THE IOWA ORTHOPAEDIC JOURNAL

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COMPLEX DORSAL DISLOCATION OF THE METACARPOPHALANGEAL JOINT: THE DEEP TRANSVERSE METACARPAL LIGAMENT AS A BARRIER TO REDUCTION

Thomas A. DeCoster, M.D.*
Deborah McGrew, B.S., O.T.R.**
George E. Omer, Jr., M.D.***

Department of Orthopaedics and Rehabilitation
University of New Mexico School of Medicine
Albuquerque, New Mexico

*Assistant Professor, University of New Mexico Albuquerque, New Mexico
**Occupational Therapist, Registered
***Professor and Chairman, University of New Mexico Albuquerque, New Mexico

ABSTRACT

Dorsal dislocation of the second metacarpophalangeal (MCP) joint may involve dorsal displacement of the deep transverse metacarpal ligament (DTMCL). In addition to retraction of the volar plate, reduction of the deep transverse metacarpal ligament to its normal volar position is necessary to achieve stable, complete reduction of the MCP joint. The factors associated with this pathologic condition are reviewed as well as an anatomic study of the mechanism of injury.

INTRODUCTION

Dorsal metacarpophalangeal (MCP) joint dislocation typically occurs from forced hyperextension. Closed reduction is possible for simple dislocations[10] but open reduction is usually required for complex dislocations[1,3,8,11]. Soft tissue interposition and button hole entrapment of the metacarpal head are associated with complex dislocations and require particular attention in open reduction[8]. The nature of the interposition and entrapment has been described in the literature but the importance of the release of transversely oriented fibers of the dorsally translocated deep transverse metacarpal ligament (DTMCL) has not been emphasized. Kaplan has described dorsal dislocation of the index metacarpophalangeal joint: "The fibrocartilaginous plate breaks away in the region of its weakest attachment, at the neck of the volar aspect of the second metacarpal; and the flexor tendons in the volar ligament are violently displaced to the ulnar side of the metacarpal head. Following this, the fibrocartilaginous plate of the joint is displaced over the head of the metacarpal, landing on the dorsum of this bone, where it becomes wedged between the base of the prxoimal phalanx and the head. The lateral collateral ligaments, which are now abnormally displaced, lock the phalanx in the abnormal position typical of this dislocation. At the same time the two groups of transverse fibers of the palmar fascia hold the head of the metacarpal. The distal group (the natatory ligament, which moves with the phalanx) applies pressure to the dorsum of the metacarpal head, while the proximal group (the superficial transverse ligament which extends across the volar aspect of the metacarpal neck) applies pressure to the volar aspect"[8]. Difficulty with open reduction led Kaplan to emphasize three steps: ". . . The first incision is made to free the constriction of the cartilaginous plate. . . . The transverse fibers of the taut natatory ligament must also be completely divided, and following this, another longitudinal incision should be made through the transverse fibers of the superficial transverse metacarpal ligament. This third incision . . . releases the constriction below the metacarpal head"[8].

Numerous authors have identified difficulty with open reduction[4,10,15] or report patients referred to them after unsuccessful open reduction (Baldwin[1] two of three cases, Murphy[11] six of ten cases, Becton[3] four of six cases). Each author references the pioneering work of Kaplan but their observations represent modification of Kaplan's description and recommendations. Murphy[11] implicated the DTMCL, not mentioned by Kaplan[8], identifying that the metacarpal head is forced through the deep transverse metacarpal ligament. Becton recommended a dorsal instead of a volar approach to visualize and release the dorsally translocated volar structures by a longitudinal incision[3]. Baldwin reported it unnecessary to incise the natatory ligament or the superficial transverse metacarpal ligament[1].

We encountered a case which differed slightly from Kaplan's description and seemed to incorporate several of the findings of other authors. The difference required a slightly different surgical release in order to obtain a com-

plete, stable reduction. In this case the DTMCL had displaced dorsally. Despite removing all intra-articular obstructions (including the volar plate) and reducing the articular surfaces, the dorsally displaced DTMCL forced the metacarpal head into the palm. Stable reduction was achieved by reducing the ligament. The case differed from Kaplan's description in that the volar plate pulled the deep transverse metacarpal ligament dorsal to the metacarpal head. This ligament then forced the metacarpal head volarly even after release of the volar plate itself and the more distal natatory ligament.

A case report will be presented as well as the results of a series of anatomic dissections. This information will substantiate the importance of the displaced DTMCL in addition to the volar plate in some complex MCP dislocations.

CASE REPORT

M. M. is a twenty-four year old male who sustained a hyperextension injury to his dominant right index metacarpophalangeal joint. The finger was held extended with puckering of the skin volarly. The neurovascular examination was normal. A lateral roentgenogram (Fig. 1) shows a dorsal dislocation of the second MCP joint. The sesamoid present in this case conveniently marks the dorsal displacement of the volar plate. Attempt at closed reduction was predictably not successful.

Figure 1.
Lateral roentgenogram of a dorsal dislocation of the index MCP joint.

Under general anesthesia in the operating room a volar and dorsal approach to the index MCP was performed. The volar plate was seen to be interposed between the articular surfaces of the proximal phalanx and metacarpal head, still attached to the base of the proximal phalanx. The volar plate was surgically split longitudinally and the proximal free end brought out of the joint with volar retraction. A longitudinal incision was then made through the distal (natatory ligament) and proximal (superficial trans-

verse metacarpal ligament) transverse fibers volarly as described by Kaplan[8], even tough they did not seem to be preventing reduction. The flexor tendons and lumbrical were lax and retracted ulnarly and radially respectively. The articular surfaces were visualized completely and MCP reduction was performed under direct vision by distracting and flexing the finger. Reduction was achieved. However, upon release of the hand, the metacarpal head spontaneously dislocated volarly. Repeat reduction was performed but a stable reduction could not be maintained.

The deep transverse metacarpal ligament (DTMCL) was seen to be dorsally displaced over the neck of the second metacarpal forcing the metacarpal head palmar. Instead of running from the radial side of the second metacarpal to the third metacarpal volar to the second metacarpal, it passed dorsally. The DTMCL could not be readily swung around the metacarpal head through the joint so it was divided dorsally in line with the metacarpal shaft. The cut ends were returned to the volar surface of the metacarpal neck. Stable reduction was easily achieved. The cut ends of the DTMCL were sutured volarly. A full stable range of motion was achieved. The incision was closed and the hand splinted with the metacarpal phalangeal joint reduced.

An extension block splint was worn for six weeks with active flexion encouraged after the first week. The patient had a stable painless joint at six months. His range of motion was from eighty degrees of flexion to ten degrees of extension. At eighteen months follow-up, he is working as a carpenter and the joint appears normal roentgenographically.

CADAVERIC INVESTIGATION OF THE MECHANISM OF DORSAL DISLOCATION OF THE DEEP TRANSVERSE METACARPAL LIGAMENT (DTML) IN DORSAL DISLOCATION OF THE MCP JOINT

A model for dorsal MCP dislocation was obtained through dissection of eight cadaveric hands previously fixed in formalin. The joint and supporting structures were exposed and dorsal dislocation obtained by forceful hyperextension. Characteristic anatomic abnormalities were identified and reduction manuevers determined. The DTMCL was identified with its attachment to the radial side of the second metacarpal, metacarpal shaft, with the volar plate being preserved. The flexor tendons were freed from their tendon sheaths and retracted to reveal the volar plate of the second MCP. The proximal and distal attachments of the volar plate were preserved. The superficial transverse metacarpal ligament and natatory ligaments were noted. The skin and superficial fascia and subcutaneous tissue were removed but the remainder of the retinacular system of the hand was left intact[6,7,12,13,14,16]. The fibrocartilaginous volar plate inserted into or was confluent with the

transverse fibers of the deep transverse metacarpal ligament as illustrated by Eaton in Green's textbook[5]. In three of eight cases there was clear demarcation of this interface but in five of eight the junction was a smooth transition.

Dorsal dislocation was achieved by extreme hyperextension of the second MCP joint. In one case, where the interface was clearly demarcated, the volar plate pulled away from the DTMCL. The volar plate became interposed in the joint without any remaining attachment to the metacarpal, but the DTMCL remained in its normal volar position. In seven of eight cases, as the MCP hyperextended the volar plate pulled the DTMCL and the proximal edge trailed over the metacarpal head. The volar plate and DTMCL became interposed in the MCP joint. With further extension the DTMCL, with its radial insertion intact, swung around the metacarpal head to lie dorsal to the metacarpal neck (Fig. 2). The true volar plate was interposed between the articular surfaces of the proximal phalanx and the metacarpal head (still attached to the proximal phalanx). The flexor tendons tore away from the tendon sheaths, displacing ulnarly and were carried dorsally with the proximal phalanx as their attachment to the finger remained intact. The lumbrical deviated radially and was carried dorsally, as again the attachments to the dorsally displaced fingers remained intact.
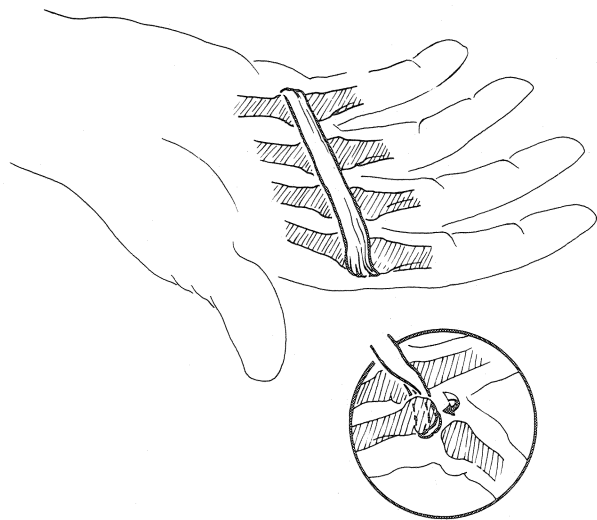

Figure 2.
Diagram of the deep transverse metacarpal ligament.
Inset: Dorsal translocation of the DTMCL.

Extension to one-hundred-twenty degrees invariably led to volar plate interposition and dorsal translation of the proximal phalanx with the tendons. No amount of manipulation, even with the joint structures under direct vision, could accomplish a reduction. Reduction maneuvers seemed

to cause the true volar plate to separate from the DTMCL by a shearing action of the metacarpal head.

If the proximal portion of the volar plate was separated by manipulation or surgically freed from its connection to the DTMCL and retracted volarly the metacarpophalangeal joint could be reduced and the volar tendons relationship would be restored. However, the reduction was unstable as the metacarpal head spontaneously displaced volarly. The condition exactly reproduced the clinical and operative findings of the case report. Similarly, reduction by division of the DTMCL dorsally and re-approximating it volarly in conjunction with the repeat reduction of the joint resulted in a stable reduction. In three of seven cases the "bucket handle" displacment could be reduced by placing a Freer elevator between the metacarpal neck and DTMCL and prying the ligament over the metacarpal head while distracting the distal phalanx. Stable reduction was achieved. In the other four cases, fractures of the metacarpal head resulted from this maneuver. The natatory ligament and superficial transverse metacarpal ligament did not have to be released to obtain reduction. If the volar plate and DTMCL were reduced it was not necessary to retract the flexor tendons or lumbrical to achieve reduction.

DISCUSSION

Dorsal dislocation of the MCP joint may require open reduction. Kaplan and others have described the common barriers to closed reduction and the steps necessary to correct them during open reduction[2,5,8]. High rates of unsuccessful open reduction continue to be suggested by the literature[1,2,3,11]. Current textbooks continue to cite Kaplan's description[4,5,15] without mention or emphasis of the deep transverse metacarpal ligament (DTMCL). We believe that it is important to emphasize that release or reduction of the dorsally displaced fibers of the DTMCL as suggested by Murphy is an important step in open reduction in some and perhaps most complex dislocations of the second MCP joint.

We are describing a barrier to reduction not specifically mentioned by Kaplan which occurs in some cases of complex second metacarpal dorsal dislocations. There is a band of transversely oriented fibers which run from the ulnar side of the fifth metacarpal across the palm to the third metacarpal and then to the radial side of the second metacarpal which have been displaced from their normal volar position relative to the neck of the second metacarpal to pass from the volar-radial side of the third metacarpal, dorsal to the second metacarpal neck to insert upon the radial side of the second metacarpal. In addition to entrapment of the volar plate the DTMCL may become displaced like a bucket handle swinging from volar to dorsal while maintaining its attachments to the third metacarpal and radial side of the second metacarpal. It comes to lie dorsal

to the metacarpal and prevents complete stable reduction. This pathologic situation was reproduced in anatomic dissections. Stable reduction required reduction of the DTMCL. Kaplan, Eaton, and Weeks all discuss release of the volar plate. This is necessary but we feel that emphasis should also be placed on release of the transversely oriented fibers which may lie on the dorsal aspect of the second metacarpal neck (DTMCL).

The radial insertion of the DTMCL, although well described in anatomic texts,[8,12,14,16] is not commonly appreciated among orthopaedists. The infrequency of the injury and distorted post-traumatic anatomy may make appreciation of the pathologic anatomy difficult for the hand surgeon attempting open reduction. An awareness of the possibility of a displaced DTMCL may facilitate the operative achievement of a complete stable reduction. Release of this dorsally displaced transverse ligament is facilitated by a dorsal approach and we recommend it for complex dislocations of the second MCP. A supplementary volar incision may be necessary.

The natatory ligament is quite distal to the metacarpal head and the deep transverse metacarpal ligament. The superficial transverse metacarpal ligament is a flimsy, almost subcutaneous structure of very limited importance in preventing reduction. After reduction of the volar plate and DTMCL it is not always necessary to retract the flexor tendons or lumbrical to achieve reduction.

SUMMARY

Difficulty with open reduction of complex index MCP dislocations continue to be reported. Dorsal displacement of the deep transverse metacarpal ligament (DTMCL) may be a component of complex dislocation of the second MCP joint which requires correction before stable complete reduction can be achieved. Retraction of the volar plate out of the joint is also necessary.

The anatomic features of the DTMCL are reviewed. The mechanism of dorsal transposition of this ligament during hyperextension is described. Knowledge of this pathologic anatomy is necessary to obtain complete correction of complex MCP dislocations when the DTMCL is displaced dorsally.

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RATIONALE FOR AUTOLOGOUS BLOOD USE IN ORTHOPAEDIC SURGERY

John J. Callaghan, M.D., MAJ
Orthopaedic Surgical Service
Walter Reed Army Medical Center
Washington, D.C. 20307-5001

Jeffrey D. Thomson, M.D., CPT
Orthopaedic Surgical Service
Walter Reed Army Medical Center
Washington, D.C. 20307-5001

Carlton G. Savory, M.D., COL
Orthopaedic Surgical Service
Walter Reed Army Medical Center
Washington, D.C. 20307-5001

Michael H. Leakan, M.D.
Pathology Department
Director, Blood Bank
Walter Reed Army Medical Center
Washington, D.C. 20307-5001

INTRODUCTION

The concern of contracting AIDS has caused the physician and the public to question the use of homologous blood transfusion in elective orthopaedic surgery. This heightened awareness is not unfounded because homologous blood transfusions are not totally benign. The probability of having a problem with a blood transfusion has been estimated at close to one in eight[22]. More importantly, based on a 1980 study, there are an estimated 5,000 deaths per year caused by non-A or non-B hepatitis from homologous blood transfusions[1].

An increased demand on the available blood supply is created by surgical advances and improved medical care. To complicate the increased demand, the blood supply is limited by decreased numbers of blood donors secondary to more stringent predonation screening programs and the unjustified fear of contacting AIDS by donating blood.

Orthopaedic surgeons should be aware of the complications of homologous blood transfusions and the methods to avoid or reduce the number of homologous transfusions. An autologous blood transfusion program is one method of accomplishing the goal of diminishing homologous blood usage. The purpose of this paper is to present some of the complications associated with homologous blood transfusions, the methods to avoid homologous blood transfusion, and our experience with a predeposit, autologous blood program.

COMPLICATIONS (See Table I)

Despite predonation screening and improved blood bank techniques, the use of homologous blood still carries significant risks. Acquired immunodeficiency (AIDS) has received most of the public's attention, however less than 1 percent of the patients who have AIDS developed the condition following transfusion[14]. This number will probably decrease as blood testing for AIDS increases.

Febrile nonhemolytic reactions are among the most common type of transfusion reaction. These reactions, which are caused by transfused leukocytes, are usually not serious and respond to antipyretic medications. However, when a patient develops a fever during a blood transfusion a hemolytic reaction must first be ruled out.

Table I

Some Common Complications of Homologous Blood Transfusions

1) Febrile Non-hemolytic Reactions

2) Allergic Reactions

3) Non-A, Non-B Hepatitis

4) Alloimmunization

5) Delayed Hemolytic Reactions

Allergic reactions are probably the second most common transfusion reaction at our institution. These reactions are typically manifested as localized urticaria although they can also present as systemic anaphylaxis. Allergic reactions are thought to be caused by plasma proteins, are usually mild, and respond to antihistamine administration[57].

Post-transfusion hepatitis, which occurs in approximately 10 percent of persons who receive blood transfusions, is the most frequent serious complication of transfusions. Hepatitis B, originally known as serum hepatitis, is associated with less than 5 percent of cases while non-A, non-B, hepatitis represents about 95 percent of post transfusion hepatitis today.

Alloimmunization to transfused red blood cell antigens initially may not produce any clinical symptoms but can make future cross-matching very difficult[33]. This could potentially be very significant in young patients, i.e., females undergoing scoliosis surgery.

Delayed hemolytic reactions are not uncommon complications of homologous blood transfusions. These reactions usually occur in patients sensitized to blood group antigens from a prior transfusion. These reactions manifest three to fourteen days after a transfusion and are usually undetected. If severe, however, the hemoglobin will decrease and hyperbilirubinemia will occur. The greatest potential danger of a delayed hemolytic reaction is exposing the patient to the possibility of an acute hemolytic transfusion reaction, where in the face of a decreasing hematocrit caused by an unrecognized immunologically mediated delayed hemolytic reaction, the patient is again transfused[57].

AUTOLOGOUS TRANSFUSIONS

The complications associated with blood transfusion and the publicity regarding AIDS have resulted in a renewed interest in autologous blood transfusion. The concept of autologous blood transfusion is certainly not new. The first reported use of autotransfusion in an orthopaedic/trauma setting was in 1886 by Duncan[17]. He amputated the leg of a patient injured in a railroad accident. The shed blood was collected in a basin with phosphate of soda as an anticoagulant and returned via the exposed femoral vein. Duncan used this same technique two months later when he assisted A.G. Miller in a hip amputation[47].

Autologous transfusion can be performed in three ways: 1) preoperative blood collection, storage and retransfusion during or after surgery, 2) artificial hemodilution with immediate preoperative phlebotomy and retransfusion postoperatively if required and 3) intraoperative blood salvage.

Preoperative blood collection and storage has been reported by several authors to be beneficial in orthopaedic surgical procedures[4,9,13,15,16,20,39,42,51,52,56,61]. Most have used liquid blood storage and oral iron supplementation. Generally,

while the patients tolerate the predeposit program well and the surgery can be performed without blood complications using liquid blood storage, there are definite drawbacks. The most significant disadvantage of liquid storage is that the shelf life limits the amount of blood that can be taken preoperatively.

Two groups have reported on their experience with a predeposit system using liquid and frozen autologous blood with oral iron supplementation[20,39]. Neither reported any complications. Furthermore, one study noted that liquid autologous blood transfusion are less expensive than homologous blood transfusions. This study compared four units of autologous blood (two frozen, two liquid) costing $368 compared to four units of homologous at $296. However, the expense of investigating homologous transfusions, as well as the additional hospitalization and medical care for post transfusion hepatitis, would raise the cost of homologous blood well above that of autologous blood[55]. All the reports note that patient acceptance and enthusiasm is high, and they agree with Mann that any patient well enough to undergo an elective procedure should be considered medically fit to donate blood preoperatively[41].

The advantages of the predeposit method are that it stimulates erythropoiesis, no extra personnel or equipment are needed in the operating room, and the blood is administered in the same fashion as homologous blood. Also, the program is flexible and can adjust for changes in the patient donation schedule or surgery schedule. Finally, patients may obtain psychological benefit by participating in their own care and may be more likely to become volunteer blood donors in the future. The disadvantage of a predeposit program is that it can pose logistical and administrative difficulties, however the AIDS issue has inspired patients and physicians to overlook this problem.

Acute normovolemic hemodilution as described by Messmer[46] in 1975 is based on the principle that hemodilution causes decreased blood viscosity which results in an improved blood flow and perfusion time, decreased stasis in the capacitance vessels and therefore a diminished risk of venous thrombosis and more importantly, a reduction in the loss of red cell mass during the operation[32]. This is achieved by phlebotomy in the immediate preoperative period with a "cc for cc" replacement of the blood volume withdrawn with either a colloid or crystalloid solution.

Laks[36], Ahlberg[2], and Dutoit[18] have reported on acute normovolemic hemodilution in orthopaedic patients. They noted that this method requires more intensive monitoring and a slight increase in the anesthesia and operating room time. They reported a 50 percent reduction in the amount of homologous blood transfused.

Intraoperative blood salvage consists of transfusing a patient's own blood retrieved during surgery[61]. Although the concept and practice are not new, it did not appear in the orthopaedic literature until 1974 when C.M. Evarts

reported on a preliminary trail of intraoperative autotransfusion in twenty-two spinal fusion cases for scoliosis[21]. The only abnormalities noted were early elevations in the plasma hemoglobin which returned to normal after twenty-four hours and depressed platelet counts during the first forty-eight hours. No serious clotting abnormalities or infections occurred and approximately one half of the estimated blood loss was retrieved. There have been several other favorable reports of intraoperative blood salvage in orthopaedic surgery. Most document a 50 percent decrease in the amount of homologous blood transfused and few, if any, complications. However, one study using intraoperative blood salvage combined with hypotensive anesthesia reported only 20 percent recovery of the blood lost hence the blood collected cost $283 per unit[35]. The economic "break even" point is reported to be between three and four units of blood[23,24,34,35,53].

The chief disadvantages of this intraoperative blood salvage system are the extra equipment needed in the operating room and the need of a skilled technician to monitor the system. Cell washing is essential to remove bone and other debris prior to reinfusion of red cells. Also, at best, the system recovers only 50 percent of the shed blood. Cases with a large blood loss would therefore require homologous blood to supplement intraoperative salvage. Finally, if more than one patient using intraoperative autotransfusion is operated at the same time, additional systems are needed. The newer model machines are becoming more semiautomated and, unlike the older models, have not been associated with adverse effects such as hemolysis, hemoglobinuria, coagulation disorders or microemboli except in massive transfusions.

Combinations of these methods as well as combining these methods with hypotensive anesthesia have been reported with good success[7,15,28,31,38,40,54,58]. Finally, some groups have reported total hip arthroplasty without transfusion[43,49,50].
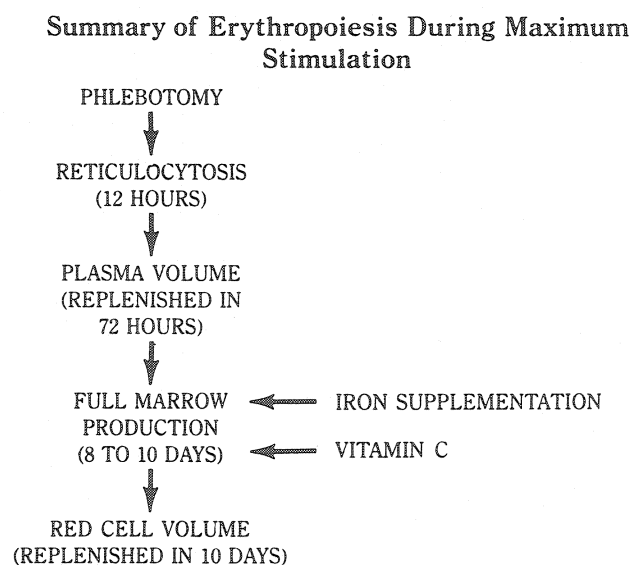
Directed donation in which patients recruit their own blood donors has become more common in recent years. Many hospitals and blood centers have instituted directed donation programs and a few states have enacted laws authorizing directed donations. However, there is no scientific evidence that demonstrates that directed donations are safer than blood from volunteer donors. Furthermore, there are several legal and ethical considerations involved with directed donations. Also, there is the potential that the blood is less safe because the pressure on family or friends to donate may result in the donor failing to admit being involved in a high-risk group such as IV drug abusers or male homosexuals. For these reasons the AABB (American Association of Blood Banks) discourages the use of directed donations[6].

PHYSIOLOGIC CONSIDERATION IN PREOPERATIVE AUTOLOGOUS BLOOD DONATIONS

In addition to eliminating the disadvantages of homologous blood, preoperative autologous blood donation has the advantage of stimulating erythropoiesis. Several investigators have demonstrated the ability of the hematopoietic system to respond to multiple phlebotomies with no significant adverse effect on hemoglobin levels[12,19,27], PT, PTT, clotting time, EKG[44,45], or serum proteins[52]. The plasma volume is rapidly replenished within seventy-two hours while the red blood cell volume is replenished more slowly and is responsive to available iron stores (Figure 1). Reticulocytes can be delivered to the circulation within twelve hours after phlebotomy, but the full level of marrow production is not reached until eight to ten days after the initial phlebotomy[8]. Numerous studies have compared patient response to multiple phlebotomies with and without supplemental iron (either oral or parental)[12,25,29,30,43]. Erythropoiesis, which is a function of bone marrow iron stores and stimulation by hypoxic stress, can increase up to three times normal if iron stores are adequate[26]. In patients undergoing regular phlebotomies and given iron supplementation, the hematocrit initially falls but then stabilizes at an adequate level[25,29,30]. Serum ferritin is a reliable indicator of marrow iron stores and permits potential autologous transfusion donors to establish phlebotomy schedules. This may be especially useful for children and menstruating females who often have low or border line marrow iron stores[26].

Another benefit of preoperative phlebotomy is that the patient will arrive at surgery in a state of maximal hema-

Figure 1.

Summary of Erythropoiesis During Maximum Stimulation

PHLEBOTOMY
↓
RETICULOCYTOSIS
(12 HOURS)
↓
PLASMA VOLUME
(REPLENISHED IN
72 HOURS)
↓
FULL MARROW ⟵ IRON SUPPLEMENTATION
PRODUCTION
(8 TO 10 DAYS) ⟵ VITAMIN C
↓
RED CELL VOLUME
(REPLENISHED IN 10 DAYS)

topoiesis. A patient who does not undergo preoperative phlebotomy will require several days before red cell production reaches a maximal level, since marrow stimulation does not begin until the surgical blood loss occurs[8]. In addition, the transfused autologous red cells which are theoretically younger will survive longer after reinfusion than transfused homologous red cells. Patients may have blood drawn at a local facility and then have the blood shipped to the "surgical" hospital where it is kept liquid if surgery is scheduled within five weeks. If the surgery is not planned within the life span of the liquid blood it is frozen. Liquid units are stored at between 1 and 6 degrees centigrade with CPD-A1 as the anticoagulant and become outdated thirty-five days after donation. A newer preservative allows liquid units to be stored for forty-two days. Frozen units are stored at not more than minus 65 degrees centigrade with glycerol added as a cryoprotective agent. Prior to reinfusion, the frozen red blood cells are thawed and the glycerol removed by washing. This process takes about thirty minutes and results in a 10 percent loss of red blood cells. Because of the risk of bacterial contamination, frozen units must be transfused within twenty-four hours after thawing. Blood from patients with erythrocyte defect such as hereditary spherocytosis or glucose-6 phosphate dehydrogenase deficiency cannot always be frozen because of red blood cell fragility. In patients with these problems a pint may be frozen then thawed on a trial basis to see if the cell will maintain its integrity. Patients with sickle cell trait require special thawing and deglycerization of their frozen cells[11].

PREOPERATIVE BLOOD PROCUREMENT

There are no rigid criteria for autologous donor selection because of the special circumstances with autologous transfusions. The suggested guidelines of the AABB include no age limits, the hemoglobin concentration of the donor patient should be no less than 11 grams/deciliter or the hematocrit should be no less than 34 percent[58].

A more structured guideline used by a community blood center required the patients to be between twelve and seventy years old, minimum hematocrit of 34 percent, no history of cardiovascular disease or recent acute asthmatic attacks, and no history of convulsions after infancy[10].

At our institution, we do not have any absolute age requirements. Instead we require the patient to be able to cooperate with repeated phlebotomies. We do not allow patients with hepatitis, malignant neoplasm, anemia, or severe cardiopulmonary disease to enroll in the program. For those patients whose history suggests that complications may occur from the donation of blood, the blood bank director may require further tests prior to enrollment.

Oral iron supplementation is recommended for autologous blood donors. Theoretically ferrous fumarate with vitamin C is the supplement of choice because the fumar-

ate form is less irritating, less constipating, and has more elemental iron while the vitamin C keeps the iron in the ferrous form which is better absorbed. Parenteral iron is not used because of its association with anaphylaxis and sudden death[65].

In general, we believe that any patient healthy enough for an elective orthopaedic procedure should be healthy enough to donate at least one or two units of autologous blood.

The following program is suggested as a general guideline for preoperative blood procurement. (Modified from Winter)[63]

1) Obtain preoperative medical evaluation concentrating on the patient's nutritional and hematological status. The medical director of the blood bank should be involved.

2) Determine the amount of blood required for surgery.

3) If less than four units are required, draw units individually at least four days apart and no sooner than seventy-two hours in advance of surgery. The patient's hemoglobin level (prior to each donation) should be at least 11 grams. These units can be kept in liquid state for thirty-five days.

4) If four or more units are required, follow the program for less than four units, drawing additional units every two to three weeks and store as frozen cells.

5) Up to five units can be collected in thirty-six days using the "leap frog" techinque. This overcomes the time limitations of liquid storage and avoids the need for costly frozen storage.

6) Whatever system is used, the patient should take one 325 mg iron sulfate tablet and one 250 mg ascorbic acid tablet three times daily with meals. A daily multivitamin should also be taken. Starting supplements before phlebotomy should be considered.

PATIENT PHYSIOLOGIC RESPONSE

The phlebotomy schedule for autologous blood donation is individualized depending on the patient's hematocrit, the length of time before surgery, and the number of units desired. For a total hip replacement we generally obtain four units of autologous blood, drawing the units every two weeks beginning about two months prior to surgery. Two units are frozen and the last two units are kept liquid. All patients receive oral iron supplementation and a daily multivitamin. Most patients tolerate this schedule very well.

We generally see the hematocrit at its nadir before the third donation, although we have not scientifically documented this. It seems that the loss of two units of blood is the necessary minimum to stimulate erythropoiesis. Most patients have their lowest hematocrit between the second and third donation. This sometimes causes the third donation to be delayed.

Because erythropoiesis is affected by bone marrow iron stores it may be useful to obtain serum ferritin levels prior to blood donation as an indicator of marrow iron stores. This may be helpful in predicting and monitoring a patient's response to repeated phlebotomies.

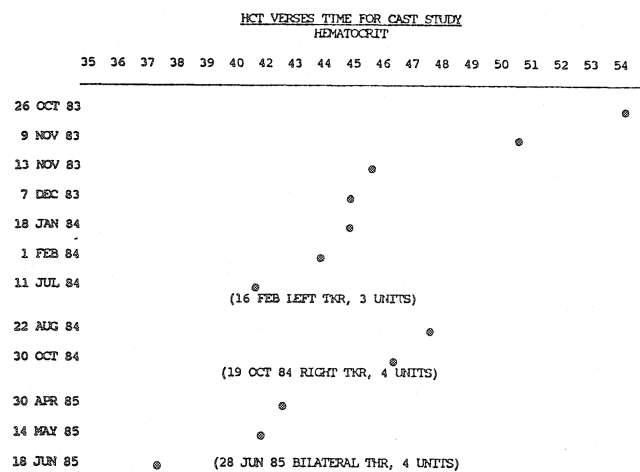## RESULTS OF WALTER REED ARMY MEDICAL CENTER STUDY

We have recently analyzed the results of our orthopaedic predeposit autologous blood program and found that 71 percent of patients enrolled had all of their blood requirements met by the autologous program[60]. We studied three groups that were composed of patients receiving autologous blood only, homologous blood only, and both autologous and homologous (mixed).

Of the 211 patients in the study, 159 actually completed the autologous blood program and had autologous blood available during surgery. Seventy-one percent of these patients received only autologous blood during their hospitalization. In the group requiring homologous blood to supplement their autologous blood, 64 percent (182 of 285 units) of the blood requirements were met by autologous blood in this mixed group. Fifty-two patients did not participate in the autologous program (forty were either not aware of the program or not interested) and twelve were rejected for medical reasons. Because of the interest in reducing homologous blood transfusions there have been several other recently published articles documenting the safety, efficacy and practicality of predeposit autologous blood programs[3,64].

CASE STUDY

A seventy-two year old male with four joint osteonecrosis underwent staged bilateral knee and one stage bilateral total hip replacements over a sixteen month period. He received only autologous blood (see Fig. 2). The points

Figure 2.

HCT VERSES TIME FOR CAST STUDY
HEMATOCRIT

| | 35 | 36 | 37 | 38 | 39 | 40 | 42 | 43 | 44 | 45 | 46 | 47 | 48 | 49 | 50 | 51 | 52 | 53 | 54 |

26 OCT 83

9 NOV 83

13 NOV 83

7 DEC 83

18 JAN 84

1 FEB 84

11 JUL 84

(16 FEB LEFT TKR, 3 UNITS)

22 AUG 84

30 OCT 84

(19 OCT 84 RIGHT TKR, 4 UNITS)

30 APR 85

14 MAY 85

18 JUN 85   (28 JUN 85 BILATERAL THR, 4 UNITS)

DISCHARGE HEMATOCRIT: 30.6, PATIENT ASYMPTOMATIC - DISCHARGE DATE 12 JULY 85

on the graph are the patients' predonation hematocrit at each donation. The patient donated one unit of whole blood at each donation.

The first six blood donations represented our typical patient response to repeated phlebotomy. That is, the hematocrit initially falls during the first two or three phlebotomies and then levels off or improves during the subsequent phlebotomies. The patient's response to the phlebotomies in 1985 is concerning but retrospectively the patient reports being much less compliant with oral iron during that time. Serum ferritin levels would have been useful but were not obtained. The patient had no illness or complication from the repeated phlebotomies or the decreased hematocrit.

CONCLUSION

A preoperative autologous blood program has several advantages over homologous blood transfusions. Primarily, it eliminates the complications associated with homologous transfusions (i.e. transfusion reactions, hepatitis, AIDS). It preserves the community blood supply and allows patients to become more involved with their care. Finally it stimulates erythropoiesis so that patients will regenerate blood more quickly in the postoperative period.

A preoperative autologous blood program is flexible, safe, and well tolerated by patients and staff. Furthermore, the system can be combined with an intraoperative salvage system to maximize the advantages of each system. It must be stressed, however, that any system chosen must be part of an overall effort to minimize blood loss and the use of homologous blood transfusion. Therefore, we believe that a patient is his (her) own best source of blood and that a predeposit autologous blood program is an excellent system, especially in elective orthopaedic surgery.

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LIMB LENGTH DISCREPANCY RECOGNITION AND PREDICTION

Stuart L. Weinstein, M.D.
Professor
Department of Orthopaedic Surgery
University of Iowa
Iowa City, Iowa

The approach to a patient with a limb length discrepancy involves a knowledge base in two main areas[1,6]. First, the treating physician must decide on the basis of all the information available, if the discrepancy warrants surgical treatment. In general, discrepancies of less than 2 cm do not require surgical equalization. In those greater than 2 cm surgical equalization should be considered[13]. Second, the physician must have a complete working knowledge of all of the equalization procedures, including their technical aspects and complications, to be able to make a decision as to what method or combination of methods would most benefit the specific patient. We will concern ourselves with the first topic in this paper.

In evaluating a patient with a limb length discrepancy, the first concern should be an understanding of the etiology. The etiology of the discrepancy is a significant factor in deciding upon the appropriate equalization procedure. Shapiro has recently demonstrated the importance of etiology in 803 patients with limb length discrepancies followed to maturity[17]. He showed that the pattern of developmental discrepancies is dependent upon the etiology of the condition and the place and time of their occurrence[16]. He also demonstrated that each etiology may have several patterns of developmental discrepancy.

In the physical exam of the patient one must determine if the discrepancy is real or only apparent. Apparent limb length discrepancies may be caused by any condition leading to pelvic obliquity. Adduction contracture of the apparent short limb or abduction contracture of the apparent long limb or scoliosis may give these apparent discrepancies. This is most commonly seen in patients with neuromuscular disorders. These apparent discrepancies are managed by treating the underlying cause of the pelvic obliquity[6].

To evaluate the degree of discrepancy three modalities are used, tape measure, elevation under the foot, and orthoroentgenograms or scanograms. Limb lengths should be measured from the anterior superior iliac spine to the medial malleolus. Measurements from the umbilicus to the heel should also be made so as to include the contributions of the pelvis and the foot to the discrepancy, in addition this helps to delineate if the discrepancy is apparent or real.

The second method of determining the degree of discrepancy is by the use of different size elevations under the short limb in an attempt to level the pelvis (Fig. 1). This method compensates for differences in size and shape of the foot or pelvis. The patient can be photographed with the correct elevation under the shortened extremity and the amount of elevation needed to level the pelvis recorded in the patient's record.
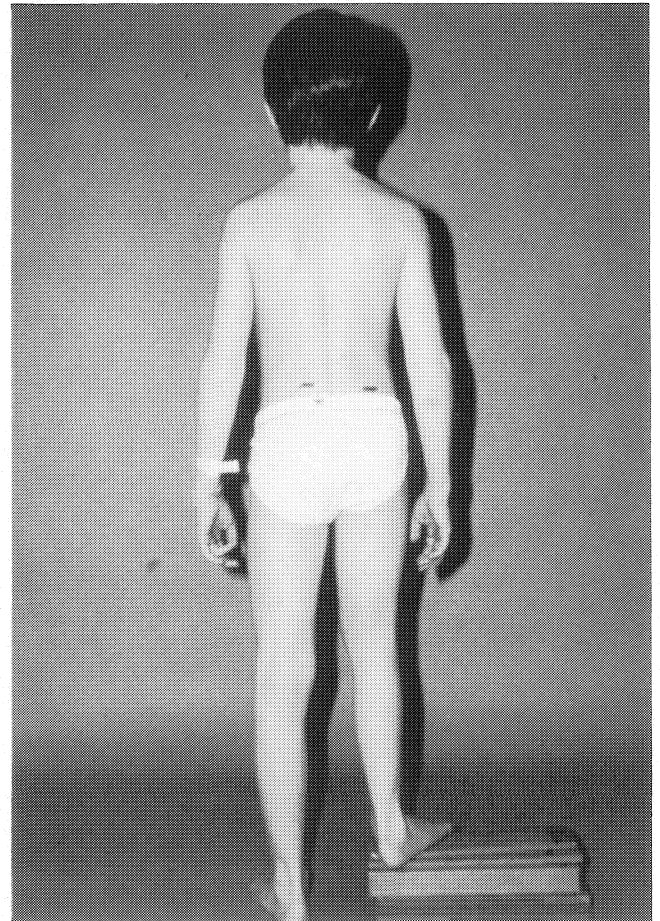

Fig. 1
Limb length discrepancy can be evaluated by using elevations under the shortened extremity to level the pelvis.

The third and most reliable method of measuring the degree of discrepancy is by orthoroentgenogram or scanogram[4,11]. These roentgenograms involve the measurement of limb length by roentgenograms taken with the limb on a measuring grid or ruler. Orthoroentgeno-

grams involve three exposures taken with the x-ray beam sequentially centered directly over the hip, knee and ankle. Scanograms involve a similar technique except that all the joints appear on one film. The total limb length is then measured from the capital femoral epiphysis to the middle of the articular surface of the tibia at the ankle. By these techniques one can also determine the location of the discrepancy, i.e. femur or tibia, by measuring their respective lengths. The femur is measured from the capital femoral epiphysis to the lateral femoral condyle, while the tibia is measured from the dense subarticular line at the tibial plateau to the middle of the articular surface at the ankle. These individual measurements may be of value if it is important to keep the segments of the lower limb equal. However, equalization of the corresponding segment may not always be possible or desirable[6].

These techniques fail to take into account the contributions of foot and pelvic discrepancies in total limb length discrepancies. In addition, they can be invalidated by any patient position changes during roentgenogram exposures.

Flexion deformities may lead to inaccuracies in measurements. In general minimal hip flexion deformities will be compensated for by increased lumbar lordosis when the patient lies on the x-ray table. With significant contractures trigonometric methods outlined by Amstutz and Sakai can be used to calculate actual discrepancies[1]. It is most important to employ all of the above methods in each patient to insure accuracy of data as each serves to validate the other.

Although difficult, the physical maturity of the patient must be assessed. Skeletal age as determined by the method of Greulich and Pyle using a hand and wrist roentgenogram is the most widely used criteria[12]. This method may be difficult to use in cases where the ossification centers appear in a different order or enlarge at rates different than described in the atlas. In certain skeletal dysplasias or congenital hand anomalies the atlas cannot be employed. There are also technique problems in that standards of comparison may be as much as fourteen months apart. Other maturity evaluation techniques have been developed which are a more precise estimate of skeletal age, but these have not yet been applied or correlated with limb length discrepancy[14,16,19].

A complete past medical history, including parental and sibling heights should be obtained. The general evaluation of the patients may reveal findings which may influence the options of treatment. Findings such as hypertension, previous history of osteomyelitis and neurovascular abnormalities may have a significant bearing on the risks of the various treatment modalities. The psychological status of the patient must also be considered. Personality defects may limit treatment options and when considering a lengthening procedure it must be determined if the patient is psychologically suited to endure the prolonged hospitaliza-

tion time and repeated surgeries often necessary with this method of equalization.

The patient should have a complete physical examination to determine their general health and more specifically, the physical status of the extremity. The physical status of the extremity plays a most important part in selection of treatment options. The range of motion and stability of the knee and ankle joints, gait patterns, neurologic status (particularly in cases where lengthening is being considered) and the strength and balance of the musculature about the shortened limb must be evaluated. For example, finding any existing deformity of the foot and ankle which would prevent the foot from being made plantigrade would be a contraindication to tibial lengthening which may accentuate the deformities. Lengthening or shortening an extremity with muscle imbalance or with already weakened musculature must be considered cautiously, for it may impair the patient's ability to ambulate.

The expectations of the patient and family must be taken into consideration. Their goals should be considered when determining which option for equalization will be selected. Cosmetic consideration must be taken into account. Limb length discrepancy relative to body proportion is important when considering lengthening or shortening procedures. Distortion of body image should be avoided. The use of full length body cutouts may help in avoiding distortion of the patient's body image such as might occur by a shortening procedure in a patient with long arms leading to an anthropoid appearance.

Once the above information has been obtained, attention should be turned to the patients pattern of growth. Two questions must be answered. What has been the pattern of past growth and has it been proportional or varible; and what is the predicted future growth?

In 1937 Todd laid the foundation for predictions of future growth by introducing the concept of skeletal maturation[20]. Gill and Abbott in 1942 emphasized that growth, maturation, and aging are different but related processes[8]. Coupled with refinement of the concept of skeletal age by Gruelich and Pyle in 1959[12] and the growth studies of Green, et al.[9-11], and Anderson et al.[2,3] the present day scientific basis for prediction of future growth was devised.

Anderson, et al., documented actual tibial and femoral lengths by orthoroentgenograms in sixty-seven male and sixty-seven female patients and developed this in percentile standard deviation charts[3]. These charts allow us to measure the lengths of the tibia and the femur and the growth remaining in relation to the standard deviation position. Anderson, Green, and Messner also developed growth remaining charts, again based on patient radiographic data[2,9,10].

The growth remaining charts allow the physician to make decisions referable to equalization procedures. This is based on the patient's growth percentile and skeletal

age. By this method the percentage of growth inhibition over a given time interval (recommended minimum three months) is determined by the formula:

percent growth inhibition =

$$\frac{(\text{growth in normal limb, length}) - (\text{growth in abnormal limb, length})}{\text{growth in normal limb, length}} \times 100$$

This is then used in conjunction with the growth remaining charts to determine the time of intervention. Past growth is most important in predicting future growth. Growth between ages four and ten is linear. If a patient is in the first standard deviation above the mean at age seven he is likely to be at that position at skeletal maturity. Therefore having as many measurements prior to the growth spurt will increase reliability. If the patient is only evaluated during the growth spurt, then the relationship of skeletal age to chronologic age becomes very important, i.e., is the patient behind or ahead of his chronologic age. Using the growth remaining method one must gather data on the lengths of the lower extremities, chart the relationship between discrepancy and age to outline the pattern of discrepancy, the percentile standing of the normal and abnormal limbs and the skeletal age[17].

Fries converted the growth remaining graphs to straight line equations (Table I). He based these on the fact that expected growth due to the epiphyses about the knee is approximately linear up to age fifteen in males and age thirteen in females. After these ages, in the period of nonlinear growth, there was less than 0.8 cm of growth expected from the knee epiphyses and he felt that this could be disregarded[7]. This method provides a quick memorizable means of utilizing the Green and Anderson growth remaining graphs.

TABLE I

Male	R distal femoral epiphysis + 1 1/2 age = 23
	R proximal tibial epiphysis + age = 15
Female	R distal femoral epiphysis + 1 1/4 age = 17
	R proximal tibial epiphysis + age = 13

R = remaining growth in cm
Age = skeletal age (Greulich and Pyle Atlas)

The newest method used to evaluate limb length discrepancy is the Moseley straight line method[14,15]. This method provides the treating physician with means of clearly and accurately documenting past growth as well as an end point in predicting further growth. It also allows the manipulation of the growth lines so as to see the effect of various means of equalization.

Moseley demonstrated in applying this method in twenty-three patients a mean error of 0.6 cm compared to a mean error of 0.9 cm when the Green and Anderson method

was used in the prediction of limb length inequality[14,15]. Others have reported similar results[5,18].

