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Volar Fracture Dislocation of the Second Carpometacarpal Joint Treated by Open Reduction through a Dorsal and Volar Approach

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ABSTRACT: An isolated volar fracture dislocation of the second carpometacarpal joint is a rare injury. This paper represents only the second reported case of its type. It is presented for its uniqueness in that two fracture fragments of the second metacarpal were removed through a volar incision and reinserted through a dorsal approach where the fragments were anatomically reduced. No evidence of avascular necrosis or fracture sequelae was noted. A review of the literature and anatomic relationships are discussed. **KEY WORDS:** Volar Carpometacarpal Dislocation; Second Carpometacarpal joint.

INTRODUCTION

Fracture-dislocation of the Carpometacarpal joints is a rare occurrance. In a review of the American literature dorsal carpometacarpal dislocations have been reported more often than volar carpometacarpal dislocations.[1] In 1948 Waugh and Yancey reviewed 31 carpometacarpal dislocations. Out of 31 reported dislocations only 5 were volar dislocations. Only one case of an isolated volar fracture-dislocation at the second carpometacarpal joint has been reported.[2]

Previous reports have indicated that successful treatment of this type of injury can be achieved by either closed methods or open reduction. This case report is presented to document this rare injury, review the anatomy involved and to illustrate an alternate method of surgical treatment for this injury.[3,4,5]

CASE REPORT

R.M. is a 15 year old male who sustained an injury to his left hand while playing football in a neighborhood lot. The patient fell forward while running and had another player land on top of his out-stretched left hand. Five days after the injury the patient reported to our clinic for evaluation.

Physical examination at that time revealed generalized swelling over the dorsal and palmar aspects of the left hand. Diffuse ecchymosis was noted on the volar aspect of the left hand with restricted motion noted at the wrist and second metacarpalphalangeal joint. Ulnar instability of the first metacarpalphalangeal joint was noted, however at the time of surgery this was demonstrated to be an old injury. Roetgenograms were taken which revealed a volar "Y" type intrarticular fracture-dislocation of the second carpometacarpal joint.(Fig. 1) Also present was a fracture at the base of the third metacarpal which was maintained in good alignment.

The patient was admitted to the hospital the day of his initial evaluation. His left hand was splinted and kept elevated for an additional four days to allow for further reduction in swelling. At the time of surgery a dorsal transverse incision was made over the second carpometacarpal joint. It became immediately apparent that all soft tissue attachments to the fracture fragments of the second metacarpal were severed and these fragments were interposed between the fibers of the Adductor Pollicis muscle.

Attempts were made without suc-

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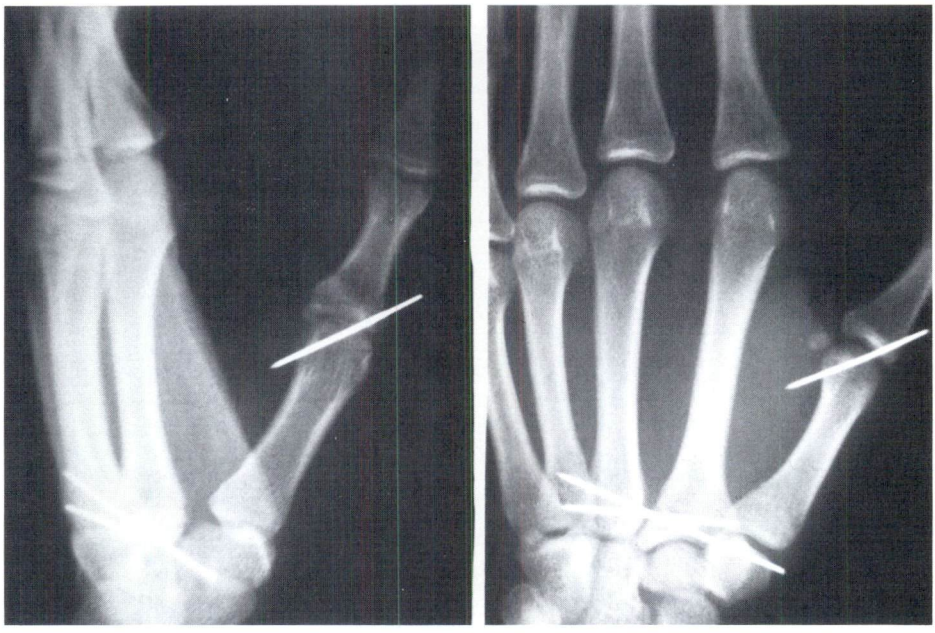

FIGURE 1A

Final reduction of fracture fragments is noted with 0.045 Kirschner wire fixation. A third wire can be seen for the repair of the ulnar collateral ligament.

Figures 1B and 1C: Demonstrate the final result at six months follow-up. The patient had full range of motion with no pain or radiographic sign of avascular necrosis.

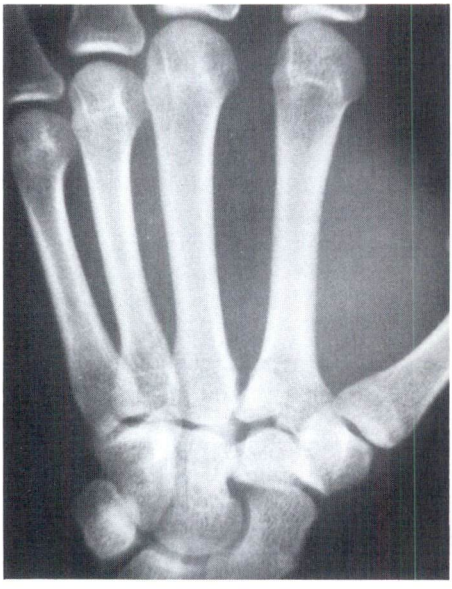

FIGURE 1B

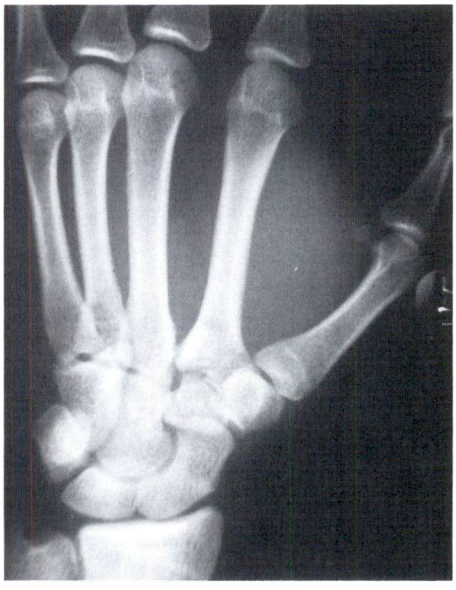

FIGURE 1C

cess to reduce the fragments through the dorsal inicision. At this point a volar curvilinear incision was made just ulnar to the thenar crease.(Fig. 2) This incision extended from the distal carpal crease proximally to the proximal palmar crease distally. The fibers of the Adductor Pollicis muscle were split and both fragments of the second metacarpal were removed. These frag-

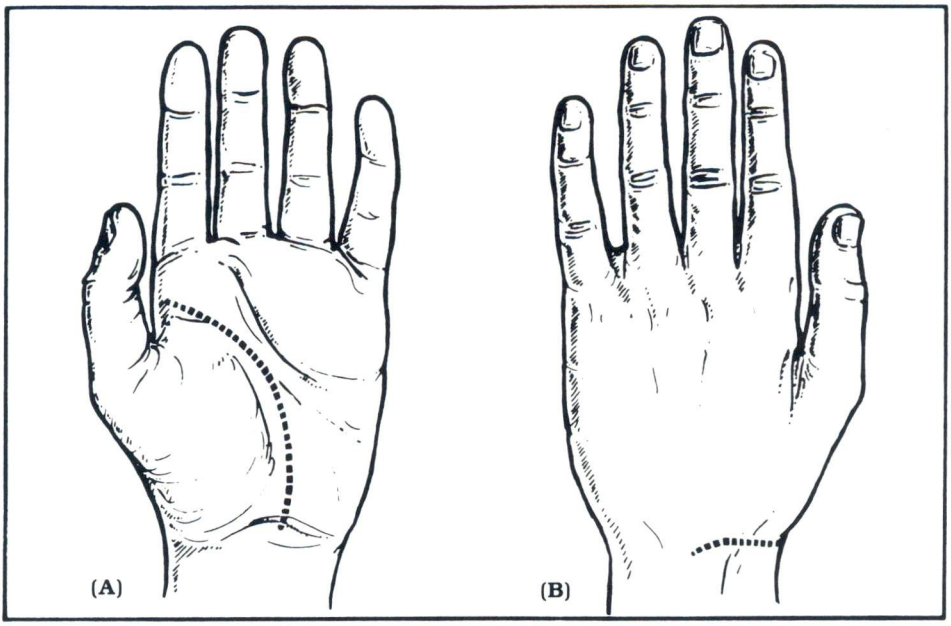

FIGURE 2

Figure 2. (A) The curvilinear Volar Incision (B) The Dorsal Incision. Both Incisions were utilized in reducing and anatomically securing the fracture fragments.

ments were then reinserted into the carpometacarpal joint through the dorsal incision.

The reduction was achieved and stable fixation accomplished by crossing two 0.045 Kirschner wires through the fracture fragments and imbedding the wires into the third metacarpal. During this surgery the ulnar collateral ligament of the first metacarpalphalangeal joint was exposed through a separate incision. An old tear of the ulnar collateral ligament was noted and reconstruction was performed.

Postoperatively the patient progressed through an initial three week period of immobilization. At six weeks motion was instituted at the second carpometacarpal joint. With continued rehabilitation complete return of function was achieved. At six months the patient had full range of motion at the second metacarpalphalangeal joint. Roentgenograms demonstrated union of the fracture with an acceptable reduction. The possibility of avascular necrosis existed, however at no time was the vascularity to the fracture fragments compromised.

The final x-rays demonstrated a congruous joint surface at the carpometacarpal joint with no evidence of avascular necrosis in the fracture fragments.

DISCUSSION

The carpometacarpal joint of the index finger relies on its tendonous, ligamentous, and bony attachments for stability. Radially, stability is achieved by articulation of the second metacarpal with the trapezium. Ulnar stability is achieved by articulations with the capitate and third metacarpal.[1]

The Dorsal, Volar, and Interossious ligaments in this concaveconvex joint serve to restrict its motion. Essentially no motion is present at the carpometacarpal joint of the index finger. The dorsal and volar carpometacarpal ligaments secure this joint in an "A" frame fashion. Interosseous ligaments are present to secure the base of the second metacarpal to the congruous portions of the first and third metacarpals.[1,5]

Direct trauma is the usual cause of

this injury. Shorbe attempted to produce carpometacarpal dislocations in cadavers. In his study volar carpometacarpal dislocations were only produced after the volar carpometacarpal ligaments had been sectioned. He noted that the metacarpals would fracture before dislocation would occur.[6] In this case report the volar carpometacarpal ligaments may have been weakened at the time of the original injury to the thumb.

A Closed reduction can be accomplished if the fracture fragments have not lost their soft tissue attachments or have become interposed in soft tissue. However because of the tremendous forces needed to produce this injury, a closed reduction may be unstable.[1,7] Nalebuff has proposed a technique of closed reduction and percutaneous pinning.[4]

When attempts at closed reduction have failed and open reduction is necessary, it is important to recognize that soft tissue interposition may occur. In achieving satisfactory reduction, both a dorsal and volar approach may be considered as a alternative.

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Adolescent Painful Scoliosis Caused by Osteoid Osteoma A Series Review

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Lloyd L. Mrstik, D.O., Garden City, Michigan

ABSTRACT: Eleven cases of Osteoid Osteoma of the spine were diagnosed between 1950 and 1980. Pain was localized to the site of the lesion and a rapid scoliosis was present in all cases. Aspirin afforded excellent relief to ten patients. Three patients had neurologic involvement. On tomography a lesion was demonstrated in six of the eleven cases. A bone scan was positive in five cases. Seven patients were treated with excision of the lesion. No patient experienced pain in followup. The spinal curvature decreased in two patients, increased in one patient, remained unchanged in one patient and disappeared in three patients. Four patients were treated without surgery. Two complained of back pain at followup. In three patients the curve decreased and remained unchanged in one patient. Despite evidence for spontaneous remission in some cases, we believe that the lesion should be removed when diagnosed, since the time interval before remission may range from two to six years, and the scoliosis originally functional, may become structural during this time period. **KEY WORDS:** Osteoid Osteoma, Scoliosis, Tomograms, Bone scan, Aspirin Excision.

INTRODUCTION

Backache without a history of trauma in children and young adults is an uncommon complaint which merits a thorough examination. A scoliosis can arise in response to pain from an osseus lesion situated unilaterally in the spine or posterior end of the ribs.[5,13]

The most common cause of a painful scoliosis is the osteoid osteoma, however other lesions such as eosinophilic granuloma, aneurysmal bone cyst, and localized pyogenic osteitis may precipitate a scoliosis.[13]

Osteoid osteoma is a benign tumor of bone that was first described by Jaffe in 1935.[8] In Dahlin's Series, 1978, osteoid osteomas comprised 11% of the benign tumors.[2] At least half of the osteoid osteomas occur in the femur or tibia.[2] Only 9 of 158 cases were localized to the spine.[2]

Osteoid osteomas are twice as common in males than females, and they most commonly affect people in the ages of 10-26.[5,9,10,18]

Pain is the most common symptom which is exacerbated at night, and is typically relieved by aspirin. Radicular pain is common, particularly if the lesion involves the lumbar spine.[9,15]

Physical findings reveal tenderness and muscle spasm at the site of the lesion.[10,15,18,20] Lordosis due to spasm of the spinal musculature is present in many cases when the osteoid osteoma is located in the lumbar spine.[18] Neurologic involvement may be present which can manifest itself as weakness of the limb, cutaneous hyperesthesia, positive straight leg raise and deep tendon reflex changes.[15,18,20]

Scoliosis of the involved segment is associated with a "C-shaped curvature",[15,18], with the apex at the level of lesion, but the typical idiopathic curvature has also been seen in osteoid osteoma.[9,15,18] The lesion is located on the concave side of the curvature and often in the posterior elements.[3,15]

The Roentgenographic features of osteoid osteoma consist of a radiolucent nidus less than one centimeter in diameter, which may contain a small amount of calcific material surrounded by sclerotic bone.[2,5,10,15]

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Tomograms are of benefit in demonstrating the nidus; however, even with multiple repeat projections a central nidus may not be visualized.[9,10,15,20,21] (figure 1) Keim reports a 40% incidence in which tomography failed to show the nidus.[9]

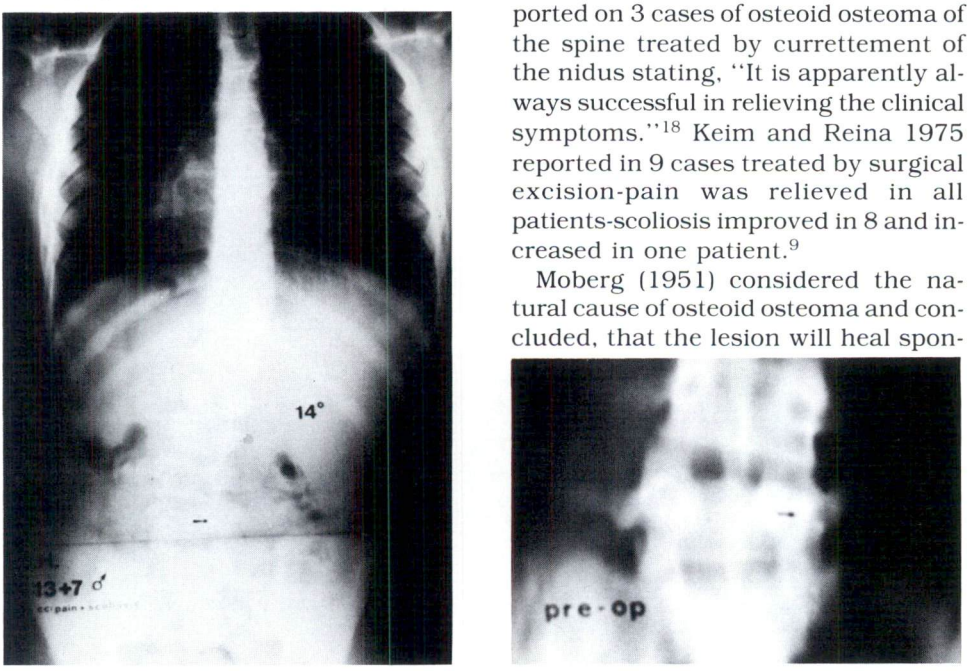


FIGURE 1

Figure 1 J.H. presented with pain and scoliosis of sudden onset (A). Tomograms (B) revealed a radiolucent nidus surrounded by sclerotic bone.

