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# THE IOWA ORTHOPAEDIC JOURNAL

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# IOWA ORTHOPAEDIC JOURNAL

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Any article relevant to orthopaedic surgery, orthopaedic science and the teaching of either will be considered 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. We request that all illustrations be 5 × 7 inch black and white glossy prints for reproduction purposes. The journal will be published annually in May or June. The deadline for receipt of articles for the 1985 journal is January 1, 1985.



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# The Iowa Orthopaedic Journal



This journal has as its primary purpose education. Those who participate in the production of each volume will undoubtedly learn the most. The challenges of such a task include the critical and constructive review of articles, organization and editing of the journal, and the production and distribution of the "final product." The residents in the department have been given the opportunity and responsibility to participate in and accomplish these goals.

We intend to publish one volume annually and hope that this journal will reflect the activity of residents, faculty, alumni, and visitors to our department. This journal should be the vehicle for the dissemination of thought. As such, we will include historical and "philosophic" articles as well as scientific and general review articles. This will provide a forum somewhat different from most periodicals now extant.

We thank the entire faculty for their help, and in particular, Drs. Pontarelli, Clark, Cooper and Weinstein for their support. We thank Jeanette Marsh, Kay Redlinger, and Cheryl Reyhons for their secretarial help, as well as Dale Clark and Sandra Bredman for their administrative assistance. We appreciate the cooperation of the authors and also the advertisers, who provided the necessary financial support. Our departmental creative ventures, such as this journal, are facilitated by outstanding administrative support from University Hospitals and Clinics, the College of Medicine, and the University of Iowa.

We hope that you, the reader, benefit from our efforts, and we welcome your response and constructive criticism.

FGM

NMN

## IGNACIO PONSETI

Nina M. Njus, M.D.  
Stuart L. Weinstein, M.D.  
Joseph A. Buckwalter, M.D.

As we publish Volume 4 of the Iowa Orthopaedic Journal, it is appropriate that we honor Dr. Ignacio V. Ponseti (Fig. 1). In 1984 he will complete forty years as a member of the University of Iowa Department of Orthopaedics, participating in its growth and assisting in its development. His contributions, through clinical work, teaching and research, have gained him world wide recognition and respect as a leader in his field and have helped Iowa to become a nationally and internationally recognized center of orthopaedics. He has treated thousands of patients, trained hundreds of residents and published over 100 papers, many of which are considered "classics" in the field. Yet in reflecting upon this man's exceptionally active and productive career, it is not these accomplishments which immediately come to mind, but the motivating force behind them: Dr. Ponseti's concern for the well being of his patients and his desire to provide them the best possible care. Many of his residents can recall sitting in an Indications Conference where this quiet man sat in the front row, listened to a case presentation, and commented that "we must do something to help this unfortunate child". Dr. Ponseti's concern for his patients underlies his achievements and his educational philosophy, which stresses understanding the lesions resulting in orthopaedic problems, the natural history of diseases and the critical evaluation of proposed treatment and results.

Dr. Ponseti was born in Cuidadela, a town in the Balearic Islands about 100 miles off the eastern coast of Spain. His formal education began at the Institute Balmes in Barcelona at the age of eleven. In 1930 he entered the University of Barcelona where he obtained his B.S. in biology in 1932, and his M.D. degree in 1936. During this time Dr. Ponseti studied with a number of "knowledgeable and inspiring teachers" including Dr. Joseph Trueta, at that time an Associate Professor in the Department of Surgery.

In 1936, the Spanish Civil War began and, just two days after graduating from medical school, Dr. Ponseti joined the Spanish Army. He worked as part of a team under the direction of Dr. Vidal, who had just returned from Vienna where he had studied with Lorenc Böhler for several years. It was at this time that Dr. Ponseti received his training in the Böhler school of fracture treatment which was "by and large very conservative with emphasis in good manual reductions, impeccable plaster cast techniques, as

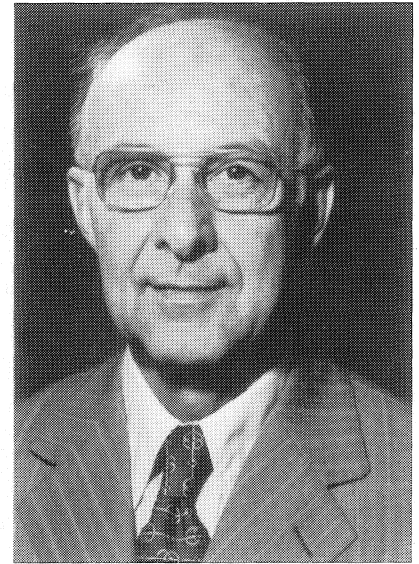


Fig. 1.  
A portrait of  
Dr. Ignacio V. Ponseti  
in 1983.

well as traction techniques for the treatment of fractures of the femur". Wounds were treated as described by Winnet Orr at the end of the First World War with extensive debridement and open treatment. Never attempting internal fixation of open fractures, the injured extremity was completely immobilized in a plaster cast. Despite the lack of antibiotics, the results of treatment were excellent. These methods of treatment were practiced almost exclusively throughout World War II. During the last year of the war Dr. Ponseti worked under Dr. Ley, a neurosurgeon who had trained with Paul Bucy at the University of Chicago. Under Dr. Ley, Dr. Ponseti repaired many peripheral nerve injuries, did tendon transfers and other reconstructive procedures, and became acquainted with the meticulous surgical technique developed by Cushing, Bucy and other neurosurgeons from the United States.

After the Spanish Civil War, Dr. Ponseti immigrated to Mexico where he practiced privately from 1939 to 1941. In June, 1941, he arrived in Iowa City to study with Dr. Arthur Steindler as a graduate student (Fig. 2). Upon completion of that year he was accepted by Dr. Steindler in a three year residency program, a rigorous and intensive training period (Figs. 3 and 4). (Dr. Ponseti has been heard to comment that "residents these days don't know what it is to be a resident", and that when he was training the title of resident was applied to the physician in training because he "resided at the hospital".) After completing his resi-



graduate students. This full year is recognized by the American Board of Orthopedic Surgeons as part of their training requirement. The lectures are given through the entire year, both semesters, and are as follows:

- Orthopedic surgery and allied subjects, 3 hrs. a week.
- Kinesiology ----- 2 hrs. a week.
- Biochemistry ----- 1 hr. a week.
- X-ray ----- 1 hr. a week.
- Anatomy ----- 3 hrs. a week.
- including lectures and cadaver surgery.
- Pathology ----- 1 hr. a week.
- Operative conference ----- 1 hr. a week.
- Literature seminars ----- 2 hrs. a week.

All graduate students are registered with the Graduate College, where they pay their tuition of about \$100 a year. Several of the students work for a degree, in which case they must write a thesis and do some extra studying in addition to the work already outlined. In general, we try not to encourage taking an advanced degree of Master of Science, because it adds at least ten more working hours a week to the already heavy burden of the graduates.

In addition to the didactic hours, the graduates take part in clinical conferences in which new patients are demonstrated or problems discussed. Special case conferences are also held by special appointment. In addition to this, the graduate has the full opportunity to familiarize himself with the work in the brace shop and the work in the physical therapy department.

After finishing his training, the graduate receives a certificate.

Fig. 2. A copy of the training requirements for graduate students in 1942.



Fig. 3. The staff and residents of the Iowa Department of Orthopaedics in 1942. This photograph was sent by Dr. William Boettcher, an Iowa graduate, in 1981. Dr. Boettcher currently practices in Seattle, Washington, where he encountered "a woman who had a club-foot deformity treated at Children's Hospital in 1942. She has had a good result from the treatment, so therefore, I assume that the casts were applied by the gentleman third from the left standing."



Fig. 4. Arthur Steindler, at right with residents and visitors at a teaching conference. Dr. Ponseti is seated on Steindler's right.

dency, Dr. Ponseti was invited to join the staff of the Orthopaedic Department at Iowa, which he did in 1944.

During his early years in Iowa City, Dr. Ponseti spent much of his free time studying bone pathology. He spent many hours studying the department's extensive collection of bone pathology started by Dr. Ernst Freund and continued by Dr. Vernon Luck. He was placed in charge of the collection and of teaching bone pathology to residents until Dr. Michael Bonfiglio arrived from the University of Chicago in 1950. He also spent many hours reviewing the "very extensive" files of the Department of Orthopaedic Surgery, and became interested in the natural history of congenital and developmental skeletal disorders and how these conditions were modified by the orthopaedic treatment given at the time.

Dr. Ponseti's primary interests have been in congenital and developmental bone and joint disorders, skeletal growth disorders in children and biochemistry of cartilage. In 1983, speaking at the International Symposium honoring Dr. Ignacio V. Ponseti, Dr. Ruth Wynne-Davies summarized Dr. Ponseti's accomplishments in these areas visually (Fig. 5), comparing his endeavors over the past twenty-five years with her own. In the early 1950's, Dr. Ponseti's pioneering work on the effect of aminonitriles on collagen cross linking laid the foundation for much of present day connective tissue biochemistry and matrix biology. For this work he received the coveted Kappa Delta Award for Outstanding Orthopaedic Research Work in 1955. He was one of the first to describe the pathologic lesion in Legg-Perthes disease. In addition, he described the biochemistry of the normal physal plate and iliac apophyseal cartilage, as well as the growth plate biochemistry, histology, histochemistry and ultrastructure in multiple skeletal dysplasias.

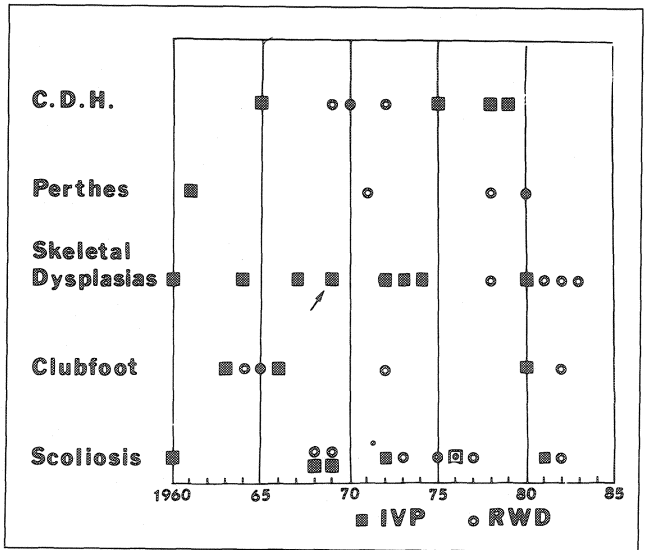


Fig. 5. A graphic summary of the accomplishments of Dr. Ponseti (squares) and Dr. Ruth Wynne-Davies (circles), comparing their endeavors over the past twenty-five years.

In the 1950's Dr. Ponseti developed a method of treatment of clubfoot deformity (Fig. 6). He recently reported on the long-term results of this method of treatment. His radiographic and anatomic studies of clubfoot have helped to define the basic nature of the deformity, and to identify those components of the deformity which are embryonic versus those which result from treatment. His studies of the normal growth and development of the acetabulum, the anatomy, histology and histochemistry of the acetabulum in congenital dysplasia of the hip, and the clinical course of congenital hip dysplasia have improved our understanding not only of the obstacles to reduction in the congenitally dislocated hip, but also of the fundamental nature of the disorder.

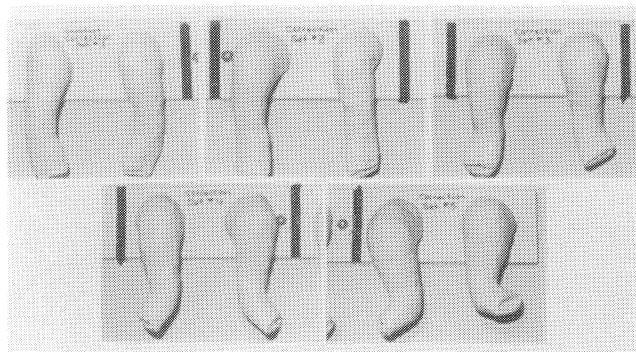


Fig. 6. Models of casts applied to correct a club-foot deformity, illustrating Dr. Ponseti's method of closed treatment.

Dr. Ponseti defined the curvature patterns in idiopathic scoliosis and was the first to demonstrate that curves progressed after skeletal maturity. His work on the natural history of this disorder demonstrated the importance of curve pattern in treatment selection. His studies of the biochemical abnormalities of the intervertebral disc and vertebral end plates in scoliosis have brought us closer to understanding the fundamental nature of this disorder.

Dr Ponseti has served as an Assistant Editor for the *Journal of Bone and Joint Surgery*, as an Examiner for the American Board of Orthopaedic Surgery and as past President of the Orthopaedic Research Society. He has been Visiting Professor at nearly 100 institutions, was awarded the Hektoem Gold Medal from the American Medical Association in 1960, and in 1975 was selected by the American Academy of Orthopaedic Surgeons to deliver the prestigious Alfred Shands Lecture. In 1979 he was selected by the British Orthopaedic Association as their guest lecturer. In 1983 the International Symposium Honoring Dr. Ignacio V. Ponseti was held in Iowa City (Figs. 7, 8, 9, 10, and 11). The guest faculty included Dr. Anthony Catterall, Dr. Sherman Coleman, Prof. J.I.P. James, Dr. Henry Mankin, Prof. Alf Nachemson and Dr. Ruth Wynne-Davies. Each of the renowned figures felt it was an honor



Fig. 7. The honoree listens to a lecture at the International Symposium honoring Dr. Ponseti.



Fig. 8. Dr. Ponseti with Dr. Henry Mankin to the right.



Fig. 9. Dr. Ponseti and Dr. Joseph Buckwalter at the dinner party after the International Symposium honoring Dr. Ponseti.

to be present, and a pleasure to see their friend and colleague, Dr. Ponseti. Finally, Dr. Ponseti was awarded an honorary doctoral degree from the University of Barcelona in 1984.



Fig. 10. The head table, Dr. and Mrs. Ponseti are seated on the far side of the table. Dr. Cooper is standing.

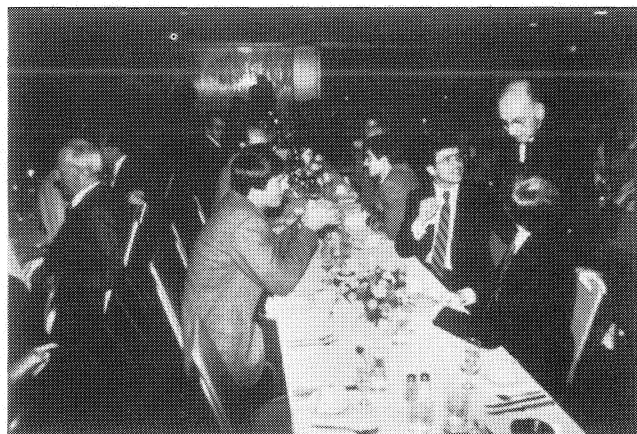


Fig. 11. Dr. Ponseti chats with some former residents.

Behind this impressive list of achievements is an incredibly gentle and compassionate man who enjoys music, art and especially nature (Fig. 12). Dr. Ponseti's interest in the arts developed when he was a student living in Barcelona, where he met many of the great personalities destined to bring about a major revolution in the arts throughout the world. Pablo Casals and other prominent musicians gave concerts. Picasso, Dimona and Miró were among the many excellent painters and sculptors exhibiting their works in Barcelona. Authors such as Garcia Lorca presented their works to enthusiastic audiences, among whom was Ignacio Ponseti, the university student. Dr. Ponseti joined the Catalonia Hiking Club and on weekends would travel to the Pyrenees to "hike through the beautiful

valleys and up the main mountains separating Spain from France". He found these expeditions "most relaxing and rewarding" and still finds hiking one of his most fulfilling hobbies.

Throughout his forty-three years at the University of Iowa, Dr. Ponseti has been a constant inspiration to his colleagues, a role model for countless numbers of students, residents and fellows, and a source of comfort for his patients.

We who continue to work and study in the Orthopaedic Department at the University of Iowa after his retirement will truly miss his gentle manner, his skilled assistance and his thoughtful guidance, and we will miss hearing that deep gentle voice with its Spanish accent say, as he adeptly applies a cast on a child, "oh, such a nice bee bee".



Fig. 12. Dr. Ponseti on an ocean cruise.



# PROGNOSIS IN IDIOPATHIC SCOLIOSIS\*

Ignacio V. Ponseti, M.D., and Barry Friedman, M.D.

The conservative attitude of this Clinic in the treatment of scoliosis has made it possible to accumulate a great number of cases of idiopathic scoliosis with prolonged follow-up studies, treated without surgery. These cases have been studied with the object of establishing criteria for determining the prognosis in idiopathic scoliosis.

Before an operation is undertaken in a young patient with scoliosis, the surgeon should have a thorough knowledge of the natural course of the curve he is going to treat and the probable outcome. The problem is so complex and so many abstract theoretical studies have been made that the orthopaedic surgeon is prone to overrate the dangers involved in scoliosis and to carry out surgical fusion of the spine which is often unnecessary. In many cases of idiopathic scoliosis, there is a natural tendency for the curve to stop progressing after a certain growth has been reached. Furthermore, the body posture is often well maintained in spite of sizable spinal curvatures. On the other hand, extremely deforming thoracic curves, requiring early and active treatment, may develop over a short period of time. It is the purpose of this paper to outline the course of the most common patterns of idiopathic curvature of the spine, and to give some basis for prognosis with any given curve.

## Material

In a group of 444 young patients with idiopathic scoliosis, spine fusion had been performed in fifty cases. This study is based on an analysis of the 394 cases of idiopathic scoliosis which were not treated surgically. Of these, 335 cases were observed through maturity. Roentgenograms taken with the patient in the standing and supine positions, photographs, and clinical examination were employed in the study. Cases with minimal curves were not included. The average follow-up period was two years and ten months.

A great number of patients in this series received conservative treatment<sup>3</sup>, consisting of exercises designed to increase muscle strength and to correct postural imbalance. Tightness of the abdominal muscles at the side of the overhang was corrected by passive and active stretching. The patients were taught to shift their thorax into

proper alignment with the pelvis. Braces were often given to help maintain body posture. It is not our intention to evaluate the results of conservative treatment in this paper. While conservative therapy may improve body posture, it has never been found—or claimed—to decrease the size of a spinal curvature. As this study is based upon the characteristics of the curves, the fact that most patients received conservative treatment did not influence the results of our observations.

## Measurement of Curves

The method used for determining the angle of a curve is as follows: The top and bottom vertebrae of the curve are identified by the width of the intervertebral space, the tilting, and the degree of rotation of the vertebrae. The intervertebral spaces are wider on the side of the convexity and narrower on the concave side. At the top and bottom of the curve, the intervertebral space is either of equal width or is wider toward the concavity of the curve. The rotation of a vertebra is indicated on the roentgenograms by the relationship of the spinous process to the body of the vertebra. The degree of rotation is greatest at the apex of the curve and diminishes toward each end, where a neutral vertebra, that is, a non-rotated vertebra, is frequently found. The top and bottom vertebrae are usually the ones which rotate the least in the curve, but an exception should be made for the cases with curves showing concave rotation. Lines are drawn parallel to the upper surface of the top vertebra and to the undersurface of the bottom vertebra. The angle formed by these lines is the angle of the curve.

## Curve Patterns

In idiopathic scoliosis, most of the characteristics of the curve or curves are present from the onset of the deformity and do not change throughout its entire course. The size of the curvature may increase considerably, and in some cases one or two more vertebrae may be included in the curve in the late stages. However, the apex, the direction of rotation, and the location of the curve do not change. Roentgenograms taken shortly after the onset, when the curvature is still minimal, point almost unmistakably to the pattern of the scoliosis (Figs. 4-A to 4-C and 7-A to 7-D). Only in exceptional cases does a change in pattern occur during the development of the scoliosis.

\*Reprinted from JOURNAL ON BONE AND JOINT SURGERY Vol. 32.-A. No. 2 pp. 381-385, April, 1950. Copyrighted by *The American Orthopaedic Association, Inc.*



Fig. 1-A

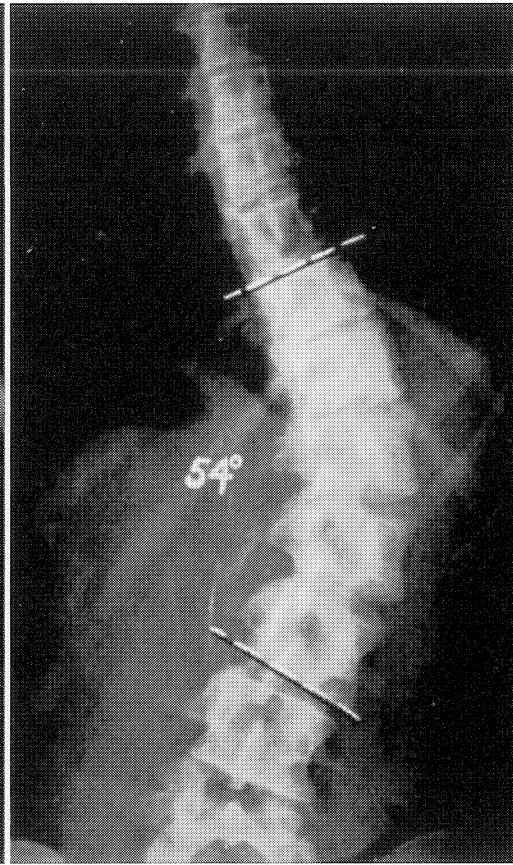


Fig. 1-B

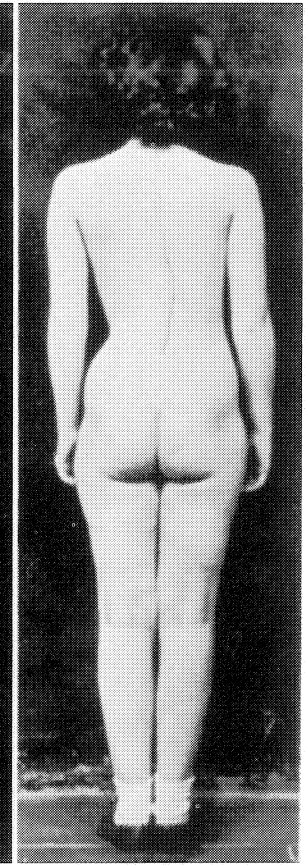


Fig. 1-C

Fig. 1-A: Roentgenogram\* showing lumbar curve in a thirteen-year-old girl. Scoliosis had first been noticed at the age of twelve. Apex is at first lumbar vertebra. Curve extends from eleventh thoracic to third lumbar.

Fig. 1-B: At the age of seventeen years, there has been only minimal increase of the curve. Lumbar curves usually increase only slightly.

Fig. 1-C: Photograph of patient at seventeen years of age. Body posture was good.

It was possible to group all the cases of idiopathic scoliosis studied into five main patterns: main lumbar, main thoracolumbar, combined thoracic and lumbar, main thoracic, and main cervicothoracic. In Table I the 394 cases are classified according to the curve patterns. These groupings correspond quite closely to the ones described by Schulthess in 1905.

The course and prognosis in idiopathic scoliosis vary considerably from pattern to pattern. For this reason, each group will be considered in detail.

#### Age at Onset

The final angular value of a curve is usually correlated with the age at which the scoliosis begins. However, it is often difficult to determine just when the curvature starts. In children who are well cared for, the scoliosis may be detected shortly after its onset; in other children, the curve is often well advanced before it is noticed. The age

of the patient at the time the curve is first noticed is used. In Table II, the cases observed until maturity were grouped according to the age of the patient when the scoliosis was first noticed. In each of the five patterns, four groups were made, according to whether the scoliosis was first noticed: (a) before the age of ten years; (b) between ten and twelve years; (c) between twelve and fourteen years; and (d) after fourteen years of age.

In each age group, the curves were classified according to their final angular values at maturity: (a) curves over 80 degrees at maturity (very severe); (b) curves between 60 and 80 degrees (severe); (c) curves between 40 and 60 degrees (moderate); and (d) curves under 40 degrees (small).

#### Curve Patterns

##### Main Lumbar Curves

Single idiopathic lumbar curves occurred in ninety-three of the 391 cases studied (23.6 per cent.). They proved to be the most benign of all types of idiopathic curves. Eighty

\*All the roentgenograms show the left side of the patient at the left side of the page. All were taken with patient in standing position and are anteroposterior views.

Table 1

Curve Pattern	No. of Cases	Sex		Average Age						Average Degrees					
				When Curve First Noticed		At First Visit		At Stabilization		Convexity to		At First Visit		At Stabilization	
				M.	F.	Yrs.	Mo.	Yrs.	Mo.	Yrs.	Mo.	Rt.	Lt.	St.	Sup.
Lumbar	93 (23.6%)	13	80	13	4	14	1	14	10	28	65	33.9	27.7	36.8	29.1
Thoracolumbar	63 (15.9%)	14	49	13	11	14		15	11	51	12	36.3	31.6	42.7	35.0
Combined															
Thoracic	146 (37.1%)	11	135	12	4	13	1	15	5	136	10	44.0	39.8	51.9	41.4
Lumbar										10	136	35.1	30.1	41.4	37.7
Thoracic	87 (22.1%)	25	62	11	1	12	10	16	1	79	8	60.2	53.7	81.4	73.8
Cervicothoracic	5 (1.3%)	1	4	15	2	16		16		1	4	32.6	30.0	34.6	32.2
Totals	394 (100%)	64	330							295	99				

Rt. = right.  
St. = standing.  
Lt. = left.  
Sup. = supine.

of the patients were females, thirteen were males. The average age at which the curvature was first noticed was thirteen years and four months. Lumbar curves became stabilized earlier than other patterns, at an average of fourteen years and ten months. Since vertebral growth in this group of cases was not complete until an average of sixteen years and eleven months, as evidenced by the completion of ossification excursion of the iliac apophyses<sup>1</sup>, it may be assumed that it is not necessary for the growth of the spine to be terminated before the lumber type of scoliosis is arrested.

The lumbar curves generally included five vertebrae and extended from the eleventh thoracic to the third lumbar (Figs. 1-A, 1-B, and 1-C). The apex of the curve was at the first or second lumbar vertebra. They occurred predominantly to the left; sixty-five (70 per cent.) were convex to the left and twenty-eight (30 per cent.) were convex to the right.

Counter curves above and below the main lumbar curve were small, but constant. The two or three thoracic vertebrae above the curve were usually tilted opposite to the vertebrae in the lumbar curve, to form the upper counter curve and realign the body. These vertebrae often rotated in the same direction as the vertebrae in the lumbar curve. Thus, a *concave* rotation in the small thoracic counter curve was present.

The lower counter curve was formed by the fourth and fifth lumbar vertebrae. These two vertebrae were also tilted in the opposite direction to the lumbar curve, so that

the undersurface of the fifth lumbar vertebra was horizontal and the sacrum was then neutral, or out of the curve. This was found in all but three of the ninety-three lumbar curves. In these three cases, the lumbar curve was lower, extending from the twelfth thoracic to the fifth lumbar, with the apex at the third lumbar vertebra. Here, the sacrum formed the counter curve.

Single lumbar curves were the least deforming of all the patterns. The gait, with the alternating rotation and oscillation of the pelvis, kept the lumbar spine limber, and extreme degrees of deformity were not seen. The average angular value after the curve had become stationary was 36.8 degrees with the patient standing, and 29.1 degrees supine. In only seven cases (8 per cent.) was the angular value of the curve over 60 degrees; but even in the most severe degrees of angulation, the curve was not very deforming.

Eighty-eight patients with lumbar scoliosis were observed until after they had reached maturity (Table II). In nine cases (10.2 per cent.), the curve was first noticed before the age of ten. In spite of the early onset, in only one case did the curve increase beyond 60 degrees. In twelve cases, the scoliosis was first noticed between the ages of ten and twelve years; in two of these cases, the curve progressed beyond 60 degrees. Of the sixty-seven cases in which the scoliosis was first noticed after the age of twelve, only four had curves greater than 60 degrees, the highest value being 67 degrees. In no case, regardless of the age at which the curve was first noticed, was clinically severe deformity observed.

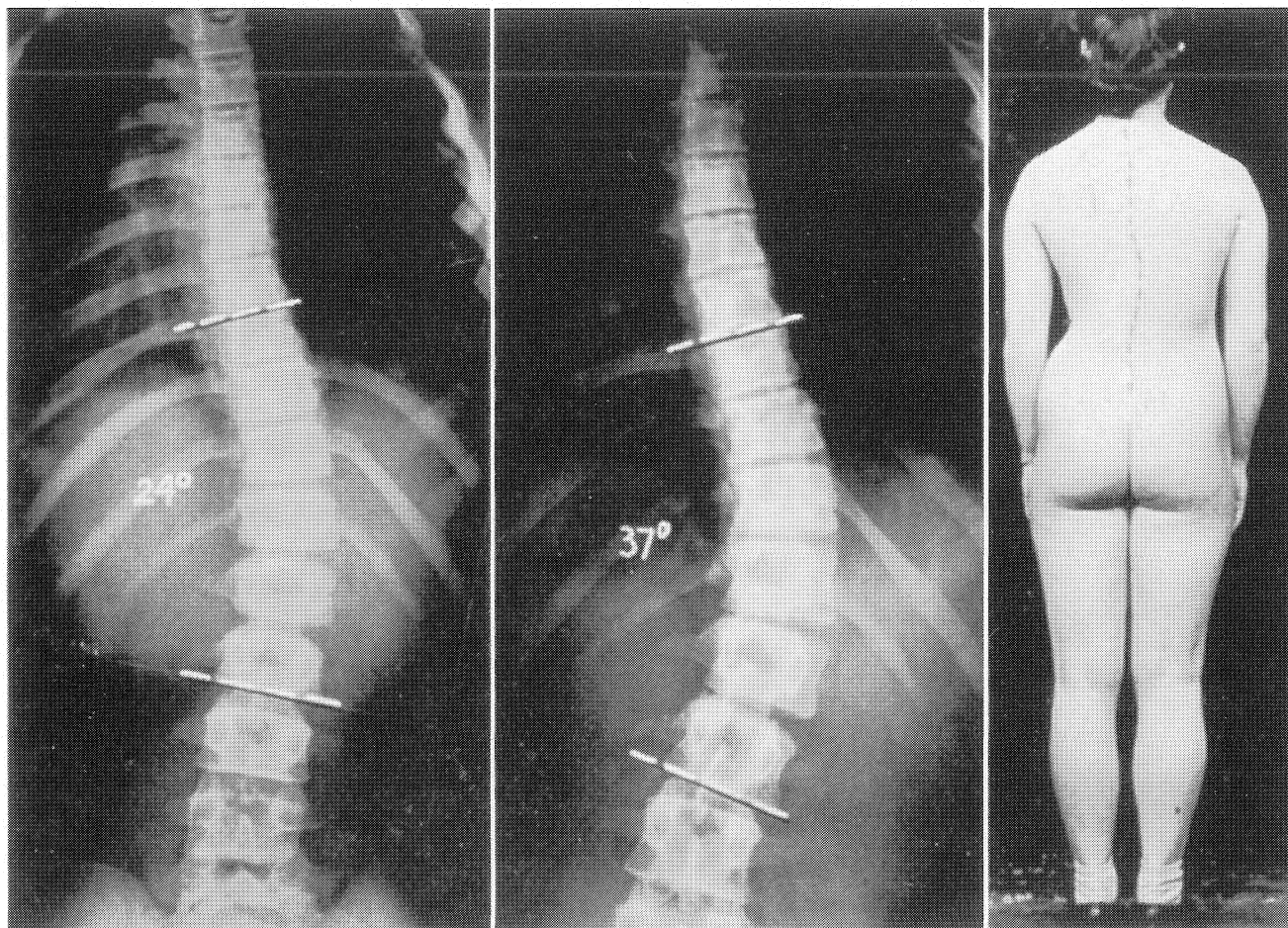


Table 2

Course of idiopathic scoliosis in 335 cases, correlated with pattern and age of patient at onset of curve

Curve Pattern	Age of Patient When Curve First Noted (Years)	No. of Patients	Angle of Curve at Maturity No. of Patients with Curves Measuring (Degrees):			
			Under 40	40 to 60	60 to 80	Over 80
<b>Main Lumbar</b> Apex: 1st or 2nd lumbar Extent of curve: 11th thoracic to 3rd lumbar	Under 10 From 10 to 12 From 12 to 14 Over 14	9 12 34 33	4 6 22 20	4 4 10 11	0 1 2 2	1 1 0 0
Total .....		88	52 (59%)	29 (33%)	5 (6%)	2 (2%)
<b>Thoracolumbar</b> Apex: 11th or 12th thoracic Extent of curve: 6th or 7th thoracic to 1st or 2nd lumbar	Under 10 From 10 to 12 From 12 to 14 Over 14	3 3 22 26	1 1 7 18	0 0 8 4	0 1 7 4	2 1 0 0
Total .....		54	27 (50%)	12 (22%)	12 (22%)	3 (6%)
<b>Combined</b> <i>Thoracic</i> Apex: 7th or 8th thoracic Extent of curve: 6th to 10th thoracic <i>Lumbar</i> Apex: 2nd lumbar Extent: 11th thoracic to 4th lumbar	Under 10 From 10 to 12 From 12 to 14 Over 14	14 28 45 30	T. 0 L. 1 T. 3 L. 10 T. 12 L. 21 T. 13 L. 26	3 4 10 16 23 18 17 4	4 3 14 2 6 6 0 0	7 6 1 0 4 0 0 0
Total .....		117	T. 28 (24%) L. 58 (50%)	53 (45%) 42 (36%)	24 (21%) 11 (9%)	12 (10%) 6 (5%)
<b>Main Thoracic</b> Apex: 8th or 9th thoracic Extent of curve: 5th or 6th thoracic to 11th or 12th thoracic	Under 10 From 10 to 12 From 12 to 14 Over 14	18 16 21 16	0 0 1 4	1 1 8 3	4 4 5 5	13 11 7 4
Total .....		71	5 (7%)	13 (18%)	18 (25%)	35 (50%)
<b>Cervicothoracic</b> Apex: 3rd thoracic Extent of curve: 7th cervical or 1st thoracic to 4th or 5th thoracic	Under 10 From 10 to 12 From 12 to 14 Over 14	0 0 1 4	0 0 0 3	0 0 1 1	0 0 0 0	0 0 0 0
Total .....		5	3	2	0	0

T. = Thoracic. L. = Lumbar.



47 Fig. 2-A

Fig. 2-B

Fig. 2-C

Fig. 2-A: Thoracolumbar curve with apex at the twelfth thoracic vertebra in a ten-year-old girl. Scoliosis was first noticed at six years of age. Curve extends from the ninth thoracic to second lumbar vertebra.

Fig. 2-B: In spite of its early onset, curve had increased only slightly by the time the girl was seventeen years of age.

Fig. 2-C: Photograph of patient at seventeen years of age. Posture was good.

Minimal lumbar curves are quite common and were not included in these statistics. They are often not discovered until roentgenograms are taken to investigate the cause of low-back pain in adult life.

### Thoracolumbar Curves

Thoracolumbar patterns occurred in sixty-three cases, or 16 per cent. of the total number of idiopathic curves studied. Of these, forty-nine were females, fourteen were males. The thoracolumbar curve had a great tendency to be convex toward the right, as did thoracic curves. Fifty-one or 80.9 per cent. were convex to the right, and twelve or 19.1 per cent. were convex to the left.

Six to eight vertebrae were included in the thoracolumbar curve, which extended from the sixth or seventh thoracic vertebra to the first or second lumbar. The apex was at the eleventh or twelfth thoracic; it was at the twelfth thoracic vertebra in forty cases and at the eleventh thoracic in twenty-three cases (Figs. 2-A to 3-C).

Although there proved to be no major differences in their eventual outcome, notable differences in the characteristics of these curves were found, depending upon the location of the apex. The curves with an apex at the eleventh thoracic vertebra had a tendency to grow larger and to penetrate into the thorax more than did the curves with an apex at the twelfth thoracic vertebra. When the apex was at the eleventh thoracic, the counter curves above and below the main curve were usually well developed and the vertebrae rotated toward the convexity of each curve (Figs. 3-A, 3-B, and 3-C). The sum of the angular values of the counter curves usually equaled the value of the main curve. The counter curves in the thoracolumbar scoliosis with an apex at the twelfth thoracic were very small or even non-existent. Usually the entire spine rotated toward the convexity of the main curve, and, if the counter curves existed, they showed a slight degree of concave rotation (Figs. 2-A, 2-B, and 2-C).