The straight line method is based on two concepts. First, that limb growth can be graphically represented by a straight line. This is done by mathematical manipulation of the Abscissa. The short limb is also represented by a straight line below that of the normal leg. The limb length discrepancy at any skeletal age is represented by the vertical distance between the two lines. The percentage growth inhibition of the shorter limb is represented by the difference in the slopes of the two lines (Fig. 2). The normal leg line slope is arbitrarily defined as 100 percent.
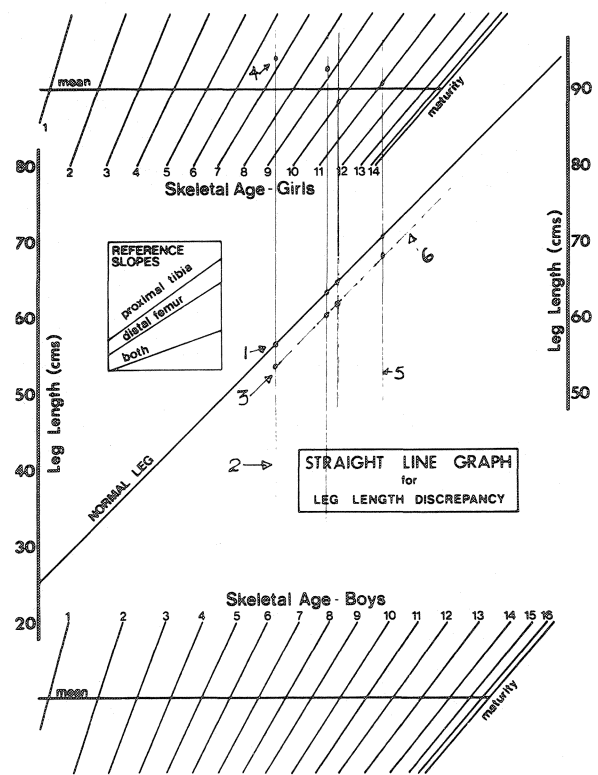

Fig. 2
Moseley Straight Line Method (patient with hemiatrophy) for depiction of past growth.

The second concept is that a nomogram relating limb lengths to skeletal age can provide a mechanism for considering the child's growth percentile and its relationship to the overall limb length discrepancy. Here skeletal age data are plotted with reference to sloping lines whose positions are based in the growth data of Anderson, et al.[3,14]. By obtaining multiple skeletal ages at different evaluations, one can gain an estimation of the child's lower extremity length at skeletal maturity. This gives a representation of the percentile in which the child's growth plot

belongs and allows the placement of a vertical line representing growth cessation (Fig. 3).
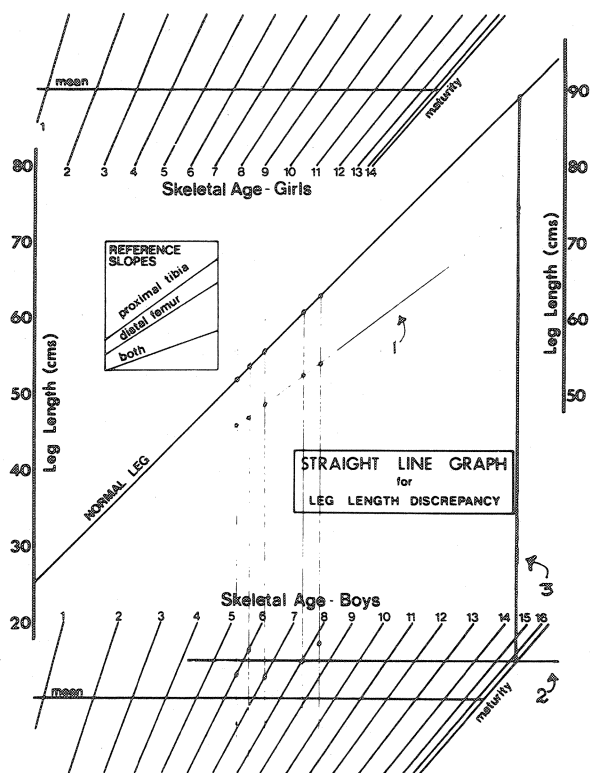

Fig. 3
Moseley Straight Line Method (patient with congenitally short femur) for depiction of future growth.

This method gives a visual picture of past and predicted future growth. Also manipulation of the growth lines, the equalization procedures can be visualized. The growth line of the shortened limb will then approximate a straight line of the same slope but displaced upwards by an amount equal to the lengthening achieved. Also the length of the lower extremity that has undergone epiphyseodesis will thereafter approximate a straight line of decreased slope, where the decrease in slope exactly equals the percent contributed by the fused growth plate to the total growth of the extremity. The proximal tibial growth plate contributes 28 percent and the distal femoral plate 37 percent to the growth of the limb[2]. Therefore one can predict the amount of inhibition produced by epiphyseodesis. The growth line of the leg operated on will therefore have a slope of 72 percent, 63 percent or 35 percent of normal depending on if the epiphyseodesis was of the proximal tibia, distal femur, or both[15].

There are several inherent problems with this method. First, it assumes that the growth of the short limb is straight. As Shapiro has demonstrated, this is not neces-

sarily true[17]. The method also assumes that a child remains in the same percentile as regards growth with respect to skeletal age. In this method (and the "growth remaining" method) such factors as nutrition, activity levels, and patterns of growth in nonwhites cannot be accounted for, nor can it account for increases or decreases in the rate of epiphyseal growth after equalization procedures[14].

The method as described by Moseley is as follows[14,15]:

Depiction of Past Growth (Fig. 2)
At each assessment of the patient three values are obtained:

1. a) length of long limb measured by scanogram from most superior part of femoral head to middle of articular surface of tibia at ankle
   b) Length of the short limb by same method
   c) Radiologic assessment of skeletal age
2. Place point for long limb on normal limb line at appropriate length (Fig. 2, Step 1)
3. Draw vertical line through the point plotted which extends into the skeletal age area of either boys or girls as the case may be (Fig. 2, Step 2)
4. Place point for short limb on vertical line at appropriate length (Fig. 2, Step 3)
5. Plot point for skeletal age along vertical line where it crosses appropriate skeletal age area — interpolating between two skeletal age lines as necessary (Fig. 2, Step 4).
6. Plot successive sets of three points in the same fashion. Each assessment will be represented by a vertical line on which three points for that visit are plotted (Fig. 2, Step 5).
7. Draw the straight line which best fits the points plotted previously for successive lengths of the short limb; this then represents the growth line for the short limb (Fig. 2, Step 6).

Prediction of Future Growth (Fig. 3)
1. Extend growth line of short limb to the right (Fig. 3, Step 1)
2. Draw a horizontal straight line which best fits the points plotted in the skeletal age area. Due to the inaccuracy inherent in estimating skeletal age these points do not closely approximate a straight line. The horizontal line should be drawn so that the total of the distance of points lying above the line is equal to the total for points lying below the line (Fig. 3, Step 2).
3. From the point at which the horizontal straight line meets the sloping line of maturity, draw a vertical line which intersects the growth lines of the two limbs. These points of intersection provide prediction of anticipated limb lengths and the anticipated discrepancy at maturity (Fig. 3, Step 3).

To use this chart as a tool to determine surgery schedules, the growth lines are manipulated either by displacing the growth line of the short limb upwards for lengthening or by decreasing the slope of the growth line of the long limb by epiphyseodesis in such a way that the two growth lines will converge at the cessation of growth.

The straight-line method has several advantages. In considering that skeletal age determinations are somewhat inaccurate, it is an advantage of this system that growth lines can be determined without reference to skeletal age. Errors made in scanogram reading can readily be seen as they do not fit previous growth lines or patterns. No calculations are required and most significantly growth inhibition is readily demonstrated and taken into account in making decisions regarding the timing of surgery[14,15].

It should be emphasized that using this method as a predictive tool requires several determinations. It should not be used with only one determination; with one determination it is impossible to deteimine growth inhibition.

Surgical treatment plans for correction of limb length discrepancies requires an accurate assessment of limb lengths over a period of time i.e., pattern of past growth, a prediction of future growth, and a proper perspective of the treatment modalities available viewed in light of the patients' and families' expectations.

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TOTAL ELBOW REPLACEMENT

Daniel E. Lee, M.D.
Richard J. Friedman, M.D., FRCS(C)
Department of Orthopaedic Surgery
Medical University of South Carolina
171 Ashley Avenue
Charleston, South Carolina 29425

Joint replacement of the hip, knee, shoulder, and interphalangeal joint for rheumatoid arthritis is now relatively routine, whereas total elbow replacement (TER) is still often considered not as successful[10]. Early attempts at replacement were fraught with a high number of complications. Improved prosthetic designs, innovations in surgical techniques, and increasing experience have provided favorable results[4,6,9,12,16].

The elbow is a complex specialized hinge joint with three separate articulations. Along with the shoulder, it allows placement of the hand in a useful position in the lateral and frontal planes of the body[1,4]. It is not possible to obtain a useful functional position with traditional arthrodesis, so other procedures are needed to relieve pain and restore function when disabled by rheumatoid arthritis[4,16].

The modern age of TER began in the early 70's with Dee's utilization of a metal constrained hinge prosthesis fixed with methylmethacrylate[2]. The predominant complication of this prosthesis was loosening (31 percent), due to the constrained hinge transmitting large torque loads directly to the bone-cement interface[4,18]. Since then there has been many changes in prosthetic design with virtual abandonment of the fixed constrained hinge model.

Two main types of prostheses have emerged: semiconstrained and nonconstrained[16]. Both permit laxity or "play" in the joint with movement possible about three axes — medial-lateral, flexion-extension, and pronation-supination (axial rotation)[4,12,13,16,18]. The semiconstrained prosthesis is a loose type hinge, or snap fit, with polyethylene bushings. The nonconstrained or resurfacing designs attempt to duplicate humeral and ulnar anatomy, relying on local soft tissues and congruous joint surfaces for stability. An inherent advantage to this type of prosthesis is that minimal bone is removed during implantation, thus allowing more options if the joint fails[1,4,7,14,16,17,19,20].

Initially, a radiohumeral prosthetic articulation, which would theoretically distribute forces more evenly to the distal humerus, was designed with these prostheses[4,16]. However, no conclusive evidence arose as to whether this imparted any advantage to the patient. In fact, the extra articulation further complicated the surgical procedure with a high failure rate, and has virtually been abandoned.

CONSTRAINED

In 1977 Dee reported on forty of his metal-to-metal elbow hinge arthroplasties with follow-up from two to five years[3]. The major complication was loosening and there were eleven of these (27.5 percent). The same year Gschwend reported on thirty-nine cases using a metal-to-metal single center hinge with an average follow-up of thirty-seven months[11]. Again, the major complication was loosening (twelve cases, 31 percent)[4].

In 1984 the Royal National Orthopaedic Hospital reported on fifty elbows replaced for rheumatoid arthritis using the Stanmore prosthesis, a metal-to-plastic hinge, between 1970 and 1982[13]. The prosthesis consists of separate humeral and ulnar components made of a chrome-cobalt alloy, which articulate across a polyethylene bearing. There was a 61 percent complication rate with nine prostheses appearing radiographically loose, but only two symptomatic enough to require revision. They reported thirty-four good results with complete relief of pain in 77 percent.

SEMICONSTRAINED

The semiconstrained prosthesis allows axial as well as varus-valgus motion. This permits the soft tissues about the elbow to absorb forces and decreases the stress at the bone-cement interface[10]. Most of these prostheses have been developed in United States[4].

In 1981, Morrey and Bryan reported on their experience with forty-seven Mayo and thirty-three Coonrad TER performed in seventy-two patients with an average follow-up of four years[18]. The Mayo prosthesis is a snap-fit articulation of metal-to-plastic design which allows play in the joint. The Coonrad prosthesis is a hinged metal to plastic design which yields several degrees of play across the articulation. Satisfactory results were obtained in 80 percent of patients with rheumatoid arthritis. There was a 55 percent complication rate with 24 percent requiring revision.

Inglis and Pellicci followed thirty-six TER in thirty-one patients for an average of 3.7 years[12]. The first seventeen replacements utilized the semiconstrained Pritchard-Walker prosthesis, while the remainder used the triaxial pros-

thesis, which has more play at its articulation. There were twenty good results, nine satisfactory, and seven failures which required revision. There was a 53 percent complication rate, with the best results obtained in patients with severe pain and altered architecture.

NONCONSTRAINED

Unconstrained prostheses have expanded most rapidly in number and represent the greatest success in total elbow arthroplasty. They rely on normal or near normal ligaments and capsular structures as well as proper alignment and congruency of the articulating surfaces for success[4,5,8,9,16].

Ewald reported on 154 capitello-condylar elbow arthroplasties performed from 1974 to 1981[6,7]. Ninety percent of the patients had a good or excellent result based on pain relief, improved function and motion. There was only one case of prosthetic loosening. Other complications requiring revision were one case of deep sepsis, four dislocations and one olecranon fracture. Partial permanent ulnar nerve palsy occurred in eight patients.

Davis et al. reported the results of thirty capitello-condylar implants in twenty-seven patients with an average follow-up of forty months[1]. There was a 93 percent success rate with moderate increases in flexion, pronation, and supination without increased extension. Complications included deep wound infection in 6.6 percent, subluxation in 13 percent, and ulnar nerve paraesthesiae in 10 percent.

Rosenberg and Turner had an 86 percent success rate with twenty-eight capitello-condylar elbow arthroplasties[20]. There was one case of loosening and four cases of postoperative dislocation requiring revision. All patients had significant increases in range of motion.

SURGICAL INDICATIONS

In the patient with rheumatoid arthritis, pain in the presence of joint destruction is the primary indication for TER (Fig. 1 and 2). Loss of motion and function are secondary indications but may become very important when there is a remarkable loss of function, i.e. inability to perform personal tasks[16].

In patients with pain, preservation of functional motion and minimal joint destruction on roentgenograms, synovectomy and radial head excision must be considered, as this gives satisfactory results in 70 percent of patients up to five years postoperatively[12,22]. Progressive joint destruction with pain, instability, decreased motion and loss of function is considered sufficient indication for replacement arthroplasty[4,16,21]. The goal of prosthetic replacement is to restore painless, functional motion (30 degrees to 130 degrees flexion and a 100 degree arc of rotation) which most of the total elbow prostheses provide[15].
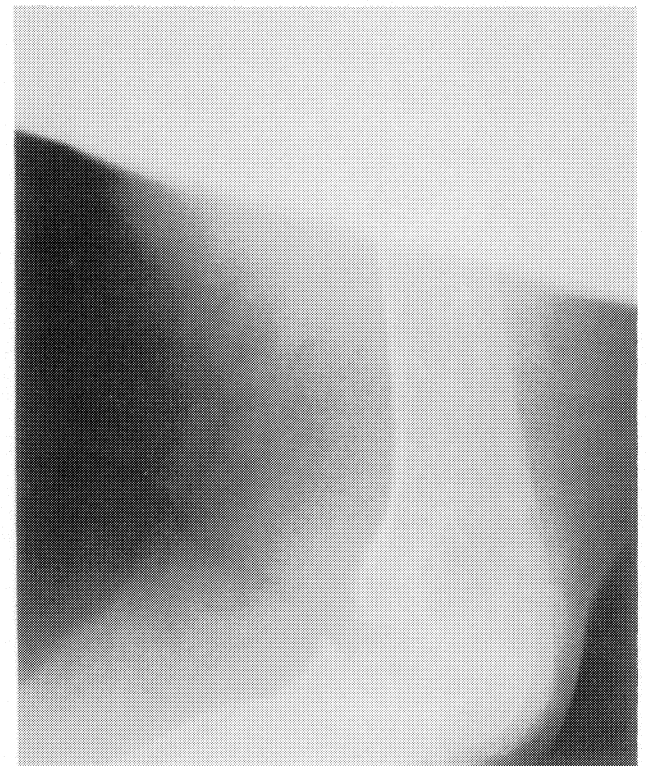

Figure 1a

Figure 1b
Anteroposterior and lateral radiographs of the right elbow in a fifty-three year old female with severe disabling functional class IV rheumatoid arthritis.
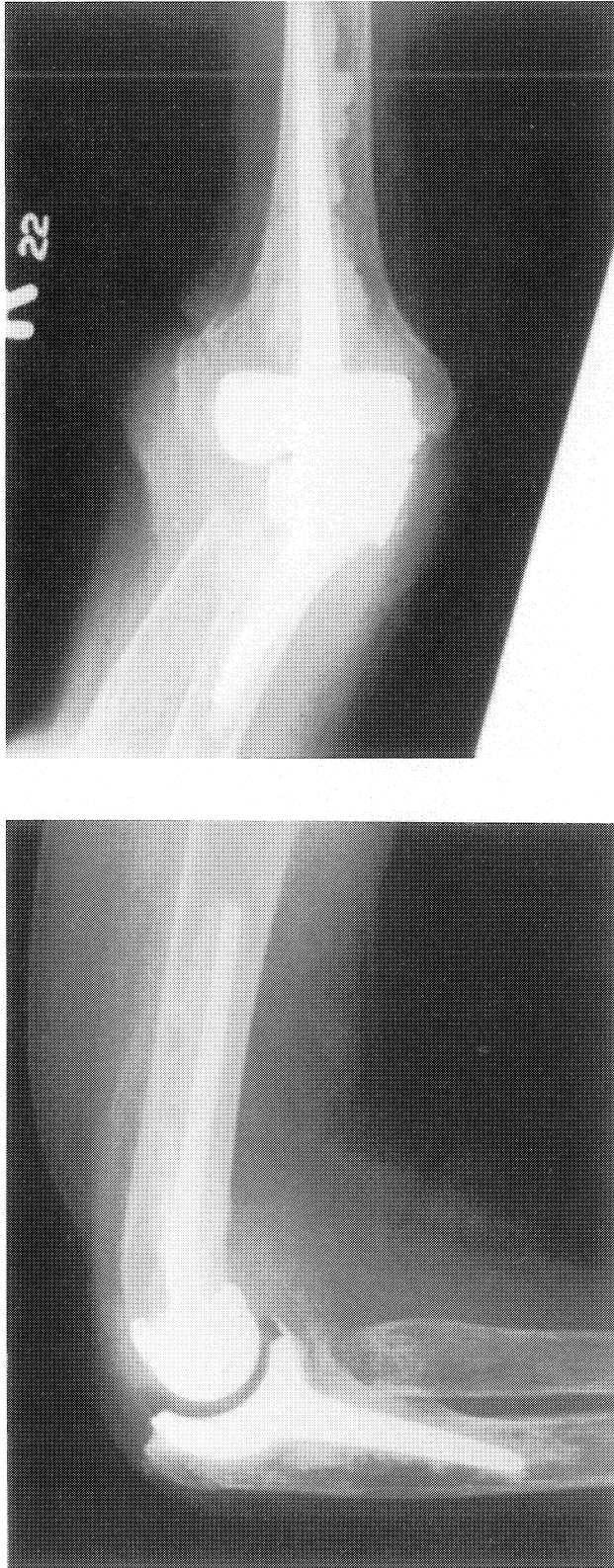
Figure 2
Anteroposterior and lateral radiographs four years following implantation of a nonconstrained capitello-condylar total elbow prosthesis.

Nonconstrained TER appears to provide the lowest rate of prosthetic loosening and other major complications compared to other designs[1,6,17,20]. The use of variable sized components and the posterolateral approach have also contributed to a high success rate and fewer complications such as loosening, sepsis, wound healing, and ulnar nerve palsy[1,6,7,17,19].

Experience and precise careful surgical technique will enhance success and minimize complications. In conclusion, a nonconstrained TER in patients with rheumatoid arthritis affords excellent pain relief, improvement in range of motion and significant functional improvement.

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THE COMPUTER IN PEDIATRIC ORTHOPAEDICS: STUDIES IN ORTHOPAEDIC ASPECTS OF MYELOMENINGOCELE

Malcolm B. Menelaus, M.D., FRCS, FRACS
Director, Department of Orthopaedic Surgery
The Royal Children's Hospital, Melbourne

Teaching, service and research are, of course, the three legs of the stool on which our various departments stand — cut off any leg and the stool falls over. In this paper, I advance the proposition that a computer program can be of value in these three areas and that multicentered studies carry advantages.

The key to computer usage is to define what you want to do with the computer. I wanted to study the natural history of a disease process influenced by many variables. Then, in 1976, I went to Berne to a meeting of the International Society for Research into Hydrocephalus and Spina Bifida. There, David Shurtleff, a pediatrician who is Head of the Congenital Defects Division of the University of Washington, Seattle, gave a paper describing the natural history of spinal deformities in myelomeningocele patients[3]. Figure one presents some of his material: the horizontal axis represents the age in years and the vertical axis represents the percentage of patients with structural scoliosis of more than 30 degrees. You will note that 85 percent of the patients with thoracic lesions had this degree of scoliotic deformity by maturity. Even more interesting than the actual figures was the fact that this graphic material was based on some 2000 observations of 500 patients. I had found a man who had an interest in the natural history of deformity in myelomeningocele patients, and who had a method of keeping accurate records of a vast number of variables in these many patients.

ACQUISITION OF SCOLIOSIS

BY CUMULATIVE PERCENT

Figure 1

It soon became clear that data from a large number of centers was necessary for such studies as this study of spinal deformity. By the time the patients are split up into the neurosegmental level of the lesion, age, presence or absence of hydrocephalus and presence or absence of various associated deformities related to that being studied, you require a large number of patients for any useful study. The need for such multicenter studies has become greater and will continue to increase as a result of an anticipated fall in the number of affected patients (resulting from antenatal diagnosis and from the birth of less severely affected children following caesarian section). The International Myelodysplasia Study Group now consists of thirteen centers in the United States, two in Canada and two in Australia.

The 1960's was the decade of establishment of myelomeningocele clinics; the 1980's is the decade for collaboration between these various clinics and the International Myelodysplasia Study Group has provided the mechanism for such collaboration.

Computerized studies have their greatest application when there is an excess of variables to be studied, an excessive number of patients and where standard methods of analysis are inadequate.

Multicenter studies are applicable when no single center can provide the patient numbers. Furthermore, ethical considerations prevent us from performing random trials of treatment methods, yet we can compare varying management elected by varying centers. Identical data collection at the varying centers ensures valid comparisons: the studies are prospective.
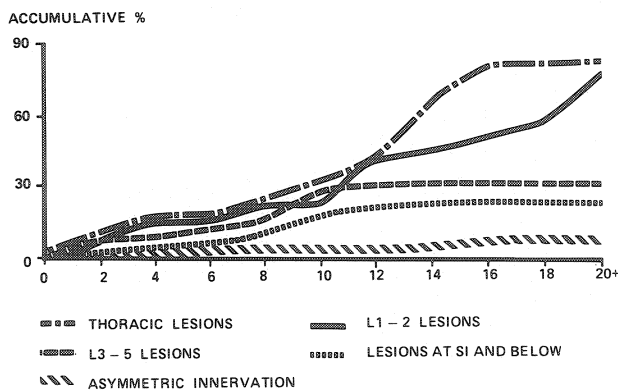
THE DATA COLLECTED

The data file was established almost thirty years ago. This long span enables studies of various aspects of the natural history of the disorder. Several million observations on nearly 7000 separate examinations of more than 1100 patients are now on file.

The data is recorded on a record sheet which contains permanent information (which has to be collected on one occasion only) and changing clinical information. The permanent information records some forty facts (relating to the circumstances of the birth of the child and of the parents) and there are some 400 available responses from which the appropriate forty are selected.

The changing and cumulative clinical information records, from the orthopaedic viewpoint, some 300 facts with some 3000 available responses. The appropriate response merely has to be circled.

From the permanent information, and in particular from the obstetrical information, some very interesting facts have emerged[2]. They are presented in Figure 2. The left column of this histogram indicates those eighty-four patients who were delivered following normal labor. The right column represents those twenty-seven patients who were delivered by caesarian section at thirty-six weeks gestation. It is to be noted that 65 percent of patients delivered by caesarian section had either sacral lesions or no neurological deficit, whereas less than 20 percent of patients born by vaginal delivery were in this fortunate situation. You will note the significant P value of this observation. In other words, caesarian section is likely to be followed by less severe degrees of neurological deficit. Some mothers state that they would not have an abortion even if a neural tube defect were diagnosed; those mothers will accept caesarian section at thirty-six weeks gestation, so we now have an alternative to abortion, an alternative which considerably reduces the risk of a major disability.
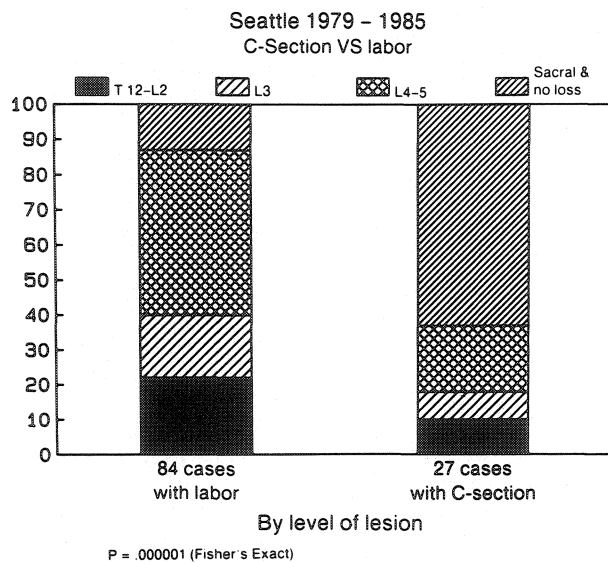

Figure 2

The changing clinical data which may be collected relates to social factors, general medical factors, occupational therapy, physical therapy, psychological factors, neurosurgery, urology, as well as orthopaedics. Members of the study group vary in the completeness with which they fill out the data sheets. Neurosurgical and neurological data is not collected at our hospital. It is important that the psychological information be recorded as, from the orthopaedic point of view, we need to have this information; intelligence scoring and other psychological data is necessary for studies of ambulation and all functional activities.

The orthopaedic data includes all those features necessary for studying the natural history of a locomotor disability and of the results of orthopaedic management. The usual method of locomotion is selected from twelve alternatives; for those who walk the type of gait is selected from twelve alternatives.

DATA COLLECTION

Clearly, there are certain prerequisites for multicenter studies. The first of these is consistency of data collection. This has been ensured by strenuous policing of data collection so that all data entering the study has been collected only by those who are suitably qualified. In general, these are physiotherapists or occupational therapists who have been trained in Seattle. The physiotherapist who has been working on this program in Melbourne visited Seattle on four occasions and has carried out inter-observer error tests in Seattle. These have been published. Of course, data is also collected by orthopaedic surgeons carrying out specific studies.

The essence of the success of the program is close cooperation at all levels; there has been close cooperation in decision making as to the definition of deformity, the data which it is appropriate to collect and the method by which it is collected. During each study, we check samples of our data by returning to data sheets and even to patients' histories.

Multi-center studies have their greatest application when they are relatively simple in structure, as when the natural history of one variable is studied, or when the management influence of one variable is under study. Studies of treatment methods are difficult to carry out when the material arises in varying centers. Seldom do two centers have the same indication for a specific procedure. This does not preclude the use of data sheets or print-outs for these single center studies, nor does it preclude comparison of results of treatment of a specific deformity in various centers. Presently, data collection is carried out at six monthly intervals up to the age of two years and then annually.

STUDIES BASED ON COMPUTERIZED MATERIAL

Dr. Shurtleff has carried out fundamental studies such as the cumulative survival rate for patients born between 1955 and 1965 compared with those born since 1975 (Fig. 3)[2]. He has further studied, in myelomeningocele patients, the changing relationship of lower extremity length to arm span as it occurs with growth and he has compared this with the normal population.

Returning to orthopaedic studies — we have designed studies to determine whether early assessment of quadriceps power is reliable. It is generally recognized that
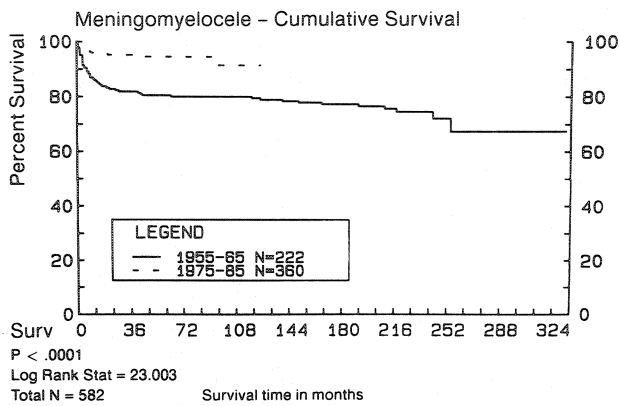
Meningomyelocele – Cumulative Survival

Figure 3

children with quadriceps muscles which are sufficiently powerful to enable them to walk in below knee orthoses remain long-term walkers into adult life. Such children benefit from more sophisticated hip surgery than is necessary for those with weak quadriceps muscles. This hip surgery is commonly best performed in the first three years of life yet during this period it is not easy to grade power accurately. In order to determine the reliability of our grading of quadriceps power in these first few years, we compared gradings in that period with that for the same child in the latter part of the first decade (Fig. 4). The first column of this histogram represents those patients whose quadriceps power remains the same or is increased in the interval between early assessment (first three years of life) and late assessment (latter part of first decade). The second column represents those patients in whom the power of the muscles deteriorated during this period. You will note that 98 percent of Grades IV and V lie in the first of these two columns: that is, for 98 percent of patients Grade IV increased to Grade V or grading as V (which by definition cannot become any stronger) remained unchanged. Thus, early assessment of quadricep power for these two grades is reliable.

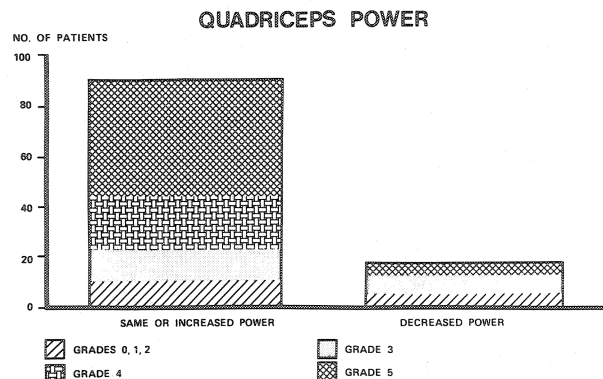Many children with myelomeningocele require teaching and repetitive practice in order to perform tasks which unaffected children can perform untaught and unpracticed. Many do not have a dominant hand, have difficulty in distinguishing the relevant objects in the foreground from the total display of objects in front of them, and frequently there is poor hand-eye coordination. These disabilities have grave implications for the performance of activities of daily living and in the ability to use walking aids.

Valuable early studies in feeding, hygiene and dressing were carried out by Seattle workers and further studies, based on the Seattle data, have been performed in Melbourne[1]. The patients were categorized as follows:

Group IA — in which there was thoracic paralysis of the lower limbs,

Group IB — in which there was thoracic paralysis of the lower limbs and spastic upper limbs,

Group IIA — in which there was spastic paralysis of the lower limbs

Group IIB — in which there was spasticity in upper and lower limbs.

Figure 5 indicates the relationship between independence in skills of daily living and neurological group. Patients with normal upper limbs (Groups IA and IIA) were more likely to be independent than those with spastic upper limbs (Groups IB and IIB). The same study indicated that spasticity had a profound influence on walking ability; when we analyzed patients with thoracic lesions we found that the number of community walkers decreased from six out of thirteen in Group IA to one out of seven in Group IB, to none in Group IIA and none in Group IIB. In patients with upper lumbar lesions, eleven out of twelve were community walkers in Group IA, none in Group IB, three out of ten in Group IIA and one out of five in Group IIB. The presence of spasticity thus has a profound influence on the quality of life of the patients with myelomeningocele. Patients who had spasticity in their upper limbs were more likely to require special schools; six out of ten children in Group IB and eleven out of eighteen in Group IIB attended special schools for the disabled.

QUADRICEPS POWER

Figure 4

Graph 2: Independence in Activity of Daily Living (ADL Skills)
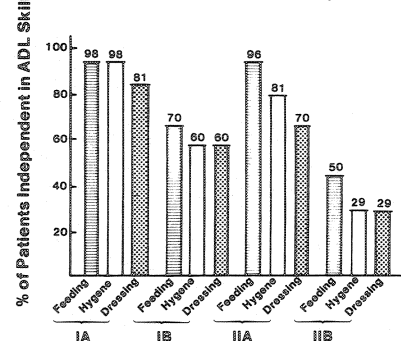
Figure 5

HIP FLEXION DEFORMITY

Various imputations of disability have been leveled at fixed flexion deformity of the hip; these include poor standing and walking ability, increased lumbar lordosis, difficulty in brace fitting, and increased risk of trophic ulceration and poor sitting balance for those with asymmetrical flexion deformity.

Figure 6 illustrates desirable posture for a patient with myelomeningocele. This girl has predominantly a low lumbar lesion but there is some quadriceps weakness requiring KAFO. Because she has no flexion deformity at the hip, her center of gravity lies vertically above her feet. Those who develop a fixed flexion deformity of the hips (Fig. 7) have their center of gravity well in front of their feet, so that theybecome crutch dependent. Figure 8 indicates the improved posture obtainable by correcting flexion deformity at the hip; in this boy extension osteotomy has been employed, commonly a soft tissue release is adequate. Figure 9 illustrates desirable extension posture at the hips.
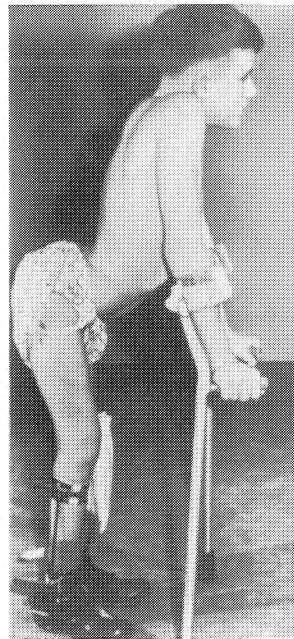

Figure 7   Figure 8

Boy, aged twelve years, with bilateral fixed flexion deformities of the hips amounting to approximately 90 degrees, despite bilateral anterior release operations.
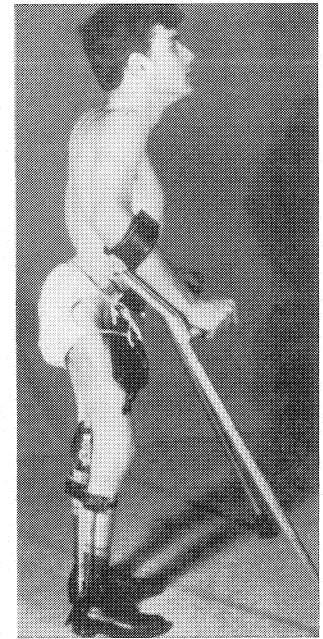
Postoperative posture following bilateral extension femoral osteotomies of the boy shown in Figure 7 at the age of sixteen years. It is important not to extend the osteotomies too much as the combination of fixed lumbar lordosis and limited flexion of the hips will prevent the sitting posture.

Figure 9

This photograph of an eighteen year old paraplegic boy indicates the advantage of hips which will fully extend; his center of gravity lies vertically over his feet.

He is now aged twenty-eight years and remains a community ambulator and, suprisingly, does not possess a wheelchair. Both hips are dislocated and no attempt has been made to reduce them.

Figure 6

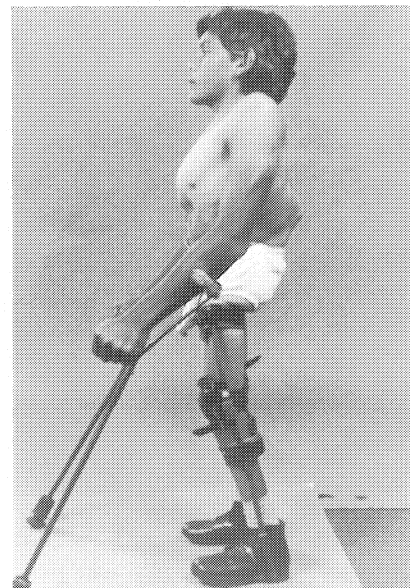Management of fixed flexion deformity at the hip has been beset with problems simply because we didn't know the natural history of the condition. For this reason, we studied, combining material from over 5000 observations of 966 patients, fixed flexion deformity in those who had not been subjected to hip surgery[4]. Figure 10 indicates hip flexion limitation (that is, fixed flexion deformity) regression lines from birth to twenty-seven months, and over the age of twenty-eight months for each of four muscle level groups for patients with a hip flexion contracture of at least 21 degrees. Concentrating first on the first twenty-seven months of life, note that all neurosegmental levels except the thoracic level patients have markedly reducing deformity in this period. This reduction, for all but those with thoracic level lesions, corresponds to that seen in normal children. Thus, for lesions other than thoracic lesions, flexion deformity in the first twenty-seven months of life is a physiological phenomenon and corrective surgery is inappropriate in these patients in this age group.
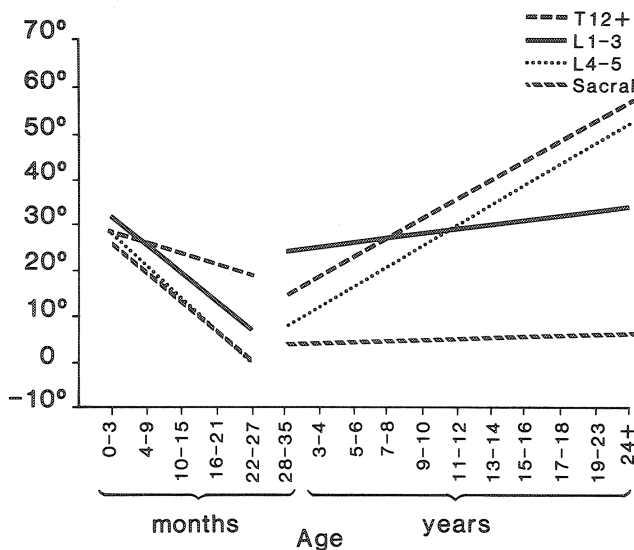

Figure 10
Hip flexion limitation regression lines for zero to twenty-seven months and greater than twenty-eight months of age for each of the four muscle-level groups for single hip examinations of 391 patients with a hip flexion contracture of at least 20 degrees.

Again, looking at the material represented in Figure 10, if we concentrate our attention on the area from twenty-eight months through maturity, we find that there is a marked increase in deformity with age for those with thoracic and low lumbar lesions, but no significant increase for high lumbar and sacral lesions. Other results from this study indicate that severe flexion deformity (defined as more than 40 degrees) occurs if physiological flexion contracture does not resolve or if hip flexion contracture of more than 21 degrees develops by the age of six years.

This data gave us very useful guidelines in our management of flexion deformity. Furthermore, we conclude from this material that treatment of muscle imbalance won't prevent flexion contracture since severe flexion contracture is more common in thoracic lesions where there is no muscle imbalance and flexion contracture is more common with minor imbalance than with major imbalance.

These findings are exciting to me. In the past I have known that some patients with flexion contracture require surgery and that it might be best carried out early. It has not been clear, however, which patients require surgery and at what age it is appropriate. There are still unanswered questions which we are currently trying to solve. Currently satisfactory information is not available on the relationship of flexion contracture to hip dislocation, to the degree of activity of the patient and to possible variations in collagen in different individuals.

A further study, performed in collaboration between Seattle and Melbourne, relates to the benefits of walking in children with high level lesions. On occasions, orthopaedic surgeons may elect to place children with high level lesions into a wheelchair without any period in which these children walk. This approach is justified by the fact that they are more effectively mobile in a wheelchair than when walking. Matched patients have had a period walking (in braces and with crutches) and then elected to use a wheelchair. A comparison of their transfer ability, independent mobility, ability in performing activities of daily living and number of fractures and pressure sores were made. The study continues, but present indications are that the former walkers are doing better on all counts except that they have required a slightly greater number of orthopaedic admissions to hospital.

BROAD IMPLICATIONS OF COMPUTERIZED DATA COLLECTION AND ANALYSIS

In the preceding paragraph, I have been stressing the research usage of this computerized program. In fact, it has been equally beneficial in terms of patient care and education.

PATIENT CARE

By having to fill out the data sheet at regular intervals, we are forced into keeping accurate records of each patient and to keep far more complete records than were kept in the past. The computer printout is there in the clinic with us, changes in deformity or function can be seen at a glance and confirmed clinically.

EDUCATION

The printout demonstrates the natural history of the disorder. Filling out the data sheet is a learning experience for medical or paramedical personnel.

RESEARCH

I have already indicated many research applications. I merely add here that each study made is virtually a prospective study since decisions on data collection are made prospectively.

Max Planck used to say to any student who asked for suggestions for a research topic — "My dear man, if I knew of a problem, I would solve it myself!". With the printout in front of you, this dilemma of finding a research project is greatly alleviated. The display of statistics commonly exposes an unexpected relationship which we are thus stimulated to explore.

The harsh focused light of mathematics which plays on properly designed computerized studies adds accuracy by reducing subjective judgements. The surgeon-investigator is relieved of some of the emotional overtones contained in such words as "poor", "fair", "good"; these terms have been replaced by much more subtly defined and unambiguous number codes.

CONCLUSION

Whilst the system I have described is most applicable to neurological disorders in childhood, we can apply it to almost any musculoskeletal disorder of childhood or adulthood. On occasion, minor modifications may be necessary. Frequently, we will wish to use a small portion of the available data sheets. For children with Legg-Calve-Perthes disease we need merely pull out the permanent information and extend in detail the hip data sheet. This is a disease which requires study yet it is not a common disorder — surely there are good reasons for many centers to be recording identical data here. Juvenile chronic arthritis is another condition which lends itself to data collection in order to characterize the natural history of various forms of the disorder and the effectiveness of treatment. It might even be that from analysis of the permanent information, we stumble on to leads as to the cause of these two conditions. Ultimately, it may be that a large proportion of our patients have data collected in this way in hospital clinical records; that is, the case histories and clinical examinations will be recorded by methods enabling computer storage and subsequent analysis. Treatment will be similarly recorded. Then, there may be great leaps forward in clinical research. Multiple centers with varying treatment philosophies for a wide variety of disorders will be able to compare results with validity.

The computer is the great catalyst for data collection analysis — without it we merely have thousands of facts on scraps of paper; with it we have the possibility for systematic analysis of facts too numerous to be analyzed by other means.

The three great leaps forward in man's capacity to expand knowledge have resulted from the development of an alphabet, of the printed word and of the computer. The latter has not been adequately applied to the study of disease processes.

At the very least, the studies that I have mentioned above indicate how the incidence of children with a severe neurological deficit may be reduced (here I refer to the caesarian section study by David Shurtleff), what the natural history of the disease process is (scoliosis study and the hip flexion deformity study), how wrong we were in the past in assuming that muscle imbalance was the cause of certain deformities (the hip flexion study), and the reliability of physical signs (the study of quadriceps power).

I conclude by stressing the value of Dr. Shurtleff's work in producing this computer program. He has been extremely creative and has led others into making good use of his contributions. It is well recognized in the world of the arts that creativity can inspire creativity in others; this is equally true in medicine. Surely one reason why the Department of Orthopaedic Surgery at the University of Iowa continues to contribute so much to our knowledge of orthopaedics is because the creativity of Dr. Carroll B. Larson inspired creativity in others.

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FRED HARK AND THE CONGENITAL VERTICAL TALUS

Scott Tromanhauser, M.D.
Richard L. Jacobs, M.D.

From the Divison of Orthopaedic Surgery
Albany Medical College
Albany Medical Center
Albany, New York 12208

One of Dr. Steindler's first residents and affiliates was Dr. Fred Hark (Fig. 1). Born in Dysart, Iowa, Dr. Hark attended the University of Iowa in a six year combined program, graduating from the College of Medicine in 1922.

Fig. 1

Fred Hark.

Dr. Hark was then affiliated with Dr. Steindler as an orthopaedic resident, and then as a member of the attending staff from 1922 until 1929. He then went to Chicago with Dr. Phillip Lewin, based at the old St. Luke's Hospital in the 1400 block on South Michigan Avenue until 1930.

He subsequently was associated with Dr. Thomas at the University of Illinois in the West Side Medical Center, and then with his successors, Fremont Chandler, and Robert Ray. He was also on the attending staff at the Presbyterian-St. Luke's after their merger in the late 1950's. After moving to Chicago, the Harks had two children. Bill, an outstanding member of the swimming team at Iowa, graduated from college and medical school there, and followed his father into orthopaedic practice in Chicago after completion of an orthopaedic residency. Jean majored in chemistry at Iowa. Her husband, also a chemist, recently retired from long service at the Quaker Oats company.

Dr. Hark is an author of thirteen major orthopaedic papers. One of the best known of these is his article on congenital vertical talus. This paper will report a typical case of congenital vertical talus treated by his technique and will review the literature.

TWO CASE HISTORIES

A three year old boy had been treated with serial casting, and then a Denis-Brown splint for a presumed diagnosis of bilateral calcaneal valgus deformity of his feet. When examined, he was found to have bilateral severe pes planus, worse on the right than on the left. There was moderate flexibility of the left foot. There was almost no subtalar motion whatsoever on the right side.

Roentgenograms of the left foot revealed dislocation of the navicular relative to the head of the talus. This reduced with forced plantar flexion of the foot. On the right side, the deformity did not correct with forced plantar flexion; the forefoot would not come into register with the head and neck of the talus (ossification center for the navicular head not yet appeared). The child had genetic and developmental consultation after impaired physical and intellectual development was noticed at the age of twenty months. Final diagnosis was hypotonic quadriplegia with mild left hemiplegia. There is also a family history of hemiplegia in the father and a maternal brother. The pediatrician noted that details of both of these conditions were obscure and tended to point toward coincidence rather than a true heritable condition.

The Hark procedure was done on the right foot at age three years and six months. The patient was seen periodically after this, and last examined ten years after the original surgery. His foot was plantigrade. He had a well formed arch and no complaints of pain in this foot. He had thirty percent loss of range of motion of the subtalar and Chopart's joint in this foot. X-rays of his foot demonstrated the navicular in register with the head of the talus (Fig. 2). He had a satisfactory, long term result of surgery, both in terms of function and in terms of avoidance of pain.

Figures 3 and 4 illustrate the unfortunate result of inadequate treatment for congenital vertical talus in a white male who first presented to us at age twenty-four. At age four he underwent two unspecified procedures on both
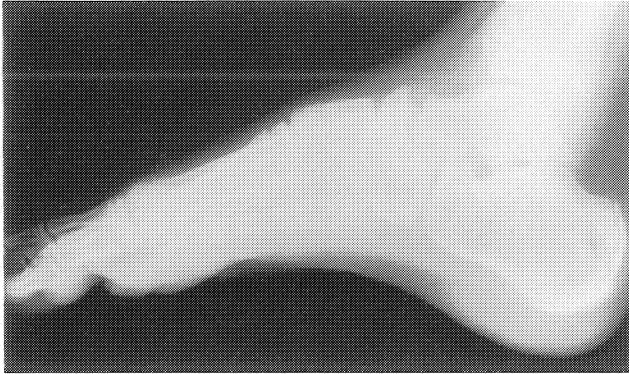
Fig. 2
Ten years after Hark procedure. No pain, good function.