Technetium 99m scintigraphy may be a more sensitive way of diagnosing osteoid osteomas at an early stage. (figure 2) If a skeletal lesion results in less than 30-50% alteration in bone, it is undetectable radiographically.[21] The bone scan reflects osteoblastic activity and increased blood flow within or at the margin of a lesion and this is independent on the material present.[21] There have been no reports of false negative bone scans in histologically confirmed osteoid osteomas.[17,20]

Angiography has also been reported to be of help in the diagnosis of osteoid osteomas.[2,15] Swee and McLeon[20] (1979) reported on two cases of histologically confirmed osteoid osteomas in which angiography was positive.

Maclellan and Wilson in 1967 reviewed the records from New York Hospital and described six spinal osteoid osteomas associated with painful scoliosis and were relieved by excision of the lesion.[12]

Sabanas, Bickel and Moe 1956, reported on 3 cases of osteoid osteoma of the spine treated by currettement of the nidus stating, "It is apparently always successful in relieving the clinical symptoms."[18] Keim and Reina 1975 reported in 9 cases treated by surgical excision-pain was relieved in all patients-scoliosis improved in 8 and increased in one patient.[9]

Moberg (1951) considered the natural cause of osteoid osteoma and concluded, that the lesion will heal spontaneously with time.[14]

MATERIALS AND METHODS

Between 1955 and 1980, eleven cases of osteoid osteoma of the spine were diagnosed at Alfred I Dupont Institute, Wilmington, Delaware and Garden City Hospital, Garden City Michigan.

The clinical data is summarized in Table 1. The ages of the patients ranged from 8 + 6 years to 15 + 10 years with the average being 14 + 10. Males outnumbered females 8-3.

The initial symptoms of the lesion were similar in all patients. Pain was localized to the site of the lesion and scoliosis was present, associated with limitation of motion of the spine. In 10 patients the pain was severe at night: one patient experienced minimal pain

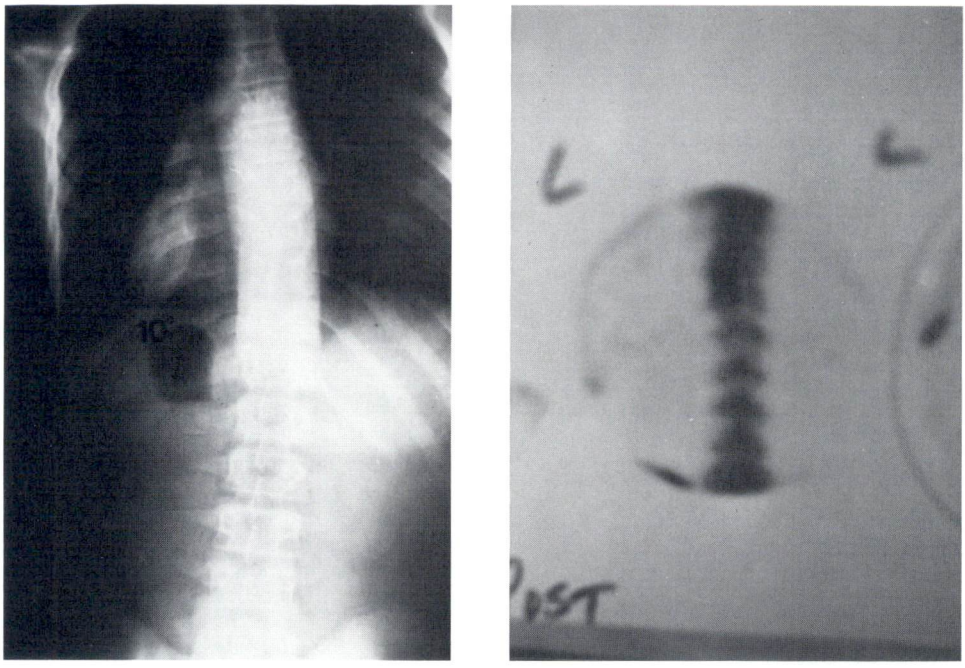


FIGURE 2

Figure 2 Scoliosis with a "C shaped" curvature commonly seen with osteoid osteoma of the spine(A). Technetium scintigraphy reveals an increase in uptake present (B).

TABLE 1

Case	Age	Sex	Vert	Loc.	Loc.	Pain at Night	Relief c ASA	Sympt. to Diag.	Neuro Sympt.	Prev. Diag.	Treatment
1	15+7	F	L_5	L	Lamina	yes	yes	1+4	no	IS	excision
2	13+10	M	T_{11}	R	Lamina	yes	yes	0+5	no	LS	excision
3	15+9	M	T_9	L	Pedicle	yes	yes	1+1	no	IS	excision
4	15	M	T_7	R	Lamina	yes	yes	1+1	yes	TS	excision
5	15+2	M	L_5	L	Pedicle	yes	yes	0+10	no	IS	conservative
6	8+6	F	T_6	R	Pedicle	yes	no	0+2	no	SD	conservative
7	13+2	M	T_{11}	L	Pedicle	yes	yes	0+5	no	00	excision
8	13+7	M	L_5	R	Pedicle	yes	yes	0+11	yes	IS	excision
9	15+10	M	T_{12}	L	Pedicle	yes	yes	1+0	no	LS	conservative
10	13+2	F	L_1	R	Lamina	yes	yes	0+8	no	IS	brace
11	14+7	M	L_2	R	Pedicle	yes	yes	1+2	yes	HNP	excision

KEY: IS—Idiopathic Scoliosis; LS—Lumbar Sacral Strain; TS—Thoracic Strain; SD—Scheurmann's Disease; HNP—Herniated Nucleus Pulposis.

at night. In 10 patients, it was stated that aspirin offered good relief of the pain.

Three patients had signs of neurologic involvement. Two patients (case 8 and 11) had radicular pain associated with a positive straight leg test. One patient (case 4) showed cutaneous hyperesthesia. No patient had reflex changes.

All of the spine lesions were in the posterior elements or pedicles. Five were in the lumbar spine and six in the thoracic spine.

The time between the onset of symptoms and diagnosis ranged from 2 months-14 months; the average being 7 months. Ten of the eleven patients were originally misdiagnosed, by the primary physician; 2-lumbar sacral strain, 5 idiopathic scoliosis, 1 thoracic strain, 1 herniated nucleus pulposa and 1 Scheuremann's disease.

Seven patients demonstrated the typical "C" curve associated with osteoid osteoma of the spine.

Seven patients were treated by surgical excision of the lesion. Spinal fusion was not performed in any case. Histopathology confirmation in the seven patients (table 3) revealed osteoid osteoma in four patients and inflamma-

TABLE 2

SCOLIOSIS DATA

	Case Roent.	Curve Pat.		Tomog.	Bone Scan	Follow-Up	Scoliosis	Eval. Final
1	Sclerosis Lamina L_5	L-T_{11}-L_4	48°	showed nidus	pos	3 yrs	L-T_{11}-L_4	45°
2	Sclerosis Lamina T_{11}	L-T_8-L_3	37°	showed nidus	not done	9 yrs	L-T_8-L_3	35°
3	Sclerosis Pedicle T_9	R-T_5-L_1 L-L_1-L_5	41° 21°	showed nidus	pos	4 yrs	P-T_5-L_1 L-L_2-L_4	46° 22°
4	Sclerosis Lamina T_5	L-T_2-T_{10}	15°	failed to show nidus	not done	18 mo	no curvature	
5	Sclerosis Pedicle L_5	L-T_3-L_2 R-L_2-S_1	28° 16°	failed to show nidus	not done	2 yrs	L-T_3-L_2	14°
6	Sclerosis Pedicle T_8	L-T_3-T_{12}	14°	failed to show nidus	not done	9 mo	L-T_3-L_{12}	6°
7	Sclerosis Pedicle T_{11}	R-T_5-L_4	10°	showed nidus	pos	16 mo	no curvature	
8	Sclerosis Pedicle L_5	R-T_8-L_1 L-L_1-L_5	16° 20°	failed to show nidus	not done	3 yrs	no curvature	
9	Sclerosis Pedicle T_{12}	R-T_8-L_3	16°	showed nidus	pos	6 yrs	R-T_8-L_3	5°
10	Sclerosis Lamina L_1	L-T_8-L_3	15°	failed to show nidus	not done	2 yrs	L-T_8-L_3	12°
11	Sclerosis Pedicle L_2	L-T_7-L_4	17°	showed nidus	pos	18 mo	L-T_7-L_4	5°

The radiographic findings (table 2) were typical of osteoid osteoma in 6 of 11 patients. These six patients, showed a radiolucent nidus containing a small amount of calcific material surrounded by sclerotic bone as seen on tomography. Bone scans were positive in 5 of 5 patients. Six patients did not receive a bone scan. Angiography was not performed in the patients in our study.

All the patients had a history of a rapid painful scoliosis. In all cases the lesion was located at the concave side of the curve. In 9 of 11 patients the lesion was at the apex of the curve.

tion in the remaining three. All patients noticed a relief of pain immediately following surgery.

One patient (case 3) had a reoccurance. Fourteen months after surgery he complained of a painful scoliosis. Radiographs and tomography demonstrated a lesion at T9. Surgery was recommended. The patient refused and he was placed in an orthoplastic jacket. Sixteen months after the pain resolved- however his curve increased 5°. Since then he has been asymptomatic. (figure 3)

Three patients (case 5,6,8) were observed and treated with conservative

therapy. One patient (case 10) was placed in an orthoplast jacket and her symptoms subsided in 6 months.

RESULTS

The average follow up was 2+9 years. (table4)

In the seven patients treated by excision of the lesion no patient experienced pain at follow up. There was one reoccurance case 3. The spinal curvature decreased in two patients (case 1,11), increased in one patient (case 3), remained unchanged in one patient (case 2) and disappeared in three patients (case 4,7,8). (figure 4)

TABLE 3

HISTOPATHOLOGY CONFIRMATION

Case	Biopsy Report
1	Osteoid Osteoma
2	Inflammation with Cancellous Bone
3	Osteoid Osteoma
4	Inflammation Cancellous Bone
7	Osteoid Osteoma
8	Inflammation with Cancellous Bone
11	Osteoid Osteoma

TABLE 4

Case	Follow-Up
1	Asymnptomatic
2	no pain curve unchanged
3	Asymptomatic curve increased 11°
4	Asymptomatic
5	Minimal pain with exercise
6	no pain
7	no pain
8	no pain
9	pain when flexing lumbar spine
10	Asymptomatic
11	no pain

In the four patients treated without surgery, two patients (case 5,9) complained of back pain. In three patients (case 5,6,9) the spinal curve decreased and one patient (case 10) treated with an orthoplast jacket, the curve remained unchanged.

CONCLUSION

The presence of a painful scoliosis in an adolescent should suggest the possibility of a vertebral osteoid osteoma.[12] A complete radiographic study of the painful area along with tomograms should be obtained. The pain associated with osteoid osteoma is due to changes in the vessel pressure registered by the abundant innervation present.[4] Another explanation is direct irritation of the nerve fibers, included in or near the calcification focus.[14] A scoliosis greater in the supine position than erect should alert the physician, since this is contrary to idiopathic scoliosis.[9,1,10]

The average length of time between onset and diagnosis in our study was 8 months. Maclellan and Wilson[23](1967) and Keim and Reina[9](1975) reported an average span of time between onset and diagnosis of 13 months-27 months. A high level of suspicion is needed in making the diagnosis. Most of our cases were diagnosed 1960-1980 so we feel that physicians are becoming more alert to making their diagnoses.

Mehta[13](1978) stated that the final outcome of the scoliosis is determined by the interaction of two factors, the age of the child in relation to his growth velocity and the duration of symptoms, ie. Early onset + short duration- spontaneous resolution and preadolescent onset + long duration- permanent deformity. Our study did not verify this.

Histologic confirmation of osteoid osteoma was not found in 3 patients, whom underwent surgical excision. There are several possible reasons for this: 1. The nidus may have been so small in relationship to the abundant surrounding sclerotic bone it was removed and not identified; 2. It was missed on histological survey; or 3. Damage to the friable tissue speciman, or separation of the nidus which does not lend itself to proper pathological interpretation.

Evidence for spontaneous remission of osteoid osteoma was made by Moberg (1951)[14] using a lesion in a metacarpal, stating that the natural cause of the tumor ceased to cause symptoms after 2 years. Other indirect

evidence of spontaneous remission is that it seldom occurs in persons over thirty years of age.[1,2,6]

Despite evidence for spontaneous remission in some cases, we believe that the lesion should be removed when diagnosed, since the time interval before remission may range from 2-6 years, and the scoliosis originally functional, may become structural during this time period.

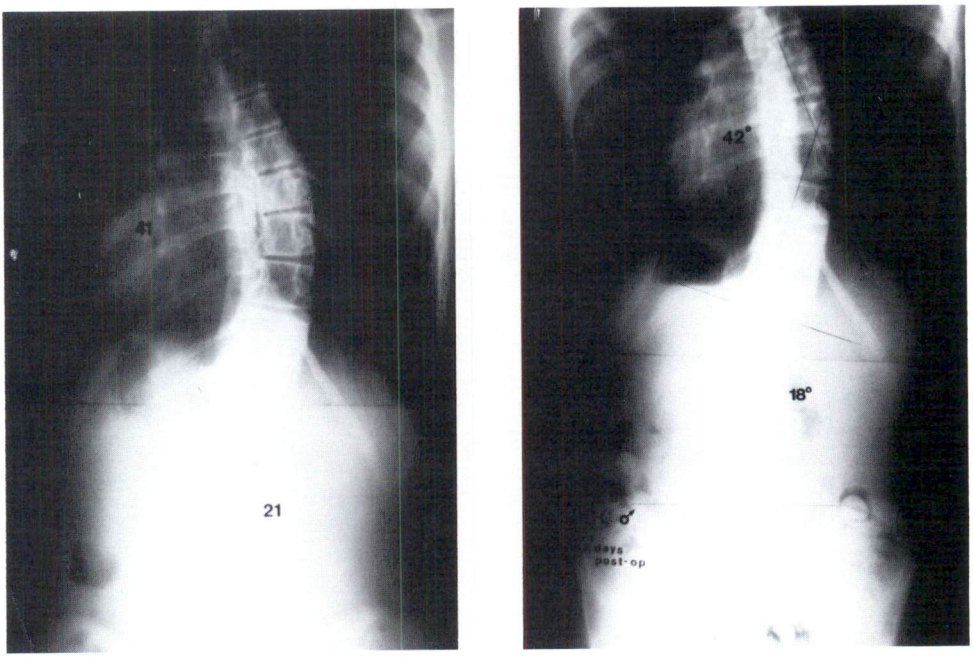
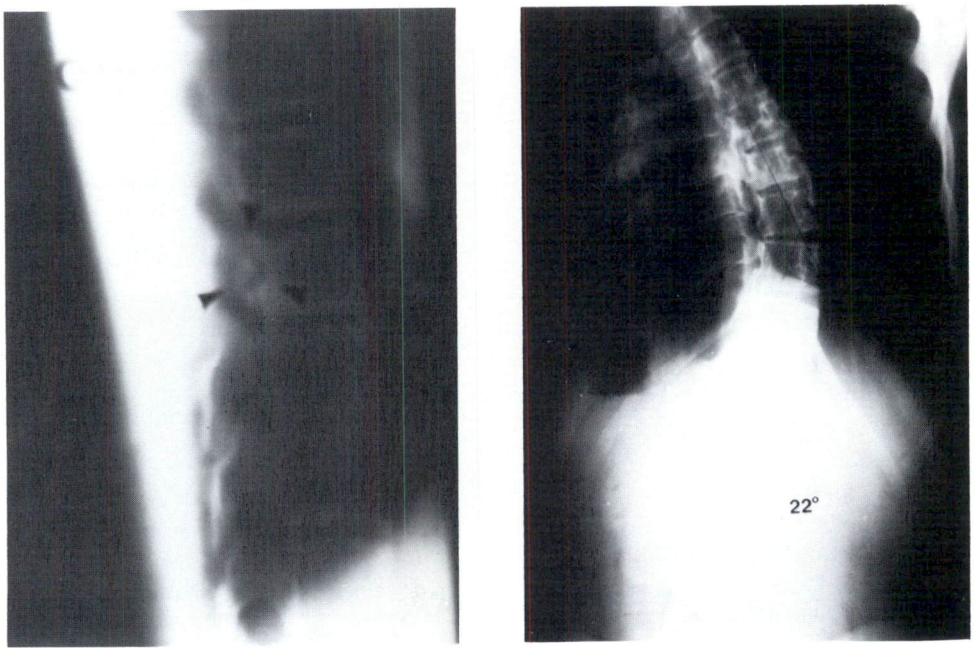