The thoracolumbar scoliosis started late. The average

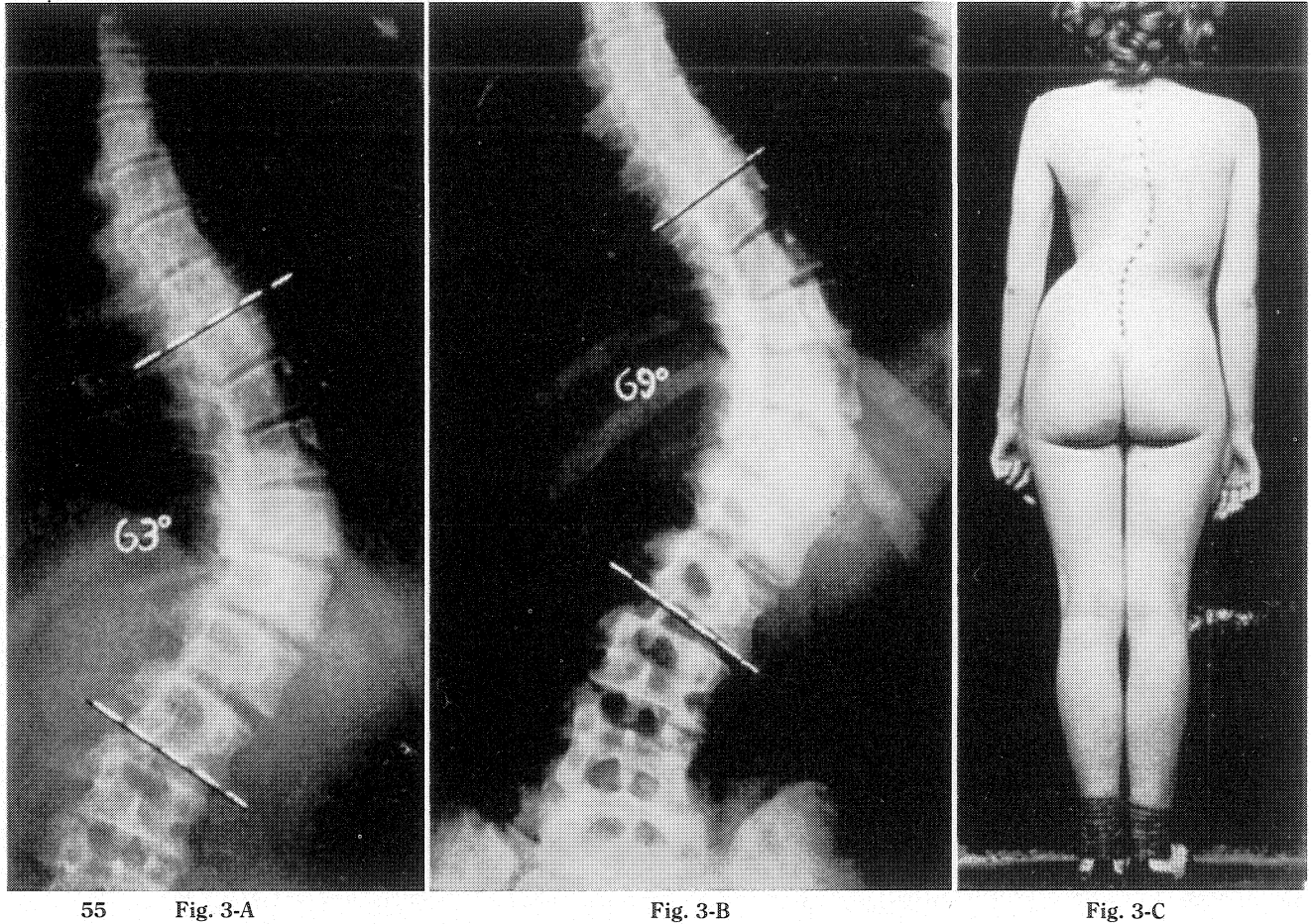


Fig. 3-A: Thoracolumbar curve with apex at eleventh thoracic vertebra in a fourteen-year-old girl. Curvature was first noticed at eleven years. Extent of curve is from eighth thoracic to second lumbar vertebra.  
 Fig. 3-B: Roentgenogram of same patient at seventeen years of age. The course is typical for curves of this pattern.  
 Fig. 3-C: Photograph of patient at seventeen years of age. In spite of sizable curvature, the general body alignment is not greatly disturbed.

age at which the curve was first noticed was fourteen years. Fifty-four patients with thoracolumbar scoliosis were observed until they reached maturity. Spontaneous stabilization of the curve occurred at an average age of sixteen years, whereas the ossification excursion of the iliac apophysis of the patients in this group was not completed until an average age of seventeen years and four months. The average angular value after the curve had become stationary was 42.7 degrees with the patient standing, and 35.0 degrees when supine.

In six cases, the scoliosis was first noticed before the age of twelve years (Table II). In three cases of this group, the angle of the curve progressed beyond 80 degrees; in one case it was 67 degrees. The curve was small in the other two cases. In none of forty-eight cases in which the scoliosis was first noticed after the age of twelve years, did the curve reach 80 degrees; eleven had curves of from 60 to 80 degrees, and in twenty-five the curve did not increase beyond 40 degrees.

The thoracolumbar curves were, as a whole, not very deforming. The lower thoracic and the lumbar segments remained limber, and a good body posture was maintained in all except eight cases. In only two of these was a major loss of body alignment observed. The scoliosis appeared at a very early age in both cases.

#### Combined Thoracic and Lumbar Curves

Combined scoliosis was the most frequent form in this series, with an incidence of 37 per cent. Of the 146 cases, 135 were girls and eleven were boys.

The cases of this group had two main recognizable curves from the very onset. One curve was thoracic, usually extended over a distance of five vertebrae from the sixth to the tenth thoracic vertebra, and had an apex at the seventh or eighth thoracic level. The other curve of opposite direction was lumbar, with an apex at the second lumbar, and included five vertebrae from the eleventh thoracic to the fourth lumbar. A few cases with six or



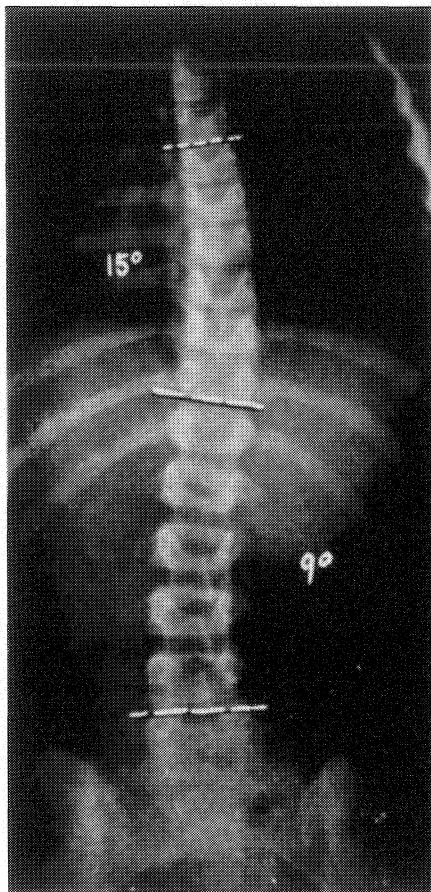


Fig. 4-A

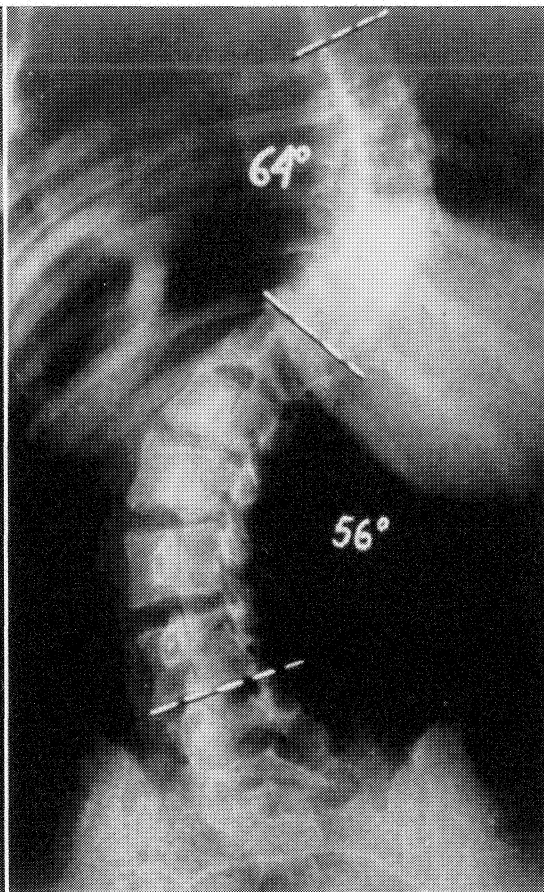


Fig. 4-B

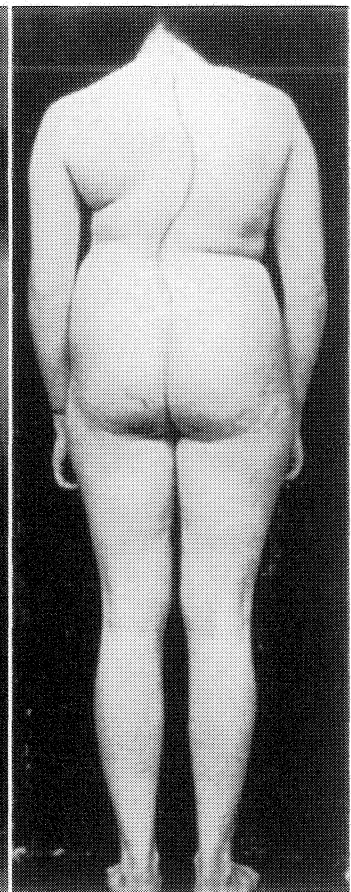


Fig. 4-C

Fig. 4-A: Combined thoracic and lumbar scoliosis in a nine-year-old girl. Roentgenogram was taken only a few weeks after curvature was first noticed, but pattern was already well defined.

Fig. 4-B: Roentgenogram of same patient at sixteen years of age, showing great increase of the curves. Increase of each curve was proportionate.

Fig. 4-C: Photograph of patient at sixteen years of age. In spite of size of the curvature, body remained well aligned.

seven vertebrae in each curve were seen. A neutral or non-rotated vertebra was often present between the two curves. The convexity of the thoracic curve was predominantly to the right and that of the lumbar curve was most commonly to the left. In the 146 cases, all but ten were right-thoracic, left-lumbar combinations (Figs. 4-A to 6-B).

Usually both the thoracic and lumbar curves had the same number of vertebrae, and the rotation in each was toward the convexity of the respective curve. The degree of rotation of the vertebrae of one of the curves matched that of the other curve from the very beginning of the scoliosis. The thoracic curve usually measured several degrees more than the lumbar curve. The vertebral rotation and the angular value of both curves increased at about the same rate, so that the basic pattern changed very little. In this series, only rare instances were seen where one curve grew more than the other.

In forty cases (27 per cent.), the apex of the thoracic curve was lower than the seventh or eighth thoracic ver-

tebra,—that is, at the ninth or tenth thoracic. The lumbar curve was, of necessity, short. When this scoliosis increased, an overhang of the body to the side of the convexity of the thoracic curve occurred and there was an upper cervicothoracic counter curve, opposite to the thoracic curve, which helped realign the body posture.

The average age of the patient when the scoliosis was first noticed was twelve years and four months. The average age at which the curve became stabilized was fifteen years and five months. The ossification excursion of the iliac apophysis in this group of cases was complete at an average of sixteen years and four months. One hundred and seventeen cases of the combined pattern were followed through maturity (Table II).

The prognosis in cases of combined scoliosis, in general, was good. The thoracic and lumbar curves were similar in length, rotation, and angular values. Consequently, the body was well aligned even when both curves reached considerable size. In many cases, curves of 60 or

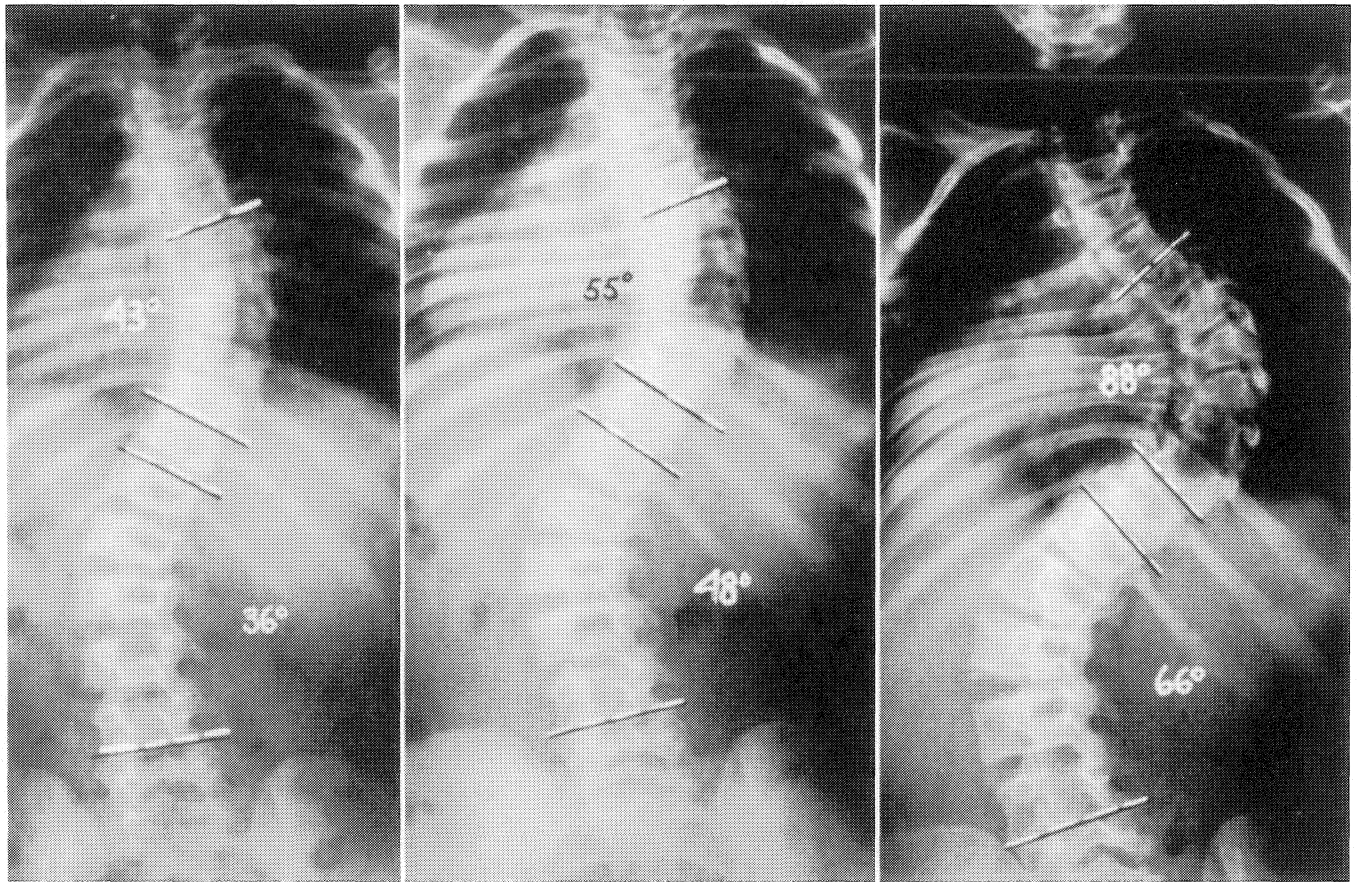


Fig. 5-A

Fig. 5-B

Fig. 5-C

Fig. 5-A: Combined thoracic and lumbar scoliosis in an eight-year-old girl. Curvature had been noticed for four months. Apex is at eighth thoracic vertebra in upper curve, and at second lumbar in lower curve. Figs. 5-B and 5-C: Roentgenograms taken at nine and eleven years show gradual increase of both curves. The curves reached great size at maturity, as is seen frequently in the combined pattern when it starts at an early age.

70 degrees (Figs. 4-A, 4-B, and 4-C) were not detectable when the patient was dressed.

The most important prognostic feature in this scoliosis pattern was the age of the patient when the scoliosis began (Table II). In seven of the fourteen cases in which the scoliosis was first noticed before the age of ten years, the curves increased to angular values of more than 80 degrees (Figs. 5-A to 5-F). This tendency of progression to extensive deformity dropped off sharply when the scoliosis began after the age of ten years (Figs. 6-A and 6-B).

Several important prognostic signs were observed in the roentgenograms (Figs. 9-A and 9-B). The thoracic vertebrae adjacent to the apex of the thoracic curve were osteoporotic and hazily outlined. The intervertebral spaces were irregularly outlined and narrow. A translatory shift was often seen, more pronounced at the transition vertebrae between the thoracic and lumbar curves. This is a sign of relaxation of the ligamentous apparatus of the spine. These roentgenographic signs were present during the progression of the curve; they were not seen in the sco-

liosis which remained stationary.

When all these features were taken into consideration, a fairly accurate prognosis of most of the combined curves could be made.

### Thoracic Curves

Main thoracic curves accounted for eighty-seven cases or 22 per cent. of the total. Sixty-two were in females, twenty-five in males. This curve was first noticed at an average age of eleven years and one month, —earlier than in any of the other patterns. The thoracic curves became stabilized at an average age of sixteen years and one month, thus allowing five years during which this curve increased. The ossification excursion of the iliac apophyses was complete at an average of seventeen years and one month.

Of the eighty-seven cases in this group, all but eight had curvatures convex to the right. Six of the eight patients with left convex thoracic scoliosis were boys.

The apex of the thoracic curve usually was at the eighth or ninth thoracic vertebra. Six vertebrae were generally

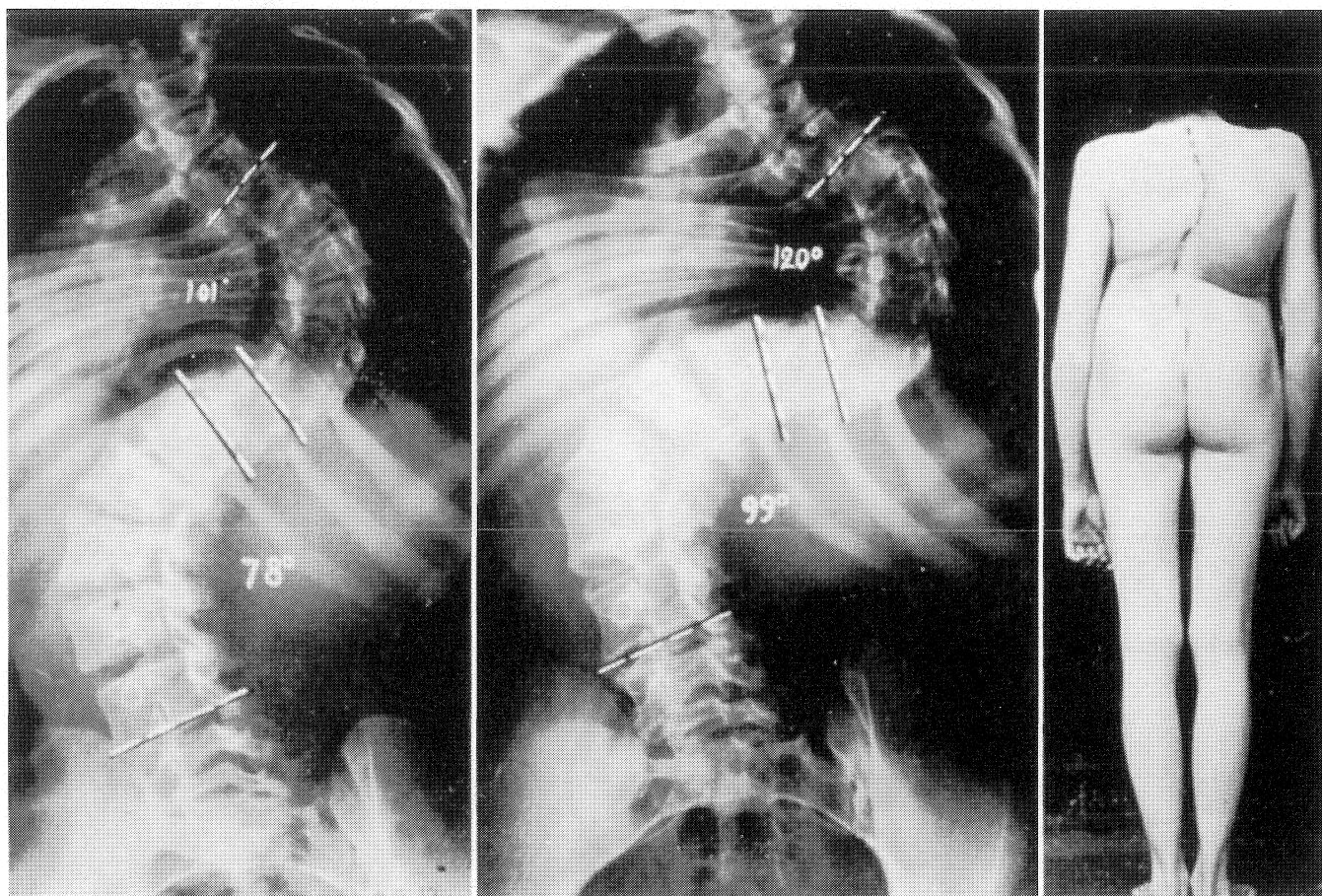


Fig. 5-D

Fig. 5-E

Fig. 5-F

Figs. 5-D and 5-E: Roentgenograms taken at twelve and fifteen years of age show further increase of the curves.  
Fig. 5-F: Photograph of patient at fifteen years of age, showing deformity.

included in the main curve, which extended from the sixth to the eleventh thoracic; extreme degrees of rotation were observed in the vertebrae. Smaller counter curves were present above and below the main curve. The rotation of the vertebrae in the counter curves was relatively mild. (Figs. 7-A to 8-C).

The roentgenograms showed significant changes in the three or four vertebrae located in the center of the main thoracic curves (Figs. 9-A and 9-B). The bodies of these vertebrae appeared osteoporotic, their outlines were hazy, and early in the evolution of the curve they showed marked wedging. The intervertebral spaces were irregularly outlined and very narrow at the site of the concavity. Often disc herniations into the vertebral body were seen. Translatory shift between the vertebral bodies was often evident. These changes were seen before and during the progression of the curve. There was a direct relationship between the intensity of these lesions and the increase of the curve. These changes were much more pronounced than, but similar to, the ones seen in the thoracic compo-

ment of the combined type of scoliosis.

Thoracic curves progressed more rapidly, grew to a greater size, and produced a greater deformity than any other type of scoliosis. The curve penetrated deeply into the thorax early in its course, producing an irreversible deformity.

When main thoracic curves started before the age of twelve years, the outlook was poor (Table II). Of thirty-four cases in which thoracic scoliosis was noticed prior to this age, in twenty-four or 70.6 per cent. the curve progressed to a final angular value greater than 80 degrees. In half of these cases, the deformity was extreme, with curves of angular values well over 100 degrees. The thoracic curve often had a tendency to progress to extensive deformity, even when it started late. About one-third of the patients whose scoliosis was detected after twelve years of age had curves measuring more than 80 degrees at maturity. However, in slightly less than half of the patients in this age group, the curves did not increase beyond 60 degrees and were not very deforming.



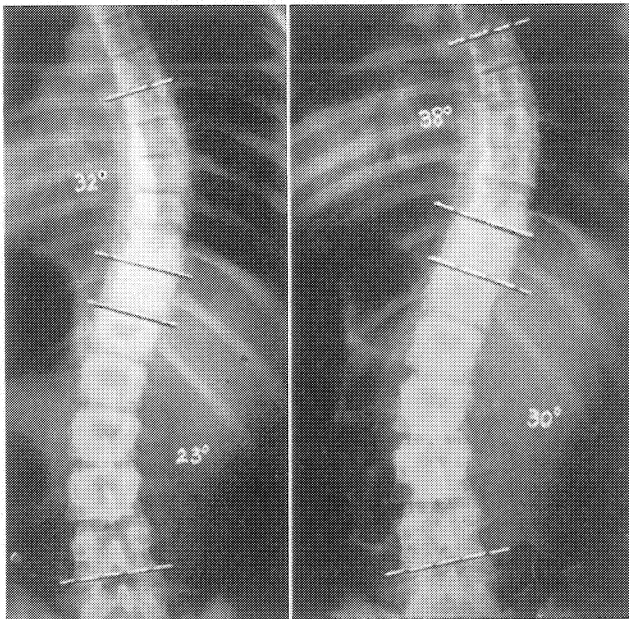


Fig. 6-A

Fig. 6-B

Fig. 6-A: Combined thoracic and lumbar scoliosis in a twelve-year-old girl. Scoliosis had been present for half a year.  
 Fig. 6-B: Same patient at seventeen years of age. Curves had increased very little. Curves of the combined pattern which start after eleven years of age usually do not increase greatly.

### Cervicothoracic Curves

In the present series there were only five cases with this type of curve. The scoliosis was first noticed when the patient was about fifteen years of age, and never became pronounced. The angular value of the curve at maturity varied from 20 to 58 degrees. The curve was convex to the left in all the cases but one. Four of the patients were girls and one was a boy.

The apex of the cervicothoracic curves was at the third thoracic vertebra. From four to six vertebrae were included in the curve, which extended from the seventh cervical or first thoracic to the fourth or fifth thoracic. The curve penetrated somewhat into the upper thorax, and its vertebrae were wedge-shaped and markedly rotated. A long, low thoracic counter curve was usually present (Figs. 10-A and 10-B).

The scapula was elevated and the shoulder was higher at the side of the convexity of the curve. In spite of these visible deformities, the body alignment was well preserved in all the cases seen by us.

### Discussion

The study of the course of idiopathic scoliosis in a sizable number of cases has given valuable data upon which to base the prognosis. Four main factors were found significant.

1. The pattern of the curve;
2. Age of the patient at onset of the scoliosis;

3. Alterations in the density of the vertebrae and abnormalities of the disc spaces, as seen in the roentgenograms;
4. Rapidity of increase in the size of the curve.

### Pattern of the Curve

The classification of idiopathic scoliosis into five main curve patterns was found to be fundamental in the determination of prognosis. These curve patterns were well established shortly after the onset of the scoliosis and, with a few exceptions, did not change throughout its course.

The pattern was easy to identify in the majority of cases. However, about 5 percent of the curves represented transition forms between two patterns, and had characteristics of each. Care was taken to classify these cases according to the pattern to which they bore closest resemblance. For example, nine cases had a main lumbar curve with a pronounced low-thoracic counter curve. These cases resembled the combined thoracolumbar patterns. However, they were classified with the lumbar curves for three reasons: First, the thoracic curve was a good deal smaller than the lumbar curve, whereas in the combined pattern, the lumbar curve is usually smaller. Second, the vertebrae of the thoracic curve were only slightly rotated, in contrast to the advanced degree of rotation usually noted in the thoracic curve of the combined pattern. Third, in the combined pattern there is a small, high thoracic or cervicothoracic counter curve, not present in these nine cases.

Transition forms were also seen between the lumbar and thoracolumbar patterns, the thoracolumbar and combined, and the combined and main thoracic. A careful study of each of these cases was necessary for their proper classification.

As a whole, the main thoracic curves increased to greater deformities than the other patterns. The prognosis in the main lumbar, thoracolumbar, and cervicothoracic curves was usually favorable. The prognosis with the combined thoracolumbar curves was usually favorable. The prognosis with the combined thoracolumbar curves was usually good if they developed after ten years of age, and poor if they started earlier.

### Age at Onset of the Scoliosis

The prognosis in cases of scoliosis depended in great measure upon the age of the patient when the curvature appeared. The most deforming curves originated at an early age. Conversely, the curves which were detected when the maturity of the patient was well advanced increased only slightly or not at all. The age of the patient when the scoliosis became apparent varied within each pattern. The main thoracic curves usually appeared at an earlier age than the other curves. The curves in the lumbar, thoracolumbar, and the cervicothoracic patterns appeared late, — often after the age of thirteen years. It



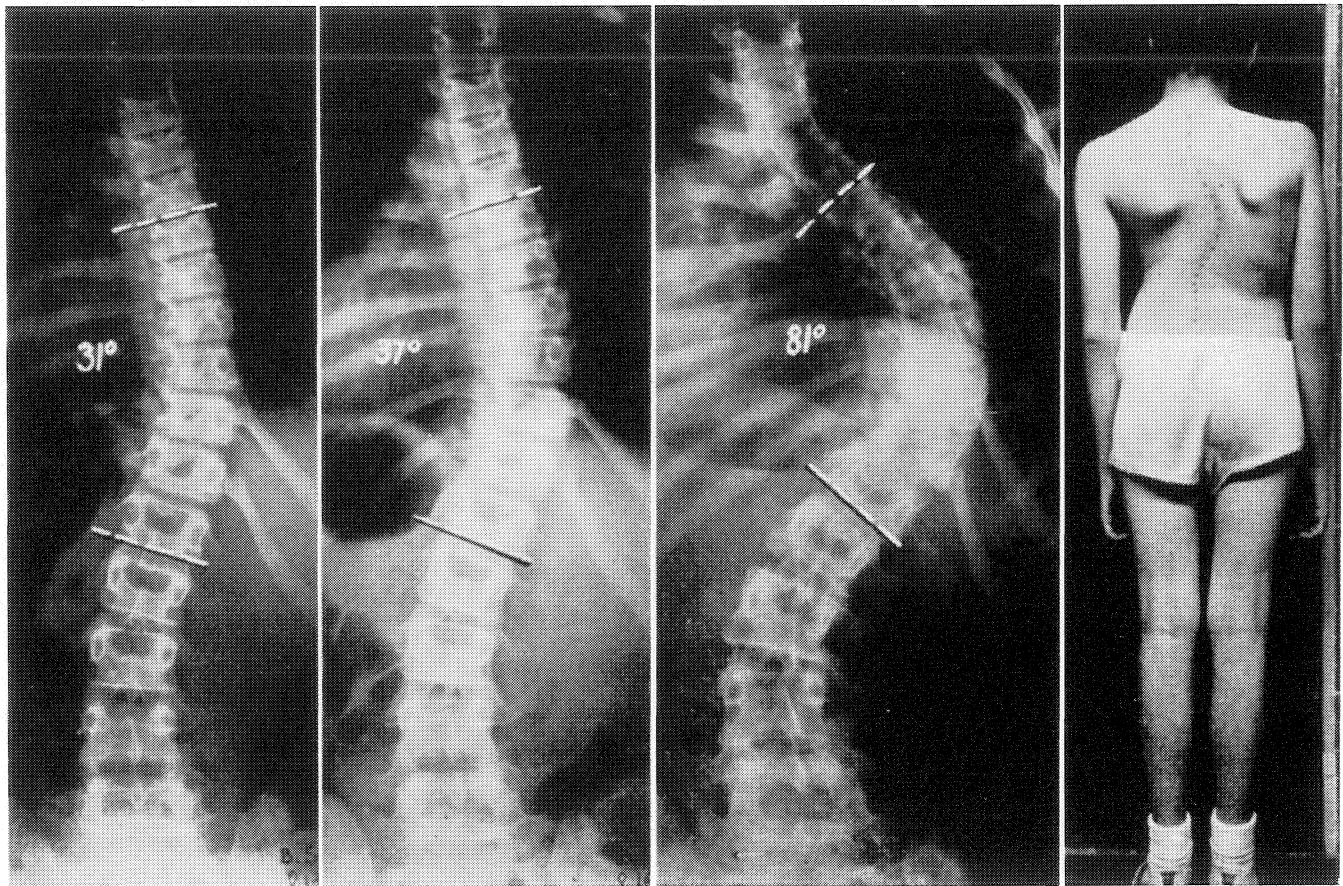


Fig. 7-A

Fig. 7-B

Fig. 7-C

Fig. 7-D

Fig. 7-A: Main thoracic curve in a thirteen-year-old girl, shortly after onset.

Figs. 7-B and 7-C: Roentgenograms taken at fourteen and seventeen years. In spite of its late onset, curve increased greatly. Cases of this pattern usually follow a similar course.

Fig. 7-D: Photograph of patient at seventeen years of age, showing the deformity.

is possible that these curves may have been present for many months without becoming clinically apparent, because they were usually not very deforming.

It would be desirable to evaluate the skeletal age of the patients and to correlate it with the growth of the scoliosis. The idiopathic curves almost always increase during growth of the skeleton, and they cease to progress about one year before the completion of ossification excursion of the iliac apophyses. Unfortunately, we were not able to determine accurately the skeletal age of most of our patients, and thus the chronological age was used instead.

Idiopathic scoliosis is much more frequent in girls than in boys. In the group of cases with main thoracic curves, however, there were many males. The scoliosis often progressed in boys about two years longer than in girls. This was probably due to the fact that the skeleton reaches maturity later in males than in females. Otherwise, there were no major differences in the course of scoliosis in boys and girls, and the data were compiled together in the tables.

#### Alterations in the Density of the Vertebrae and Abnormalities of the Disc Spaces

Changes in the vertebrae and in the intervetebral spaces were often seen in the roentgenograms of the scoliotic patients. The vertebrae adjacent to the apex of the thoracic curves were most frequently affected (Figs. 9-A and 9-B).

Most of these changes were apparent in the thoracic curves when the first roentgenograms were taken, shortly after the onset of the scoliosis. They were seen during the entire progress of the curve. The intensity of these vertebral alterations was usually directly related to the rapidity of increase of the curve. Thus, these spinal changes were of major prognostic significance. The texture of the bone of the lumbar vertebrae appeared to be fairly normal, even in the most extensive lumbar curves. Translatory shift, however, was often seen between the lumbar vertebrae.

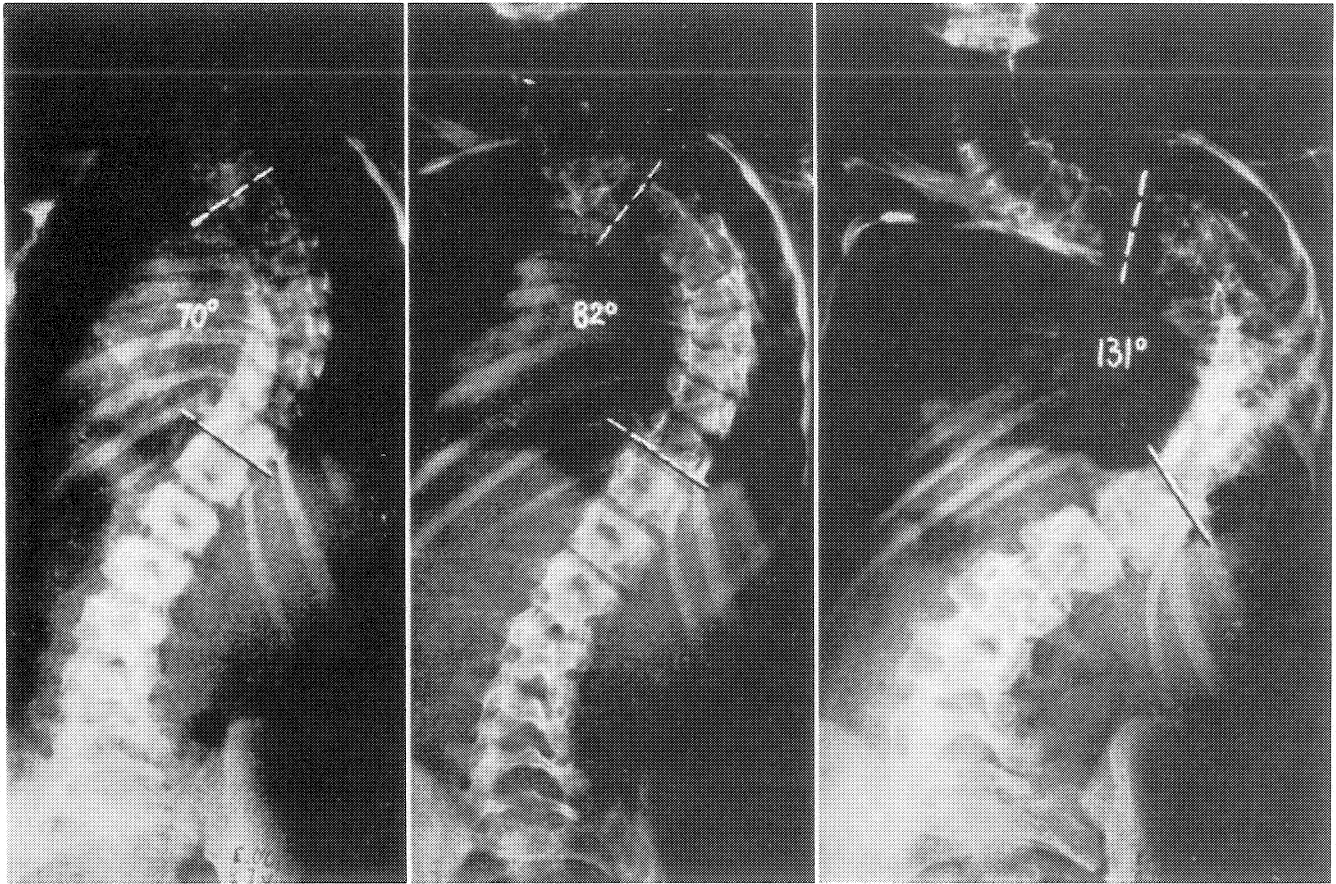


Fig. 8-A

Fig. 8-B

Fig. 8-C

Fig. 8-A: Main thoracic curve in a seven-year-old girl. Scoliosis was first noticed when patient was six. Apex was at eighth thoracic, and curve extended from fourth to eleventh thoracic vertebra.

Fig. 8-B: Same curve when patient was eleven years of age, showing some increase.

Fig. 8-C: Roentgenogram taken when patient was fifteen years of age shows great increase of the curve. The scoliosis progressed irregularly; it increased more rapidly during the period between eleven and fifteen years.