Fig. 3
Thirty years after incomplete correction of deformity, left foot. Pain, limited function.
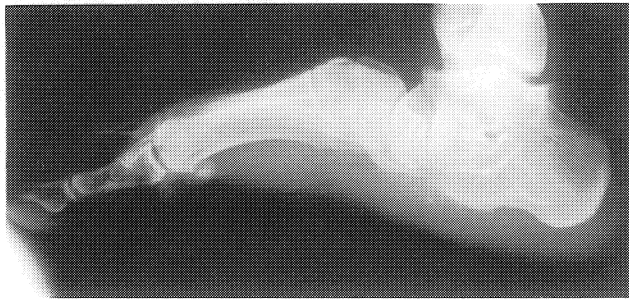

Fig. 4
Twenty years after incomplete correction of deformity, right foot. Pain, limited function.

feet followed by cast treatment for one year, followed by corrective shoes. He now complains of long arch and heel pain that becomes severe with prolonged standing and of lack of flexibility in both feet. He is unable to walk on his toes but with great pain can run and walk distances.

Physical examination reveals bilateral calf atrophy heel equinus and lack of subtalar motion. Ankle motion is limited to fifteen degrees of dorsiflexion and thirty degrees of plantar flexion. His metatarsals are functionally elevated on both feet with no dorsiflexion at the metatarsophalangeal joints and only fifteen degrees of plantar flexion. The metatarsal heads do not bear weight, and there is no push-

off in gait. No heavy corns or callouses were noted, and pulses were good in both feet except for an absent dorsalis pedis on the right. Roentgenograms reveal that the talar head is not aligned with the navicular in either foot. There is deformity of these bones, and the dome of the talus is flattened. The ankle mortise is shallow.

DISCUSSION

The talonavicular joint is dislocated in Congenital Convex Pes Valgus. The navicular articulates with the dorsal surface of the neck of the vertically oriented talus. Secondary changes involve contractures of tendons and ligaments that complicate the talonavicular dislocation. Synonyms are congenital vertical talus, vertical talus, congenital rocker-bottom flatfoot, and rocker bottom foot. Tachdjian suggests a more accurate description would be teratologic dislocation of the talonavicular joint.

The first description was by Henken[3] in 1914. Lamy and Weissman[3] presented an excellent review in 1939. The condition tends to be familial[1,4,5], but presently is presumed to be the result of an insult to the developing fetus during the first trimester of pregnancy. Joachimsthal[4] claims that congenital vertical talus constitutes 4.3% of all foot deformities. The incidence is closer to 10% in meningomyelocele patients[7]. The disorder has also been found to be associated with other central nervous system, neuromuscular or genetic disorders[2,6,7,8,9].

The anatomical abnormalities can be subdivided into bone, ligament, and muscle or tendon changes. Major bony changes are in the talus and calcaneous. The talus is tilted vertically and is misshapened. The head of the talus forms an oval rather than spherical shape, and its navicular and calcaneal facets are correspondingly abnormal. The corresponding changes in the calcaneous are abnormal facet development, and blunting of the sustentaculum talus. The navicular is hypoplastic.

Corresponding ligamentous changes prevent reduction, and necessitate release. The relative dorsiflexion of the forefoot shortens the ligaments surrounding the anterior part of the ankle joint, i.e. the tibionavicular and dorsal talonavicular ligaments. The calcaneocuboid ligament is shortened, but the spring ligament is attenuated.

Along with this are musculotendonous contractures. The tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneal brevis are major obstructions to correction. The posterior tibial and peroneal tendons may bowstring across the malleoli, acting as dorsiflexors of the foot rather than plantar flexors.

These combined changes cause a distinctive "rocker bottom" configuration. The forefoot is adducted and dorsiflexed, and the hindfoot is in equiovalgus. The head of the talus is palpable medially on the plantar surface of the foot.

At birth, roentgenograms show the talus vertically oriented and the calcaneus in equinus. The talocalcaneal angle is increased (the angle formed by the intersection of two lines along the longitudinal axis of the talus and calcaneous as seen on the lateral view). An equally acceptable method is determining the axis of the metatarsus and the talus. A line is drawn through the longitudinal axis of the first metatarsal, and another line drawn through the longitudinal axis of the head and neck of the talus. In the normal foot, these two axes are in alignment. In the vertical talus, there is an appreciable angle. All X-rays should be taken in multiple modes: standing anteroposterior, standing lateral, and lateral plantar and dorsiflexion views. If the relationships between the axis of the talus and the calcaneous fail to correct, or if the axis of the first metatarsal and the head and neck of the talus fail to align, then the diagnosis is confirmed.

Congenital convex pes valgus should be differentiated from other foot deformities. Talipes calcaneovalgus is one of the main differential diagnoses. The deformity does correct on plantar flexion of the forefoot. A similar clinical picture can be seen with muscular contractures or paralytic pes valgus due to neurologic causes; vertical talus, however, is a rigid deformity as confirmed by roentgenograms. At least seventeen different approaches to surgical treatment have been recommended[1]. Hark's paper in 1950[2] described two major defects. First, moderately severe equinus, and second, dislocation at the midtarsal joint. He recommended that the heel equinus be corrected first, by heel cord lengthening and posterior capsulotomy of the tibiotalar joint as needed. The calcaneous is then held down in a corrected position by a transverse wire passed over the top of the tuber. Two longitudinal incisions are made, one lateral to the extensor tendons over the sinus tarsi, and the other medial to the anterior tibial tendon. Fascia, tendon, and capsule abnormalities make reduction impossible. Fascial strands, therefore, are incised, Z-plasty lengthening of the tibialis anterior and extensor hallucis longus tendons is done along with the lengthening of the tibialis posterior and flexor hallucis longus as needed. The lesser toe extensor tendons were then cut, two proximally and two distally. The two long tendons were sutured together and the two shorter then sutured to the sides of the longer. The capsules of calcaneocuboid and talonavicular joint are then incised to whatever degree needed to obtain reduction, and the navicular was reduced. The smooth Kirschner wire is placed through the navicular into the head of the talus and brought out through the skin (to be removed in three weeks). Soft tissue and skin are then closed. A long leg cast is applied, first only the thigh and leg portions with the knee at ninety degrees flexion. The transverse pin above the os calcus is incorporated into this proximal portion of the cast, pulling down the calcaneous.

The forefoot, usually plantar flexed and now in register with the head and neck of the talus, remains held there by the previous placed Kirschner wire. The forefoot is now immobilized with plaster which is incorporated into the rest of the cast with the K-wires for the heel and the talonavicular joint. The talonavicular pin is removed at three weeks. The thigh portion of the cast and the calcaneal pin are removed at six weeks. The patient then is allowed to be weight bearing. The cast is typically removed after a total period of immobilization of four months.

Two cases were presented here. In the first, performing the Hark procedure, the dislocation was completely reduced. Soft tissue releases sufficient to give tension free correction of deformity were done. The end result was a foot with good function and free of pain.

In the second case, full releases were apparently not performed, and the patient remained with appreciable soft tissue and bone deformity and malalignment. Specialized orthotics are being tried, but wedge osteotomies may need to be considered. If satisfactory reduction is not achieved in the operating room when the original surgery is performed, time and prayer will usually *not* improve the results!

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TREATMENT OF GIANT CELL TUMOR OF BONE

Yu-pu Lu, M.D.
Quing-yu Fan,
Qing-liang Wang

Department of Orthopaedic Surgery
First Affiliated Hospital
Fourth Military Medical University
Xian, China

Though uncommon in the Western countries, giant cell tumor (GCT) is one of the most common primary bone tumors in China (ranked first or second) and accounts for fifteen to twenty percent of the registered bone tumors [1,2]. The tumor occurs most often in the ends of long bones especially near the knee. Up to now the tumor was thought to be difficult to eradicate without sacrificing joint function.

Metastases to the lung may occasionally occur from histologically benign GCT. The tumor is considered therefore to be potentially malignant or of low grade malignancy. Because of a high recurrence (from thirty to sixty percent) following conventional treatment by curettage and bone grafting[3,4] other methods of treatment have been sought. Wide excision of the tumor has been routinely used in those situations where joint function would not be affected. However, a dilemma exists in those cases in which the tumor is juxta-articular. En-bloc excisions may compromise joint function while curettage with bone grafting is associated with an increased incidence of recurrence. Many authors prefer a drastic approach with resection of the tumor and the joint. This would require prosthetic replacement, allograft reconstruction or arthrodesis of the joint. Serious complications may follow these procedures eventually leading to an amputation.

The ideal treatment is to eradicate the tumor and to preserve a normal joint with a low risk of recurrence. Remarkable progress has been made toward this goal by the method proposed by the senior author. This article reports on eighty-six cases (sixty-nine previous untreated and seventeen recurrent giant cell tumors) of giant cell tumor.

MATERIALS AND METHODS

Clinical Data:

Sex and age: Out of the eighty-six cases in this series, forty-five were male and forty-one female. Their ages ranged from fifteen to seventy years old. Seventy-seven cases or ninety percent were between twenty to forty years old. Site of lesions: Out of eighty-six cases, seventy-four were adjacent to a major joint and fifty-five, (seventy-four percent) were located above or below the knee as shown in Table 1.

Table 1

Distribution of Eighty-six Cases

Upper extremity:	Humerus:	proximal	3
			1
	Radius	distal	5
	Ulna		3
	Hand		1
Lower extremity:	Femur:	proximal	6
		distal	35
	Tibia:	proximal	20
		distal	1
	Fibula:	proximal	5
	Talus:		2
Sacrum:			2
Ilium:			1
Acetabulum:			1
TOTAL			**86**

CLINICAL MANIFESTATION

Pain was the most common complaint varying from a few months to two years duration. In twenty-five cases (thirty percent) pain was severe and worse at night, not unlike that of an osteosarcoma. Sixty-four cases (seventy-five percent) had limited joint motion because of swelling and pain. Fifteen cases (eighteen percent) had rapidly growing masses. Radiographically, thirteen cases revealed expanding osteolytic lesions without new bone formation. Thirteen cases (fifteen percent) because of extensive destruction were reported as probable malignant neoplasms by the roentgenologist. They were not malignant histologically. In twenty-one cases (twenty-four percent) the cortices were penetrated. Seven had pathological fractures. Microscopically, except for five cases with frank malignancy, all were confirmed to be benign giant cell tumor of bone, Jaffe's Grade I or II.

TREATMENT AND RESULTS OF EN-BLOC RESECTION

In the twelve cases with the lesion located in non-weight bearing bone, i.e. head of fibula, lower end of the ulna, iliac crest, sacrum, en-bloc resections were performed.

Nine of them were followed for two to twenty-seven years with no recurrence noted. Two had early satisfactory results followed for six months. One GCT of the sacrum recurred two years after the first operation. A partial resection of the sacrum was performed with preservation of the S1-2 nerves. The follow-up is only four months.

TREATMENT AND RESULTS OF CURETTAGE AND BONE GRAFT

The other seventy-four cases with benign features include the seventeen recurrent cases referred from elsewhere and were treated by our presently recommended method. These all had benign features and were located adjacent to major joints.

Adequate exposure and thorough curettage for eradication of all tumor tissue was performed under direct vision through a large window in the bone. Good vision of the whole cavity is necessary in order to prevent any gross tumor residual. The defect in the bone was cauterized with a fifty percent aqueous solution of zinc chloride soaked sponges. This solution has been shown experimentally[8] to destroy all live tumor cells as deep as 5mm. yet not affect the healing process of bone grafts. The corrosive liquid should not be allowed to touch any soft tissue especially nerves and vessels. The cavity is then grafted and the wound is closed with a drain left in place for forty-eight hours. Post operatively, a heavy (pressure) dressing for hemostasis and absorption of oozing blood is placed followed by a plaster cast. Precautions are taken to provide protection for a long enough period of time for those cases with potential for fracture and in whom a varus or valgus deformity might occur.

RECURRENCES

Of the sixty-nine cases operated on in our department, there were five recurrences. Two recovered after a second operation and two had metastasis to the lungs. One at the upper end of the right humerus recurred after a second operation which could not be completed by the standard method because of uncontrollable bleeding. This patient recovered after radiation therapy and has done well after a three year follow-up.

The remaining sixty-four cases had satisfactory bone healing and joint function. The recurrence rate in those cases followed for greater than two years was 10.8% (five recurrences in forty-six patients).

Three out of five cases with malignant histopathological features had an amputation and were followed for thirteen, fourteen and sixteen years respectively. One died of pulmonary metastases nine months after amputation. One had an en-bloc resection of the lower end of the left femur and an arthrodesis of the knee with a short follow-up.

RESULTS OF TREATMENT OF RECURRENT CASES

Twenty-two recurrent cases including seventeen referred from elsewhere were treated. The majority of them had recurred within one year and all of them were treated by this method. After the second operation, nine of them were doing well with a follow-up of two to twelve years. Each had a satisfactory result (acceptable bone healing and joint function). Nine cases followed for less than two years had early satisfactory results.

Two required a third operation and have been followed three and six years respectively without any evidence of recurrence. Two patients had recurrences both in the bone and soft tissue. After repeated operations these two developed pulmonary metastases.

Table 2

Period of Follow-up

15-21 years	4 cases
10-15 years	5 cases
5-10 years	9 cases
2-5 years	28 cases
1-2 years	12 cases
Within 1 year	11 cases
TOTAL	69 cases

ILLUSTRATIVE CASES

Case 1 (Fig. 1A-D) Z.X.: 22 year old female with severe osteolytic lesion in the upper end of the right humerus with a pathological fracture. A malignancy was suspected.

Date of operation: May 20, 1978.
Diagnosis: giant cell tumor of bone, Grade I.
Bone graft: autograft
Follow-up: 7 year follow-up with complete healing of the lesion, remodeling of the bone graft and excellent joint function.

Case 2 (Fig. 2A-F) L.B.: 23 year old male diagnosed as giant cell tumor in the proximal end of the left tibia and had two previous operations.

Date of operation:
1st operation
  (elsewhere)   May 1972, 3 years, 9 months earlier
2nd operation   February 26, 1976, curettage, ZnCl2 and autogenous bone graft.
3rd operation   February 1979, 3 years later

The patient was followed for 6 years after the last operation with complete healing of the lesion and excellent function of the knee.
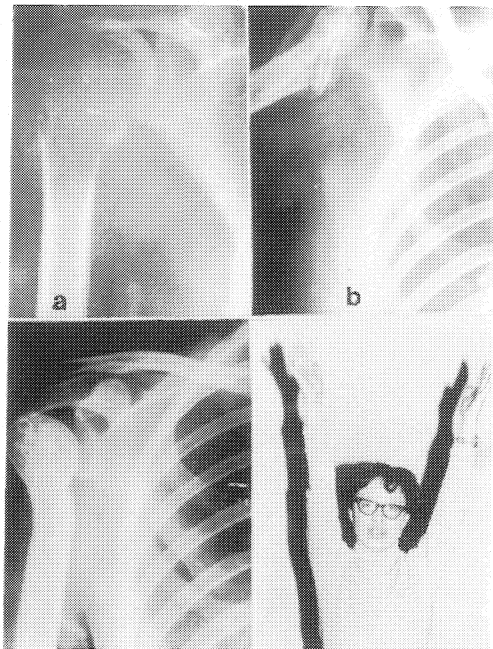
Fig. A X-ray before operation. Fig. B Roentgenogram 2 months postoperatively. Fig. C Roentgenogram 7 years postoperatively. Fig. D Followed for 7 years, the function of the shoulder is excellent.

Fig. 1A-D
Zhang Xiu-feng, a 22 year old female, giant cell tumor of bone of right shoulder.

Fig. A, B
X-ray shows the recurrent appearance before 3rd operation.

Fig. C, D
Roentgenogram 6 years postoperatively.

DISCUSSION

Routine amputation for GCT of bone was indicated in the 19th and early 20th century before the standard treatment of curettage plus bone graft was developed. High local recurrence rate and the occasional "benign" metastasis to the lung have caused considerable debate and confusion over the benign or aggressive nature of the tumor. In order to reduce the recurrence rate, en-bloc resection and reconstruction procedures were advocated by many authors. These techniques, cadaver allograft or prosthetic replacement, have not been without complications[9,10].

The cause of recurrence is incomplete removal of the tumor. Cauterizing the bony wall with a chemical agent can destroy the residual live tumor cells. Liquid nitrogen has been used successfully and has reduced the local recurrence rate. However, it is difficult to use and is associated with complications such as skin necrosis, late fracture and neurovascular damage[11,12,13]. In light of these complications and our experiments, zinc chloride is better suited for eradication of GCT.

During our study, in some cases tumor penetrated the cortex and protruded into the soft tissue. After curettage a defect may be seen in the bony wall which increases the
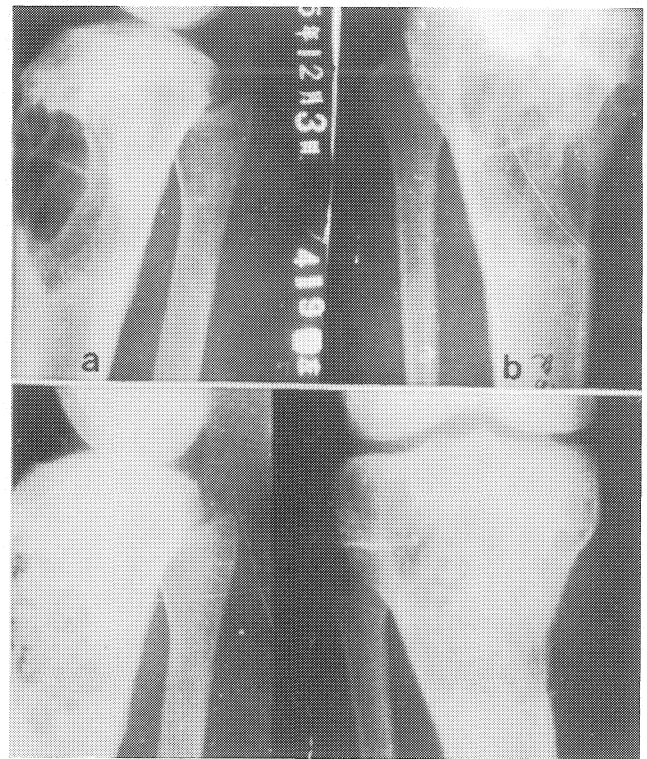

Fig. E, F
Excellent function of the knee at 6 year follow up.

Fig. 2A-F
Li Bao-qing, a 23 year old male with a giant cell tumor of the proximal left tibia.

risk of cryotherapy, whereas zinc chloride can safely be used. The operation should be performed by an experienced surgeon should a satisfactory result be expected.

Articular cartilage usually seems to be a natural barrier against tumor invasion into the joint. All except two cases were without joint involvement despite the fact that in many cases the bone was almost entirely destroyed except for the transparent cartilage. This should be saved because it is preserving joint function after the bone graft has healed.

Jaffe's histologic classification has proved to be of limited clinical value. Benign appearing giant cell tumors (Grade 1) may display locally aggressive behavior and may even metastasize. The frankly malignant giant cell tumors (Grade 3) do behave like malignant sarcomas without correlation to clinical or radiographic findings. At times it may be difficult to differentiate it from fibrosarcoma of bone. Based on our limited experience, less emphasis should be placed on Jaffe's classification and it is more advisable to use two categories, benign and malignant.

CONCLUSIONS

Complete removal is important to prevent recurrence of giant cell tumors. From these results curettage and cauterization with zinc chloride followed by bone grafting offered an acceptable method of treatment with a low incidence of recurrence in those giant cell tumors that were adjacent to major joints. En-bloc resection for GCT was successful in those lesions that were located in non-weight bearing bones.

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MANAGEMENT OF ACETABULAR DYSPLASIA

L.T. Staheli, M.D.
Department of Orthopaedics
Children's Hospital and Medical Center
4800 Sand Point Way, N.E.
P. O. Box C5371
Seattle, Washington 98105

INTRODUCTION

The human hip joint is inherently defective. Our upright posture creates an anterior deficiency reducing the load bearing area. Our increasing longevity exceeds the design limits of the hip joint and the vascular vulnerability of the upper femur further complicates the problem.

Nearly 100 years ago Konig performed the first acetabular reconstruction with a "Shelf" operation. This was followed by other capsular arthroplasties such as the Colonna and Chiari procedures. Later, redirection and pericapsular osteotomies were added to the options and more recently new imaging techniques have allowed precise characterization of the dysplastic acetabulum. Today the surgeon can chose from nearly a score of procedures to reconstruct the dysplastic acetabulum.

Hip dysplasia remains as one of the foremost challenges for the orthopaedic surgeon as the choices are many and the marginal hip joint is unforgiving of errors in management.

ETIOLOGY

Acetabular dysplasia may be divided into primary and secondary types. Primary dysplasia is of genetic origin. Family and racial factors produce predisposition to degenerative arthritis by variability in configuration and metabolism. Secondary acetabular dysplasias are due to a variety of problems:

1. *Congenital Hip Dysplasia* — The most common cause of acetabular dysplasia is congenital dysplasia of the hip. This includes a spectrum of disorders from a simple shallow acetabulum to frank dislocation. The causes of CDH are multifactorial, but commonly associated with ligamentous laxity and abnormal intrauterine position.
2. *Neuromuscular* — Muscle imbalance about the hip produces chronic adducted positioning with secondary subluxation and acetabular dysplasia.
3. *Vascular* — Necrosis of the head of the femur is a complication of CDH management or Perthes disease and produces deformity of the upper femur which is mirrored by the deformity of the acetabulum.

CLINICAL FINDINGS

Acetabular dysplasia is silent during the first decade. Occasionally mild discomfort or delayed Trendelenburg test are found. The diagnosis and severity can only be made by radiographic studies.

RADIOGRAPHY

Conventional radiographs provide the basic assessment and show coverage and congruity. In childhood the most valuable study is the "acetabular index". The angle should decrease with advancing age. As the joint becomes more ossified the "center edge angle" becomes most useful. In both of these studies normative data with standard deviations are available. The mean for the CE angle is 35 degrees with a range from 25-45 degrees. Special additional studies may be needed. The abduction internal rotation view (AIR view) shows congruity anticipated after repositioning procedures. Other studies may be necessary: 1) CT scans for complex deformity, 2) MRI for avascular necrosis, and 3) arthrography for the immature hip.

DIFFERENTIAL DIAGNOSIS — PATHOLOGIC ANATOMY

The pathology of the dysplastic acetabulum can be characterized by several features.

1. Maldirection: In the frontal plane the normal acetabulum is oriented laterally about 15 degrees. Often the dysplastic acetabulum is directed more anterolateral than normal.
2. Insufficiency: Coverage describes the extent of the weight-bearing area and is best assessed by the "CE angle" measurement.
3. Configuration: The acetabular shape is either spherical or aspherical. Furthermore, it can be either congruous or incongruous. Incongruity leads to uneven weight distribution and cartilage deterioration.

MANAGEMENT

The treatment of acetabular dysplasia is age dependent. In infancy and early childhood, the objective is to achieve an early concentric reduction without inducing avascular necrosis. Following reduction, the use of casting, bracing and splints to position the femur in abduction and flexion may promote improvement of acetabular competence.

The indication for operative correction of acetabular dysplasia in early childhood is a failure of satisfactory acetabular development. The rate of development should be documented over several years. The hip is reduced at the earliest possible time and the acetabular development is assessed periodically. If the acetabular index fails to improve, redirectional or pericapsular osteotomy is indicated.

Better results from acetabular procedures are achieved if the operations are performed in early rather than late childhood. The timing remains controversial and some elect to wait while others feel that early intervention gives the best long-term prognosis.

If unresolving dysplasia exists, what procedure is best? This selection is a complicated one and should be guided by a number of principles.

1. Correct the primary deformity when possible. If the defect is primarily acetabular, it is better to approach the pelvis rather than creating a compensating deformity in the proximal femur.
2. The correction should be adequate. Partial correction may be of help, but adequate correction to bring the acetabulum to within the normal range is important for function and durability.
3. Conserve hyaline cartilage by reposition or redirectional procedures when possible.
4. Maintain or improve joint congruity.
5. Consider the patient's age. Remodeling may improve congruity in the young child, but may be inadequate in late childhood and adolescence. Prophylactic correction of acetabular dysplasia is often indicated in the immature patient. In late adolescence it may be prudent to wait until symptoms develop.
6. Consider the patient's psychosocial and vocational needs. Individual needs may vary and this may influence the correction technique.
7. Consider future procedures. It is unlikely that any acetabular reconstruction will be permanent and avoid creating additional deformity, leaving residual hardware or inadequate bone stock may jeopardize later procedures.
8. Consider the risk factors. The skill and experience of the surgeon as well as the inherent risk of the procedure should be considered.

OPERATIVE CHOICES

OPERATIONS TO REPOSITION HYALINE CARTILAGE

Redirectional osteotomies are the most useful of acetabular procedures. They provide hyaline coverage, have a wide range of application and do not alter the shape of the acetabulum. They do require spherical congruity and the double and triple innominate osteotomies are technically difficult.

1. *Single Innominate Osteotomy.* The Salter procedure is probably the most useful acetabular operation. The procedure is indicated for mild and moderate dysplasia in the child older than nine months. At best it provides about 25 degrees of anterior and 10-15 degrees of lateral coverage.
2. *Double Innominate Osteotomy.* This employs a second osteotomy to the pubis to improve correction in the older child or adolescent.
3. *Triple Innominate Osteotomy.* Is most useful in the second decade when greater correction than either the single of double innominate osteotomy can provide. This is technically difficult. Modifications include the Tonnis posterior and Tachdjian anterior approach to the pubic and ischial osteotomies.

PERICAPSULAR OSTEOTOMIES

These pericapsular osteotomies parallel the joint surface and allow repositioning and reshaping of the acetabulum.

1. *Marginal "Lip" Osteotomy (Lance Procedure).* An acetabular lip osteotomy extends a variable distance over the acetabulum.
2. *The Pemberton Osteotomy.* The procedure extends to the triradiate cartilage. It changes the acetabular configuration by reducing the radius of curvature. It is appropriate in the younger child where remodeling may overcome incongruity. It is ideal for patients with more severe dysplasia, particularly bilateral cases, as both sides can be corrected concurrently. It is also useful in the neurogenic dislocations in cerebral palsy and myelodysplasia as it reduces the radius of curvature of the enlarged acetabulum.
3. *Extended Pericapsular Osteotomy (Weston Procedure) and Complete Pericapsular Osteotomy (Eppright Procedure).* These procedures are less commonly employed.

CAPSULAR ARTHROPLASTY

1. *Acetabular Augmentation (Shelf).* This procedure is a capsular arthroplasty utilizing a graft to enlarge the volume of the acetabulum. It is the oldest acetabular procedure. Recently its role has been better defined. A modern refinement is the "slotted augmentation". Cancellous bone is extended laterally from the aceta-

bular slot to provide a congruous extension. The procedure allows great flexibility, improves the bone stock, is relatively safe, and requires no metal fixation.

2. *Total Acetabular Capsular Arthroplasty (Colonna).* This procedure is seldom performed.

3. *Displacement Osteotomy (Chiari).* This procedure medializes the lateralized acetabulum to reduce joint loading. It is often supplemented by a lateral augmentation to improve congruity, and to avoid the need for excessive displacement to provide adequate coverage.

CLINICAL APPLICATIONS

1. *CDH.* Nearly all the procedures described have some role in this disorder. The Pemberton and Salter procedures are most commonly used during infancy and early childhood, whereas the Steel, Chiari and Shelf procedure are most useful during the second decade.

2. *Myelodysplasia and cerebral palsy.* The Pemberton procedure is useful in early childhood whereas the Chiari and Shelf operations are used mostly in late childhood and in the second decade. As the Salter operation uncovers the acetabulum posteriorly this procedure is contraindicated in neuromuscular disorders.

3. *Perthes Disease.* As this deformity primarily involves the proximal femur, operative correction is achieved best by a varus osteotomy. The Salter osteotomy is an alternative procedure and may be combined with the femoral osteotomy to provide extensive containment.

SUMMARY

A dysplastic acetabulum is likely to result in degenerative arthritis. Correction often requires an operation, which may be prophylactic, in childhood. The selection of the procedure should be based on many factors, but primarily on the acetabular pathology. Careful characterization of the acetabulum, thoughtful selection of the procedure and skillful performance of the operation are necessary for a satisfactory outcome.

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MANAGEMENT OF DEFORMITIES SECONDARY TO BONE DYSPLASIA

Stuart L. Weinstein, M.D.
Professor
Department of Orthopaedic Surgery
University of Iowa
Iowa City, Iowa 52242

In evaluating a patient with skeletal dysplasia the orthopaedic surgeon must keep several general concepts in mind. Children with a skeletal dysplasia have disproportionate growth of various portions of the skeleton. These disproportions may change during growth, i.g. in spondyloepiphyseal dysplasia congenita the trunk grows at a relatively slower rate than the limbs during the first decade[23]. The portion of the skeleton primarily affected tends to have the greatest number of problems. Thus short trunked dwarfs tend to have more problems with spinal deformity and atlantoaxial instability while short limbed dwarfs tend to have more problems with joint malalignment, joint contractures, etc. The more severely involved the individual is, the greater the likelihood of having associated problems. In addition, once the patient becomes ambulatory secondary deformities may be superimposed as the effects of weight bearing are exerted on the dysplastic bones and joints.

It would be a monumental task to discuss the management of all of the musculoskeletal dysplasias. Therefore we will attempt to discuss the management of the musculoskeletal problems of the most common skeletal dysplasias[23]. The basic principles used in the management of these conditions can then be applied to other skeletal dysplasias.

Achondroplasia

Achondroplasia is the most common form of dwarfism[2,16,20,23,34,36,40,43] and is the prototype of the short limbed dwarf. It is an autosomal dominant disorder but 80 percent of the cases represent spontaneous mutations[40]. These children are easily recognizable at birth. Their characteristic features include an enlarged head with frontal bossing, mild hypoplasia of the mid face, prominent mandible, broad short hands with a space between the third and fourth digits (trident hand), rhizomelic shortening of the extremities with ligamentous laxity and slight hypotonia[23,40]. These patients commonly have delays in reaching milestones secondary to their disporportionate enlarged head and trunk relative to their extremities and relative hypotonia.

Roentgenographic features of the cranium include overall cranial enlargement with shortening of the base of the skull while the foramen magnum is small and the frontal area is prominent. There may be occipitalization of the atlas. The spine shows characteristic decrease in the interpedicular distances on the AP view proceeding cephalad to caudal. On the lateral view, the pedicles are broad and thick. The vertebrae are often bullet shaped. The AP diameter of the body is small with scalloping of the posterior body margins (secondary to dural compression). The pelvis has short square ilia, the sciatic notches are narrow and deep and the acetabulum is horizontal. The long bones are short and broad with wide flaring of the metaphyses. The growth plate is like an inverted V and the fibula is longer than the tibia. The radial head may be dislocated.

Most achondroplasts will develop a thoracolumbar kyphosis in the T10 to L4 region (Fig. 1). These curves have been reported to reach 70 degrees[23,40]. These curves are usually supple and 90 percent will regress when independent gait is achieved[23]. However, some achondroplasts will have anterior vertebral body wedging or body recession leading to kyphosis. These kyphotic curves may progress and lead to anterior spinal cord compression requiring anterior decompression in addition to anterior and posterior fusion. If the deformity is less than 60 degrees and if the patient has no neurologic deficit, the kyphosis should be treated with a Milwaukee brace with kyphosis pads. Should the deformity reach greater than 60 degrees, consideration should be given to anterior and posterior spine fusion[5,14,23].
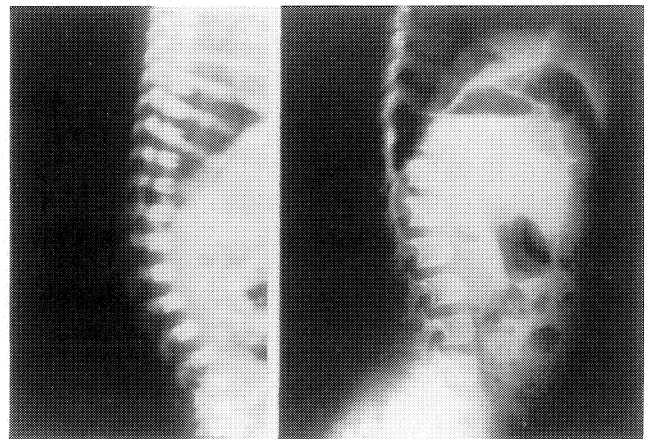

Fig. 1
Achondroplasia. (A) Newborn with thoracolumbar kyphosis. (B) By age twenty-three months the kyphosis is resolving.

Scoliosis is uncommon in achondroplasts and if present is usually insignificant[5,23]. Odontoid hypoplasia is also not a problem in these patients.

Genu varum is a common problem in achondroplasia[23]. Ponseti feels that the genu varum is secondary to fibular overgrowth[36]. The bowing primarily affects the proximal leg but occasionally may be evident distally. Patients may complain of pain where the fibula comes in contact with the calcaneus if there is heel valgus to accommodate the genu varum. If the deformity is distal, this should be treated by a supramalleolar osteotomy. Ligamentous laxity contributes to the deformity although this tends to decrease with age. Bracing will not correct this deformity and is not recommended. These patients' extremities are extremely difficult to fit with an orthosis and the closeness of the joints make them cumbersome. In addition, the brace acts through lax ligaments rather than bone[14,23]. We have treated the genu varum deformity by proximal fibular epiphyseodesis in the preadolescent patients with mild deformity. In older children with knee discomfort, resection of the prominent fibular head and, in the more severe deformities, opening wedge spike osteotomies have been done to correct the malalignment (Fig. 2)[36]. The deformities tend not to recur. Flexion contractures are present about the hip and elbow and are usually well tolerated.
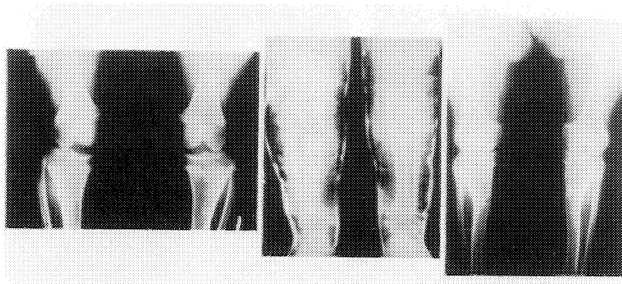

Fig. 2
Achondroplasia. Left, preoperative roentgenogram demonstrating genu varum and fibular overgrowth. Center, roentgenogram immediate postoperative opening wedge spike osteotomies and proximal fibular head resections. Right, roentgenogram one year postoperatively.

The most significant problem facing the achondroplast is spinal stenosis. Approximately 40 percent of adult patients are affected; however, the number is significantly greater if one takes into account those with mild symptoms[5]. The etiology is the narrowed canal in the frontal and sagittal planes (Fig. 3). Other contributing factors may include increasing lumbar lordosis, herniated nucleus pulposis, bulging annulae, hypertrophied facet joints and osteophytes[1,5,23,28]. The average age of symptom onset is thirty-eight years[1,28]. The patients may present with either signs or symptoms of nerve root compression, cauda equina syndrome, paralysis or claudication[28].
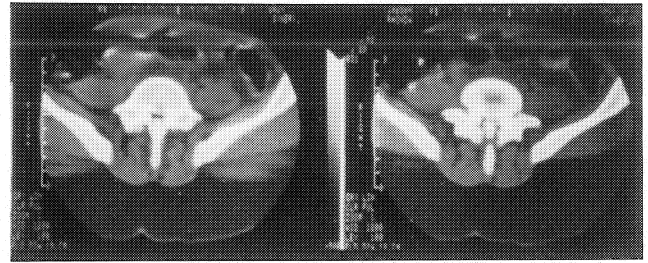

Fig. 3
Achondroplasia. CAT scan of a twenty-three year old with symptoms of spinal stenosis. Note the narrowed canal in both AP and transverse planes.

Lumbar myelography may be difficult to perform and even hazardous because of the small canal. Typically it will show pooling of dye at the body levels and filling defects at the disc levels and possibly a block at L3.

The treatment is adequate laminectomy. This usually encompasses five to ten levels and may require partial pediculectomy, facetectomy and formaninotomy. An inadequate decompression will often lead to increasing symptoms necessitating extension of the laminectomy in length and width. The facet joints are very close to midline in achondroplasts and thus postlaminectomy instability is usually not a problem. Therefore fusion is usually only needed if the facet joint congruity is disrupted[5,23,28].

Pseudoachondroplasia Dysplasia

Pseudoachondroplasia dysplasia was first described by Maroteaux and Lamy in 1959[29]. Subsequently four forms have been delineated[5,17] with both autosomal dominant and recessive inheritance and varying severity of musculoskeletal problems. It represents the second most common type of short limbed dwarf after achondroplasia. The disorder can be differentiated from achondroplasia because the patients have a normal sized and shaped head, a greater degree of ligamentous laxity, particularly about the wrists, ankles, and knees, and severe lower extremity deformities (Fig. 4). The disorder is not detectable at birth, the rhizomelic shortening becoming evident at age two to three. They have a waddling gait and excessive lumbar lordosis.

Roentgenographically there is generalized involvement of both the epiphyses and metaphyses of the tubular bones. The epiphyses are delayed in their appearance and are small and fragmented when they do appear[8]. The central portions appear normal but the periphery is markedly deformed, especially at the sites of ligament insertions[9]. The metaphyses are splayed and frayed. The vertebrae show varying degrees of platyspondylia, being biconvex with anterior tonguing. The severity of these changes decreases with age. The pelvis demonstrates large ilia and short ischia and pubi.
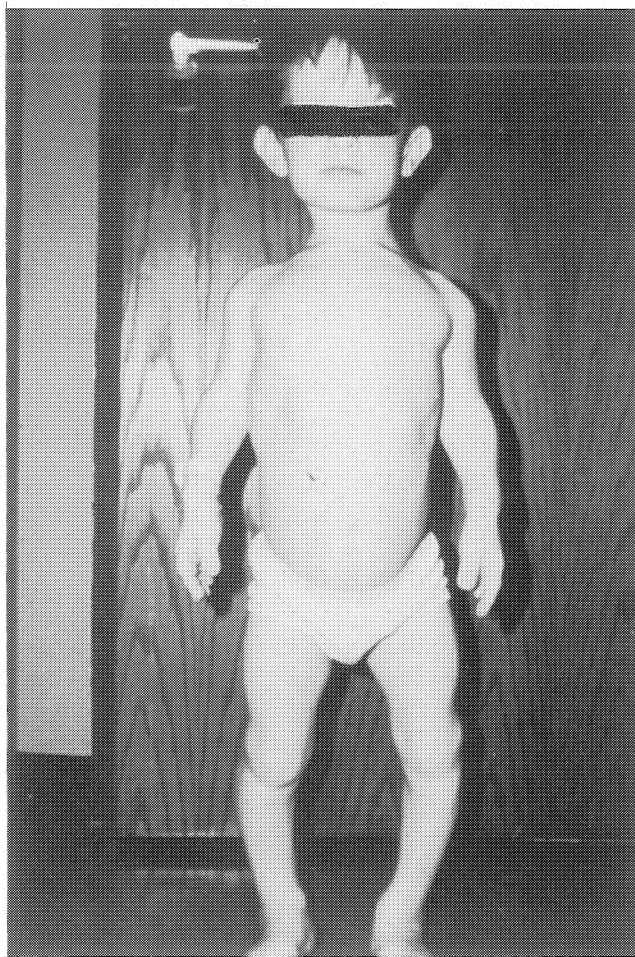
Fig. 4
Pseudoachondroplasia Dysplasia. Seven year old male. Note the rhizomelic shortening and genu varum. The head and face are normal.

Over 80 percent of these patients develop malalignment deformities of their lower limbs[23]. The most frequent picture is that of the windswept deformity with hip adduction and knee valgus on one side and hip abduction and knee varus on the other[23]. This may lead to hip subluxation and pelvic obliquity. Patients may also have bilateral genu varum and fibular overgrowth or bilateral genu valgum. Genu recurvatum may also accompany these deformities. Bracing treatment is ineffectual for the same reasons mentioned in achondroplasts. Wrist instability caused by ulnar deviation secondary to bowing of the radius and ulna in combination with ligamentous laxity may be a functional problem that can be managed by splints.

Surgical correction of the deformity is aimed at correcting AP and sagittal plane deformity so as to achieve a level pelvis with the ankle and knee joints parallel to the ground. At surgery, one must take into account the extreme degree of ligamentous laxity that contributes to the deformity. In addition, the delayed and incomplete ossifi-cation of the epiphyses may require the surgeon to use arthrography at surgery to insure proper joint alignment. These deformities have a tendency to recur; therefore slight overcorrection with the opening wedge osteotomies should be obtained at surgery to prevent recurrence (Fig. 5)[2]. Patients with pseudoachondroplasia dysplasia have a tendency to develop degenerative joint disease[2,9,43] which may require joint replacements.
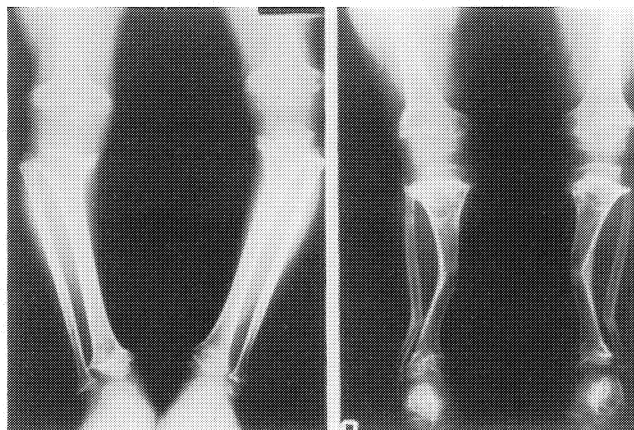

Fig. 5
Pseudoachondroplasia Dysplasia. Left, preoperative roentgen-ogram demonstrating genu varum. Note also the splaying and fraying of the metaphyseal regions. Right, roentgenogram one year postoperative bilateral opening wedge tibial and fibular osteotomies.

Scoliosis may develop but it is usually not severe and if progressive should be treated by standard Milwaukee brace therapy. Correction of the infra pelvic causes of pelvic obliquity is also helpful. If progression continues, surgical stabilization by standard techniques is recommended[5].

Hypoplasia of the odontoid is the most common dysplasia of the odontoid seen in patients with pseudoachodnro-plasia if they have significant spine involvement. The tip of the odontoid is cone shaped and does not extend cephalad to the ring of C1. Mild atlantoaxial instability may occur in these patients and it is uncommon for them to develop cervical myelopathy, however, it has been reported[23].

Metaphyseal Chondrodysplasia

Metaphyseal chondrodysplasia refers to a group of disorders of defective enchondral ossification with major roentgenographic manifestations in the metaphyseal region[26]. This disorder encompasses the disorders formerly referred to as metaphyseal dysostosis of the Jansen (autosomal dominant)[19] or Schmid type (autosomal dominant)[11,39], McKusick type — Cartilage-hair hypoplasia (autosomal recessive)[31], and metaphyseal chondrodysplasia with pancreatic insufficiency and neutropenia (autosomal recessive)[6,8,13,45]. These disorders all have a common

bowing of the long bones with the lower extremities being affected to a greater extent than the upper extremities. The bowing is slowly progressive and will lead to malalignment in over 50 percent of the cases[23]. Cooper, et al., reported dilated cisternae of chondrocyte endoplasmic reticulum filled with a granular precipitate. They hypothesized that this represented a defect in enzyme transport mechanism inhibiting intracellular transport of protein polysaccharide or a similar material from the rough endoplasmic reticulum to the golgi apparatus[10].

Metaphyseal chondrodysplasia of the Schmid type is the most common. These children are normal appearing at birth. In the toddler years, short stature becomes evident as does bowing of the legs, a waddling gait and excess lumbar lordosis. The head size is normal as are all blood chemistries. Metaphyseal chondroplasia of the Jansen type is evident in infancy. The head may be slightly enlarged.

In these dysplasias, roentgenographic changes are confined to metaphysis and growth plate regions (Fig. 6). The metaphysis is widened, irregular and stippled, while the physis is widened and irregular[10]. All long and tubular bones are affected with the most severe changes occurring at the most rapidly growing bones. Normal bone curvatures are accentuated. The Jansen type may show more extreme changes with abnormal zones of ossification extending down into the diaphysis. The skull may show evidence of increased sclerosis with prominent supraorbital ridges and small paranasal sinuses[10]. These patients, particularly the McKusick type, may also have problems with varus foot deformity secondary to distal fibular overgrowth.

Coxa vara represents the most common problems in these disorders. When the neck shaft angle is less than 100 degrees surgical correction by intertrochanteric valgus osteotomy is indicated (Fig. 7). Postoperatively this deformity tends not to recur and patients walk with less effort and report increased work tolerance[2,23].

Genu varum is another problem in many of these patients. Bailey reports some success in treatment of genu varum with a Blount brace[2]. This has not been our experience.