FIGURE 3

Figure 3 O.H. 15 + 9 year old male = painful scoliosis (A). Diagnosis of Osteoid osteoma was made and surgical excision was carried out (B). Fourteen months later the patient complained of back pain, tomograms (C) revealed a reoccurence of osteoid osteoma. Surgery was recommended, the patient refused. Sixteen months later his pain resolved. However, his curve increased 5°. (D)



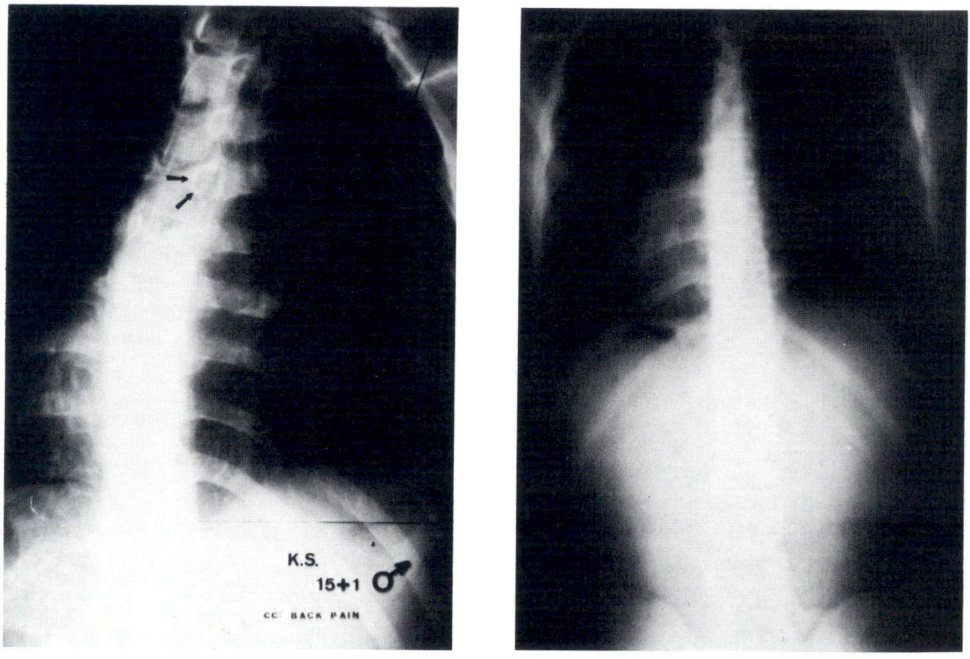


FIGURE 4

Figure 4 K.S. 15 + 1 year old male with a painful left thoracic scoliosis 15°. (A). Excision of an osteoid osteoma was carried out without difficulty. Eighteen months later no scoliosis was present (B).

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Medial Malleolar Fractures Complicated With Disruption of the Posterior Tibial Tendon

Mark J. Reiner, D.O., Cherry Hill, New Jersey

ABSTRACT: Medial Malleolar fractures are frequently seen in an Orthopedic practice. An unusual complication, rupture of the posterior tibial tendon with this fracture is presented. The diagnosis and treatment are discussed in the following case presentation. **KEY WORDS:** Malleolar Fracture, Tendon Rupture.

INTRODUCTION

Ankle fractures are one of the more common traumatic injuries seen by Orthopedic surgeons. Attention is primarily placed on the boney injuries and not to the soft tissue. Operative intervention to these fractures is becoming more commonplace and has made us aware of the associated soft tissue injuries. Recently, I have treated a patient who had a displaced medial malleolar fracture of the right ankle and associated rupture of the posterior tibial tendon. I had personally not encountered this combination before and review of the literature did not reveal any such combination. A recent report in the Journal of Bone and Joint Surgery, February, 1983, revealed two similar cases with disrupted posterior tibial tendons and displaced bimalleolar fractures. This is apparently the first such reported case of a posterior tibial tendon rupture with a medial malleolar fracture alone.

CASE PRESENTATION

The patient is a 23 year-old white male who sustained a displaced medial malleolar fracture to the right ankle secondary to a motor vehicle accident. The patient also sustained head trauma, a fracture to the right thumb and a fracture to the jaw with facial trauma. Initially, due to the severe facial trauma and swelling, a tracheostomy was performed in the operating room several hours following the emergency room evaluation. He was then admitted to the Intensive Care Unit for monitoring and observation. The right ankle was initially splinted in the emergency room. Two days later, when his condition stabilized, he was taken to the operating room for an open reduction, internal fixation of the displaced medial malleolar fracture utilizing an AO malleolar screw. At the time of exploration, there was noted to be a disruption of the posterior tibial tendon. The incision was extended and the sheath of the posterior tibial tendon was opened protecting the neurovascular bundle. The tendon was repaired utilizing 3 0 nylon suture in a Bunnell fashion. The tendon sheath was repaired, the wound closed and the leg splinted with the ankle in a slight plantar flexion.

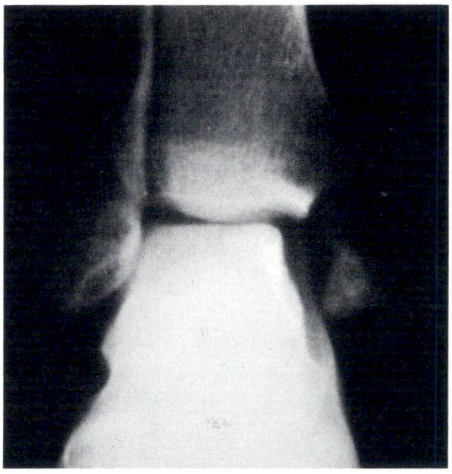

FIGURE 1A

Initial Fracture Displacement

Address correspondence and reprint requests to Dr. Reiner, Cherry Hill Hospital, Cherry Hill, NJ 08002.

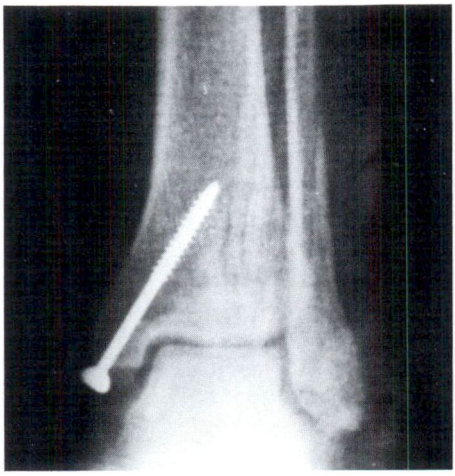

FIGURE 1B

Open Reduction with Medial Malleolar Screw Fixation and Cast

Two days later the dressing was changed. The wound was clean and dry with a minimal amount of swelling present. Neurovascular status to the foot was intact. At this time, a below knee cast was applied maintaining the foot in slight plantar flexion to remove tension from the sutured posterior tibial tendon. At the end of six weeks the cast was removed and the patient was started on physical therapy for range of motion and muscle strengthening to the left leg. The patient responded well and six months later the malleolar screw was removed. Radiographs taken at the time of the screw removal revealed primary healing of the fracture with maintenance of a normal ankle mortice. Clinically, the patient continued to do well ambulating without any problem or any evidence of disability.

CONCLUSION

Although tendon injuries associated with ankle fractures appear to be rather rare, they apparently do exist. It is quite possible that failure or recognize these soft tissue injuries could compromise the functional result of the healed ankle fracture. I am certainly not suggesting that all ankle fractures should be explored for a possible tendon disrupture, but that if one feels it is necessary to perform an open reduction, internal fixation for an ankle fracture the soft tissue should be examined for possible injury.

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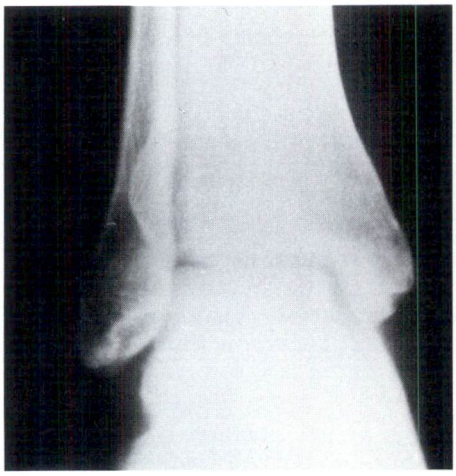

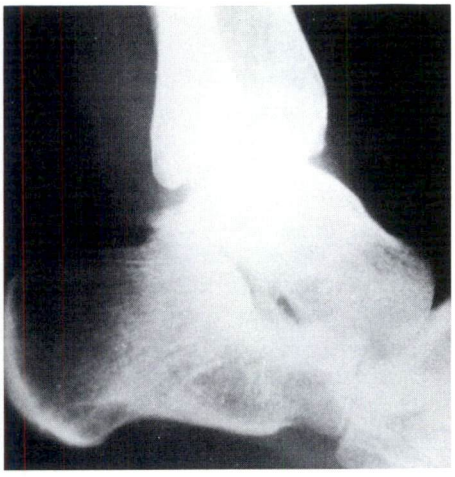

FIGURE 2A & 2B

Follow-up after Screw Removal

The Treatment of Unicameral Bone Cysts With Injections of Methylprednisolone Acetate (A Case Study and Review of Literature)

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Michael J. Fugle, D.O., Pontiac, Michigan
Andrew B. Limbert, D.O., Pontiac, Michigan

ABSTRACT: Unicameral bone cysts have been treated in many fashions through-out the years. In 1973, Scaglietti began directly injecting unicameral bone cysts with methylprednisolone. He has hypothesized that the steroid injection causes destruction of the lining of the cyst wall, allowing osteoblastic repair to begin. A double puncture technique is used to allow easy egress of cystic fluid and prevent hemorrhage within the cyst. Such favorable results have been obtained by this form of treatment that Scaglietti has begun work on other bone lesions using the same technique.
KEY WORDS: Unicameral Bone Cyst, Methlyprednisolone (Depo Medrol), Active Cyst, Latent Cyst.

INTRODUCTION

Unicameral bone cysts occur within the first two decades of life affecting long bones. Most commonly, these lesions are usually asymptomatic, however, pathologic fractures may occur at the site of the cyst causing pain as the chief complaint. At one time these fractures were thought to initiate the healing process within the cyst. With more exacting follow-up, it was found that children suffered subsequent fractures from previously fractured cysts that did not heal and often enlarged post-fracture. Recently, direct steroidal injection into the cyst cavity has indicated a strong healing potential with relief of discomfort caused by the cyst. Figure 1.

PATIENTS AND METHODS

Patient was treated for expanding bone cysts of the proximal humerus. Patient had been previously treated for fractures in or about the cyst in the proximal humerus. Previous pathologic fractures had gone on to complete healing, but the cysts remained and had enlarged from initial evaluation.

Case 1 BL 264263

3-year-old white male presented with a pathological fracture of the right humerus associated with a solitary bone cyst. Fracture was nondisplaced and was treated by conservative therapy with shoulder immobilization sling. History revealed a similar fracture in the same area approximately one year prior, also associated with a solitary bone cyst. Conservative treatment at that time provided complete healing of the fracture. Initially the bone cyst seemed to be healing also. At this time the cyst has enlarged to in-

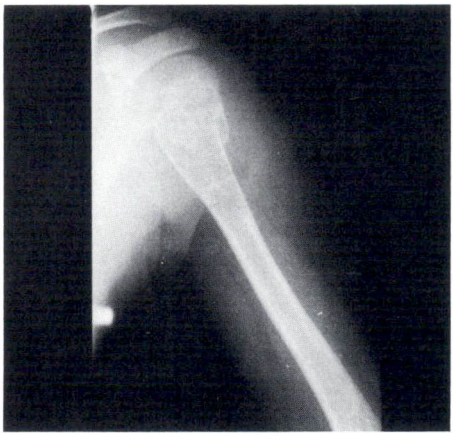

FIGURE 1

At one time fractures through unicameral cysts were thought to induce healing. This fracture did not induce healing in our patient.

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volve the right humerus from the epiphyseal plate to the mid diaphysis. Thinning of the cortex as well as expansion of the cortex was also noted. The lesion appeared to extend into the non-ossified portion of the epiphysis. Physical examination revealed minimal tenderness about the proximal right humerus. Circulation and sensation were within normal limits as was the range of motion of the shoulder and elbow.

Conservative therapy was instituted for the present pathological fracture. Because of the recurrence and the enlargement of the cyst, it was recommended that the cyst be injected with methylprednisolone acetate. Because of the close proximity of the cyst to the epiphyseal plate and the possible damage to the plate during curettage and grafting, injection was recommended.

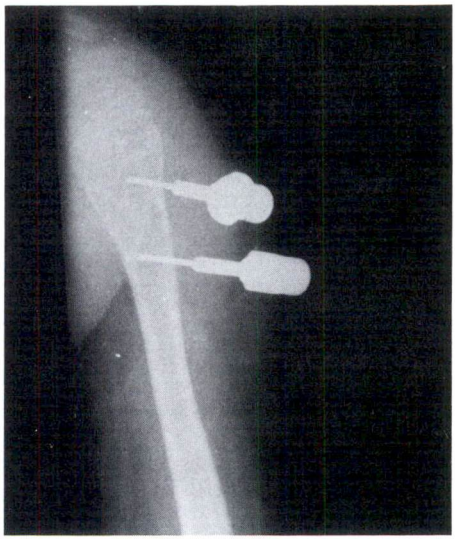

FIGURE 2

The two puncture technique is used to prevent hemorrhage within the cyst and allow easy egress of cystic fluid.

TECHNIQUE

Patient was given general anesthetic in the Department of Radiology. Single stab incisions were made over proximal and distal cyst. Then, under fluoroscopic visualization, Jam-Shidia biopsy needles were placed within the proximal and distal aspects of the cystic lesion. Contents of the cyst were aspirated and sent to the laboratory for evaluation. The cyst was then lavaged with sterile saline until clear. Then 140 mg. methylprednisolone acetate was injected through the proximal needle. The needles were withdrawn and sterile bulky dressings were applied. Patient tolerated the procedure well and was taken to postoperative recovery room in satisfactory condition.

The double puncture technique is used to allow spontaneous escape of cystic fluid; a single puncture with forced aspiration often causes profuse hemorrhage, impairing diagnostic studies on cystic contents.[3] Figure 2

RESULTS

Healing was demonstrated on follow-up radiographic examination by decreased lucency, cortical thickening and increased trabeculation, as represented by the ground glass appearance throughout the cyst. Complete healing cannot be diagnosed until complete skeletal maturity has occurred.