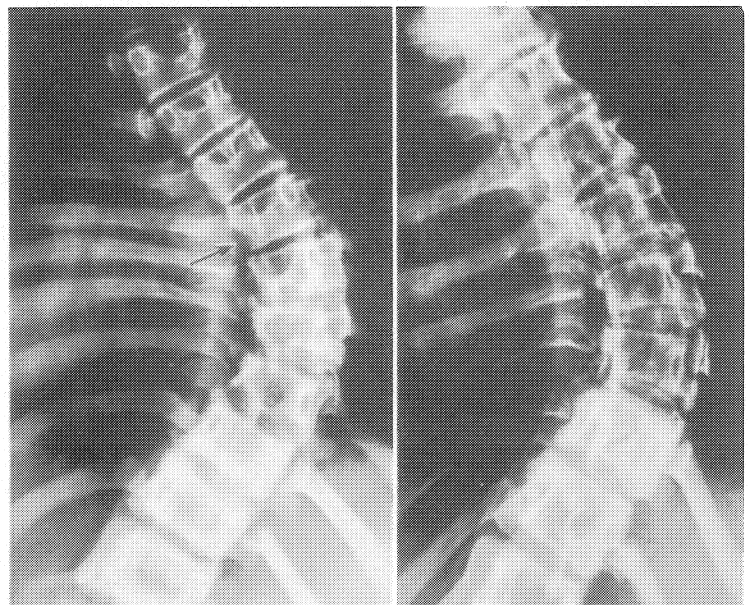
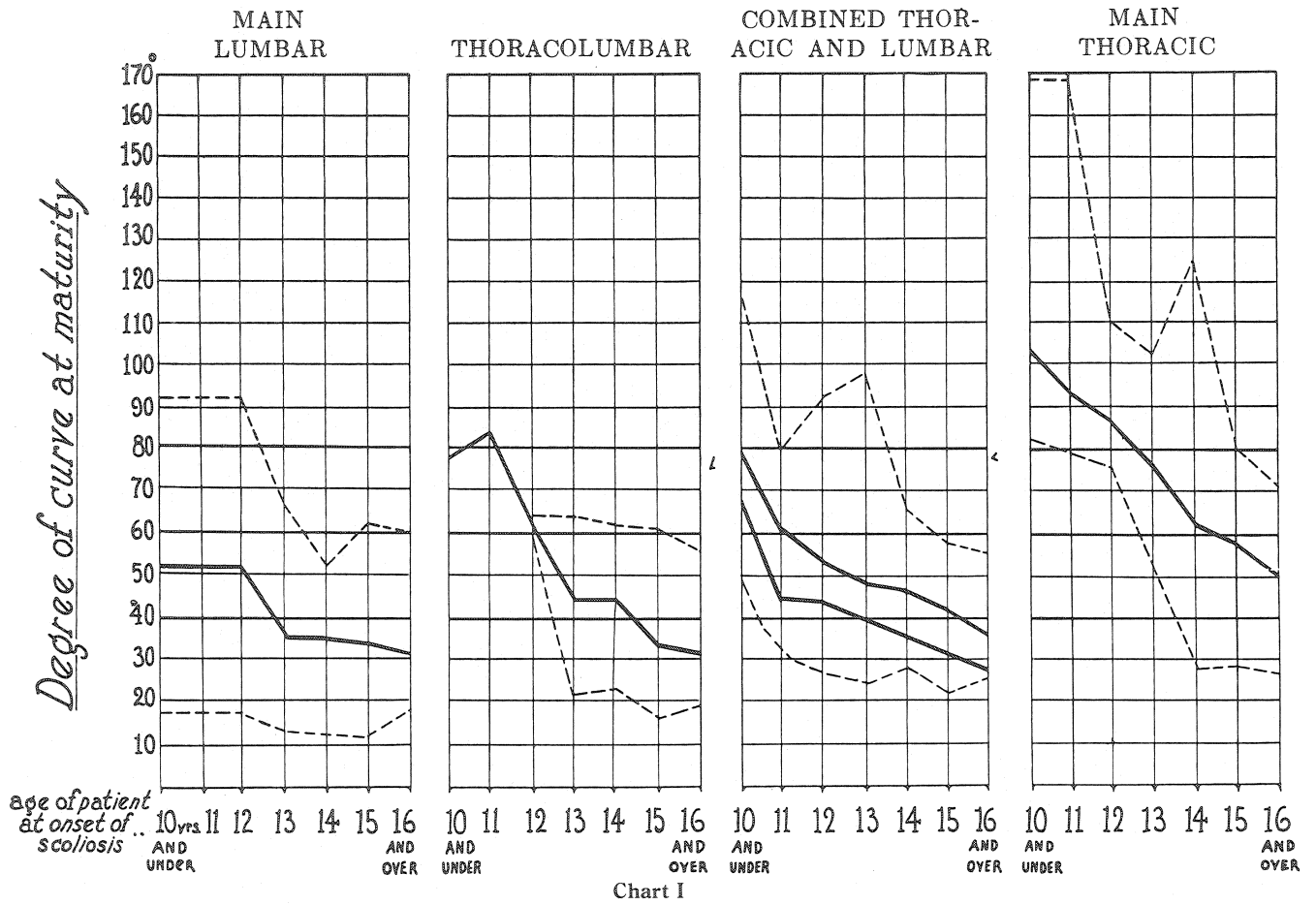


Fig. 9-A (At Left)

Fig. 9-A: Roentgenogram showing lesions of the vertebrae in a main thoracic curve. Defects of the vertebral bodies on the concave side of the curve may be seen.

Fig. 9-B (At Right)

Fig. 9-B: Roentgenogram of the thoracic component of a combined thoracic and lumbar scoliosis. The vertebral bodies adjacent to the apex of the curve are osteoporotic, wedge-shaped, and poorly outlined.



Angle of curvature at maturity in each curve pattern, as related to the patient's age at onset of scoliosis. The solid lines represent the average angle; the broken lines, the extremes. In the graph of the combined thoracic and lumbar pattern, there are two solid lines. The upper one represents the thoracic component, the other represents the lumbar; only the extremes of the thoracic curve are shown. The small number of cases of cervicothoracic scoliosis were not included in the graph.

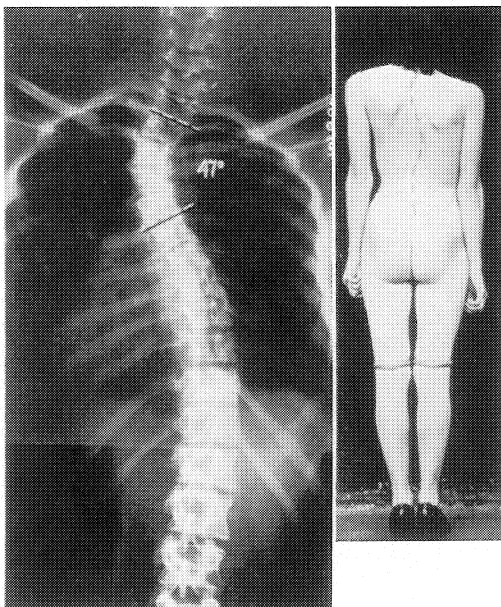


Fig. 10-A

Fig. 10-A: Composite roentgenogram showing a cervicothoracic scoliosis in a woman of twenty years. Curvature was first noticed when patient was fourteen and one-half years old. There is a long, low thoracic counter curve.

Fig. 10-B

Fig. 10-B: Photograph of patient, showing elevation of left shoulder.

### **Rapidity of Increase in the Size of the Curve**

When the scoliosis started after ten years of age, the increase was gradual in most of the patients. The prognosis, then, could often be best evaluated after two or three successive examinations, taken at intervals of three months. If the roentgenograms showed marked increase of the curve, the prognosis was usually poor. On the other hand, if there was only a minor progression of the curve during this period of observation, the prognosis was much better.

When the scoliosis started in children under ten years of age, it often did not increase much for a few years and then increased suddenly. This scoliosis of early onset usually carried a poor prognosis, in spite of its slow increase during the first years.

For practical usage, a graph was constructed for each curve pattern, to show the size of the curvature which might be expected at maturity, as related to the age of the patient at the onset of the scoliosis (Chart 1). The central

line represents the average angular value; the secondary lines, the extreme curves. Data for this chart were obtained from the study of the 330 cases of idiopathic scoliosis followed through maturity. Angular values of the curves in the standing position were recorded. The ages referred to in the graph are the chronological ages of girls.

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# CONGENITAL CLUB FOOT: THE RESULTS OF TREATMENT‡

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Since 1948, a uniform system of treatment has been applied to all cases of congenital club foot on the Orthopedic Service of the State University of Iowa. Our aim has been to obtain a supple, well corrected foot in the shortest possible time. An end-result study of severe club-foot deformities in otherwise normal children treated initially in this department from 1948 to 1956, with a follow-up period from five to twelve years, is here presented.

Three hundred and twenty-two patients with club-foot deformity were treated during this period. The following were not included in this study: One hundred and forty-nine patients had been originally treated in other clinics and were referred to us for further correction. Ten patients had arthrogyposis; four had a complete or partial absence of the tibia; and eighteen had a myelomeningocele. The sacrum was absent in two and congenital constriction was present in the legs above the malleoli in two patients. In forty-six patients, the foot deformity was mild and was corrected by simple manipulations or the application of one to three plaster casts. Of the remaining ninety-one otherwise normal children with severe untreated club-foot deformities, twenty-four were lost to follow-up, usually at the end of the initial treatment.

We were able to evaluate the results of treatment in only sixty-seven patients with a total of ninety-four club feet. All these deformities were severe, although many variations in the degree of rigidity of the feet were present. The age of the patient at the onset of treatment ranged from one week to six months, and the average age was one month. Of the sixty-seven patients studied, ten were female and fifty-seven were male. The deformity was, therefore, almost six times as prevalent in male as in female children. Forty patients had only one foot deformed (60 per cent) and twenty-seven patients had both feet deformed (40 per cent). In the patients with unilateral involvement, the right foot was deformed in eighteen and the left foot in twenty-two cases. Anteroposterior and lateral roentgenograms and photographs of the feet of all

patients were made at the onset of treatment and again at the time of the final examination.

## Method of Treatment

We aimed at an early and full correction of all the components of the deformity by gentle manipulation and well molded, thinly padded plaster casts which were changed every four to seven days. Anesthesia was never used. The plaster cast was applied in two sections, the first section extended from the toes to just below the knee and the second covered the knee and thigh. The knee was immobilized at a right angle while the leg was gently rotated outward to correct tibial torsion.

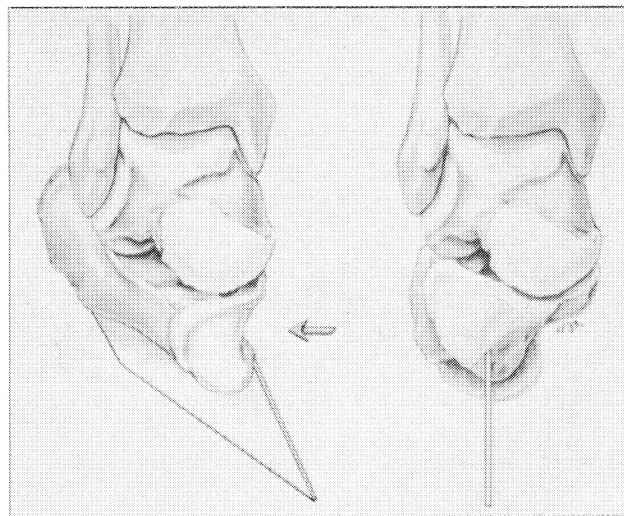


Fig. 1-A

Fig. 1-B

**Fig. 1-A:** In club foot, the anterior portion of the calcaneus lies beneath the head of the talus. This position causes varus and some equinus deformity of the entire calcaneus.

**Fig. 1-B:** Lateral displacement of the anterior portion of the calcaneus to its normal relationship with the talus will correct the heel varus deformity of the club foot.

A clear understanding of the club-foot deformity is possible after identifying by palpation the position of the bones in the foot and their relationship to one another and to the leg. The foot is displaced and rotated medially beneath the talus. The head of the talus is palpable on the lateral aspect of the dorsum of the foot, owing to the inward and backward displacement of the navicular. The calcaneus is in severe equinus deformity with its anterior portion lying directly beneath the head of the talus. This displacement is responsible for the severe varus deformity of the heel

\*For the past four years, Denis Browne splints have been worn at night until the age of three to five years.

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(Figs. 1-A and 1-B). The cuboid is also displaced inward in front of the calcaneus. The cuneiforms are displaced downward and inward in front of the navicular. The medial displacements of the navicular, cuboid, cuneiforms, and metatarsals contribute in different degrees to the severe adduction deformity of the club foot. The varus deformity of the calcaneus and the adduction of the mid-tarsometatarsal bones together are responsible for the inversion. The fore part of the foot, although adducted and inverted, is not as severely inverted as the hind part. As a result, the front of the foot is somewhat pronated with respect to the back of the foot, and this relationship causes the cavus deformity. The cavus deformity is thus produced by the slight downward displacement of the cuneiforms and by the fact that the first metatarsal is plantar flexed to a greater degree than the fifth metatarsal. The cavus deformity is sometimes erroneously designated as equinus of the fore part of the foot. Excessive plantar flexion of the anterior part of the foot occurs primarily on its inner aspect. The plantar flexion of the outer aspect of the front part of the foot may be normal, as evidenced by the fact that the calcaneus, cuboid, and fifth metatarsal are in a straight line, even though the club-foot deformity is severe (Figs. 2-A through 2-F).

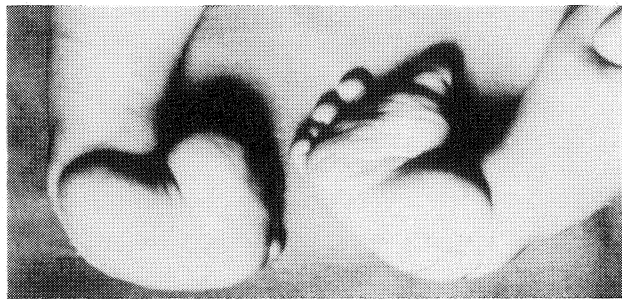


Fig. 2-A

Fig. 2-A: Severe bilateral club-foot deformity in a six-week-old infant. The heel is in severe varus deformity. The fore part of the foot is adducted and inverted. The cavus deformity results from the slightly pronated position of the fore part of the foot in relation with the heel.

Brockman noted that in a club foot there is subluxation of the talocalcaneonavicular joint and alterations in position of the navicular and calcaneus with respect to the talus like those which occur in the normal foot when it is adducted, inverted, and plantar flexed, but they are exaggerated in degree. However, a normal foot cannot adopt a true club-foot position because even in the extreme degrees of plantar flexion and inversion, the fore part of the foot moves with the hind part, and a cavus deformity does not develop since there is no discrepancy in the degree of inversion of the front and back part of the foot.

The cavus deformity must be corrected with the first cast. Since the cavus deformity is related to the pronation of the fore part of the foot with respect to the hind part,

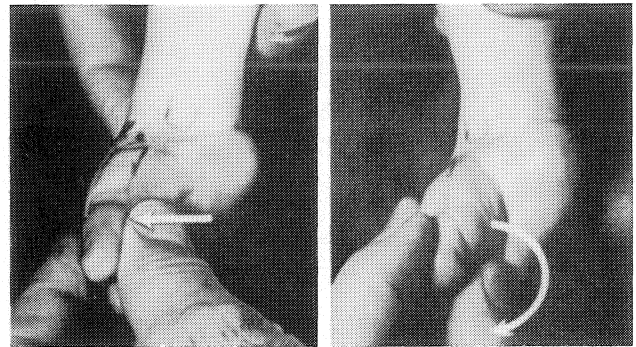


Fig. 2-B

Fig. 2-C

Fig. 2-B: Manipulation to correct the cavus deformity. The fore part of the foot is slightly supinated to be placed in proper alignment with the hind part of the foot.

Fig. 2-C: Wrong maneuver to correct the inversion. This maneuver increases the cavus deformity and fails to correct the varus deformity of the heel.

the cavus is corrected by placing the fore part of the foot in supination in proper alignment with the hind part. An attempt to correct the inversion of the foot by forcible pronation of the anterior part of the foot will increase the cavus deformity as the first metatarsal is further plantar flexed. This common maneuver is harmful because it hinders greatly any correction of the club-foot deformity by increasing the pronation of the fore part of the foot and thus making it very difficult to mobilize the navicular and displace it laterally in relation to the head of the talus. The navicular, cuneiforms, and metatarsals should be placed in straight alignment to form the lever arm needed for the correction of the inversion (Figs. 2-B and 2-C).

To correct the inversion of the foot, all of the foot distal to the talus must be made to rotate laterally underneath the talus which is fixed in the ankle mortise. A thumb

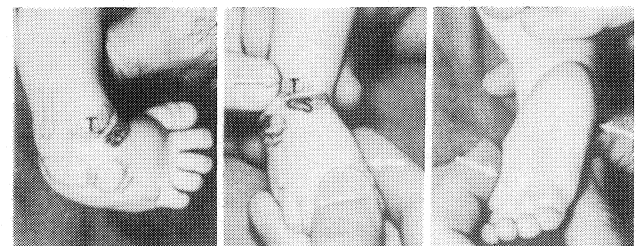


Fig. 2-D

Fig. 2-E

Fig. 2-F

Fig. 2-D: The outlines of the bones were drawn on the skin. The head of the talus (*T*) was palpable in the dorsolateral aspect of the foot in front of the ankle joint. The navicular (*S*) was displaced medially and its tuberosity was palpable just anterior to the medial malleolus. The first metatarsal is almost in a straight line with the cuboid and calcaneus.

Figs. 2-E and 2-F: Manipulation to correct the inversion: outward pressure is exerted on the first metatarsal and counter pressure on the lateral aspect of the head of the talus (*T*). When the navicular and cuboid and the entire fore part of the foot are displaced laterally in relation with the head of the talus, the anterior portion of the calcaneus follows; thus, the heel varus deformity is corrected. While the inversion is corrected the fore part of the foot should not be pronated to prevent recurrence of the cavus deformity.

placed on the lateral aspect of the head of the talus is used as a fulcrum while outward pressure is exerted on the first metatarsal and first cuneiform. During this manipulation an attempt is made to realign properly and simultaneously the calcaneocuboid, the talocalcaneonavicular, and the posterior talocalcaneal joints. When the navicular and cuboid are displaced laterally, the anterior portion of the calcaneus will be displaced outward and upward from its initial position underneath the head of the talus, and thus the varus deformity of the heel will be corrected (Figs. 1-A and 1-B). Care is taken not to pronate the fore part of the foot during this manipulation to prevent recurrence of the cavus deformity (Fig. 2-E). The manipulations should be gentle and followed by the application of a well molded thinly padded light plaster cast. Four to five plaster-cast changes are usually sufficient to correct the inversion of the foot.

The equinus deformity is corrected next by dorsiflexing the foot with the heel in a neutral or slight valgus position. This is the most difficult deformity to correct because of the great shortening of the tendo achillis which resists stretching. Two to three casts are often applied after manipulations in an attempt to correct equinus deformity. If it then becomes evident that many more casts will be necessary for a complete correction, a simple subcutaneous tenotomy of the tendo achillis is performed with the patient under general anesthesia. A toe-to-groin cast with the foot in maximum dorsiflexion and the knee at a right angle is then applied for three weeks. The equinus deformity is thus immediately corrected, obviating a rocker-bottom deformity which often results from prolonged forceful manipulation. When the plaster cast is removed three weeks later, the defect in the tendon is healed. The scar in the tendon after this procedure is minimum, as observed

in several instances where a tendo achillis lengthening was performed several years later to correct a recurrence.

Medial tibial torsion of variable degree is present in most patients with club feet and is a tenacious deformity if below-the-knee casts are used during treatment. Tibial torsion can be gradually corrected when toe-to-groin casts are applied with the knee in 90 degrees of flexion. To do this, the leg portion of the cast which includes the foot is held in slight outward rotation while the thigh portion hardens.

From five to ten (average 7.6) plaster casts worn for periods of from five to twelve weeks (average 9.5 weeks) were necessary for the correction of all the club-foot components in our cases. A subcutaneous section of the tendo achillis was done in seventy-four of the ninety-four feet. To prevent recurrences of the deformity, Denis Browne splints with high-top shoes with well molded heels were applied after the plaster-cast treatment. These splints were left on full time for an average period of three months and at night for an average of twenty-one and a half months more (ranging from ten to thirty months)<sup>†</sup>. Ordinary high-top shoes were used for walking. No sole wedges were prescribed.

*First Recurrence*

The deformity recurred in fifty-three feet (56 per cent) (Table I) at ages ranging from ten months to five years, with an average of two and one-half years. Some authors<sup>1</sup> stated that recurrences only occur when the club-foot deformity is not completely corrected at the initial treatment. However, when we reviewed the roentgenograms

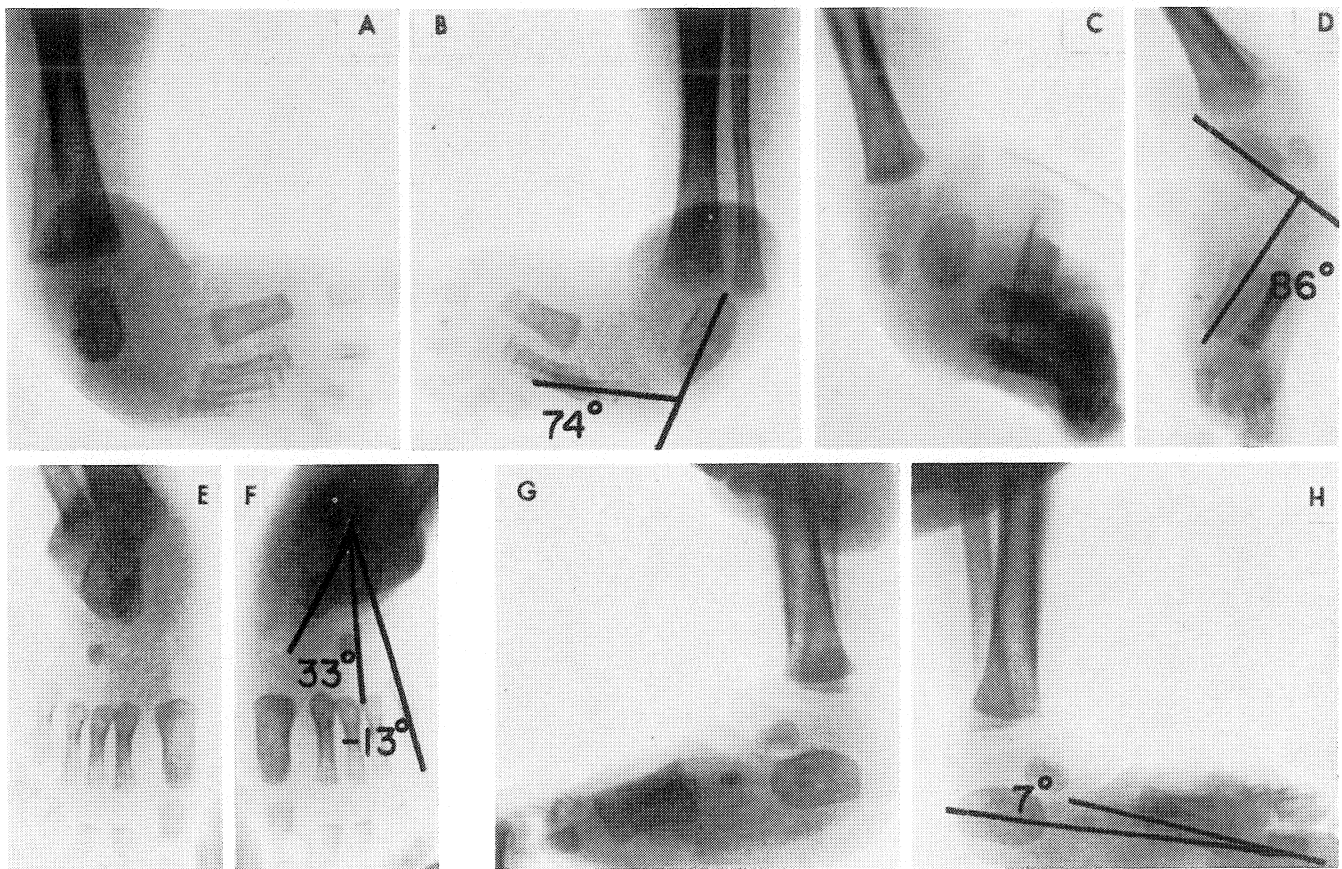
<sup>†</sup>For the past four years, Denis Browne splints have been worn at night until the age of three to five years.

**Table 1**

	Treatment of Recurrences			
	First	Second	Third	Fourth
No. of Patients	37	12	7	1
No. of Feet	53	17	9	1
Average Age (years)	2½	3	4½	7
Treatment	No. of Feet	No. of Feet	No. of Feet	No. of Feet
Plaster casts	47 (6.4 wks.)	17 (8 wks.)	9 (7 wks.)	1 (6 wks.)
Denis Browne splints	6 (11 mos.)			
Subcutaneous section, tendo achillis	5*	4**	1*	
Tendo achillis lengthening	5**	1	1*	
Anterior tibial transfer	27	5	6	1
Recession, extensor hallucis longus	3	1		1
Recession, extensor digitorum longus		1		
Subcutaneous plantar fasciotomy	1	1		1
Medial release		1	2	
Lisfranc capsulotomy	1			

\*One foot had subcutaneous tendo achillis tenotomy with the initial treatment.

\*\*Two feet had subcutaneous tendo achillis tenotomy with the initial treatment.



Part I

Fig. 3, A, B, C, and D: Anteroposterior and lateral roentgenograms of the feet of a six-week-old baby boy with severe congenital club foot. In the anteroposterior roentgenograms, the talus and calcaneus are superimposed and their axes coincide indicating a severe heel varus deformity. The angle between the axis of the calcaneus and that of the fifth metatarsal (adduction of the fore part of the foot) measures 74 degrees. In the lateral views, the angle formed by the long axis of the calcaneus and that of the first metatarsal (cavus) measures 86 degrees. The calcaneus is in severe equinus and the talus is subluxated forward in relation with the tibial mortise. Treatment consisted in manipulation and application of five plaster casts for the correction of the cavus, the adduction, and the heel varus deformities. This took a total period of one month. A bilateral tenotomy of the tendo achillis was then performed followed by a plaster cast applied for three weeks.

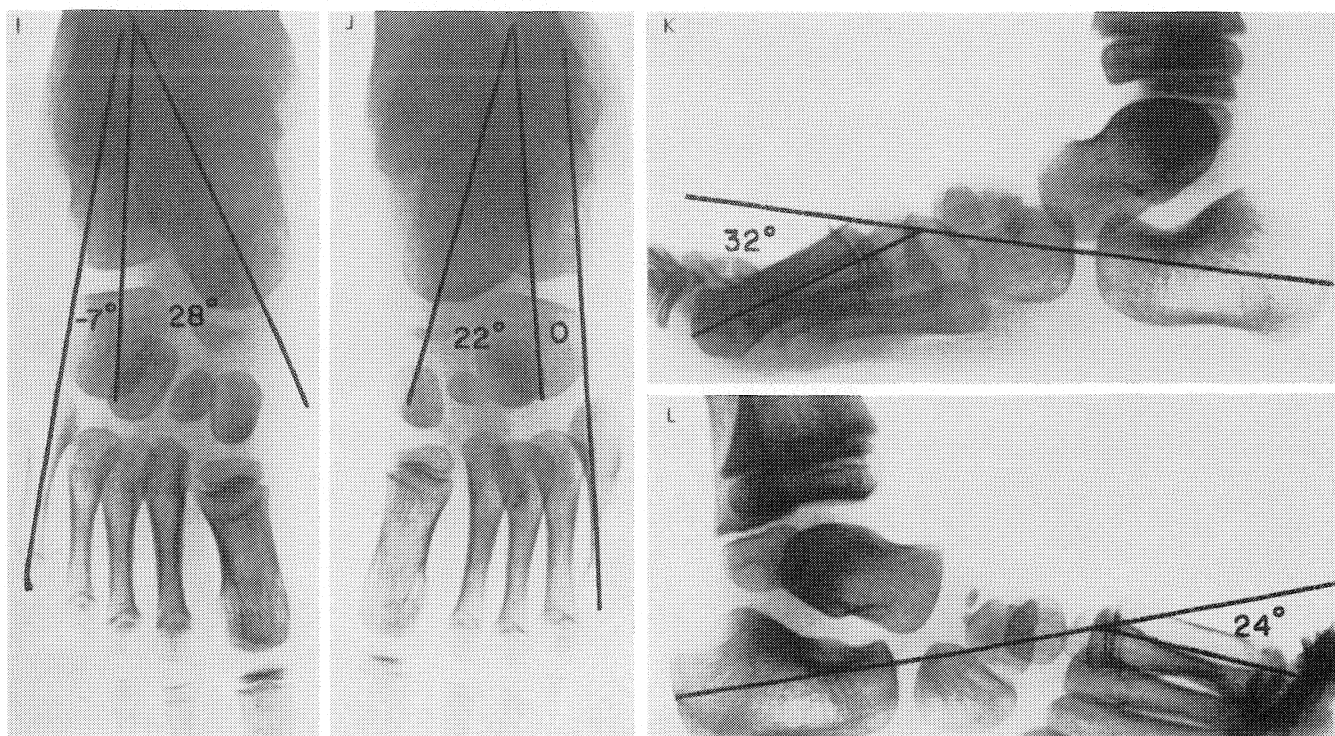
Fig. 3, E, F, G, and H: Anteroposterior and lateral roentgenograms made after removal of the last plaster cast, seven weeks after onset of treatment. In the anteroposterior roentgenograms the angle between the long axis of the talus and that of the calcaneus measures 33 degrees, indicating correction of the heel varus deformity, and the angle between the calcaneus and the fifth metatarsal measures 13 degrees, indicating correction of the adduction of the fore part of the foot. In the lateral roentgenograms the equinus deformity appears corrected and the angle between the long axis of the calcaneus and that of the fifth metatarsal measures 7 degrees, indicating correction of the cavus deformity. Denis Browne splints on shoes were worn full time for ten months and at night until four and one-half years of age.

made at the end of the primary treatment, we found that the relationship of the talus to the calcaneus had not been completely corrected in only five cases. The causes for the recurrences are difficult to determine. In the forty-one feet permanently corrected with the first treatment the deformity tended to be less rigid, the leg muscles better developed, and the length of Denis Browne splint treatment longer than in the feet with recurrent deformities. Also, the family of patients without recurrence tended to be more cooperative. About half of the recurrences occurred from two to four months after the Denis Browne splints were discarded, usually on the family's own initiative; the recurrences could be blamed on the neglect of

follow-up treatment with these splints. In other patients the recurrence was associated with a severe initial deformity and apparently poorly developed leg muscles; these recurrences seemed to be related to the severity of the primary aberration which caused the deformity.

In six patients the recurrence was treated with the Denis Browne splint worn at night and during napping hours. In forty-seven patients, the recurrence was more severe and was treated with manipulation and toe-to-groin plaster casts changed each week. The cast treatment lasted from three to twelve weeks (an average of 6.4 weeks). In the majority of recurrences, the equinus deformity was mild and responded to conservative treatment. Of the





Part II

Fig. 3, I, J, K, and L: Anteroposterior and lateral roentgenograms made when the boy was seven years old. A good result was obtained in the right foot (I and K) and an acceptable result in the left (J and L). In the anteroposterior roentgenograms, the angle between the long axis of the calcaneus and that of the talus measures 28 degrees on the right and 22 degrees on the left, indicating correction of the heel varus deformity on the right and incomplete correction on the left. The angle between the calcaneus and fifth metatarsal measures -7 degrees on the right and 0 degree on the left, indicating correction of the adduction of the fore part of both feet. In the lateral roentgenograms the angle between the long axis of the calcaneus and that of the first metatarsal measures 32 degrees on the right and 24 degrees on the left, indicating correction of the cavus deformity in both feet.

seventy-four feet treated with heel-cord section at primary treatment three required further surgery. The tendo achillis was sectioned subcutaneously again in one patient and was lengthened through a short medial longitudinal skin incision in the other two patients. The equinus deformity was resistant in seven feet not surgically treated initially. The tendo achillis was sectioned subcutaneously in four of these and lengthened in the other three. A subcutaneous section is preferred in patients under one year of age and tendo achillis lengthening in the older patients. In many of the recurrences, the varus deformity of the heel was more resistant to conservative treatment than the equinus deformity. A transfer of the anterior tibial tendon to the third cuneiform was done in twenty-seven feet in which there was a tendency for this muscle to supinate the foot strongly after the correction<sup>3</sup>. The tendon was transferred to the third cuneiform in nineteen feet and to the cuboid in eight. The tendon was attached to the bone through a drill hole using a Bunnell pull-out suture in twenty-one feet and a silk suture and osteoperiosteal flap in six feet. Two skin incisions were made, one along the distal one or one and one-half inches of the anterior tibial tendon, the other shorter incision on the dorsum of the foot at the

level of the third cuneiform. The tendon was transferred to its new attachment without changing its position underneath the ankle retinaculum. The foot was immobilized in a toe-to-groin plaster cast for four weeks. An overcorrection of the club-foot deformity after this procedure was not observed in this series. In three feet in addition to the anterior tibial transfer the extensor hallucis longus was recessed to the neck of the first metatarsal after suturing its distal stump to the tendon of the short extensor of the big toe. The recession of the extensor hallucis longus was done in cases with severe plantar flexion of the first metatarsal and hyperextension of the first metatarsophalangeal joint. In one foot with severe cavus deformity, a subcutaneous plantar fasciotomy was performed. In another, a Lisfranc capsulotomy was done to correct a severe metatarsus adductus<sup>4</sup>.

#### Second Recurrence

A second recurrence was observed in seventeen feet (18 per cent) at ages ranging from fourteen months to five years, the average being three years. In five of these seventeen feet the initial club-foot deformity was very rigid and the leg muscles were atrophic. Four other feet

were short and stubby and hence difficult to treat. The first recurrence in these four feet and the first recurrence in three other feet with deformities of average severity had been incompletely corrected by the application of only two to four plaster casts. In the five remaining feet the second recurrence was observed shortly after the Denis Browne splint was discarded prematurely.

The second recurrence was treated with reapplication of toe-to-groin plaster casts changed every one to two weeks for periods ranging from four to twelve weeks (average, eight weeks). This was followed by a subcutaneous section of the tendo achillis in four feet, a lengthening of the tendo achillis in one foot, and a transfer of the anterior tibial to the third cuneiform in five feet. In one foot, the extensor hallucis longus was recessed to the neck of the first metatarsal, all the tendons of the extensor digitorum longus were recessed to the third cuneiform, and a subcutaneous plantar fasciotomy was done to relieve the cavus deformity. A medial release operation was necessary in another foot with a severe recurrence which had been treated previously by plaster-cast applications and transfer of the anterior tibial tendon.

#### *Third Recurrence*

The deformity recurred for the third time in nine feet (10 per cent) at an age ranging from three to eight years (average, four and a half years). In five of these feet, the recurrence was mild and was apparently caused by the strong supinatory action of the anterior tibial muscle. These recurrences were permanently corrected by lateral transfer of the anterior tibial tendon after the application of two or three plaster casts. The other four feet were somewhat rigid and the leg muscles were very atrophic. In one of these, a medial release operation, as well as a subcutaneous section of the tendo achillis, was necessary; in another, a medial release was combined with a transfer of the anterior tibial tendon to the third cuneiform; in the third, a tendo achillis lengthening was performed; and in the fourth treatment consisted in five plaster-cast changes which were followed by a recurrence of the deformity.

#### *Fourth Recurrence*

The last foot, just mentioned, was the only fourth recurrence observed (1 per cent). Treatment consisted in the application of one plaster cast followed by a subcutaneous plantar fasciotomy, a transfer of the anterior tibial tendon to the third cuneiform, and a recession of the extensor hallucis longus to the neck of the first metatarsal.

### **Results**

The correction obtained in each of the components of the club-foot deformity was evaluated clinically and roentgenographically. Both evaluations correlated closely with

respect to ankle dorsiflexion, heel varus, and adduction of the fore part of the foot. Therefore only the clinical measurements of these components are charted (Table II). All the clinical measurements were performed by the senior author for the sake of uniformity.

On the anteroposterior roentgenograms, the degree of heel varus deformity was estimated by measuring the angle formed by the long axis of the talus and the calcaneus. A 30-degree angle was considered normal and was classified as 0 degree of heel varus deformity. Thus a measured talocalcaneal angle of 20 degrees corresponded to 10 degrees of heel varus deformity. The adduction of the fore part of the foot was also estimated on the anteroposterior roentgenograms by measuring the angle between the long axis of the calcaneus and that of the fifth metatarsal. On the lateral roentgenograms, the cavus deformity was estimated by measuring the angle between the long axis of the calcaneus and that of the first metatarsal (Fig. 3). The correction of the equinus deformity was estimated by measuring the degree of ankle dorsiflexion. The degree of tibial torsion was estimated clinically by having the patient seated on the edge of the examining table with the knees at 90 degrees of flexion and the feet in slight plantar flexion. In the normal foot, the head of the talus can be palpated in front of the ankle mortise in the same plane as the axis of the thigh. Medial or lateral tibial torsion is indicated by the orientation of the head of the talus in respect to the patella and axis of the thigh.

The cavus deformity was corrected in most feet by the first plaster-cast application. The recurrences of this deformity were usually mild and responded to manipulation and plaster-cast applications with upward pressure on the first metatarsal head. However, a subcutaneous plantar fasciotomy was necessary in three feet with severe cavus deformity. In two of the three feet, this operation was combined with recession of the extensor hallucis longus tendon to the neck of the first metatarsal. Recession of this tendon was done in three other feet with severe plantar flexion of the first metatarsal. All these operations were successful, and in the final examination the cavus deformity was corrected in all cases.

The heel varus deformity was completely corrected in seventy feet (74 per cent). A slight degree of heel varus deformity of less than 10 degrees persisted in twenty-four feet (26 per cent). The heel varus deformity was corrected after the initial treatment in all feet but it recurred in fifty-two. Anterior tibial transfer was done after plaster-cast correction in thirty-nine of these feet. The operation was successful in thirty feet but from 1 to 10 degrees of heel varus deformity persisted in nine feet. In six of these feet, the tendon pulled loose from its insertion (in three the wire broke and in the other three the silk stitches apparently came loose). In three feet the tendon was

transferred to the third cuneiform and probably should have been transferred to the cuboid. The thirteen remaining recurrences had slight residual heel varus that did not require treatment.

Adduction of the fore part of the foot was completely corrected in seventy-two feet (77 per cent), was less than 20 degrees in twenty-one feet (22 per cent), and was severe in one foot. In one foot with severe adduction, the deformity was corrected by capsulotomy of the Lisfranc joint. Of the twenty-two feet with residual adduction, seven had an anterior tibial transfer and fifteen did not. It appears then that anterior tibial transfer may help to correct not only the heel varus deformity but also the adduction of the fore part of the foot.

Tibial torsion was completely corrected in seventy-eight feet (83 per cent). In fifteen feet (16 per cent) a moderate residual medial tibial torsion of less than 10 degrees was observed, and in one there was tibial torsion of 20 degrees. In no instance was an osteotomy of the tibia performed.

Dorsiflexion of the ankle of more than 10 degrees above a right angle with the knee in extension was observed in seventy-five feet (80 per cent). In this group are included ten feet in which neither section nor lengthening of the tendo achillis was performed and sixty-five feet which had this tendon sectioned at the initial treatment. In eighteen feet (19 per cent) dorsiflexion of the ankle was limited to from 0 to 10 degrees. Eight of these feet had the tendo achillis sectioned at the initial treatment. A second section of the tendon was done in four to treat a recurrence, and in three the tendon was lengthened. The other ten feet of this group had either a section or a lengthening of the tendo achillis at the time of the first or second recurrence. One foot in which the heel cord was sectioned at the initial treatment and sectioned again at the third recurrence had a 5-degree residual equinus deformity. Mild flattening of the superior articular surface of the talus was observed in the final roentgenograms of this last foot and in fourteen feet of the preceding group. The over-all rating of results is summarized in Table II. Good results were obtained in 71 per cent of the feet (Figs. 4 and 5), acceptable in 28 per cent (Figs. 6 and 7), and poor in 1 per cent.