Fig. 6
Metaphyseal Chondrodysplasia. Four year old with Schmid type metaphyseal chondrodysplasia. Coxa vara is present. The physes are widened and irregular.
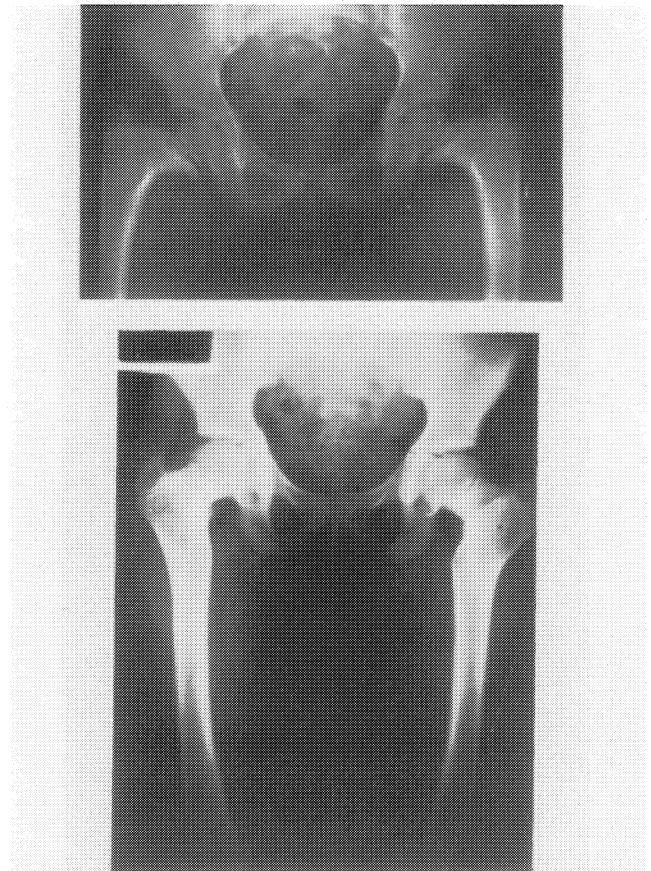

Fig. 7
Metaphyseal Chondrodysplasia. Top, preoperative roentgenograms of a four year old female with Schmid type metaphyseal chondroplasia demonstrating severe coxa vara. Bottom, same patient two years after bilateral valgus intertrochanteric osteotomies.

When the genu varum is significant enough to cause malalignment of the knee and ankle joints or cause pain, it should be corrected with the appropriate osteotomy. Scoliosis and odontoid dysplasia are not usual but have been reported in metaphyseal chondrodysplasia[2,5].

In a variant of this disorder, spondylometaphyseal chondrodysplasia (autosomal dominant), the proximal femoral metaphysis is severely affected and the spine shows evidence of platyspondylia with anterior beaking[25,26]. These patients may develop scoliosis and thoracolumbar junction kyphyosis[5,26]. Kyphosis at the thoracolumbar junction should be managed in the Milaukee brace with kyphosis pads. Progression in the brace requires surgical fusion to prevent the neurologic sequalae of progressive kyphosis. If the kyphosis is greater than 60 degrees staged anterior and posterior fusion is recommended[5]. Progressive scoliosis should be managed in a Milwaukee brace regardless of age. However, when progression exceeds 45 to 50 degrees, surgical stabilization is recommended[5].

Diastrophic Dwarfism

Diastrophic dwarfism is an autosomal recessive disorder described in 1961 by Lamy and Maroteaux[27]. The word diastrophism is derived from the Greek word for twisted or tortuous. The patients with this disorder are recognizable at birth by an abducted hypermobile proximally placed thumb (hitchiker's thumb). Other deformities include short broad hands with ankylosis of the PIP joints, clinodactyly of the little finger, absence of flexion creases, shortness of stature with micromelia (proximal segments affected most) and severe bilateral clubfoot deformities[18,13,46]. During the neonatal period these patients have acute swelling in the pinnae of the ear which eventually go on to calcification or ossification.

The patients develop multiple complex problems with joint contractures, subluxation and dislocations, degenerative joint disease and spinal deformity. The mortality rate of this disorder is fairly high with Bailey reporting eight deaths in ten affected children in three families[2]. Roentgenographically there is involvement of virtually all articular and epiphyseal cartilage. The metaphyses are flared and the epiphyses are late appearing and flattened.

The clubfoot deformity is difficult to treat. Serial casting should begin soon after birth. Once the maximum benefits of casting have been achieved, any residual deformity should be corrected by soft tissue surgery and prolonged casting. The equinus component is difficult to correct and maintain, particularly in those patients with hip and knee contractures and probably fail unless the hip and knee problems are corrected. If a recurrence occurs, or if the patient is first seen in the toddler years, it may be best to observe the patient and accommodate the deformities in appropriate shoes. This is in light of the fact that some patients may function adequately despite incomplete correction. When the patient is into the second decade and is hampered by foot deformity, bony surgery such as triple arthrodesis, foot narrowing procedures, and supramalleolar osteotomies may be considered[2].

Progressive hip dysplasia is seen in 80 percent of the patients[18]. The hips are generally normal at birth, but may progressively subluxate and even dislocate. The acetabular roof is slanted laterally (Fig. 8). Kopits has demonstrated the femoral head to be flattened and bilobulated and the femoral neck to be short[23]. The patients may develop symptoms of degenerative joint disease prior to the full ossification of the femoral head. Valgus intertrochanteric osteotomies alone or in combination with an acetabuloplasty may prevent or delay degenerative joint disease[23]. Once the joint develops degenerative changes total hip replacement is the only alternative. Total joint replacement in these patients may require the use of specially designed femoral components as well as acetabular augmentation procedures similar to those techniques used in joint replacements for congenital hip dysplasia.

Fig. 8

Distrophic Dwarfism. Natural history of hip dysplasia in a single patient is demonstrated from roentgenograms in infancy and early childhood on the left through the development of degenerative joint disease by age twenty-eight years in the far right. Note the stunting of lateral acetabular growth.

The joint contractures (hip, knee, and elbow flexion contractures), may be very severe. They are secondary to cartilaginous joint deformity as well as soft tissue contracture. Knee flexion deformity is probably secondary to the hip deformity and is accompanied by misshappened joint surfaces[23]. Release of soft tissue contractures about the hip is associated with recurrence and increased joint stiffness[2]. Should correction be indicated, extension osteotomy may offer the best solution.

Spinal deformity developed in over 80 percent of the patients[18]. In one-half of the patients this may be limited to excessive lumbar lordosis, which may be a compensatory mechanism adjusting for hip flexion deformity[2,4,47].

The onset of scoliosis occurs during the first five years of life[4,5,17,18]. These curves are progressive, become rigid rapidly and will reach considerable magnitudes causing cardiopulmonary compromise if left untreated[4,5,17]. The double curve pattern is most common. In early childhood the curves are small and flexible and should be managed by a Milwaukee brace to arrest or at least limit progression. If progression occurs despite bracing, early posterior fusion is indicated. Delaying the procedure only serves to increase the magnitude and rigidity of the curve[4,5,17]. For some young patients with progression a Moe subcutaneous rodding and bracing without fusion may be an alternative[4].

Cervical kyphosis may be a significant problem in patients with diastrophic dwarfism and is not reported in other forms of dwarfism[4,5,17,23,46]. In its extreme form it may lead to quadriplegia and death[5]. It is usually associated with hypoplastic cervical vertebrae. Three-fourths of the cases of cervical kyphosis will resolve without treatment[4,5]. If the deformity is progressive, threatening neural function, surgical intervention is indicated. Since the majority of these patients have cervical spina bifida occulta, posterior fusion alone may prove difficult and consideration should be given to anterior strut grafting[4,5,17].

In the lumbar spine the pedicles are shortened, particularly in the lower levels as in achondroplasia. The interpedicular distance may or may not be decreased[4,47]. These patients may develop symptoms of spinal stenosis in their fourth and fifth decades and should they develop, decompression via laminectomy is the treatment of choice[23].

Genu valgum, flexion contracture and dislocating patellae are problems that occur about the knee in diastrophic dwarfs and may contribute to the loss of ambulation in the second decade[24]. These may require soft tissue release of contracture and/or osteotomy and extensor mechanism realignment.

The hand deformities rarely require treatment. Interphalangeal joint spaces usually synostose with time. The shortened, ovoid or triangular first metacarpal contributing to the "hitchiker" deformity may cause some difficulty with pinch, but can be compensated for by using the ulnar

side of the IP joint. Rotational osteotomy of the proximal phalanx may be of some benefit[23].

Morquio's Syndrome

Morquio's Syndrome (mucopolysaccharidoses IV) is an autosomal recessive disorder that was the prototype of the short trunk dwarf. It was described in 1929 by Morquio and Brailsford[7,3] and is the most common of the mucopolysaccharidoses. In addition to musculoskeletal abnormalities these patients may have aortic insufficiency, corneal opacities and keratosulfaturia[21,30,35,37,48]. Older patients may not secrete keratan sulfate in the urine and McKusick has described a nonkeratan sulfate excreting variety[32]. The patients often have a broad maxilla, widely spaced teeth, cuspids slightly more prominent than usual and a thin enamal cap[21,37]. All cartilage is involved with the disorder. Ligament, tendon and capsular insertion sites seem to be particularly involved with delayed and faulty ossification[30,35,37]. The disorder is not diagnosed at birth (fetal cartilage has no keratan sulfate) but skeletal lesions may be seen by eight months of age[37].

By two years of age short stature and a waddling gait become apparent. Pectus carinatum develops secondary to disproportionate growth between the spine and the sternum[23]. The patients quickly develop hip flexion contractures, tightness in the tensor fascia latae and genu valgum. With age these deformities and resultant disabilities increase.

The bases of the metacarpals are conically shaped and carpal bone ossification is retarded. The ulna is shorter than the radius with its distal end being radially inclined. The ulnar side of the distal radial metaphysis has a particularly jagged appearance. Ligamentous laxity about the wrist may allow dorsal-volar displacement and distraction of up to 2.5 cm[23]. This combination leads to decrease grip and pinch strength and disability. Wrist stabilization by fusion would seem the logical choice, however, attempts at fusion have been unsuccessful[23]. Thus, wrist splints offer the best alternative.

The pelvis in Morquio's Syndrome is usually narrow. The acetabular roof has a defect in ossification of the lateral margin (Fig. 9). The femoral heads are small, unevenly ossified in young patients and gradually become flattened and fragmented. The hips may go on to subluxate or dislocate but usually remain asymptomatic and require no treatment[23]. The greater trochanters are poorly ossified and all patients have some degree of coxa valga[37].

Genu valgum may reach disabling proportions in patients with Morquio's Syndrome by eight to ten years of age. The proximal lateral tibial epiphysis growth is stunted (Fig. 10). This in combination with severe ligamentous laxity, contracted tensor fascia lata and iliotibial band leads to increasing deformity. In time fragmentation may occur in
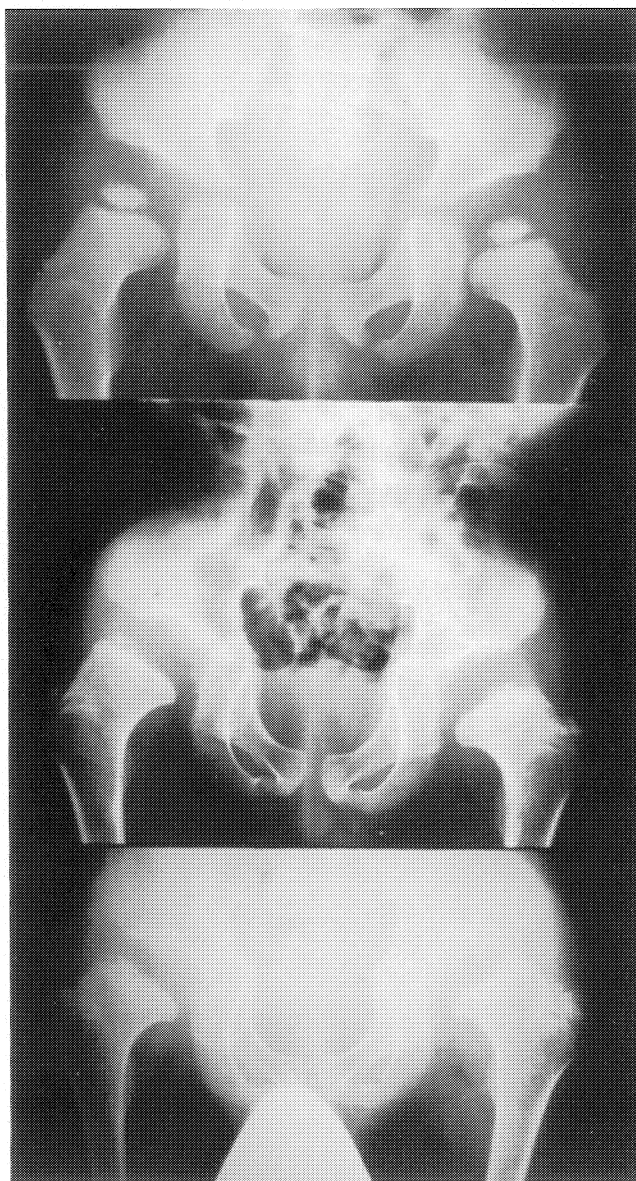
Fig. 9
Morquio's Syndrome. Roentgenogram of the pelvis in a patient with Morquio's Syndrome. Upper, roentgenogram age five. Middle, roentgenogram age ten. Lower, roentgenogram age fourteen. Note the narrow pelvis, the small unevenly ossified femoral heads that become flattened and fragmented. The greater trochanters are poorly ossified.
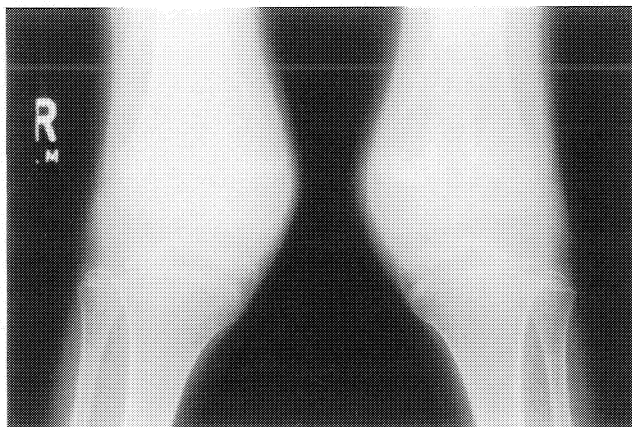

Fig. 10
Morquio's Syndrome. Fourteen year old male with Morquio's Syndrome and severe genu valgum with stunting of the growth of the lateral portion of the proximal tibial epiphyses.

the lateral femoral condyle as well as the proximal lateral tibial plateau. Releasing the tensor fascia lata contracture early to prevent the deformity has been tried without success[37]. Kopits reports good results without recurrence using realignment osteotomies performed at eight to ten years of age. The reason for lack of recurrence is that the patient usually stops growing about this time[23]. Correction may also take into account the extreme degree of ligamentous laxity present.

The spine in Morquio's Syndrome is severely affected with platyspondyly consistently present. In young patients the vertebral height is greater centrally than peripherally. On the lateral view, the typical flame-shaped contours are seen with failure of ossification of the anterior superior and inferior vertebral margins[37]. Some vertebrae are smaller than adjacent vertebrae and are recessed usually at the thoracolumbar junction, thus narrowing the spinal canal. Scoliosis and kyphosis may ensue, but only to a mild degree[23]. In the thorax, the sagittal diameter is increased due to horizontal ribs and its mobility is severely restricted[21,23]. In the late second decade the spine will become rigid.

The most significant problem in Morquio's Syndrome and the one most often recognized late is cervical myelopathy secondary to atlantoaxial instability. The instability is secondary to odontoid hypoplasia or aplasia and ligamentous laxity. More than 70 percent of patients with odontoid dysplasia have C1-C2 instability and 50 percent of these will develop cervical myelopathy[2,14,23].

Symptoms of myelopathy are usually present between the ages of six and ten years[14,23]. The first signs are usually decreased physical endurance despite a normal neurologic exam. These symptoms are often erroneously attributed to the lower limb deformities. Patients begin to do more sedentary activities. Gradually pyramidal tract signs develop (hyperactive reflexes, clonus, positive Babinski, spasticity) generally involving one side of the body with the lower limbs primarily affected[14,23]. The patients may complain of paresthesias in the legs but sensation to pain and touch remains intact. Vibratory sensation decreases as the myelopathy progresses. The pectus carinatum may protect the patient from hyperflexion but sudden death has been reported[23]. The etiology of the myelopathy is cord compression and lateral displacement

(accounting for the unilateral symptoms in 90 percent of the patients) by a hypertrophic mass composed of posterior longitudinal and transverse ligaments[14,22,23]. These ligaments hypertrophy as a result of abnormal motion and produce a large mass of tissue situated posterior to the dysplastic odontoid.

All patients with Morquio's Syndrome (as well as all those with skeletal dysplasia) should have upright lateral flexion extension cervical spine films to evaluate instability. The presence of odontoid aplasia or hypoplasia does not necessarily indicate atlantoaxial instability. When myelopathy is suspected it can be further evaluated by magnetic resonance imaging, metrizamide myelography with polytomography or CT scan. The instability is reduced by halo traction and the reduction maintained by posterior fusion. If there is no mobility at the occiput C1 junction, the occiput may be included in the fusion. Postoperatively the patients are maintained in a halo cast or jacket until the fusion is solid, usually three to four months. Atlantoaxial instability with myelopathy should be treated prior to any extremity realignment procedures, because of the dangers of anesthetic intubation. Failure to treat this problem secondary to lack of recognition may account for the inability of patients to regain ambulation after lower extremity realignment procedures[14,22].

Spondyloepiphyseal Dysplasia Congenita

Spondyloepiphyseal dysplasia (SED) congenita is an autosomal dominant disorder in which the patient presents as a short trunked dwarf without visceral features (as distinguished from Morquio's Syndrome)[41,42,44]. The diagnosis is possible, although difficult at birth, for initially the limbs are disproportionately short. However, in the preschool years the growth of the trunk lags behind the extremities. These patients may have mild frontal bossing, a long flat face with wide set eyes, normal sized hands and feet, pectus carinatum, and occasionally clubfeet[23,43]. They may exhibit hyptonia and walking may be delayed.

On standing, patients with spondyloepiphyseal dysplasia congenita exhibit an excessive lumbar lordosis and a protuberant abdomen. In gait they may walk with their heads hyperextended to compensate for the pelvis being set posterior to the plane of the shoulders. Their hips are held in a flexed, abducted and externally rotated posture[23,43,44].

Roentgenographically the disorder is characterized by a marked delay in the appearance of the epiphyseal ossification centers of long bones, especially the femoral heads and also a delay in ossification of the iliopubic ramus. The vertebral bodies have a biconvex appearance on the lateral view. In the lumbar area the vertebral height is greater anteriorly, the pedicles are very long and the posterior elements are diminished in height. This in combination with the hip flexion contracture may cause the excess

lumbar lordosis[23]. At the thoracolumbar junction the vertebrae may be stunted anteriorly leading to kyphosis.

Coxa vara usually develops leading to a waddling gait and if left untreated may lead to hip subluxation. To avoid these complications, as well as, degenerative joint disease a valgus intertrochanteric osteotomy is recommended. A psoas lengthening at the time of surgery may help to improve the patient's lumbar lordosis.

Scoliosis and kyphosis may occur in patients with spondyloepiphyseal dysplasia congenita. These deformities should be treated with standard Milwaukee bracing techniques. Should the curve be progressive despite nonoperative methods, surgical stabilization is indicated.

Aplasia of the odontoid is another common finding in SED congenita (Fig. 11). One-third of the patients will develop cord compression. Cervical myelopathy is evident earlier in children with SED congenita than in those with Morquio's Syndrome. Symptoms may be evident in the newborn or neontal period. Cases of respiratory arrest with head flexion in the newborn period as well as quadriplegia secondary to minimal head trauma have been reported[23]. Cervical myelopathy secondary to atlantoaxial instability should be suspected in any child with SED congenita who is late in reaching psychomotor landmarks. Treatment consists of reduction with halo traction, posterior fusion and stabilization.

The management of skeletal deformities in patients with skeletal dysplasia is very demanding. The goals of treatment must be individualized for each patient. Awareness of the problems associated with skeletal dysplasias allows the orthopaedic surgeon to identify these problems early and institute prompt treatment and hopefully to ultimately alter the natural history of these problems and improve the patient's quality of life.
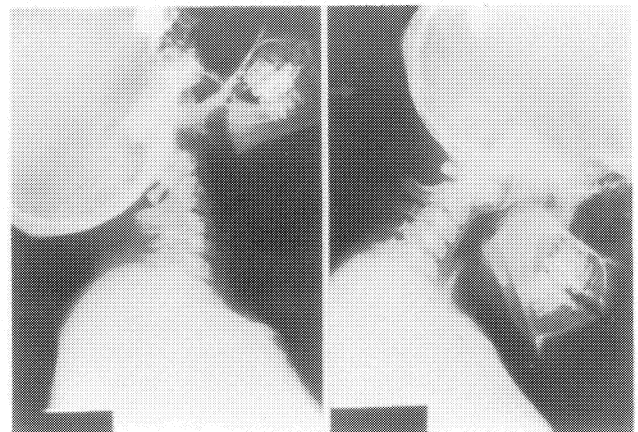

Fig. 11
Spondyloepiphyseal Dysplasia Congenita. Twenty-two year old patient with SED congenita demonstrating aplasia of the odontoid and atlantoaxial instability on lateral flexion and extension roentgenograms.

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AN ESSAY ON SIMPLE BONE CYSTS
THE MICHAEL BONFIGLIO LECTURE 1987

Jonathan Cohen, M.D.
Kennedy Memorial Hospital
30 Warren St.
Brighton, Mass.

I have chosen, for this essay, a well known lesion, the unicameral bone cyst as it occurs in children. It exemplifies, in its mystery as regards pathogenesis and etiology, a phenomenon that is fairly frequent in Orthopaedics, namely, a wealth of empirical information, from which no essential understanding is forthcoming. Several other lesions come to mind that illustrate the same paradox — Paget's disease, eosinophilic granuloma, osteoid osteoma, aneurysmal bone cyst, fibrous dysplasia, and metaphyseal fibrous defect. The phenomenon has stimulated many an orthopaedic surgeon in academia to study a particular lesion in the hope of clarifying the nature of a basic physiological process in bone, the idea being the detection, in what went wrong, of the reason for the deviation, or at least, that step in the normal sequence of a process where something failed to occur. A caricature lends us insight by exaggerating features that usually serve merely as elements of recognition. My choice of topic not only falls within the field of expertise of Dr. Bonfiglio, orthopaedic pathology, but also permits me to explore lines of investigation analogous to those he has pursued in his own studies, e.g. on avascular necrosis.

My studies on bone cysts began in 1946, when, as a resident in Pathology, I tried to make some sense out of the cases available at the Children's Hospital in Boston. In that institution, thanks to a meticulous saving of slides and x-rays, and record keeping that verged on an obsession, about fifty cases could be retrieved from the files. The search was aided in no small measure by the fact that the diagnostic terminology had remained constant for many decades. There were no other names to search for in the files, and, once a case came to light, only rarely would it be discarded as misdiagnosed or lacking in essential information.

The histology of the lesions, in the initial stage of analysis, required that there be separation of features attributable to fractures and their repair, from features to be considered the essence of the cyst. Nearly all the cases were characterized, or, more properly, came to light, when the pathologically thinned wall of the cyst underwent fracture, but often, in the operative material, the specimens represented lesions in which the fractures preceded the operative specimen-taking by weeks or months. The chronology of the repair processes therefore had to be analyzed apart from the cyst itself.

As was well known then, and for more than a century before, the lesion had as a sine-qua-non a thin membrane, a sac, that contained many milliliters of a fluid, which, in the absence of recent fracture, was straw-colored, and slightly viscous. Its contents had not been characterized beyond the statement that it was essentially without cells but was rich in protein. The membrane, when at its thinnest was known to consist of a lining of flattened cells, morphologically more similar to the lining cells of bursas and synovial sheaths than to the endothelium of vessels, or the mesothelium of peritoneum or pericardium. The membrane, it was known, varied in thickness, and in the German literature in particular, there were elaborate descriptions of the details of the membrane as seen in several individual cases. The membrane contained, on occasion, a focus, or more than one focus of bone that had a primitive or immature pattern, now usually called woven bone. Where there were trabeculae in these foci they were thin and short, and they did not anastomose as frequently as do trabeculae in the cancellous bone of adults.

From this pattern, arose the speculation that the lesion was, in essence, a disturbance of bone formation, a dysplasia, or a dystrophy. I did not find, in the descriptions, mention of the existence in any case of a ball of this dysplastic bone, but I did find sections of such a ball in a few of my cases. These balls of tissue were not large enough to be noticed grossly, and in the slides although they appeared to be freely floating in the cyst cavity, I could not be sure that they weren't attached by a pedicle out of the plane of the section, or that they merely represented an excrescence whose base was above or below the plane of the section. The bizarre histology of these foci, which do not resemble any normal stage of bone formation, even in embryos, is still to be explained. The explanation should, ideally, be easily related to the explanation of the pathogenesis of the cyst cavity and its fluid.

The most favored "explanation" for a cyst then, in 1946, when found in a child's bone was a disturbance of growth. This presumption was largely dependent on the observation that most of the cysts arose close to the epiphyseal cartilage, and particularly in the proximal end of the humerus. The cyst seemed to grow as did the bone, but, inexplicably, sometimes a cyst would grow at its diaphyseal end too. In truth, the "explanation" is a cloak for ignorance: it merely hints at some reason why osteoblasts and osteo-

cytes have an organizational behavior in groups that is faulty. Their individual behavior in producing matrix may be normal, but instead of the matrix being patterned in trabeculae, it forms blobs, an error in design.

There are several items of clinical observation which must be taken into account and which should fit in with the histological findings, just mentioned, if experimental approaches are to be devised to clarify the pathogenesis of cyst formation. They have previously been discussed[8]. A few further observations are a propos as they affect the present discussion. The first relates to the model that is needed for any experimental approach. There is no lesion in any animal commonly available for experimentation that is at all similar to the lesion in humans. Patients who present for therapy, not experimentation, have to undergo the experimental manipulation, which must be atraumatic and without appreciable risk. There is no need to elaborate on these restrictions.

Another restriction does deserve mention. That is the availability of suitable patients for the experimental protocol. Starting de novo, that is, without a group of patients under observation who have not had operations or other therapy that might skew the experimental results, one must have some idea about how many subjects might come along within a reasonable period of time. One also must take into consideration the following two facts which have been established empirically. One is that lesions in different sites do not behave in the same way. The other is that lesions in adults are too diverse to allow a protocol to be established with confidence. A third observation may also pertain, namely, that a lesion encountered in its advancing, active stage, may pose different conditions than will obtain with a lesion in its inactive, latent stage.

It would be logical, therefore to choose for one's experiment, lesions at the upper end of the humerus, the most common site for simple cysts in children. This would confer on the protocol some semblance of purity of the sample. It would be nice to use only active or only latent lesions, but for practical purposes that is impossible. There will be too few subjects, and the criteria to establish activity or latency are too inexact. The important question that arises is "how many suitable subjects can one expect to become available?" That question can be answered as an approximation, by a study of the several large series of cases that have been reported.

That study, supplemented by inquiries directed at several large centers, allowed me to come to this interesting conclusion. If the past record is any indication, here as well as abroad, about two cases per year will present in any large academic center. This means that one must anticipate a rather long interval of time for conduct of the experiment, that interval being further extended because, often, the experiment will include longitudinal observa-

tions of the clinical behavior of the cyst over several months.

My own studies, begun about 1950[6], were first directed at a characterization of the fluid in the cyst. I found that, with the techniques of analysis that were easily available then, the fluid corresponded closely to serum, when not contaminated by hemorrhage, or residuals of hemorrhage. These data, more than 25 years old, and confirmed by others[11] are not to be taken at face value now, because the advances in analysis of proteins as a class of organic compounds that includes antibodies, complement and other compounds involved in clotting, hormones, etc., would require that there be a more sophisticated analysis to reveal how similar the fluid is to serum, etc. None of those compounds was sought for in the published studies, as might well be the case today. Obviously, the fluid in cysts is a subject that should be reinvestigated.

Those interested in pursuing this topic should become aware of a recent Japanese attempt[5] at characterizing cyst fluid. In six patients, aged nine to fourteen, at the time of aspiration the fluid pressure was recorded. It was noted to be higher (slightly) than the pressure obtained in the marrow of the contralateral humerus. The chemical analysis done included pCO_2, pO_2, HCO_2^-, pH, and content of bases, all of which were normal except for the pO_2 which was subnormal. These data were considered supportive of the theory of pathogenesis that I proposed in 1960, which will be further discussed below. The higher pressure indicated venous obstruction, and the lowered oxygen content indicated a more prolonged exposure of the fluid to metabolic demands of adjacent tissues as compared to normal demands from interstitial fluid.

Another of my experimental approaches to determine what causes cysts, more physiological in its method, was injection of a radio-opaque medium into the cyst and sequential observation of its disappearance. It is worth pointing out that only two subjects presented over a period of two years who were suitable for this experiment. In one, the cyst was in the latent stage, and in the other it was active. The results in the case of the latent cyst, were that the veins that drained the cyst and the proximal end of the metaphysis were those at the site of the previous fracture, and, in the case of the active cyst, those draining into the metaphysis. In other words, no metaphyseal veins were demonstrated. That finding was emphasized, but only now does it seem important that there were different patterns of drainage associated with active vs. latent cysts. Of course, one swallow doesn't make spring, and at least a few cases should be tested. Nevertheless, given the results obtained with analysis of the fluid, the results in both cases supported the postulate that the normal veins either were obstructed or were nonexistent. Putting together the clinical evidence with these observations and with the findings that in both cases the radio-opaque medium

was absorbed very slowly, the theory of anomalous development with venous obstruction was advanced. This theory allowed for a spontaneous reversal of the developmental defect, so that spontaneous disappearance of the cyst would be "explained".

It should be pointed out that the theory, as advanced, merely postulates trouble in the circulatory process, but omits any consideration of the nature of the trouble. One is free to speculate about thrombosis, or trauma, or whatever, and also speculate on the peculiar site of predilection of the lesion. However, whatever the basic etiological agent, it is not likely that effective therapy would be targeted at the precipitating cause. A better target would be the secondary effects.

In 1968 Scaglietti proposed that instead of using any of the several surgical treatments advocated, injections of steroid hormone in the form of microcrystals of methyl prednisolone be tried. Mention of that treatment was made in a review article I wrote in 1977[8] in which the treatment is characterized as "under study" and without "sufficiently detailed reports to allow further discussion". In his first article on the subject[10], Scaglietti states that the same treatment was tried on a number of other benign lesions of bone. Later, in 1979, he reported his results (mostly favorable) and theorized that the treatment possibly was effective because the steroid's "microcrystals caused destruction of the connective tissue coat of the cystic wall, thus allowing secondary osteogenic repair".

Another speculation made in the same article, is that "injection of a corticosteroid into the cavity would cause resorption of the cystic fluid in the same manner as it causes resorption of transudates in a joint." Neither of these explanations makes much sense, but some accounting for the frequent success of the treatment should be formulated, supported by credible evidence. In my view, as argued below, the steroid probably is irrelevant as an active agent in the treatment.

Only a few articles[1,2,3,4] have been written, reporting on the actual rate of success of the steroid treatment in large series of cases, and none of them allow the reader to judge precisely the percentage of success beyond the round figure of perhaps more than half. I hope to show, in this essay, what may be the reason for the imprecision, and in so doing, to point the way to better success.

In the Japanese article previously cited, Chigara et al.[5] treated seven children (in 1983) with the idea of allowing prolonged drainage of the fluid. A majority had excellent results. The lesions were not restriction to the proximal end of the humerus, and in those that were, only two out of four had excellent results. The treatment consisted of multiple drilling with 2 mm Kirschner wires which were left in situ for a period not specified.

This experience, plus that with the steroid treatment (often repeated, I might add) suggested to me that the

common factor in the two treatments was the perforation of the cyst wall, including the thin layer of bony cortex, and the true explanation of success with either treatment might be the development of venous channels in the callus which must form in the iatrogenic holes in the bone. This explanation jibes well with the experimental results I have described with the radio-opaque medium. If true, it also lends some significance to the venous pattern as associated with activity or latency of the lesion.

It should be noted that there was general agreement, prior to 1974, that many cysts would heal after a fracture, but in that year Galasko[9] reported a study on that point, and could not corroborate the general impression, based as it was on anecdotal "experience". The study not withstanding, I now believe that when the steroid treatment succeeds, or when a cyst heals spontaneously after trauma, the success depends on the generation of veins in the callus that are large enough or numerous enough to allow the fluid to transude back into the circulation at a rate high enough to prevent stasis. If all this is true, the frequent, but not invariable, success obtained with the Kirschner wire treatment does not depend on the transient period of drainage of cyst fluid while the wires are in place, but upon the succeeding drainage by transudation of the fluid into the circulation.

Pursuing this line of thought further, the amount of callus that develops might be the critical factor in the success of treatment, and it would be of the essence to insure that there be profuse callus. So, as a practical suggestion, the treatment might well consist of the use of a Terkel needle, or other trocar of similar design, instead of small-bore injection needles. The obvious variations on this theme would be multiplying the perforations, inserting absorbable materials in the holes e.g. sutures, etc.

One further thought occurred to me while I was doing my experiments, and it may be worthwhile to record it here. Mention has been made of the frequent occurrence in the cyst wall of woven bone where there should be cortical or cancellous bone. Might this abnormality be the consequence of long-standing hypoxia, and/or prolonged, but slight elevation of venous pressure in the intraosseous milieu? If there is a vascular mediation of the lesion, might it not be true that other lesions that contain woven bone do so because of venous stasis?

Such speculation, if it is not to be idle, although interesting, must evoke experimental studies, and I am suggesting that the microvascular structures and physiology would be a fruitful subject for investigation. In bone, the methods for studying the microanatomy and physiology, in the normal as well as in particular lesions are particularly uncertain, mostly because of the mineralized impediment to methods suited to soft tissues. The unique feature of the vascularity of bone, the absence of lymphatic vessels,

makes for difficulty in explaining the return of tissue fluids to the circulation, and that was one of the enigmas that faced me in my initial attempts to understand how bone cysts form. It is my hope that with further experiment, with new methods, that enigma will be resolved.

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FUNCTION OF KNEE LIGAMENTS:
AN HISTORICAL REVIEW OF TWO PERSPECTIVES

Randall R. Wroble, M.D.
Cincinnati Sportsmedicine, Deaconess Hospital
311 Straught Street, Cincinnati, OH 45219

Richard A. Brand, M.D.
Department of Orthopaedic Surgery
University of Iowa, Iowa City, IA 52242

The gross form of ligaments naturally suggests a passive structural function. Since the beginning of recorded observations, anatomists exclusively recognized this function and clinicians based their treatment on that function. More recent concepts and observations, however suggest neurosensory functions, perhaps relegating the structural functions to a secondary role.

While this synthesis of ideas and observations suggests a need for re-evaluation of current clinical concepts, the model of the ligament as a neurosensory organ is not a new one. Herein, we will present historical aspects of knee ligament function. First, we will discuss the separate historical development of the structural and neurosensory models. Following this, we will explore some reasons why these models have developed in essentially a mutually exclusive way. Scientific evidence for each has accumulated during the same time period but nonetheless, the ideas have been connected only on rare occasions. To date, they remain parallel and distinct — an intriguing dichotomy.

Clinicians, especially surgeons, and some early biomechanicians[15] interpreted the anterior cruciate ligament (ACL) as a check rein or guide rope only — the structural mode. The mechanisms of injury, indications for treatment, and rationale for surgical technique were based strictly on these precepts.

At roughly the same time, another group, primarily physiologists and anatomists, developed another way of looking at ligaments. In this view, the ACL (and other ligaments) act as sensory organs, providing the central nervous system with information about stresses applied to the ligaments. Efferents then could cause appropriate muscles to contract in response. This is the neurosensory model.

In the 100+ year history of these models, there have been only rare attempts at synthesis of the two. The rest of the time they have existed in parallel, never meeting.

Ligaments were known to ancient Greek anatomists. Galen[19] described their structural function quite clearly. In his work, "On the Usefulness of the Parts of the Body," he provides a succinct delineation of the structural role of ligaments. ". . . if the articulating bones were not strengthened by ligaments, there would be nothing to keep them from being displaced from their proper situation in every movement and turned now to one side and now to the other. To prevent anything of the sort from happening, Nature surrounds every diarthrosis of bones on all sides with ligaments which are strong, indeed, but which also permit a considerable degree of relaxation."

The first clinicians interested in the ACL thought only of its mechanical role. Stark,[48] who in 1850 first described a case of ACL rupture, mentioned what he considered to be the ACL's functional role. He said that "if the crucial ligaments, however, be cut, there is nothing to prevent the joint from being bent to a certain extent in every direction, more especially backwards and forwards." In 1907, Pringle[43] published the first English language study directed toward experimental determination of ACL function. His interests lie in determining the mechanisms for cruciate injury. In a series of cadaver experiments, he found that he was able to rupture the ACL by combined flexion, abduction, and internal rotation. He recounted the similar but earlier experiments of Pagenstecher and Honigschmied. In addition, he pointed out the role of the ACL in rotational motion of the knee when he said that "it is round the anterior cruciate ligament, tightened up as it is by the extension of the joint, that the inversion of the femur takes place, as round a pivot, in the last movement of locking the extended knee." This appears to be one of the first descriptions of the knee's terminal "screw home" mechanism.

Hey Groves and Jones, two prominent names in the early history of the ACL treatment, were also quite clear about confining their description of ACL function to purely structural aspects. Hey Groves[23,24] noted that the ACL checked the forward motion of the tibia and that it was made tense with extension of the knee. Jones[28] elaborated on this and said, in fact, that "from the mechanical stand-

point nothing could be more beautiful and efficient than the action of these (cruciate) ligaments in stabilizing the joint." He also states that the ACL was tight in extension and in flexion and that it prevented the anterior movement of the joint.

Milch[34] concluded that the cruciates function mainly during flexion and that with the knee extended, the stability of the knee was maintained by the collateral ligaments and the capsule. Horwitz came to quite different conclusions; he felt that the ACL was taut in flexion and relaxed in extension and that the role of the ACL was a secondary one. He stated that "the action of the cruciate ligaments is shown to be either accessory or prominent only during the flexion phase of knee joint motion and not indispensable to the stability of the knee joint"[26].

In a landmark 1941 paper, Brantigan and Voshell summarized the various disputes about the function of the cruciate ligaments[6]. They pointed out the literature at the time revealed no unanimity of opinion regarding ligament function, commenting apocalyptically that "a study of the literature on this subject leaves one bewildered". Their very elegant series of cadaver experiments clearly delineated in detail the mechanical function of the ACL. Their paper represents a prime example of the structural model of the ACL brought to full flower.

Helfet[22], another structuralist, felt that cruciate function was not so much to prevent anterior-posterior displacement, but rather was to act as guides for rotation of the tibia on the femur. He refers to them as guide ropes which during the last 30 to 40 degrees of extension keep tibial rotation in a set path.

Augustine[3] described the first dynamic ACL reconstruction in 1956. He felt the role of the cruciates was to hold the tibial plateau opposed to the femoral condyles, to resist anterior and posterior drawer, but to permit some varus and valgus motion along with tibial rotation. He felt that in a flexed position, knee stability was more determined by dynamic rather than static structures.

This, of course, represents just a small fraction of the work done on assessing ACL function. What is apparent from this summary is that all these investigators thought of the cruciate ligaments as a mechanical obstacles to abnormal joint movement. None of them appeared to regard any other facet of ligament physiology as important. In fact, there appears no suggestion that they even acknowledge the existence of other such functions.

The first line of evidence supporting an additional nonstructural function of ligaments was first expressed by Delpech and by Hunter[4]. They separately formulated what became known as the "law of the ligament". They felt that muscles rather than ligaments acted as the primary support for joints. Ligaments only came into play when muscles were incapacitated by injury, fatigue, or disease.

Muscles served as the means for postural maintenance, in other words, proprioception. They further stated that ligaments could not provide continuous support and only limited motion when normal muscle support was absent.

Although these authors expressed their views in the 18th and 19th centuries, experimental verification of this idea was not forthcoming until 1981. At that time, Lewis[31] demonstrated that during normal walking and trotting only minimal force was generated in canine collateral ligaments. He concluded that during normal loads and normal motion, ligaments and capsule play a less important mechanical function than during extremes of load or motion. Joint stability derived mostly from the bone geometry and from the muscles. At the extremes of motion, he suggested that both mechanoreceptor stimulation and increased tension of the ligaments made them play important mechanical and sensory functions.

In 1965, Freeman[16] suggested that dysfunction following ankle sprains resulted from motor incoordination, consequent to articular deafferentation. Twenty-five percent of his patients had proprioceptive deficits. He employed proprioceptive training to reduce their symptoms of giving way and reported successful results.

The neurosensory role of ligaments has also been investigated from an anatomic perspective. While the gross anatomy of the innervation of joints was defined in the 19th century, Gardner[20] reported the first detailed study of the intraarticular distribution of nerves to the human knee in 1948. He demonstrated nerve endings in association with blood vessels within the pericapsular tissues, but was unable to identify mechanoreceptors (though he did find them in feline joint capsule). He did find that the human knee was innervated by branches from the femoral, obturator, and sciatic nerves, thus providing an experimental validation of Hilton's law. Freeman and Wyke in 1967 delineated four different types of articular receptors and identified nerve endings in all of the knee ligaments[18]. O'Connor[35] confirmed that a variety of encapsulated mechanoreceptors existed in the cat medial collateral ligament (MCL). In 1982, Kennedy[30] found nerve fibers in the tibial insertion of the human ACL and in the synovial covering of the ACL but no receptors in the ligament. Conte[8] demonstrated mechanoreceptors in the human MCL. Schultz[44] in 1984 first demonstrated mechanoreceptors on the surface of human cruciate ligaments, while Schutte[45] in 1987 identified mechanoreceptors deep in the substance of the human ACL.

Other investigators discussed the proprioceptive function for ligaments in a more speculative manner. Payr in 1917, wrote about "Die Kinetische Kette" — the kinetic chain. He said the nerves act as a medium between the joint and the muscles. He postulated that irritation of the sensory endings of the ligaments via increased ligament

tension would cause protective contraction in the muscle groups functionally connected with the part of the joint involved[37,40]. Abbott[1], in 1944, published one of the first clinically oriented works in this regard. He pointed out that ligaments have a rich sensory innervation and stated that

> "impulses arising in the ligaments are transmitted through the CNS back to the effector muscles, thus maintaining the normal, smooth, corrdinated motion of the joint. Abnormally strong impulses, such as are initiated when the joint is forcibly overstretched, result in contraction of the allied muscle groups, thereby protecting the ligament and preventing further injury and subluxation of the knee".

He also used the term "the joint team" in which he regarded the ligaments as only one of many important elements, including nerves, which contributed to controlling joint motion.

Palmer, another clinician, also explored the proprioceptive functions of ligaments[37-40]. He conceptualized a functional unit—"The physiologic joint"—which included menisci, synovium, ligaments, and muscles with their nerve supplies. A lesion in any one of these parts could result in injury to the entire joint. He believed that the sensory function of ligaments provided the joint with protective muscular reflexes and said that "a harmoniously functioning joint is the result of an intimate cooperation between muscles, ligaments, and articular surfaces controlled by the nerves". In 1944, he even more specifically stated that ligaments do not act as check-reins alone but "serve as terminal apparatuses for the neurogenic component of the joint". He clearly stated the sensory role of the ligament—

> "They constitute an exciting organ for the muscular defense reflexes of the joint. The tensing of the ligament activates a group of muscles with which it is connected functionally. It is through this mechanism alone that the [ligaments] are able to resist the often violent strain to which they are submitted during the course of a lifetime".

Kennedy[30] also thought that mechanoreceptors within ligaments allowed ligaments to function not only as mechanical stabilizers but also as initiators of protective relexes. He said that "failure of stretched or damaged ligaments to provide adequate feedback in the injured knee may contribute to unpredictable giving way and result in progressive ligamentous laxity". Conte[8] suggested that the integrity of the knee may depend on "the possibility of instantaneously and automatically regulating the contractions and extensions of different muscle groups". Schutte[45] stated that the ACL "has a mechanoreceptor system that is able to respond to the tension of the ligament. . . . Information provided by this population of intraligamentous receptors allows the CNS to appreciate speed, accel-

eration, direction of motion, and position of the joint".

A final line of evidence revolves around physiological experiments done to document the reflex functions of ligaments and other pericapsular tissues. Hilton paved the way for research in this area. He stated what came to be known as Hilton's law when in 1863 he said,

> "the same trunks of nerves, whose branches supply the groups of muscles moving a joint, furnish also a distribution of nerves to the skin over the insertions of the same muscles; and — what at this moment more especially merits our attention — the interior of the joint receives its nerves from the same source".

He also suggested a relation between muscles and articular nerves. He stated "muscles, indeed, appear to be told, through the medium of the nerves of the interior of the joint, that its articular structures are overtasked".

According to Dee and Wyke[50], Goldscheider in the late 19th century first proposed that joint position sense was due mostly to receptors from joints. Shortly thereafter, Sherrington, while agreeing that joint receptors contributed to position sense, stated that muscular and cutaneous receptors were the principle reflex regulators of muscle tone and the major contributors to postural sense[50]. Stopford, in regard to the fingers joints, felt that joint receptors were more important than those in the tendons[41].

Keith, in 1919, suggested that knee ligaments and capsule have receptors which are affected by changes in pressure and tension of the joint[29]. After injury, these receptors send abnormal signs to muscles. In 1924, Partridge[41] elaborated further on Hilton's work saying that, for example, knee extension would continue until the tension in the ACL and the posterior capsule is great enough to signal the flexors to contract, in this way protecting the joint.

In the 1950's, Mountcastle, unable to find cortical representation of muscle afferent, suggest that postural stability was due to joint receptors[11]. Some studies further supported the role of joint receptors in a wide variety of functions. Andrew[2] showed that receptors in the MCL of various animals responded to tension applied. Likewise, Ekholm[12] showed that both tension and increased intraarticular pressure activated joint receptors in cats. Skoglund[46] in 1956, showed two types of receptor response in cat ligaments, slowly adapting receptors which were active only at certain joint angles and rapidly adapting receptors which were active throughout the entire range of motion of the knee. He thus felt that joint receptors functioned for position sense, speed and direction of motion, and detection of acceleration or deceleration. He steadfastly maintained that muscle receptors do not contribute to kinesthesia[47]. Ferrell found receptor activity through a full range of knee motion, and concluded that joint receptors could adequately signal joint angle[14].

Wyke[50,51], describes two general functions of joint

receptors: 1) the kinesthetic function, giving awareness of joint position and movement, and 2) the function in arthro-kinetic reflexes. In these latter, he felt that mechanoreceptors can contribute to regulation of muscle tone and provoke either facilitation or inhibition during joint displacement. He described them as "brakes" that limited excessive displacement. In 1981, however, he admitted that the work of Burgess, McCloskey, and others indicated that joint receptors were supplementary to skin and muscle receptors in regard to postural sense.