DISCUSSION

Solitary cyst (unicameral bone cyst) usually occur in the first two decades of life; particularly between the years 3 and 14. The long bones of actively growing skeletons are invariably involved. The proximal humerus and femur comprise 80% of total tumor sites. Traditionally, cysts which border the epiphyseal plate are referred to as "actively-enlarging" cysts and cysts more distally located, with a separation of normal trabeculated bone between the cyst and the epiphyseal plate, as "latent" cysts.[6] Attempts have been made to determine cyst age by the distance between the cyst and the epiphyseal plate, but data has been inconsistent to this point. Figure 3

Because the solitary bone cyst is a slow, growing lesion, it rarely causes acute onset of pain. Pain is usually caused when the thinning of the cortex progresses to the point of pathological fracture or periosteal sensory struc-

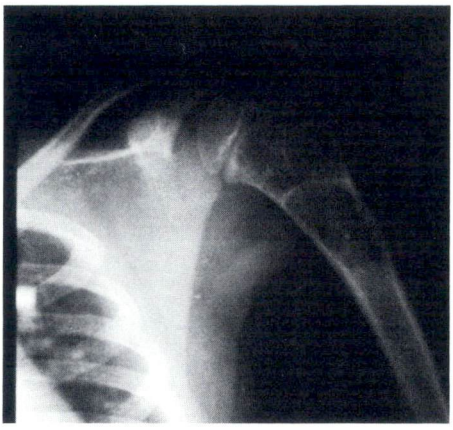

FIGURE 3

Solitary cyst (unicameral bone cyst) proximal humerus.

tures become involved.[8]

Lesions presumably begin in the metaphyseal regions of the long bones. Expansion of the cyst is at the expense of the medullary cavity causing erosion from the interior of the bone. As the cortex is eroded, some external new bone is laid down by the periosteum giving a somewhat fusiform look of expansion. External new bone is in no way able to keep up with internal destruction and the cortex ultimately weakens to the point of fracture.

On dissection, the periosteum is easily lifted away from the intact wall which has a bluish discoloration to it. On penetration of the wall, a typically amber fluid is found within the confines of the cyst.[9] This fluid may change in appearance from a clear to that of pure blood depending on the occurrence of fracture. The cyst is lined with a thin layer of a red-brown membrane, no more than a millimeter in thickness. The membrane consists of moderately young fibrocysts of rather loose texture intermixed with masses of fibrin. Pools of extravasated blood are sometimes present. Xanthoid cells may occur singly or in small clusters. Giant cells occur in varying numbers diffusely scattered throughout the granulation matrix.[8]

Roentgenographic features of a solitary cyst are:

1) Radiolucent lesion involving the metaphyseal portion of long bones.

2) Bone destruction is from the medullary cavity.

3) Subperiosteal new bone formation gives a fusiform appearance of cortical expansion.

4) The defect is round, giving a unilocular appearance and is of soft tissue density due to the thinness of the wall and the fluid contents.

In the past, it was thought that pathological fracture through a solitary cyst would be curative and, in some cases, this has been the case, however, in all too many cases, multiple pathological fractures have occurred with no indication of healing of the cyst and in some cases, the cyst has enlarged. In the past, surgical intervention was initiated due to the diminished strength of the bone in an effort to prevent multiple pathological fractures and possible skeletal abnormalities.[2] 50% of solitary bone cysts are associated with pathological fractures. The active cysts, as mentioned earlier, have had a greater tendency toward recurrence than the latent forms.

The traditional method of treatment has consisted of curettage and grafting the remaining cavity with any of a variety of materials (autogenous bone, bone from bone bank, plaster-of-Paris pellets). This form of treatment has realized a recurrence rate from 25-30%. Cauterization following curettage with either phenol or zinc chloride prior to packing has not decreased the recurrence level. Recently the use of a high-speed bur on the cyst walls after curettage has led to some limited improvement in results.[6] A more aggressive subtotal resection of the entire cyst wall (with or without grafting) has reduced recurrence to 5-10%. In 1973, Scaglietti et. al. began directly injecting methylprednisolone into the cyst cavities with impressive results. Scaglietti confirmed that the fluid contents of the solitary cyst were similar to that of a transudate.[3] Using past knowledge of the efficacy of steroid injection in treating synovial processes associated with transudates, Scaglietti felt that injections of steroids into the cystic cavity would cause resorption of

the cystic fluid. Scaglietti hypothesized that the steroid injections caused destructive changes within the connective tissue lining of the cyst wall, thus allowing osteoblastic repair to begin.[1,3]

Dissection carried out two months post-injection with methylprednisolone demonstrated the cyst cavity to be filled with edematous fibroblastic connective tissue and active proliferation of trabeculae and osteoid bone on the wall of the cyst. This is in direct contrast to the usual histological findings within a solitary cyst; a thin, fibrous, poorly visualized lining with little active proliferation with a few giant cells.[1]

When radiographs failed to reveal satisfactory healing at six months, additional injections were performed up to a maximum of nine times. No abnormalities were found due to excessive doses of corticosteroids. No damage was done to the epiphyseal plate in association with steroid injections. In all cases studied by Scaglietti, any risk of pathological fracture was eliminated from the time of the initial injection of corticosteroid.

The dosage of methylprednisolone acetate was individualized for each patient depending on the age of the patient and the size of the cyst. In general, 40-80 mg. was used in smaller cysts and younger patients. As much as 200 mg. was used in larger cysts.[3]

Methylprednisolone acetate was chosen because it is a microcrystalline suspension that is relatively insoluable and therefore has a prolonged pharmalogical effect.

Following local injection of methylprednisolone into the cystic cavity, the following radiologic signs were used to imply healing: Reduced cyst size and lucency, cortical thickening of both internal and external walls with remodeling of the surrounding bone, and finally increased internal density of the cavity, taking on a diffuse ground-glass opacity. Growth at the epiphyseal plate, usually delayed by the presence of the cyst, became normal once the cyst began to demonstrate signs of healing and shift towards the diaphysis.[2] Figure 4

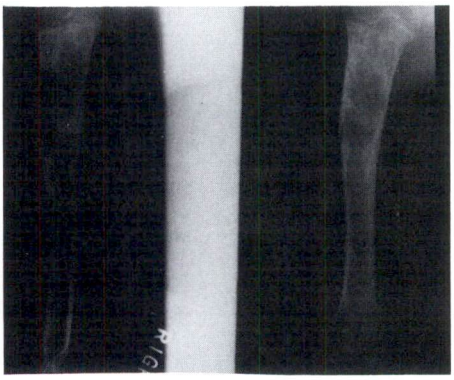

FIGURE 4

Decreased cyst size, and lucency, cortical thickening, diffuse ground-glass opacity with a shift towards the diaphysis indicate early stages of healing.

Few radiographic changes are noted over the first three months, therefore studies during this period were felt to be unnecessary. Healing was defined by complete disappearance of the lesion or the presence of a static lesion showing partial remodeling and increased internal density. Of the 96% of patients with positive results, 60% showed healing within two years. Figure 5

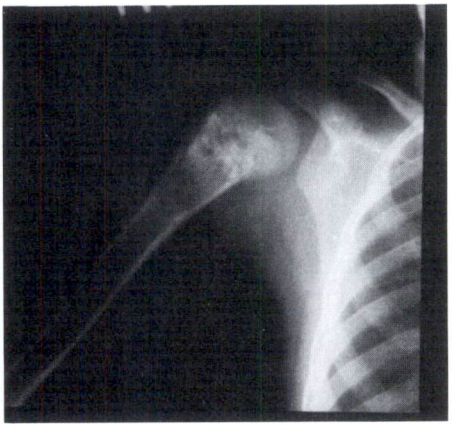

FIGURE 5

Complete opacification of cyst in later stages of healing.

Scaglietti noted that small lucent areas retained within the cyst may become a nidus for renewed cyst formation and should be treated with further injections.

Scaglietti et al. have also treated other osteolytic lesions with injections of methylprednisolone. Through their experiences, eosinophilic granulomas, nonossifying fibromas as well as epiphyseal chondroblastomas have demonstrated favorable repair of cystic lesions with injection.

SUMMARY

Favorable repair of multiple forms of osteolytic lesions have been demonstrated using intercavitary injections of methylprednisolone acetate (Depo Medrol). The mechanism by which local injection promotes bone replacement is not yet clear but present hypothesis indicates that the corticosteroid exerts a destructive action on the pathologic tissue which lines the cystic cavities, allowing osteogenic repair.

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Dislocation of the Triceps Tendon: Is It Really the Triceps? A Case Report and Theory of Etiology

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ABSTRACT: The dislocation of tendons is an uncommon occurrence. Those most frequently encountered in a clinical setting are the tendons of the peroneal muscles, the quadriceps femoris, and the long head of the biceps brachii. In a review of the literature, only five cases involving a snapping triceps tendon have been reported (Table 1). [4,11,14,17] Three occurred in men and one in a woman. Their ages ranged from eighteen to twenty-six. Their complaning of a snapping sensation at the elbow, existed for several weeks to several years in the case of the twenty-six year old patient. It was not the snapping at the elbow which prompted these patients to seek medical attention. Rather, the complaint of ulnar neuritis, which existed for only a few months prior to examination, appeared to be their primary complaint. Only one patient had a history of previous trauma. This was a supracondylar fracture twenty years prior to the onset of symptoms. No boney abnormalities existed in these elbows as has been reported in other types of snapping syndromes. [13] The history of one patient was not reported.

The etiologies and mechanisms, for ulnar neuritis at the elbow, have been extensively researched and reported in the literature. [1,3,5,7,8,12,18,20] The literature, however, lacks an explanation of the causes and biomechanics of this snapping tendon. It is the purpose of this article to present a case report of a dislocating tendon at the elbow and offer a theory for its etiology.

KEY WORDS: Dislocation, flexion, tenography, dorsoepitrochlearis.

CASE REPORT

A seventeen year old, right hand dominant, mentally retarded white male was admitted to the hospital for an intracranial hemorrhage. Initially he was combative, with a decreased level of consciousness and a right hemiparesis. During passive range of motion exercises by the physical therapy department, a snapping was palpated at the right elbow. On the tenth hospital day an orthopedic consult was obtained. Physical examination of the right upper extremity revealed no swelling or ecchymosis at the elbow. The range of motion at the elbow was 0° extension to 140° flexion. Supination and pronation were unrestricted. The elbow had a valgus carrying angle of 5°. There was no apparent atrophy of the intrinsic muscles of the hand. On passive flexion of the elbow to 90°, a subcutaneous, flat structure was felt to slip anteriorly over the medial humeral epicondyle. With continued flexion of the elbow, a larger, cylindrical, subcutaneous structure, which appeared to be the long head of the triceps, became taut. It continued to bowstring and then slipped anteriorly over the medial humeral epicondyle at 130-135° of elbow flexion (Figs. 1 & 2).

Both structures would spontaneously reduce with extension of the elbow. Because of the patient's combativeness and altered state of consciousness, it was hard to determine if this manuever was painful. The family denied any history of trauma to the elbow. A

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TABLE 1
REVIEW OF CASES, INCLUDING THE AUTHOR'S

Patient	Age	Sex	Duration of Snapping	Symptoms Neuritis	History of Trauma	Treatment
1	21	M	weeks	weeks	no	Anterior transposition of ulnar nerve. Resection of muscle/tendon.
2			Not Reported			Anterior transposition of ulnar nerve. Medial epicondylectomy.
3	25	M	3 yr.	few months	no	Diverted muscle through triceps tendon.
4	26	M	years	few months	supracondylar fracture 6 y.o.	Anterior transposition of ulnar nerve. Diverted muscle through triceps tendon.
5	18	F	1 year	three months	no	Anterior transposition of ulnar nerve. Resection of muscle/tendon.
6	17	M	new	new	no	None.

similar shaped structure as that which dislocated on the right elbow at 90°, dislocated at 120° on the left elbow. Plain radiographs of the right elbow were unremarkable. Electrodiagnostic examination of the right and left ulnar nerves revealed an axonocachexia of the right ulnar nerve at the elbow. The left ulnar nerve was normal. Tenography of the dislocating structure demonstrated its position anterior to the medial humeral epicondyle on complete flexion (Figs. 3A,B,C,D). Because of the patient's medical condition surgery was not indicated. Six months after his intracranial hemorrhage, he had fully recovered neurologically. he denied any pain or instability at the elbow. The ulnar nerve and the tendinous structure continued to dislocate anteriorly with flexion.

DISCUSSION

The technique of tenography has been described for those tendons encased in sheaths.[15,16] To the best of the author's knowledge, it has not been attempted on a tendon without a sheath, such as the triceps. Under aseptic conditions, with local anesthesia of 1% plain Lidocaine; approximately two cubic centimeters of Renografin 60 (Squibb) via a twenty-two gauge needle was injected directly into the substance of the dislocating structure with the elbow in acute flexion. Radiographs in the AP and lateral planes were then taken in various degrees of flexion (Figs. 3A,B,C,D). These radiographs clearly show a cylindrical, tendinous structure inserting onto the medial side of the olecranon and originating on the posterior, medial side of the humerus, well above the medial epicondyle.

The triceps consists of two aponeurotic laminae which begin about the middle of the muscle. One covers the superficial surface of the muscle, the other is more deeply seated in the substance of the muscle. The two laminae join together proximal to the olecranon and Anomalous muscles arising from the deep fascia of the forearm and inserting either with the flexor digiti minimi or the abductor digiti minimi have been reported.[10,19] These anomalous muscles passed through the canal of Guyon, where they caused an entrapment neuropathy of the ulnar nerve.

Two anomalous muscles have been described on the extensor, surface of the arm: the apitrochlearisanconeus[6] and the dorsoepitrochlearis.[6] The former is a small, flat muscle which arises from the medial humeral epicondyle and inserts into the medial border

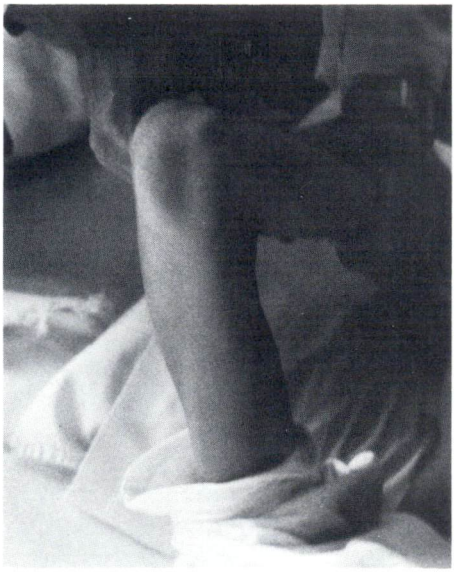

FIGURE 1

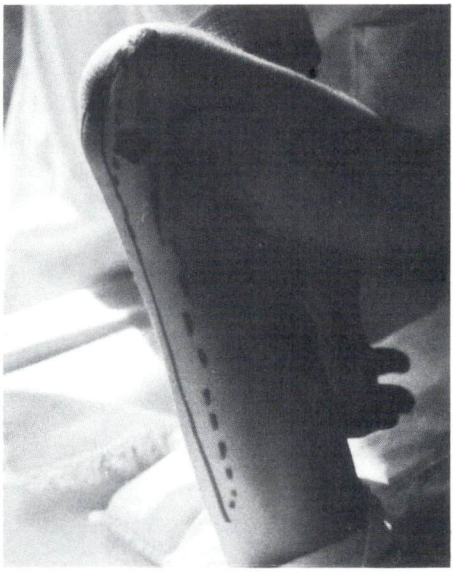

FIGURE 2

Figure 1. With flexion, a band of soft tissue becomes prominent on the medial side of the arm.

Figure 2. The dark circles identify the olecranon and medial epicondyle. The dark line follows the course of the anteriorly dislocated ulnar nerve. The solid and broken lines represent the location of the dislocating muscle prior to and during dislocation respectively.

of the olecranon. It has been responsible for an entrapment neuropathy of the ulnar nerve[7], but has not been associated with displacement anterior to the medial epicondyle. In primates, including man, the dorsoepitrochlearis arises from the inferior border of the tendon of the latissimus dorsi, near its insertion, and inserts on the long head of the triceps brachii. The homologus muscle in carnivores, ie. cats and dogs, is the epitrochlearis.[9] In these mammals, it takes its origin from the latissimus dorsi and inserts on the medial side of the olecranon (Fig. 4). In rabbits, the epitrochlearis takes its origin more distally, on the medial side of the long head of the triceps brachii, and inserts on the medial side of the olecranon.[9]

If the dislocating structure described in this case report represents an anomalous muscle, namely the dorsoepitrochlearis, several factors would predispose it to dislocate anteriorly around the medial epicondyle. Firstly, it is not bound down by the tethering fibers of the medial head of the triceps.