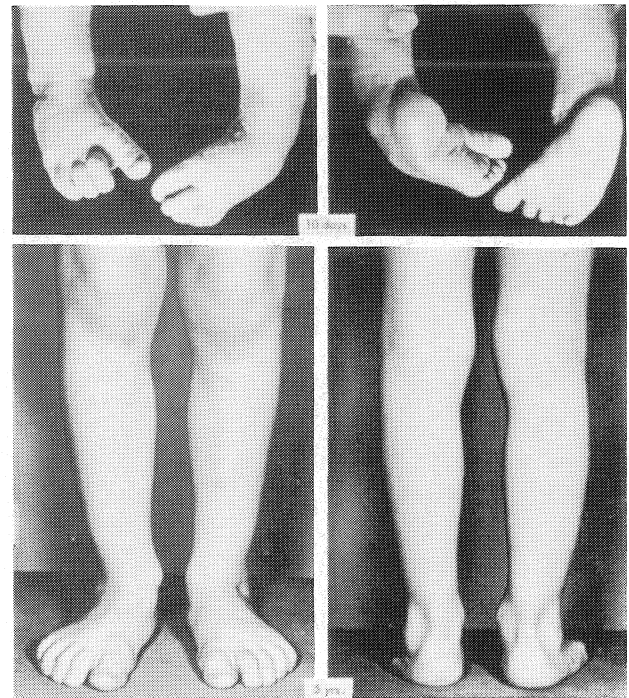


Fig. 4

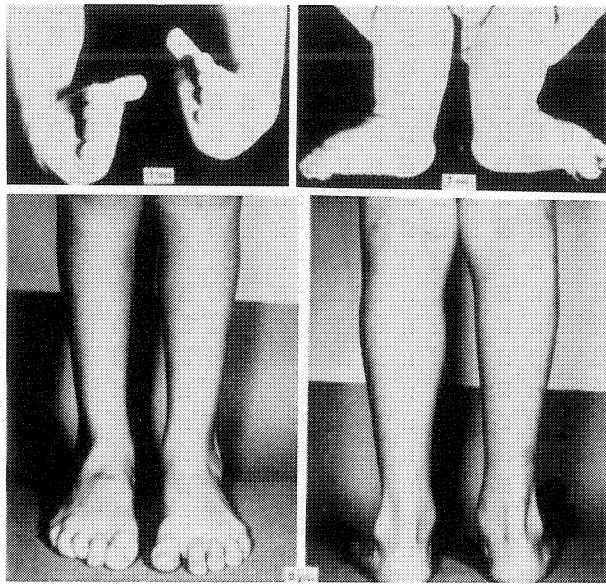
*Above:* Club foot in a ten-day-old male infant. Photographs made after removal of the plaster casts which had been applied three days previously. Cavus deformity is completely corrected on the right side and partially corrected on the left side. Five more plaster casts were applied in a period of three weeks. The child wore Denis Browne splints part time for four years. *Below:* At five years of age both feet were well corrected.

### Discussion

Although the treatment of a mild congenital club foot may be easy, the complete and permanent correction of a severe and rigid club foot is often difficult. In this study, we have been concerned with the severe cases only. The early months of life offer a golden opportunity for the correction of club feet since the skeleton, which is to a great extent cartilaginous, is little deformed, and the joint capsules, ligaments, tendons, and muscles can be stretched without damage. Early correction of all the components of the deformity in the shortest possible time is necessary for the proper development of the foot, since plaster-cast treatment prolonged for many months interferes with

Table 2

Results				
Ankle Dorsiflexion (Degrees)	Heel Varus (Degrees)	Adduction of the Fore Part of the Foot (Degrees)	Tibial Torsion (Degrees)	Result
>10	0	0-10	0	Good: 67 feet (71 per cent)
0-10	0-10	10-20	Moderate	Acceptable: 26 feet (28 per cent)
0	Over 10	Over 20	Severe	Poor: 1 foot (1 per cent)



**Fig. 5**

*Above, left:* Bilateral club-foot deformities in a one-month-old female infant. Six plaster casts were applied in a period of one month, followed by bilateral subcutaneous tendo achillis tenotomy. *Above, right:* At three months of age both feet were corrected. At fourteen months of age, the deformity recurred on the left and was treated by application of two toe-to-groin plaster casts. A bilateral recurrence of the deformity occurred when the child was five years old and was treated by transfer of the anterior tibial tendon to the third cuneiform after the application of three plaster casts. *Below:* At eight years of age, both feet remained well corrected.

growth and may cause stiffness of the joints.

Correction of a severe equinus deformity can be radically shortened by subcutaneous section of the heel cord followed by the application of a plaster cast (with the foot in maximum dorsiflexion) for three weeks. After this procedure, the tendon always heals with little scarring; and, if it is done early, a posterior capsulotomy of the ankle joint is unnecessary and rocker bottom and flattening of the upper articular surface of the talus are prevented. However, the heel cord should be sectioned only after the other components of the club-foot deformity are completely corrected. Of seventy-four feet with severe equinus deformity treated with a subcutaneous section of the heel cord in the primary treatment, only eight required further surgery to treat a recurrent equinus deformity. Only seven tendo achillis lengthening operations were performed in the entire series.

Recurrence of the heel varus deformity and adduction of the fore part of the foot are common even after complete correction. Denis Browne splints on shoes worn full time for the first two or three months after correction, and part time thereafter until the child is from three to five years of age, are useful to prevent recurrences in many cases. However, only half of the recurrences could be blamed on the neglect of follow-up treatment; often

these could be corrected by the reapplication of a few plaster casts. Some form of surgical treatment was necessary to prevent further recurrence in the more severe cases. A transfer of the anterior<sup>3</sup> or posterior<sup>2</sup> tibial tendons to the third cuneiform or to the cuboid in these feet seems to be the most effective procedure to prevent further recurrence of the heel varus deformity. The tendon transfer should be performed only after the foot is well corrected either by the application of several corrective plaster casts or, if necessary, by a medial release operation. However, the medial release operation often leaves extensive scarring and stiffness in the mid-tarsal joints and, when possible, should be avoided. Early transfer of the anterior tibial tendon in the very severe cases reduces greatly the need for this operation. It was performed in only three of our feet. The anterior tibial tendon was preferred over the posterior tibial for transfer because the operation is easier to perform and the anterior tibial functions in phase with the foot dorsiflexors, thereby making unnecessary postoperative training. Excessive plantar flexion on the first metatarsal and cock-up of the big toe was observed in only six of our feet; three of them before and three after anterior tibial transfer. This deformity was corrected by the transfer of the long extensor of the big toe to the neck of the first metatarsal. The nine failures of the anterior tibial transfer to correct permanently the varus deformity of the heel could be blamed on surgical errors.



**Fig. 6**

Bilateral club-foot deformities in a five-day-old male infant treated with the application of ten plaster casts in a period of two months. A recurrence of the equinus deformity at one year of age was treated by the application of two toe-to-groin plaster casts and subcutaneous tenotomy. All the components of the club-foot deformity recurred at three years of age. Three plaster casts were then applied, followed by the transfer of the anterior tibial tendon to the third cuneiform. When the child was eight years old the left foot was well corrected. On the right a 20-degree metatarsus adductus and 10-degree heel varus deformity persisted.



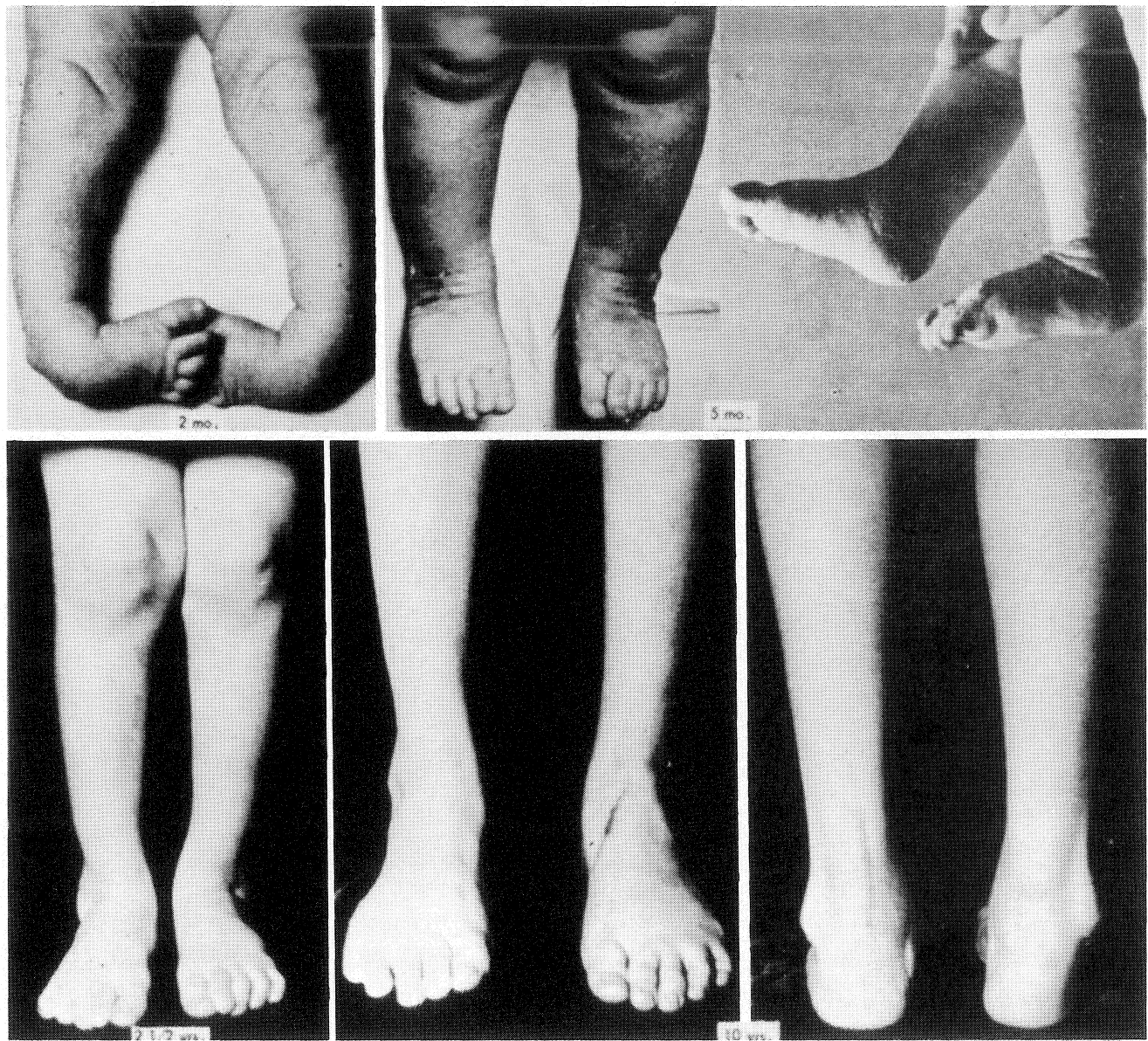


Fig. 7

Bilateral club-foot deformities in a female infant treated at two months with six plaster casts in a period of six weeks, followed by a bilateral subcutaneous tendo achillis tenotomy. Denis Browne splints on shoes were worn for four years. Photographs made when she was five months old, and later, when she was two and one-half years old, show all the components of the deformity well corrected. A mild recurrence of the deformity occurred when she was five years old, which was treated by the application of four plaster casts which were changed every ten

days. Then, Denis Browne splints on the shoes were worn for three years. When she was nine years old the deformity recurred again. The left foot was treated by lengthening of the heel cord and transfer of the anterior tibial to the third cuneiform. *Below, right:* In the photographs made four months after operation, the deformity is well corrected on the left side. A 15-degree varus deformity of the heel and 5-degree equinus deformity persisted on the right. Subsequently, a lengthening of the heel cord and transfer of the anterior tibial tendon was done on the right foot.

Tibial osteotomy was not necessary in our patients because medial tibial torsion was corrected during the application of toe-to-groin plaster casts followed by the use of Denis Browne splints. Tibial torsion will remain uncorrected if below-the-knee plaster casts are inadvisably used in the treatment.

### Summary

The results of treatment in sixty-seven patients with a total of ninety-four severe congenital club feet were evaluated five to thirteen years after the initial treatment. The primary treatment consisted in the application of several

plaster casts changed frequently for an average period of 9.5 weeks. In many instances a subcutaneous tendo achillis tenotomy was performed in the primary treatment to obtain a complete correction of the equinus deformity. Denis Browne splints were used in the follow-up care in all patients.

In fifty-three feet the deformity recurred and required further treatment. The recurrences of the equinus deformity were usually mild and responded to conservative treatment. Only seven tendo achillis lengthening operations were performed. A transfer of the anterior tibial tendon to the dorsolateral aspect of the foot was performed in thirty-nine feet to prevent further recurrences of the heel varus deformity. Medial release operations were necessary in only three feet. In no case was bone surgery performed.

The results in 71 percent of the feet were good; in 28 percent a slight residual deformity persisted; and in one foot a poor result was obtained.

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# THOUGHTS ON THE ETIOLOGY OF PERTHES' DISEASE

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Despite everything that has been written on the subject of Legg-Perthes' disease, it must be admitted that the cause of this condition remains unknown. However, it is pertinent to review the facts concerning its etiology to see whether any conclusions might point towards avenues of fruitful research.

The basic facts about this condition should be briefly summarized.

1. *Incidence.* The incidence of this condition is approximately one in 12,000 children in the age "at risk" (Males 1:8,000, females: 1:30,000)(Table 1). Recent studies<sup>1</sup> have shown that Perthes' disease is more common in the northwest area of England and Scotland and has a higher incidence in towns than in rural communities. The condition seems to be related to social class with an incidence of 2.6/10,000 in social class 5 compared with 0.7/10,000 in social class 1.<sup>9</sup> There is no evidence of inherited factors<sup>16</sup> but Hall et al. demonstrated an increased incidence of minor congenital anomalies<sup>8</sup>.

**Table 1**

## Incidence of Legg-Calve-Perthes' Disease in the British Isles

Source	Area	Overall	Boys	Girls
Harper 1976	South Wales	1:4,750	1:3,000	1:11,800
Catterall 1970	Scotland	1:5,590	1:4,060	1:14,830
Barker 1980	England	1:12,500	1:8,064	1:30,300
	Merseyside		1:5,917	1:20,000
	Trent		1:8,333	1:33,000
	Wessex		1:11,494	1:50,000

2. *Age and Sex.* The onset of the disease occurs between the ages of four and nine years in 80 percent of patients. Boys predominate in the ratio of four to one<sup>4</sup> (Table 2). Eighteen percent of cases are bilateral with the sex ratio of seven to one in favor of males stressing the extreme rarity of these bilateral cases in girls. The average age of onset in bilateral cases is younger than unilateral cases<sup>4</sup>. The outcome of this condition is known to be less favorable in girls than boys<sup>12</sup>. The explanation for this is

that there are more girls with Groups III and IV disease that have less favorable prognoses. Within the groups the outcome for boys seems similar to that for girls. There may be additional unrecognized factors accounting for the poor prognosis for girls.

**Table 2**

	No.	Male	Female	Sex Ratio	Age of Onset
All cases	388	305	83	3.67:1	71.75
Unilateral	323	248	75	3.3 :1	72.36
Bilateral	65	57	8	7 :1	68.7

3. *Environmental Factors.* There is a strange contrast between cases with unilateral and bilateral disease (Table 3). In the unilateral case there is an increased incidence of hernia in the patient and his first degree relatives<sup>3</sup>, together with an increased incidence of prematurity and breech position or version in the last trimester of pregnancy. In the bilateral case there is a high incidence of genitourinary anomalies and social class 5 origins.

**Table 3**

	No.	Renal Anomalies	Hernia and Undescended Testicle
All cases	388	22	10
Unilateral	323	12	8
Bilateral	65	10	3

4. *Growth Disturbances.* It has been known for many years that children with Perthes' disease may be of short stature and may have delayed bone age<sup>1,15</sup>. Observations by Burwell<sup>2</sup> suggest that the head, shoulders, trunk and proximal limbs of these children are normal; the main loss of height is in the short distal extremities including the hands and feet. Wynne-Davies and Gormley<sup>16</sup> showed that the short stature in younger children often corrects in adolescence, whereas children presenting at an older age will continue to be small throughout life. In parallel with these growth disturbances, bone age is often delayed. According to Harrison et al.<sup>10</sup> this delay was present in the index patient but not in their first degree relatives. With healing of the disease the bone age returns to normal in most cases, but in others may persist in its retarded state. This failure of normal skeletal maturation may be part of the growth disturbance in the distal limbs or it may reflect an overall failure of normal ossification. The latter

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is supported by the finding of thickened articular cartilage on the uninvolved hip in children with unilateral Perthes' disease<sup>4</sup>. In his studies of the growth plate morphology in this condition Ponseti<sup>14</sup> originally suggested that one of the primary abnormalities was in growth plate changes similar to those induced in latherism by aminonitriles. In their morphologic study of this condition Catterall et al.<sup>4</sup> looked at the uninvolved opposite hip in two cases and compared it with normal controls. These apparently uninvolved hips showed ossification abnormalities with loss of cellular columns and masses of cartilage remaining unossified in the primary spongiosa.

5. *Pathology.* Due to the paucity of clinical material available for study, detailed accounts of the morphology during various stages of Perthes' disease remain few. In most cases the epiphysis undergoes avascular necrosis and the overlying articular cartilage becomes thickened, particularly in the anterolateral aspect of the femoral head. In many cases metaphyseal lesions produce a growth disturbance in the proximal femoral metaphysis. Jensen and Lauritzen<sup>11</sup> reported the only case without definite evidence of avascular necrosis in which the defect produced was a consequence of a growth disturbance. This growth disturbance is in essence a failure of normal endochondral ossification producing a central femoral head defect of thickened articular cartilage. The recent review by Catterall et al.<sup>4</sup> has shown morphologically that the process is a spectrum of disease in which the radiological Group I cases are represented by a growth disturbance. In Groups II and III there is an infarction of the anterior bony epiphysis leaving the posterior aspect without evidence of avascular change. In the Group IV cases, not only is the whole of the bony epiphysis infarcted, but it seems to have occurred on several occasions. The articular cartilage overgrowth is greatest in the Group IV case. Following the epiphyseal infarction, the radiographic changes are the consequence of a repair process in which loose necrotic trabecular bone is removed and intact necrotic trabeculae replaced by a process of creeping substitution. The thickened articular cartilage is gradually re-ossified. The growth disturbance is proportional to the extent of the epiphyseal infarct, the presence of subluxation of the femoral head and the range of motion in the hip joint. Interestingly, inspection of most specimens reveals excess fat in the normally haemopoetic marrow of the proximal femoral metaphysis. There is no evidence of femoral neck infarction and the fatty tissue appears to have replaced the normal haemopoetic marrow.

### Conclusions

No final conclusions can be drawn concerning the cause of this condition. The evidence would support the view that there are a number of normal children susceptible to Legg-Perthes' disease. These "Susceptible Children" would

be primarily boys between ages five and nine years who are, smaller than their friends and first degree relatives and have delayed bone age. In all other respects they are normal, healthy, and very active children; many of them are hyperactive. This "Susceptible Child", when subjected to a particular inciting event, incurs the pathological changes in the upper femoral epiphysis that we call Legg-Perthes' disease.

If this concept of the "Susceptible Child" is accepted, there are various questions that must be immediately asked and which may provide fruitful avenues of future research. Why should they have a growth disturbance? Why should they have so much fat in their proximal femur and why is the pathology so variable with an incomplete infarct in many cases?

The accepted theories of infarction are those of embolism, tamponade, and extreme position. Although all have produced epiphyseal infarction in animals there is no clinical evidence in susceptible children of acute femoral head embolism or of sufficient excess hip joint fluid to produce tamponade. Although episodes of hip irritability occur, tense effusions sufficient to produce tamponade would cause very severe pain. This does not occur clinically. A more attractive possibility is that of raised marrow pressure producing intraosseous tamponade within the epiphysis. For this theory to be accepted, it must be shown that it is possible to raise pressure within the epiphysis either directly or via pressure changes in the proximal femoral metaphysis. McPherson<sup>13</sup> has demonstrated very considerable changes in marrow pressure following muscle stimulation via the femoral and sciatic nerves in the cat. Following injection of saline into the proximal femur, Green and Griffen<sup>6</sup> noted increased pressures in Perthes' disease compared to normals. The observations by Gershuni et al.<sup>7</sup> suggest an acute effusion in the hip could not be sustained long enough to produce bony infarction, which requires a period of eight to ten hours of total ischaemia.

The causes of this condition remain unsolved, but fruitful avenues of research would investigate the cause of this ischaemia and the biochemistry of fat metabolism.

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# REFLECTIONS ON SCOLIOSIS AND MIDDLE EASTERN MEDITATIONS\*

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When after protracted wanderings, Dr. Weinstein's letter found me in Saudi Arabia, I could hardly contain my delight. Having retired as Professor in Edinburgh four years ago, I had grown accustomed to the notion that I was perhaps unlikely again to visit the United States where I have been so often, learnt so much and have found so many friends. There is not one among them, however, that I would rather see than Dr. Ponseti and his wife. We have seen each other many times in various places since I first met him in 1948 while visiting Dr. Steindler. My delight was complete when I saw that all my fellow guests are well known to me, and to meet them again is good fortune beyond reasonable expectation, but then the whole occasion has that quality.

Before I return to Dr. Ponseti, perhaps I may digress a moment and explain why I was in Iowa then and why I owe so much to American Orthopaedic Surgery. Like Dr. Ponseti, I graduated from medical school just before World War II. After an abbreviated general surgical training, I joined the Army and spent the next few years in the Middle East and the Balkans; an exciting life, but with few opportunities to learn. However, I was able to determine that my interests, though not my training, were to be orthopaedic surgery. On leaving the Army, I was appointed to the staff of the Royal National Orthopaedic in London and soon found that I knew less of elective orthopaedic surgery than my residents. The fortunate award of a Rockefeller Fellowship brought me to North America for a year, during which I gained what formal orthopaedic training I have had. Even more, it allowed me to meet the giants of that time, amongst whom I was most influenced by Jo Barr, R. I. Harris, John Cobb, Sterling Bunnell and many others. During this time I met Albert Key, whose somewhat fearsome reputation was known to me, though he was not. Somewhat hesitatingly I asked him what were his special interests in orthopaedics. He replied "the skin and its contents boy", a definition of orthopaedic surgery I have subsequently used many times, particularly in battles with general surgery.

To meet so many great surgeons was a timely and stimulating experience and a turning point in my surgical life. However, even more important and lasting in its effects was to meet my American and Canadian contemporaries, then chief residents or newly on the staff. They, like me, are retired or soon to do so. Amongst them are almost all the leaders of the last two decades, including Dr. Ponseti. The interrelationships of American, Canadian and British orthopaedics have traditionally been close. In due time came my opportunity to foster these friendships and knowing the surgeons in the U.S. was the essential catalyst. The Travelling Fellowships continue, and I believe they still have as perhaps their most important role the bringing together of the likely future leaders in our six countries.

I would like to recall that in 1948 the first British Travelling Fellows came as the result of a remarkable act of generosity by American and Canadian orthopaedic surgeons led by R. I. Harris, a Canadian, then President of the American Orthopaedic Association. He and others were aware of the immense gap in British opportunities to travel and learn from you caused by the War years of 1939-45. Indeed, travelling remained almost impossible until 1948; a huge gap causing our surgeons to remain in ignorance of what was happening here.

It is more than time I returned to Dr. Ponseti, the reason we are all here. When they first visit America, the British are at a peculiar disadvantage. Perhaps because we and you both speak English, we come expecting to find many differences, yet a basic similarity. This is somewhat an illusion. It is perhaps the first time some of us learn that we are basically European. I am no longer aware of this, but in those first few months I felt it keenly. Thus when I first met Dr. Ponseti, then not many years in America and still clearly Spanish and European, I felt an immediate close relationship which has continued.

As we all know, since then he has contributed over a wide panorama of orthopaedic problems, seeming always to take on the most difficult and rarely failing to illuminate those difficulties. I will confine myself to his work in scoliosis, since it is an area of orthopaedic surgery we have both tried to elucidate.

## I. Reflections on Scoliosis

I am primarily a clinician and it is to two papers on clinical features of scoliosis to which I will first refer. In 1950 Dr.

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Ponseti published a study which differentiated various patterns of curves within the entity of idiopathic scoliosis. I had been impressed by the same patterns and a year later published a study with very similar findings. This differentiation of these varied curve patterns with very different behavior, although of one etiology, has proved to be very important. The safe, effective treatment of scoliosis is only possible when early curves are treated either by brace or surgery. The selection for this is based on the natural history which allows us to separate the progressive curve from the many benign. When this study of different curve patterns was extended to other etiologies, such as paralytic and congenital scolioses, their treatment for the first time became rational. When the age of onset of the curve, the curve pattern and the etiology had been studied in subsequent years it seemed that for the first time the surgeon could know the prognosis of almost any scoliosis he might see. He could then deduce what should be done. Scoliosis has a vast array of different causes and within each etiological group extremely different behavior. Looking back, I think for too long, and perhaps particularly in the States, the presence of scoliosis was too often an indication for surgery. The knowledge of the behavior of different scolioses was often ignored even after Ponseti's paper.

In the present day, when few severe scolioses are seen and early curves can frequently be managed without surgery, the problems that presented thirty years ago hardly seem comparable. Perhaps now our knowledge of curve behavior is less relevant, but in earlier years this knowledge was an absolute necessity and became my own main line of study.

A more recent clinical paper of great practical importance was Ponseti's long term study of the various idiopathic curve patterns that had not been corrected or fused. The patients were seen twenty years after the end of growth. To the best of my knowledge, this remains the only study of all types of idiopathic curves followed for many years after the end of growth with measured curves at maturity. Later Ponseti showed that a majority of curves did not change dramatically, though some, particularly the thoracic curves, progressed so much and so consistently that this had to be taken into account when an adolescent with a thoracic curve on the borderline for correction and fusion was seen. In recent years there has been a surgical fashion to advise many adult patients to have correction and fusion, or to advise adolescents with small curves to have surgery because of late deterioration. Those of us with much experience from earlier years when scoliosis was not treated are aware of many older patients with scoliosis and no problems. Dr. Ponseti's study gave us factual evidence of the late outcome of curves left unfused. Though many that we might have left alone we now know

should be fused, the wholesale correction of adult scoliosis is shown to be based on dogmatic opinion, not fact.

I continue to find it remarkable that such a common and intensely investigated deformity as scoliosis should remain an unsolved mystery. Idiopathic scoliosis can, in a relatively short time, cause horrific deformity in children who are, to all investigations, normal. It is perhaps the most mysterious problem of them all.

The academic surgeon has many directions in which he should excel. He must be an adequate surgeon, an excellent teacher and writer, a skilled administrator and be able to make a major research contribution. Few achieve success in more than two of this quartet of necessary virtues. Important as Ponseti's clinical observations have been, we all know that this is but a part of his work. Over many years he has led a team which has probed deeply into the collagen and protein constituents of the disc and apophyseal cartilages in an attempt to elucidate the cause and mechanism of scoliosis. That he and we remain baffled is almost immaterial. The immense contributions of his team is clearly based on a sound laboratory discipline. There are few clinicians who can also excel in the laboratory. I doubt in orthopaedic surgery whether there is his equal. That some clinician should be able to do and direct laboratory research is essential, though the difficulties are immense. Without their direction the clinical problems cannot be perceived nor solved.

The work from Iowa has shown differences in the chemical constituents of disc and cartilage in patients with scoliosis. Muscle fibers have been shown to have microscopic differences in the type of fiber. Burwell's painstaking measurements have shown many asymmetries in the scoliotic: the upper limb on the convex side is longer; fat is thicker on the concavity of the trunk. We can cause an idiopathic-like scoliosis in animals by cutting the costo-transverse ligaments or the sensory roots. There are some fifty diseases in which scoliosis is a manifestation yet there is no identifiable common link. Apart from congenital scoliosis and scoliosis after poliomyelitis, we have not the remotest idea what abnormality causes such gross and varied deformity. I could go on almost endlessly citing these odd and varied differences. They have so often seemed to be the vital clue, only to disappear like a mirage in the desert. There seems to be no possible common factor between all the diseases that can cause lateral curvature of the spine.

I am delighted to see Dr. Wynne-Davies here. She made the field of orthopaedic genetic disease very much her own while in the orthopaedic department in Edinburgh. The genetic studies indicating a familial incidence in idiopathic scoliosis seemed to be perhaps the first clear indication where to look to solve this mystery. The basis of these views was a population incidence of some 2:1,000 contrasted with a much higher incidence in those families

with children with idiopathic scoliosis. I would like to ask her views now that school surveys have shown such a high population incidence of minor curves. Does this apparent family preponderance continue to be significant?

Despite so many (perhaps too many) clues from clinical observations, anatomy, histology, biochemistry, genetics and animals models, we remain not only ignorant of what causes scoliosis of virtually any etiology, but we do not even know which tissue is primarily affected. Even more baffling is our ignorance of the mechanism of curvature or how deformity occurs. I first became interested in scoliosis thirty-five years ago after meeting that gifted, obsessive teacher, John Cobb. Since then I have attempted to put together what we know, to provide an explanation, but fruitlessly. I am in good company. Despite being distanced from the conundrum for several years, a position which sometimes leads by a curious process of mental digestion to seeing the wood from the trees, no miraculous enlightenment has come upon me.

## **II. Middle Eastern Meditations**

As I warned Dr. Weinstein, I have been away too long to contribute scientifically, other than by these meditations. However, it may interest you to hear an account of the medical and orthopaedic problems, the cultural and social modes of the two Middle Eastern Arab countries where I have worked since I retired, Kuwait and Saudi Arabia.

Whilst the challenge of very poor, underdeveloped, Third World countries is tremendous and rightly attracts many, there are almost insuperable problems. Disease, trauma and crippling are rife and facilities and money so scarce, what can be achieved is very limited and relatively primitive. This is not to diminish its importance and the urgent need to help.

In the oil rich countries of the Middle East the problems are quite different. Before their sudden wealth neither Kuwait nor Saudi Arabia had any significant medical services. In Kuwait there was a small American mission hospital, the only source of modern medical help. In Saudi Arabia, since all other religions are prohibited, there was even less. Within a generation they were able to afford and demand medicine at its highest technical level. The opportunity to advise and guide this unprecedented, indeed meteoric progress, is very important and very exciting. Guiding this development along common sense lines is not always easy. The expensive, technically impressive methods can be far more attractive than basic clinical medicine. It is our job to ensure by example that the latter is not lost to the glamour of technology. As can be appreciated, there are many difficulties and frustrations in bringing a country with nothing to modern standards.

Kuwait is a small country wedged between Iraq and Saudi Arabia, with Iran a very near neighbor across the

narrow Gulf. Despite proximity, the war that has raged now for some three years has hardly impinged on the outward life of this area. Kuwait is wholly desert. Before oil was discovered the small population lived on trade by camel caravan, by sea voyages to India and Zanzibar and from pearl fishing. The huge Middle Eastern oil fields extend from Iraq and Iran in the north to Saudi Arabia in the south. The Burgan oil field in Kuwait is surely the most accessible oil there is. The oil is near the surface and under such pressure that it need not be pumped. The field lies on a ridge, and from the collecting tanks on the ridge it runs by gravity to the ships. This could not be in greater contrast to the hazardous exploitation of North Sea or Alaskan oil.

The main problem has perhaps been how to manage the vast wealth now pouring in. It has changed Kuwait from one of the poorest countries, to the one with the highest per capita income in the world. Perhaps because of their long tradition as traders and businessmen they have managed this enormous problem well, with an emphasis on schools, universities and hospitals.

Having had some medical association with Kuwait from dealing with cases of paralytic scoliosis, I was invited to be Director of their National Accident and Orthopaedic Services when I retired. This was a fascinating task. In the years I was there, five large new general hospitals were opened, one a teaching hospital. They served the whole country. Each hospital had its own accident/orthopaedic service. The orthopaedic staff also worked at a preexisting central orthopaedic hospital of 300 beds, which will be replaced this autumn with perhaps the most superbly built orthopaedic hospital in existence. Though administrative in title, my job was fortunately largely clinical.

One particular feature I would like to comment upon are the genetic diseases which are so common. In many Arab countries an arranged marriage between cousins (often first cousins) is the norm. This has been going on for centuries. McKusick's genetic gold mine that he found amongst the Amish pales into insignificance. However, for the geneticist it is complicated by the multiple wives and numerous related children, a particular feature of Saudi Arabia. As might be expected, recessive diseases rare with us were common. Skeletal diseases of dominant inheritance were also common. Ehlers-Danlos and severe osteogenesis imperfecta (often with elastic skin as seen in Ehlers-Danlos) were more frequent than I had seen before. The sickle cell trait occurred in some tribal groups, though not as severe as in Negroes of West African origin. As Negro slave concubines were common, they may well be the source. Albinos were a frequent sight in the streets. Dupuytren's contracture was seen only once.

After two and a half years in Kuwait we had decided to return home, but persuasion by an old Edinburgh col-



league took us to Riyadh in central Saudi Arabia. This has been a fascinating contrast to Kuwait and other Arab countries. Saudi Arabia is dominated by the Royal family who rule largely through the 5,000 Princes. The most powerful have been the Sudairi brothers, sons of one of the wives of the remarkable Abdul Aziz Ibn Saud, who created the modern Kingdom by tribal conquest. The strong cohesive force is Islam. There is no civil law as we know it. Life is governed by the Sharia, the religious laws. Riyadh is dominated by the traditional outlook of the stern puritan Wahibi sect, the moral force behind Abdul Aziz. Life pauses for half an hour five times daily for Salah (prayer time). All shops must close. Any shopkeeper who ignores this will be in serious trouble with the Mutawa, the religious police. Women may not drive. Virtually all Saudi women are veiled, even in the clinic. European women must wear long dresses to the wrist and ankle, and the hair must be covered. Women may not be alone in a car with a man who is not their husband or brother. This poses a considerable problem for the many single men and women expatriates working in Riyadh. As in many Moslem countries, the theater does not exist, for religious reasons. In Saudi Arabia there are no cinemas, though approved television programs are now broadcast. Expatriates live mostly in compounds which allow a reasonably normal Western social life to go on behind walls. Despite these restrictions there are many things to enjoy.

Until a few years ago, Saudi Arabia was a country centered around Bedu nomads totally unacquainted with Western life. The Bedu are the core of the population, only now becoming acquainted with Western medicine and Western outlook. There is much religious concern over modernization. On Thursdays and Fridays, the Islamic weekend, many of us explore the Central Arabian desert. Four wheel drive vehicles (never less than two), compasses and water are the essentials for exploring and camping. The escarpment of the wadi in which Riyadh lives are reminiscent of Utah and New Mexico. Camels are still important for meat and milk. Wheat fields are now large. They are irrigated by "fossil" water, originally derived from the Anatolian mountains and which has lain underground for thousands of years. Some lie not far from the Rhub Al Khali, the Empty Quarter.

Earlier Arabia lay under the sea and there are many fossils to be found including coral reefs, even on the mountain tops. Several thousand years ago it was savannah country and the hunters of those times pursued deer, ostrich and other animals no longer able to live in the arid desert.

Some time before Christ, a people called the Nabateans grew powerful along the spice route going North through Saudi Arabia into what we now call Jordan. They carved elaborate tombs in the sandstone rocks. Petra, their main

city now in Jordan, was easily defended as it was approached by a long defile. The southern most Nabatean city was Medain Saleh, scarcely known to the world even now because of difficulties in entering the Kingdom and visiting near the Holy cities of Mecca and Medina. Entry to the latter two cities is totally forbidden to Christians and other infidels. We were able to go to Medain Saleh. Remains of the Hejaz railway include sabotaged tracks still lying twisted as they were left by T. E. Lawrence, the British officer who led the Arab revolt against the Turks in the First World War.

I have left discussion of the disease patterns until now because, although Kuwait and Saudi Arabia have many similarities, the special medical characteristics of the Middle East are much more marked in the latter, modern medicine came later and the population is still often nomadic.

One of the very surprising features in this sunny land is the prevalence of rickets. The pediatricians talk of it as an epidemic. One sees severe infantile rickets, but babies may be born with marked features of this deficiency, so-called congenital rickets. This is due to a vitamin D deficiency in the mother, often still an adolescent. Very severe vitamin deficiency rickets may even be seen in older children. Severe osteomalacia may be seen in the adolescent. This paradox is largely due to the fact that older girls and women now living in houses are completely veiled and are never exposed to sunshine. Even the two square inches of skin, enough to synthesize vitamin D, are not permitted. Another factor is that the traditional foods are low in vitamin D.

Congenital deformities are much as we know. Most dislocations of the hip first come to us when the child is walking. An adult with congenital talipes equinovarus may come walking on the dorsum of the foot. After a triple arthrodesis he can wear the normal sandal worn by all Arabs.

Tuberculosis of bone and joints is still common and is most atypical in its behavior. Vertebral pedicle destruction, which to us would be pathognomonic of tumor, is common. One learns that tuberculosis is a great imitator and must be excluded in many unlikely lesions. It responds well to antibiotics; surgery is confined to the relief of paraplegia. Brucellosis from the many goats is far from rare and hydatids indirectly from the many sheep is another diagnostic problem.

Poliomyelitis is a common problem, still almost totally confined to children. Hygiene is generally good in hot dry countries but many children do not escape subclinical infections as babies (when still protected by maternal antibodies) so that paralysis still occurs, mostly in infants. Immunization programs are advanced, but nomadic Bedu are hard to trace and the vaccine is very heat labile. Now the government wisely does not issue a birth certificate without full immunization. Without the birth certificate,

passport and schooling are not provided. The poliomyelitis has usually not been treated and severe contractures are common. The tensor fascia lata contracture is almost universal. Upper limb paralyses and paralytic scoliosis are quite rare. I believe most patients with these potential problems die in the acute stage from respiratory paralysis. However, some paralytic scolioses have come my way and it is interesting again to see the dropped ribs, asymmetrical intercostal and lateral abdominal flexor paralysis that was the causative deforming force when we saw this in the West. All these patients, as most of the ones we see, have extensive cautery marks, still the universal Arab treatment.