Grigg, Burgess, and McCloskey[21,7,33] have generated a large body of evidence which excludes joint receptors as being entirely responsible for kinesthesia. Burgess demonstrated that articular nerves were almost exclusively active at the extremes of flexion and extension (in the cat knee), and found very little activity at mid-ranges of motions. He concluded that these receptors were not useful for maintenance of posture. Grigg found receptors in the posterior capsule of the cat knee which were sensitive to stretch, the amount of discharge from the nerve being proportional to the applied stress. However, since the receptors were almost entirely localized to the posterior capsule, he felt they served as "limit detectors" only. McCloskey cites several studies indicating that joint receptors normally make little or no contribution to the sense of position or movement. At the very least, he felt that any sensory input they provided was duplicated adequately by other sources such as a muscle or cutaneous receptor. He cites the work of Burgess and Grigg showing that they were not capable of providing information over most of the working range of the joint. He also noted the fact that anesthetizing joints changes kinesthesia only when the overlying skin is anesthetized. Finally, he cites evidence that in patients with total joint replacement and, therefore, virtually totally denervated joints, kinesthetic sensation is only minimally impaired. Interestingly, one may surmise from the fact that cutaneous and muscular receptors facilitate proprioceptive signals from joints that one of the functions of knee braces may be to facilitate the function of these nonarticular receptors.

One could conclude from limited experimental evidence that joint receptors play a small role in maintenance of posture and muscle tone. However, it is important to realize that different joints in different species exhibit differing mechanisms and structures related to differing functions. Furthermore, their role in arthrokinetic reflexes is considered important. In 1959, Stener[49] tried to verify the existence of ligamento-muscular protective reflexes. Unable to demonstrate this in either cats and humans, he suggested that muscular responses seen in ligament injuries originated from pain receptors rather than mechanoreceptors. Ekholm[12], in a series of similar experiments, demonstrated activation of joint receptors with tension on the cat MCL. He felt Stener failed because of using too excitable a preparation (the decerebrate rather than the decerebrate, spinalized animal). deAndrade[10] studied joint distention and its effect on muscle inhibition. He concluded that stimuli from the knee joint could reflexly inhibit motor neurons, particularly those to the quadriceps. Freeman and Wyke demonstrated that partial deafferentation leads to alterations in postural reflexes in cats suggesting that capsule and ligament mechanoreceptors normally contribute to these[17]. Lindstrom and Norrsell, however, later repeated these experiments and could not reproduce their results[32]. In 1979, Pope[42] also attempted to find a muscular reflex in response to stretching the MCL. He tested reaction times to various stimuli and concluded that ligament musculoprotective reflexes are too slow to protect against many sports injuries. Finally, O'Connor[36] studied the development of neuropathic arthropathy in dogs. He compared three groups of animals, one with deafferentated knees, a second with isolated ACL transections, and a third with deafferentation plus ACL transection. In the third group, degenerative changes developed more rapidly and were more severe than in either of the other two groups. He concluded that "ipsilateral sensory input may play a role in eliciting joint reflexes and behavior that retard the rate at which an injured (ACL-deficient) joint degenerates".

Why has this dichotomy between the structural and neurosensory models continued to exist? Why has there been so little work attempting to synthesize the two views? And furthermore, why has the neurosensory view apparently received so little attention from clinicians? First, there are some advantages to considering the ACL a mechanical stabilizer alone. Clinical diagnosis, at least theoretically, should be straightforward. The ACL restricts anterior tibial translation; therefore, we attempt to displace the tibia forward to evaluate its status. We even have instrumented arthrometers available to assist us. On the other hand, there is no way to clinically diagnose subtle neurosensory deficits in the knee joint. Second, we can easily think of ways to treat a mechanical deficit. As we all well know, a variety of surgical procedures are purported to do this. We have no current way of treating proprioceptive deficits in the knee. Third, it is relatively straightforward to design experiments to test the structural hypothesis.

This is not the case with the neurosensory hypothesis —witness the complexity of and variation in results obtained by the neurophysiologists just described. It should not be surprising then that clinicians have not delved into the ACL's neurosensory function more enthusiastically.

Another idea stopping most clinicians from looking at alternative hypotheses is that from the beginning, the ACL has been viewed only as an isolated anatomic structure.

How it fits into the so-called "kinetic chain" has not been considered. It seemed easy to look at the ACL merely as a rope that one needs but to pull or twist to determine its properties. From the outset of experimentation with joint proprioception, however, investigators viewed ligaments as part of a system containing multiple elements, all of which interacted with each other and had interrelated functional roles—"the joint team".

Clinicians may have failed to look beyond the ACL's structural importance because their experience was derived, as a matter of course, from the observation of the "abnormal". In this scenario, the temptation exists to attribute the problem to the obvious defect—the torn ligament. In a complex system such as the knee, a simple one-to-one correspondence such as this may not hold true. Physiologists looking at "normals" have come to quite different conclusions reached in a distinctly different manner.

It seems apparent from the simple volume of literature being generated that there has been an increase in interest in ligaments' proprioceptive functions. Perhaps this resulted from the structural model's apparent failure to explain all the facts regarding knee ligaments and their injuries. It may be simply because the concept is intellectually attractive.

In any case, one is tempted to agree with the statement that there is "little doubt that ligaments provide sensory input to the CNS. How much this contributes to reflex muscle control and functional joint stability are issues needing resolution"[5]. Furthermore, incorporating the neurosensory model into our thinking may help to explain some of the confusing and contradictory observations made about knee ligaments, particularly the ACL. As Cooper[9] pointed out to the American Academy of Orthopaedic Surgeons in his presidential address, "establishment of key questions must precede successful solutions". With this model, we may indeed establish what those key questions are.

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ANTERIOR KOSTUIK-HARRINGTON DISTRACTION SYSTEMS FOR THE TREATMENT OF KYPHOTIC DEFORMITIES

John P. Kostuik, M.D., FRCSC
Professor University of Toronto
Head, Combined Division of Orthopaedic Surgery
Toronto General/Mount Sinai Hospitals
Director of Spinal Unit
Toronto General Hospital
Toronto, Ontario
Canada

INTRODUCTION

Treatment of kyphotic deformities has always presented a challenge to Orthopaedic Surgery. The work of Hodsgon[2] et al. from Hong Kong ushered in the modern era of the treatment of kyphotic deformities. Subsequent work by Moe, Winter and Bradford[6] clearly demonstrated that anterior surgical decompression of the dural canal, when indicated, together with anterior strut grafting provided the maximum possible success in the treatment of rigid kyphotic deformities of the spine. Malcolm and Bradford[5] found that failure rates from anterior fusions were as high as 50 percent in the treatment of post-kyphotic deformities of the spine. They recommended anterior correction of the deformity together with anterior grafting followed by a secondary posterior instrumentation and fusion in order to decrease the incidence of pseudoarthrosis.

Recently, an increased interest in anterior approaches to the spine for fractures has developed due to an improved understanding of the mechanics of kyphosis, a better mechanical classification of fractures, a better understanding of the anatomy and improved imaging techniques of the spine. Until recently, most anterior procedures required supplementary posterior procedures in order to assure postoperative stability. The advent of anterior fixation devices may preclude the necessity of posterior instrumentation and fusion, except when the posterior column is also destroyed, when at approximately ten days later supplementary posterior fusion and instrumentation is required

BIOMECHANICS OF KYPHOTIC DEFORMITIES

Definition

In the thoracic spine, angulation in the saggital plane greater than 40 degrees is considered as abnormal. In the cervical and lumbar spine, 5 degrees or more of fixed posterior angulation is defined as a kyphotic deformity.

Anatomical Considerations

The spinal column can be thought of as consisting of the anterior elements and the posterior elements. Everything anterior to the posterior longitudinal ligament is considered part of the anterior elements.

When evaluating burst fractures of the spine, the three column concept of Dennis[2] is used. The anterior column consists of the anterior longitudinal ligament and the anterior two-thirds of the body. The middle column consists of the posterior third and cortex of the body and posterior longitudinal ligament. All elements posterior to the posterior longitudinal ligament are in the posterior column. The resting positions of the spine are dictated by the osseous and ligamentous components. The physiologic thoracic kyphosis is determined primarily by osseous structures and the lordotic curves of the cervical and lumbar spine are determined by ligamentous structures.

BIOMECHANICS OF KYPHOSIS—(WHITE AND PANJABI)[8]

The posterior elements are considered to be under tension and the anterior elements are under compression. Kyphosis may occur when either of these two components are disrupted. Posteriorly, the laminae and ligamentae flavum are major structures resisting tension.

Non-physiological loads, both in magnitude and direction may also result in a kyphotic deformity. An increase in the moment arm (Fig. 1), that is the amount of angulation present, also plays an important role in the production of kyphosis. Under static load conditions, wedging accentuates angulation and effectively increases the moment arm which in turn increases eccentric loading and a vicious cycle ensues.
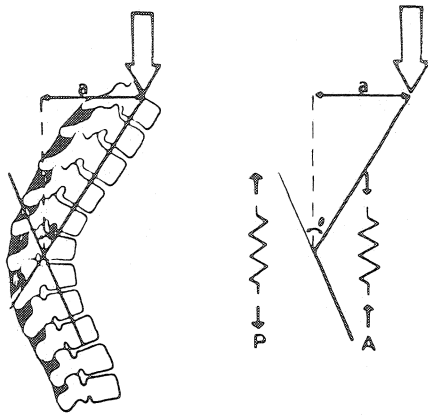
Figure 1
BIOMECHANICS OF KYPHOSIS: On the left, there is a kyph-
otic deformity showing a Cobb angle of 58 degrees. Vertically
directed physiological forces shown by the large arrow work at
a moment arm at length (a). On the right, the deformity is
depicted schematically, showing that the posterior elements (P)
resist tensile loading. The anterior elements (A) resist com-
pressive loading. Factors tending to contribute to kyphosis
include:
1. increase in physiologic load (white arrow)
2. increase in moment arm (a)
3. weakening of posterior elements
4. weakening of anterior elements

BIOMECHANICAL CONSIDERATIONS INVOLVED WITH TREATMENT

The treatment of severe kyphosis involves adequate
decompression of spinal cord, if necessary, and the appli-
cation of forces to correct deformity. These corrective
forces consist of axial traction and sagittal plane bending
moments (transverse loading). Axial loading is greater than
transverse loading when the Cobb angle is greater than
53 degrees. These correctional forces reverse the role of
the anterior and posterior structures.

PATHOMECHANICS OF FUSION (GRAFTS)

Posterior fusions are generally under tension and are
usually thin, susceptible to stress fractures, and may bend.
The stability of a posterior fusion increases in proportion
to its length. Despite this, pseudoarthrosis rates and fail-
ure to maintain correction are as high as 40 percent in the
treatment of Scheuermann's disease in adults[4]. Con-
versely, anterior fusions are under compression and are
therefore ideal.

If one considers kyphotic deformity as a bent column
and the middle of the column the neutral axis, the more
one moves away from this neutral axis towards the con-
cavity, the more the moment arms are reduced, thus, the
more effective the support. Ideally, bone grafts for fusion
kyphosis should be placed as far as possible from the
neutral axis on the compressive side and include all ver-
tebrae that are in the deformity.

THE RATIONALE FOR ANTERIOR INSTRUMENTATION

Iliac grafts (bicortical and tricortical) incorporate well
but cannot withstand the compressive loads encountered
in the erect position in the lumbar spine. These compres-
sive loads can approach three to four times the body weight[8].
Fibular strut grafts are sufficiently strong, but do not incor-
porate and revascularized quickly enough. Internal fixation
devices enhance stability.

INSTRUMENTATION

The Kostuik-Harrington instrumentation provides ade-
quate rigidity and stability provided that it is used in a
rectangular or parallelogram fashion (Fig. 2). The assets
of the system are in its versatility, ease of application, and
adapatability. The system allows for correction of deform-
ity and early ambulation. The system utilizes standard
Harrington distraction instrumentation with a crimper for
the screw heads when heavy compression rods are used
in conjunction with distraction (such as in burst fractures).
Crimping the collar-ended heads over the heavy compres-
sion rod is faster and as effective as using nuts. Equipment
consists of a collar-ended screw and a distraction screw.
The heads of either screw are compatible with the stan-
dard round end of the Harrington rods. The screw heads
are attached to cancellous threads which come in three
lengths. Ideally, the maximum length is used and the excess
cut after measuring the depth of insertion required with a
depth gauge and adding 2 to 3 mm in order to assure
penetration of the contralateral cortex of the vertebral
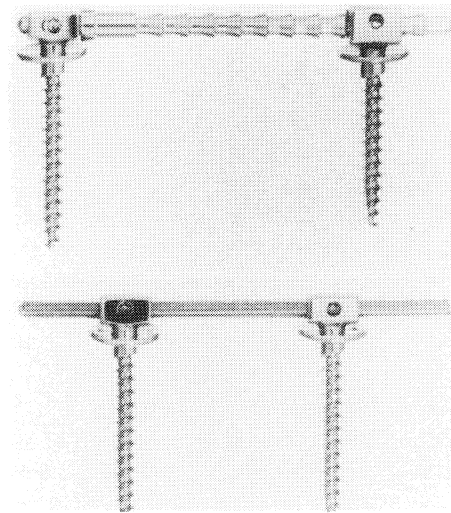body.

Figure 2
Kostuik-Harrington system. Round ended Harrington rod dis-
traction screw (rachet end).
Collar-ended screw
Washers
Heavy compression Harrington rod

INDICATIONS

The anterior Kostuik-Harrington system is used for all forms of acute and chronic kyphotic deformities. The indications for its use are: 1) burst injuries of the spine, 2) post-traumatic kyphosis, 3) Scheuermann's disease, 4) rigid round back, 5) acute rigid kyphosis, 6) post-laminectomy kyphosis and instability, 7) iatrogenic lumbar kyphosis (flat back syndrome), 8) kyphosis secondary to tumor, and 9) kyphosis secondary to osteoporosis with fracture.

CLINICAL STUDIES

Since 1981 the author has employed anterior distraction consisting of distraction and collar-ended screws together with rounded compression rods in 279 cases (Table 1). Additionally, the screws have been used posteriorly for pedicle fixation for a variety of indications (pseudoarthrosis, multiple level degenerative disease, tumor) in fifty cases.

Table 1

Clinical Applications of Kostuik-Harrington Instrumentation

Indication	# of Cases
acute burst injury	100
post-traumatic kyphosis	45
Scheuermann's disease	36
rigid round back	3
acute rigid kyphosis	4
post-laminectomy kyphosis	21
flat back syndrome	56
kyphosis secondary to fusion	10
kyphosis secondary to osteoporosis with fracture	4

COMPLICATIONS RELATED TO INSTRUMENTATION AND FUSION

To date, the instrumentation has been used in 279 cases anteriorly and fifty cases of pedicle fixation posteriorly with the following complications:

1. Screw breakage totaled thirty-five. The majority (twenty-four), were with the older, thin shank unta-pered screw. Twenty-three of these occurred with burst fractures and four in Scheuermann's kyphosis, three in post-traumatic kyphosis, and four in pedicles. Only four cases lost correction.
2. Two distraction rods fractured at the junction of the rachet rod area, a well recognized point of stress concentration. There were no abnormal sequelae to this problem. Additionally, two heavy compression rods fractured.
3. Vertebral body fracture occurred in eight cases. All occurred intraoperatively in osteoporotic bone with three in the same person. All cases were salvaged by the insertion of methylmethacrylate bone cement. Addi-

tionally, screws were applied within the fractured body in three instances. In two cases, a more proximal vertebral body was used. The use of bone cement in osteoporotic vertebral bodies is recommended and appears to provide excellent purchase. The body is drilled with a standard drill and measured for depth. The drill hole is then enlarged with the aid of a curette and cement is packed in. The screw is then inserted over a washer. The screw can be turned after the cement hardens. There have been no vascular injuries and no neurologic injuries.

TECHNICAL POINTS— GENERAL

1. Use an awl to start the screw holes
2. Place the screws at mid-body (slightly anterior in fractures—slightly posterior in other kyphoses).
3. Pierce the far cortex.
4. Use staples where possible (screw length should be measured with depth gauge and add 2 to 4 mm).
5. Place the distraction screw(s) where the spine proximally or distally is level in order to allow for ease of rod insertion.
6. During distraction apply manual pressure posteriorly.
7. Distract as with posterior Harrington instrumentation.
8. In Scheuermann's disease place both rods first and distract both concurrently.
9. In burst fractures distract to a predetermined length (normal body plus two disc heights as measured from lateral x-rays of patient). Place the bone graft, preferably iliac crest bicortical or tricortical grafts, slightly longer than gap. Rib graft can be added. Add a second heavy Harrington compression rod and crimp screw heads lightly.
10. Cut excess rod length.
11. In burst injuries and other short kyphosis, e.g. post-traumatic kyphosis, the compression rod is essentially a neutralization rod.

CASE ILLUSTRATIONS AND SURGICAL TECHNIQUE

Technique varies with the type of kyphosis.

A) BURST INJURIES (Figure 3)

Indications

1. Early injuries with neurological signs. Early is defined from hours to days.
2. Late injuries (ten to fourteen days) with or without neurological signs. The analogy here is to a Colles' fracture which are recognized as being difficult to reduce because of the cancellous nature of the bone after ten days. It is felt that posterior instrumentation cannot correct a kyphotic deformity after seven to fourteen days.
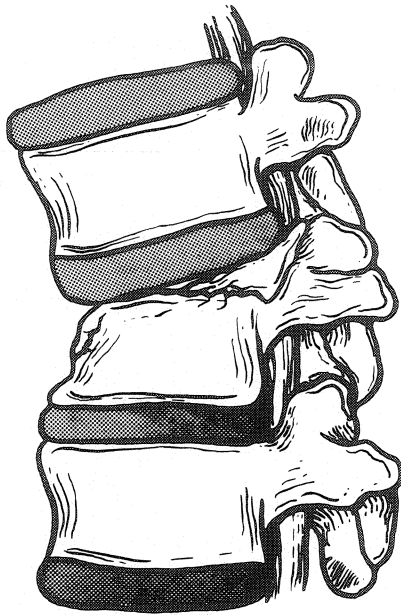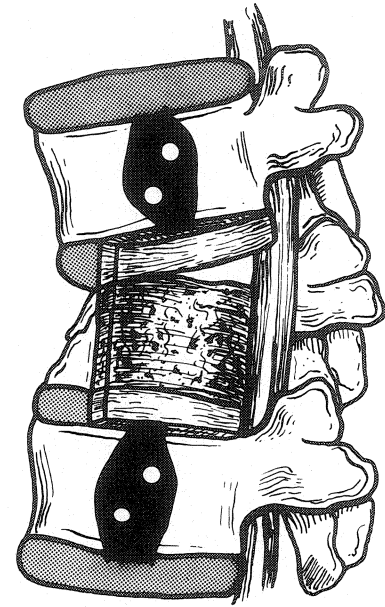
Figure 3
Burst fracture.

Figure 4
Dura decompressed. Anterior one-fourth of body is left as graft.

3. Computerized tomographic evidence of significant canal intrusion by bone fragments. It is recognized that this point may still be controversial as it has not yet been decided what degree of canal intrusion is important from a neurological viewpoint.
4. Post-traumatic kyphosis.
5. Burst injuries at L3 or lower are preferably treated by anterior methods in order to preserve as many distal vertebrae as possible.

Anterior instrumentation and fusion involves two disc levels whereas posterior distraction instrumentation with Harrington or other rods involves considerably more. The use of posterior instrumentation without fusion or fusing over a short area is not recommended since there is good clinical and experimental evidence to suggest that immobilization of the posterior facet joints results in early degenerative changes even with early rod removal.

SPINAL FRACTURES

Technique

A lateral decubitus left sided approach is preferred. The incision is usually two levels above the fracture (i.e. tenth rib for fracture at T12). The anterior one quarter of the body is left and the dura is fully decompressed (Fig. 4). The Kostuick screws are inserted. Distraction is then done. C-clamps are applied to the rachet end of the distraction rod after distraction is completed. The bone graft is then inserted (Fig. 5). A rib strut, if available, is added.
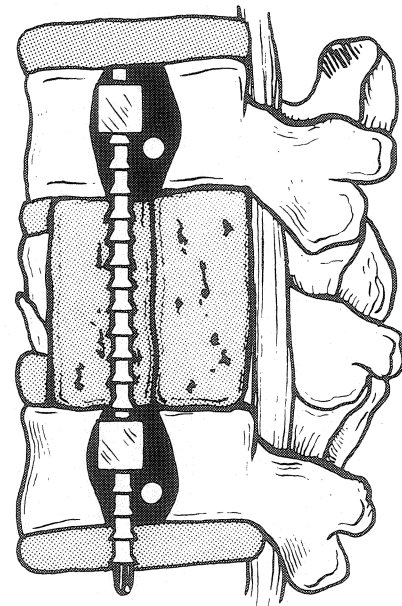
Figure 5
Anterior lateral rod is inserted to reduce kyphosis. Bone graft (iliac crest) is added.

A heavy compression rod is inserted into the collar-ended Kostuik screws which are angled forward from the near posterior part of the body to the contra-anterolateral cor-

tex of the body (Fig. 6). Slight compression is applied and the screw heads are lightly crimped.
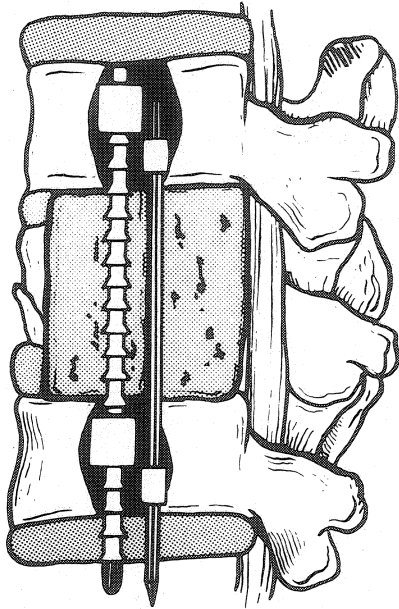
Figure 6
Second heavy Harrington compression rod is added to increase stability. Slight compression is applied. Collar-ended screw heads are crimped.

Case illustration (Fig. 7-12) — C.D. A twenty year old male fell 12 meters, sustainining a burst injury of L2, Frankel grade B. Decompression, correction of deformity, grafting and stabilization were carried out with in twenty-four hours. At two years following he was functionally graded as per Frankel.

Results

A total of 100 cases were dealt with by the above technique. There were four nonunions, including two cases early in the series. In retrospect, because of severe posterior column comminution, a second stage posterior fusion and instrumentation should have been added.

Screw fracture occurred in twenty-three screws, the majority of which were in the earlier model of untapered screws. One rod fractured.

Average neurologic improvement was 1.6 grades in the partial paraplegics with a range from 2.0 to 1.0. Greater improvement was seen the earlier the case was done. There were no early or late neurovascular problems.
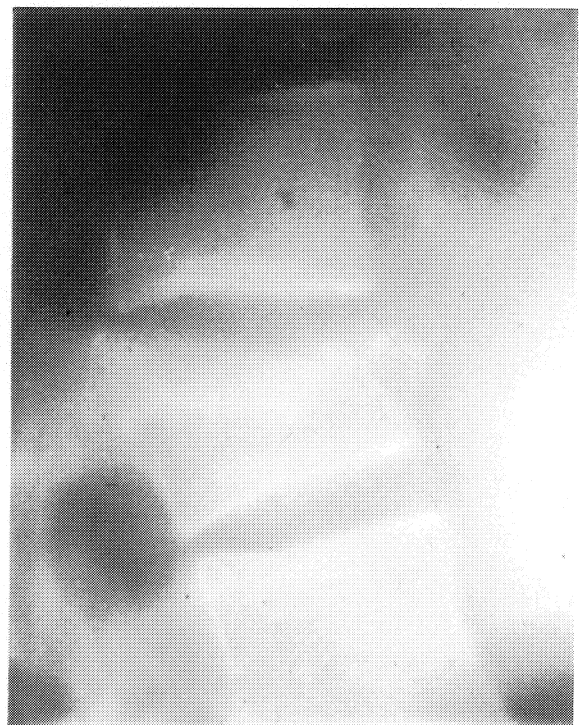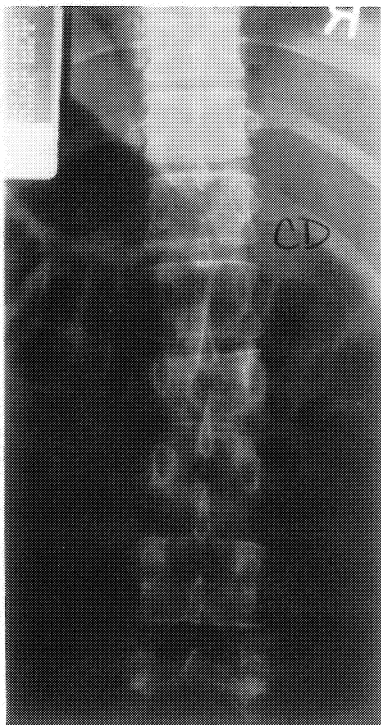
Figure 7-8
AP and lateral views of a burst fracture of L2. Neurological assessment Frankel Grade B.
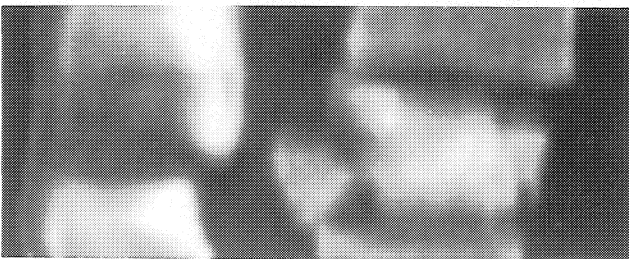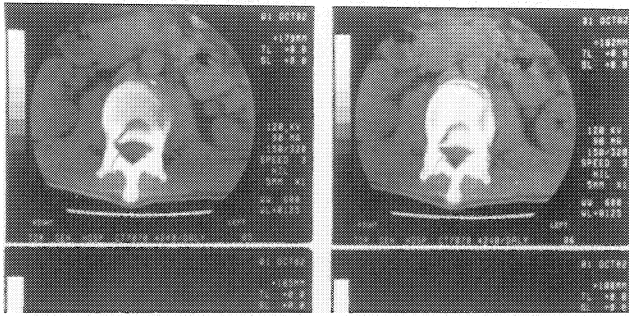
Figure 9-10
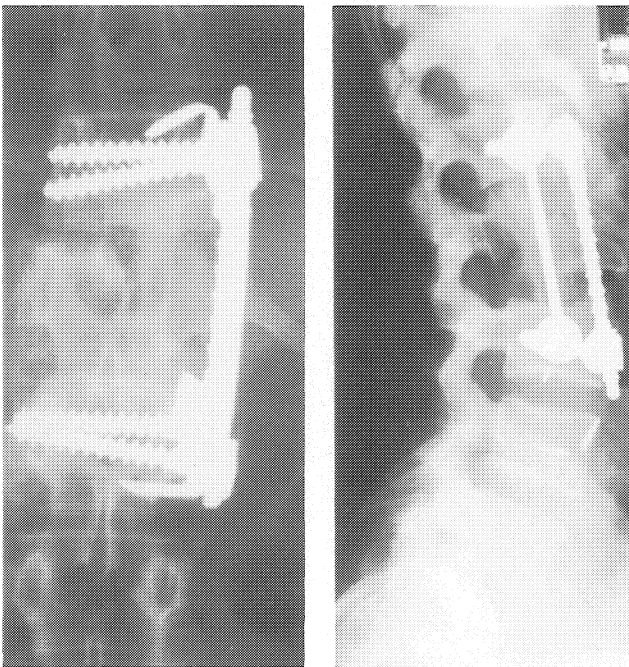CT scan with reconstruction demonstrates significant canal occlusion.

Figure 11-12
Postoperative AP and lateral demonstrate fusion at two years with good correction of deformity after anterior decompression, grafting and stabilization.
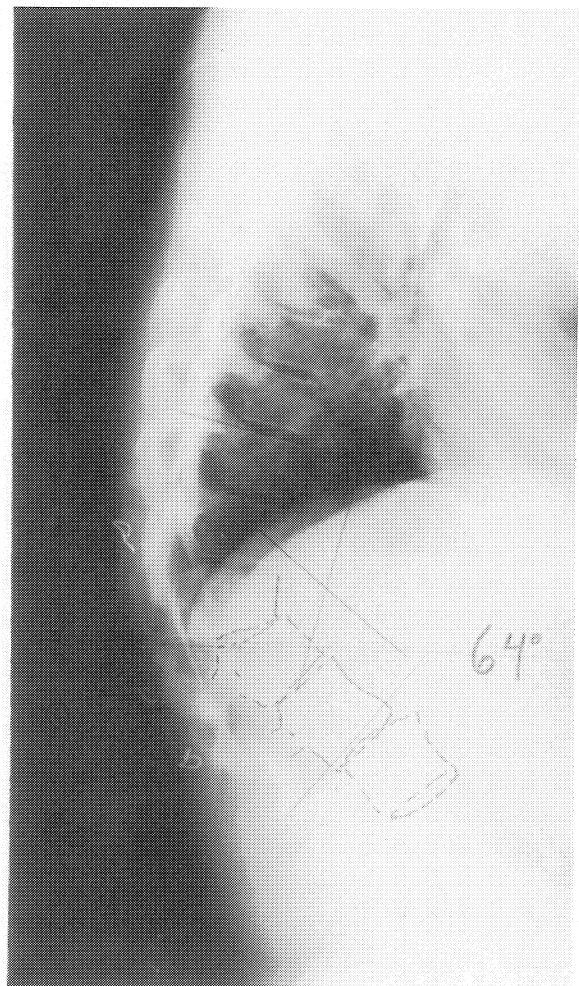
B) POST-TRAUMATIC KYPHOSIS

Indications

The indications for surgery included: pain and deformity which was present in all forty-five patients. Neurological lesions occurred in twenty-two patients, including ten with residual paraparesis from the original injury and twelve who developed slowly and progressively signs and symptoms of spinal stenosis.

Preoperative assessment included: CT scanning, metrizamide myelography, discography—above and below the fracture (four levels) and facet blocks (if no previous posterior fusion). Discograms and facet blocks are done in order to make sure all painful levels are incorporated in the subsequent surgery.

The technique is as with burst fractures. If CT scanning shows no or little canal intrusion the dura does not need to be decompressed. Previous posterior fusions do not need an osteotomy (Fig. 13-14).

Figure 13
Female age 34. Kyphosis post-traumatic — previous posterior fusion with no pain relief.
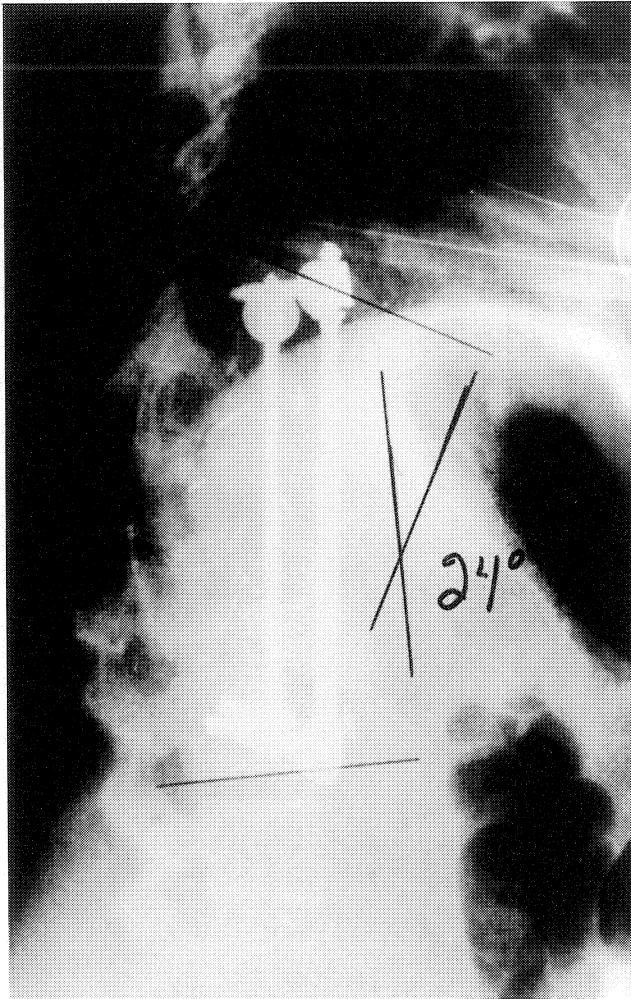
Figure 14
Postoperative two years with solid fusion. No loss of correction and pain relieved. Curve corrected from 64 degrees to 24 degrees without osteotomy of the posterior fusion.

Results

Screw breakage occurred in three of forty-five patients. Pain relief was good to excellent in thirty-seven of the forty-five cases. Neurological improvement can be seen twenty years after the initial fracture.

Neurological Recovery

Of the ten residual paraparetics four improved more than one grade on the Frankel scale after decompression. All spinal stenoses improved after decompression.

C) SCHEUERMANN'S KYPHOSIS

Studies of the surgical treatment of Scheuermann's kyphosis by posterior instrumentation indicate a progressive loss at long term follow-ups. Loss of correction is due to: 1) fusion on the tensile side of the spine, 2) high pseudoarthrosis rates (up to 40 percent)[4] and 3) late stress fractures due to repetitive cyclic loading on the tensile side of the spine. Anterior interbody fusion alone does not provide an adequate correction of the deformity either.

The relatively unsuccessful technique of posterior instrumentation and fusion with high pseudoarthrosis rates, complications and subsequent surgical procedures due to loss of correction lead to the development of our technique of anterior instrumentation and interbody fusion. The general aim is to stabilize and correct the kyphotic deformity by a mechanically sound procedure, with minimal immobilization, with minimal complications, that will not deteriorate within or out of the fusion mass. The biomechanical basis for this lies in providing axial distraction and reducing bending moments and producing a fusion mass which is: 1) under compression, 2) long, 3) far from the neutral axis, and 4) interbody.

Surgical Indications

Surgical indications in the skeletally mature are: 1) pain (apical or low back), 2) deformity (75 degrees or greater), 3) spinal cord compression (rare) and 4) progression of kyphosis. In the skeletally immature the indications for surgery include: 1) failure of bracing techniques, and 2) for deformity of 65 degrees or greater.

Surgical Technique

For thoracic deformities a thoracotomy via the fifth or sixth rib is performed. The scapula is mobilized. If there is an associated right thoracic scoliosis a left sided approach is preferred. The segmental vessels are clipped and the spine is exposed from T3 to T12 (or where appropriate). All discs and end plates back to the posterior annulus are removed. Rachet and collar-ended screws are inserted. The screws must pierce both cortices of the body and are placed as far posteriorly as possible. The rods are inserted and may be contoured. Distraction of both rods is carried out concurrent with application of manual pressure posteriorly. C-clamps are used to secure the rods. Bicortical iliac crest grafts are inserted under compression. The grafts should be slightly larger than the interspace. Supplementary rib graft is also used. In osteoporotic bodies, cement may be used to hold the screws in place. Postoperatively patients were immobilized in a plastic orthosis for six months. The average hospital stay was twelve days.

Early Results

The preliminary early results of anterior interbody fusion and modified Kostuik-Harrington anterior instrumentation from 1982 to 1987 included thirty-six patients with an average age of 27.5 years. The preoperative curve averaged 75.5 degrees. Postoperatively the average curve

was reduced to 56 degrees with instrumentation. After follow-up the average curve was 60 degrees (Fig. 15-16). Six patients had progression of their curves within their fusions. One patient underwent subsequent surgery and one patient had a pseudoarthrosis. The complications were minimal and included four screw fractures.
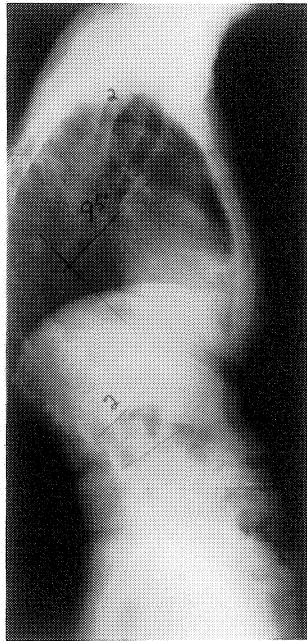

Figure 15   **Figure 16**

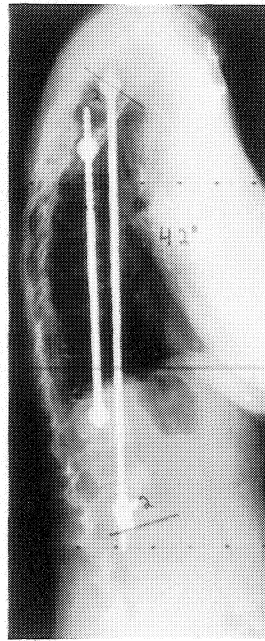**Preoperative, female age 51. Scheuermann's kyphosis 95 degrees (T2-L2). Progressive deformity and pain.**

Postoperative — solid union at two years. Kyphosis is physiologic (T2-L2 = 42 degrees).

Conclusions

The preliminary results of anterior distraction and interbody fusion indicate minimal loss of correction, minimal complications with minimal morbidity and low hospital stay. This is attributed to the fusion mass being under compression. Anterior instrumentation prevents collapse of interbody grafts. Preliminary results indicate that anterior instrumentation and fusion appear to yield satisfactory results.

D) ACUTE RIGID KYPHOSIS

Moe, Winter and Bradford[7] have clearly outlined indications and a treatment protocol for the treatment of acute angular kyphosis of either a congenital, developmental or infectious nature. General principles in the treatment of acute angular kyphosis consist of decompression of the neural canal, traction for correction of deformity if indicated, strut grafting anteriorly, and supplementary posterior fusion with instrumentation. The use of anterior

instrumentation has decreased the need for posterior instrumentation and fusion in the treatment of this problem. The principles of decompression and strut grafting, plus or minus traction when indicated, remain the same.

We prefer to use iliac crest strut grafts, substantiated with rib grafts rather than fibular grafts. Fibular grafts take up to two years to revascularized and are quite weak, particularly at their ends six months following implantation. Iliac grafts revascularized quickly. Bicortical grafts are generally used in treatment of all forms of kyphosis, but tricortical grafts may be used in the presence of osteoporosis.

E) SURGICAL LOSS OF LUMBAR LORDOSIS

Posterior instrumentation (Harrington) to L5 and S1 may result in excessive loss of lordosis. Pseudoarthrosis rates for fusion to the sacrum in adults with Harrington rods are as high as 50 percent and may lead to loss of lordosis. Pseudoarthrosis rates in adolescents with fusions to L5 or S1 are 12 percent with Harrington rods and may lead to loss of lordosis[5]. Loss of lordosis (flat back) occurred in 50 percent of posterior fusions to the sacrum in adults and was significant in half of these[4]. Children with flat backs develop problems as adults as they no longer can compensate for the loss of lordosis after age thirty-five. Frequently the development of degenerative changes below a previous fusion ending at L3-L4 or L5, as shown by Cochran and Nachemson, may lead to loss of anterior disc height and loss of lordosis and a flat back.

The prevention of iatrogenic lumbar kyphosis or flat back syndrome can be achieved with attention to strict detail if fusion to the sacrum is necessary. The use of prebent Harrington rods together with sacroiliac R hooks or midline hooks is suboptimal, since distraction is still necessary. When fusion to the sacrum is necessary, segmental wiring with Luque contoured rods into lordosis has helped and has decreased the pseudoarthrosis rate. However, the incidence of pseudoarthrosis remains too high.

In mobile curves, anterior Zielke instrumentation followed by a second stage posterior pedicle fixation from L3 to the sacrum will assure correction, fusion and preservation of lordosis. In rigid curves, especially kyphoscoliosis, the procedures of choice are: 1) multiple level anterior discotomies, filling the disc spaces with morsalized bone graft followed by, 2) a second stage posterior Cotrel-Dubousset instrumentation and fusion in order to derotate the spine and restore lordosis, performed ten to fourteen days after the first stage.

Materials

This study consists of a retrospective review of fifty-six scoliotic patients, four of whom were female and fifty-two were males. The average age was forty with a range

of fifteen to sixty. Of the fifty-six patients, forty-five were idiopathic. The number of previous operative procedures was twenty-seven. Previous posterior instrumentation extended to L4 (four), to L5 (five) and to S1 (forty-seven). The interval between previous surgery to osteotomy was 4.7 years (range: one to seventeen years). Nine patients had an associated posterior pseudoarthrosis, prior to osteotomy. Previous anterior fusions had been done in eleven patients.

Surgical Technique

A combined single stage posterior and anterior approach is used incorporating two incisions, flank and posterior. Incisions are joined if a quadrilateral wedge removal is required. An anterior osteotomy in the presence of a previous fusion is done. Alternatively, the disc and end plates at the selected level are removed (usually L3-4 or at the same level as a preexisting pseudoarthrosis). The anterior instrumentation is inserted (Figs. 17-20). The posterior osteotomy is done with 1.0 to 1.5 cm of bone removed. Posterior instrumentation consisting of Dwyer screws and cables placed in the fusion mass lateral to the dura is used. The anterior osteotomy is opened with the anterior Kostuik-Harrington system simultaneously as the posterior osteotomy is closed with the Dwyer system. An iliac crest bone graft is applied anteriorly in the open wedge. A contoured neutralization plate is applied centrally posteriorly for rotational control together with a posterior bone graft.
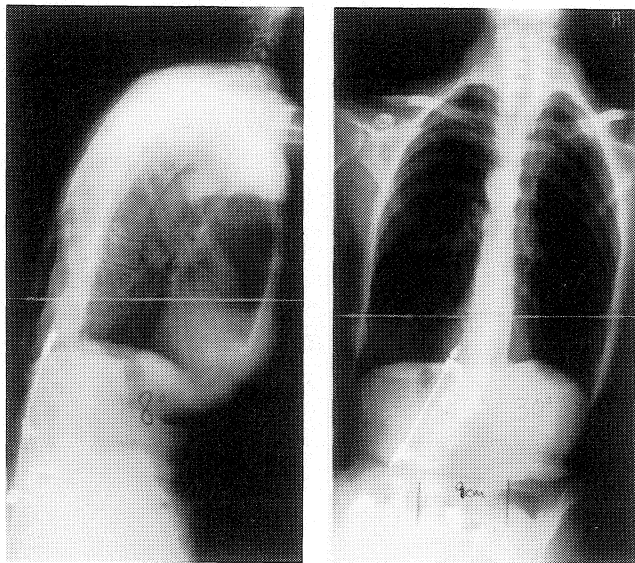

Figure 17-18

Female age 41. Pre-correction painful lumbar kyphosis—iatrogenic to lumbar. Distraction—distraction rod has been removed. Note imbalance in AP plane of 9 cm. Lordosis (L1-S1) measures 8 degrees.
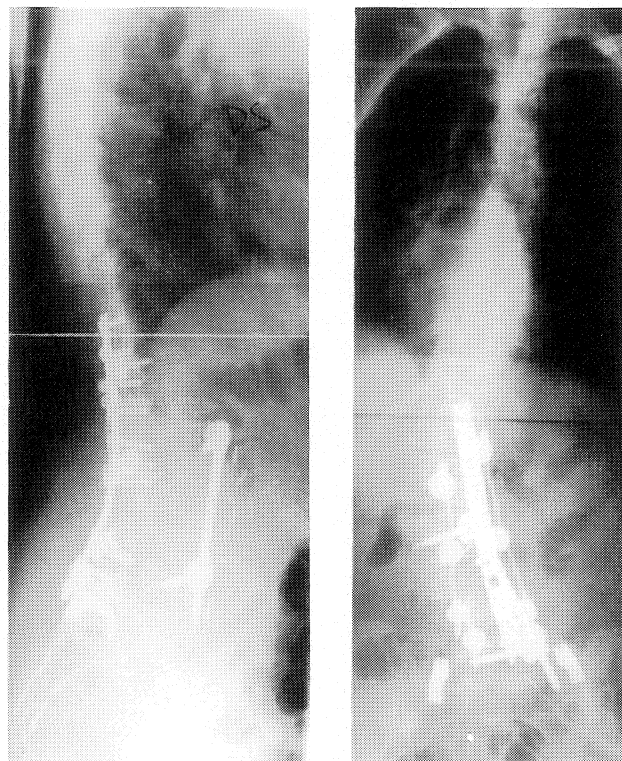

Figure 19-20

Post-osteotomy — note: restoration of lordosis with complete relief of pain. Lordosis measures 37 degrees. Balance has been restored in the AP plane.

RESULTS

Preoperative lordosis prior to initial surgery measured from L1 to S1 averaged 49 degrees. Prior to surgical correction the range was 10 degrees of kyphosis to 42 degrees of lordosis with an average of 21.5 degrees following osteotomy. At a minimum of two years follow up, the average lordosis was 49 degrees with a range from 5 to 78 degrees. Bone union occurred in all cases. Pain relief was obtained in forty-eight of fifty-six patients.

Major complications included one death. There were three intraoperative hemorrhages secondary to major left common iliac vein tears. All three had undergone previous anterior surgery. Two neurological complications occurred, one patient had persistent loss of bowel and bladder function. Three patients lost partial correction due to partial anterior graft collapse.

CONCLUSIONS

Anterior Kostuik-Harrington instrumentation has been used in a total of 279 cases anteriorly with a minimum morbidity. The wide range of application, ease of adaptability, and versatility has modified treatment of acute and chronic kyphotic deformities of the spine. The use of posterior instrumentation and fusion, we feel, is rarely necessary in the presence of anterior instrumentation.

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CT ANALYSIS AND CLASSIFICATION OF INTRA-ARTICULAR CALCANEOUS FRACTURES

J. L. Marsh, M.D.
Assistant Professor
Department of Orthopaedic Surgery
University of Iowa Hospitals and Clinics
Iowa City, IA 52242

J. V. Nepola, M.D.
Assistant Professor
Department of Orthopaedic Surgery
University of Iowa Hospitals and Clinics
Iowa City, IA 52242

Intra-articular fractures of the posterior facet of the calcaneous are difficult problems to treat and frequently lead to long term disability. In a long term follow-up of non-reductive treatment by Pozo[6], over seventy-five percent of patients who sustain this injury have a flat broad valgus heel and twenty percent will have a permanent limp and are disabled from work.