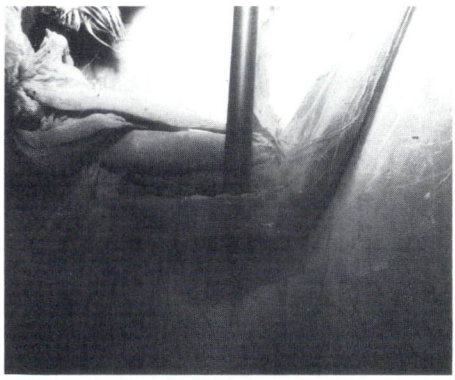

Figure 4. The epitrochlearis muscle of a cat, (ventral view of L. foreleg), lying superficial to triceps (and black pointer). Notice origin from tendon of latissimus dorsi and insertion onto medial side of olecranon.

Secondly, it does not participate in the extensive aponeurosis of the triceps tendon. Thirdly, it inserts more medially on the olecranon than the triceps. Finally, the medial rotation of the proximal end of the humerus is much greater in man than in other primates and carnivores (Figs. 5 and 6).[6] This rotation is termed the angle of torsion

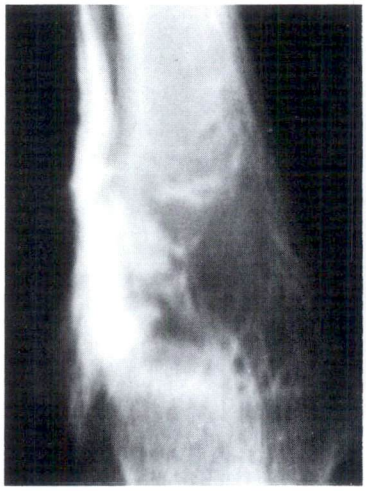

FIG. 3A

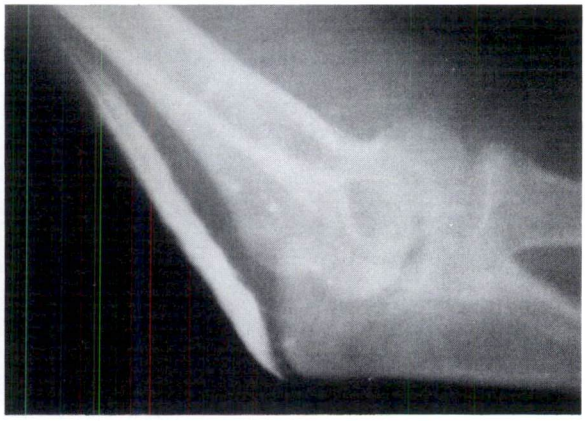

FIG. 3B

FIG. 3C

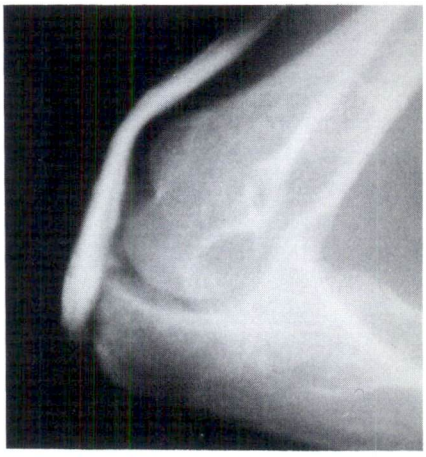

FIG. 3D

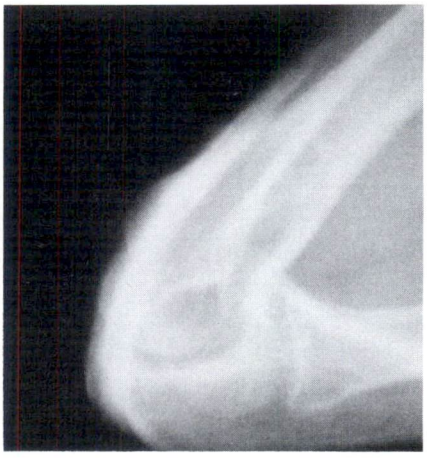

Figure 3A. AP view of contrast outlined tendinous structure inserting into medial side of olecranon. 3B: Lateral view of contrast outlined tendinous structure in extension. 3C: With moderate flexion, the tendinous structure begins to bowstring against medial epicondyle. 3D: With complete flexion, the tendinous structure is seen anterior to medial epicondyle.

of the humerus. The angle of torsion of the humerus ranges from 95° in carnivora, to about 100° in monkeys and 120° in apes. In man it ranges from 135° to 165° or more. It is greater in men than in women, and greater in adults than children. That is to say, the angle of torsion continues to increase until the head of the humerus unites with the diaphysis at about twenty years of age in males and about eighteen years of age in females. As the angle of torsion increases, the insertion of the latissimus dorsi is brought anterior. Therefore, the origin of the dorsoepitrochlearis is more anterior in man than other mammals. As the elbow is flexed, the olecranon process comes to lie anterior to the medial epicondyle, therefore the shortest distance between the origin and insertion of the muscle is on a plane anterior to the epicondyle. This forces the tendon to dislocate or "snap" around the boney prominence.

CONCLUSIONS

From a review of the literature and a study of the case presented, the following conclusions can be drawn: 1) The anatomy of the triceps muscle and tendon makes it particularly difficult to dislocate. 2) Single or repetitive trauma to the elbow is not a factor in producing this dislocation. Only one of the five previously reported cases described any trauma to the elbow and that

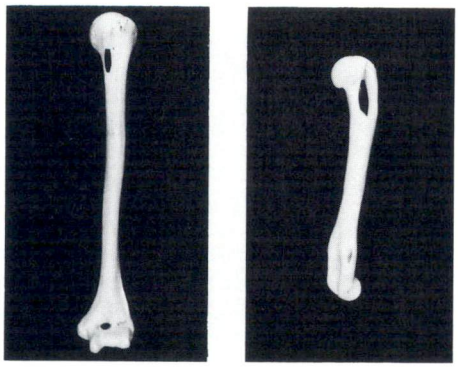

Figure 5. Anterior view of L. human humerus. Black mark just inferior to lesser tuberosity denotes insertion of latissimus dorsi, anterior to medial epicondyle. Angle of torsion places the axis of the humeral head and condyles nearly parallel. Figure 6. Medial view of L. cat humerus. Black mark just inferior to lesser tuberosity denotes insertion of latissimus dorsi, posterior to medial epicondyle. Angle of torsion, places the humeral head at 90° to axis of condyles.

trauma was several years antecedent to the patient's symptoms. 3) Anomalous muscles do exist and the dorsoepitrochlearis, when present, is found on the extensor surface of the arm. 4) The biomechanics of this anomalous muscle would lend itself more readily to dislocation. 5) The age at which this spontaneous dislocation presents itself coincides with the timing of the achievement of the greatest angle of torsion of the humerus. 6) The preponderance of cases documented in males also correlates with a greater angle of torsion of the humerus. 7) The tenographs show a tendinous structure with apparently the same diameter in the AP and lateral planes. If this represented a part of the triceps tendon, one would expect it to have a much smaller diameter in the lateral projection.

SUMMARY

A case report of a dislocating tendon on the extensor surface of the arm has been presented. The possibility that this represents an anomalous muscle, the dorsoepitrochlearis, and that the anatomical features of this muscle predispose it to anterior dislocation is offered as the etiology of this disorder. Furthermore, the possibility exists that the other five reported cases also represent the dorsoepitrochlearis when the anatomy of the triceps is remembered and comparisons are made of the age, sex, and spontaneity of the dislocation.

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The Ace Colles Fixator in the Treatment of Comminuted Fractures at the Distal Radius

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Edward Loniewski, D.O., Farmington Hills, Michigan

ABSTRACT: During a 2 year period at Botsford General Hospital all Colles fractures were evaluated for their stability and classified according to the Frykman classification. Ten patients with unstable distal radius fractures were placed in either the Ace Colles Fixator or the New Ace Colles Fixator.

Patient assessment revealed that 80 percent of the patients had good functional results. Complications included late collapse of two fractures with one of the fractures also developing radioulnar arthritis. **KEY WORDS:** Colles Fracture; Frykman Classification; External Fixator; Radio-Ulnar Joint.

INTRODUCTION

The unstable Colles fracture has been recognized as a difficult fracture to manage. These fractures inherently will shorten, displace, or collapse with angular deformities of 20 percent or greater.[1,2] It becomes a challenge to maintain alignment without losing the reduction or developing angulation. Particular concern arises over the comminuted-intra-articular fracture where loss of normal articular congruincy has developed.

Various methods of treatment have been advocated to deal with this problem. In 1929 Bohler advocated the fixed traction technique utilizing pins in plaster.[3] This has seen several modifications by Darrach[4], Marsh, and Teal[5], and Cole and Obletz[6] In 1944 Anderson and O'Neal developed a triangular external fixator device.[7] In 1952 DePalma encouraged insertion of a rush rod to maintain length of the distal radius.[8] More recently Mital and Patel published their results with percutaneous pinning of the Colles fractures fragments utilizing Kirschner wires.[9] In 1975 Sarmiento developed the functional cast bracing technique with the forearm held in supination.

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In 1979 William Cooney and Ronald Linscheid published their results utilizing a quadrilateral Roger Anderson frame for the fixation of comminuted intra-articular Colles fractures.[10] Their study led them to devise a quadrilateral frame which provided tham with better fixation and greater stability than the Anderson device. This frame has been marketed as the "Ace Colles Fixator".

In this study 10 patients were treated utilizing either the original Ace Colles fixator of the "New Ace Colles fixator" as the preferred method of treatment.

MATERIAL

In treating Colles fractures at Botsford General Hospital between 1981 and 1983 each fracture was assigned a Frykman classification number.[11] (Table 1)

TABLE 1

Classification of Colles' Fractures

	Distal Ulnar Fracture	
Fractures	**Absent**	**Present**
Extraarticular	**I**	**II**
Intraarticular (radiocarpal joint)	**III**	**IV**
Intraarticular (radio-ulnar joint)	**V**	**VI**
Intraarticular (radiocarpal & radio-ulnar joint)	**VII**	**VIII**

Traditional methods of closed reduction and casting were preferred for the stable fractures. Those fractures that were comminuted, unstable, and had a classification number of VI or greater were treated with the Ace Colles fixator.

Treatment by the external fixator was limited to patients with: 1) comminuted, displaced fractures that were inherently unstable. This was determined at the time of initial reduction. If more than 20 degrees of dorsal angulation or 10mm of radial shortening were seen with multiple comminuted fragments, these fractures were then considered unstable. 2) Loss of reduction following closed treatment with a cast. If more than 5 degrees of dorsal angulation or 5mm of radial shortening was noted following closed treatment, the patient was converted to the external fixator.[10] The average period for retention of external pins was 6.5 weeks, with a range of 6 to 8 weeks.

TECHNIQUE

Following the administration of anesthesia the patients were placed in finger traps with counter traction supplied by 4 to 6 kilograms of weight suspended from a swathe applied above the elbow. The elbow was flexed to 90 degrees and image intensification was utilized to check the initial alignment. (fig. 1)

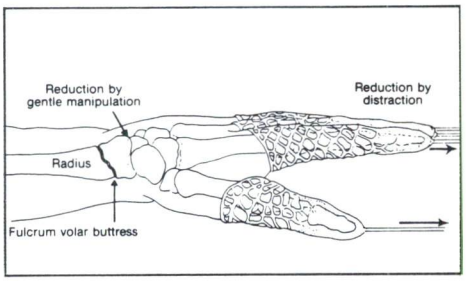

FIGURE 1

Reduction of the unstable fracture is achieved by distraction with 4-6 kilograms of weight and gentle manipulation. Stability can be achieved by molding the volar buttress. Supination must be maintained during application of the frame to stabilize the radio-ulnar joint.

Exact alignment of the volar cortices with full restoration of length was expected. Attention to the radioulnar joint was made to allow for anatomic restoration of that joint as well. The forearm was maintained in slight to full supination during application of the fixator. This was necessary to achieve and maintain reduction in a stable position. Without supination the radioulnar joints were prone to subluxation and loss of radial length would be more likely to occur.[10] The original Ace Colles fixator required drilling a 2.7mm hole at the base of the second and third metacarpals at an angle of 60 degrees to each other. The self tapping 3mm pins could then be inserted through the couplers at the distal end of the frame. Proximally two drill holes are made in the radial shaft paralelling the original 2 holes.

Traction is maintained during insertion of the pins. A final correction of fracture alignment was made following application of the frame. The frame was then tightened with some distraction introduced to the fracture initially. At two weeks the distraction was decreased by tightening the bolts at the proximal end of the frame. Early motion of the digits and elbow was encouraged. With the wrist immobilized in neutral position and ulnar deviation, flexion and extension of the digits could be accomplished. The pins were removed in the office without an anaesthetic. Either a cast or plaster splint was then applied.

RESULTS

The subjective response of these ten patients indicated that seven were fully satisfied, two were partially satisfied, and one was unsatisfied. The one unsatisfactory result involved late displacement of the fracture which required a darrach procedure with a sylastic cap of the ulna.

Measurements of the wrist motion revealed a mean dorsiflexion of 51 degrees compared to 56 degrees on the uninjured side. Palmar flexion averaged 48 degrees on the fractured side and 64 degrees on the uninjured side. Radial deviation of 23 degrees was

noted compared to 25 degrees on the uninjured side. Ulnar deviation demonstrated 22 degrees on the injured and 31 degrees on the uninjured side. The average grip strength was 38 kilograms on the injured side compared to 58 kilograms on the uninjured side. Eight out of the 10 patients had grip strengths greater than 50% of the uninjured side.

CASE REPORT

R.M., a 32 year old male, was seen in the emergency room at Botsford General Hospital following a motor vehicle accident. The initial assessment demonstrated multiple abrasions and contusions with a closed Frykman VIII fracture of the distal left radius and ulna (fig. 2).

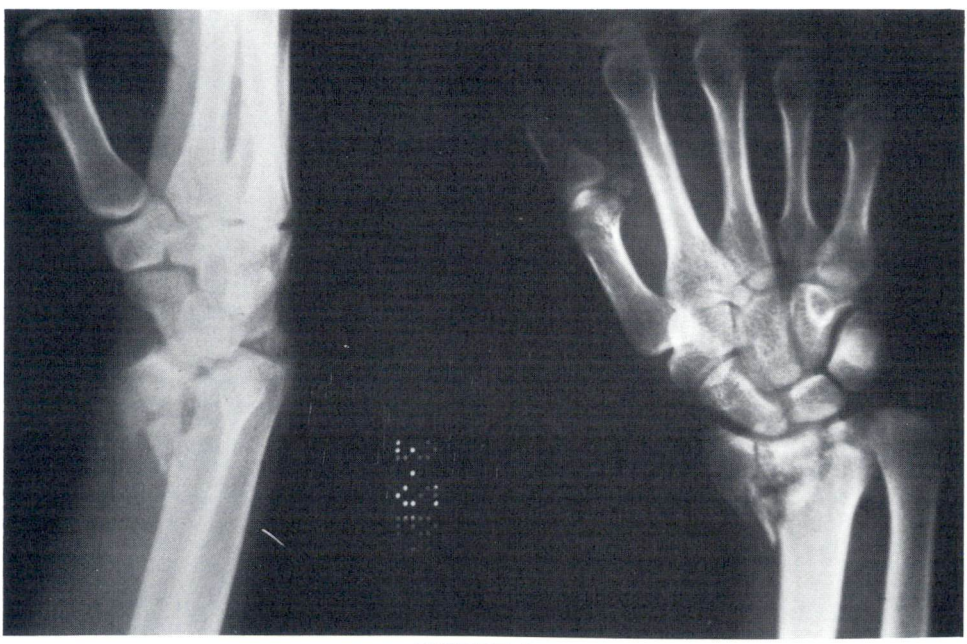

FIGURE 2

The original x-rays demonstrate a Frykman VIII fracture of the distal radius and ulna. Note the volar/dorsal fragmentation of the distal fracture fragment.

As part of the initial treatment rendered in the emergency room, a closed reduction and application of a long arm cast was performed. Post reduction films were noted to be satisfactory (fig. 3). On the fourth day of hospitalization the fracture was noted to be displaced within the cast. At this point application of the new Ace Colles fixator was elected.

Under a general anaesthetic the left hand was placed in finger traps with 6 kilograms hung from a swathe about the arm to act as counter traction. Utilizing image intensification closed reduction was achieved although the distal volar fragment remained displaced. Following application of the fixator two 0.045 Kirschner wires were placed through the distal fragments to maintain congruity of the volar cortex (fig. 4).