Driving in Saudi Arabia is terrifying. The death rate is many times that of Western countries. Since women may not drive, one may see a small boy driving a large American automobile taking his mother shopping. High speed head-on collisions on desert roads are numerous and the roads are littered with thousands of abandoned cars. It is common to see in the remote desert two cars pancaked flat, one on each side of the road. They destroyed each other and now lie in adjacent graves. In many accidents there are no survivors but we still get many horrible injuries, particularly quadriplegias. The problems of rehabilitation of a Bedu quadriplegic living in the desert are daunting.

Whilst we see many diseases and injuries now disappeared from the Western World, we are largely spared the ravages of rheumatoid arthritis. Though occasionally seen in its florid form, it is not only less common but when seen, joint destruction is often minimal or nonexistent.

Primary osteoarthritis of the hip is very unusual. In contrast, osteoarthritis of the knees is almost universal in those over forty-five years of age. I believe this is related to the habit of squatting for hours during which the patellofemoral and medial compartment articular cartilages must be without synovial fluid nutrition. It is a great problem, as the Muslims in Saudi Arabia must pray five times a day. Patients seek relief more from this difficulty than for pain.

One very interesting difference between Kuwait and Saudi Arabia lies in the provision of medical care. In Kuwait it is free to all in the many government hospitals already providing a reasonable standard. Both the many very rich and the few poor use them. Private medicine is minimal. Hospitals in Saudi Arabia, often staffed by American, British and other European doctors, provide high class but wholly separate service for the Armed Services, Public Security and the National Guard. Each is competing with the other in providing a service, causing problems from expense and duplication. For the general population, government hospitals are crowded and with limited facilities, but are improving all the time.

I hope a brief look at these problems has interested you. It has enabled me to continue to teach and train for a few extra years. I am profoundly delighted to be present on this occasion to honor one of the great scientific investigators in orthopedic surgery, a great yet delightful man whom I am honored to call a friend. I can only wish him well in what I was heartened to hear was to be semiretirement. I must finally thank Dr. Weinstein and his colleagues for inviting me to this very special occasion.

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## RESEARCH AND PATIENT CARE\*

Michael Bonfiglio, M.D.†

Dr. Alfred R. Shands, in whose honor this lectureship was endowed, clearly recognized the role of research in Orthopaedic surgery when he said "interest in research has always been deep, my desire to be an active participant in investigation has been great and my realization of what research means to Orthopaedic surgery has been clear. For without research there can be no progress."

Talking to members of the American Orthopaedic Association about research and patient care is like "bringing coals to Newcastle." Each of you has pursued an idea or question to a conclusion or you would not be members of this Society which recognizes the leaders of Orthopaedics. Any one of you could, I am certain, present cogent thoughts on the subject. My purpose is to share with you an idea which, while not new, is worth reemphasizing from time to time; namely, that an attitude of inquiry and scientific method is essential in the daily care of patients. The literature is full of observations based on fresh insights made by physicians caring for patients with obscure or unusual conditions, as well as common ones.

Dr. Shands acknowledged, as we do, that his major contribution to Orthopaedic research is what he did in planning and administering research programs. He was the prime mover in founding and steering the Orthopaedic Research and Education Foundation. He was a leader in the formation and organization of the Orthopaedic Research Society, he actively served on the Board of the Easter Seal Research Foundation, and was Chairman of the Skeletal System Committee of the National Research Council. His were consummate political and organizational skills which produced a much stronger position for orthopaedic research than existed when he gave his AOA presidential address entitled "Responsibility and Research in Orthopedic Surgery" twenty-nine years ago.

As a beneficiary of funds from OREF, I trust that my remarks will express my appreciation for the efforts of Dr. Shands on behalf of orthopaedic surgeons and their patients.

Earlier, in an article on Research in Orthopaedics, Dr. Shands wrote that "we have many laboratory men with clinical backgrounds and clinical men with strong laboratory leanings to thank in recent years for their contribu-

tions, such as Fuller Albright, Dallas Phemister, Gallie, Jaffe, Copeland, Steindler, Henderson, among others."

It is therefore out of the *milieu* of clinicians with a laboratory background that much of the research affecting patient care has developed in the three decades since. These men and women believed that in the resolution of a specific patient condition or the resolution of a question, the scientific method was the most effective way of thinking. They thought it should be as much a part of patient care in the office or at the bedside as in the conduct of laboratory or clinical research.

My research experience began during the Phemister-Hatcher years at the University of Chicago. I continued my involvement with clinical as well as laboratory research at the University of Iowa Children's Hospital. It was supported and encouraged by my late chief Carroll Larson, my good friend Ignacio Ponseti, and later my present chief Reginald Cooper, each of whom has actively promoted orthopaedic research of the highest quality.

The experience involved in doing research enhanced my awareness and appreciation of the work of others. I tried to transmit the excitement of discovery to students, residents and practitioners and to stimulate them to evaluate patients with a spirit of scientific inquiry. The process by which a question, an idea or a problem is resolved, after thinking it through, or after a thorough discussion with a colleague, is critical to planning an investigation whether basic or clinical. The heightened interest in the discussion and planning of an experiment is not diminished when the experiment fails. It often leads to a better plan for the next experiment. The steps required to state or revise a provisional diagnosis are similar, particularly when the data gathered in the evaluation of a clinical problem do not seem to fit. We review the information continually in discussions with colleagues, residents or students and often decide to consult with others. The value of these discussions at the office, in the clinic, on rounds, or in the operating room cannot be underestimated. The process involved in planning an experiment is useful in the diagnosis of a clinical problem and planning the care of a patient. The approach to either is similar and, in my judgment, is equally stimulating and essential. Just as research and teaching are closely intertwined, so are research and patient evaluation and treatment.

Phemister recognized the importance of basic surgical research. He also recognized that a considerable proportion of research work of a teaching hospital and medical

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school faculty should be clinical investigation, in order to justify the enormous expenditure which is necessary to maintain any research effort mounted in the hospital. Unless the members of the staff remain reasonably active in the care of patients, their research problems are likely to have little direct connection with patient care.

As I returned to the University of Chicago from the military service, Dr. Howard Hatcher had coaxed Dr. Phemister to become a Professor Emeritus of Orthopaedic Surgery after his retirement from the Department of Surgery. He also provided Dr. Phemister his office, a secretary and a resident. It was my good fortune to be his resident during that year.

When it came time to consider a project for study, Dr. Phemister told me of his experience with five patients upon whom he had performed the drilling and bone grafting procedure for aseptic necrosis of the femoral head. Four patients were treated for necrosis after fracture of the femoral neck and one for idiopathic aseptic necrosis. The results seemed promising.

It is not surprising, therefore, that when he suggested that we study the femoral head changes which occur with the procedure, I eagerly accepted the challenge (since, as you are aware, few of us come to a residency with ready ideas for a research project). Numerous histological studies had been previously reported on aseptic necrosis of the femoral head after trauma and only a few after unknown causes, but there was little known or done to preserve the necrotic femoral head. At Dr. Phemister's suggestion, many hours were spent then (and since) over a microscope studying the repair of aseptic necrosis from as many femoral head specimens as were available. In our discussion, we considered the following questions: How does the necrotic femoral head repair after drilling or grafting? What effect does drilling or coring alone have on the repair? What role do the grafts play in the repair process? I began work on the answers to these questions.

It seemed that the quickest and most direct way to study the effect of drilling and bone grafting on femoral head necrosis would be in a laboratory animal, so we chose to perform the experiment in the dog. Total femoral head necrosis was produced by dislocating the hip and osteotomizing the femoral neck, since others had shown that doing less than that produced an inconsistent pattern of necrosis. A 5 mm hole was drilled in the anterior and lateral half of the head and neck of the femur after dislocation of the hip and a 3 mm threaded wire was inserted in the posteromedial area of the head and neck for stability. A graft obtained from the ipsilateral tibia was inserted in the channel from lateral cortex to the subchondral cortex.

Photomicrographs from a three week specimen show that granulation tissue began to revascularize and replace necrotic marrow and bone with living tissue along the tibial

bone graft in advance of the repair from the osteotomy site (Fig. 1A, B). A high power photomicrograph demonstrates vascular invasion through a trabeculum into a necrotic marrow space (Fig. 1C). In addition, new bone united the bone graft to the channel walls in the femoral head (Fig. 1D). Reconstruction was complete for all the bone and marrow at one year, as shown in this dog femoral head (Fig. 2). The articular cartilage was mostly preserved but degenerative changes were seen.

That experiment indicated drilling and bone grafting aided the repair of a necrotic femoral head in the experimental animal. The union of the graft to the channel stabilizes the necrotic area during its revascularization and replacement.

We know of many ways to study research problems. If the most direct and fundamental approach to a clinical problem is through basic research, that should be the method of choice. The story of the development of the polio vaccine was eloquently presented to the members of the AOA by Dr. Albert Sabin when he was president Mark Coventry's guest lecturer in 1977. For orthopaedists, this represented a most dramatic change in the practice of our speciality. Similar stories can be recounted for the reduction of morbidity and mortality of osteomyelitis and tuberculosis by the discovery of antibiotics and chemotherapy.

For most of us, however, *clinical* rather than laboratory research forms the foundation for our advances in knowledge. The information gathered is more likely to have a direct application to patient care since it usually inspired by and deals with problems encountered in the patient. The experimental animal can provide useful biological information. The answer to the difficult question of how much research should be performed in experimental animals, and how much should be done with patients, depends on the problem and the talents and interests of the investigator. Information can be acquired from patients, but this is a difficult and time consuming method. Knowledge is usually obtained from available laboratory tests, functional examinations, roentgenograms or therapeutic tests gathered over a long period of time.

Clinical studies are full of vexing pitfalls related to incomplete data, inadequate samples and wide variability of data. Many an hour has been spent attempting to resolve such concerns before submission for publication.

The clinical problem I studied, i.e. the effectiveness of Dr. Phemister's procedure in treating femoral head necrosis, has been tested in the experimental animal. The observed sequence of events observed indicated a satisfactory repair of the femoral head could be achieved. I believed this confirmed what was noted in a few patients. The problem now needed further resolution of such questions as: 1) when is the procedure indicated; 2) how often and how long will aseptic necrosis of the femoral head



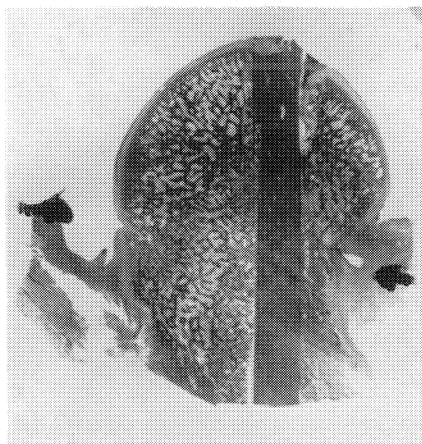


Fig. 1A. Dog femoral head three weeks after drilling and bone grafting.

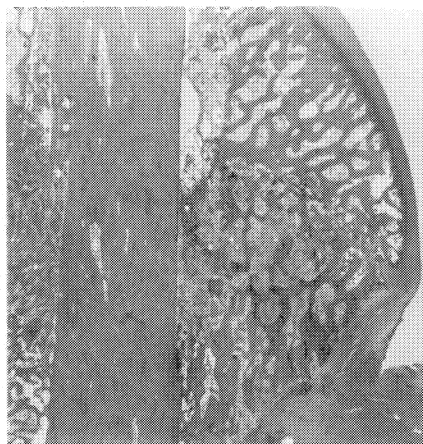


Fig. 1B. Low power view of same specimen.

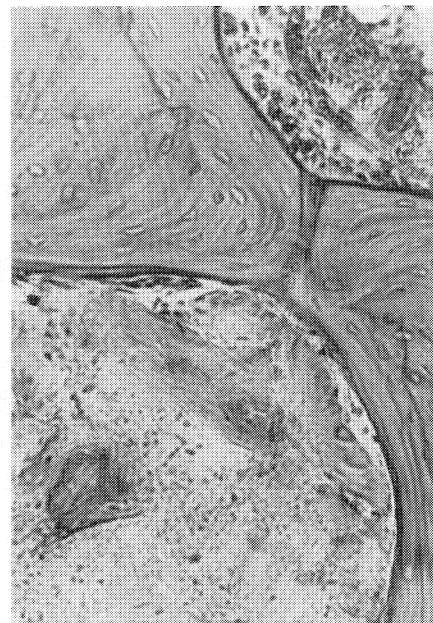


Fig. 1C. High power view of same specimen.



Fig. 1D. Low power view showing graft to head union.



Fig. 2. Whole head repair one year after grafting.

respond to the procedure. These cannot be resolved except by application of the procedure to more patients.

Clinical investigation should follow the scientific method whenever possible, whether the study is retrospective (as it was in analyzing results of the procedure) or prospective (as it was in the study of etiology). Reports of a procedure should include an adequate number of cases telling us the natural history of the condition, how often a given procedure works, why, for how long and at what risk to the patient. Otherwise, our judgement is based upon how strongly we accept the conclusions of the presented material.

I agree with Milton, who wrote that "where there is much desire to learn, there of necessity will be much arguing, much writing, many opinions; for opinion of good men is but knowledge in the making". However, by application of the scientific method to solve a problem, we

should develop more valid evidence to support or refute our position.

The experience I gained at Iowa with the Plemister procedure, along with the experience of others, has helped delineate answers to these questions. After the initial presentation of our clinical experience twenty-five years ago, I was encouraged by Dr. Sam Banks, who discussed the paper, to continue the long term clinical study. The following brief report will serve as a follow up note on our experience.

It was shown that four out of five hips with necrosis after fracture or dislocation can repair and function for ten or more years. In three patients out of four, the hip with nontraumatic femoral head necrosis functions for an average of fourteen years, and for as long as twenty-eight years. Twenty years after the procedure, as degenerative arthritis ensues, the percentage of satisfactory hips decrease

to sixty per cent.

One example of a post-trauma patient is a fifty-four year old woman who sustained a femoral neck fracture. Two years later she had developed nonunion of the fracture and aseptic necrosis of the femoral head (Fig. 3A). The Phemister procedure achieved union and repair of the femoral head as shown in the three year postoperative roentgenograms (Fig. 3B). Function was excellent with an Iowa hip rating of 96 points at eleven years. The roentgenograms showed minimal degenerative changes (Fig. 3C).

his Iowa Hip rating is ninety-four points with points lost for fatigue pain at the end of a ten hour workday and minimal restriction of motion in internal rotation and extension. The roentgenogram shows preservation of the articular cartilage shadow and a residual 2x3 mm central subchondral cortex density. The repaired femoral head demonstrates well incorporated grafts in the anterolateral half of the head (Fig. 4C, D).

The best long term results were in patients with unilateral involvement of the hip in which preoperative collapse was absent or minimal and in whom the preoperative hip



Fig. 3A. Neck fracture nonunion with femoral head necrosis two years after injury.



Fig. 3B. Same patient three years after bone grafts with healed nonunion and repaired head.



Fig. 3C. Same patient eleven years postoperatively.

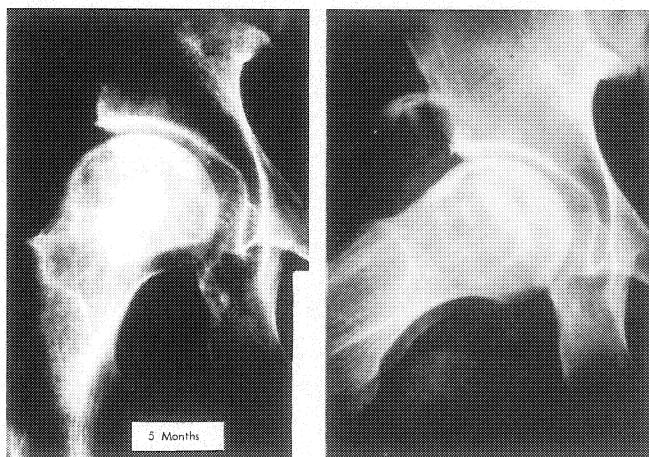


Fig. 4A,B. Preoperative AP and lateral roentgenograms taken five months after onset of symptoms.

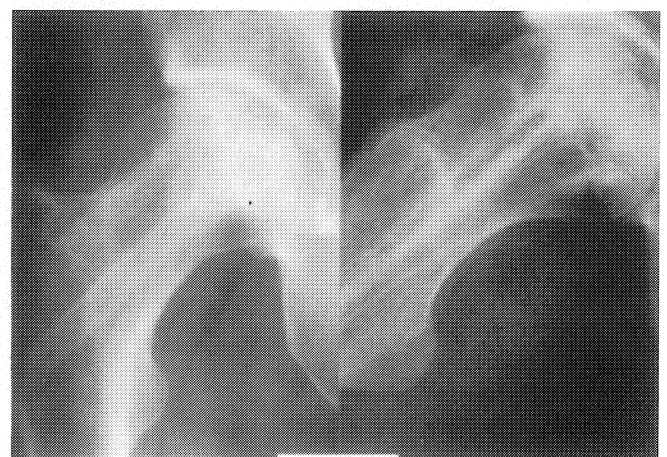


Fig. 4C,D. Roentgenograms taken twenty-two years postoperatively.

A thirty-one year old male dentist with hypocoagulability problems from drug induced pancytopenia had pain in his right hip for five months. The preoperative roentgenogram shows a large right femoral head infarct with minimal collapse (Fig. 4A, B). Twenty-two years postoperatively

rating was satisfactory—a score of eighty points or more on the Iowa hip rating. This assumes accurate placement of the drilled channels and tibial grafts.

Histologic studies begun under Dr. Phemister's guidance continue to bear fruit. Evidence relating to the orig-

inal question on repair of the necrotic femoral head has gradually been accumulated from femoral heads removed after failed procedures, but more importantly, from hips which had developed degenerative arthritis after many years of satisfactory function. Dr. Edward McCarthy will be reporting to us at this meeting on the repair of femoral head necrosis in humans.

Until now we have concentrated on the repair of the necrotic femoral head and the results of the procedure but have said little about the etiology or pathogenesis. Studies on the etiology and pathogenesis of aseptic necrosis also require clinical observation. The possible causes of non-traumatic aseptic necrosis have intrigued many individuals. Interruption of the blood supply to the bone and marrow was thought most likely by Freund, Axhausen, Chandler and Phemister, but the etiology and pathogenesis remains obscure.

Again, back to the patient. Our study of this aspect of the problem began on a chance observation of one patient who bled excessively during surgery for the necrosis. From this experience, my colleagues from the Hematology section at Iowa, Drs. Hamilton, Sheets, Connor and I then developed a two part protocol for a prospective study of every patient. One was a battery of tests emphasizing blood coagulability, and the other was to gather information on associated diseases and conditions which could be of significance in causing aseptic necrosis. We noted two significant factors, abnormal platelet counts, and hyperuricemia with gout.

We developed a theory reflecting the results of that study. Femoral head necrosis is a skeletal expression of systemic disease which by a constellation of events results in sludging, thrombosis or hemorrhage in an area of susceptible blood supply. It is one expression among several. There is no doubt that we are dealing with an incompletely resolved multifactorial problem.

Another clinical experience which occurred as we gathered data on the intake of alcohol proved to be a valuable lesson in the art of history taking. A patient had responded to a question from a resident that, yes, he was a social drinker. He usually had one or two drinks before dinner and one after dinner. Following this visit, his wife approached the resident to say, "I heard him tell you that he had one or two drinks before dinner. He forgot to mention that he also has one or two drinks *with* dinner, and one or two after dinner—and sometimes he never gets to dinner."

From this simple observation, we took note of the fact that we had not been asking the questions in such a manner as to elicit a complete reply. We learned that it is important to talk to family members regarding drinking and drug habits. The asking of questions in more detail showed us that there was a higher than expected or previously reported association of alcohol ingestion and aseptic necrosis of the

femoral head. This was a clear example of the value of clinical studies to improve clinical skills and patient care!

Since the associated risk factor of alcohol abuse was noted in 74% of our patients with aseptic necrosis, we tried to determine the incidence of aseptic necrosis in a known population of alcoholics. We thought that a prospective comparison of roentgenograms of the hips from alcoholics who were admitted to a treatment center on our campus with those of a group of nonalcoholic (control) subjects might be a valuable study. We wanted to detect early lesions for possible early treatment. We compared the roentgenograms of the hips from 705 alcoholics with those from 100 age matched controls for radiographic changes consistent with aseptic necrosis as outlined by the Decompression Sickness Panel of the Medical Research Council of Great Britain.

None of the findings showed any statistical significance between the controls and the alcoholics. The only instances of structural failure, however, (three hips for a 0.4 per cent incidence) were in the femoral heads of alcoholics. The demonstration of a lack of a significant difference in the incidence of early changes of aseptic necrosis came as a surprise. Not everything we study shows expected results.

Although I have personalized the theme of the role of research to solve a clinical problem by emphasizing the none too dramatic efforts on one problem, I trust it has characterized the nature of the solution to such problems. It is only by the gradual accumulation of bits of knowledge that most problems are solved. Only rarely does someone come along with the insight to synthesize such data into a significant biological theory.

Major problems await solution. While the efforts to alleviate the effects of arthritis by medical and surgical means are commendable and essential, until biological studies on cell function and matrix control are known and deficiencies corrected, we will be doing mostly symptomatic treatment. Fundamental research is required on the biologic mechanisms by which arthritis occurs. It is critical that any clinical investigation be directed to provide information upon which the patient and doctor can make a reasonable judgement based on known probabilities.

Each of you can apply your particular talent in the research arena to help identify and solve problems, whether in the clinical or basic science area, or as a residency program director or instructor. While Dr. Shands was not inclined to play any of the usual roles required for successful laboratory research, he did use his special skills to stimulate, promote and support research. We have an obligation to continue the tradition of scholarly pursuits which nurtured us. We should inspire and encourage the interest of our students and residents to study a problem in depth. The ideas for a project often arise from a patient who should

be the beneficiary of the solution to that problem. The ultimate pleasure in solving a problem occurs when the solution to that problem results in its successful application in a patient.

The triad of patient care, teaching and research, which I learned from Drs. Hatcher and Phemister and continued with my colleagues at Iowa over the past three decades, is one which you share and one often reiterated by Dr. Shands. It has served us well as a foundation on which to build for those who will follow.

Since we are in Virginia, perhaps it is fitting to close with Thomas Jefferson's motto for the University of Virginia:

"For here we are not afraid to follow truth wherever it may lead, nor to tolerate error as long as reason is left free to combat it."

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# CONTINUING PROBLEMS IN SEPTIC ARTHRITIS OF THE HIP: ANALYSIS OF RESULTS AND CURRENT TREATMENT RECOMMENDATIONS

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Dennis R. Wenger, M.D.†

Much has been written about the diagnosis, treatment, prognosis and management of acute suppurative arthritis of the hip in infancy and childhood. Although the general principles of treatment of acute cases are fairly uniform, the specifics of medical and surgical management vary widely.

Our management plan has evolved from our close association with the Children's Medical Center and Parkland Memorial Hospital in Dallas and the careful documentation by Nelson, McCracken, Bucholz and co-workers, of 528 cases of septic arthritis in pediatric patients<sup>1,6,9,12</sup>. Their series includes 128 septic hips. This large series, carefully analyzed and up-dated yearly by Nelson and McCracken, serves as our reference point in making recommendations for medical management. Additionally, we recently reviewed the long term consequences of hip sepsis by reviewing thirty-six cases of septic arthritis whose chronic sequelae were managed at the Texas Scottish Rite Hospital. This experience with both acute management and orthopaedic treatment of chronic problems provides the perspective for this review.

## Etiology

Most hip sepsis in children is secondary to hematogenous bacterial seeding of the synovium or contiguous spread from the proximal femoral metaphysis. The pathophysiology and differences in blood supply according to age have been well documented and will not be presented<sup>13</sup>.

## Diagnosis

Without question, delay in diagnosis is the single most important cause of severe complications in childhood hip sepsis. Most clinicians state that the longest possible delay in treatment without significant joint injury from virulent bacterial infections (i.e. *Staphylococcus aureus*) is four to five days. Obviously, immediate diagnosis is preferred to minimize joint changes. The need for early diagnosis is further clarified by noting that other authors<sup>2,10,11</sup> have

reported satisfactory results in cases treated with early antibiotics and/or aspiration alone. These hips survived because of *early* treatment.

Diagnostic delay has been classically reported in infants who do not present with the typical clinical picture of painful hip motion, fever and leukocytosis. They are often irritable, afebrile, have a normal white blood count and show only minimal limitation of motion in the affected hip. Missed sepsis in this age group often results in severe bone destruction, trochanteric overgrowth, pathologic dislocation and significant limb length inequality.

We have seen several infants with hip sepsis who were referred with diagnoses of congenital hip dislocation (Fig. 1). The two conditions are distinguished by carefully determining whether the infant has pain with passive hip motion. Congenitally dislocated hips are never painful. Soft tissue swelling may help but is a later sign. In hip sepsis, roentgenograms usually show relatively normal acetabular development, while in a congenitally dislocated hip the acetabulum is abnormal. The frequency of this diagnostic error suggests that making the distinction in an infant remains a challenge even for experienced orthopaedic surgeons.

Our review of the sequelae of childhood hip sepsis suggests older children have a similar delay in diagnosis and treatment and an associated poor prognosis. Of thirty-six children with the diagnosis of pyarthrosis of the hip, seven (19.4 per cent) had the onset of their disease between the

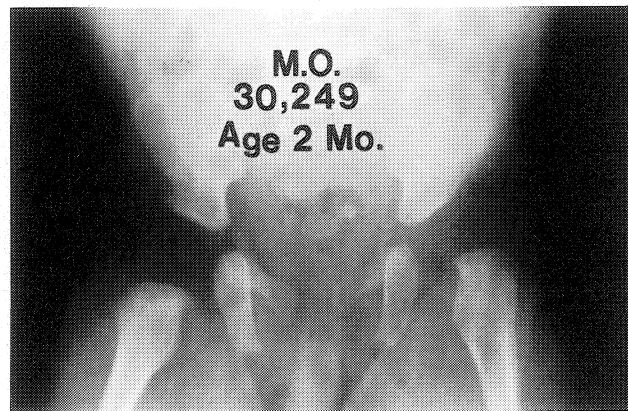


Fig. 1A. Patient age two months incorrectly diagnosed as having CDH by an outside physician.

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†Texas Scottish Rite Hospital, 2222 Welborn Street, Dallas, Texas 75219.

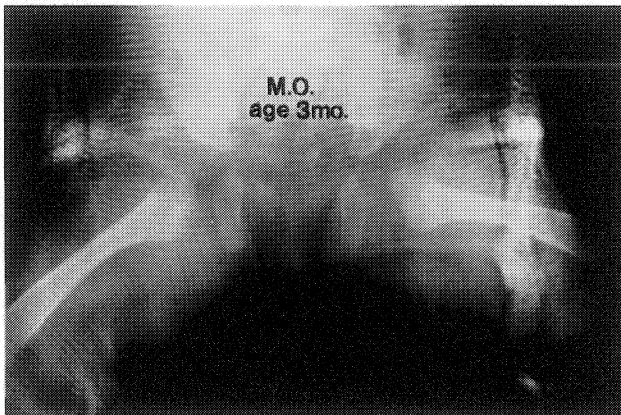


Fig. 1B. In synthetic spica cast applied to treat supposed CDH.



Fig. 1C. After presentation to our hospital. Radiographs demonstrate lateralization and avascular changes.



Fig. 1D. Arthrogram showing flattening of the femoral head.

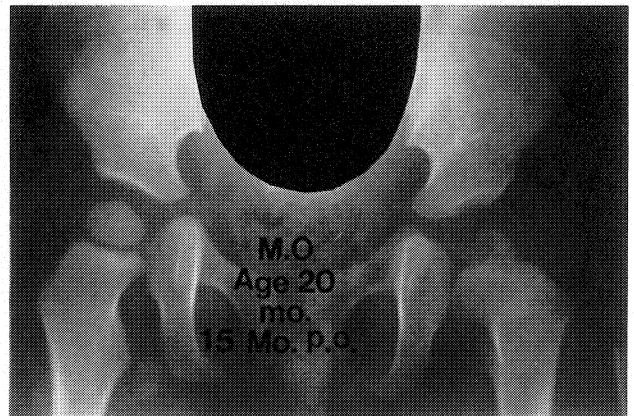


Fig. 1E. Fifteen months after delayed surgical drainage via an anterior approach. The changes of avascular necrosis are apparent.

ages of nine and thirteen years. All were seen by a physician within seven days of the onset of symptoms, but in many cases the physician did not consider hip sepsis upon first encountering the patient. Definitive treatment was delayed from seven days to fourteen months. Excluding one patient with a fourteen month delay, the remaining six patients had a mean delay of eighteen days from the onset of symptoms to definitive treatment. Many physicians apparently think of hip sepsis as a disease only of early childhood, thus the great delay in diagnosis.

In our series the typical history was one of fever with vague groin, thigh or knee pain that was treated with oral or intravenous antibiotics on an empiric basis. In one case (Fig. 2) the diagnosis of hip sepsis was considered on the second day of symptoms. This patient was given intravenous antibiotics for eight days before surgical drainage and later developed avascular necrosis and a slipped capital epiphysis. Two subsequent operations were required before a solid, painless fusion was obtained. In many patients the correct diagnosis was not considered until fulminant sepsis developed, leading to comprehensive reevaluation.

Previous reports have not emphasized the poor prognosis in older children. Patterson did report an eleven year old with a thirty-five day delay in diagnosis and at least one other author has noted the overall poor outcome<sup>4,13</sup>. A brief history of our older patients is given in Table 5.

#### Approach to Diagnosis

Suspicion is paramount. The typical child with hip sepsis is febrile and irritable with a flexed, abducted hip that is extremely painful to move. Initial studies should include a complete blood count with differential, erythrocyte sedimentation rate, blood cultures and roentgenograms of the pelvis. The white blood cell count is usually elevated although it may be normal. The sedimentation rate becomes elevated two to three days after onset. It is in the 50–100 mm/hr range in 85–90 percent of patients but is less than 20 mm/hr in 10–15 percent. Roentgenograms may demonstrate capsular swelling, an important early sign. Lateral subluxation of the femoral head suggests that the process is at least several days old. Nuclear scanning with

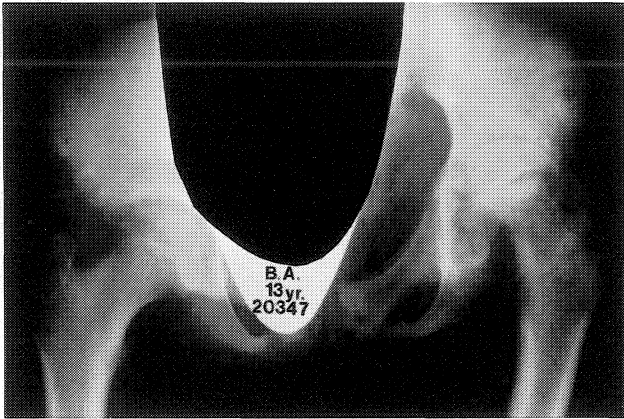


Fig. 2A. Thirteen-year-old child who presented to our center four months after left hip sepsis in which drainage was delayed by ten days. Note severe destruction and avascular necrosis.

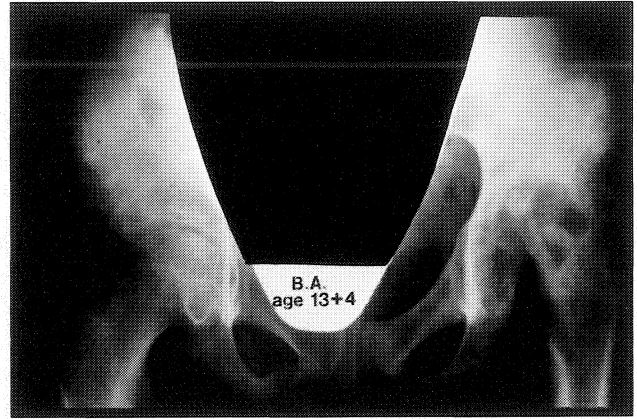


Fig. 2B. Further spontaneous dissolution of the head.

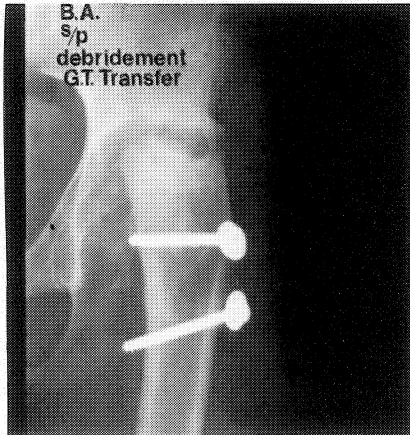


Fig. 2C. One year status post debridement and transfer of the greater trochanter. This provided initial stability but the hip later became painful.

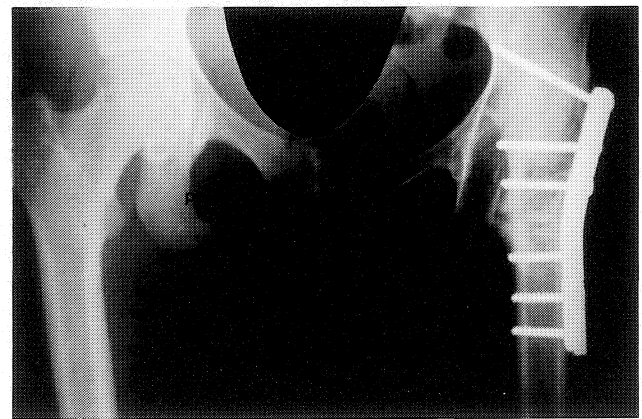


Fig. 2D. After further grafting of the joint to obtain fusion and subtrochanteric osteotomy to correct adduction deformity.

Technetium-99mm polyphosphate may aid in differentiating transient synovitis of the hip or osteomyelitis of the pelvis from septic arthritis. In addition to synovitis and pelvic osteomyelitis, the differential should include rheumatoid arthritis, rheumatic fever and hemophilia<sup>13</sup>.

Once sepsis is suspected, the hip is aspirated from a medial or lateral approach under fluoroscopic control, ideally under general anesthesia. A distended hip is usually not difficult to enter. When necessary, injection of radiopaque dye will confirm the intraarticular position of the needle tip. Any fluid obtained should be gram stained. Aerobic, anaerobic, mycobacterial and fungal cultures are obtained. Immediate transport of all cultures to the laboratory by a member of the surgical team will greatly improve the yield. The laboratory should save the pathogen for future serum bactericidal and inhibition testing. Study of synovial fluid with immunoelectrophoresis or latex agglutination for bacterial antigen will occasionally be helpful in determining the probable pathogen when cultures are negative<sup>9</sup>.

## Treatment

If the results of aspiration confirm sepsis, immediate surgical drainage is indicated. Occasionally aspiration will be equivocal or negative. As noted by Patterson, aspiration of a hip is not always reliable and the morbidity of an unnecessary arthrotomy is negligible compared to the potential catastrophe of neglected infection. Accordingly, he recommends surgical drainage for all suspected septic hips, even if the aspiration is negative. This philosophy led to fifty normal hips at follow-up in a series of fifty septic hips. This 100 per cent success rate is probably related to his policy of early drainage of all hips with suspected sepsis. During the same period, eleven additional hips which were not infected were drained unnecessarily with no adverse sequelae<sup>14</sup>. This policy of surgical drainage is similar to the general surgery philosophy of exploring all suspected infected appendices. They accept that they will operate on a certain percentage of normal appendices to avoid the excessive morbidity associated with delayed sur-

gery. The severe morbidity associated with delayed drainage in hip sepsis validates Patterson's aggressive philosophy. A surgeon should not be embarrassed by occasionally draining a suspected septic hip which proves not to be infected. Since medicine is not an exact science, erring on the side of over-diagnosis and treatment is accepted in conditions where failure of early diagnosis and treatment leads to unacceptable morbidity.

We routinely use an anterior approach to the hip with a cruciate capsulotomy and copious irrigation with normal saline. We prefer the anterior approach over the posterior because it affords better visualization, but more importantly, it allows for concentric reduction in subluxed hips. The anterior approach avoids further embarrassment of the blood supply to the femoral head as well as postoperative subluxation which may result from posterior exploration<sup>8</sup>.

With delay of several days, the femoral head may become fixed in a subluxed position (Fig. 3). Surgeons often assume that this lateral position is due only to the increased intraar-

ticular volume (pus) and that the hip will spontaneously reduce once it is drained. This is clearly not the case (Fig. 3). Synovial hypertrophy, capsular stretchig, and secondary adductor and psoas spasm may cause the hip to remain subluxed following drainage in patients with even a modest delay in diagnosis. The alert surgeon will recognize this picture prior to drainage and will use an anterior approach to facilitate reduction. In more difficult cases, a percutaneous adductor tenotomy and anterior intramuscular psoas tenotomy may be added to assure maintenance of reduction. Postoperative cast immobilization is necessary, with reduction confirmed radiographically. We use the anterior approach to the hip in all cases. In patients with very early diagnosis and no lateralization on radiograph, a posterior approach is probably acceptable.

In patients with suspected femoral neck osteomyelitis, drilling of the metaphysis below the growth plate should be considered at the time of drainage. The specific indications for neck drilling remain unclear and we do not have a strong opinion regarding its use.



Fig. 3A. Radiograph demonstrating residual subluxation immediately following surgical drainage via a posterior approach for left hip sepsis.



Fig. 3B. At the time of drainage, the hip would reduce with flexion and abduction.



Fig. 3C. Attempt to maintain hip reduction in a commercially available splint.



Fig. 3D. The hip remains subluxated.