Attempts to alter this prognosis have developed advocates of both closed and open reductive methods. The analysis of pathologic anatomy and the results of reductive maneuvers have been hampered by difficulty in defining the complex three-dimensional anatomy of the calcaneous and its intra-articular facets radiographically. The routine measuring of Bohlers[2] and Gissanes[4] angles on the lateral view add little to the understanding of the pathology. An axial and oblique views as recommended by Athmonsen[1] and Broden[3] may show the major fracture lines but these views are difficult to obtain and often difficult to interpret. Several authors have recommnded the use of the CT scan in the evaluation of intra-articular fractures of the calcaneus and this method is now widely employed[5,7]. The purpose of this paper will be to discuss the normal and the pathologic anatomy of the fractured calcaneus as demonstrated by CT scanning. It will first be necessary to review the location of the major fracture lines.

Major Fracture Patterns and Mechanism of Injury

The primary fracture line in intra-articular fractures of the calcaneus is always the same. The medial wall of the calcaneus beneath the medially projecting sustentaculum is concave and allows passage of the neurovascular structures and the flexor hallucis tendon. The sustentaculum supports the middle facet which is anterior and medial to the larger posterior facet. This medial overhang of the sustentaculum, when subjected to axial load, produces a sheering force through the body of the calcaneus separat-

ing the sustentaculum from the remainder of the calcaneus and forms the primary fracture (Fig. 1A). This fracture line usually includes a variable portion of the posterior facet with the sustentaculum. The primary fracture is always present and is the key to understanding intra-articular calcaneus fractures.

The secondary fracture is a joint depression fracture and involves the portion of the posterior facet left intact with the tuberosity fragment (Fig. 1B). It is depressed by impaction with the lateral process of the talus and may include a projection posteriorly through the tuberosity. This forms a "tongue type" fracture as defined originally by Essex-Lopresti[4]. Lateral wall comminution and expansion to a variable degree accompanies this joint depression.

These two major fracture lines produce the following deformities of the calcaneus (Fig. 1C). 1) The sustentaculum which remains intact and attached to the talus through the talo-calcaneal interosseous ligament is driven towards the plantar surface of the foot. This produces loss of vertical height of the calcaneus. 2) Separation of the primary fracture line between the sustentaculum and the tuberosity, produces widening of the calcaneus in the medial-lateral direction. This is further aggravated by the lateral wall comminution. 3) The secondary fracture combined with the primary fracture produces posterior facet intra-articular incongruency. 4) The tuberosity fragment is usually tipped into varus but the widening of the heel produces an overall valgus appearance. 5) There is usually some posterior displacement of the sustentacular fragment on the tuberosity fragment, producing decrease in length of the calcaneus in the longitudinal axis of the foot.

These deformities are all relatively constant and mostly a consequence of the primary fracture between the sustentaculum and tuberosity. The intra-articular incongruity will depend on the exact location of the primary fracture line in relation to the posterior facet and on the degree of depression in the secondary fracture. As can be seen in

Fig. 2, a diagramatic view of the superior surface of the calcaneus, the primary fracture line can occur anteriorly, leaving the entire posterior facet with the sustentaculum. Most commonly the primary fracture line occurs through the posterior facet[8].

CT Anatomy of the Normal Calcaneus

CT scans done in thirty degrees semicoronal and transverse planes are recommended. The middle and posterior

facets form sixty degree angles with the longitudinal axis of the foot and the calcaneus and therefore a thirty degree semicoronal scan is perpendicular to the articular surface of these important intra-articular structures (Fig. 3). With this technique, the facets are shown on the maximum number of cuts, and the length of the body of the calcaneus as well as the major fracture line are more clearly demonstrated than using straight coronal sections which may miss the salient pathology.
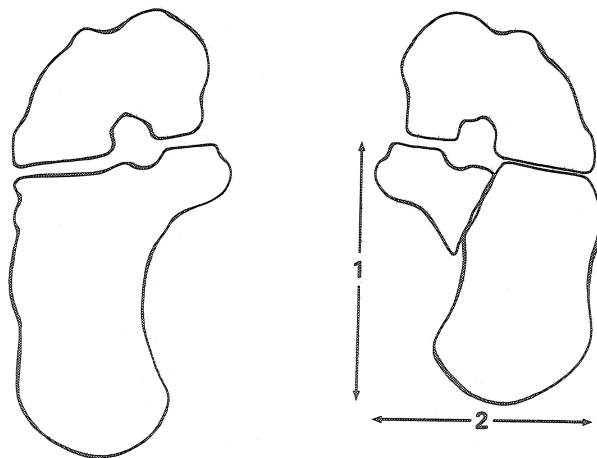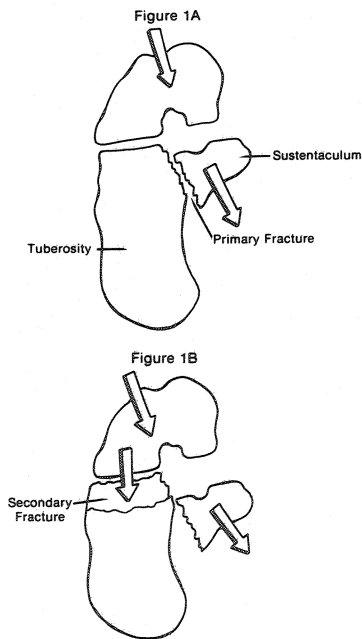

Fig. 2
Schematic view of the superior surface of the calcaneus shows the primary fracture can be (1) anterior to the posterior facet (Type I) (2) through the posterior facet (Type II) or (3) posterior to the posterior facet (Type III).

Fig. 1A-C
(A) Diagram of the primary fracture caused by axial force through the talus to the overhanging sustentaculum. (B) The secondary fracture formed by impaction of the posterior facet by the lateral process of the talus. (C) The primary fracture causes (1) loss of height and (2) increase in width of the calcaneus compared to the opposite side.
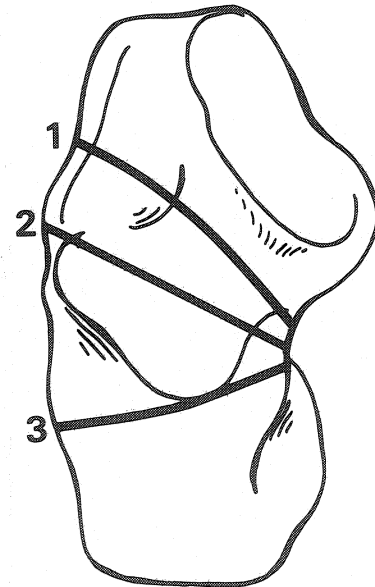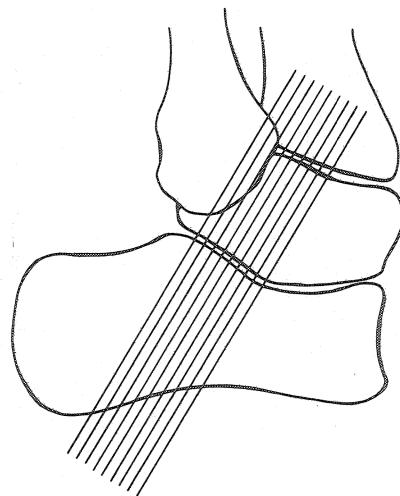

Fig. 3
The ideal scanning plane is perpendicular to the posterior facet and the primary fracture line.

A series of cuts from posterior to anterior is demonstrated in Fig. 4 with the relevant anatomy. The most posterior cut (Fig. 4A) shows the tuberosity, the back of the posterior facet and the relationship of the fibula to the lateral wall of the calcaneus. Fig. 4B demonstrates the medial-lateral width of the mid-portion of the posterior facet. Fig. 4C shows the middle facet and posterior facet on the same scan. The middle facet is on the sustentaculum and forms the superior aspect of the concave sweep of the medial wall. The medial wall contains more dense subchondral bone than the lateral wall. Fig. 4D, showing a cut anterior to the posterior facet, reveals only the middle facet and sustentaculum.
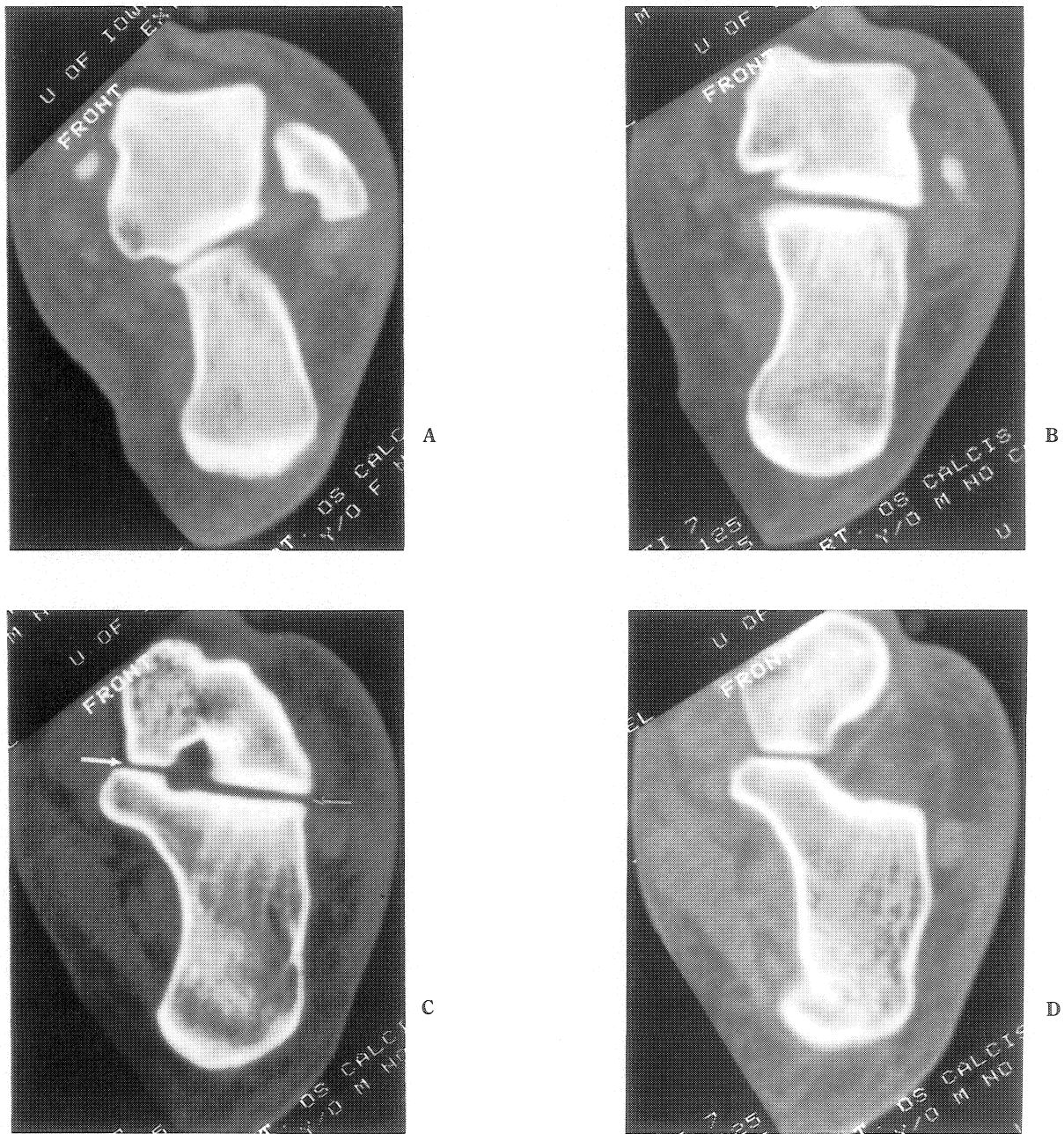

Fig. 4A-D
Successive thirty degree semicoronal cuts of a normal calcaneus from posterior to anterior showing (A) the posterior aspect of the posterior facet and fibular relation to the lateral wall. (B) The middle of the posterior facet. (C) The middle facet (white arrow) and posterior facet (black arrow) on the same image. (D) The middle or sustentacular facet anterior to the posterior facet. Note the concave medial wall with dense subchondral bone, the overhanging sustentaculum and the thinner convex lateral wall.

The transverse plane is perpendicular to the calcaneal-cuboid joint and is the best for demonstrating this articulation (Fig.5). Note again the thinness of the lateral wall compared to the medial wall and the slight concavity of the medial wall. Here also the length of the calcaneus in the longitudal plane of the foot is well demonstrated.
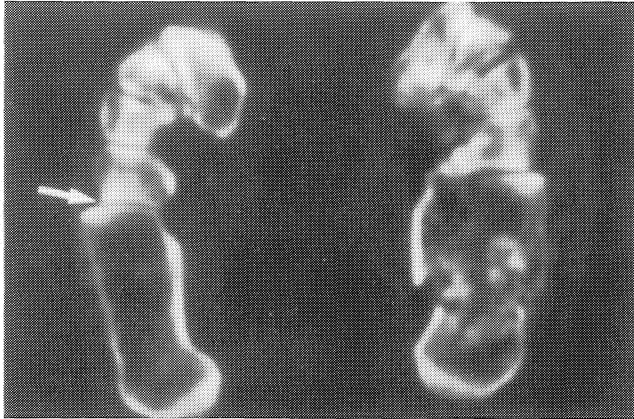

Fig. 5
The transverse scan is less useful but demonstrates the calcaneal-cuboid joint nicely (arrow).

Pathologic CT Anatomy of the Fractured Calcaneus

The major fracture line is easily identified on the thirty degree semicoronal scan and is seen to separate the calcaneus into two major fragments: the sustentacular and the tuberosity fragments as seen in Fig. 6. The fracture line consistently runs from the concave section of the medial wall in a superolateral direction and usually enters the posterior facet. However, the position of the primary
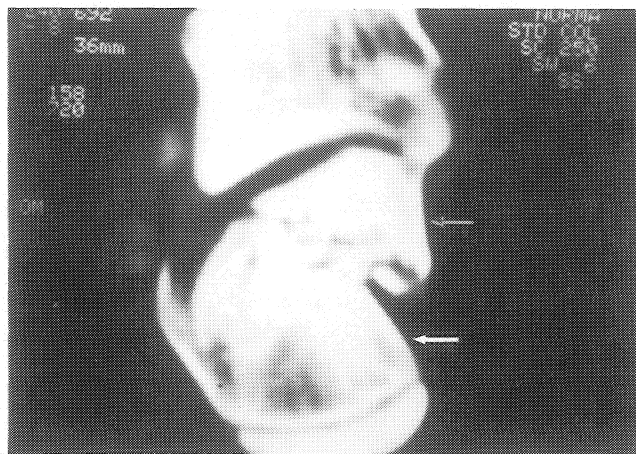

Fig. 6
The primary fracture divides the calcaneus into two major fragments, the sustentaculum (black arrow) and tuberosity (white arrow).

fracture line in relation to the posterior facet can vary. Fig. 7A is a badly comminuted fracture, but the primary fracture line passes behind the posterior facet which remains intact with the sustentacular fragment. In Fig. 7B, the primary fracture is anteromedial to most of the posterior facet which is depressed by the secondary fracture. It is
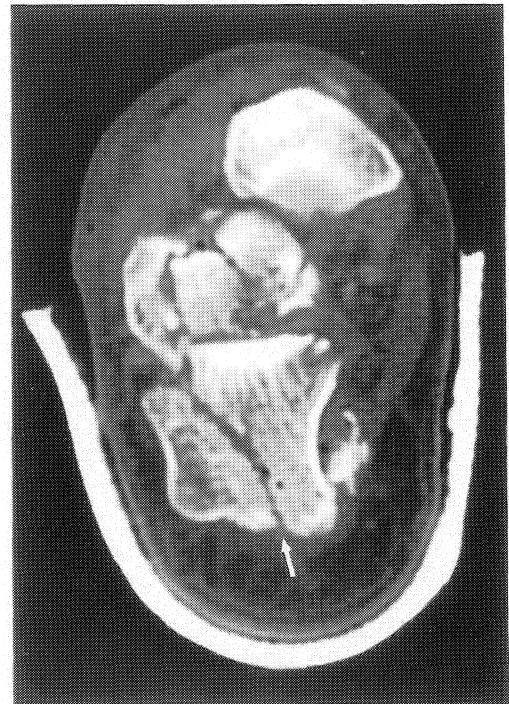
A

B

Fig. 7A-B
(A) The entire posterior facet remains intact with the sustentaculum anterior to the primary fracture line (arrow). (B) The primary fracture line (black arrow) passes anterior to the posterior facet which is depressed and rotated by the seondary fracture line (white arrow).

the location of both the primary fracture line and the secondary lateral facet depression and rotation that determines the congruency of the posterior facet as seen in Fig. 8.

Fig. 8
The primary fracture line is through the posterior facet with the lateral part of this facet (arrow) displaced and rotated by the secondary fracture.
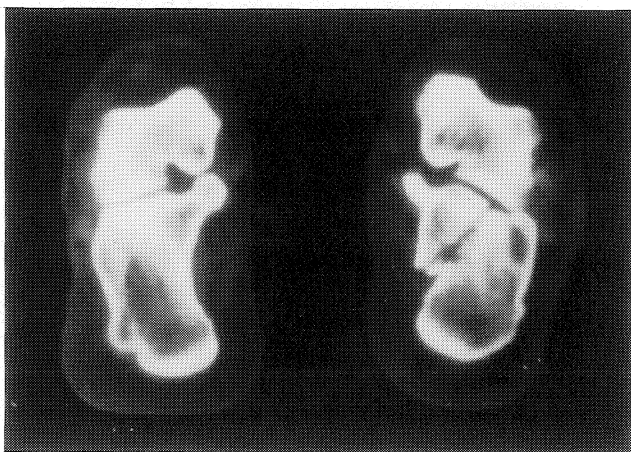

Fig. 9
The primary fracture causes shortening and widening of the heel (diagramatically shown on Fig. 1C). The secondary fracture causes posterior facet intra-articular incongruity.

The foot deformity is not caused by intra-articular incongruity but by loss of height and increase in width of the hindfoot. In Fig. 9 we can see these deformities compared to the opposite side. They are predominantly determined by the medial wall displacement between the sustentaculum and the tuberosity fragment. The loss of height of the heel is caused by the downward displacement of the talus and the attached sustentacular fragment and is easily measured on the medial wall. This lowers the fibula and contributes to peroneal impingement. There are two factors that contribute to widening of the heel. One is the lateral wall expansion, but more significant is widening caused by the diastasis between the sustentacular and the tuberosity fragments in the medial-lateral direction.

On the transverse plane scan (Fig. 5), the shortening of the longitudal length of the calcaneus is apparent as well as the loss of the concave sweep of the medial wall, the widening of the hindfoot and the lateral wall bulge. The intra-articular displacement in the area of the calcanealcuboid joint is well demonstrated.

SUMMARY

The CT scan gives simple accurate visualization of the major displacements of the fractured calcaneus. The primary fracture and its subsequent distortion on the shape of the hindfoot is easily identified. The secondary fracture lines and their relationship to intra-articular displacement of the posterior facet are likewise evident.

Studying the location of the primary fracture line in relation to the posterior facet on the thirty degree semi-coronal scan leads logically to a classificiation of intra-articular fractures. In Type I fractures the primary fracture line is anterior to the posterior facet which remains with the tuberosity fragment. In Type II fractures the primary fracture line is through the posterior facet. In Type III fractures the primary fracture line is posterior to the posterior facet which remains intact with the sustentaculum. Type I and II fractures can be further subdivided into A & B. In Types IA and IIA, there are no secondary fracture lines. The posterior facet congruency is determined entirely by the primary fracture. In Types IB and IIB, there is a secondary fracture producing depression of the posterior facet lateral to the primary fracture line. Fig. 8 and 9 are Type IIB fractures through the posterior facet with secondary lateral depression. Fig. 6 and 7A are Type III fractures. Note that the posterior facet remains intact with the sustentaculum. Fig. 7B is a Type IB fracture with an anteromedial primary fracture line but a major depression of the posterior facet caused by the secondary fracture.

This classification based on thirty degree semicoronal CT scans has relevance in planning treatment. If a reduc-

tive approach is chosen, planning is essential and greatly simplifed by studying the lcoation of the major fracture lines. Types IA, IIA, and III fractures are best reduced through a medial approach. Type IB and IIB usually require medial and lateral approaches for optimum reduction and fixation. Fig. 10A-E is an example of a Type IIB fracture reduced through combined medial and lateral approaches. The CT demonstrates the major medial wall displacement as well as the lateral posterior facet rotation and depression.

In future studies, with CT aided classification of fractures and measurement of degress of displacement, similar fractures can be grouped together and studied prospectively to determine the utility and indication for these reductive methods. In addition, CT analysis of experimental fractures will allow determination of the precise mechanism of each type of intra-articular fracture as defined here, and further validate this classification.

Figure A

Fig. 10A-E

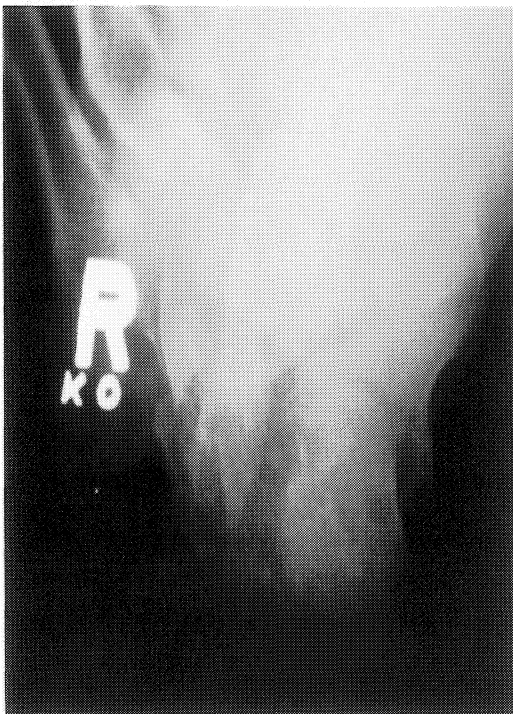(A and B) Lateral and Harris view of an intra-articular calcaneus fracture (C) 30 degree semicoronal scan demonstrates a Type IIB fracture with the primary fracture line through the posterior facet with a secondary fracture causing rotation and displacement of the lateral posterior facet. (D and E) Postoperative lateral and Harris views after ORIF with combined medical and lateral approaches.
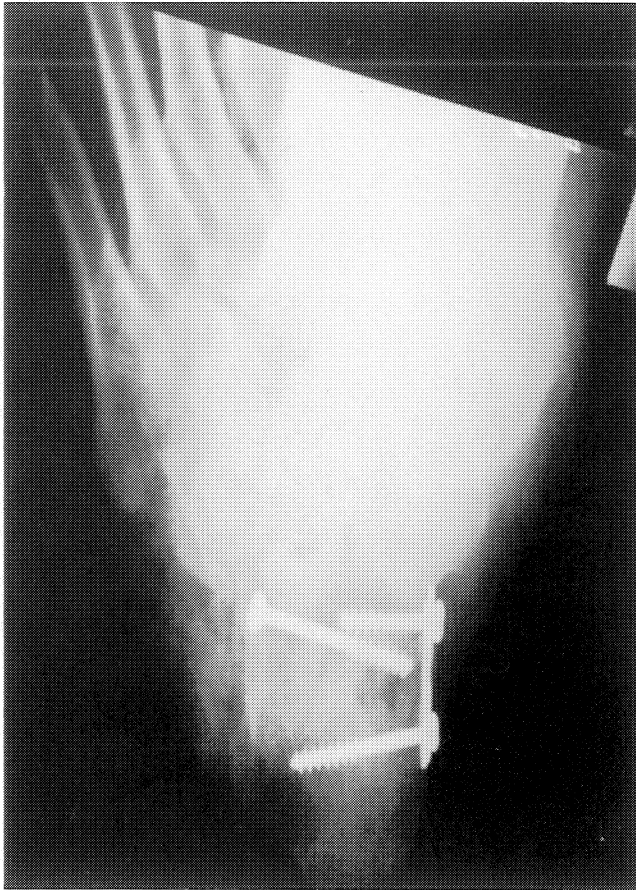

Figure C

Figure D

Figure 10E

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JOHANN FRIEDRICH AUGUST VON ESMARCH: HIS LIFE AND CONTRIBUTIONS TO ORTHOPAEDIC SURGERY

John E. Herzenberg, M.D., F.R.C.S.(C)
Instructor in Orthopaedic Surgery
University of Michigan Hospital
Ann Arbor, Michigan 48109-0328

INTRODUCTION

Orthopaedic surgeons make daily use of the Esmarch rubber bandage to create a bloodless field of surgery, enabling completion of complex extremity surgery. However, the rubber tourniquet which bears the eponym of Esmarch is but one of many contributions made by this man who was one of the most innovative and respected surgeons of his day. His remarkable personal life and professional accomplishments which served as an inspiration to his students at the University Kiel can equally inspire the modern day orthopaedic surgeon.

BIOGRAPHY

Johann Friedrich August von Esmarch (Fig. 1) was one of the last great surgeons of the nineteenth century German school. He was primarily a military surgeon and has been therefore called the "Ambroise Pare of Germany", after the sixteenth century French military surgeon. Born on January 9, 1823, in the small town of Tonning, on the west coast of Schleswig-Holstein, he was the son of a district surgeon. As a nine-year-old schoolboy, he dissected frogs, and accompanied his father on his rounds. His schooling was in Redsburg and Flensburg, though he was less than a model student. Esmarch was a proud German patriot, and participated in the struggle to free his native Schleswig-Holstein from Danish control.

He studied medicine at the University of Kiel and Gottingen and recived his medical degree from Kiel in 1848, passing the state examination with the highest grade. Esmarch graduated medical school during the era in which ether and chloroform were introduced into Germany, making surgery a vastly more attractive specialty for the young Esmarch. Upon graduation, Esmarch began working as an assistant to the great Professor Bernhard von Langenbeck. Langenbeck was recognized as the greatest surgeon of his time, and profoundly influenced Esmarch (Langenbeck later left Kiel for Berlin and founded the German Society of Surgery, the Archiv fur klinische Chirurgie, and trained most of the famous German surgeons of the nineteenth century).

Immediately following his graduation from medical school, war broke out between Denmark and Germany, and Esmarch began his career as military surgeon. In the initial
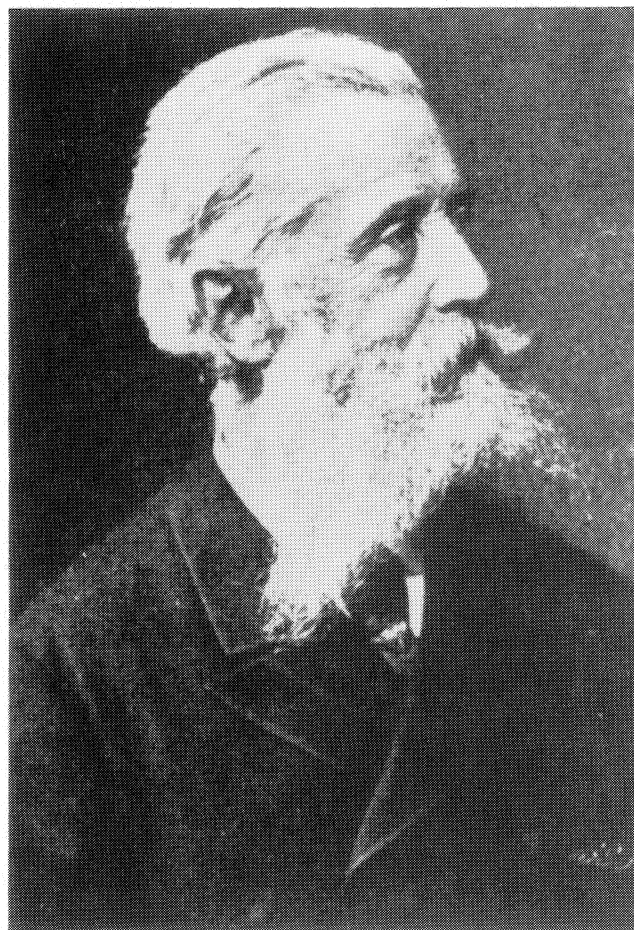
Fig. 1
Friedrich von Esmarch (from the British Medical Journal 1:719, 1908).

battles, he was a line officer (lieutenant), having joined with other young German patriots in secret arms training prior to the outbreak of hostilities. His talents, however, lay in his ability to provide expert and compassionate care for his wounded comrades. During the battle of Bau, while trying to control a severed brachial artery in a fellow German soldier, Esmarch was captured by the Danes. He was held on the prison ship "Droning Maria" for nine

weeks, until he was exchanged for a Danish doctor. During the wars of 1848 and 1850, he worked alongside Professors von Langenbeck and Stromeyer in the field hospital at Flensburg, his boyhood home. After the cease fire of Malmo, he set up a private practice in Kiel. Soon thereafter, he left on a study tour of the great European medical centers; Prague, Vienna, Paris, Brussels and London. Upon his return to the University of Kiel, Esmarch was appointed privat dozent (equivalent to associate professor).

Working alongside Esmarch in the struggle for independence from Denmark was Professor George Stromeyer, who succeeded Langenbeck as Chief of the Surgery at Kiel when Langenbeck was called to Berlin. Stromeyer was also a great military surgeon, but is most remembered for popularizing the operation of subcutaneous tenotomy. One of Stromeyer's more famous patients was William Little of England, who later described Littles' disease (cerebral palsy).

Esmarch's association with Stromeyer proved to be beneficial both personally and professionally, as he married Stromeyer's daughter in 1854. When Stromeyer left to become General staff doctor of the Hanover Army, Esmarch succeeded him as Director of the clinic. Although Esmarch was Chief of Surgery at the University of Kiel, the Danish Minister of Education would not give Esmarch the title of full Professor until three years later in 1857. Although this wait had to be frustrating for him, he spoke of it in typically lighthearted terms. When told that he was too young to be a full Professor, Esmarch responded by saying "that was a well known fault that would disappear with every year." Esmarch held the position of Director until his retirement in 1899. His marriage was a happy one, until his wife's death on May 30, 1870 after a severe, chronic illness. Their son Edwin von Esmarch, a bacteriologist, later became Professor of Hygiene at the University of Gottingen, 1899.

During the turbulent period from 1866 to 1870, war broke out again, and Germany fought first against Denmark, then Austria, and France. Esmarch served an important role in the Franco-Prussian war as consulting surgeon and public health officer supervising the military hospitals near Berlin (Fig. 2). In the later years of the campaigns, Esmarch was unable to serve in the field, as he was recovering from an illness he contracted while operating. In 1871, at age 48, he became Surgeon General of the German army. In 1872 he married his second wife, the Princess Henriett von Schleswig-Holstein-Sonderburg-Augustenburg, who was an aunt to the German Emperor William II. She had been a friend to Esmarch, but it was unusual in those days for a member of the royalty to marry a University professor. Henriett bore Esmarch at least one son, in addition to the two children from his previous wife. It was Emperor William who titled von Esmarch "Excellency" in 1899, when at the age of seventy-
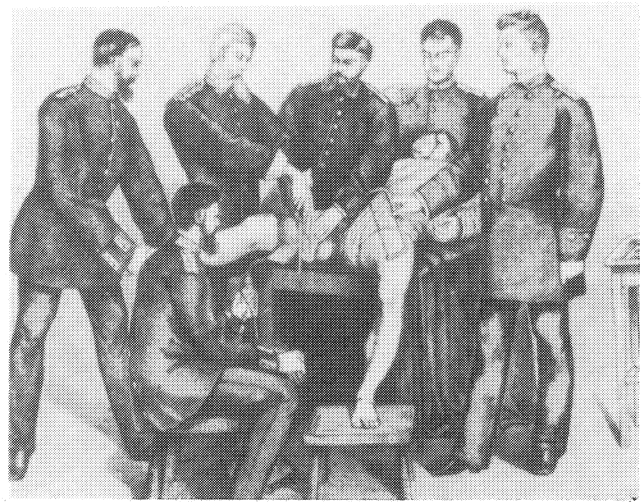

Fig. 2
Esmarch amputating a soldier's leg. Note the thigh tourniquet and the use of carbolic spray antisepsis. (From the Surgeon's Handbook on the Treatment of Wounded in War, 1878.)

six he retired from active practice. Nine years later, on February 23, 1908, he died of pneumonia following influenza. Even before his death, a statue was errected in his hometown Tonning to commemorate him.

Esmarch's household, across from the hospital, was graceful and comfortable, but not ostentatious, despite his marriage to royalty. His wife adroitly juggled her dual roles as royalty and dutiful housewife to the University professor. It was said that Esmarch was a great sportsman, mountaineer, and hunter with numerous trophies to his credit. His voice was low, and he spoke in short sentences. While not excelling as a public speaker, Esmarch was a spirited story teller in the company of small groups, both at home and at work.

Despite its small size and out of the way location by the Baltic coast, Esmarch built up the Kiel surgery clinic and attracted patients and students from far away. Esmarch was described as technically talented and very practical minded. His surgery was fast, confident, yet careful. In forty years of practice, Esmarch performed over twenty-thousand major operations. In addition to his technical prowess, he was recognized as an unusually insightful diagnostician. He was particularly adept at recognizing the orthopaedic manifestations of psychiatric illness such as hysteria and neurosis. Esmarch was loved by his patients, and had a gentle way with small children. Even patients with terminal illness found comfort in his kind manner.

Esmarch taught his students by demonstration rather than lecture, and trained them to be practical, effective physicians rather than helpless theorists. He maintained an extensive collection of pathological teaching material,

and spared no expense to have artists draw pictures of unusual cases. His monograph on elephantiasis was particularly well illustrated.

CONTRIBUTIONS

Esmarch was a prolific writer, publishing a large number of monographs dealing with military and trauma surgery. He was an early convert to Lister's antiseptic technique, after having visited England and Scotland. At the 1881 Seventh International Medical Conference, Esmarch spoke against the overly aggressive surgical approach to war wounds, despite the advances made in antisepsis. Esmarch felt that Lister's techniques reduced morbidity and mortality for elective civilian surgery, but not for battlefield surgery. It was around this time that the importance of wound debridement was being rediscovered by the Russian military surgeon Carl Reyher in the Russ-Turkish war. Ultimately, it was wound debridement and not antisepsis that proved to be the most critical factor in reducing mortality from war wounds to the extremities.

Esmarch urged against an overly invasive approach to gunshot wounds. He reviewed the medical treatments rendered to the U.S. President James Garfield after an assasination attempt. Esmarch concluded that the bullet, which shattered the twelfth thoracic vertebra, but did no damage to the spinal cord, should have been left alone. He wrote that the numerous wound probings by fingers, catheters, and instruments caused the abscess which eventually eroded the President's splenic artery and caused his demise eleven weeks after the original wounding.

During the Franco-Prussian war, Esmarch worked with the great pathologist Rudolf Virchow to further develop the battlefield hospital pavilion system, first initiated by the Americans in the Civil War. Esmarch pioneered the treatment of gunshot wounds to the extremities by conservative local resection of damaged parts, in contrast to the common practice of radical amputation of the entire limb. In 1851, following his experience in the Danish war, he published a treatise on "Resection of Bullet Wounds." In it, he described conservative resection of the injured part, as opposed to amputation of the entire limb. Esmarch was not, however, dogmatic in this approach, and he recognized the relatively good results obtained from amputation surgery.

Esmarch displayed deep concern for wounded soldiers, and became an innovator in battlefield first aid. His sense of humanity was revolted by the horrors of war, and he wrote a treatise "Against the Terror of War." In his typically pragmatic fashion, he channeled his concern for the misery and suffering of the wounded into efforts to teach lay people to render appropriate immediate first aid. He was particularly interested in preventing needless death from exsanguination before the physician arrives (Fig. 3).

FIG. 414. APPLYING A TOURNIQUET SUSPENDER

Fig. 3
Immediate first aid to the wounded -- a lifelong concern of Esmarch. (From Surgical Technic, 1901.)

As Surgeon-General, Esmarch required every German soldier to have an "Antiseptic Dressing Package". This is described in the German Military Sanitary Regulations of 1886 as containing, "two antiseptic muslin compresses 40 cm. X 20 cm., a cambric bandage 300 cm. X 5 cm., a safety pin, and waterproof material 28 cm. X 18 cm., for covering."

Esmarch also developed his "von Esmarch triangular cloth bandage", not to be confused with the Esmarch tourniquet bandage. This three-cornered bandage had detailed illustrations of wounded soldiers printed on the cloth itself (Fig. 4). The illustrations depicted its appropriate use on any part of the body, so that the soldier could properly apply the bandage according to the illustration. Some of the non-medical military authorities objected to distributing this graphic display of battlefield horrors to the soldiers, but Esmarch prevailed. A later modification of the arm issue first-aid package included Esmarch's triangular bandage, along with its pictures of six naked wounded soldiers, each with an Esmarch triangular bandage binding a different part of the anatomy (Fig. 5).

Esmarch published a pamphlet titled "First Dressings on the Battlefield" (1869) and later adapted his work to civilian life when he published a layman's handbook of "Early Aid in Injuries and Accidents" (1875). This was translated into English in 1883 by HRH Princess Christian and published in America. It was considered at the time to be a classic work, and was translated into twenty-three languages. In this pamphlet Esmarch explained his philosophy of first aid:
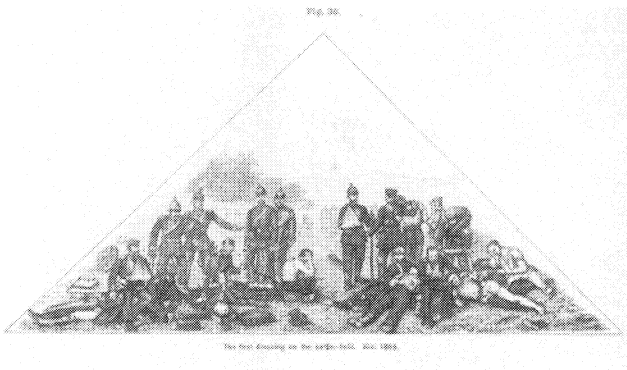
Fig. 4
Esmarch's triangular bandage for soldiers' first aid kits. (From Surgeon's Handbook on the Treatment of Wounded in War, 1878.)
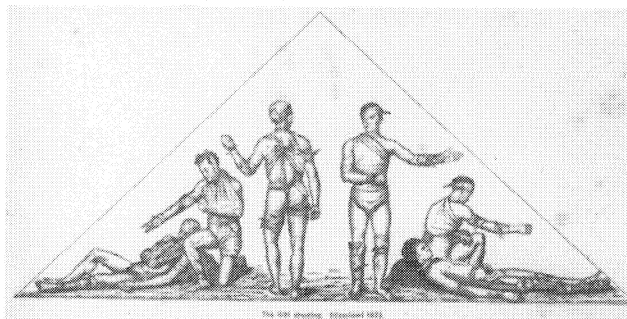

Fig. 5
Revised printing of Esmarch's triangular bandage; felt by the German military authorities to be less demoralizing than the previous version with the battlefield scene. (From Surgeon's Handbook on the Treatment of Wounded in War, 1878.)

"Though I have invited you here to teach you how to render first aid to the injured, I do not in the least aim at rendering a doctor's services unnecessary; on the contrary, I hope to convince you how important the immediate help of a doctor is in most cases. What I want to do is enable you to give the right kind of aid before the doctor arrives — without which, irreparable damage might be done, and perhaps even a valuable life be lost".

"When I look back at my career as a surgeon, I can with truth say that many and many are the times I have deplored that so very few people know how to render the first aid to those who have suddenly met with some injury . . . of the thousands who have flocked thither in their desire to help, so few have understood how to render aid."

"How many there are every year who die a miserable death, and who might have been saved by prompt aid, had anyone been near who knew how to give it."

Esmarch further devoted his energies to the founding of the Samaritan Movement in which first aid was taught to civilians from all walks of life. He was inspired by the order of St. John Ambulance Association which he had observed in London. On returning home, Esmarch gave some simple emergency medical lectures to the laymen of Kiel, and was overwhelmed by the enthusiastic response. There was some resistance from Esmarch's medical colleagues who feared that the laymen trained in first aid would be dabbling in medicine. Esmarch persisted however, and eventually there was a Samaritan School in virtually every village in Germany. Appropriately, they used Esmarch's "Textbook for Samaritans" as training material. This tradition is being continued in modern times in the form of the ever popular cardiopulmonary resuscitation courses.

Esmarch was particularly interested in teaching laymen how to control bleeding to prevent death from exsanguination. In addition to describing the various points where finger pressure would control a named artery, Esmarch advocated the use of tourniquets to control extremity bleeding. To this end, he advised every male citizen to wear specially designed suspenders that could be quickly removed and used as a tourniquet in the field.

Esmarch is most well remembered for his description of the "bloodless technique of surgery" employing the tourniquet that bears his name. He first presented his method to the Congress of the German Surgical Association in 1873. Esmarch very humbly and humourously related that hardly any of his colleagues at the Congress paid any attention to his paper, at least in partly due to the fact that Esmarch's paper was positioned on the program just before dinner. Within a year, however, his method was standard operating procedure throughout the hospitals of Europe. He published "The Art of the Bloodless Operation" (1873), and later devoted an entire chapter in his textbook "Surgical Technic" to the history and varieties of tourniquet control. This textbook was Esmarch's magnus opus, and for it he received a prize from the Empress August. In the second edition, it was expanded to three volumes and translated into several languages, including English.

The original Esmarch bandage was an elastic band 5 X 140 cm. made of pure India rubber which was wound around the limb from distal to proximal, overlapping one-half of each turn (Fig. 6). Esmarch noted that it is unnecessary to wrap toes and fingers individually. He makes the important point that local infection or malignancy are contraindications for the elastic bandage, proposing an alternative:

"Such parts as contain pus, sanious matter or soft tumor tissue, must not be bandaged, because thereby infectious matter might be pressed into the cellular tissue and the lymph channels. In such cases, the operator must be satisfied to hold the limb up perpendicularly for a few minutes until it has become visibly pale."
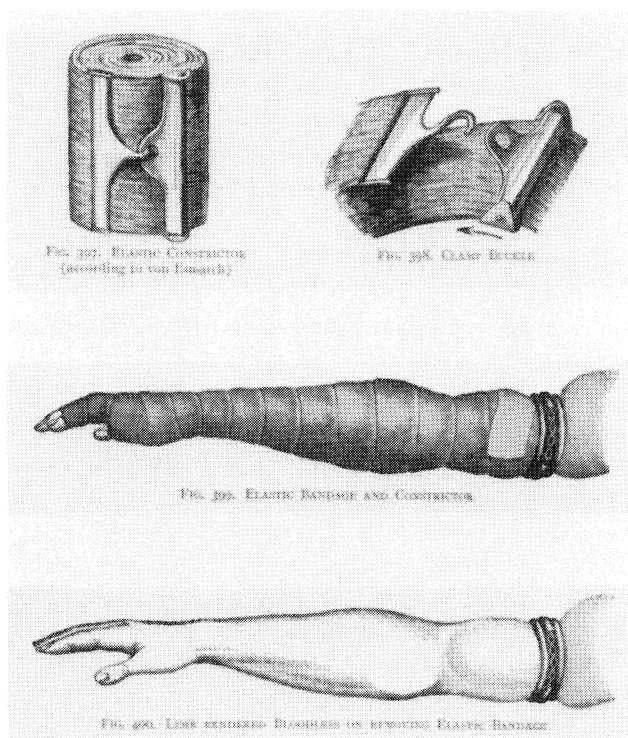
of course, most violent if the constrictor is removed slowly, because the blood immediately enters the arteries . . . but since it cannot return immediately through the veins, which are still compressed by the last turns of the bandage, venous congestion is likely to occur. Hence, it is necessary to remove the constrictor not slowly, but quickly".

Esmarch recommended that his bloodless technique be used for operations of the extremities, and even of the penis (Fig. 7). Although not in usage by modern day urologist, the technique of bloodless surgery has proved absolutely essential for extremity surgery.

FIG. 410. BLOODLESS METHOD USED IN OPERATION ON PENIS AND SCROTUM   FIG. 411. BLOODLESS METHOD IN HIGH AMPUTATION OF THIGH
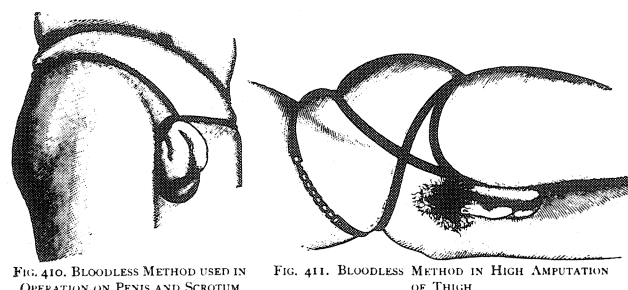
Fig. 7
Esmarch's bloodless technique for urologic surgery and thigh surgery. (From Surgical Technic, 1901.)

Fig. 6
Esmarch's elastic bandage for exsanguination; demonstrated on the upper extremity with a brass chain tourniquet. (From Surgical Technic, 1901.)

Interestingly, one of Esmarch's early case descriptions was the use of the exsanguinating bandage for a "necrotomy" for bilateral chronic osteomyelitis. Once rendered bloodless, a constrictor band was placed as high up as possible to prevent blood from re-entering the extremity. Esmarch used a variety of different constrictors, but today a mamometrically controlled blood pressure cuff is most frequently used. Esmarch advised that the constrictor could be left on for "several hours" safely, and even sites cases where the extremity was bloodless for "seven to twelve hours without resulting in gangrene or paralysis."

Esmarch recognized the well known phenomena of secondary hyperemia that follows removal of the constricting band, and proposed a remedy:

"When the constriction bandage is removed at the end of the operation, the limb, which until then presented a deadly pallor, turns as red as a boiled lobster, and a very considerable hemorrhage occurs in the wound, because the walls of the blood vessels were in a state of paresis and had become flaccid from the continued pressure upon the vasomotor nerves; hence, they allow more blood to pass through them than in their normal condition. The consequence is that the blood gushes forth from the operating wound as from a sponge. The hemorrhage is,

Esmarch had a variety of other contributions, including a treatise on chronic inflammation of the joints, on illnesses of the rectum and anus, on elephantiasis, on orthopaedic manifestations of neuroses, on the circulation of air within human dwellings, and the care of scrofulous children. He was an innovator, designed a face mask for ether administration, and numerous traction-splint devices (Fig. 8).

FIG. 225. VON ESMARCH'S PLASTER OF PARIS SUSPENSION SPLINT FOR RESECTION OF ANKLE JOINT

Fig. 8
One of the numerous traction-splinting devices that Esmarch invented. (From Surgical Technic, 1901.)

He was the first to describe intracranial epidermoid tumors, and was a pioneer in the use of cryotherapy (Fig. 9).