The patient remained in the external fixator for six weeks and to date has maintained full length of the radius without angular deformity.

COMPLICATIONS

One patient had clinical and roentgenographic evidence of post traumatic arthritis involving the distal radio-ulnar joint. Two patients had dorsal angulation of greater than 10 degrees. Two pin sites developed in-

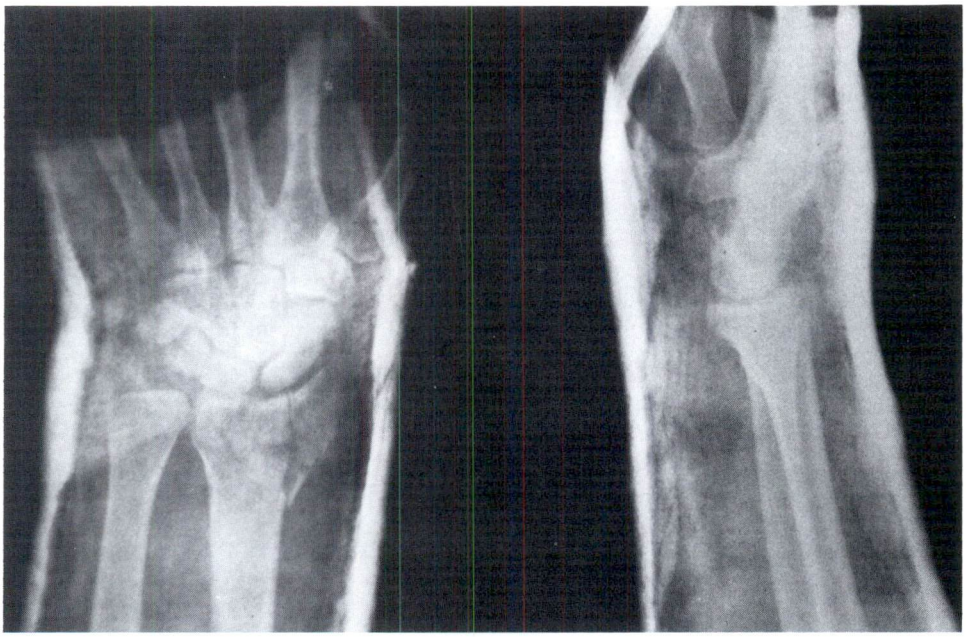

FIGURE 3

Following closed reduction and casting these x-rays were taken which demonstrate good alignment of the fracture with some radial shortening. After three days the reduction was lost and a second reduction became necessary.

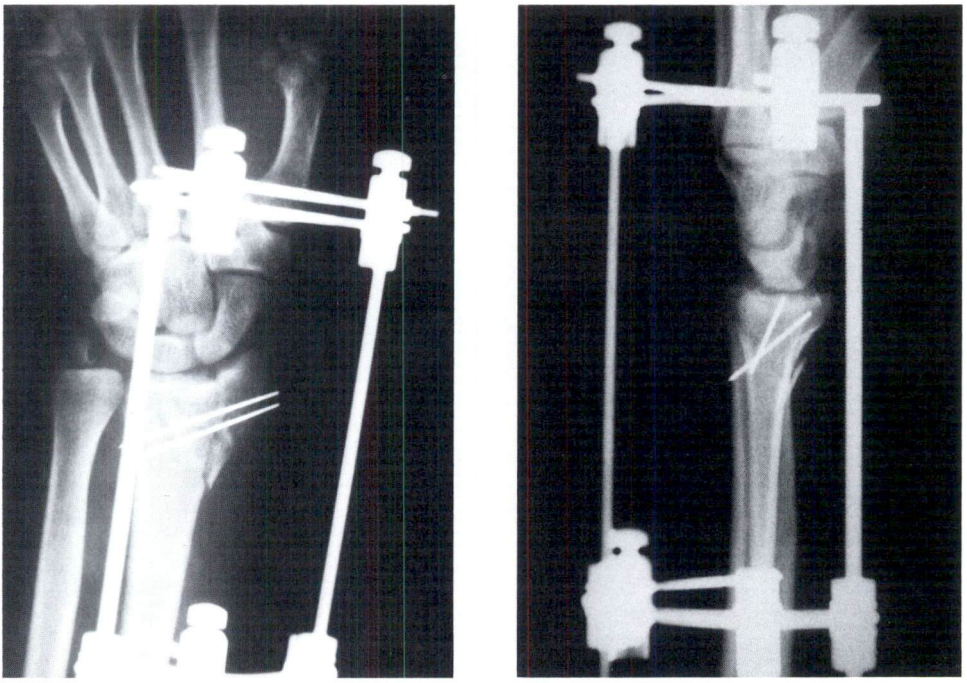

FIGURE 4

When a second reduction became necessary, the arm was suspended using finger traps and the New Ace Colles' Fixator was applied. Two 0.045 Kirschner wires were also inserted to enhance stability.

flammation and drainage, however these cleared with pin removal. Despite the serious nature of these injuries no one experienced acute carpal tunnel syndrome, rupture of flexor tendons or the extensor pollicus longus tendon.[12,13,14] Fractures through the pin holes have been reported, however this was not seen in our study.

DISCUSSION

The Ace Colles fixator has demonstrated rigid fixation capabilities in the treatment of the comminuted intra-articular Colles fracture.[10,15] This devise can be utilized (like the Hoffman frame) in a distraction, compression, or neutralization mode. The mechanical stability is firmer than can be achieved by other methods of pin fixation.[10,17]

The technique that was used allows for volar stabilization of the comminuted fragments. When alignment is maintained, union of the volar elements would occur thus preventing angular deformities. It is important to examine the radio-ulnar joint following assembly of the frame in order to maintain a stable reduction.[1,10,15]

With the introduction of the new Ace Colles fixator at the convention in Anaheim this year, two distinct improvements were noted to have been added to the original design. The arc of radius in each ring segment has been increased from 40 degrees to 84 degrees. This gives the surgeon greater versatility in using the frame. Also new with this device is the introduction of predrilled pins and drill sleeves to minimize soft tissue damage. It was our constant concern with the original fixator that the extensor tendons of the radial nerve would be irritated at the time of pin placement. The serrated end of the drill sleeve will also minimize the risk of sliding off of the bone during drilling.

The new Ace Colles fixator offers more versatility in pin placement. With the pre-drilled pins and drill sleeves we have minimized soft tissue damage during application of the frame which decreases the risk of extensor tendon or radial nerve irritation.

***Acknowledgement must be made to Dr. Robert Mandell, Dr. Edward Loniewski, Dr. B.J. Page II, and Dr. John Swienckowski for their participation in the production of this paper.**

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Pigmented Villonodular Synovitis: A Case Report

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ABSTRACT: Pigmented villonodular synovitis is a rare disease that usually affects young adults. The disease is monoarticular. Almost any joint can be involved, although the knee is most commonly affected. The etiology as well as the optimal treatment is unknown. **KEY WORDS:** Hemarthrosis, synovitis, juxtrarticular bone erosion.

INTRODUCTION

Pigmented villonodular synovitis was first named by Jaffe in 1941. Prior to the studies done by Jaffe this disease was felt to be neoplastic in nature. It is now felt to be an inflammatory reaction.

Many theories have been put forth regarding the etiology of the disease. These include: neoplasm, inflammation, disturbance of lipid metabolism, trauma, hemorrhage, immune aberation and autoimmune reaction.

Prior to Jaffe's work, the disease was felt to be due to tumorous proliferation of osteoclasts in or around sesamoid bones. It was also felt to be a benign lesion that could undergo malignant degeneration.

The theory of inflammation was based on the cytology of the lesion but the precise etiologic agent was unknown. Wyllie[11] in 1969 added support for Jaffe's theory of inflammation with electron microscopic studies. Two basic cell types were demonstrated. Fibroblasts predominated and showed an ultrastructure suggesting that the cells were synthesizing collagen and proteoglycan. Macrophages were present and contained hemosiderin.[5] From this data Wyllie implied that the process was inflammatory.

Hirohata in 1968 proposed the theory of abnormal lipid metabolism as the etiology of pigmented villonodular synovitis. Biochemical and ultrastructural studies of affected tissues showed a marked increase in cholesterol concentration and intra-cellular lipids in histeocytes. The foam cell, a characteristic part of the histology, was felt to be the primary pathologic cell. It exerted it's influence by abnormal biosynthesis of cholesterol and phospholipids. The inflammatory response was secondary to the disturbance of lipid metabolism.

Singh,[10] et.al. published a paper in 1969 which proposed tr;auma and hemorrhage as the etiology of pigmented villonodular synovitis. Trauma with hemorrhage was felt to be a possible etiology because of the heavy accumulation of hemosiderin. They injected the knees and ankles of rhesus monkeys with blood to experimentally produce the lesion. The lesion produced was simular but not identical to pigmented villonodular synovitis. Some authors have named trauma as the inciting factor with hemarthrosis, in some susceptible patients, causing an autoimmune response with a sustained inflammatory reaction. Roy and Ghadially in 1969 produced chronic hemarthrosis in rabbits and found the lesion produced to be simular to the chronic hemarthrosis of hemophilic arthropathy. Giant cells and foam cells were not found. Although trauma has been implicated as the cause, a history of trauma is present in only 22 to 50% of the cases.[9] Trauma is considered an incidental finding.

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Kinsella, in 1975, studied immune responsiveness in a patient with pigmented villonodular synovitis and described an immune aberation.[8] The synovial fluid and synovium contained large numbers of immunoglobulin containing cells. These cells were identified by immunofluorescent studies as immunoglobulin-synthesizing plasma cells. The in vitro blastogenic response of the patient's blood lymphocytes to phytohemagglutinin was abnormal. Loss of in vitro blastogenic response to PHA with maintenance of cell-mediated immunity in vivo has been observed with acute surgical trauma, viral infections and neoplasm. Both neoplasia and viral infection have been considered as possible etiologic factors in pigmented villonodular synovitis.

The synovial fluid in pigmented villonodular synovitis was found to be rich in plasma cells, indicating that antibody formation was possibly part of the local reaction. Bhawan in 1980 suggested that pigmented villonodular synovitis was an autoimmune reaction. He proposed that hemarthrosis in susceptable individuals may lead to a sustained inflammatory response based on an autoimmune reaction.[1]

CASE REPORT

W.C. is a fifteen year old black male seen in March of 1983 with a complaint of pain, stiffness, and swelling of the right knee. He also complained of weakness of the knee on prolonged standing. The history was positive for right knee trauma two years prior when he ran into a wall while playing football, striking his right knee. No workup was done at that time. For approximately one year prior to seeing us the patient noticed increasing right knee pain. Marked swelling and stiffness had occured over the past three months. The hemarthrosis was aspirated twice in the two weeks prior to admission. Each time the fluid quickly accumulated. An outpatient arthrogram was performed as well as a plain film of the right knee. The arthrogram was positive for a large joint effusion and questionable tear of the posterior horn of the lateral meniscus. Plain films revealed no bony pathology. The articular cartilages were smooth and regular.

At the time of admission, all presurgical lab work including CBC, PT, PTT, UA, SMA12, SMA6 was within normal limits. The RPR was negative. Aerobic and anaerobic cultures of the synovial fluid were negative for growth. Diagnostic arthroscopy with arthrotomy and open formal biopsy was carried out on March 16, 1983. At the time of surgery an additional one hundred cc of bloody fluid was withdrawn. Both menisci, cruciates, and ligaments were found to be intact. The synovium was heavily pigmented with hemosiderin deposits and was made up of villous fronds.

Pathology of the synovium microscopically revealed a markedly edematous and hemorrhagic synovia. Proliferation of synovial tissue formed numerous well vascularized villous structures. The formation of cleft-like spaces lined by synovial cells was seen within the proliferating zones. Occasional multinucleated giant cells were seen within the cleft-like spaces. The villi were lined with cuboidal cells with enlarged centrally placed nonpleomorphic nuclei. Deposits of hemosiderin were scattered throughout. There was a proliferation of muscular as well as thin walled arterioles. There were numerous foamy histeocytes scattered throughout. There was also a heavy infiltrate of round cells. Based on this histology, the diagnosis of pigmented villonodular synovitis was made.

After having received this pathology report, the patient was taken back to the operating room for a synovectomy. Post-operatively the patient's recovery was uncomplicated and he was sent home.

The clinical presentation of pigmented villonodular synovitis is fairly characteristic. The patients are usually in their thirties and have involvement of a joint on the lower extremity. Articular swelling is present and associated with warmth and loss of motion with atrophy of adjacent muscles. The joint swells intermittently or may be chronically swollen. Few

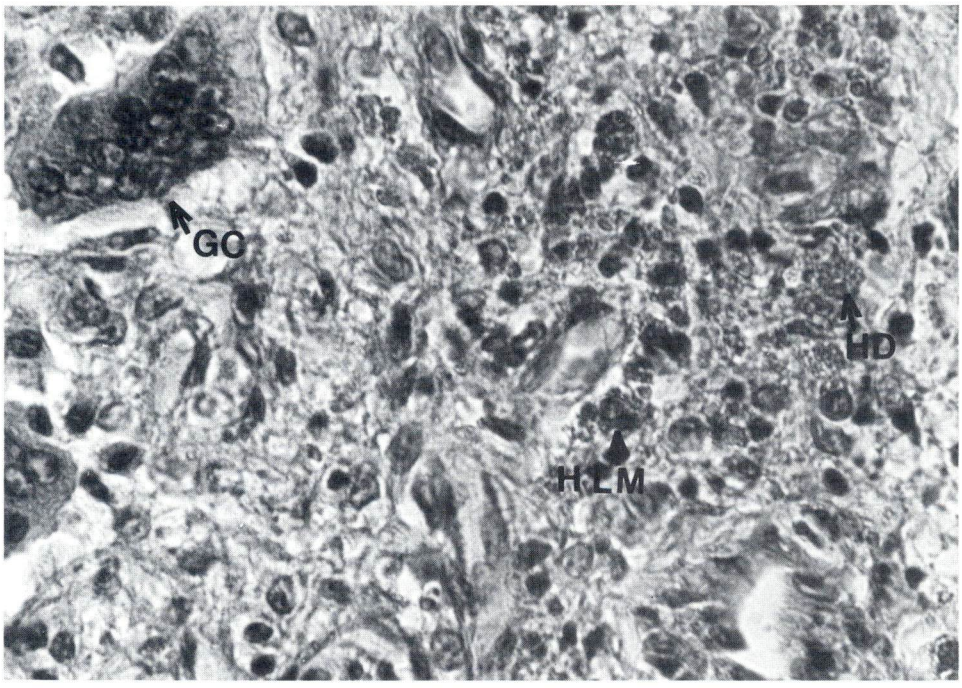

FIGURE 1

GC—Giant Cell. HLM—Hemosiderin-laden macrophage. HD—Hemosiderin deposits.

complain of severe pain, but, rather, complain of stiffness and a dull ache that worsens as the joint becomes more swollen. All laboratory values are found to be within normal limits, including the ESR, CBC, and serologic assays for RF and ANA. Synovial fluid aspirate will appear serosanguinous in the diffuse form and straw colored in the circumscribed form. The fluid will accumulate quickly following aspiration.

Pigmented villonodular synovitis occurs in three forms. Pigmented villonodular tenosynovitis is an isolated discrete lesion which occurs within a tendon sheath. This extra-articular lesion is most often found in the hand but is sometimes found in the foot and ankle. The intra-articular lesion presents as either the localized or diffuse form. The localized, circumscribed form macroscopically shows a synovial membrane with single or multiple nodular growths. The nodules are yellow-brown in color and have stalks. Gross villi are not seen. It may present as internal derangement of the knee. A palpable mass may be found in either the medial or lateral compartment. Prognosis is excellent following local excision. The third presentation is diffuse pigmented villonodular synovitis. The entire synovium appears brownish in color and is covered with multiple nodules and villous growths. The colors result from accumulation of hemosiderin, lipid, and blood. Prognosis is poor following synovectomy. A 46% recurrence rate has been reported.

The histologic appearance of pigmented villonodular synovitis is variable, reflecting maturation of the inflammatory process. Early on, the lesion is cellular and hypervascular. This phase gives way to hyalinization and fibrosis. Early fibrosis is either scattered within nodules or perivascular in distribution. These lesions with little fibrosis have abundant vascularity and cellularity. Many hemosiderin-laden macrophages are present. The synovium with marked papillarity but little nodule formation. With increasing

fibrosis, nodule formation becomes more prominent with less prominent vascularity. Extreme fibrosis is present when only islands of cells remain. Few vessels remain. Broad bands of collagen are present with little or no iron deposition.