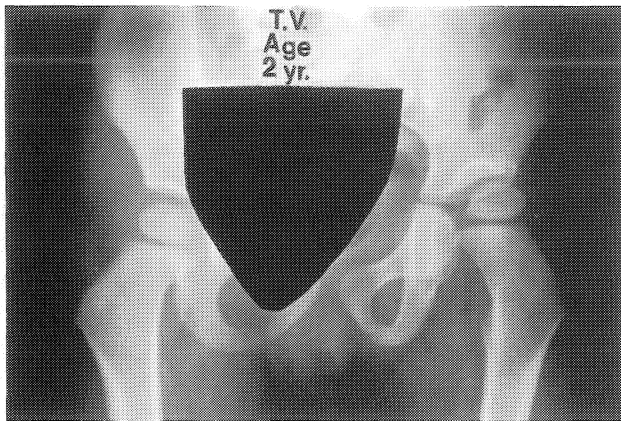


Fig. 3E. One year after drainage, the hip remains lateralized.

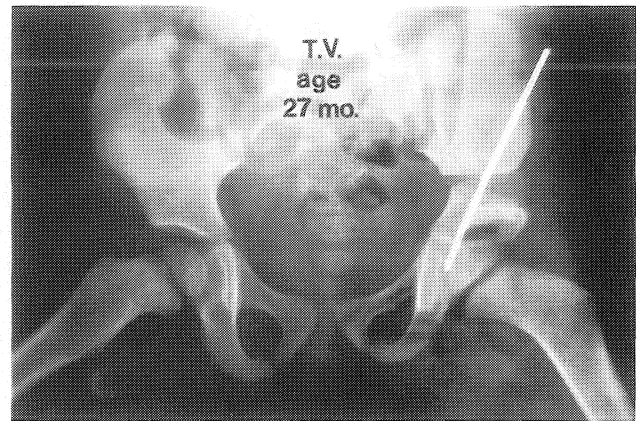
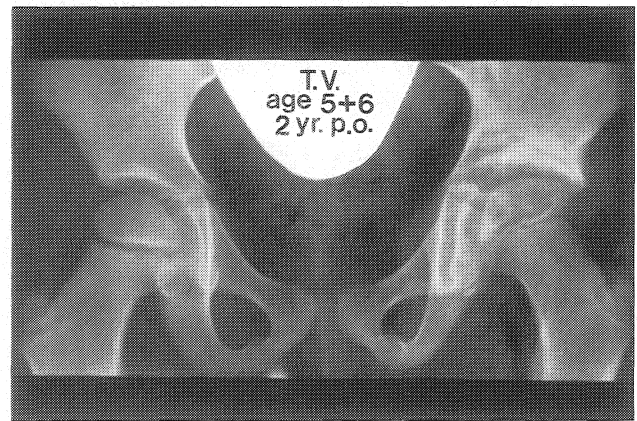


Fig. 3F. After innominate osteotomy and capsulorrhaphy performed at age twenty-seven months.

Fig. 3G. Two years later the hip remains well centered and function is good.



After arthrotomy, the hip is closed over a non-collapsible drain which is left for twenty-four to forty-eight hours. Excellent results have been reported with use of suction irrigation, passive drainage or no drainage at all. We doubt that the method of drainage is an important factor in a hip which has been diagnosed early and adequately drained. Although traction, triple diapering or use of a commercially available splint may be adequate immobilization in very acute cases, we feel that spica immobilization for four to six weeks in a stable reduced position is the safest post-operative policy in any case with preoperative subluxation.

### Antibiotics

Our recommended medical management is derived from the experiences of Nelson, McCracken, Bucholz, et al.<sup>1,9</sup>. The initial intravenous antibiotic is determined by the gram stain and the most likely pathogen according to the patient's age (Tables 1 and 2). This selection is important since in most series approximately 30 per cent of patients have negative bacteriologic studies<sup>4,5,6,11</sup>. When cultures are positive, antibiotics are changed appropriately.

Intravenous antibiotics are continued for a minimum of five days, then a transition to appropriate oral antibiotics

Table 1

#### Most common bacteria in septic arthritis according to age:

Premature neonates	} S. aureus, gram negative
Healthy newborn (up to 1 month)	
Under 2 years of age (overall)	H. influenza
Over 2 years of age	S. aureus

(Bucholz, Nelson, et al.<sup>1,6,12</sup>)

Table 2

#### Recommended initial antibiotic therapy:

Premature infants and neonates	Methicillin + gentamicin
Children less than 5 years of age	Nafcillin + chloramphenicol
Children greater than 5 years of age	Nafcillin

(Bucholz, Nelson, et al.<sup>1,6,12</sup>)

is made, based on culture and sensitivities (Table 3). An early change to oral antibiotics is made only if: (1) the patient is improving clinically; (2) the patient is able to swallow medication; (3) the organism has been identified and is sensitive to oral medications; and (4) the laboratory is able to perform serum bactericidal levels. The course of antibiotics varies according to the pathogen but usually

Table 3

**Suggested dosages for oral antibiotics following initial intravenous therapy for the treatment of hip sepsis:**

- Amoxicillin – 100 mg/kg/day in 4 doses
- Ampicillin – 150 mg/kg/day in 4 doses
- Cephalexin – 100 mg/kg/day in 4 doses
- Chloramphenicol – 75 mg/kg/day in 4 doses
- Cloxacillin – 100 mg/kg/day in 4 doses
- Dicloxacillin – 75 mg/kg/day in 4 doses
- Cefaclor – 150 mg/kg/day in 4 doses

(Bucholz, Nelson, et al.<sup>1,6,12</sup>)

runs for a minimum of three weeks. The recommended duration of antibiotic therapy according to bacteria is listed in Table 4.

Table 4

**Recommended *minimum* duration of antibiotic therapy according to organism.**

Gonococcus	}	10 days
Streptococcus		
H. flu		
Staph aureus		3 weeks
Coliforms & pseudomonas		4-6 weeks

(Bucholz, Nelson, et al.<sup>1,6,12</sup>)

If the patient's clinical response is slow or if the weekly white blood cell count and sedimentation rate do not fall to within the normal range, other foci of infection must be considered. In most cases, medical management is best performed by the pediatric service and not the orthopaedic surgeon. Oral antibiotic therapy is difficult and tedious and requires expertise not only on the physician's part but also on the part of the laboratory and other ancillary personnel. If the surgeon elects this early transition to oral antibiotics, he must have a sound working knowledge of the method and its pitfalls. Oral therapy is less expensive due to shorter hospitalization and is more comfortable for the child<sup>9,12</sup>

**Reconstructive Procedures**

Late complications from hip sepsis range from mild irregularity of the hip joint to total head and neck destruction with an unstable hip and pathologic iliac dislocation. Limb length inequality is commonly 5 to 6 cm or more at maturity in patients with neonatal sepsis and complete hip destruction. Therefore, children with severely involved hips should be followed with orthoroentgenograms on a regular basis. In our experience epiphysiodesis has often been performed too late with residual undercorrection.

Whether late operative procedures to reduce and stabilize a damaged hip are effective remains controversial. Traditionally little has been done and the femoral head is allowed to remain dislocated and high-riding. Hallel and

Salvati recommend stabilization by trochanteric arthroplasty including varus osteotomy<sup>7</sup>. When successful, this procedure should improve the limb length inequality and hip stability, and provide a better anatomic condition for possible later hip reconstruction. They recommend this procedure in young children with an unstable hip and insufficient head or neck for open reduction. To our knowledge they have not published results of long-term function. Freeland et al. has the largest series of trochanteric arthroplasties and report their best results when surgery is performed at or near skeletal maturity<sup>3</sup>.

We feel that reduction may be indicated in certain cases which have a residual femoral neck and/or head. Figure 4 illustrates this technique, which includes a subsequent varus osteotomy to direct the limb into the weight-bearing position while retaining hip stability.



Fig. 4A. Right hip dislocation two months after the onset of sepsis with delayed drainage.

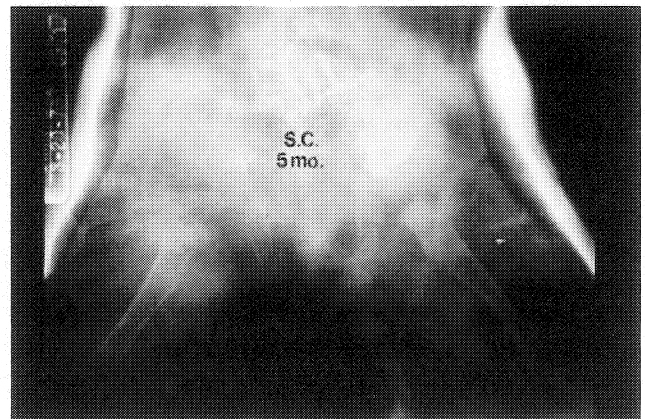


Fig. 4B. Reconstructive open reduction of head remnant including capsulorrhaphy performed at age five months with post-operative immobilization in wide abduction.

In less severe cases with a deformed, enlarged or partially subluxed femoral head, acetabular reorientation (Salter) or enlarging (Chiari) procedures can be considered. Figure 5 demonstrates a child with hip subluxation and

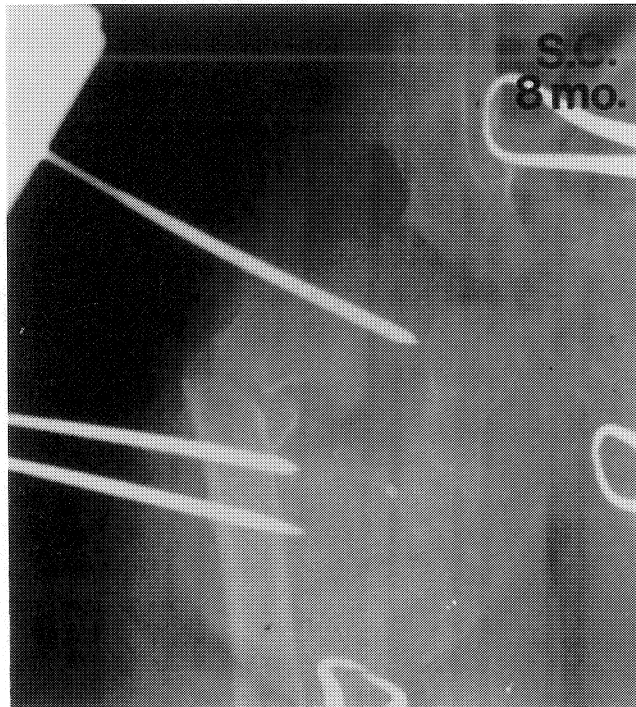


Fig. 4C. Subtrochanteric osteotomy performed two months later to improve limb position yet maintain reduction.

limb length inequality secondary to sepsis who was treated by quadrilateral type innominate osteotomy. This procedure improved femoral head coverage and, in effect, lengthened the limb. The Chiari osteotomy is a more forgiving and effective method for improving coverage of an enlarged, deformed femoral head and is usually selected when improvement of head coverage is desired.

The issue of greater trochanteric arrest or transfer for correction of overgrowth remains unsettled. Hallel and Salvati recommend neither, and instead advise abduction (valgus) osteotomy to improve the mechanical efficiency of the hip abductors. All considerations for late surgery to reduce hips, improve coverage, etc., must be weighed against the finding that most patients with high iliac dislocations following sepsis remain pain-free without significant functional limitations.

#### Sequelae in Older Children with Hip Sepsis

Total joint destruction in the older child is a severe problem noted in our patients (Fig. 2) and the report of Gillespie<sup>4</sup>. The hips are usually painful, in poor position and undergo progressive femoral erosion and shortening. Gillespie noted that early massive debridement in these children was often excessive, contributing to severe limb length inequality. He noted that not all radiographically "dense" bone is truly sequestrum and that conservative debridement may allow for ankylosis in a good position. Most older patients in our series were treated by hip fusion

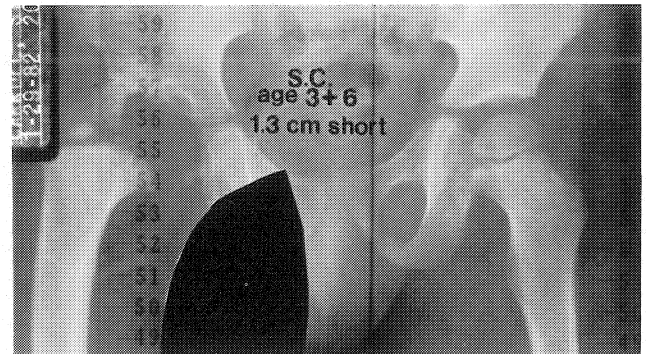


Fig. 4D. Subsequent orthoroentgenogram. The neck remains reduced which minimizes instability and limb length discrepancy.

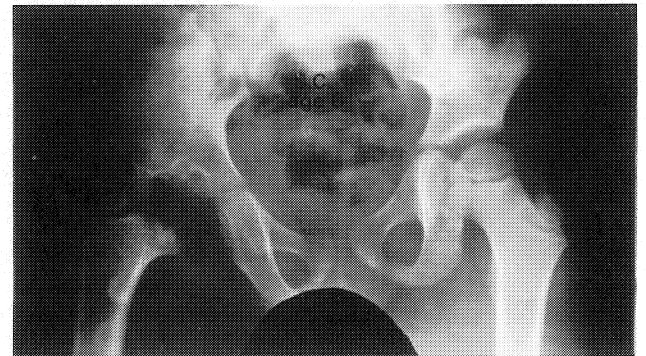


Fig. 4E. Last follow up at age six years.

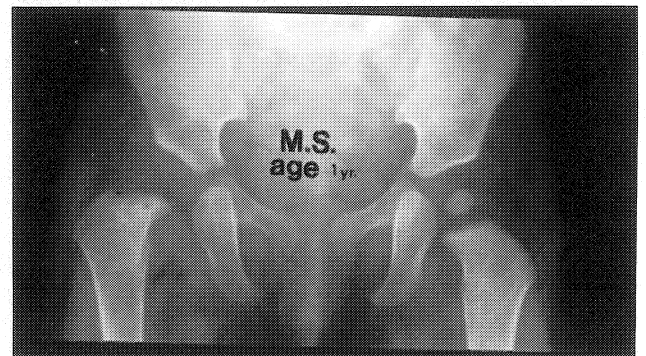


Fig. 5A. One year old child who presented with a several week history of right hip pain. The hip was immediately drained anteriorly.

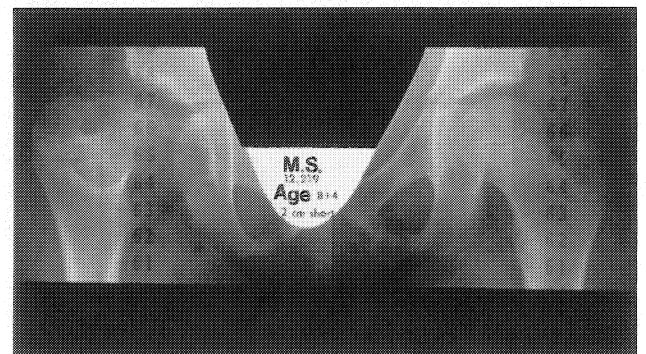


Fig. 5B. Age eight years, four months. Patient has coxa vara and a lateralized, deformed head. Greater trochanteric overgrowth is also noted. The limb is 2 cm short.



Table 5

Analysis of children with onset and treatment of hip sepsis after age nine years						
TEXAS SCOTTISH RITE HOSPITAL REVIEW						
Diagnosis	Age At Diagnosis	Delay in Diagnosis	Etiology	Number Surg.	Outcome	
1. BK	Septic R hip	11+1	8 days	S. aureus	2	Fusion with 1.5 cm LLD
2. BA	Septic L hip	12+7	11 days	S. aureus	4	Hip fusion—6 cm LLD
3. BW	Septic L hip	13	8 days	S. aureus	6	Painful cup arthroplasty—2.5 cm. LLD
4. JV	Septic R hip	11	14 days	B. strep	6	Hip fusion—lost to F/U
5. SL	Septic R hip	9	14 months	No growth	2	Hip fusion—lost to F/U
6. CP	Septic L hip	10+8	7 days	S. aureus	4	Hip fusion
7. NW	Septic R hip	11+6	2 months	S. aureus	4	Unstable hip

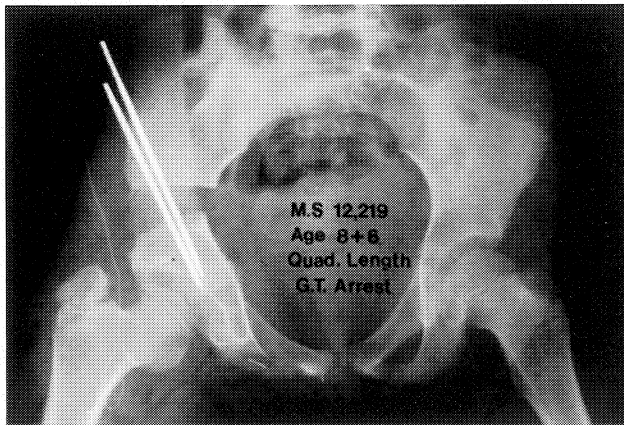


Fig. 5C. Intra-operative view after variant of Salter osteotomy using a quadrilateral bone graft to improve limb length. The greater trochanter was also arrested.

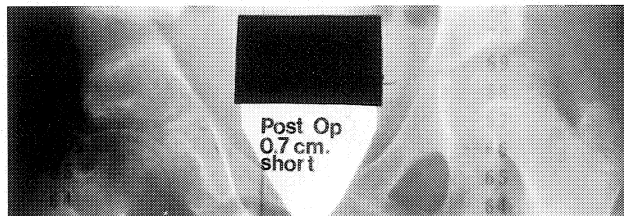


Fig. 5D. Immediately after surgery. The discrepancy is now 0.7 cm.

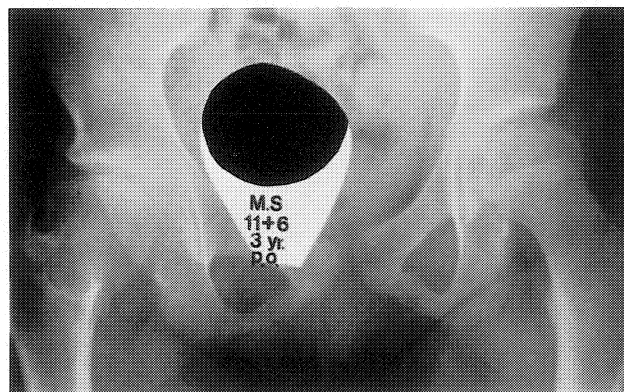


Fig. 5E. Three years after surgery. The damaged head remains covered and the child has no symptoms.

with satisfactory functional results, although more than one attempt at fusion was sometimes necessary.

### Summary

With the increasing availability of well trained orthopaedic surgeons and pediatricians, early diagnosis and effective treatment of hip sepsis is now common. Despite this improved circumstance, poor results continue to be seen. We have outlined our approach in acute cases and feel that the principles of early surgical drainage and appropriate antibiotics provide excellent results in most cases. Empiric surgical drainage in cases where hip aspiration results are equivocal is advised. Post-surgical residual subluxation remains a significant problem in subacute cases and is best avoided by an anterior approach for drainage, adductor and/or psoas release as indicated and cast immobilization in a reduced position.

Hip sepsis can occur in adolescents and teenagers. Because of low index of suspicion in this age group, diagnosis and treatment are often delayed, resulting in a poor outcome in many patients.

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# MYOFIBROBLASTS AND THE CARPAL TUNNEL SYNDROME

William D. Engber, M.D.\*

In 1971, Gabbiani and associates<sup>1</sup> described a type of cell which had characteristics of both fibroblasts and smooth muscle. They coined the term myofibroblast to describe this hybrid cell. Morphologically (Fig. 1), these cells are characterized by 1) a well organized system of cytoplasmic microfilaments containing electron-dense bodies similar to those seen in smooth muscle, 2) nuclei with surface irregularities usually not present in fibroblasts, and 3) surface differentiation with well formed intercellular attachments or desmosomes in some areas and basal lamina in others. Guber and Rudolph<sup>2</sup> presented a comprehensive discussion of this hybrid cell, including its life cycle and physicochemical characteristics.

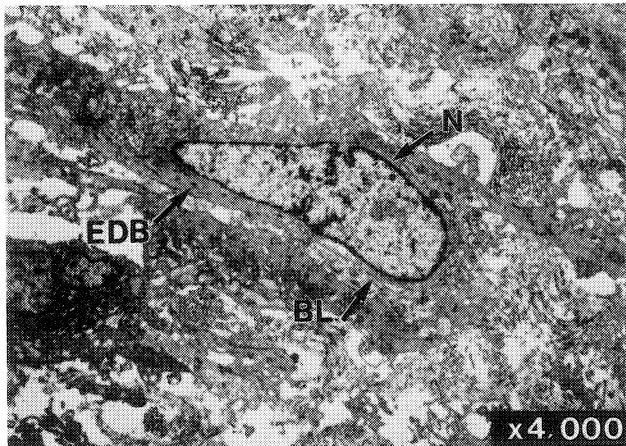


Fig. 1A. Electron micrograph of a typical myofibroblast from a proliferative nodule in a patient with Dupuytren's contracture. Note the morphologic characteristics of a myofibroblast including: 1) cytoplasmic microfilaments containing electron-dense bodies (EDB), 2) nucleus with surface irregularities (N), and 3) surface differentiation such as basal lamina (BL).

The presence of these cells in human tissue and their possible role in pathologic processes have been documented by several groups. Ryan and associates<sup>3</sup> identified these modified fibroblasts in human granulation tissue and suggested they may be the active component in wound contraction. These cells have been identified in Peyronie's disease and in the capsular scar tissue around surgical implants<sup>4</sup>. Myofibroblasts have also been documented in

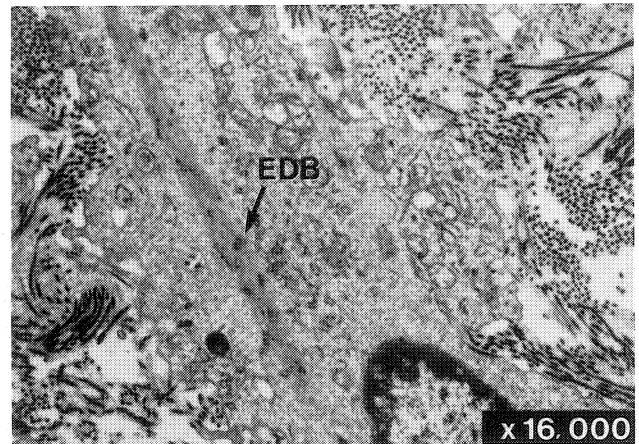


Fig. 1B. Higher magnification showing microfilaments containing electron-dense bodies (EDB).

both Dupuytren's disease and Ledderhose's disease<sup>5</sup> and correlated with recurrent contractures in surgically treated patients with Dupuytren's disease<sup>6</sup>. Legge, et al.<sup>7</sup> have proposed a mechanism by which the longitudinal contractures occur in the palmar aponeurosis in Dupuytren's disease. Their hypothesis suggests the myofibroblast is an active contractile cell that produces "a small amount of shortening" in the collagenous stroma. The cumulative effect of many such cells functioning sequentially is a macroscopic longitudinal shortening or contracture of the palmar aponeurosis. As a result of this shortening, the collagen fibers assume a thicker helical configuration.

The carpal tunnel syndrome is a well known clinical entity characterized by numbness and paresthesias in the median nerve distribution of the hand. Frequently the symptoms are worse at night and may be accentuated by forced flexion of the wrist. Motor involvement may also be present with weakness and atrophy of the median innervated thenar muscles. The known etiologies of this syndrome include rheumatoid arthritis, pregnancy, amyloidosis and myxedema, but most cases are idiopathic.

The anatomic cause of the carpal tunnel syndrome is compression of the median nerve at the wrist as it passes through the rigid canal formed by the carpal bones and the deep transverse carpal ligament. Also passing through this canal are the flexor digitorum profundus, flexor digitorum sublimis and flexor pollicis longus tendons. Thus the median nerve may be compressed either intrinsically by increasing the volume of the contents of the canal (Fig. 2) or extrinsically by decreasing the size of the canal (Fig. 3).

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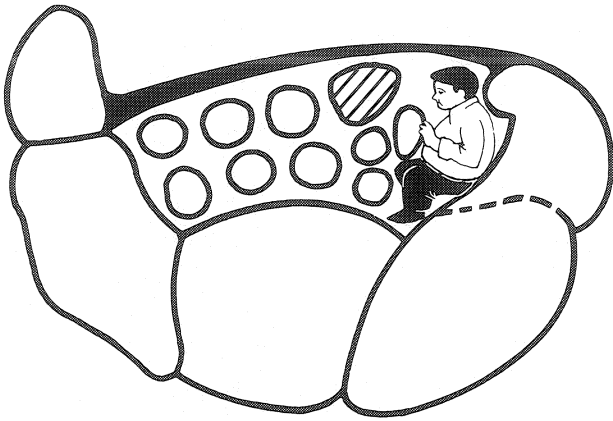


Fig. 2. Cross section of the carpal canal demonstrating intrinsic compression by increasing the contents of the canal.

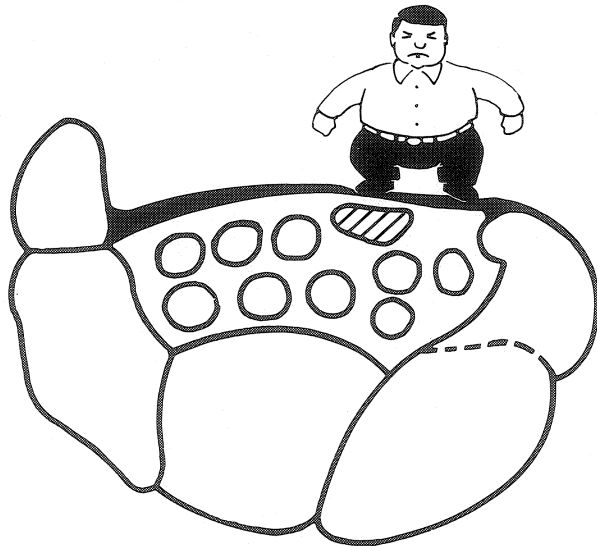


Fig. 3. Cross section of the carpal canal demonstrating extrinsic compression by decreasing the size of the canal.

Most systemic causes of the carpal tunnel syndrome can be explained by intrinsic compression. In rheumatoid arthritis, the proliferative synovitis around the flexor tendons increases the volume of canal contents. Since the nerve is more susceptible to pressure than the tendons, symptoms of nerve compression are seen.

The role of extrinsic compression has not been well defined. Decreased canal size is most likely due to contracture and thickening of the deep transverse carpal ligament. In the absence of trauma or significant osseous pathology the bony carpal base of the canal remains unchanged. Yet, conflicting opinions exist as to the possible etiologic role of the transverse carpal ligament in this clinical entity. Lipscomb<sup>8</sup> reviewed histologic material from a "large" number of cases from the Mayo Clinic and stated

that the transverse carpal ligament was never abnormal. He did not mention whether these specimens were studied on an ultrastructural level. Madden<sup>9</sup>, on the other hand, has described abnormal cells with "many myofibroblastic characteristics" which were found in the transverse carpal ligaments in patients with the carpal tunnel syndrome.

The association of carpal tunnel syndrome with Dupuytren's disease<sup>10</sup> (a known myofibroblast associated disease) raised the possibility that myofibroblasts may be related to idiopathic carpal tunnel syndrome. Since myofibroblast mediated thickening and contracture of the transverse carpal ligament would offer an attractive explanation of the etiology of idiopathic carpal tunnel syndrome, we studied specimens of this ligament in twenty patients with this syndrome.

### Methods and Materials

All specimens were collected at operation by one surgeon (W.E.). They were taken from the cut edge of the deep transverse carpal ligament after its division. Each specimen was fixed in phosphate buffered glutaraldehyde and postfixed in cacodylate buffered osmium tetroxide. The tissue was further processed and embedded in epon. One micron sections were stained with toluidine blue and examined by light microscopy. Sixty millimicron sections were stained with uranylacetate and lead citrate and examined by transmission electron microscopy. All specimens were examined in the same way by the same microscopist (D.N.) to assure uniformity of examination.

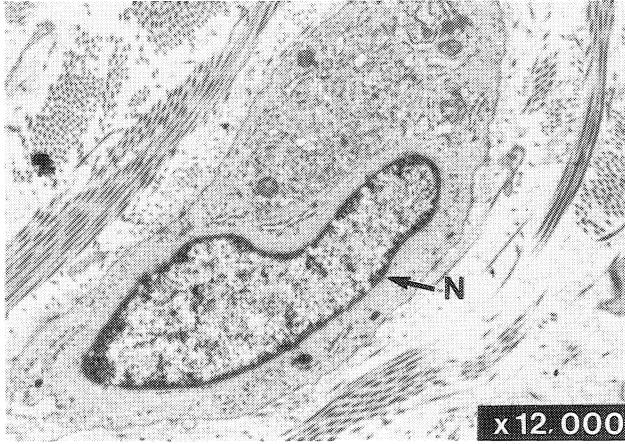
### Results

Cells were found to be sparse in the dense collagenous tissue of the transverse carpal ligament, except in areas where they were the component parts of blood vessels. Vascular tissue had to be carefully avoided when the tissue was scanned, as tangential sections show smooth muscle cells which could give rise to false positive results.

Cells with some myofibroblast characteristics were identified in all specimens (Fig. 4). These cells commonly had cytoplasmic microfilaments but did not contain the electron-dense bodies which characterize myofibroblasts. Pinocytotic vesicles and nuclear lamina were also commonly found. Nuclei with infolding and surface irregularities were occasionally observed. Although the cells in question did have some characteristics of myofibroblasts, the absence of cytoplasmic microfilaments with electron-dense bodies prevents their classification as myofibroblasts.

The absence of evidence supporting the presence of a structure, however, does not prove that the structure is absent. False negative results may arise from either sampling error or time frame error. The chances of sampling error are minimal due to the large number of sections taken on each of the twenty specimens. Gelberman et al.<sup>5</sup>

identified myofibroblasts in Dupuytren's samples only in the nodules. In no instance were myofibroblasts identified in the thick collagenous cords which are similar in composition to the deep transverse carpal ligament.



**Fig. 4.** Electron micrograph of a typical cell from the deep transverse carpal ligament. Note the nucleus with minimal surface irregularity (N) and the absence of microfilaments with electron-dense bodies.

Time frame errors are another potential source of false negative results. Myofibroblasts could possibly be present early in the course of the disease but not be present in later, symptomatic stages when operations are performed. We doubt that this is the case since the preoperative duration of symptoms in these patients ranged from six months to fifteen years. The corresponding nerve conduction studies in these patients showed distal latencies (both sensory and motor) ranging from normal to unavailable evoked potentials.

### **Conclusions**

Although extrinsic compression of the median nerve by contracture and thickening of the transverse carpal ligament is an attractive explanation for idiopathic carpal tunnel syndrome, our observations do not support this

hypothesis. Although examined cells did have some characteristics of myofibroblasts, they did not possess the electron-dense bodies or cytoplasmic microfilaments which are necessary for the unequivocal identification of these cells. Thus, we cannot substantiate that idiopathic carpal tunnel syndrome is a myofibroblast mediated phenomenon.

### **Acknowledgement**

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# TRACTION LESIONS OF THE BRACHIAL PLEXUS: A CRITICAL APPRAISAL OF THE METHODS OF DIAGNOSIS AND A GUIDE TO MANAGEMENT

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Much experience in the treatment of brachial plexus lesions has been gained from high velocity penetrating wounds in wartime. In peacetime the pattern of injury is different; most lesions are caused by high speed traction injuries. Formerly, these lesions were considered irreparable. Prognosis of various lesions served as a guide to possible reconstructive surgery or early amputation<sup>1,2</sup>. This pessimistic approach was confirmed at the 10th Congress of the International Society of Orthopedic Surgery and Traumatology in 1953. All agreed that the nature and severity of the lesion made it unsuitable for operative repair.

Several factors have brought about a change in philosophy. Amputation and upper limb prostheses leave much to be desired. Amputation does not relieve pain as originally thought and prostheses are often rejected on both functional and aesthetic grounds. Significantly, functional recovery has occurred in some patients who have refused amputation. Obviously, methods of diagnosis and determination of prognosis have not been wholly reliable.

With the advent of improved suture techniques, nerve grafting, better diagnostic aids and safe prolonged anesthesia, repair may be feasible and worthwhile<sup>3</sup>. Doubt remains regarding the value of such treatment because of inadequate documentation of the lesion being treated. We have therefore critically appraised the various methods of evaluation and suggest a method of documenting these injuries.

## Clinical Material

One-hundred-twenty patients were seen at the Peripheral Nerve Injury Unit of the Royal National Orthopaedic Hospital under the care of Mr. Donal Brooks. Twenty-nine patients with closed traction injuries of the brachial plexus were considered for surgery and entered a pro-

spective trial. Twenty-six patients were male and three were female. The average adult age was 22.2 years (range fourteen to forty-one). There were two children, one three months old and the other six years. We excluded two patients from operation because of poor medical condition. One other patient was excluded from surgery because of unrealistic expectations and an immature personality. Eighteen patients underwent exploration. The average age of the explored adults was 19.7 years (range fourteen to thirty-five years). The six year old child was also explored. The time elapsed from injury to operation averaged eight months (range two to seventeen months). Twenty-one of the twenty-nine patients had been involved in motorcycle accidents.

All patients had functional assessment, electrical studies, myelography and somato-sensory evoked potential studies (SEP). Peripheral conduction and SEP studies of the exposed brachial plexus were performed intraoperatively. Four patients sustained complete lesions with eighteen out of twenty-five nerve roots avulsed.

## Neurologic Assessment

Horner's syndrome is a reliable sign of lower nerve root avulsion. However, Narakas has shown that plexus exploration revealed a repairable lesion of the middle or upper trunks in 28 per cent of patients with Horner's syndrome<sup>10</sup>. Of the nine patients with Horner's syndrome that we explored, four had at least one other repairable root (44 per cent). Skin temperature and sweating are noted, for return of neurotrophic function may be the earliest indication of recovery.

Assessment of neuromuscular function is usually considered the most important step in evaluating brachial plexus injuries, but undue emphasis may have been placed upon it<sup>4,5</sup>. Nevertheless, it should be recorded meticulously. The chart shown is a modification of that used in the Clinica Ortopedica at Modena. The strength of each muscle is recorded. The chart is constructed with the muscles in descending order of innervation as related to the plexus. Once it has been completed, the level and severity of the lesion can be determined. The chart includes the medial cutaneous nerves of forearm and arm because these help

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to avoid confusion between a peripheral nerve injury and a more central lesion. Our series included fifteen patients who had brachial plexus lesions without peripheral nerve injuries. Three had the upper trunk alone affected, eight had upper and middle trunks affected and four had infraclavicular lesions. The accessory nerve was ruptured in one patient, and the ulnar nerve lacerated in another.

Of the sensory functions, only light touch is tested. The sensory distribution of the brachial plexus is variable and a standard sensory chart has not been universally accepted. One patient had no sensory loss despite complete C5 and C6 root avulsions. The sensory overlap may be explained by her young age at the time of injury.

The Tinel sign is used to indicate nerve regeneration. Tapping along the course of the nerve produces a pins and needles sensation in the nerve distribution. It is *not* painful to elicit and is therefore distinct from the local painful sensation produced when a neuroma is tapped. A neuroma in the presence of regenerating axons will produce both pain and a Tinel sign when tapped. If the Tinel sign persists but fails to progress, complete discontinuity is suspected and spontaneous recovery doubtful. A positive Tinel sign in the supraclavicular fossa is an important indication that at least part of the lesion is postganglionic<sup>9</sup>. Pain elicited when tapping in the posterior triangle of the neck indicates either a recovering cervical plexus injury or postmyelographic inflammation from collection of contrast medium in a myelocoele. Strict interpretation of these signs is important when considering exploration. By this method we were able to distinguish between complete nerve rupture and lesions in continuity in fourteen of nineteen cases. The Tinel and neuroma signs can therefore indicate the type of injury, whether the lesion is pre- or postganglionic and the likelihood of spontaneous recovery.

### **Associated Lesions**

Four patients sustained Volkmann's ischemic contracture. All were associated with conventionally treated forearm fractures where loss of sensation masked compartmental ischemia. One patient with cervical plexus involvement had facial dysesthesias. Three patients had residual symptoms from central nervous system injury.

Twelve patients with brachial plexus lesions had scoliosis not associated with a bony injury. Sternoclavicular subluxation was present in two patients with severe plexus lesions. This discontinuity of the skeletal system indicates a great traction force was exerted on the brachial plexus. Inferior subluxation of the shoulder is always present following complete brachial plexus lesions and often causes the patient concern. Two patients had rupture of the long head of biceps. Narakus reported five cases of rotator cuff rupture associated with a brachial plexus lesion<sup>11</sup>.

Major vascular injuries are sometimes associated with

lesions of the brachial plexus. Swelling in the supraclavicular fossa should make one suspicious of vascular damage. Interestingly, none of our patients with complete avulsions had vascular problems. Major vascular damage was always associated with bone or joint injury. If a clavicle fracture or sternoclavicular dislocation is present, the subclavian artery may be in jeopardy. However, the skeletal injury usually occurs more distally, as in three of our patients where the vascular injury was associated with an infraclavicular lesion of the brachial plexus. The site of the damage is inferior to the clavicle where the neurovascular bundle is tethered by the costo-coracoid membrane. Fracture allows the traction force to be exerted on the neurovascular structures at this level.

### **Further Investigations**

Plain roentgenograms of the cervical spine can demonstrate foraminal encroachment or rotational spinal injury, which may present as abnormal head posture and be incorrectly attributed to the plexus injury.

Other investigations attempt to determine whether the lesion is pre- or postganglionic. The histamine test has been shown to be unreliable. Electromyographic recordings of the paraspinal muscles may be confusing because of the overlap of small muscles in this area.<sup>12</sup>

A sensory action potential (SAP) detected in a nerve supplying an area of anesthetic skin indicates at least one of its roots has sustained a preganglionic injury. The somatosensory evoked potential (SEP) is based on the same principle<sup>6,14</sup>. The median and ulnar nerves are stimulated at the wrist and elbow and recordings are made by means of electroencephalograph electrodes on the contralateral centroparietal region. Recording of potentials distally but not proximally (on the scalp) indicates a preganglionic lesion. The limitation of these two investigations is that they only give useful information about C6, C7, C8 and T1. C5 is checked by determining the nerve action potential (NAP) of the musculocutaneous nerve. A second limitation occurs when a peripheral nerve injury occurs in conjunction with the plexus injury. If the proximal lesion is also postganglionic, electrical conduction studies would indicate only that a postganglionic lesion is present. They cannot demonstrate both lesions.