FIG. 63. ESMARCH'S COLD COIL
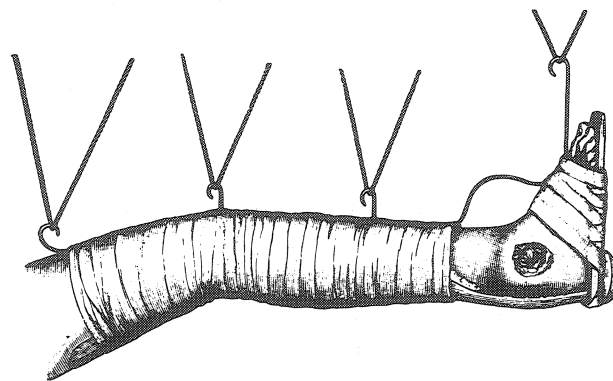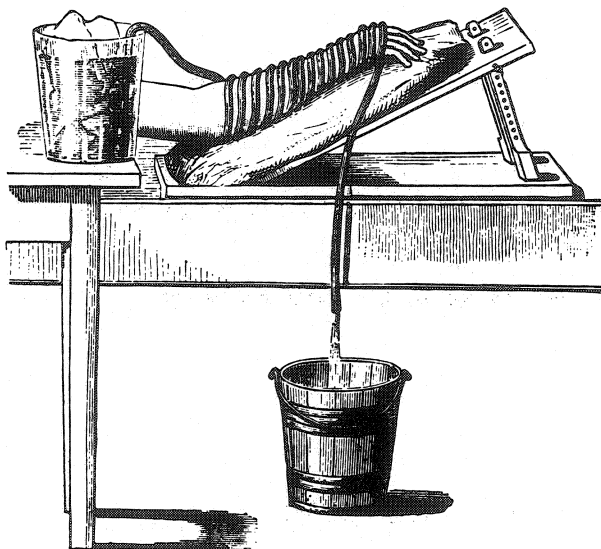
Fig. 9
Esmarch's Cold Coil for anti-phlogistic treatment of inflammation. (From On the Use of Cold in Surgery, 1861.)

Esmarch was proud but not boastful of his many accomplishments. His escutcheon, bestowed in 1887 by Emperor William I, included the Esmarch family arms, the Samaritan Cross, and a bloodless arm, exsanguinated by the Esmarch bandage. Indeed an appropriate collection of heraldic symbols for such a remarkable man who contributed so much to surgery, and to the compassionate and speedy care of the wounded.

ACKNOWLEDGEMENTS

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SYNOVIAL CHONDROMATOSIS OF THE HIP
WITH NORMAL PLAIN FILMS

From the Department of Radiology[1] and Orthopaedic Surgery[2]
The University of Iowa Hospitals and Clinics
Iowa City, IA 52242
Thomas J. Barloon, M.D.[1]
Raymond G. Harre, M.D.[1]
Georges Y. El-Khoury, M.D.[1]
Brian D. Adams, M.D.[2]

A case of synovial chondromatosis of the hip with normal plain films is presented. CT scanning may be helpful in detecting early phases of synovial chondromatosis when the diagnosis is strongly suspected on clinical findings and the radiographs remain normal. The pathologic findings are described and several disorders in the differential diagnosis are discussed.

CASE REPORT

This twenty-seven year old female presented with a nine year history of intermittent left hip pain that radiated into the upper thigh and increased with prolonged ambulation. The patient described occasional popping and locking of the left hip while walking. The patient denied any pain in the other joints. There was no history of significant trauma or inflammatory arthritis.

On physical examination of the hip there was limited range of motion especially in flexion and pain in the hip at extremes of motion. There was tenderness on palpation over the femoral head. Extreme abduction of the left hip elicited a loud popping sound. The following radiographic examinations were performed: anteroposterior (AP) view of the left hip (Fig. 1), computerized tomographic (CT) scan (Fig. 2), arthrogram (Fig. 3), and CT following the arthrogram (Fig. 4).

Arthrotomy revealed multiple round, white cartilaginous tissue fragments ranging in size from 0.2 to 0.5 cm. A synovial biopsy and histologic examination demonstrated multiple small areas of calcified cartilaginous material in the synovial membrane (Fig. 5). No significant cellular atypia was noted.

DISCUSSION

Synovial chondromatosis is a condition of metaplastic synovium with cartilaginous foci in synovial joints, tendon sheaths, and bursae. These metaplastic foci can become detached and form cartilaginous and osteocartilaginous loose bodies that are nourished by synovial fluid [1,3,6].

According to Milgram[4], synovial chrondromatosis is an evolving disease characterized initially by proliferation of the synovial membrane with no loose bodies. Subse-

quently, transitional lesions develop characterized by both active intrasynovial proliferation and free loose bodies. This phase is followed by multiple loose bodies without demonstrable intrasynovial disease.

In the initial phase of synovial chondromatosis, the radiographs may either be normal or reveal only soft tissue swelling about the affected joint. In Milgram's[4] series of eight patients in the initial phase of synovial chondromatosis, three patients failed to demonstrate any calcification on the preoperative radiographs. Therefore, the correct diagnosis may not be suspected from plain film studies even though symptoms may have been present for many years.

In the transitional cases with intrasynovial involvement and loose body formation, plain films may either demonstrate multiple loose bodies or remain normal. Milgram had nine patients in this second phase; four patients showed no demonstrable calcifications on plain films.

In the last phase with multiple free bodies due to synovial osteochrondromatosis, all patients demonstrated calcified loose bodies surrounding the involved joint. Surgical therapy consisting of removal of all accessible chondromata and subtotal synovectomy is usually curative. Recurrence is quite unusual, being reported in one of ten cases by Giustra et al.[2]. In their case, removal of the pathologic tissue was only partial. Cases of spontaneous regression have been reported.

The surgical findings in our patient most closely correlated with the second phase of synovial chondromatosis. Pathologically, both intrasynovial involvement and free loose bodies were present. Plain films of the hip remained normal even though the symptoms were present for nine years (Fig. 1).

The CT scan was especially helpful in our patient in demonstrating multiple intraarticular loose bodies of the hip (Fig. 2). The CT findings subsequently led to an arthrogram that demonstrated multiple intraarticular filling defects (Fig. 3)[6]. A CT scan immediately following the arthrogram confirmed the location of the loose bodies (Fig. 4).

Differential diagnosis must include other possible causes of loose bodies and intraarticular soft tissue masses. Frag-

mented osteophytes presenting as loose bodies in osteoarthritis are usually fewer in number and larger in size. The same distinction applies to the loose bodies in osteochondritis dissecans. In pigmented villonodular synovitis calcification is absent. Although calcification occurs in synovial sarcoma, this entity causes more bone destruction as opposed to the erosion sometimes seen in synovial chondromatosis. There have been reports of transformation of synovial chondromatosis into synovial chondrosarcoma, although this is rare. Neurotrophic arthropathy and postinfectious arthritis can give rise to intraarticular calcifications; however, their associated findings are not similar to synovial chondromatosis. Hemangiomas of the synovium or joint capsule occasionally produce intraarticular calcifications (phleboliths).

This case suggests that CT studies may be useful in the noninvasive detection of the early phases of synovial chondromatosis when the disease is suspected on clinical evidence yet the plain radiographs are normal.
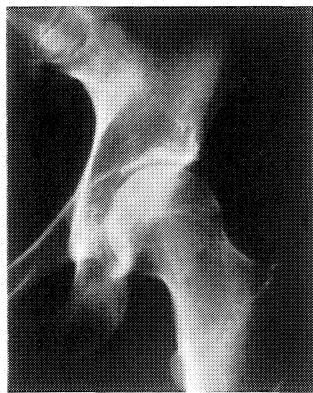
Figure 1
A plain anteroposterior radiograph of the left hip is normal.
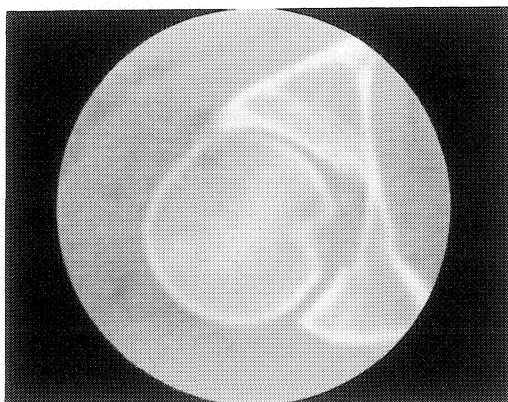
Figure 2
A CT scan without intraarticular contrast demonstrates several small calcified loose bodies.

Figure 3
A left hip arthrogram following the hip CT demonstrates multiple filling defects.
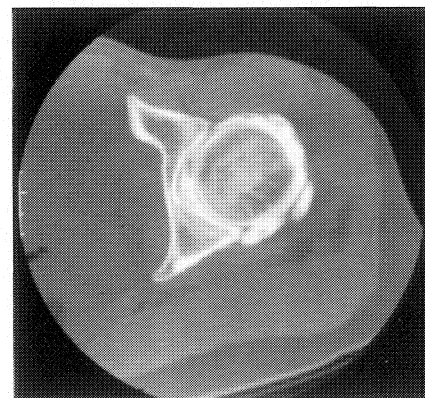
Figure 4
A CT scan with intraarticular contrast demonstrates several filling defects.
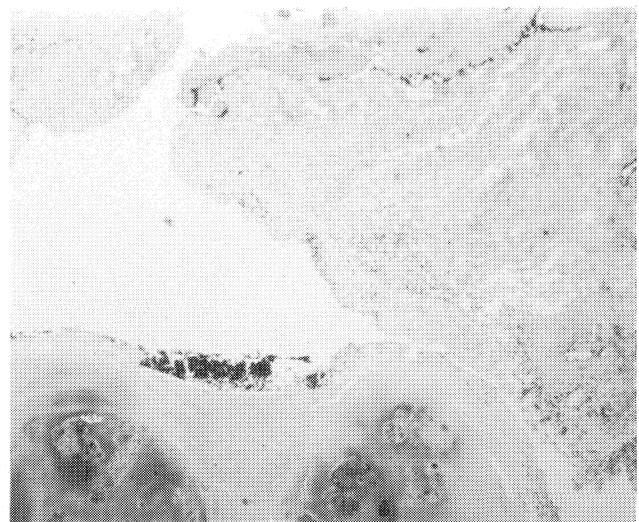
Figure 5
A synovial biopsy specimen (H & E stain 100X) demonstrates calcified cartilaginous material in the synovial membrane.

T. J. Barloon, R. G. Harre, G. Y. El-Khoury, B. D. Adams

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COMPARISON OF ENERGY COST AND GAIT EFFICIENCY DURING AMBULATION IN BELOW-KNEE AMPUTEES USING DIFFERENT PROSTHETIC FEET

David H. Nielsen, L.P.T., Ph.D.
Donald G. Shurr, L.P.T., C.O.
Jane C. Golden, L.P.T., M.S.
Kenneth Meier, C.P.

INTRODUCTION

Attaining efficient, upright, locomotion marks a milestone in the development of an individual[30]. Persons with acquired locomotor dysfunction such as lower extremity amputations spend significant time and effort attempting to regain their lost walking proficiency. In some of these indivudals ambulation is difficult and may not be feasible or practical. Important factors cited for ambulation failure are the relative high exercise intensity required and associated excessive energy cost. Even though the impairment may prevent completely normal walking, with appropriate treatment intervention, most lower extremity amputees can still achieve an efficient gait within the limits of their disability. For optimum gait efficiency, it is imperative that prosthetic devices keep energy expenditure to a minimum.

The gait of normal subjects has been extensively studied by means of motion and force analysis as well as energy cost techniques[5,13]. Comprehensive descriptive and analytical data concerning normal gait have been obtained. Gait in several categories of disabled subjects has also been studied, though less completely[11,17].

Results from available studies indicated that amputees walking with leg prostheses consume more energy than normals at comparable walking velocities[12,14,23,32]. Waters indicated that the increased energy cost was a function of the level of amputation[34]. Until the development of an energy storing design, the type of prosthetic foot assumed minor concern. The recent introduction of the Flex-Foot, a dynamic foot prosthesis appears to offer some advantages to the conventional prosthetic foot. Wagner's biomechanical analysis revealed improved ankle range of motion and gait symmetry for the Flex-Foot contrasted to the SACH foot[33].

Research is limited with no information available concerning differences in energy cost or efficiency of ambulation between these two types of prosthetic feet. The purpose of this study was to investigate differences in self-selected walking velocity, relative exercise intensity, oxygen consumption and gait efficiency in below knee amputees during ambulation with the Flex-Foot versus the conventional prosthetic foot.

METHOD

DESIGN

A two factor treatment by subjects repeated measures design was used in which all treatments were administered to each subject[15]. The two factors were walking velocity (graded walking speeds on a motor driven treadmill) and type of prosthetic foot (Flex-Foot versus conventional prosthetic foot). Self-selected walking velocity was also measured on each individual. This research is an ongoing study. Self-selected walking velocity data has been collected on seven subjects. Complete data collection has been obtained on three subjects.

SUBJECTS

The subjects of this study were healthy adult males with unilateral traumatic below-knee amputations (mean age = 26.7 ± 7.1 years, mean weight = 172.7 ± 33.0 pounds). All subjects had the Flex-Foot and a conventional prosthetic foot and were proficient walkers with both types of prostheses. In accordance with the Human Subjects Review Committee of the College of Medicine at the University of Iowa, informed written consent was obtained from each subject prior to participation in the study.

PROCEDURES

Three one hour sessions (one orientation and two test periods) on separate days were required of each subject. The initial session comprised of completion of paperwork, measurement of self-selected walking velocity and practice walking on the treadmill. The two following sessions involved the graded speed treadmill tests using the Flex-Foot and conventional prosthetic foot on alternate days with the treatment order randomized between days according to the type of prosthesis.

A 15 meter long segmental walkway was used for the self-selected walking velocity measurements. An electronic timer with portable lights and photoconductive switches was used to obtain time measurements. Five repeated time measurements were taken over the mid 5 meter section of the walkway at the end of five minutes of self-selected, steady state walking with each prosthetic foot. The mean of the five time measurements was used

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to calculate self-selected walking velocity.

In order to standardize walking velocity and to facilitate measurement procedures, the actual walking tests were performed on the treadmill. A progressive graded testing protocol with three to five minutes of walking at each of seven walking velocities (1.0, 1.5, 2.0, 2.5, 3.0, 3.5, 4.0 mph) was adopted. Physiological steady state heart rate and oxygen uptake measurements were used to calculate percent MHR (maximum heart rate), our criterion measure of relative exercise intensity. The oxygen uptake values were reported directly as well as used for the calculation of gait efficiency (ml 02/kg.m).

Heart rate was monitored by ECG radiotelemetry. The system consisted of three small disposable chest electrodes, a miniature radiotransmitter worn on a belt around the subject's waist, a remote FM receiver and a standard single channel electrocardiograph recorder which was connected to a digital cardiotachometer. Oxygen uptake was determined by the open-circuit method with a semiautomated on-line computer system. The method involved timed collection, volume measurement and electronic gas analysis of the subject's expired air. A printer connected to the computer provided typed summary tables of the oxygen uptake results.

RESULTS

SELF-SELECTED WALKING VELOCITY

Table 1 represents the results of the self-selected walking velocity tests. Included are the values for our sample of seven subjects as well as for the smaller group of three subjects. For comparison, the data reported by Waters are also included[34]. As the table indicates, the Flex-Foot values compared to the conventional foot were higher for both groups of our subjects with mean percent increases of 9 percent and 7 percent respectively.

Table 1

Self-Selected Walking Velocity

Subjects	Meters/Min (SD)	mph (SD)
Normals	74 — 83	2.8 -3.1
Traumatic BK Amputees		
Waters (1976) (N-14)	71	2.6
Conventional Foot (N = 7)	71.4 (15.8)	2.7 (.59)
Flex Foot (N = 7)	77.8 (16.9)	2.9 (.63)
Conventional Foot (N = 3)	80.5 (19.0)	3.0 (.71)
Flex Foot (N-3)	85.8 (22.5)	3.2 (.84)

ENERGY COST

As illustrated in Figure 1, the energy cost of ambulation increased systematically with increases in walking velocity. In all cases the oxygen uptake values for our three ampu-

tees were higher compared to normals[26]. Energy cost differences related to type of prosthetic foot were minimal at the low speeds. However, at walking speeds of 2.5 mph and above the energy cost of walking with the conventional foot was higher.
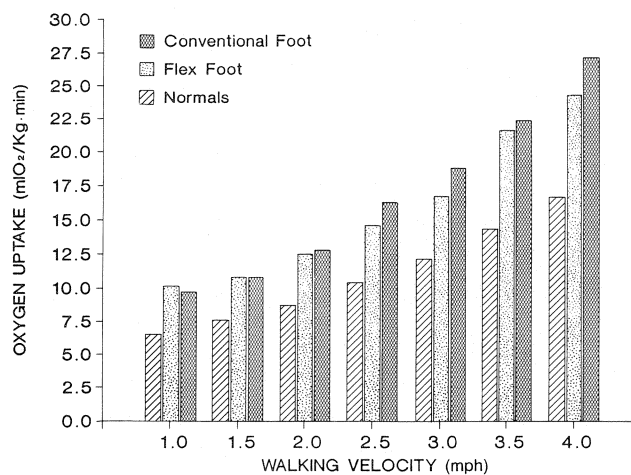
Figure 1
Energy cost differences of amputee walking based on type of prosthetic foot with comparison to normal values.

RELATIVE EXERCISE INTENSITY

As was expected, changes in percent MHR, our criterion measure of relative exercise intensity, mirrored the energy cost responses (Fig. 2). Exercise intensity increased systematically with increases in walking velocity. Again, between-prosthetic foot differences were greatest at the higher walking speeds with a maximum difference at 4.0 mph.
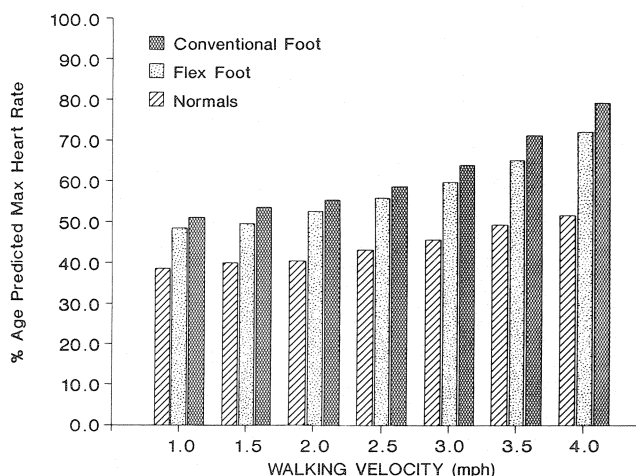

Figure 2
Relative exercise intensity of amputee walking based on type of prosthetic foot with comparison to normal values.

GAIT (DISTANCE) EFFICIENCY

As shown in Figure 3, gait efficiency for amputee walking paralleled the response curve for normal subjects[26]. In all cases the individual values of energy cost per meter traveled were upwardly displaced for amputee walking. Little difference in gait efficiency was observed between the two types of prosthetic feet at the slower speeds. For speeds equal to and above 2.5 mph the values for the Flex-Foot were generally lower.

DISCUSSION

As described in the company's product literature, the Flex-Foot is a dynamic prosthesis designed to store and release energy during the normal course of locomotion[31]. Accordingly, the fiberglass and carbon (graphite) pylon compresses during heel contact and extends during heel off increasing forward momentum during toe-push off. The company maintains that these energy absorption and releasing features should make walking and running with the Flex-Foot easier and theoretically should result in reduced energy comsumption.

In support of these claims, Wagner reported increased ankle range of motion and generally improved walking biomechanics for the Flex-Foot compared to the SACH foot[33]. Based on our search of the literature no definitive information is available concerning the energy cost of ambulation with the Flex-Foot.

Several investigations have shown that people spontaneously self-select an optimally efficient walking speed referred to as the self-selected or free-paced walking velocity[1,17,18,23,24,25,37]. The energy cost per meter traveled is higher for speeds slower and faster than the self-selected walking velocity (Fig. 3). For normal persons the

most efficient average self-selected walking velocity is approximately 80 meters/minute (3 mph) with a range from 74 to 83 m/min (2.8-3.1 mph)[11,26]. Persons with abnormal gait usually walk slower but, also, tend to select the most optimally efficient walking speeds[11,34]. This optimal speed, however, may not be possible if the total energy cost and relative exercise intensity are excessive.

Wagner indicated that self-selected walking velocity for both Flex-Foot and SACH foot ambulation was below normal values[33]. This was in agreement with our findings as well as other reports on below knee amputee walking[27,34]. The 71.4 meters/minute (2.7 mph) value for ambulation with the conventional prosthetic foot for our sample of seven subjects was essentially identical to the self-selected velocity reported by Waters, 71 meters/minute (2.6 mph)[34]. For our group of three subjects who were quite active and physically fit individuals the value was higher: self-selected walking velocity for the conventional foot was 80.5 meters/minute (3.0 mph).

In contrast to Wagner who reported no between prosthetic foot differences, our subjects produced higher self-selected walking velocities using the Flex-Foot compared to the conventional prosthetic foot[33]. Since Wagner did not report any numerical values, no specific comparisons could be made. Variations in testing protocols may help explain the inconsistency. Wagner's velocity measurements were determined from repeated individual, motion analysis walking trials. Our measurements were based on continuous steady state five-minute walking tests for which we have previously established within session and between session measurement reliability[28]. Between subject differences in physical fitness status could have been another contributing factor.

Research on the energy cost of walking with normal subjects in our lab as well as other studies has shown a curvilinear increase in oxygen uptake with increases in walking speed[2,8,26]. One could speculate that the altered biomechanics in amputee walking would produce corresponding changes in gait efficiency and subsequent elevations in energy cost. Accordingly, oxygen uptake for ambulation in our below-knee amputees was higher than normal as found in other studies involving amputee walking[12,14,34]. Increases in walking velocity tended to augment these differences. Expressed as a percentage above normal values the elevations ranged from 48 percent at 1.0 mph to 61 percent at 4.0 mph.

Of particular interest in the present study was the lower energy cost values observed at the higher walking velocities for Flex-Foot ambulation compared to conventional foot walking. The largest decrease was 2.5 ml 02/kg.min corresponding to a 10 percent difference occuring at 4.0 mph. These results suggest that the energy storing-releasing design characteristics of the Flex-Foot were of negligible consequence at slow walking speeds, but at speeds
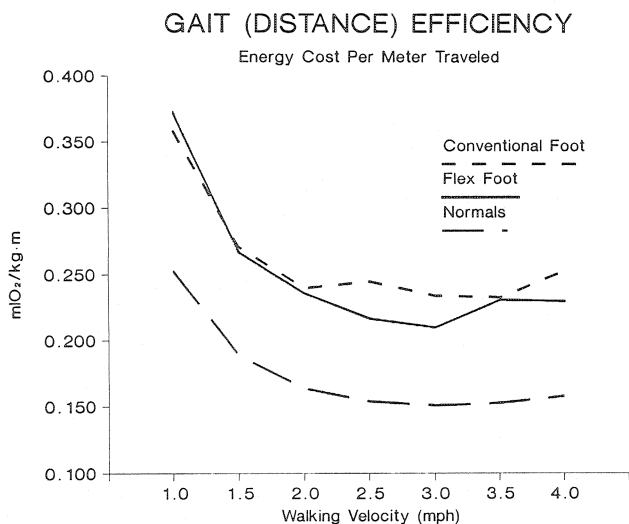

GAIT (DISTANCE) EFFICIENCY

Energy Cost Per Meter Traveled

Figure 3
Gait efficiency differences of amputee walking based on type of prosthetic foot with comparisons to normal values.

equal to and above 2.5 mph walking performance was enhanced.

The relative exercise intensity of gait is the relative workload of walking which can be expressed as a percentage of the person's age-predicted maximum heart rate (percent MHR):

$$\text{percent MHR} = \frac{\text{steady state exercise heart rate}}{\text{age-predicted maximum heart rate}} \times 100$$

or as a percentage of the individual's maximum aerobic power (percent MAP):

$$\text{percent MAP} = \frac{\text{steady state energy cost of walking}}{\text{experimentally determined maximal oxygen uptake}} \times 100$$

Relative exercise intensity (percent MHR and percent MAP) has been used to evaluate gait performance in various patient groups[1,17,23,28,34]. It has been stated that ambulation may be too demanding for some handicapped disabled individuals[3,7]. The general guideline is that the relative exercise intensity for ambulation at the self-selected walking velocity should not exceed 70 percent MAP or 80 percent MHR values[17,22]. For practical reasons, we did not want to do maximal graded exercise tests on our subjects; we elected to use percent MHR as the criterion measure of relative exercise intensity.

The range in percent MHR for our amputee subjects was from 48 percent at 1.0 mph to 80 percent at 4.0 mph. The average percent MHR value at the subject's self-selected walking velocity was approximately 65 percent. These results suggest that the exercise stress associated with below-knee amputee walking was well within tolerable physiological limits. The reduced percent MHR values for ambulation with the Flex-Foot which occurred at all walking velocities indicated decreased levels of exercise stress.

Efficiency is technically defined as the ratio of the work output to the work input or the ratio of the power output to the power input[20].

$$\text{Efficiency} = \frac{\text{work output}}{\text{work input}} \text{ or } \frac{\text{power output}}{\text{power input}}$$

Numerous approaches to studying gait efficiency have been described in the literature, but there is little agreement about which approach is best[4,10,18,21,25,35]. Part of the problem is related to inconsistency and inadequate definition of terminology. The complexity of the motor task and the inherent difficulty of objective measurement of the associated work component of walking are confounding factors.

The work output of ambulation can be simply expressed as the product of the person's body weight times the vertical displacement of the body's center of gravity. The work input is reflected by the energy cost of walking[35].

$$\text{Gross Efficiency of Ambulation} = \frac{\text{body weight} \times \text{vertical displacement of center of gravity}}{\text{energy cost}}$$

Displacement of the body's center of gravity during walking, however, is difficult to measure, usually requiring sophisticated cinematographical measurement systems, e.g. high speed 16 mm cameras or special videotaping-motion analyzers[6].

A more easily determined alternative criterion measure of gait efficiency is the term we refer to as "distance efficiency". Gait (distance) efficiency is the energy cost per distance traveled. It is calculated simply from the ratio of the oxygen uptake to the walking velocity and may be expressed in milliliters of oxygen consumed per kilogram of body weight per meter traveled.

$$\text{Gait (Distance) Efficiency} = \frac{\text{ml } O_2/\text{Kg.min}}{\text{m/min}} = \text{ml } O_2/\text{Kg.m}$$

In this context, a decrease in calculated gait efficiency reflects improved overall work/exercise efficiency. Although the specific term "distance efficiency" has had limited employment in the literature, the concept behind the term has been used by numerous authors who studied gait[1,8,9,17,19,27,34,36].

The gait efficiency graphs that we obtained on our three amputee subjects appear to be quite reasonable. The response curves were a little erratic, probably due to the small subject sample size. The profile of the graphs was the same for the amputees and normal subjects. The upward displacement of both amputee curves indicated decreased efficiency for amputee compared to normal walking. The separation of the amputee curves suggested improved gait efficiency for Flex-Foot ambulation at speed of 2.5 mph and above.

Optimal efficiency, i.e. the minimal energy cost per meter traveled, for all three curves occurred at approximately the same speed, 3.0 mph, which interestingly corresponded to the self-selected walking velocity of both amputee and normal groups. The optimal gait efficiency value for ambulation with the conventional foot was .24 ml 02/kg.m. at 80 meters/minute (3.0 mph) which was higher than the value Waters reported, .20 ml 02/kg.m. at 71 meters/min (2.6 mph) for his traumatic below-knee amputee subjects[34]. Interestingly, the optimal vlaue for the Flex-Foot was .21 ml 02/kg.m. at 85.8 meters/minute (3.2 mph). These results suggest that the Flex-Foot accommodates faster walking velocities without compromising gait efficiency.

Subjective feedback from the subjects supports these findings. The comments in general were positive regarding their use of the Flex-Foot. The most common responses were that the Flex-Foot allowed faster walking and improved general balance and stability while walking on uneven ground. Ambulation with the conventional foot was possibly better at very slow walking speeds and during downhill walking. Based on these observations future research could include the effect of uphill and downhill grade walking.

SUMMARY AND CONCLUSIONS

The present study focussed on the gait performance of traumatic below knee amputees during walking with the Flex-Foot versus a conventional prosthetic foot over a functional range of walking velocities from 1.0 to 4.0 mph. Although the results are based on only a small number of subjects, several conclusions appeared to be warranted:

1. Ambulation with the Flex-Foot tended to facilitate faster walking approximating more normal values of self-selected walking velocity.
2. Little difference was seen in gait performance during slow walking velocities (less than or equal to 2 mph) between the conventional prosthetic foot and the Flex Foot.
3. Ambulation with the Flex-Foot at higher walking velocities (greater than or equal to 2.5 mph) tended to conserve energy resulting in lower relative levels of exercise intensity and enhanced gait efficiency.

ACKNOWLEDGEMENTS

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PISCINE ORTHOPAEDICS

Twisted Trout, Crooked Catfish and Other Finny Funnies

By David D. Scherr, M.D., Ph.D.
1111 Madison St.
Jefferson City, Missouri

I wish to aquaint readers of this paper with a phase of orthopaedics not well known by the usual orthopaedist, and with a medical organization some readers would enjoy joining. The particular phase of orthopaedics is that which concerns skeletal defects in fish, and the organization is the American Medical Fly Fishing Association. This paper contains information which I learned while preparing to deliver a paper of the same title to the annual meeting of that association in West Yellowstone, Montana, on August 29, 1986. I must add that the majority of papers delivered there each year concern human medical topics.

The skeleton of bony fishes is almost exclusively axial, so orthopaedic abnormalities in fish involve scoliosis, lordosis and kyphosis. In addition, cranial abnormalities also occur. Descriptions of the best known skeletal deformities in fish follow.

WHIRLING DISEASE OF TROUTS

This is perhaps the most interesting disease process causing skeletal deformities in fish. Its cause is infection of trouts and salmonids by *Myxosoma cerebralis*, a unicellular parasite. Whirling Disease was first identified in this country in 1956, affecting factory reared rainbow trout (*Salmo gairdneri*). It had first been described in Germany in 1903 when rainbow trout became diseased after having been imported to Germany. Investigation showed that the native European brown trout (*Salmo trutta*) commonly is infected by the parasite but shows no outward evidence of the infection. The term "whirling" has been applied to the disease because spores of the micro-organism develop within the pre-osseous cartilage skeleton of the fish and result in cranial deformity which alters the function of the intracranial balance organs. When the affected fish accelerates its swimming motion such as when pursuing prey, its defective sense of balance will cause it to whirl in a tail-chasing manner (rather than spiraling axially as is characteristic of several other diseases not involving skeletal deformity). As the vertebral column ossifies, various spinal deformities may also occur. These not only alter the configuration of the body of the fish, but if the infection involves the twenty-sixth post cranial vertebra, a condition called "blacktail" occurs. Deformity at that vertebral level can interrupt the nerves supplying color control to the caudal section. Spores of *M. cerebralis* have been identified in diseased fishes and in their rearing ponds, but, interest-
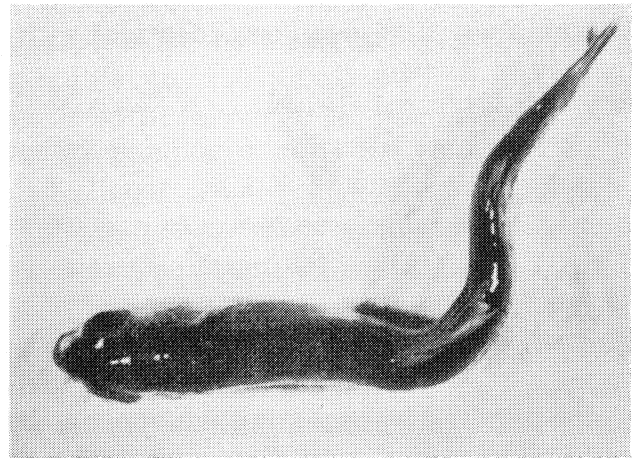
Figure 1
Yearling Rainbow Trout with scoliosis caused by *Myxosoma cerebralis*. From Whirling Disease of Trout caused by *Myxosoma cerebralis* in the United States by Hoffman, Dunbar, and Bradford.

Figure 2
Brook trout with spinal deformity and blacktail, caused by *Myxosoma cerebralis* infection. From Whirling Disease of Trout Caused by *Myxosoma cerebralis* in the United States by Hoffman, Dunbar, and Bradford.

ingly, artificial transfer of spores or infected tissues into uneffected fish has never been shown to produce the disease, so Koch's Postulates have never been fulfilled in this disease. Brown trout harbor the parasite without outward signs of disease, whereas rainbow trout are severely affected, and brook trout (*Salvelinus fontinalis*) are less severely affected than are the rainbows. The disease also affects several species of true salmons[3].

Figure 3
Rainbow trout with blacktail caused by *Myxosoma cerebralis*, about three months post infection. From Whirling Disease of Trout Caused by *Myxosoma cerebralis* in the United States by Hoffman, Dunbar and Bradford.
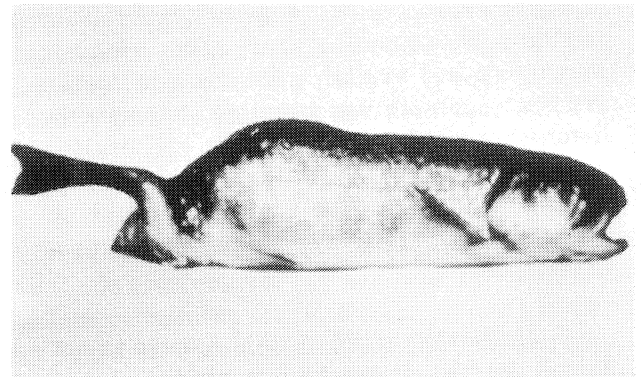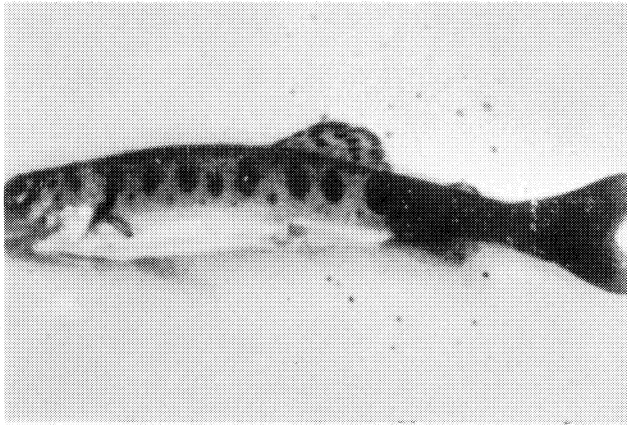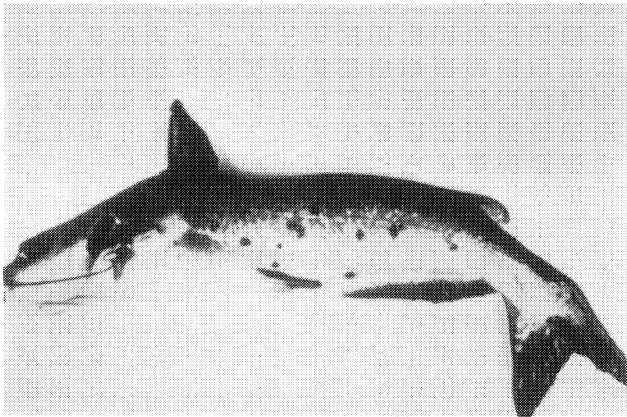

Figure 4
Channel catfish difficient in ascorbic acid, showing retracted head and lordosis at base of tail. From Nutrient Requirements of Trout, Salmon, and Catfish. National Academy of Sciences.
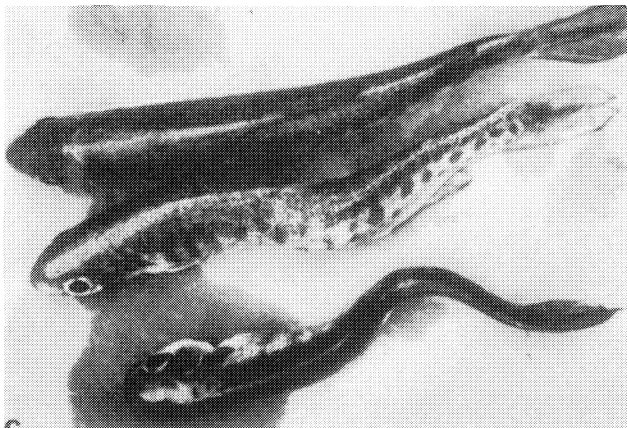

Figure 5
Coho salmon fingerlings. The fingerling at the top of the photo is normal. The other two are deficient in ascorbic acid. From Nutrient Requirements of Trout, Salmon and Catfish. National Academy of Sciences.

POLLUTANTS

Skeletal deformities in fish are known to result from toxicity of heavy metals and chemicals produced by man[1,2], including organophosphorous pesticides such as Kepone, and the defoliant Trifluran.

The skeletal system of some fish is so sensitive to pollutants that fish have been proposed as biological indicators of water purity or pollution. In Japan, the red medaka (*Oryzias latipes*) was found to develop spinal abnormalities in proportion to the concentration of two heavy metals and two agricultural chemicals to which the young fish were exposed, so the authors suggested that the fish be used to indicate levels of pollution. In addition, those investigators stated that the medaka cannot live in waters where zink and cadmium are present in quantities considered unsafe for human consumption[2].

Finally, as might be expected, piscine skeletal deformities can occur in the wild, without known etiology. Fig. 6 shows an anteroposterior roentgenogram of a grossly scoliotic salmon caught by a fellow orthopaedist.
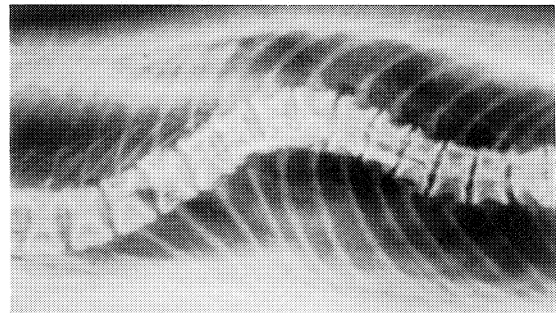
Figure 6
Roentgenogram of a wild salmon caught by orthopaedist Glen McElroy, M.D., Columbia, Missouri. Photograph contributed by Robert Gaines, M.D., Division of Orthopaedic Surgery, University of Missouri Hospitals and Clinics, Columbia, Missouri.

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ISOLATED ATROPHY OF THE SUPRASPINATUS AND/OR INFRASPINATUS MUSCLES

Report of Three Cases and Review of the Literature

M. Mysnyk, M.D.
J. L. Marsh, M.D.

Department of Orthopaedics
University of Iowa Hospitals

Isolated weakness and atrophy of the supraspinatus and/or the infraspinatus muscles is not a common problem, but has been reported with increasing frequency. We are presenting three cases which have similar presentations, but, have different etiologies and therefore need to be treated quite differently. The most common cause is entrapment of the suprascapular nerve at the suprascapular notch as seen in our first case. A much rarer cause, demonstrated by our second case, is isolated compression of the inferior branch of the suprascapular nerve by a ganglion. The third etiology, not previously reported in this context, is isolated weakness of the spinati muscles due to a C5-6 disc (case 3).

CASE REPORTS

Case One

A forty-six year old right handed female was referred for evaluation of a one and one half year history of posterior right shoulder pain. The pain was burning in nature and occasionally radiated to her neck. Activities involving arm abduction or internal rotation aggravated her symptoms. Non-steroidal medications were ineffective.

Physical exam revealed significant atrophy of the right supraspinatus and mild atrophy of the infraspinatus muscles. She had 5-/5 strength in abduction and external rotation. The remainder of the upper extremity neurologic examination was normal.

EMG's demonstrated denervation of the right supraspinatus and questionable denervation of the infraspinatus muscles. Nerve conduction velocities of the brachial plexus were normal, but NCV's of the suprascapular nerve were not done. A diagnosis of suprascapular nerve entrapment at the suprascapular notch was made and the patient elected surgical treatment.

At the time of surgery, the patient was placed in the lateral decubitus position and a transverse incision 1 cm. superior and parallel to the spine of the scapula was made. The trapezius was split in line with its fibers and the lateral aspect of it raised from its insertion on the spine of the scapula. The supraspinatus was then elevated from the supraspinous fossa until the transverse scapular ligament was identified. This was sectioned and the nerve was identified and seen to be intact. No widening of the notch was done. Post operatively, the patient had immediate, complete relief of her pain. Over the course of the next seven months her strength returned to normal.

Case Two

A thirty-seven year old right handed male presented with a four month history of severe left shoulder and scapular pain accompanied by weakness. On close questioning he recalled some left shoulder weakness four years previously. His severe symptoms began acutely during an aerobics class while using hand-held weights. In subsequent months, he noted increased weakness especially in external rotation (difficulty opening the car door), and one month prior to his initial evaluation he became aware of decreased muscle mass in the scapular region. He had no neck or arm pain.

Physical examination revealed marked atrophy (Fig. 1) and weakness of his left infraspinatus muscle but the supraspinatus muscle was normal as well as the rest of his upper extremity musculature. Neck and shoulder range of motion, sensory exam, and deep tendon reflexes were normal. EMG's demonstrated isolated denervation of the left infraspinatus. Nerve conduction velocities of the brachial plexus were normal. Based on this examination and EMG findings the etiology was most likely compression of the inferior branch of the suprascapular nerve in the infraspinatus fossa.
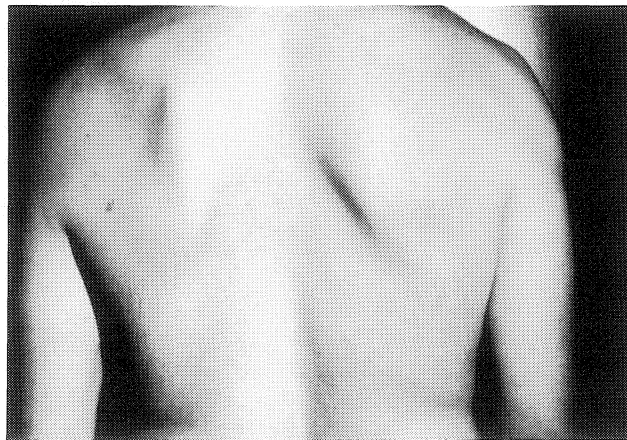

Fig. 1
Case 2. Patient with infraspinatus atrophy on the left side.

The patient underwent exploration of the suprascapular nerve. He was positioned prone with his left arm draped free. A transverse incision directly over the spine of the scapula was made and the posterior aspect of the deltoid was raised from the scapula. The infraspinatus was elevated subperiosteally until the spenoglenoid notch could be seen. This revealed a 4 X 3 cm. cystic mass, apparently arising from the glenohumeral joint, which was compressing the inferior branch of the suprascapular nerve as it coursed around the spenoglenoid notch (Fig. 2). The cyst was excised and histologic examination was consistent with a ganglion cyst. Post operatively the patient's pain immediately resolved but at the six week follow-up he had no return of external rotation strength or increased mass of the infraspinatus muscle.
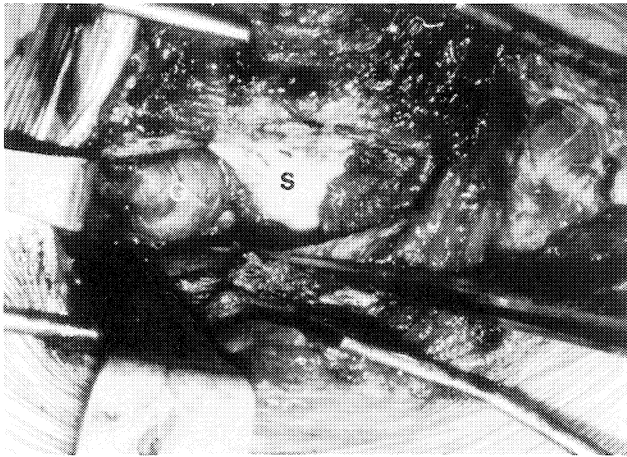

Fig. 2
Intraoperative photograph of case 2. The infraspinatus is reflected caudad and the ganglion (labeled G) is noted at the spenoglenoid notch and into the infraspinatus fossa of the scapula (labeled S).

Case Three

A thirty year old woman was referred for evaluation of left shoulder pain. She stated she had episodic left sided neck pain for several years, and three months prior to her initial evaluation she had acute onset of this pain while lifting weights. The severe pain resolved, but she continued to have persistent, but milder, left sided neck pain. She also developed shoulder and lateral arm pain with occasional pain into her forearm with episodic numbness in her left thumb. Just prior to her evaluation, she had noticed left shoulder weakness.

The physical examination demonstrated severe atrophy of her left supraspinatus and infraspinatus musculature. External rotation and abduction strength were diminished (4/5) and she clearly became worse over the next few weeks. Strength throughout the rest of the upper extremity was normal including the biceps. Deep tendon reflexes were normal and two point sensation was 4-5 mm. in all digits. Neck and shoulder motion was full.

Anteroposterior, oblique and lateral flexion-extension cervical spine plain films were normal. EMG's revealed isolated denervation of the left infraspinatus. The EMG's of the supraspinatus, biceps, rhomboids and deltoid were normal. NCV's were done only of the median and ulnar nerves and these were normal.

Since the EMG's did not completely explain the clinical picture (atrophy of both the supraspinatus and infraspinatus), and since she had subjective complaints consistent with a possible cervical radiculopathy (though not confirmed on physical examination or EMG's), an MRI was done. This revealed a left sided extra-dural defect at C5-6 consistent with a ruptured disc. A cervical myelogram confirmed this. At the time of this writing, she was scheduled for disc excision.

DISCUSSION

The suprascapular nerve arises from the upper trunk of the brachial plexus and is comprised primarily of C5 and C6 nerve fibers. It runs deep to the trapezius and omohyoid muscles to reach the suprascapular notch. At this point, the nerve passes into the supraspinous fossa by coursing under the transverse scapular ligament while the suprascapular artery, which accompanies the nerve throughout its course, runs over the ligament. The nerve runs deep to and supplies motor fibers to the supraspinatus muscle and continues laterally to supply sensory branches to the acromioclavicular and glenohumeral joints and also the subacromial bursa. It then curves around the spenoglenoid notch (which may be covered by the spenoglenoid ligament in up to fifty percent of cases[10]) to supply the infraspinatus muscle. It has no cutaneous sensory branches.