Histopathology of pigmented villonodular synovitis reveals a synovial lining which is two or three layers thick, not hyperplastic, and often thrown into numerous microscopic villi.[2] Hemosiderin is found in both intracellular and extracellular locations. Collagen may appear as an open reticular network or condensed into broader bands; occasionally totally acellular, collagenized areas are seen.[2] Giant cells are common and may possess up to fifty or sixty nuclei. On electron microscopy, the giant cells have numerous mitochondria but few lysosomes or inclusions. Highly vascular areas occur and are unremarkable in appearance. The most prevelant cell is the histeocyte. Lymphocytes and plasma cells are also present. Mitosis are infrequently seen and cellular atypia does not occur. Foam cells are present and occur in clusters and sheets. Both diffuse and localized pigmented villonodular synovitis may show extension into surrounding tissues. Many of the histopathologis features are nonspecific, but the histeocytes and foam cells of the cellular infiltrate is distinctive.[2] The prominant feature in pigmented villonodular synovitis in the subsynovial tissue is the occurrence of many closely packed subsynovial macrophages containing: erythrophagosomes and erythrophagolysosomes, solitary and compound siderosomes, erythrocyte ghosts and lipid droplits.[3]

The cytochemistry of pigmented villonodular synovitis reveals changes in the metabolism of synoviocytes. These include a massive rise in the activity of glucose-6-phosphate dehydrogenase to levels greater than those found in rheumatoid arthritis.[7] There is also a significant rise in the activity of two glycolytic enzymes; glyceraldehyde 3-phosphate and lactate dehydrogenase.[7] Lysosomal napthylamidase showed increased activity.[7] The sulphydryl content in the synoviocytes was raised, as was the amount of available phospholipid.[7] This pattern is simular to the metabolic alteration in human rheumatoid synoviocytes.

Bone lesions associated with intraarticular growths are almost always from the diffuse form. X-ray changes in pigmented villonodular synovitis include lobular soft tissue swelling in a monoarticular distribution, with preservation of cartilage space and no regional osteoporosis.[2] In 15% of cases there will be cystic changes in the subchondral bone. The intraosseous cysts appear in nonmarginal locations, occasionally at some distance from the joint line.[3] The rim of the cysts is thin, smooth, an sclerotic.[3]

The exact manner in which pigmented villonodular synovitis leads to bony destruction is unknown. Some researchers have suggested that osseus invasion occurs via the vascular foramina. The other theory is that the pressure on the articular cartilage by the synovial tissue causes erosion of the cartilage and subsequently, of the underlying bone into which the synovium expands. Bone invasion is more common in the hip than in other joints possible due to the small articular cavity which allows limited room for growth of the synovial tissue.

Two other modes of radiographic examination include arthrography and CAT scanning. Arthrography will demonstrate synovial proliferation. CAT scanning may demonstrate iron deposition in the soft tissues due to recurrent hemorrhage.

The differential diagnosis of pigmented villonodular synovitis includes synovial hemangioma, rheumatoid arthritis, osteoarthritis, synovioma and giant cell tumor. The differentiation of PVS from synovial hemangioma must be done at the microscopic level. PVS exhibits synovial proliferation with few thin walled sinusoids where as the synovial

hemangioma has many thin walled sinusoids with no synovial proliferation. Rheumatoid arthritis differs from PVS clinically by it's polyarticular distribution, uniform synovial swelling, and presence of osteoporosis. Osteoarthritis exhibits loss of joint space with hypertrophic spurring. Loss of joint space is a very late finding in PVS. Synovia on x-ray reveals irregular bony erosion with spotty osteoporosis. Classic finding in PVS include a monoarticular distribution with synovial swelling which is lobulated in appearance. No synovial calcifications are seen. There is normal mineralization of bone surrounding the joint with preservation of joint space. Cystic lesions with thin sclerotic margins are seen in the juxta-articular bone of the joint involved.

Treatment differs depending on the type of PVS. Total synovectomy is accepted as the best treatment for diffuse PVS. In the presence of bone lesions the synovectomy should be combined with curretage of the bone cysts which when large, must be filled with cancellous bone chips. In the knee, manipulation under general anesthesia two weeks after synovectomy is recommended to decrease the amount of stiffness that will result. In the hip, when extensive bony and cartilaginous destruction is present synovectomy and bone grafting may not be sufficient. Total hip arthroplasty or arthrodesis may be necessary.

Significant recurrence rates as high as 46% have been reported following synovectomy alone. Radiation therapy has been used on recurrent cases and is recommended only if synovectomy is not curative.

Total surgical excision has proven to be a successful treatment for localized nodular pigmented synovitis. The recurrence rate has been low following excision.

In conclusion, PVS is a rare disease that usually affects young adults. The disease is monoarticular. The knee joint is most commonly affected but, almost any joint can be involved. The presentation is not dramatic as inferred by the finding that some patients may have the disease six years or more prior to diagnosis. The most recent theories on etiology include viral infection or an immune response. Treatment of PVS varies depending on the form of the disease present. At present, it is reasonable to treat localized PVS with local excision and diffuse PVS with synovectomy. Repeat synovectomy may be required if an unacceptable amount of pain and stiffness ensues. Radiotherapy should be reserved for older, symptomatic patients with recurrent disease despite two or more efforts at surgical excision. At the present, both the etiology and optimal therapy are unknown.

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The Treatment of Infected Nonunion with Bone Deficit

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ABSTRACT: The treatment of infected nonunion is discussed as well as three case presentations. Each case presentation demonstrates a different form of fixation to achieve fracture stability with the resultant union enclosing of the wound deficit. Four principles of treatment of the affected nonunion are discussed and include radical debridement, fracture stability, antibiotic usage, and leaving wounds open with delayed primary enclosure. Aggressive cancellous grafting is the key in bridging bone deficits and should be done at the time of definitive bone stabilization procedures. Two future surgical techniques are discussed to aid the surgeon in performing appropriate sequestrectomies and in the management of bone deficits. **KEY WORDS:** Fracture Stabilization, Corticocancellous and Cancellous Grafting, Osteomyelitis, Bone Deficit.

INTRODUCTION

The treatment of infected nonunion has always been a difficult problem. Since the AO Group has stimulated a more aggressive approach toward the open fracture, as well as expanded our attitude toward the open treatment of closed fractures, a more difficult problem has presented itself. The infected nonunion with defective bony bridge i.e. substantial bone loss, will at some time plague the orthopedist who makes full use of AO principles and techniques. Fortunately, AO has not left us without treatment alternatives to these problems. Papineau[11] discussed his treatment of chronic osteomylitis with excision and cancellous bone grafting on a bed of granulation tissue in an open technique. Gustillo[7] has reviewed Papineau's technique and expounded on some variations. However, it was the AO group that proved to the orthopedic world that stability in the face of infection, will allow a fracture to heal.[12] Three cases will be presented below that will illustrate various types of bone deficits in extremity trauma in the face of infection.

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CASE REPORT

Case 1: L. K. is a 52 year old white female who sustained a type III open fracture of her radius and ulna eleven years ago. (6/23/71). She presented herself to the traumatology service at Bremen's Zentralkrankenhaus on 1/8/81 (Fig. 1) after having 4 previous surgeries performed on her right radius resulting in a pseudo-rush rods internal fixation, b) cancellous bone grafting, c) a second cancellous bone grafting, d) and corticocancellous bone grafting. She underwent a radical debridement with excision of scars and the fibrous union, leaving her with a substantial bone loss. This was treated by using an overlapping multiple one-third tubular plating technique and the use of corticocancellous strips of bone on 2/10/81. (Fig. 2a-d) After bone union, the plates were removed on 2/4/83. (Fig. 3) Her ROM was supination 5° from neutral, pronation 35°, wrist dorsiflexion 15°, wrist palmar flexion 20°, wrist ulnar deviation 20°, radial deviation 0°, and no neurocirculatory deficit.

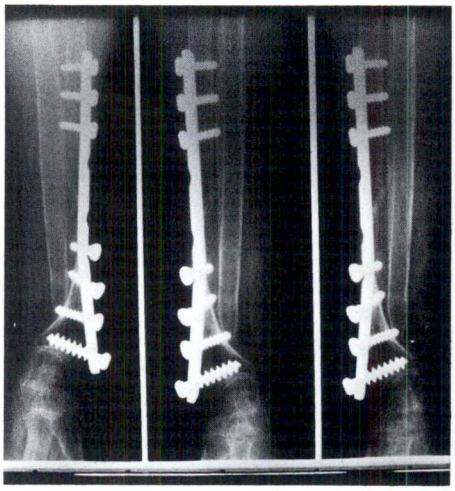


FIGURE 1

L.K. on initial presentation to Traumatology Clinic. Note the bone deficit present and internal fixation using semitubular plate and the use of 6.5 mm cancellous screws and 4.5 mm cortical screws.

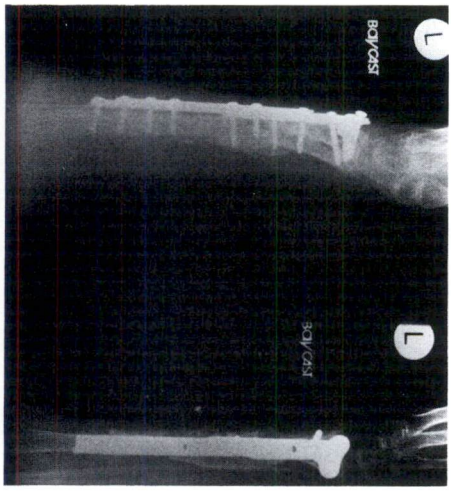
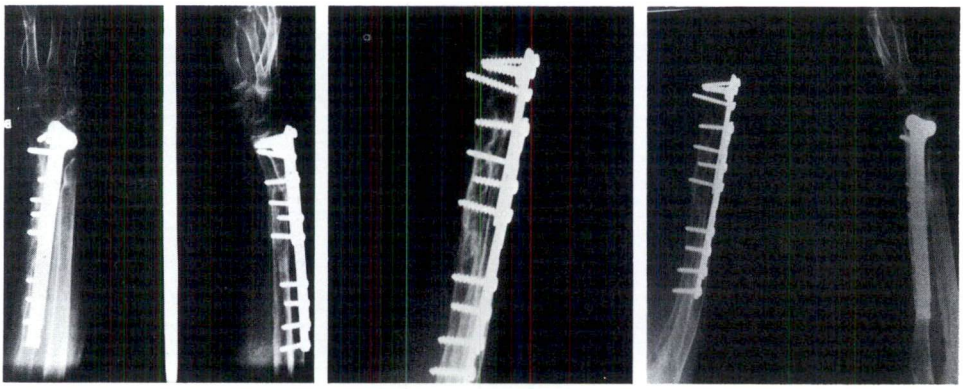


FIGURE 2

a) upper right — L.K. after removal of semitubular plateand screws, followed by corticocancellous bone graft and application of overlapping one-third tubular plates.



b, c) Sequential radiographs showing healing of fracture. b: 4/23/81 c: 2/4/83 d) Close up radiograph prior to removal of plates and screws.

Case 2: F. M. was a 68 year old white female who was struck by a car while walking along the sidewalk in December of 1981. She sustained a type III open fracture of the right tibia with comminution. (Fig. 4) She was treated initially by debridement and irrigation, plating, circlage wiring and screws on 12/4/81. (Fig. 5) She subsequently became infected and had the plate removed because of instability. (Fig. 6) She reported to the Traumatology Clinic in Bremen in March of 1982 where a diagnois of infected nonunion was made. Bone deficits were incurred after sequestrectomy and removal of screws on 3/20/82 which was simultaneously treated with a bone bridging plate and a large homologous cancellous bone graft. (Fig. 7a-d) Healing of soft tissues quickly occurred, and she was placed in a walking cast on 4/20/82. Healing was subsequently uneventful. Her current physical findings one year postoperatively were ROM right knee 0-65° flexion, 30° plantar flexion, -8° neutral dorsiflexion of right ankle, 2 cm shortening of her right tibia. (Fig. 8) She currently uses crutches and is allowed 40 Kg weight on her right leg, and has no pain with weight bearing.

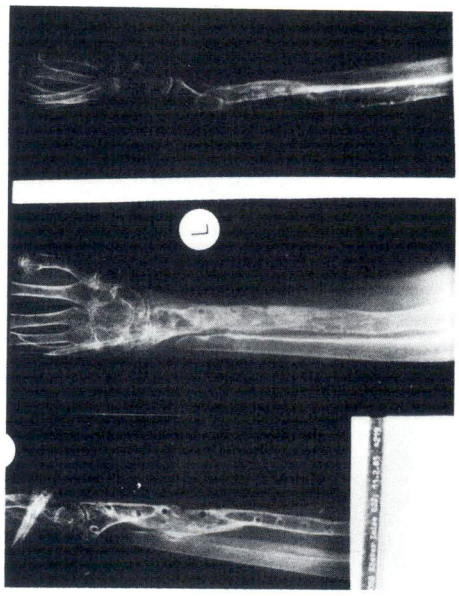


FIGURE 3

Post operative radiographs following plate removal. Note well incorporated grafting material at former bone deficit site.

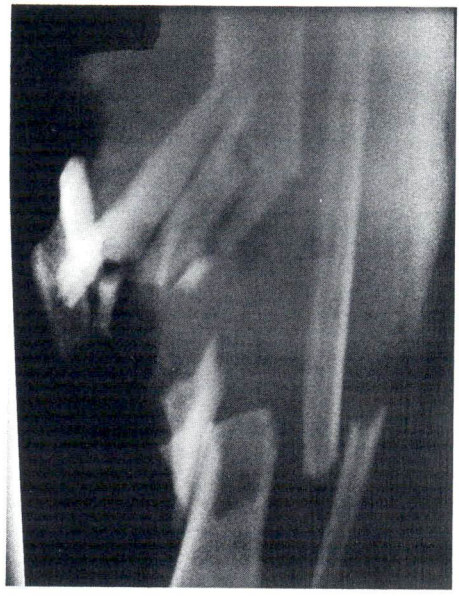


FIGURE 4

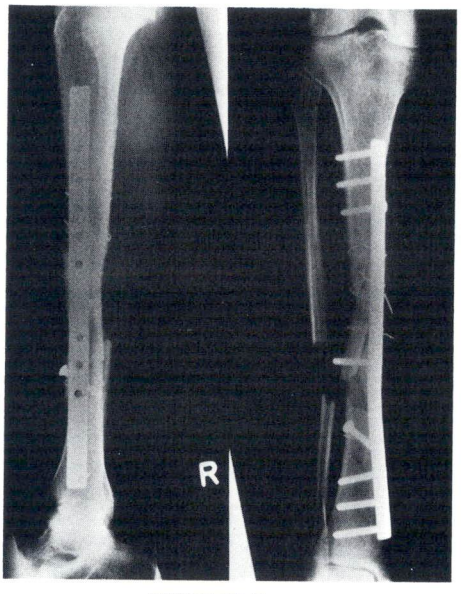


FIGURE 5

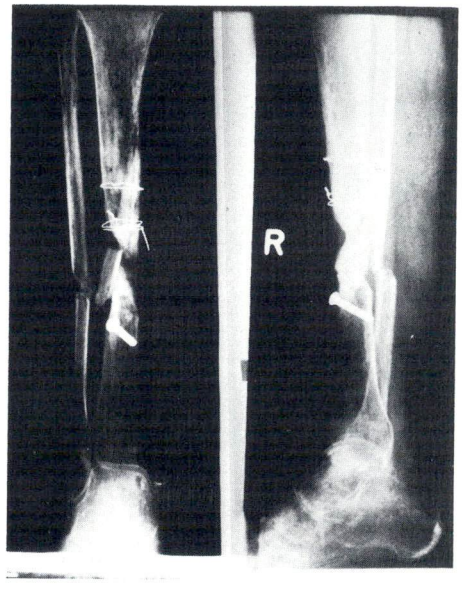


FIGURE 6

F.M. presented to emergency room after sustaining this type III open tibia and fibula fracture. Immediate open reduction and internal fixation of the fracture with plates, screws, and circlage wire. Following infection the removal of plate and most of screws.