Myelography was used in the investigation of lesions of C5 and C6 but it was correct in only fourteen of twenty-four root avulsions (58 per cent). If lesions have occurred at two different levels of the plexus, electrical studies would indicate only the more distal lesion. Myelography may demonstrate the more proximal lesion.

### **Provisional Diagnosis**

A summary of findings is made. How the neurologic picture changes over time can be important. Most of the

**NEUROMUSCULAR FUNCTION**

LEVEL	NERVES	ROOTS	MUSCLES	DATE	
ROOT	Accessory		Sterno-mastoid		
	Trapezium n.	(C2)-C3-C4	Trapezius		
	Dorsal scapular n.	C3-C4-(C5)	Levator scapulae		
	Rhomboids n.	C5	Rhomboids		
	Phrenic n.	C3-C4-C5	Diaphragm		
UPPER TRUNK	Long thoracic n.	C5-C6-C7	Serratus anterior		
	L. pectoral nerve	C5-C6-C7	Pectoralis major (clavicular head)		
LOWER TRUNK	Suprascapular n.	C5-C6	Supraspinatus		
			Infraspinatus		
LOWER TRUNK	M. Pectoral n.	C8-T1	Pectoralis major (Sternal head)		
		C7-C8-T1	Pectoralis minor		
POST CORD	Upper subscapular m.	C6-C7	Subscapularis		
	Lower subscapular m.	C5-C6	Teres major		
	Thoraco-dorsal	(C6)-C7-(C8)	Latissimus dorsi		
	AXILLARY NERVE	C5-C6	Deltoid (Anterior (Medial (Posterior		
	RADIAL NERVE		C5-C6	Brachioradialis	
			C5-C6	Supinator	
			C5-C6	Extensor carpi radialis (longus brevis)	
			(C6)-7		
			C6-C7-(C8)	Abductor pollicis longus	
			C6-C7-C8	Triceps	
			C6-C7-C8	Extensor digitorum communis	
			C6-C7-C8	Extensor pollicis (longus) (brevis)	
			(C6)-C7-C8	Extensor carpi ulnaris	
			C7-(C8)	Extensor digiti minimi	
C7-C8	Extensor indicis				
LATERAL CORD	MUSCULO-CUTANEOUS	C5-C6	Biceps		
MEDIAL CORD	MEDIAN NERVE	C6-C7	Pronator teres		
		C6-C7	Flexor carpi radialis		
		(C7)-C8-T1	Palmaris longus		
		(C7)-C8-T1	Flexor pollicis longus		
		(C7)-C8-T1	Flexor digitorum superficialis		
		(C7)-C8-T1	Flexor digitorum profundus II		
	ULNAR NERVE	C6-C7(C8-T1)	Thenar muscles		
		(C7)-C8-T1	Flexor carpi ulnaris		
		(C7)-C8-T1	Flexor digitorum profundus III,IV		
		C8-T1	Hypothenar muscles		
		C8-T1	Abductor pollicis		
		C8-T1	Interossei		
	Medial cutaneous/arm	C8-T1			
	Medial cutaneous of forearm	C8-T1			

**Reflexes**

- a) Scapulo-humeral (?C4-C5)
- b) Pectoralis (C5-T1)
- c) Biceps (C5-C6)
- d) Triceps (C6-C7)
- e) Brachioradialis (C6-T1)
- f) Pronator (C6-T1)
- g) Finger flexors (C6-T1)

**Present Absent Associated Lesions**

- 1) Volkmann's
- 2) Cranial nerve
- 3) Cervical plexus
- 4) Cervical cord damage
- 5) Braininjury

spontaneous recovery from neuropraxia occurs during the first month. A detailed account of the sequence of improvement and the pattern of recovery may be a guide to the anatomical site of injury. For example, recovery may indicate peripheral nerve distribution or root distribution. In the case of complete paralysis, isolated recovery in one nerve root distribution may indicate only that the other roots were more severely damaged. Based on all the available information, a provisional diagnosis is made. The decision to operate can then be made rationally.

### **Patient Expectations**

For most patients, amputation of a limb is a last resort. They are prepared to undergo operations that permit retention of the limb and give a reasonable prospect of the return of limited function. The surgeon should help the patient understand the injury and its implications. The outlook must be explained in realistic terms. The patient must understand the limited return of function and be well motivated before surgery is performed. Patient motivation is an important determinant of the outcome. A paralyzed arm may impose only small limitations in the motivated patient. He may continue his life with minimal handicap. Some patients readily accept mechanical aids and splints, but to others these are totally unacceptable.

Pain may be the patient's main concern and its treatment may be more important than return of function. Its site and nature should be carefully recorded. Severe pain was present in twelve patients. Ten had a complete lesion associated with lower root avulsion, but four other patients had similar lesions without pain. Pain is often assumed to be due to cervical cord involvement, but it can be due to a more peripheral lesion. Several patients have experienced relief of severe causalgic pain after excision and grafting of the damaged plexus.

### **Intraoperative Investigations**

Electrical conduction studies of individual roots and branches are done at the time of operation. Individual roots must be evaluated for possible grafting, whether ruptured or in continuity. More proximal lesions must be identified or ruled out. A somato-sensory evoked potential (SEP) can be elicited by stimulating the stump and recording from the scalp. This is compared to the preoperative SEP<sup>7</sup>.

In four patients the intraoperative SEP was interpreted as indicating the stumps were centrally connected and therefore suitable for grafting. This was confirmed by signs of recovery in all four cases. In three patients root grafting produced useful recovery. C6 grafting in the fourth patient produced EMG recovery eleven months after surgery, but no functional return. This may have been related to his neurofibromatosis.

C5 and C6 were in continuity and appeared normal in three patients. However, SEP indicated a more proximal lesion was present and therefore neurotization was done in two. In one patient the absence of the SAP with a normal musculocutaneous NAP indicated intramedullary rupture of these roots. Neurotization was not done in this patient because of the time elapsed since injury.

Nerve conduction studies were also used as an aid to surgery in cases of lower root avulsion. The median nerve can be microscopically divided into three groups of fascicles and recordings made to isolate the group with the major sensory component. This can be anastomosed to the intercostobrachial nerve to restore some sensory function to the hand.

A descriptive and diagrammatic recording of the operative findings and procedure is made.

### **Comparative Analysis of Investigations**

A comparative study was done in twelve patients who underwent all investigations. We attempted to distinguish between pre- and postganglionic lesions with each of the investigations and compared results to the operative findings.

Clinical judgement using Tinel sign<sup>8</sup>, Horner's syndrome and motor evaluation was correct in forty-three of forty-nine roots tested (87 per cent), myelography in thirty-three out of forty-nine (67 per cent), SAP and EMG in thirty out of forty-one (73 per cent) and SEP in twenty-two out of thirty-five (64 per cent). Considering all these factors, our final judgement was correct in forty-three of forty-nine (87 per cent). Preoperative investigations did not enhance our clinical accuracy of 87 per cent.

### **Conclusion**

The brachial plexus lesion is one part of a multifactorial problem. Consideration must be given to associated lesions, especially when considering surgery.

Clinical evaluation is the most reliable form of assessment of the type (pre- or postganglionic) and nature (continuity or rupture) of the plexus lesion. Intraoperative investigations (SEP) are of great value because they permit diagnosis of intramedullary avulsion of the brachial plexus without the need for intraspinal exploration. The SEP reliably assessed stump function and was helpful in planning the specific surgical treatment.

The complexity of each individual brachial plexus injury may be understood by more systematic recording before, during and after operation. Surgery, if indicated, can be planned in advance and designed to meet the functional needs of the individual patient. With more uniformity of recording, comparisons can be made between results obtained at different centers.



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# TREATMENT OF THE COMPLETE BRACHIAL PLEXUS PALSY

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Injuries to the brachial plexus present major problems in diagnosis and treatment. Often, associated life threatening injuries initially overshadow the brachial plexus injury. The evaluation and treatment of these patients involves many health care team members. Close cooperation and communication among all parties including the patient is essential. Once a definitive diagnosis is made, the majority of the elective treatment involves the prosthetist-orthotist, therapist, and vocational counselor or social worker, in cooperation with the managing orthopaedist.

This paper reviews our existing knowledge of the complete brachial plexus palsy and methods of treatment. The elective care described pertains to those complete injuries which present no opportunity for return of normal nerve function. Since few centers treat large numbers of these injuries, this discussion will be based in part upon the data and experiences of other clinicians.

## Mechanisms of Injury

The two major causes of brachial plexus palsy are childbirth complications and motor vehicle accidents. During childbirth, downward traction on the shoulder increases the angle between the head and shoulder, resulting in injury to or avulsion of the upper (C-5 and C-6) roots. Upward traction on the shoulder increases the angle between the arm and lateral thoracic wall, injuring the lower (C-8 and T-1) roots. Complete injuries involving the entire plexus from C-5 to T-1 may occur.

Motor vehicle accidents, especially motorcycle accidents, cause most brachial plexus injuries. Fletcher<sup>1</sup> reported 180 brachial plexus injuries and noted that 81 per cent of the patients were under the age of twenty-four years and that 77 per cent of the injuries resulted from motorcycle accidents. The injury may result from traction across the arm or across the head. Barnes<sup>3</sup> stated that root tension varies with position of the arm; elevation tenses the lower roots while adduction tenses the upper roots. When the shoulder is forcibly depressed with the arm by the side, as in a motorcycle accident, the greatest

tensile stress falls on the upper roots. During arm abduction and extension the axillary portion of the plexus, particularly the posterior cord, may be stretched across the head of the humerus as it dislocates anteriorly and inferiorly. When the abducted limb is forced behind the trunk and the head is thrust to the opposite side, tensile stress is exerted on all roots. The most severe brachial plexus lesion, a complete palsy, may result.

Traction across the head may also be an important mechanism of brachial plexus injuries. In Fletcher's series<sup>1</sup> most motorcycle accident victims wore helmets and were involved in head-on collisions. The head was forced laterally, away from the shoulder, injuring the plexus.

## Classifications Of Injury

Brachial plexus injuries may be classified by the roots involved, by division of plexus injured, and by Sunderland's severity of injury to specific nerves<sup>5</sup>. Sunderland's five degrees of injury best correlate with prognosis for recovery of the injured nerve. A first degree injury produces temporary loss of nerve conductivity at the site of injury with loss of motor function and muscle tone, and a reduction in proprioception. First degree injuries recover completely and spontaneously. Second degree injuries involve the fascicles, resulting in complete loss of motor, sensory and sympathetic functions. Axon regeneration proceeds distally from the site of injury with proximally innervated muscles returning first. Third degree injury results in interruption of the internal structure of the fascicles. Regenerating axons are not aligned with appropriate tubules and clinical recovery is never complete. Fourth degree injury results in disruption of all fascicles. Complete loss of motor, sensory and sympathetic function occur and no motor or sensory function returns spontaneously. Fifth degree injury is severance of the nerve trunk with loss of motor, sensory and sympathetic function. Although neuromas form, no neurologic recovery is possible.

## Evaluation and Diagnosis

Early, accurate assessment of the plexus injury is necessary but difficult. It requires various neurological examinations and tests. Most important is a thorough physical examination. Additional tests, including myelograms, electromyographs and nerve conduction velocities, are helpful but require experienced interpretative skills. A relatively

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new technique developed by Dr. Steven Jones in England, the spinogram, involves stimulation of the peripheral nerves at the wrist while recording over the plexus at the root of the neck. This non-invasive test departs from the usual procedure of stimulating proximally and recording distally in order to demonstrate preganglionic or root avulsion injuries.

Pain following brachial plexus injury is a common problem. Pain is often not experienced until two to three weeks after injury and increases in intensity until it reaches its peak about six weeks post-injury. It may persist at this level for years. The pain may be described as burning, crushing, stabbing or like severe electric shocks. According to Wynn-Parry<sup>2</sup>, the presence of severe burning pain indicates a preganglionic lesion with root avulsion from the spinal cord. Dermatomal pain distribution correlates with the avulsed root.

### Non-Operative Treatment

Treatment begins with physical therapy to prevent joint stiffness, prevent soft tissue contractures and assist in relief of pain. Pain relief is a monumental challenge. Neuromodulation may be beneficial in some patients. Splints are used to prevent joint contractures or to optimize limb function. These programs require careful reevaluation of the patient at regular intervals to determine changes in muscles and joints. The therapist and orthotist share in encouraging the patient to comply with the prescribed regimen. The exercise program can often be performed by the patient or family at home and only requires checks by the managing team to assess progress.

Careful attention to detail and accurate communication among the team members will clarify goals and alert the physician to change or lack of progress. Decisions are made by the patient based on a sound understanding of all options; thus the patient becomes the controlling factor in care management.

### Operative Treatment

A *complete* brachial plexus lesion implies that all parts of the brachial plexus are involved: all five roots, all three trunks, all three cords, or a combination of root, trunk and cord lesions. If no neurologic recovery has occurred by six weeks after injury, and if physical findings (Horner's sign), paraspinal EMG's, or cervical myelography indicate a preganglionic component to the injury, the prognosis for recovery is poor. The treatment described will concern only the severe, complete injuries.

Given a permanent complete plexus palsy, the pivotal issue is whether the patient will become a successful prosthetic user. Patient sex, age, limb dominance, associated injuries, motivation, experiences with mechanical equipment and the support and experience of the medical care team may influence this decision.

A positive relationship may exist between prosthetic use and the amputation of the patient's dominant hand, especially for those who are unable to transfer dominance to the non-dominant hand. Ransford and Hughes<sup>11</sup> state that if the patient is a manual worker, he or she will more likely use a prosthesis if he or she has difficulty converting hand dominance. They reviewed twenty cases at ten years. Thirteen patients were supplied prostheses, seven were dominant and six non-dominant. Only two of the seven were true prosthetic users. Since only two of twenty cases resulted in true prosthetic users, they recommended amputation and prosthetic fitting only if the dominant limb was affected.

The treatment plan is simplified if the patient is clearly not destined to use a prosthesis. No surgery may be indicated. The patient may elect to retain the extremity for cosmetic reasons. If the patient is athletically inclined or if the flail nondominant limb is a nuisance, above elbow amputation is an accepted option<sup>11</sup>. It may also be indicated for the dominant limb in the patient who will not be a prosthetic user, who has carefully considered the alternatives and who requests the procedure for convenience (Case 1).

Careful consideration of surgical alternatives (including above elbow amputation and shoulder arthrodesis) is important for the potential prosthesis user (Case 2). Rorabeck<sup>10</sup> stated that amputation and fitting done within one year after injury are more likely to result in successful prosthetic fitting than are alternative approaches. He evaluated nineteen patients, fourteen with above elbow amputation alone, and compared them to five patients with above elbow amputation and shoulder arthrodesis. Only one of the five returned to gainful employment while six of fourteen returned to work. Yeoman and Seddon<sup>10</sup> believe that combined amputation and arthrodesis are the treatment of choice within two years of the injury. They reported on seventeen cases of above elbow amputation and shoulder arthrodesis. They compared their results to either no surgical treatment or to total limb reconstruction, but not to above elbow amputation with early prosthetic fitting. For those using a prosthesis the average interval between injury and amputation was sixteen months; for those not using a prosthesis it was three and a half years.

Wynn-Parry reported on fourteen patients who underwent above elbow amputation and arthrodesis within six months of injury. Of these, ten returned to work within one year. Further follow-up revealed that these patients were working without their prostheses, leading Wynn-Parry to a more conservative attitude towards early amputation.

Ransford and Hughes<sup>11</sup> felt that shoulder arthrodesis was necessary for the true prosthetic user. Because the true prosthetic user is rarely seen in clinical practice they

recommended the procedure cautiously. They noted that arthrodesis of the shoulder produces potential for skin irritation over bony prominences, but that the procedure resolved the problem of humeral head subluxation. Prosthesis fitting must be delayed until after fusion has occurred.

### **Prosthetic Fitting Time**

The elapsed time between elective amputation and initial prosthetic fitting is important. Burkhalter<sup>13</sup> believes that early or immediate fitting does not adversely affect wound healing and helps maintain the two handed pattern for activities of daily living. However, only three of the eighty-seven patients in his series had brachial plexus injuries and none of these were using a prosthesis at follow-up. These data suggest that the patient with a brachial plexus injury may differ from other amputees treated similarly.

Rorabeck<sup>10</sup> states that amputation and fitting should be done within one year of injury, suggesting that the two parts together play an integral role in successful prosthetic wearing and return to work.

Leal and Malone<sup>14</sup> report that myoelectric fitting decreases rehabilitation time when compared with conventional immediate fitting. This suggests the important factor is prosthetic control. Patients used to support this conclusion were all working prior to injury and returned to work after fitting. However, no job description or dominant hand data were reported. Additional follow up is needed to clarify long term results.

### **Patient Satisfaction**

Perhaps the most interesting and perplexing data reported deals with patient satisfaction. Fletcher<sup>1</sup> reported on seventy-three patients contacted by questionnaire one year post-amputation. Ninety-one per cent reported wearing the prosthesis regularly at work, and all were glad they chose to have the arm amputated. We are not told what procedures each patient underwent.

Brewerton and Daniels<sup>12</sup> reported that at one year post-injury only 16 per cent of the patients recalled talking with their managing physician about long-term options and outcomes. They emphasized the existence of this void in the care of the brachial plexus injured patient.

### **Case Reports**

Case 1. A 27-year-old male employed as an unskilled laborer suffered a complete avulsion of his nondominant extremity brachial plexus in a motorcycle accident. No spontaneous recovery occurred in six months. When offered amputation with early prosthetic fitting, he replied "I wouldn't use it if I had one." The patient later requested amputation since the arm was "always in the way." An uncomplicated above elbow amputation was completed eighteen months after the patient's accident. Since no prosthetic fitting was

planned, shoulder arthrodesis was not performed. He described mild pain prior to and unchanged since the operation.

Since the accident he has remained unemployed, is now divorced and is currently residing with his parents. He has adapted to one handed activities of daily living. He believes that his care was satisfactory.

Case 2. A 34-year-old male semiskilled service station attendant sustained a complete brachial plexus avulsion when a truck wheel exploded. He suffered a C5 through T1 preganglionic injury to his dominant extremity. He was advised of his poor prognosis. No recovery had occurred within fourteen months. He felt the arm was a nuisance and requested amputation, but sincerely wanted a prosthesis to aid him in his hobbies and with his wheel repair business. An above elbow amputation was completed fourteen months following his injury. A shoulder fusion was not performed, allowing early prosthetic fitting. He was fitted with a conventional above elbow body-powered system. He is able to control the elbow position and the terminal device. He uses both a stainless steel terminal device and an Otto Bock cosmetic hand.

Since his injury the patient has changed extremity dominance and can eat, write and tinker in his shop. He stated that he was never athletic but enjoyed fishing and fishing reel repair. The patient is satisfied with his treatment and continues to gain skills with his above elbow prosthesis.

### **Conclusions**

Brachial plexus trauma results in a spectrum of palsies, including the severe, complete plexus palsy. When this injury includes a preganglionic component, the prognosis for recovery is poor. Early, accurate diagnosis is critical to planning treatment and counseling the patient. Non-operative treatment of the complete brachial plexus palsy, under the supervision of the physical therapist, includes neuromodulation for pain control and prevention of joint contractures. Operative treatment includes above elbow amputation in the nonprosthetic user. For the potential prosthetic user, above elbow amputation and/or shoulder arthrodesis may facilitate prosthetic fitting and use.

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# TREATMENT OF ACROMIOCLAVICULAR SEPARATION

James L. LeNoir, M.D.†

Acromioclavicular separation occurs as a result of a force applied to the superior acromion either by a falling object striking the acromion or by a fall onto the point of the shoulder<sup>1,2,3,5</sup>. Four distinct soft tissue disruptions may allow acromioclavicular separation. First, the conoid and trapezoid ligaments may tear anywhere along their lengths. Second, the lateral clavicle may ride upward after being avulsed from its periosteal bed. Third, the acromioclavicular ligaments may be torn, although this is of lesser significance. Fourth the conoid-trapezoid ligament origin may avulse from or fracture the coracoid. The coracoacromial ligament is not usually torn.

To properly assess the degree of separation, anterior posterior roentgenograms (using hand-held weights and possibly local anesthesia) is required<sup>2</sup>. In partial acromioclavicular separation non-surgical treatment may be acceptable. If complete separation is demonstrable some favor open reduction and fixation between the clavicle and coracoid process<sup>4</sup>. We recommend open ligamentous-periosteal repair, with internal fixation using a Knowles pin across the acromioclavicular joint. We have developed an instrument called an acromioclavicular reducer/maintainer and pin guide\* to make the technique simpler and more accurate (Fig. 1). The instrument anatomically reduces a separated acromioclavicular joint and maintains reduction. A scale on the instrument indicates the required

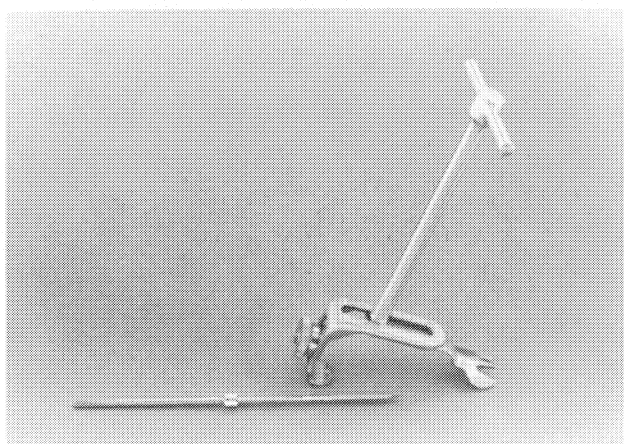


Fig. 1. Acromioclavicular reducer and pin guide.

length of Knowles pin. It also acts as a guide for the acromioclavicular transfixion pin.

The operative procedure is as follows:

1. Routine preparation and draping of the shoulder is performed with the patient in the semisitting position and a sandbag between the scapulae. A sterile stockinette is placed over the arm.
2. An anterior transverse incision is made along the outer one-third of the clavicle and then around the lateral acromial border to the posterolateral process of the acromion.
3. The torn conoid and trapezoid ligaments are exposed. Braided 00 silk Bunnell sutures are placed in the stumps of each ligament and left untied until the AC joint fixation is complete. If torn, the AC ligaments are prepared for repair.
4. The hooked tooth end of the instrument is placed under the depressed acromion process at the site allowing alignment of the acromion with the lateral one-fourth of the clavicle.
5. The instrument is adjusted so that the tip of the clavicular pointer lies over the center of the clavicle near its outer concave curve. This is accomplished by unscrewing the transverse handle to allow either shortening or lengthening of the clavicular pointer arm as necessary. The handle screw is then tightened. Upward pull on the handle elevates the acromion with fulcrum pressure from the clavicular pointer end. Anatomic reduction of the separated acromioclavicular joint is accomplished. It is held in reduction and prepared for pin placement.
6. The proper length Knowles pin is read from the pin scale on top of the instrument. The measurement is from the outer acromion to the pointer tip (Fig. 2).
7. The threaded guide is positioned correctly over the entry site on the lateral acromion edge by adjusting the controlling screw mechanism (Fig. 3).
8. The Knowles pin is advanced into the threaded guide and through the acromion, across the reduced acromioclavicular joint and into the medullary canal of the clavicle. The pointed pin end always falls within the medullary canal of the clavicle. It is advanced as far as the junction of the middle and distal thirds of the bone.

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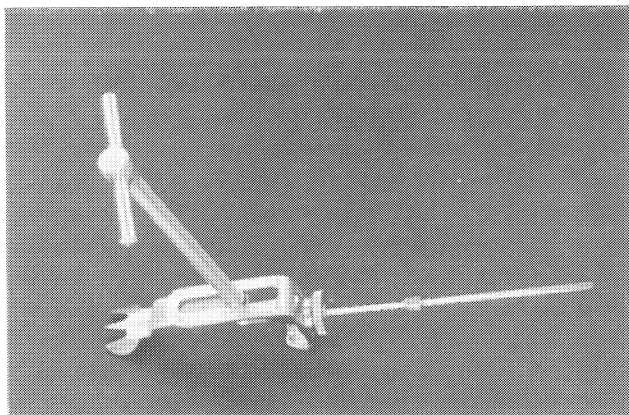


Fig. 2. Pin length scale.

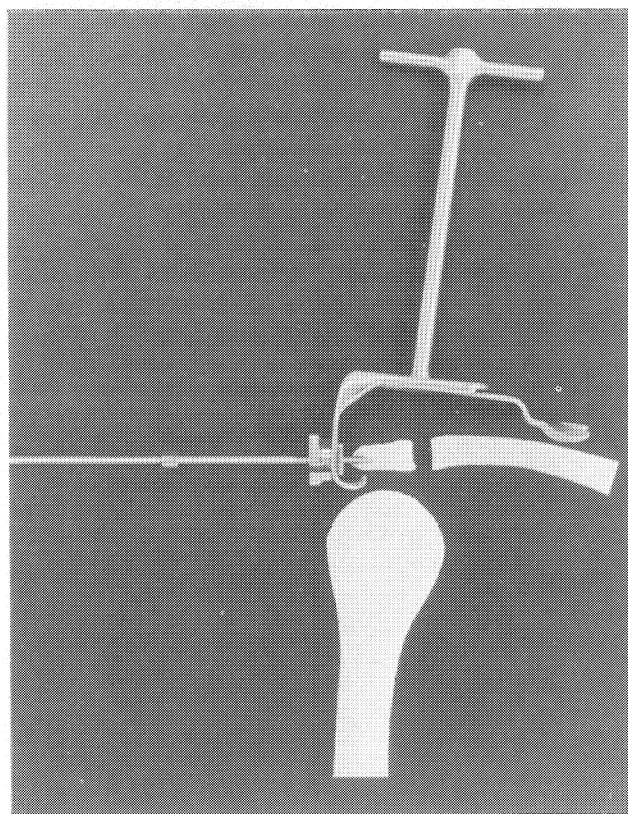


Fig. 3. Representation of pin guide.

9. The threaded acromial pin *guide* is unscrewed and the pin released. The instrument is then lifted off the transfixing Knowles pin.
10. The transfixing pin is advanced into the medullary canal until the hexagonal enlargement of the pin shaft contacts the acromial process or is slightly countersunk (Fig. 4).
11. The previously inserted sutures in the conoid and trapezoid ligament stumps are tied to approximate the torn ends. The periosteal tear and torn acrom-

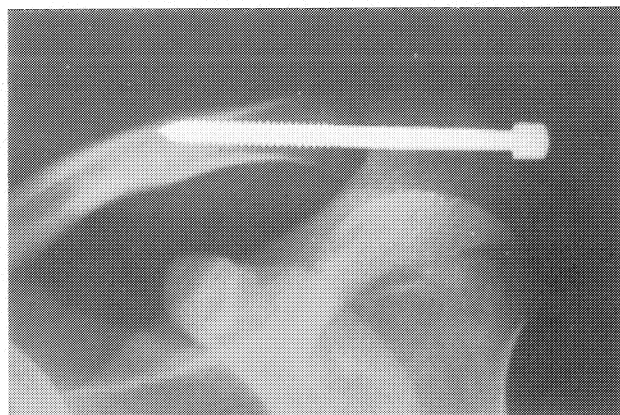


Fig. 4. Roentgenogram showing pin fixation.

ioclavicular ligaments are repaired.

12. The protruding Knowles pin is then bent and divided in the groove just outside of the hexagonal pin head.
13. Wound closure is completed by anatomic layers. The arm is placed in a sling and strapped to the chest using a circumferential elastic bandage. The sling is worn until postoperative pain subsides. Early physiotherapy prevents stiffness.
14. When soft tissue repair is complete, the pin is removed using local anesthetic.

We have treated AC separations with ligament and periosteal repair and transfixion Knowles pins for many years. We have observed excellent maintenance of reduction after pin removal with full recovery of shoulder motion (Fig. 5). The acromioclavicular instrument presented here is designed to make this procedure simpler and more accurate.

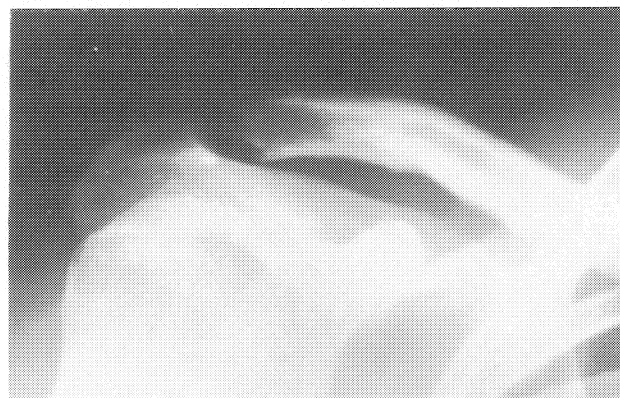


Fig. 5. Acromioclavicular joint after pin removal.

A final word of caution — “Treat the problem, not the coach or the supposed need of the player who does not happen to have a spare shoulder to present to the cause”.

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## BACK SCHOOL: ORGANIZATION, METHODS AND PRINCIPLES

Wayne E. Janda, M.D.\*

Back Schools have been initiated and developed primarily to help back-injured patients return to work or a normal life pattern, and secondarily to prevent future injury through patient education and community outreach. I have been involved with one since 1979. The Instructional Course Lectures on Low Back Treatment<sup>1-4</sup> were the catalysts stimulating our endeavor. The following is an overview of the organization, methods and principles used in developing this Back School.

The Back School utilizes the health team approach that includes nurses, physical and occupational therapists, biofeedback therapists, psychologists and physicians. They all may play a role in either hospital or outpatient care of the back-injured patients. Diagnosis and treatment is initiated by the physician either in the office or in the hospital. The back-injured patient's complaints usually fall into one or more of the following categories:

1. altered sensation, e.g. numbness, tingling or pain in the back or legs;
2. limited mobility, e.g. stiffness or catching in the back or hip (buttock);
3. decreased strength, e.g. weakness in the back or legs; and
4. diminished function, e.g. change in bowel, bladder or sleep habits; or inability to bend, stoop, lift, sit, stand, walk or work.

A pain drawing is usually done by the hospitalized patients. It may also be done by outpatients in the physiotherapy department or the physician's office. This helps the patients define and communicate their pain characteristics to the health care team and it helps the physician categorize the patient's perception of pain into either pathophysiologic or psychologic patterns. The team substantiates the subjective complaints with objective findings. The various team member's assessments or diagnoses are correlated and synthesized by the physician, the team leader.

Initial treatment is conservative and includes one or more of the following:

1. restricted activities or bed rest;
2. medication(s);
3. physiotherapy; and
4. biofeedback training.

To prevent behavioral changes from prolonged bed rest, patients' requests for bathroom or shower privileges are usually granted, but other activities are limited. Medication is used as indicated to reduce inflammation and muscle spasm, to relieve nausea, vomiting or pain, and to promote bowel function and sleep. Physiotherapy (various combinations of ice packs, moist hot packs, ultrasound diathermy, massage, traction, neuroprobe and/or transcutaneous electrical stimulator) may be initiated either as an outpatient after the acute pain symptoms have subsided or as an inpatient at the bedside. These modalities are frequently helpful in relieving or controlling pain. The patient may be instructed to use the TENS or traction, and if effective, manage and control his or her pain without narcotics or other pain medications. Biofeedback training has been used for pain management in selected cases.

Educational materials such as the manual on "Back Care"<sup>5</sup> and the soundslide program on "Back School"<sup>6</sup> are offered to patients by the physiotherapy and education departments. These materials are discussed with the patients by the nurses, physical therapists, or physician. There is no formal or written examination.

When and if pain is controlled, increased activities and therapeutic exercises are initiated to improve mobility and strength. These may be initiated by the patient or the physician and then monitored by nurses, physical therapists and physicians. Hot showers or hydrotherapy in a whirlpool or Hubbard tank are sometimes helpful in improving mobility and allowing progressive strengthening exercises. Williams exercises for the back<sup>5-7</sup> and modified DeLorme progressive resistance exercises for the legs<sup>8</sup> are started. Situps, pushups and wall slides are introduced later as tolerated.

Improved function is simulated through a modified obstacle course in physical therapy including the exercise bicycle, the isokinetic exercise apparatus and weight lifting with barbells. The energy costs of pedaling a bicycle at various loads are measured in terms of METS, the ratio of cardiac work (or metabolism) during the activity compared to the basal value when resting supine<sup>9</sup>. A submaximal exercise test uses a target heart rate which is 70 per cent of the predicted maximal heart rate. Work on the isokinetic apparatus (Orthotron) during contractions of the knee extensors and flexors at varied repetitions per minute are measured in foot-pounds of torque<sup>10</sup>. We do not use the

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recording device displaying maximal isokinetic torque curves. Weight lifting performance is measured by weight and repetitions. A large barbell may be lifted from floor to waist and waist to chest. For heavy weight lifting, the need for a corset or further abdominal muscle strengthening exercises is assessed. During these activities, it is important to observe patient tolerance, postural and lifting techniques, and achievement level. These testing methods allow for performance analysis and they provide, if necessary, a rational basis for work restriction.

Finally and concurrently, just as the student is evaluated by the teacher, the patient's attitude, application and achievement are assessed by the health care team. Physical achievement can be documented by physical or occupational therapists, nurses, and the physician. Attitude and application can be observed and, if indicated, they may be further assessed by Minnesota Multiphasic Personality Inventory (MMPI) testing, psychologic interview or psychiatric consultation to evaluate the patient's motivation toward return to work or a normal life pattern.

If initial conservative treatment fails, alternatives include further diagnostic workup (eg. bone scan, CT scan, myelogram, MMPI, etc.), continued but modified conservative treatment, injections, surgical procedures, or no further treatment with possible vocational retraining.

The unifying principle of the whole man concept was expounded by Beals and Hickman<sup>11</sup> in 1972 when they discovered patients return to work when they feel able to do so, and not when they are medically ready. These authors studied vocational, psychologic and physical factors and found both physical and psychologic factors play an important role in the return to work of both extremity-injured and back-injured patients. They attributed greater complexity of psychopathologic findings in the back-injured patient as the reason why psychologists rather than physicians were more accurate in predicting return to work in this group of patients. They concluded that their studies "document the importance of psychological evaluation for optimum rehabilitation effort and suggest that the whole man concept is a useful, and perhaps necessary, consid-

eration in the rehabilitation of the industrially injured workman"<sup>11</sup>. Although their study was confined to the industrially injured worker on compensation, they believed the various factors applied to other patients as well. These statements are a direct challenge to the practitioner who wishes to limit his evaluation and treatment to the pathophysiologic process in back-injured patients while ignoring the psychosocial processes.

In conclusion, the development of the Back School has been educational, enlightening and intellectually challenging. It has the potential to be a valuable adjunct in diagnosis and treatment of back-injured patients and the prevention of further injuries. It has proved helpful in patient evaluation and selection before surgery or vocational retraining. Our Back School has been modestly successful in its efforts.

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# BONE SCANNING FOR THE EVALUATION OF PRIMARY BONE NEOPLASMS\*

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Bone scans have an established role in the orthopedist's armamentarium for evaluating musculoskeletal disease such as tumor, trauma and infection. Radionuclides and imaging devices have evolved over the past twenty years to provide information about the biologic activity of a given lesion with minimum radiation exposure to the patient.

Technetium-99m, the metastable product of molybdenum-99, has an ideal energy for capture by the gamma camera and has a half-life of six hours which allows satisfactory imaging at a safe radiation exposure of approximately 0.15 rads total body<sup>12</sup>. The Technetium-99m requires binding to one of several phosphorus complexes which are the actual "bone seeking" molecules. Currently the most popular complex in use is methylene diphosphonate (M.D.P.). This combination molecule, Tc99m MDP, is thought to chemisorb to developing crystals of hydroxyapatite within fifteen to thirty minutes of intravenous injection. Bone imaging is delayed two to three hours to allow soft tissue clearance and renal excretion of nonabsorbed radionuclide<sup>16</sup>.

There are two additional potentially useful phases of a bone scan study obtainable from the same injection and radiation dosage<sup>3</sup>. The vascular phase of a lesion is studied by immediate imaging as the radionuclide is injected. During the next sixty seconds, serial images can be obtained as the radionuclide is concentrated in the vascular system. Lesions with increased blood flow will be "hotter" than neighboring soft tissue or bone.

The blood pool image is another early study of the area of interest. It is made one to ten minutes post-injection and reflects more subtle increased vascularity than is seen in the flow study<sup>6</sup>. The low blood flow to bone is reflected in the negative image it projects in this phase.

The static or delayed bone scan is essentially a biologic photograph of the skeleton indicating areas of abnormal bone activity. It can be used semiquantitatively by comparing the bone activity in question to the sacroiliac joint region, as described by Simon and Kirchner<sup>14</sup>. Increased activity is consistent with bone formation seen microscopically whether it represents fracture repair, response to

infection, pagetoid bone formation, epiphyseal plate activity, periosteal reaction or neoplastic bone. Scan activity is dependent upon a vascular supply to carry the radionuclide to the site of cellular activity<sup>1,4</sup>.

For evaluation of primary malignant bone tumors, such as osteosarcoma and chondrosarcoma, the bone scan is effective in localizing or defining the extent of the primary sarcoma<sup>8,11</sup>. Several authors caution that in delayed views tumor-induced regional hyperemia may give the impression of tumor extension beyond its gross and microscopic extent<sup>2,14,16</sup>. This reactive hyperemia is seen primarily in delayed images and, if recognized as such, need not be confused with tumor. High quality gamma camera images obtainable today provide improved resolution and minimize this drawback. If activity is intramedullary, it should be considered part of the tumor or its reactive zone and may actually contain malignant cells. This finding in a six year old girl with osteosarcoma prompted a radical amputation (hip disarticulation) rather than through-bone amputation (Fig. 1). Pathologic assessment proved this interpretation correct.