The most common case of suprascapular neuropathy is trauma. This is occasionally direct, but most frequently it is an indirect traction injury of the nerve at the suprascapular notch due to the mobility of the scapula. Although this injury may be acute, it is more commonly secondary to chronic irritation at the notch as in our first cast. Rengachary and Swafford described variations in the anatomy of the suprascapular notch which may predispose patients to this injury. Other documented etiologies of suprascapular neuropathy resulting in atrophy of both the spinati include shoulder dislocations[22], radiation induced sarcoma in the suprascapular region, and scapular fractures involving the suprascapular notch[3,18].

Isolated atrophy and weakness of the infraspinatus may also be due to chronic irritation of the suprascapular nerve at the spenoglenoid notch rather than the suprascapular notch. Aiello et al. described shoulder pain and isolated infraspinatus atrophy on the dominant side of a fencing instructor[1]. At surgery, they found a hypertrophied spenoglenoid ligament which was excised. Ferretti et al also

described isolated, asymptomatic infraspinatus wasting on the dominant side in twelve of ninety-six world class volleyball players[4]. They believed that repeated traction at the spenoglenoid notch during the serve caused the deficit and believed either a spenoglenoid ligament, or a variant of the terminal motor braches of the suprascapular nerve, as described by Mestdagh[10], could predispose certain players to this.

The patient in case two presented with shoulder pain and isolated weakness and atrophy of the infraspinatus muscle. This was secondary to a ganglion in the infraspinatus fossa and at the spenoglenoid notch compressing the inferior branch of the suprascapular nerve. Six previous cases of suprascapular nerve compression by a ganglion have been reported in the English literature[5,7,8,11,20]. Three of these patients presented similarly and the surgical findings were nearly identical[5,20]. In the other three, the ganglion was superior to the notch. In two cases, both the infraspinatus and supraspinatus muscles were affected[7,8] and in the remaining case, only the infraspinatus muscle was affected[11]. Unlike the other five reported cases in which the suprascapular nerve was compressed under the ganglion, in this case the nerve was stretched over the mass.

The patient's history, physical exam, response to injection, radiographs and EMG/NCV's should be used to determine the etiology of the atrophy and the weakness. The most common presenting symptom, especially when the compression is at the suprascapular notch, is deep-seated shoulder pain, which is usually posterior. Six of the nine cases presented by Post, however, also had arm or neck pain. Others have confirmed that the pain due to suprascapular nerve entrapment is frequently referred to the neck or lateral arm[2,7,8,21] (presumably due to the nerve's constant origin from C5[10]). Occasionally, the pain may even be referred to the extensor muscles of the forearm secondary to a traction injury on the radial nerve axis[9].

On physical exam, atrophy of the supraspinatus and infraspinatus is frequently seen as is weakness of abduction and external rotation of the shoulder. Two of our patients presented with atrophy and weakness of both the supraspinatus and infraspinatus. Since rotator cuff injuries (impingement syndrome or tears) may also present this way and are seen more frequently, the work-up and exam must rule these out. An arthrogram should be obtained if any question exists.

Diagnostic and therapeutic injections have been tried with varied success. Injection of a local anesthetic in the area of the suprascapular notch has been advocated as a diagnostic aid[6,12,13,18,21]. However, since the suprascapular nerve sends sensory fibers to the subacromial bursa, and the acromioclavicular and glenohumeral joints, relief of the pain would not differentiate between rotator cuff or impingment pain and suprascapular nerve entrapment. In

fact, Rose described using such injections to treat shoulder pain, noting it is "most applicable for the tendonitis-bursitis type of shoulder involvement". Koppel suggests using a steroid preparation (rather than a local anesthetic) for the injection for both diagnostic and therapeutic measures, implying it would not relieve the pain if a rotator cuff injury is present[9]. Although others agree that steroid injections may be therapeutic[7,14,21], we suspect that relief of rotator cuff pain could still be seen with steroid injections, especially considering the lack of control over the exact area of the injection (even with fluoroscopic control). In short, a suprascapular notch injection with local anesthetic agents and/or steroids may be therapeutic but it does not differentiate pain due to suprascapular nerve compression from that due to rotator cuff pathology. If the clinical setting makes the latter a possibility, an arthrogram is indicated.

Probably the most important diagnostic aids for suprascapular nerve entrapment are electromyographic (EMG) and nerve conduction velocity (NCV) studies. Post and Mayer specifically emphasized the need for nerve conduction studies of the suprascapular nerve, since in six of their nine cases the EMG's were negative but the NCV's were positive[12]. In our three cases, NCV's of the suprascapular nerve were not done, although EMG's were positive in all three cases. However, in the third case the EMG findings proved to be misleading and deserve special comment.

The third case demonstrates that if the history, physical examination and EMG's conflict then other etiologies other than entrapment need to be considered. In this case, the patient presented with symptoms of shoulder pain with radiation to her back and arm which are consistent with entrapment. However, she also initially had severe neck pain which later largely resolved, and episodic tingling in her thumb which are atypical symptoms for entrapment. Her physical exam revealed wasting of both her spinati muscles on the right while the EMG's demonstrated isolated denervation of only the infraspinatus. A possible explanation for this descrepancy between physical exam and EMG's may be due to the fact that the trapezius (which overlies the supraspinatus) was tested instead of the supraspinatus. NCV's of the supraspinatus might have helped and in the future these will be obtained. Because of these descrepancies, an MRI followed by a cervical myelogram were obtained which revealed a herniated C5-C6 disc.

MRI and cervical myelogram have rarely been mentioned in the work-up of spinati atrophy. Clem stated that these should be obtained whenever there is any question[2]. Rask felt that a myelogram should be obtained in patients that had continued symptoms after suprascapular ligament release[13]. Beyond these two reports there is very little mention in the literature of any further work-up, other than plain films, to rule out cervical pathology. In fact,

Weaver states, "myelography has no place in the diagnosis of a suprascapular nerve lesion"[21]. We disagree with this and the third case demonstrates why the cervical roots must be carefully evaluated.

Once the diagnosis has been made, then therapy should be planned. If the site of entrapment has been localized to the suprascapular notch, simple resection of the ligament (without widening the notch) via the posterior approach to the supraspinous fossa gives consistently good results[2,6,7,12,14,16,19]. Rask has suggested widening the notch routinely[13], but this is not necessary in most cases and could potentially cause deleterious scarring.

If there is isolated involvement of the infraspinatus muscle, the infraspinous fossa should be explored. In the second case the posterior aspect of the deltoid was raised from the spine of the scapula and the infraspinatus was subperiosteally elevated so the spenoglenoid notch could be visualized. This gave excellent exposure and because the insertion of the infraspinatus was not taken down post operative rehabilitation was simplified.

Rengarchary stated that the sequence of improvement with resection of the suprascapular ligament was relief of pain, return of strength, and lastly, if at all, return of muscle mass[16]. Others have reported similar results, including the return of EMG's to, or at least towards normal[2,19].

Results following excision of a ganglion are similar. Of the six cases reported in the literature, five presented with pain. Post operatively, pain fully resolved in three and partially in one. Four of six had improved strength, but atrophy persisted in most[5,7,8,11,20]. At the time of this writing, this patient was only six weeks status post surgery. His pain resolved immediately, but no strength or muscle mass had returned.

SUMMARY

In a patient presenting with shoulder (and/or neck and arm) pain, weakness and atrophy of the supraspinatus and/or infraspinatus muscles, suprascapular nerve entrapment must be considered along with rotator cuff injury, brachial plexus injury, and cervical radiculopathy. EMG's and NCV's are helpful to localize the site of entrapment as well as to rule out other possibilities in the differential. If a descrepancy still exists, then cervical spine MRI may be helpful to rule out cervical spine pathology. Once the diagnosis of supraspinatus entrapment or compression is made and localized, then surgery has been consistently effective in relieving the pain. Weakness may improve although atrophy often persists.

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SPINAL DEFORMITIES SECONDARY TO TUMORAL PATHOLOGY IN CHILDREN: PATHOLOGICAL CONSIDERATIONS AND TREATMENT

J. Dubousset
A. M'Rabet

Hospital St.-Vincent-de-Paul Paris

Pedriatric oncology is a relatively new science born in France in the early 1950's. Tremendous progress has been made in the cure of the tumors, primarily malignant, during these last thirty years. This progress has been related to a better understanding of the tumor disease, and improvements in the timing of application and coordinated management of surgery, chemotherapy, and radiation therapy. The overall result of this progress has been an increasing rate of survival in children. With a complete cure of a tumor there are often undesirable sequelae. Sometimes these sequelae are negligible, however significant pathology can develop from the treatment itself.

Spinal deformities secondary to the treatment of a tumor illustrates well such secondary pathology. While the tumor may induce spinal deformity, the deformity may be potentiated by factors such as biopsy and local incision techniques as well as adjuvant therapy.

During the last fifteen years, we were able to study and follow more than three hundred such patients in the largest institution for cancer in Paris (IGR and Curie Institutes). Spinal fusion, with and without instrumentation, was the treatment of spinal deformity in one hundred and three of these patients. The purpose of this paper is to attempt to identify the etiology of these deformities in these patients and to discuss subsequent treatment options.

In the evaluation of the etiology of tumor related spinal deformity, a differentiation must be made between deformities arising directly from the lesion or in the immediate vicinity, from deformities secondary to factors such as paralysis or general growth disturbances. The etiology of spinal deformities has been related to the type of primary tumor, its location, and relationship to the spine. We can distinguish four principle locations of primary tumors: extra-spinal, juxta-spinal, and intra-spinal and combinations of the above:

1) Extra-spinal designates a tumor removed from the spine itself with no direct anatomic relationship, such as a Wilms tumor.
2) Juxta-spinal designates the tumor outside of the spinal canal itself, but with close proximity to the bony or soft tissue of the spine, such as a neuroblastoma or rhabdomyosarcoma.

3) Intra-spinal is defined as a tumor that arises within the spine itself either as bone tumors or in the nervous structures within the spinal canal (tumor of the cord, roots, or meninges).
4) Combinations of the above locations such as when a juxta-spinal tumor grows inside the canal through a foramen or other structure such as the dumbell tumor in neuroblastoma or neurofibromatosis.

In our one hundred and three cases, fifty six percent of these tumors developed essentially inside the spinal canal requiring laminectomies of varying extent. Adjuvant radiation therapy was used in the majority of these cases. Only twenty-one of the one hundred and three patients were limited to surgical treatment; chemotherapy as a single adjuvant was administered in two cases. Kyphosis, either pure or combined with varying degrees of scoliosis, was the dominant spinal deformity noted in ninety percent of the patients. Additionally, seventeen patients developed paralysis in the lesional zone, leading to deformities requiring fusion. As expected, the paralysis stemmed from intra-spinal lesions. Review of these records allowed a detailed analysis of factors predisposing the spinal deformity from locales: lesional and extralesional (especially infra-lesional).

If we look first at the lesional area, we can see direct lesions of the spinal structures arising from several sources. Destruction of a vertebral pedicle or posterior arch can occur in lytic lesions of bone tumors, both malignant and benign, such as osteoblastoma or other pathologic processes such as aneurysmal bone cyst. Destructive permeation of a tumor, such as neuroblastoma, into adjacent bony structures such as adjacent pedicles, lamina, transverse processes or even ribs, often explains the development of rather sudden spinal instability. Direct lesions may arise from the surgical approach to remove the tumor. If the tumor involves the spinal canal such as an intra-spinal lesion, excision may lead to significant spinal deformity. This was the principle complication of laminectomies in children in a previous study where we were able to demonstrate that a bilateral facet joint removal at one level of the growth plate uniformly resulted in kyphosis. The influence of this factor is related to age, as this deformity progresses with growth. The younger the patient is at the

time of surgery, the more severe the deformity will be at skeletal maturity. In contrast, mild deformity will result if similar surgery is performed when skeletal maturity has been achieved. The level of the laminectomy is also related to the degree of deformity. In the cervical spine, kyphosis and instability will never develop if only the posterior arch of C1 is removed. However, a patient has a twenty-five percent chance of developing kyphosis and dislocation if the C1 and C2 posterior arches are removed. The chance of development of deformity increases to one hundred percent if the laminectomy is extended to C7. Kyphosis can appear at every level of the spine, but the greatest frequency is found in the cervico-thoracic area and thoroco-lumbar area. When a laminectomy is done in the lower lumbar region, where the spine is normally lordotic, there is a lower incidence of secondary kyphosis. Finally, the resultant deformity can be related to the amount of posterior resection, including resection of the ligamentum flavum, which as determined experimentally is sufficient to initiate kyphosis. Of course, the wider the resection, the worse the instability and deformity. If we can keep one half of the posterior arch intact, such as in hemilaminectomy, even at several consecutive levels, we can prevent kyphosing deformity.

The surgical approach and removal of the tumor can damage the soft tissues surrounding the spine and give direct or indirect insults to the spine, resulting in deformity. For the completely extra-spinal lesion, such as a Wilms tumor, the volume of the tumor or the degree of invasion may increase the need for removal of soft tissues (part of the diaphram, vena cava, etc.). We have seen for example in a series of Wilms tumors previously reviewed and published, that more extensive surgery leads to more severe deformity, especially when radiation therapy was given soon after surgery. In such cases, it was interesting to notice that the concavity of the deformity was always on the same side as the surgery was done. We have observed this in tumors of the chest wall away from the spine or sarcomas of the abdomen as well. In the case of juxta spinal tumors, especially neuroblastomas, involving the thoracic area with or without invasion of the spinal canal, we have uniformly observed that the concavity of curvature coincides with the region of surgery. This is independent of adjuvant radiation therapy. In a previously published work, twenty-five of twenty-seven patients with pure thoracic neuroblastoma developed thoracic scoliosis with a convexity on the side of the thoracotomy. Additionally, the apex of the curve was at the level of the thoracotomy. It is feasible that paralysis of small posterior muscles of the spine during surgery initiates the convexity of the curve.

Finally, we know very well that direct lesions may result from the adjuvant therapy, especially radiation therapy. The effect of radiation on soft tissue gives progressive sclerosis with limitation of mobility and contracture of the skin, muscles, etc. The most visible effect of radiation is on the growing structures. This was very well studied in the Wilms tumors and reported by many authors. The three-dimensional assymetrical doses of radiation received by the vertebra explains why the deformities always retard growth on the side of the tumor laterally and in front of the spine. These growth retardations result in anterior wedging with predominant kyphosis and lateral wedging with predominant scoliosis. In general, comparing equivalent doses, more deformity results from irradiating younger patients.

Asymmetric dosimetry is more or less constant to protect adjacent vital structures such as vessels of the contralateral kidney, spinal cord, etc. Mechanical damage produced by asymmetric pressure on the growing structure, asymmetric sclerosis and biologic damage produced by direct avascular necrosis secondary to surgery and/or radiation therapy, lead to growth disturbances. This explains why most of the spine deteriorate progressively with a clear increase during the adolescent growth spurt.

When looking at the infra-lesional area, two types of damage can be seen: those resulting from hormonal disturbances and those resulting from paralysis. When the tumor is located high in the cervical region and requires adjuvant radiation therapy, a disturbance of the thyroid gland or thalamus and pituitary gland may be seen. This may interfere with the function of growth hormone leading to alterations in the height of the spine. Similarly, complications may be encountered with treatment of tumors of the retroperitonum involving the adrenal glands. When the tumor involves the spinal canal, the spinal cord or the roots and results in permanent damage of nervous structures, the effects are often not purely symmetrical and the underlying spine may develop a paralytic scoliosis. The scoliosis may progress and require specific treatment.

TREATMENT

The treatment of instability and protection of spinal cord function is the same as for all kinds of pathology and is mandatory. We have to distinguish between immediate instability and potential instability. Immediate instability is defined as a radiographic demonstration of abnormal motion between two adjacent vertebra. Potential instability cannot be demonstrated by dynamic roentgenograms, but is evident as an increased risk of instability by sudden injury or gradual progression of a deformity at a particular vetebral level. Both types of instability result in a mechanical danger for the spinal cord and require immediate treatment.

Posterolateral fusion with and without instrumentation can be used for the treatment of either immediate or potential instability. One can expose and decorticate the necessary elements at the time of laminectomy such as facets and transverse processes. We fuse one or two intact

levels above and below the level of the lesion. Postoperative immobilization is achieved by a cast. Common instrumentation includes the Harrington rod and the Cotrel-Dubousset systems. Sometimes it is necessary to treat potential instability in a delayed fashion after a laminectomy has been performed. Progressive deformity is reduced as much as possible with a halo system prior to posterolateral fusion. Because of altered anatomy from the previous surgery, it is necessary to begin the dissection superior and inferior to the level of the laminectomy, where normal anatomy should be encountered. We then work towards the level of the lesion. Sometimes it is necessary to be extremely careful in the region of the lesion because either the tumor has destroyed bony elements or previous surgery has resulted in loss of bony elements. Careful preoperative radiographic evaluation will aid in determining such conditions. Autogenous bone graft harvested from the iliac crest is used in the fusion. In some cases, anterior fusion alone can be sufficient. When the lesion has destroyed the vertebral body and the posterior elements, it is necessary to fuse anteriorly and posteriorly. This is especially true when radiotherapy was used for the treatment of the primary tumor.

We have tried to treat the asymmetrical growth disturbances secondary to irradiation by early posterior fusion done at a young age. The results have been poor because the growth potential of the irradiated growing cartilage of the vertebra is low and a high percentage of fibrous scarring is present. I now feel that for deformities where growth imbalance is secondary to radiation therapy, it is better to treat by cast or brace and fuse the entire segement of the spine involved when growth is close to maturity. In some cases such as an associated paralytic spine, we can augment bracing by subcutaneous rodding.

Posterior fusion alone with or without instrumentation has been used in the final treatment for deformities near growth arrest. We tended not to instrument the spines early in our experience. The treatment period was long, often requiring almost one year of casting and bracing, always with the concern of development of secondary pseudoarthrosis. We now attempt to instrument the spine in order to enhance the correction and to reduce the time of external immobilization. Instrumentation is most useful when there is no or very little kyphotic component of the deformity or if the kyphosis is mainly rotational and corrects with reduction of the scoliosis.

Most of the cases we have seen develop predominantly kyphotic deformities. These deformities can be pure or develop with various amounts of scoliosis. In treatment of these deformities with combined anterior and posterior fusion, we determine the order of procedures by obtaining lateral dynamic flexion and extension spine roentgenograms. If satisfactory correction of the deformity is noted with extension of the spine, we begin with posterior instrumentation followed eight to fifteen days later by anterior fusion. On the other hand, if the curve is demonstrated rigid on the dynamic hyperextension view, we start by anterior approach, release, and fusion and follow this eight to fifteen days later with the posterior fusion and instrumentation to augment the correction. Between and following the procedures, the patient is maintained in halo traction until circumferential fusion is achieved.

In some cases, we have used anterior and posterior fusion without instrumentation. Some years ago, we used Harrington instrumentation with distraction rods on the concave side and compression rods on the convex side. Now, of course, we have switched to Cotrel-Duboussset instrumentation without external immobilization.

In cases of neurologic disorder in relation to the deformity, it is always difficult to know what is secondary to radiation and what is mechanical. If the deformity is slightly flexible on the hyperextension roentegenogram, it is advisable to start with gentle traction and hyperextension or with a distraction cast.

Among a study group of forty patients with extra-spinal tumors and associated kyphoscoliosis without spinal defects, we found:

1) Fourteen were treated by posterior fusion with or without instrumentation. Preoperative mean angle of scoliosis was fifty-seven degrees corrected to thirty-one degrees and maintained at thirty-six degrees with six years mean follow-up. Kyphosis component was thirty degrees preoperative corrected to twelve degrees and maintained to seventeen degrees at follow-up.

2) Twenty-six patients were treated by anterior and posterior fusion with mean preoperative angle of kyphosis at sixty-seven degrees and twenty-five degrees scoliosis (this explains why the anterior fusion alone was done). The immediate results were thirty-two degrees average kyphosis and fourteen degrees average scoliosis with only three degrees and one degree loss of correction at four years follow-up, respectively.

For patients with laminectomy, we excluded the patients where fusion and/or instrumentation was done at the time of primary surgery. For these patients with or without radiation therapy, we had a mean preoperative angle of seventy-nine degrees kyphosis corrected to forty-six degrees immediately after surgery and maintained at forty-nine degrees at the mean follow-up of four years. For the scoliotic component, preoperative angles measured thirty-four degrees, corrected immediately postoperatively to twenty-one degrees and 22.5 degrees at follow-up.

At the infra-lesional area, the management of the spinal deformity has been done with subcutaneous rods and bracing during the growth period. Following completion of the growth period, fusion to the sacrum has been carried out if the paralytic spine was the principle problem with pelvic obliquity.

COMPLICATIONS

In our patient population treated with radiation, with and without neurologic deficits, the following complications have been observed. In terms of the fusion, stress fracture of the posterior fusion occurred in one patient, pseudarthrosis in two, fracture of the anterior and posterior fusion which was ultimately repaired with an anterior vascularized rib graft, and progression of kyphosis above and below the early fusion level in two patients. One patient was referred to us from another hospital with significant difficulty in ambulation secondary to neuroblastoma with anterior and posterior fusion done at too early of an age. We performed three posterior osteotomies and compression instrumentation and followed this with extension of the anterior fusion. We have also encountered skin problems including skin necrosis with secondary healing without significant loss of correction, infection secondary to skin damage with protrusion of the instruments from beneath the irradiated skin, and commonly atrophic fragile muscles, fat and subcutaneous tissue secondary to radiation therapy. As noted above, the instruments have a tendency to protrude from beneath the skin when not covered by sufficient soft tissue. We have also had experience with three hooks which dislodged. We felt that this was an especially high risk in Cotrel-Dubousset instrumentation where the tissues in the patient are atrophic or the patient is in a pediatric age group. Pediatric size instruments are not as strong as the adult counterparts and have a smaller contact surface area. We have also seen one case of acute kyphosis developing below the level of the instrumentation with the necessity to secondarily extend the fusion to the sacrum.

The major complications for these patients is related to pulmonary function. Respiratory problems are always present when radiation therapy has been instituted at a young age in the thoracic region. Some patients functionally only have one lung and this is important to determine preoperative as an approach for an anterior fusion should be done on the side where the lung function is worse. In our group of patients where anterior and posterior fusions were performed, the mean preoperative vital capacity was fifty-nine percent. At follow-up at two years, the mean preoperative vital capacity was fifty-one percent. Some of our patients treated with combined surgery and radiation therapy for neuroblastoma, for example, now have permanent tracheostomies in order to augment their ventilation, as well as using ventilatory support while sleeping. It is mandatory for every patient to be evaluated preoperatively by a pulmonary function laboratory in order to secure adequate postoperative care and plan for long term respiratory control and assistance for the remainder of their life. It is also necessary to be aware of renal dysfunction, either from blood pressure anomalies or stenosis of the renal artery observed after irradiation of the con-

tralateral kidney. We also attempt to evaluate hormonal function each time that the neck and the base of the skull have been irradiated to rule out problems with thyroid or growth hormone.

From the above discussion regarding potential problems including failure of early fusion in irradiated spine, it is pertinent to discuss the opportunity for use of a brace or cast for conservative management of deforming problems of the spine encountered in these patients. It is apparent that brace treatment alone will not improve and cure the deformity when the deformity is significant. However, bracing begun at the time of diagnosis is able to prevent deformity or to reduce the extent of the deformity is some patients. Various types of braces can be designed, but in our experience, the Milwaukee brace with specially designed cervical extensions gives good resistance to development of swan neck deformities as well as allowing satisfactory posterior surgical approaches. When the patient is paralytic or nearly paralytic, the Garches-type brace is better suited from the parents and the child. It seems to offer advantages when dealing with respiratory difficulties, enhanced fitting of the abdominal wall, easier access for fixation of calipers if necessary, and presents options for improving control of the spine either in the coronal or sagittal plane.

CONCLUSION

Many of the problems outlined above can be solved or improved significantly if a few key methods of prevention are identified and applied.

SURGERY:

It should be emphasized that laminectomy in children should be carried out with care taken to minimize damage to both facets at one level. If the facets are interrupted, simultaneous immediate posterolateral fusion should be carried out. As an alternative, reconstruction of the lamina and posterior elements at the termination of intraspinal surgery may be attempted. With cervical surgery, reconstruction of soft tissue planes is critical.

POSTOPERATIVE CARE:

In patients without significant kyphotic posture, it will be necessary to brace them or place them on bed rest perioperatively. It is also important to exercise spinal structures in a controlled fashion.

LOWER RADIATION THERAPY DOSES:

It is important to carefully culminate and reduce the radiation exposure in an attempt to irradiate only the malignant tissue and to avoid extension of the radiation field to surrounding structures, particularly those that are vital.

PULMONARY FUNCTION:

Pulmonary toilet with or without the the the aid of ventilators, as soon as the primary lesion is removed, regardless of the age of patient, should be carried out vigorously.

THE FRENCH CONNECTION—
THE LIFE AND TIME OF JACQUES AND FRANÇOIS

by

Richard L. Jacobs, M.D.
Albany Medical College
Albany, New York

"Doctor, doctor, will I die?"
"yes, my child, and so will I!"
Children's jumprope verse—apochryphal

The study of history, they say, puts things in perspective. At the same time, it beats fiction all hollow; here are things that really happened and to people you know existed. Claude Manceron, Mitterand's Minister of Culture, found this out. Starting as a writer of fiction, he read widely in order to furnish a better background for his novels. In the end, what he found interested him so much more that he became a historian instead[14]!

With that explanation, disclaimer or whatever, then a little local history. I remeber a quiet, modest young man (maybe not *too* modest!) when he first came to Iowa out of the hills of West Virginia back in July, 1955. That was *thirty-two* years ago, and *that's* history. Historians like to correlate, so at the same time let me tell you of an early health maintenance organization and their brochure, printed in 1896. It was the Pennsylvania Medical and Burial Company. It claimed, "If you don't respond to medical treatment, you are at least assured of a decent burial". It also noted, "As medical care is cheaper than funeral expenses, the company will no doubt strive at all times to keep its patrons alive". (Albany, New York Times Union, 16 December 1961). Any good historian could relate these two separate little stories like *that*, but I've never claimed to be a historian!

Eponyms give a sense of history, intimatcy and acquaintance. For example, try Dupuytren. Everyone knows about his *contracture*, but what about the man? Imagine this; He was chief of surgery at Hotel Dieu. Among his staff were Lisfranc (joints, amputation), Roux (remember the Roux en Y?), Clouquet (the nodes of), Nelaton (the line of), and Velpeau (dressing of). These are all names that we still use eponymically. Dupuytren, a jealous man, systematically tried to supress the fame of all these men. Now, imagine him on hospital rounds, wearing a white butcher's apron and a red night cap with a blue silk tasse. When he encountered a patient who hadn't followed directions, he often would flatten him on the spot[1]! He later became a Baron of the French empire. They say he had a personality more to be respected than admired[10].

You can remember Nelaton for his line if you like. I'll also remember him for a hemorrhoidectomy he did, without anesthesia, on the famous French artist, Gustave Courbet. Courbet said that it "lasted forty-five terrible minutes" but was satisfied with the result. He paid Nelaton with a painting of the Duval garden[13].

So, eponyms give flavor, give life. Two of my early favorites were Chopart's (joints, amputation) and Lisfranc's (joints, amputation). Other places don't use eponyms as much as we did at Iowa, but that was a part of our particular style!

Today, I want to discuss the life and times of Francois R. Chopart (1743-95) and of Jacques Lisfranc (1790-1847). Their lives covered just over one hundred years in the history books during a turbulent era. Francois died at age fifty-two, Jacques at age fifty-seven. As I write this I am fifty-six, so permit me a feeling of at least small kinship!

Chopart was born the same year as Thomas Jefferson, Jean Paul Marat (French revolutionist, assassinated by Charlotte Corday as he sat in his bath. Remember David's painting?). Turbulent era, no?

Doctor Joseph Ignace Guillotin was a humanitarian who fought a proposed tax on vinegar and set forth a plan to drain the swamps[14]. Beheading with a sword or axe could be difficult. The executioner often missed the neck altogether and then had to take as many as three or four strokes to get the job done; sometimes a knife had to be used to finally stab the victim to death! Guillotin made a sketch, Louis designed the mechanism and a German pianomaker, Tobias Schmidt, made the prototype of the execution apparatus[21].

Lisfranc was born the same year as James Fenimore Cooper, Lord Byron and Louis Daguerre (pioneer in photography). Ben Franklin died this year.

Medicine was only beginning to rise above the superstitions and fallacies of the middle ages. The profession was rightfully accorded grudging small respect[16].

The writings of another French physician, Francois Rabelais (1490-1553), were still in the center of literary attention. The adventures of Gargantua and Pantagruel attracted much more favorable comment than the medical literature of the time! Rabelais (Fig. 1) practiced medicine most of his life; for many years he was of the faculty at

Figure 1
Rabelais[23]

was no real reform in French medicine until after the revolution. Moliere (pseudonym of Jean Baptiste Poquelin, 1622-1673; see Fig. 2) in his plays "Tartuffe" and "L'Amour Medecin" heaped scorn on contemporary physicians[16].

Figure 2
Moliere and his leading lady, Madeleine Bejart, probably posed for this paining by Mignard[21].

Guy Patin's famous letters also exposed the hypocrisy of his fellow physicians in the pre-revolutionary era. They relate an altogether lively picture of the customs of his time in Paris[16].

Goethe (1749-1832) also lived in this era. His immortal words "All medical skill is for naught when an angel pisses in the touch hole of your musket!" *deserve* to live on! He also mentioned an early upper prosthetic wearer, Gottfried von Berlichingen (an illustrious German knight with an "iron hand" in a velvet glove), in one of his dramas[8].

So, a little flavor of the era in which Chopart and Lisfranc lived. Early mention of amputation adds to the story. The word amputation is derived from the Latin preposition "ambi" meaning around and "putare", to prune.

Ever hear about poor Hegesistratos? Heroditus (484 B.C.) tells us that he was imprisoned by the Spartans and condemned to die. He cut off his foot, which chained him to his death cell, fled thirty miles to the city of Tigea (presumably on foot <singular>) where he fitted himself with a wooden leg after his wound had healed[8]. I just know they don't grow them like that any more, even though an occasional orthopedic resident at Albany claims to have had a similar experience!

Horuk Barbarossa, corsair and pirate, had an artificial hand after injury in battle[8].

Lord Horatio Nelson rose to heights as an admiral after loss of both an eye and an arm.

Technology can set limits, but intuition counts for much. Peter Lowe came to Galsgow as Professor of Medicine after being a master surgeon at the College de St. Come in Paris. His classic "Chyurgerie" was published in 1597.

the great medical school at Montpelier[10]. I still remeber the great sense of relief I felt in my college days on realizing one could write this type of thing and get away with it! The hygienic practices of Gargantua were a sophomoric high spot for me (After Greek sophos, wise plus moros, foolish! Further, how does one ever forget it when his professor tells him, in front of a large class, that "no, scatalogical does not refer to a form of improvisational jazz singing — it refers to ——, pure and simple ——"!). Pantagruel's description of the burning at the stake of Professor Caturce by the authorities was truly based on fact. Many of us in academic medicine are (should be) haunted by the possibilities this suggests!

Often despised, Alexandre Dumas, pere (1802-1870) was also a physician, although better known for his literary endeavors. His book, "The Memoirs of a Physician" contains observations of a physician in the era of Lisfranc[7].

Members of the Faculte de Medecine of Paris in the 17th century were inbred, hypocritical and affected. There

He didn't have anesthesia or (formal) antisepsis, but listen to this; he advocated use of a tight "ribben" (tourniquet) because "it stupifies feeling of the part", gives hemostasis "stayes fluxe of bloude", and when loosened holds skin and muscle in place for healing! He also mentioned use of warm claret wine to irrigate the wound (early disinfectant!) and to pacify the patient. Remember how some of the classic sages sat on pillars to dispense their wisdom? Peter Lowe did a unwanted variation well over a millennium later; he was sentenced to stand on a pillar for three Sundays in a row for offending ecclesiastical authorities[5].

This was the era of Chopart. Born in Paris 30 October 1743, he attended Mazarin College (named after the Catholic statesman and Cardinal) and led his class with many honors. In 1761 he also recieved his Master of Arts before enrolling in the medical school at Hotel-Dieu. After graduation he interned at Hospital de la Pitie and Bicetre. At Bicetre he conducted an extensive study of syphillis[3,12].

Early in life, he developed repeated episodes of abdominal pain and vomiting which plagued him for the rest of his life.

French hospitals of his time were considered better than English hospitals, but this is small compliment! Separate studies of French hospitals by Tenon and by Cross relate that la Salpetriere (originally a munitions works) was a prison, insane asylum and hospital sheltering as many as eight thousand souls[3].

Hotel Dieu held only fifteen hundred to two thousand patients. For this volume of work there were eight or nine attending physicians, one hundred "dressers" and twenty "les eleves" internes (guess who did the work?). Internes served for two years, and were given room, board and a yearly stipend of twenty guineas (around $60.00). The overcrowding was scandalous; over half the beds were "large beds", designed to hold two or more patients. On busy days, these beds often held six persons, and were vermin-infested[3].

Chopart's studies led him to become an expert in legal medicine, and he studied surgery with Dupuid. During his student days, he earned a double prize for his exposition on the tumor known as lupus, and got an honorable mention for a paper "Counterblow in Lesions of the Head" from L'Academie de Chirugie in 1761. Later he earned his master of Surgery degree with great distinction, writing his thesis in Latin on "Injuries of the Head"[12].

He was named a professor at Ecole-Pratique where he taught anatomy and surgery. When Bordenave died in 1782, Chopart took the chair of Professor of Physiology, and later took charge of Hospice de les Ecoles[12].

We orthopedists remember him mainly for his description of partial amputation of the foot. The final refinement of his technique was described by the otherwise unknown writer Lafiteau in "Observation sur un amputation partielle du pied", Med. Eclaire par les Sciences Physiques, 4, 85-88, 1792[3].

Chopart's major contributions were in other areas. His principle written work was on diseases of the urinary tract, and some call him "the father of urology". His two volume set "Traite des maladies des voies urinaries" was a classic in its time, published in Paris in 1791. Publications were his strength; his lectures were said to be boring in the extreme[23]!

Other papers were on "fungous tumor of the dura mater" and on scorbutic gingivitis. With Desault, he published a two volume treatise on surgical diseases and their operative treatment[12].

Chopart developed strong friendships with many of the leading physicians of his day. Traveling to London on two different occasions, he became a lifelong friend of John Hunter (did you know that John Hunter limped because of an untreated rupture of the heelcord[8]?).

He was also a friend of Astley Paston Cooper. Cooper (Fig. 3) visited Paris in June 1792 to attend the lectures and surgery of Desault and of Chopart. As an added attraction, he visited meetings of the National Assembly, hearing the orations of Danton (later guillotined), Marat (later stabbed to death by Charlotte Corday) and the murderous Robespierre (later guillotined) (Figs. 4, 5, 6). Cooper recorded that he was watching Chopart operate on 10 September 1792 when he heard the first cannon fired as the French revolution began! On two different occasions a mob threatened Astley's life, accusing him of being a French aristocrat. In what had to be the grand finale of his visit, he was there and watched the mob escorting Louis XVI and Marie Antoinette (Fig. 7) to the Temple[2]. One need not go to Las Vegas for excitement at conventions!
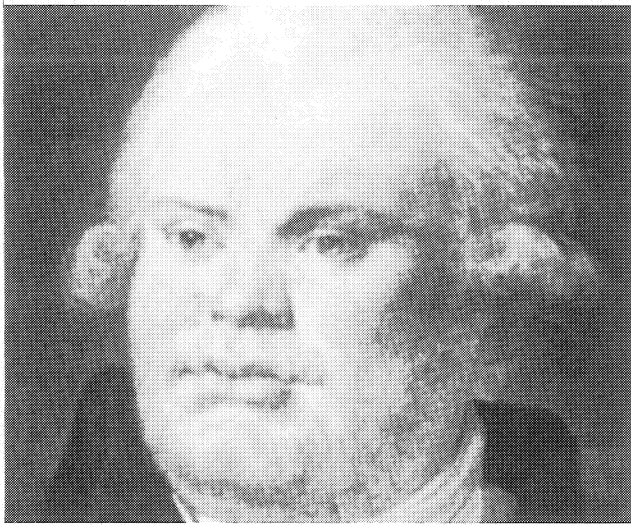

Figure 3
Astley Paston Cooper[2]

Figure 4

Danton[2]

Figure 6

Robespierre[21].

Figure 5
Marat, after being stabbed by Charlotte Corday. (Courtesy of
Life/Time Books. The World of DeLacroix-Tom Prideaux and
editors, 1966).

Figure 7
Louis XVI, Marie Antoinette with the Dauphin and Marie
Therese[21].

Chopart was a member of the French Academy of Sciences, and presumably was acquainted with many of its members. I found an interesting book[17] discussing the academy and containing the eulogies of its deceased members (1699-1791). As Chopart did not die until 1793, he is not included. Some who were:

Denis Diderot, author, critic and editor of the encyclopedia. A wonderful quotation of his; "Virtue commands respect, and respect is a liability!" (Rameau's Nephew[6]).

Giovanni Morgagni (1772) anatomist.

Carl von Linnaeus (1779) the Swedish botanist and his system of classification.

William Hunter (1784) English surgeon, mentioned earlier.

Leonhard Euler (1783), Swiss mathematician. Remember calculus in undergrad? Are you happy nowadays?

Benjamin Franklin (1790), American statesman and sci-

entist. Condorcet's euology included Turgot's words about Franklin "He seized the lightning from the skies and the scepter from tyrants!" Many other famous names are included; these are the ones that caught my fancy.

Everything changes, and nothing changes; Chopart had his share of busy work as councilor of the Comite de L'Academie de Chirugie and later as their corresponding secretary[12]. Reg can tell you about this.

Chopart was chronically ill, had a sad demeanor, and never married. He got some "relief" of his intermittent hemoptysis by venesection (if you have less blood, less to cough up). Biographers tell us he had frequent episodes of "colick" and what they finally called "cholera morbus". He ate a large dinner on 23 May 1795, followed by a violent colicky attack and died. His autopsy revealed death due to volvulus of the sigmoid colon[12]. Remeber the old saw "A surgeon is a doctor who knows nothing and does everything. A pathologist is a doctor who knows everything, but a day too late."

Anyhow, that's a glimpse into the life of Chopart. Jacques Lisfranc (Fig. 8) was only three years old as this famous physician died. Napolean Bonaparte was twenty-one years of age, and poised for his plunge into history. Lisfranc became a brilliant surgeon and lecturer, although not so productive of written works as Chopart.

This was the era of Guillame Dupuytren, already mentioned as Lisfranc's chief at Hotel Dieu.

Despite the bushel Dupuytren placed over his light, Lisfranc bloomed and contributed. He became the first surgeon to surgically excise carcinoma of the rectum, done through a perineal approach. Six of his nine patients survived[15].

In a classic report "nouvelle methode operatoire pour l'amputation partielle du pied dans san articulation tarso-metatarsienne; methode precedee des nombreuses modifications qu a subies celle de Chopart" (Paris, 1815), he devised a more physiologic amputation with better survival than the usual above or below knee amputations done in that era.

Of course, when partial foot amputations are mentioned, the *Pirogov* amputation comes to mind. Pirogov (Fig. 9) knew Lisfranc, meeting him during a visit to Paris in 1838. The amputation is essentially through the ankle, but the calcaneous is conserved, turned up and fused to the plafond. He called this "resectioning" and mentioned his attempt to keep the largest number of uninjured parts. (Pirogov, N.I.:Osteoplastic amputation of the foot, Voyenno M. Journal, 63; second series, 83-100, 1854. for English accounts, London M. Times and Gos. 11;1621, 1855. 15:274. 1857.) Plaster dressings were used in association
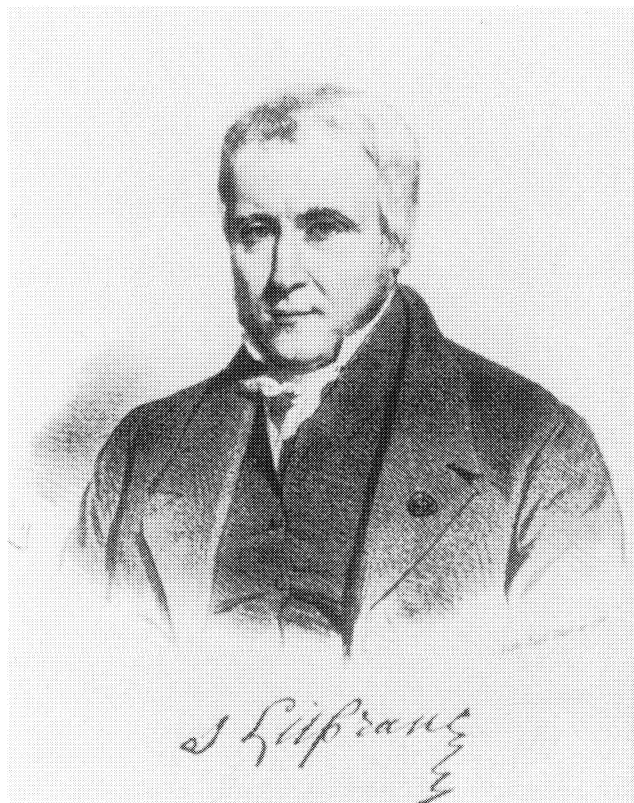

Figure 8
Lisfranc (courtesy New York Academy of Medicine)

Figure 9
Pirogov[19]

with his amputations, and he was (at the least) one of the first to use plaster in this way. Nikoli Pirogov (1810-1881) is called the founder of the present day Russian system of surgery. A child prodigy, he graduated from medical school at age eighteen after his father lied about his age on his birth certificate. He became professor of surgery at Dorpaty at the ripe old age of twenty-six! He wrote a classic work on arterial and fascial anatomy, and later when at St. Petersburg did more than twelve thousand autopsies over a period of fourteen years. Such was his fame, that the Italian patriot Garabaldi came to him in Russia in 1862 with a bullet wound. None of the European physicians were able to locate the bullet. Not only did he find the bullet, but he treated Garabaldi until he recovered. Like John Hunter, Pirogov developed a mania in his later years; a fear of the dark, associated with hallucinations. When he died of carcinoma, his body was embalmed and laid in a vault in Chernigovskaya, Gubernia province in Russia. It rests there to this day, under a glass cupola, visible to those who go to pay tribute[19]. This may well be the precendent for the later enshirnement of the body of Lenin (V. I. Ulyanov) in Moscow.

It may be inappropriate to discuss modes of interment here, but listen to what Winston Churchill had to say when someone asked him about the best method of handling the funeral of one's mother-in-law; "By all means, have her embalmed, cremated and buried. One must be sure!"

Lisfranc also became known for his techniques of resection of the jaw, and of shoulder disarticulation[4] ("Nouvelle Methode Operatoire", 1815). He invented a two-bladed

vaginal speculum which was described in his "Maladies de l'Uterus" which was published by Jean Pauly in Paris, 1836[15].

Lisfranc knew other interesting people. One was the famous, unhappy and notorious Dr. Robert Knox. Knox was the Scottish physician caught up in the scandal of body-snatching for anatomic dissection in medical schools. His name is forever tied to the murderers Burke and Hare. It is recorded that Knox spent a year of postgraduate study in Paris in 1821. Mainly at La Charite Hospital, he also spent some time at two other public hospitals, Ecole de la Medecine and Hopital de la Pitie. He was said to have returned to Scotland a confirmed Francophile, full of contempt for all Scottish doctors save John Hunter and John Bell! In June 1815 Knox was sent to Brussels to attend English soldiers wounded in the Battle of Waterloo. Sir Charles Bell was there, and Knox noted that only one of Bell's secondary amputations survived (no mention of how many he did)[18]!

Another famous though senior contemporary of Lisfranc was Baron Deminique-Jean Larrey (1776-1842)[9,12,20]. Larrey (Fig. 10) trained at Hotel Dieu and Hotel des Invalides in Paris. He came to North America on a French navy frigate in 1787 during his tour of duty. On his return he studied surgery with Sabatier, and later during army service devised the "ambulance volante", the flying ambulance service (Fig. 11) that brought first aid to the battlefield for the first time. This is a detail of a painting showing the Grand Army fleeing Russia. Before, the injured faced a random death on the field. Larry instituted major advances in military surgery.

Figure 10

Larrey[8]

Figure 11
One of Larrey's "flying ambulances" during the Russian campaign[21]

His "Memoirs of a Military Surgeon" are just fascinating reading[11]. Hold an old gold and leather-bound book, and read "if gangrene be traumatick (sic), no time should be lost in removing the limb above the sphacelated part". Then, half a book later he talks of conservation, "In general, if the body of a bone be crushed by a ball and there is no loss of substance of the soft parts amputation is not required and preservation of the limb should be attempted according to the indications". Remember the time and the circumstance. How would you handle it if you were there, with the same knowledge and physical limitations?

Napoleon made Larrey surgeon-general of the army in 1805. He was made a Commander in the Legion of Honor and made a Baron, both in 1812. Wounded and captured at Waterloo, he found fame to be fickle. When the Bourbons returned to power Larrey was stripped of his honors and privileges because of his association with the "little corporal". From exile, Napolean thought highly of him and left him one-hundrend-thousand francs in his will. Larrey was the first to describe trenchfoot, and most surely did more amputations than any other man as a result of his battlefied experiences (sixty battles and four-hundred engagements). He did over two-hundred amputations at Borodino alone[8]! He died a rather mundane death at home in bed, probably of congestive failure, in Lyon in 1842.

Lisfanc died, full of honors, in 1847. Even as he died, another revolution began in Paris; the King abdicated and Louis Napolean was elected President of the French Republic. Everything changes, and nothing changes.

For the French, Lisfranc is still a living presence. See Lisfranc, J., "Memoir sur la Rhynoplastie, ou l'art refaire le nez", Ann. Chir. Plast., XXVII(2), 189-93, 1892. Do you think that anyone will really be interested in how any of us did a nose job one-hundred thirty-five years after our death?

Finally, rhetoric demands some serious, meaningful conclusion. Try this;

"This festered joint cut off, the rest rests sound,
This alone, while all the rest confound—"
Richard II-Shakespeare

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