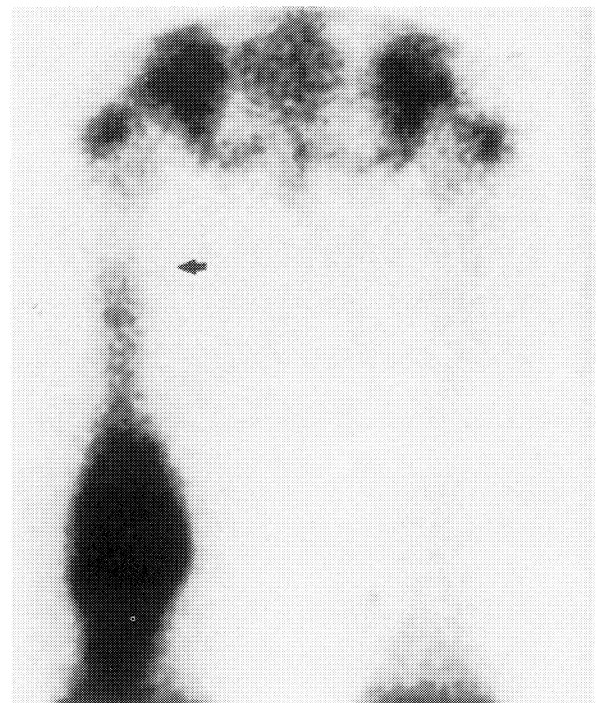


Fig. 1. AP bone scan of the pelvis and femurs. The proximal scan activity corresponds with intramedullary extension of the osteosarcoma (arrow).

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The bone scan also surveys the skeleton for other sites of involvement as part of the staging work-up for malignant bone disease. This is particularly important in tumors where metastases to bone occur, such as in Ewing's sarcoma. Asymptomatic sites of involvement may need to be biopsied to accurately stage the disease. Telfer reports greater accuracy with gallium-67 in identifying occult skeletal disease in a large series of Ewing's sarcoma patients<sup>16</sup>. One might consider gallium scanning in this circumstance.

In most high grade primary bone malignancies there is a significant extraosseous component which may not be apparent on plain radiographs due to lack of mineral density. The vascular phase and blood pool scans aid in defining the soft tissue extent, provide information about its relative vascularity and to a lesser degree show its relationship to large regional vessels (Fig. 2). This easily obtained information may preclude the need for an arteriogram, especially where amputation is planned.

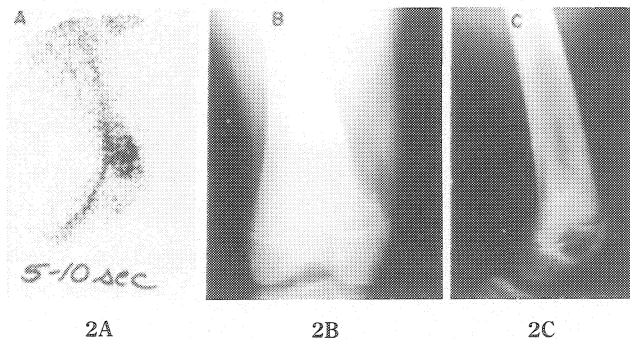


Fig. 2A. Early flow study of the femur demonstrates rapid filling of the tumor. Fig. 2B. AP radiograph of the distal femur. Note cortical destruction and thickening of the soft tissue planes medially. Fig. 2C. Lateral view of the distal femur. Open biopsy revealed Ewing's sarcoma.

The use of bone scan patterns for diagnosis of radiographically benign appearing bone tumors was first suggested by Gilday<sup>6</sup>, but has since received little attention in the literature, except for osteoid osteoma. Attempts to differentiate benign from malignant tumors via bone scan have not produced encouraging results<sup>14,15</sup>.

The three phase bone scan used in conjunction with roentgenograms may narrow the differential diagnosis of bone tumors. Osteoid osteoma is a lesion that radiographically and clinically may be difficult to distinguish from a Brodie's abscess. In our experience the osteoid osteoma bone scan image is like a target with a diffuse zone of increased uptake surrounding a central area of intense activity (Fig. 3), whereas the Brodie's abscess produces diffuse uptake without a "bull's-eye". The diagnosis is confirmed if the flow study reveals increased vascularity to a central nidus. Acute osteomyelitis will produce increased regional blood flow<sup>1</sup> and will not show the "bull's-eye" appearance on delayed views.

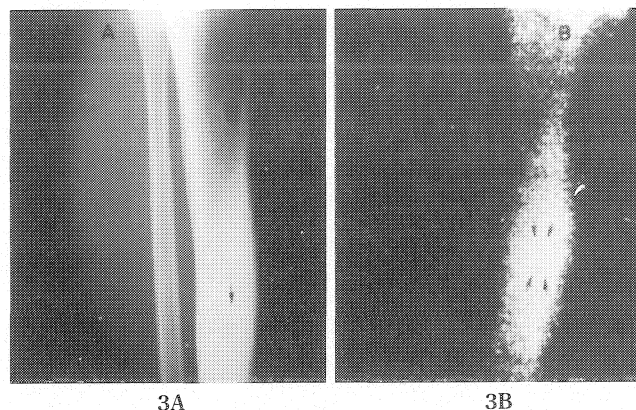


Fig. 3A. Oblique roentgenogram of the tibia reveals a large area of diffuse sclerosis with a radiolucent nidus (arrow). A fibrous cortical defect is seen proximally. Fig. 3B. The skeletal image anterior view reveals intense uptake of the radionuclide in the region of the nidus (arrows). The zone surrounding the nidus is less intense suggestive of a target with a bullseye.

Another benign lesion whose radiographic appearance may give rise to concern is the osteochondroma or exostosis. Although these lesions may enlarge through childhood, their growth in an adult is cause for concern. In a series of twelve patients with multiple hereditary exostoses, we surveyed the skeleton with bone scans to assess biologic activity of individual lesions. We classified lesions as either quiescent or active based on scan activity<sup>10</sup>. Quiescent lesions were those with scan activity equivalent to the parent bone and active lesions revealed activity greater than the parent bone. When biopsied for mechanical reasons the "quiescent" osteochondromas had thin cartilage caps, negative tetracycline fluorescence and no active enchondral bone formation histologically. Scan "active" exostoses frequently appeared mushroom-shaped with an enlarged knobby or bulbous end radiographically. Grossly, these exostoses had thick 4.0-8.0 mm cartilagenous caps, were fluorescent with tetracycline labeling and revealed areas of active enchondral bone formation in the immediate subchondral zone suggestive of a growth plate. Whether any of these "active" lesions were destined to become malignant is unknown. Our feeling was that they should be electively removed in the skeletally mature patient.

Our experience with malignant transformation in an exostosis is limited to three cases, all of which were scan positive. However, there is no clear pattern in the bone scan indicative of malignancy<sup>9,10</sup>. Malignancy is often suggested by pain, clinical signs of tumor growth and cartilaginous tumor size on roentgenograms.

The asymptomatic enchondroma has modestly increased uptake of radionuclide. The increased scan activity is consistent with enchondral bone formation seen microscopically. Scan activity alone should not be the basis for a diagnosis of malignancy. A radionuclide with an affinity for cartilage would be helpful in assessing cartilage growth.



Lesions that appear cystic on plain radiographs have some distinguishing scan characteristics. In the absence of fracture, simple or unicameral bone cysts are avascular on the flow study and relatively "cold" on the static bone scan<sup>6</sup>. Recently this observation proved helpful in evaluating a patient with the presumptive roentgenographic diagnosis of bilateral humeral fibrous dysplasia (Fig. 4). Bone scan revealed bilateral "cold" lesions. Needle biopsies and cystograms confirmed the cystic nature of both humeral lesions.

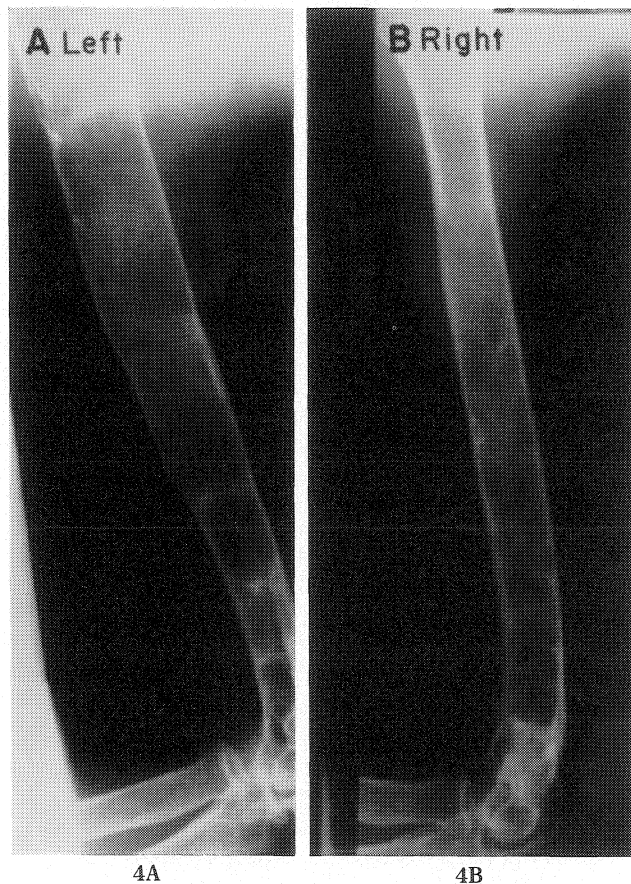


Fig. 4A. The lateral roentgenogram of the left humerus reveals a thinned and expanded cortex throughout most of its length. Fig. 4B. The lateral roentgenogram of the right humerus shows similar extensive involvement but with less cortical thinning and deformity.

Aneurysmal bone cysts show peripheral increased uptake on delayed scan views, but less uptake than one might expect from the relatively aggressive radiographic appearance. The flow study and blood pool images reveal a modest increase in activity peripherally, corresponding to the vascular lining. Early views should allow differentiation between simple and aneurysmal type cysts if the lesions are studied when asymptomatic.

Giant cell tumor is a lytic, aggressive appearing lesion that may be in the differential diagnosis of cystic lesions.

Within seconds, the vascular scan images are distinctly "hot" throughout the lesion (Fig. 5). Static images are intensely hot at the bone-tumor interface. The vascular nature of giant cell tumors helped differentiate a soft tissue recurrence from a bone recurrence because only the soft tissue was "hot" on the vascular phase (Fig. 6).

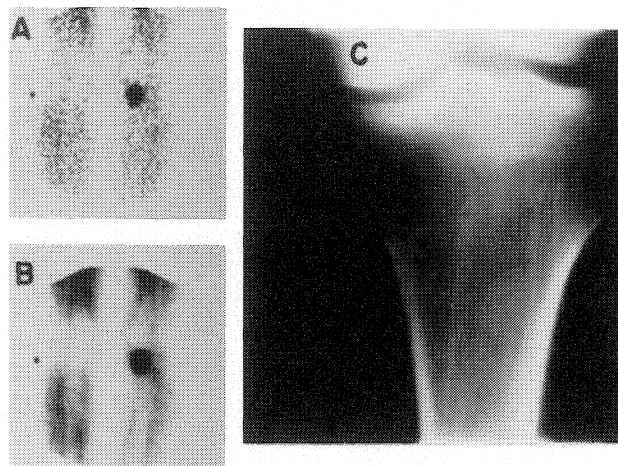


Fig. 5A. The flow study of a proximal tibial giant cell tumor reveals marked vascularity. Fig. 5B. The blood pool image obtained approximately five minutes post radionuclide injection is similar. Fig. 5C. AP tomogram of the proximal tibia.

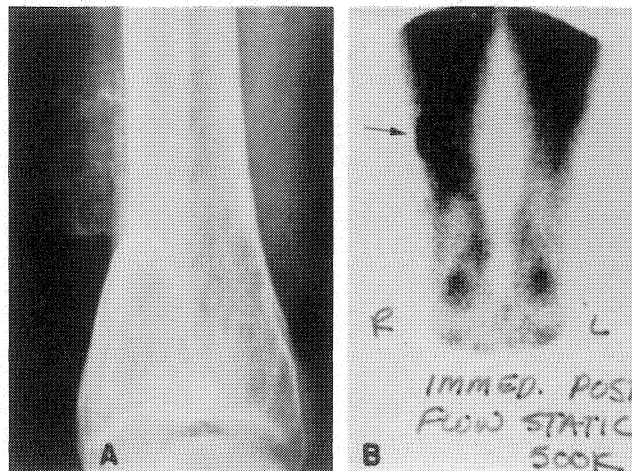


Fig. 6A. Lateral roentgenogram of a soft tissue recurrence of a giant cell tumor. Fig. 6B. The anterior blood pool image reveals tracer uptake corresponding to the soft tissue mass (arrow). The distal tibia has no increased flow and was free of recurrent tumor. The standard skeletal images (not shown) did not differentiate reaction and repair from the recurrence.

The most exciting and potentially helpful application of bone scans in tumor management is the use of a nuclear probe intraoperatively<sup>5,7,13</sup>. A standard dose of Tc99m M.D.P. given approximately four hours before surgery allows intraoperative localization of an osteoid osteoma nidus that is not apparent grossly. A modified probe

(approximately 15.0 cm × 2.5 cm in diameter) and portable monitor are brought to the operating room. A technician and nuclear radiologist interpret the counts as the surgeon moves the sterilely covered probe along the exposed bone. This technique was applied to a rib lesion which was "hot" on scan but not apparent radiographically or grossly at surgery. The surgeon removed the normal appearing segment of rib that was sensed as "hot" by the probe. When the specimen was bisected the lesion was found.

In summary, the physician needs to utilize every tool at his disposal for bone tumor diagnosis and localization. The bone scan is only one of several useful studies to aid the treating surgeon. Generally it will not indicate a specific diagnosis but rather be a piece in the diagnostic puzzle. Usefulness of the bone scan can be maximized by obtaining dynamic flow studies as well as static skeletal images, both of which provide an image of the biologic activities of the tumor, namely vascularity and osteogenesis.

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# OPERATIVE TREATMENT OF CHONDROMALACIA OF THE PATELLA CORRELATED WITH EXPERIMENTAL STUDIES OF CARTILAGE REPAIR IN RABBIT PATELLAE

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Hu Yun-yu\*  
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Chondromalacia of the patella, a common painful disorder, may be caused by trauma. Clinical manifestations vary according to the severity of pathologic changes in the patellofemoral joint. Diagnosis can be established from history, physical examination and roentgenographic study. Early symptoms may subside with rest, limitation of motion and physiotherapy. Operative treatment may be indicated for more advanced and persistent cases. Superficial or deep shaving of the eroded and softened areas of the patellar cartilage, patelloplasty and patellectomy have been used.

Numerous studies deal with repair of articular cartilage defects. Most indicate poor endogenous regeneration by adjacent chondrocytes<sup>1,2</sup>. According to Calandruccio<sup>3</sup> and DePalma<sup>4</sup>, the healing process of chondral defects involves exogenous regeneration via fibrocartilaginous metaplasia of fibrous ingrowth from underlying bone marrow.

Questions such as how cartilaginous defects repair, the time required to complete the repair, and the nature of the regenerated cartilage remain unsolved. Moreover, measures to promote cartilage healing need further investigation. This article presents a clinical study of chondromalacia patellae treated by chondral shaving, as well as experimental work on the repair of patellar chondral defects and the effect of L-dopa on repair of these defects in rabbits.

## Clinical Study

### Material

Seventy-four patellae in fifty-seven patients were operated upon by various methods from 1955 to 1980. There were thirty-eight male and nineteen female patients. Ages ranged from thirteen to forty years with thirty-seven patients from twenty to thirty years old. Patients usually presented with discomfort and aching in the knee associated with pain and crepitus on squatting or stair climbing.

Acute pain frequently occurred after vigorous sports. A positive patellar compression test was often noted. Tenderness was more marked at the undersurface of the medial patellar edge than at the lateral edge. There were no significant roentgenographic findings except in cases with intraarticular loose bodies or marginal osteophytes. Concomitant findings in the knees included meniscal injury in twenty-nine cases (51 per cent), osteoarthritis in fifteen cases (26 per cent) and intraarticular loose bodies in four cases (7 per cent).

Pathologic changes in the patellar cartilage commonly included loss of luster, softening and fragmentation. Denuded bone was noted in the four knees with loose bodies. The medial facet was involved in forty-seven knees (55 per cent), the mid-posterior area in twenty (23.5 per cent) and the lateral facet in five (5.9 per cent). A "mirror" lesion of the femoral condyle was present in forty knees (47 per cent).

## Results

Twenty-five of forty-eight knees treated with superficial shaving were followed for an average of 7.8 years. Good to excellent results were seen in nineteen (76 per cent), fair in four (16 per cent) and poor in two (8 per cent). Twelve of fifteen knees treated with deep shaving to subchondral bone were followed for an average of 10.5 years. Good to excellent results were seen in eleven (91.6 per cent) and fair in one (8.4 per cent).

Approximately one-half of the patients with satisfactory results felt much relief shortly after the operation. However, residual discomfort or pain in the knee persisted for months or years, then gradually subsided.

## Experimental Studies of Articular Cartilage Repair in Rabbit Patellae<sup>7</sup>

### Methods

In thirty-two mature rabbits, a 1.5 mm wide transverse defect in the patellar articular surface was made with a scalpel. On one side the defect extended to the subchondral bone. On the other side the defect was one-half the

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thickness of the radiate layer. Rabbits were sacrificed at one, two, four and six months after operation. The patellae were removed, fixed in neutral buffered formalin and decalcified in 5 per cent nitric acid. Histologic sections were stained with hematoxylin and eosin, periodic acid-Schiff reagent, toluidine blue and Alcian blue.

Autoradiographic study was performed by intraarticular injection of 300 uci of radioactive sodium sulfate ( $L^{35}S$ ) twenty-four hours prior to sacrifice. Four specimens were placed in the solution containing 1.0 ml 199# cell culture solution, 50 uci tritiated thymidine and 50 units kanamycin. Autoradiography was completed according to techniques described by Boyd<sup>5</sup> and Rogers<sup>6</sup>.

### Results

Patellae with superficial defects showed necrosis of chondrocytes and depletion of glycosaminoglycans (GAG) beneath the defect from one to four months after injury. At four months the defect was covered by flat fibrocytes from adjacent cartilage (Fig. 1). Healing was incomplete



Fig. 1. Histologic section of a superficial defect four months postoperatively shows poor staining of the superficial layer due to GAG depletion. Toluidine blue.

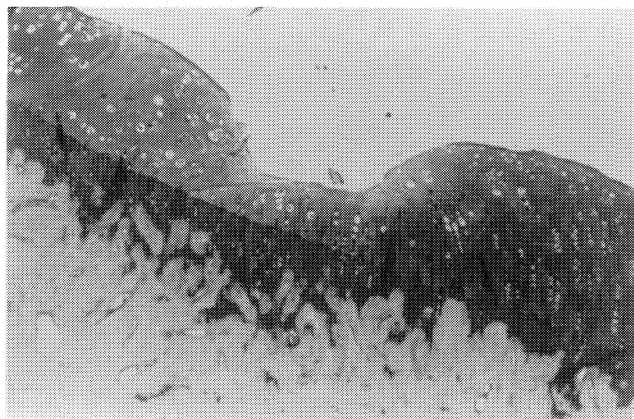


Fig. 2. Histologic section of a superficial defect six months postoperatively shows the defect covered by a layer of flat cells. The area of GAG depletion has disappeared. Toluidine blue.

at the end of six months (Fig. 2). In patellae with full thickness defects, chondrocytes from the radiate layer appeared to lose their normal array and began growing and converging on the defect at the end of one month (Fig. 3). Autoradiography with tritiated thymidine revealed active proliferation of chondrocytes as evidenced by uptake of radioisotope into many cells (Fig. 4). At the end of four to

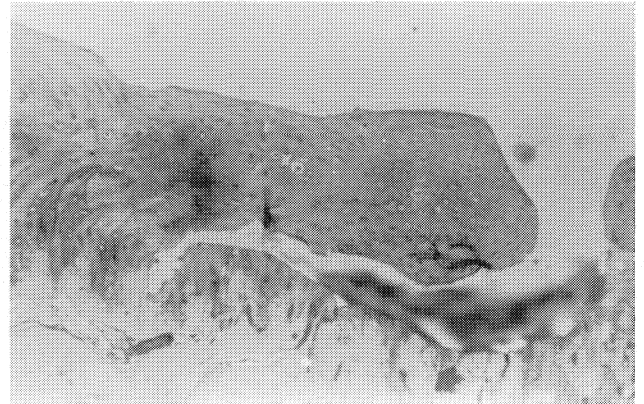


Fig. 3. Histologic section of a full thickness defect two months postoperatively reveals new reparative tissues. Alcian blue.

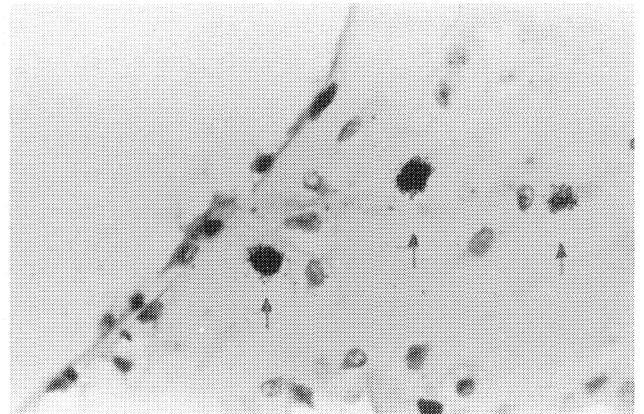


Fig. 4.  $^3H$ -Thymidine autoradiograph shows uptake by proliferating chondrocytes in the regenerating tissue. H and E.

six months the defects were nearly filled except for a tiny fissure between the bone and regenerating tissue (Fig. 5). Glycosaminoglycan matrix of the regenerating tissue appeared similar to hyaline cartilage in its uptake of  $^{35}S$  (Fig. 6).

### The Effect of Oral Administration of L-Dopa on Cartilage Repair in Rabbit Patellae<sup>8</sup>

#### Methods

Full thickness defects in patellar cartilage were created in fifty-seven mature rabbits as described above. The rabbits were divided into four groups: Groups 1 and 2 received 0.1 mg/kg and 0.2 mg/kg of L-dopa respectively for two



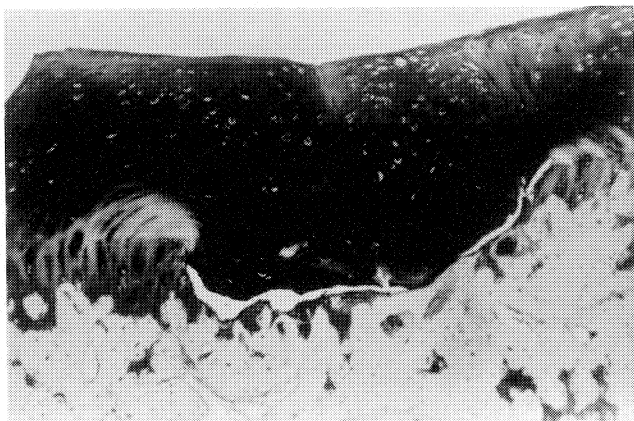


Fig. 5. Histologic section through a full thickness defect six months postoperatively reveals nearly complete healing, however, a narrow fissure remains unfilled at its base. Toluidine blue.

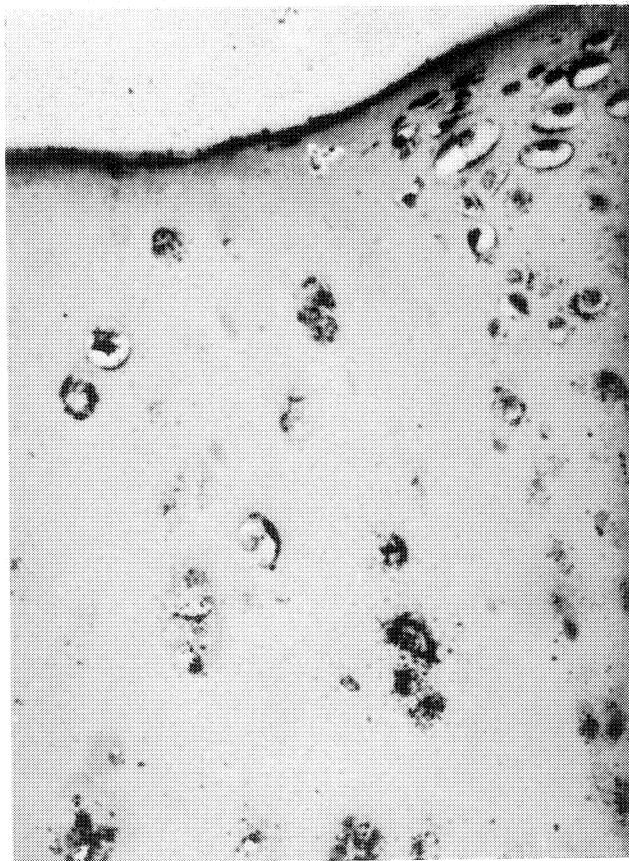


Fig. 6. <sup>35</sup>S autoradiograph reveals uptake of sulphur around each cell in the regenerating tissue. H and E.

weeks, Group 3 received 0.1 mg/kg of L-dopa for four weeks and Group 4 served as a control group. Rabbits were sacrificed and patellae removed two, four, eight, twelve and sixteen weeks after surgery. In specimens obtained at four and eight weeks, mitotic activity of chondrocytes were studied eight hours after intravenous injection of 2 mg colchicine.

## Results

Defects filled gradually with regenerating cartilage from the proximal or both proximal and distal edges of the radiate layer. Regeneration from both edges occurred in 87 per cent, 80 per cent and 83 per cent of Groups 1, 2 and 3 respectively. This occurred in only 23 per cent of the control group. Proliferation and maturation of chondrocytes was noted four weeks earlier in the L-dopa groups than the control group (Fig. 7, 8). By sixteen weeks the defects appeared healed in the L-dopa groups (Fig. 9), while the control group showed incomplete healing even at six months (Fig. 5). Chondrocyte mitoses were present in most specimens from L-dopa groups (Fig. 10) while none were found in the control group. Administration of 0.2 mg/kg L-dopa for two weeks (Group 2) appeared to give the best results in promoting chondrocyte regeneration and maturation.

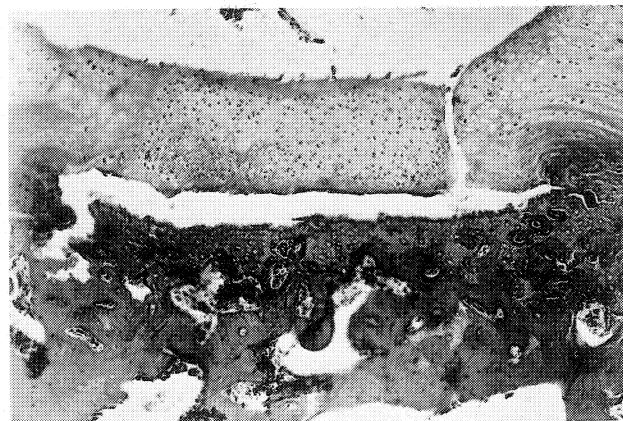


Fig. 7. Control group. Histologic section through a full thickness defect with regenerating cartilage from the proximal (left) edge. Cell density is increased. H and E.

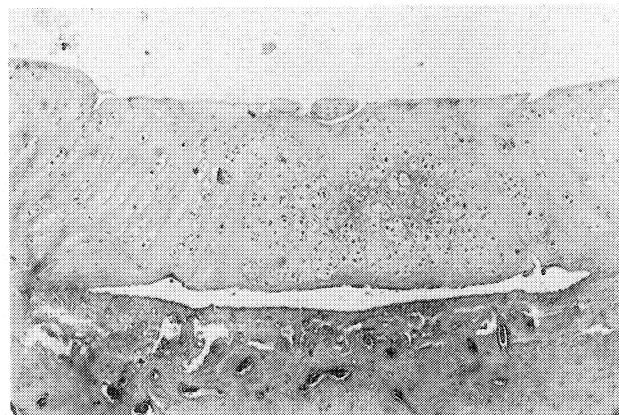
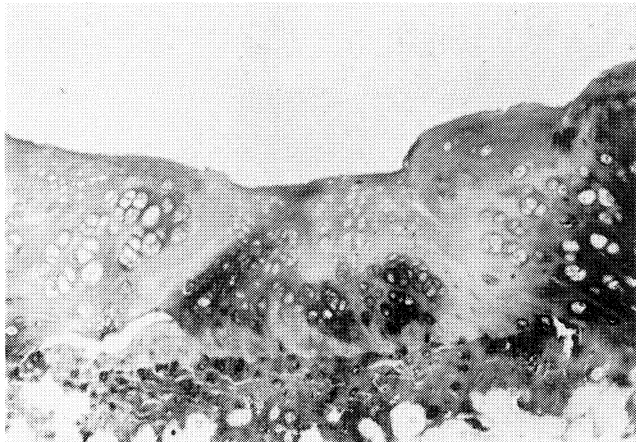
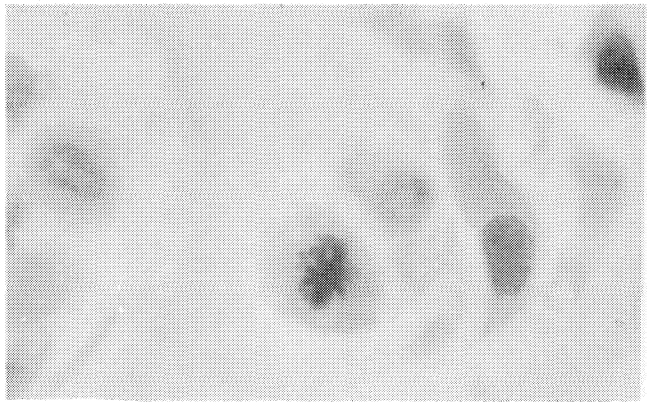


Fig. 8. L-dopa treated group. Histologic section through a full thickness defect shows cartilage bridging the defect from both the proximal (left) and distal (right) margins. The number of chondrons has decreased and cartilage cells show a columnar orientation. H and E.



**Fig. 9.** L-dopa treated group. Sixteen weeks postoperatively the defect has healed completely. Alcian blue.



**Fig. 10.** L-dopa treated group. Four weeks postoperatively histologic sections reveal cells in the regenerating tissue undergoing mitosis. H and E.

### Discussion and Conclusion

An experimental model demonstrated that created defects in rabbit patellar cartilage were repaired by ingrowth of chondrocytes from the radiate layer of the contiguous cartilage. Deep shaving to subchondral bone yielded superior results when compared with superficial shaving. The newly regenerated tissue resembles hyaline cartilage in autoradiographic studies with tritiated thymidine and  $^{35}\text{S}$ . Thus, tissue possessing characteristics of hyaline cartilage developed in the defects through endogenous repair from contiguous chondrocytes.

Clinical results correlate well with our experimental work. Deep shaving of the eroded and softened articular cartilage in patellar chondromalacia appeared to be a simple and effective treatment modality yielding 91 per cent satisfac-

tory results on long term follow-up. Due to the slow repair process, residual symptoms persisted for months or even years in many of our patients but eventually subsided in the majority.

Proper use of L-dopa is generally without serious complication and may result in marked elevation of growth hormone (GH) in blood<sup>10</sup>. GH has been shown to be useful in promoting repair of articular cartilage in animal models<sup>11</sup>. Our experimental work revealed a remarkable promoting effect on regeneration and maturation of chondrocytes associated with chondrocyte mitoses. Clinical use of L-dopa to promote healing of articular cartilage offers exciting prospects for further study. By strict comparison of control and L-dopa groups, we should be able to determine its efficacy in the future.

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# A METHOD OF ARTHRODESIS FOLLOWING FAILED TOTAL ANKLE ARTHROPLASTY

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Obstacles to the conversion of a failed total ankle arthroplasty to an arthrodesis include insufficient bone stock after prostheses and cement removal, difficulty in obtaining adequate fixation, and the gamut of problems which may arise when autogenous bone grafting from a separate site is performed. We overcame these obstacles with an operative technique which has not been reported.

## Case Report:

A moderately obese, thirty-five year old woman complained of left ankle pain with weight bearing. She had undergone open reduction and internal fixation of a left trimalleolar fracture. Severe pain with weight bearing prompted removal of the fixation devices eight months later. A total ankle replacement was performed nine months thereafter. Following the arthroplasty she had only a few weeks of pain relief. After another fourteen months her ankle was explored. Scar and a loose piece of cement were removed and her orthopaedist reported no evidence of infection or loosening of the components. The debridement did not provide significant pain relief. She presented to us twenty-eight months following the debridement complaining of inability to stand or walk longer than ten minutes at a time.

Physical examination of her left ankle revealed no evidence of infection. Roentgenograms showed a radiolucent

line between the tibia and its contained cement (Fig. 1). We decided to fuse the ankle.

At surgery the longitudinal mid-anterior surgical scar was opened. No evidence of infection was seen. The loose tibial and firmly fixed talar prostheses and cement were removed. This left a defect in the anterior tibial cortex. Only a thin shell of cortex and medullary bone remained at the distal end of the tibia. The superior surface of the talus was smooth and flat. We anticipated that the fibula could be inserted into the hole in the tibia.

A longitudinal incision was made over the mid-lateral aspect of the distal one-third of the fibula. After transection of the fibula approximately 8 cm above the distal tip, it was released from the tibia and surrounding structures. Its attachments to the talus and calcaneus were left intact (Fig. 2). Soft tissue, including periosteum, was removed from the distal fibula, except for those most distal ligamentous attachments. A rongeur was used to cut into the fibular cortex in several places and a drill was used to perforate the fibula many times to allow granulation tissue to penetrate the fibular cortex during healing.



Fig. 1. Initial AP and lateral radiographs of the left ankle. Note radiolucency at the tibial bone-cement interface.

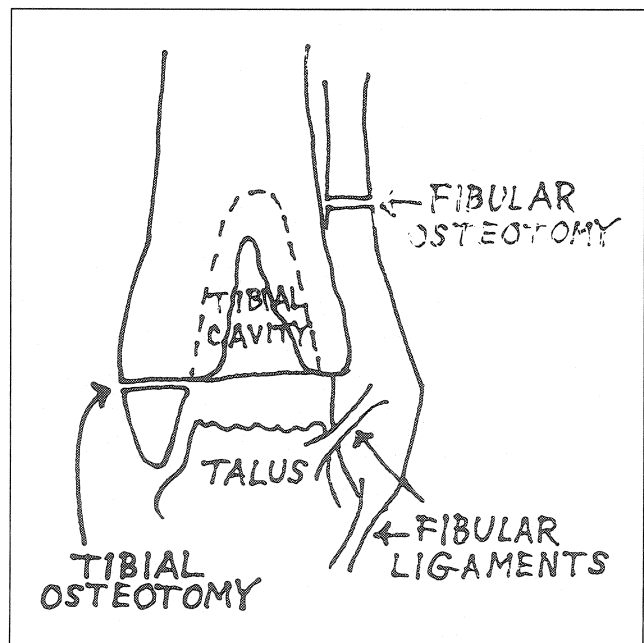


Fig. 2. Diagram illustrating the transected left fibula with intact attachments to the talus and calcaneus. The medial malleolus was osteotomized at the level of the tibial plafond.

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We could not move the talus far enough medially for the fibula to fit into the hole in the tibia without excising the medial malleolus. The malleolus was osteotomized at the level of the tibial plafond (Fig. 2). This allowed the talus to move medially under the distal tibia and the fibula to slide through the anterior slot in the tibia into the tibial cavity. The talus had good contact with the distal end of the tibia, and the fibula fit well in the tibial defect (Fig. 3). The foot was only slightly medially translocated and in about 5 degrees of equinus. The ankle was stable when there was a compressive force across it.

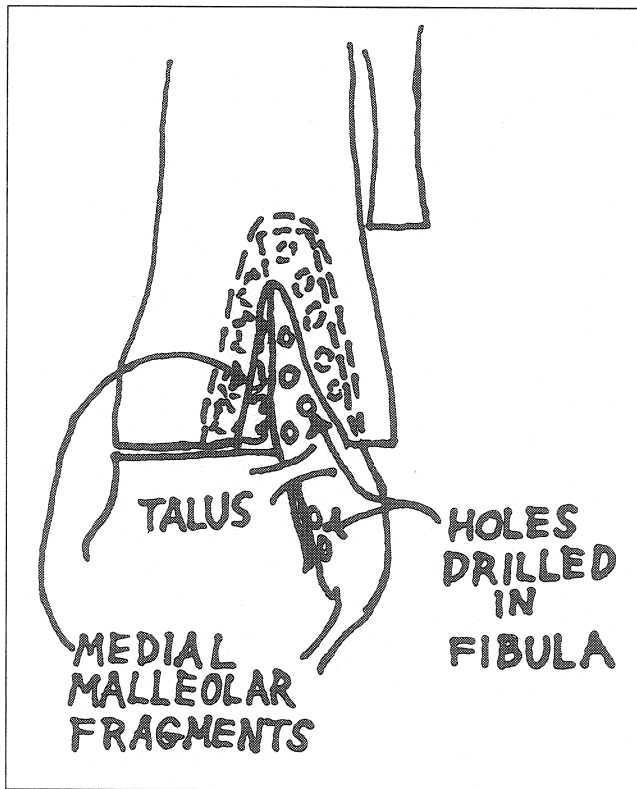


Fig. 3. Diagram illustrating placement of the distal left fibula in the anterior tibial defect. The medial malleolus was cut into small fragments and used as bone graft.



Fig. 4. Radiograph of the left ankle fourteen months postoperative demonstrating a solid arthrodesis.

The medial malleolus was cut into small fragments and used as additional graft (Fig. 3). Good apposition of all major bone fragments and filling of the tibial cavity were obtained. No bone graft from a distant site was necessary.

After copious irrigation, deep tissues and skin were closed with sutures. A compression dressing and a posterior long leg padded plaster splint were applied. The tourniquet was deflated after eighty-seven minutes.

One week postoperatively we applied a patellar tendon bearing short leg cast. She was instructed to weight bear to pain tolerance (for bony compression) and to elevate her left foot when not walking. For about seven months she wore either a laced-on rigid splint or a short leg cast. During that time she fell while unsplinted and required a second period in a cast. She had roentgenographic (Fig. 4) and physical evidence of solid healing of the arthrodesis by fourteen months after our surgery.