Case 3: This is a 14 year old white male who fell riding am motorcycle in November 1982 and sustained a closed comminuted fracture of the distal one-third of his right tibia. He was treated by open reduction internal plate fixation at another institution and subsequently was referred to the Traumatology Clinic in Bremen after his right tibia became infected. In January of 1983 he underwent several debridements, including plate removal,

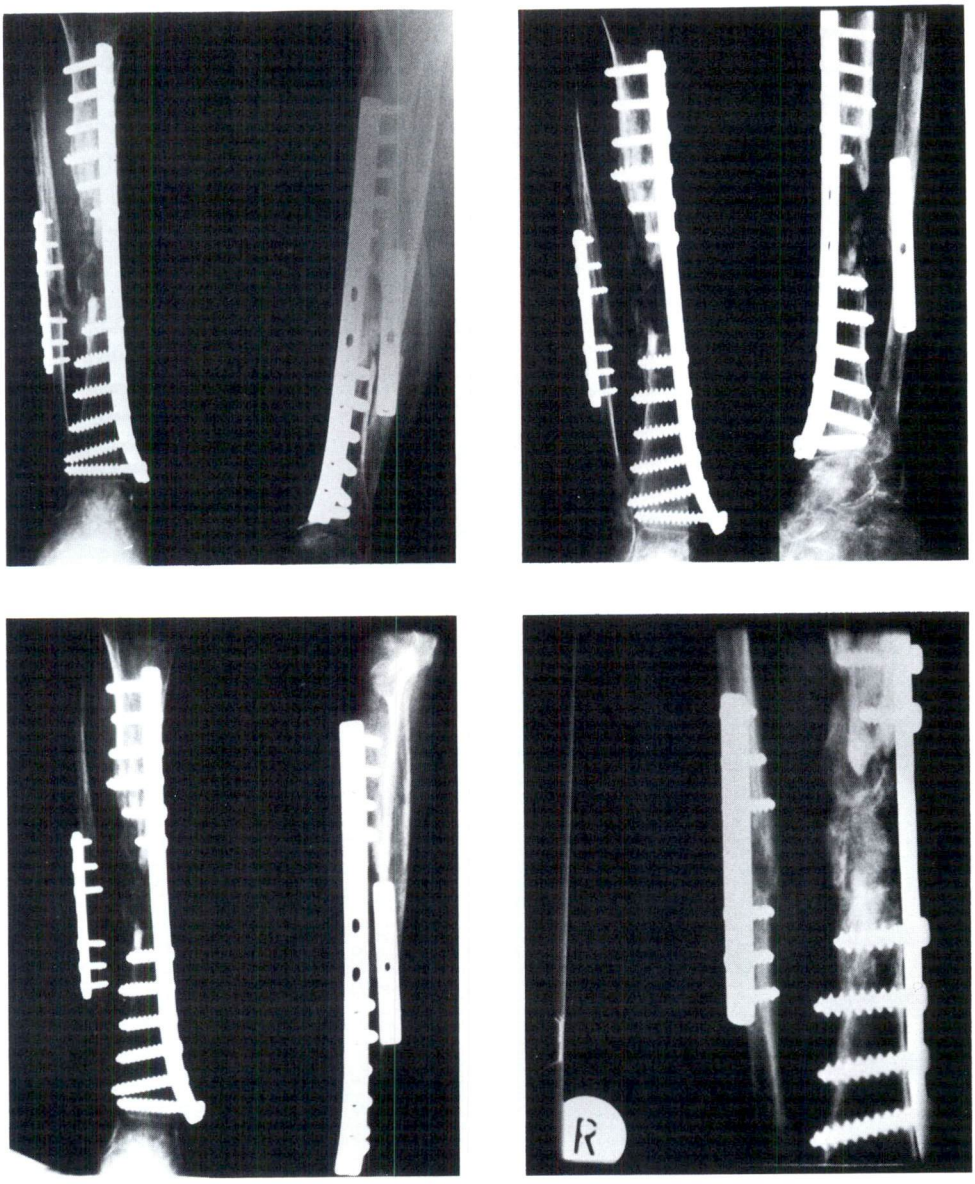


FIGURE 7

a) Debridement and irrigation procedure followed by open reduction and bridging homologous bone graft to right tibia and fibula on 3/20/82. b) Nine months postoperative radiograph showing incorporation of cancellous bone graft. c) One year postoperative radiograph. d) Close-up radiograph one year postoperatively.

leaving him with a bone bridge deficit. While being maintained in skeletal traction, he was treated with an open irrigation system until his bone deficit was covered with granulation tissue. (Fig. 9) At that time (2/18/83), he underwent further debridement, fibular osteotomy and cancellous grafting to create a tibial-fibular synostosis with application of an AO external fixator placed in a triangular half pin position. (Fig. 10)

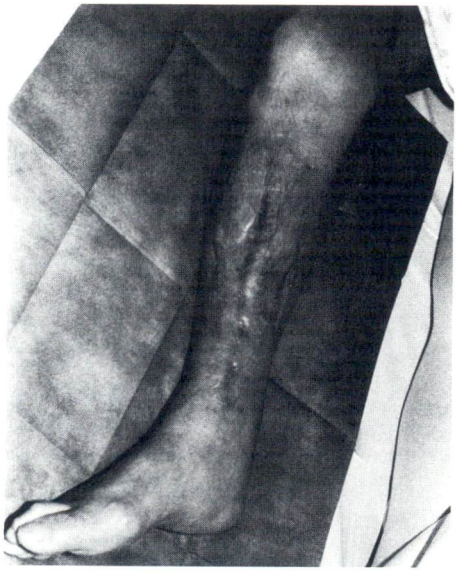


FIGURE 8

Photograph of right lower leg one year postoperatively.

PRINCIPLES OF TREATMENT

The principles of the treatment of infected nonunion or infected fractures apply to bone deficits as well, and are well outlined by Gustillo[7]. Radical debridement is the initial approach. This includes bone (sequestrectomy) and this is frequently the cause of a major bone deficit. The wound at this point should be left open. If metal (i.e. plate, rod) is in the wound and is maintaining stability, it should be left in place. If the metal is loose, then it must be removed.

The decision at this point, centers around how to maintain fracture stability. This leads to the second principle. Fracture stability can be maintained by immediate re-plating, renailing, or by temporary external fixation or the less desirable skeletal traction. The choice of implant depends on the extent of infection, amount of bone loss, previous fixation, and current conditions of soft tissue and skin. But again, the goal is to, maintain stability.

The third principle is an appropriate antibiotic regimen. This has been helped in recent years by knowledgible men in the field of infectious disease and particularly with the advent of clinical pharmicokinetics.

Fourthly, wounds should always be left open after treating infected nonunions. McNeur[9] closed many of his wounds primarily with an 85% healing rate of his open fractures (treated open) and only one infection; however, this has not borne out true in other studies.

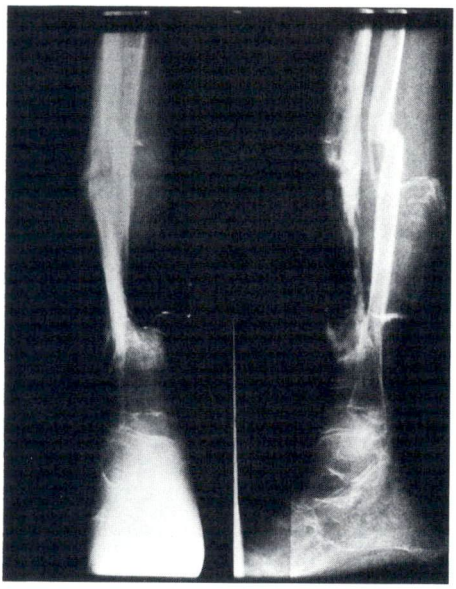


FIGURE 9

Following removal of plates and screws leaving bone deficit.

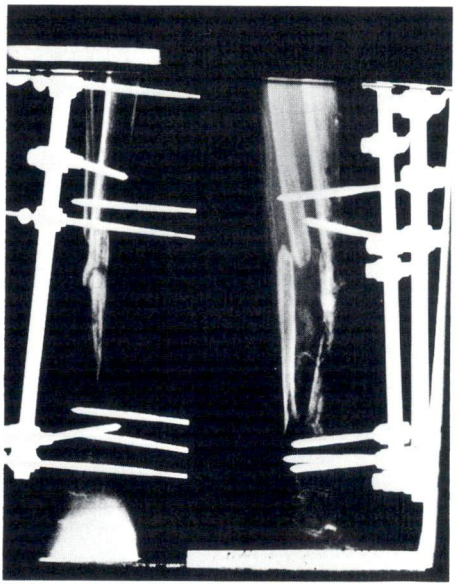


FIGURE 10

Following final definitive procedure of homologous bone grafting and tibial-fibular synostosis.

In treating non-infected pseudoarthrosis, this of course, does not apply.

Delayed primary closure frequently cannot be accomplished in treating infected nonunions with or without bone defects. Not infrequently these require plastic procedures including skin grafts and muscle flaps.

Open and closed suction irrigation systems do have their place. Many studies advocating their use[5,8] have been refuted by other[3]. We used open irrigation in one of our cases because of its medial location on the tibia. Continuous irrigation stimulates granulation tissue and acts in debriding at the same time. The surrounding skin is protected from maceration by the use of zinc oxide. Closed irrigation systems can be used in areas where the bone itself or a wound may lead to a large dead space. It should only be used for a short period of time i.e. 3-5 days.

Cancellous grafting is the key in bridging bone deficits and should be done at the time of definitive bone stabilizing[10] in most cases. In procedures conducted on noninfected wounds, corticocancellous, or cancellous bone can be used followed by immediate closure of the skin. Infected wounds, however, should have a granulating bed on which a cancellous bone graft can be placed. These wounds can be covered secondarily or be allowed to granulate completely. The tibia lends itself well to a synostosis salvage procedure. This helps to decrease the incidence of recurrence of infection [2] by bypassing the involved site.

DISCUSSION

There truly are many alternatives within the above framework with which to treat this most difficult entity. They center around the modes of fixation, primarily, and one must use all of the AO techniques at their disposal, and not limit themselves. An example may be combining internal and external fixation for a short period of time, or initially stabilizing the fracture site with an external fixator prior to later plating and grafting. The tibia is unique in that union can be obtained directly, or occasionally indirectly via a synostotic bridge with the fibula as previously mentioned. The femur lends itself to a potential diaphyseal osteomylitis with significant bone loss in the femur, it may well be limb saving since it can provide excellent stability. Kostinuk[4] achieved an 80% union rate with nailing of infected nonunion of the femur combined with bone grafting (corticocancellous chips) 2-3 weeks after nailing.

The forearm can be plated with overlapping plates, lending increasing stability to the fracture. This was done in our Case I, but it can also be done in the femur, and less frequently, the tibia. The fibula can be plated in instances where maintaining length of the lower extremity is important.

Aggressive cancellous and corticocancallous grafting combined with AO methods to achieve stability are the keys to reaching union. Just as early bone grafting is being advocated in the treatment of open fractures[1] (as early as months if no signs of healing), the result of healing in these fractures with bone deficits could only be accomplished with this aggressive approach.

Some new techniques and chemical products may soon be at our disposal to aid in the approach to the infected nonunion as it may pertain to resulting bone deficits. The first is a product called Disulphine Blue, which when taken intravenously, turns the skin green. More importantly, it does not impregnate nonviable tissue; it also turns all living tissue green but does not impregnate nonviable tissue. It then becomes easier to perform sequestrectomies because the nonviable bone (and soft tissue) is not stained. Bone, in particular, remains remarkably white. A second material is currently being used experimentally in dentistry. It is mixed like a cement and poured into bone cavities to take up dead space. It then hardens, but because of its chemical makeup, it is absorbed by the body like absorbable suture material and is replaced by fibrous tissue. Secondary procedures may then be performed at a later date.

COMMENTS

An easy alternative to avoid having this dreaded complication is to either avoid caring for the traumatized victim or to treat all fractures closed. Unfortunately, the medical legal aspects of medicine teach us to avoid the possibility of a complication sometimes at all costs and not infrequently with a resultant lesser functional result. Open treatment of closed fractures give better anatomical results, and frequently a shorter healing time[10] with less morbidity (plaster disease). Though infection rates will always temper ones surgical inclination, the answer does not lie in complacency and in finding the best medical-legal course of treatment. The topic of this paper lends one to consider the initial causes of this complication. In that search, one finds the not infrequent gray zone of open versus closed treatment of a fracture. Even Gustillo and Anderson progressed from a position of no metal[6] to its judicious uses in open fractures.[1,7] As Aristotle said, "Virtue is situated at an equal distance between two opposite views."[13]

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Instructional Course
Dupuytren's Contracture: Concept of and Approach To Treatment with Series Review

Terry L. Weingarden, D.O., Garden City, Michigan

ABSTRACT: Dupuytren's contracture is best treated surgically when the fibrotic band produces a flexion contracture of a digit. There are many types of surgical treatments as well as incisions that can be used. In choosing the best surgical approach the surgeon must keep in mind what procedure will produce the least complications. I have found that performing a partial palmar fasciectomy using a zig-zag incision has given me excellent results with minimal complications. **KEY WORDS:** Contracture; Fibrotic Palmar Fascia; Fasciotomy; Fasciectomy; Fibratic Pathologic Band; Zig-Zag Incision.

INTRODUCTION

Dupuytren's contracture is a disease affecting the palmar fascia of the hand. In 1823, Sir Astleyn Cooper, an English surgeon, noted the contracture in the palm. It wasn't until 1832 that Baron Guillaume Dupuytren, a French surgeon, reviewed the pathology and noted that the pathological changes occurred on the palmar fascia as he described the palmar fascia contracture that now bears his name. Dupuytren's disease is characterized by progressive contractures of the palmar aponeurosis. The contracture is due to fibrotic changes that occur in the palmar fascia beneath the skin. It is first noted by nodular formation in the palm which may stay dormant for years. This nodular formation is usually non tender; however, the disease may progress to form proliferation and fibrosis of the fascia in the palm. This thickened strand of fibrotic fascia begins at the base of the palm and can extend distally to the volar aspect of the proximal interphalangeal joint of the digit involved. The disease progresses from a cellular nodule to a dense fibrotic cord, thus producing a contracture of the involved digit.

Dupuytren's contracture appears usually in the fifth and sixth decades of life and is commonly seen in Caucasian males. The cause of this disease is unknown. According to Ling[9], Dupuytren's contracture is a genetic disease due to a single dominant gene. There are many predisposing factors. It does have a male sex link, although it can rarely be found in females. It is also seen in alcoholics, epileptics and diabetics. Fibrosis of the plantar fascia of the feet, knuckle pads of the dorsal aspect of the proximal interphalangeal joints of the fingers and peyronie's disease have also been noted with Dupuytren's disease. There are some who also feel that tramua may be associated with this disorder. Dupuytren's contracture usually affects the ring finger while the little finger is the second most involved digit. The thumb, index and middle finger is rarely associated with this disease (Fig. 1).

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PATHOLOGY

Hueston[4] believes that the disease

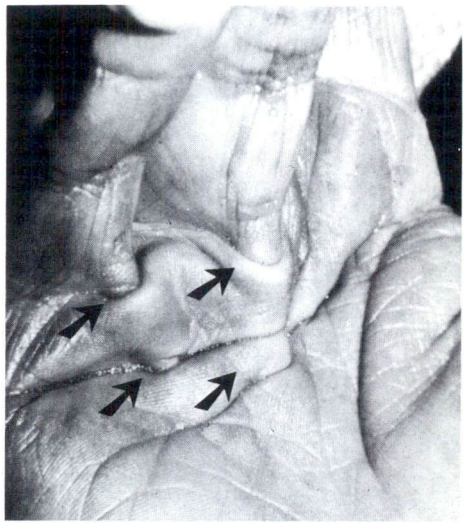

Fig. 1.

Demonstrates Dupuytren's contracture involving the fourth and fifth digits. Note the fibrotic band in the palm extending to the proximal phalanx of the ring and little fingers.

begins in the subdermal tissue as a cellular nodule. Luck[10] believes that the nodule is the contracting structure which produces joint contracture.

Gabbiani and Majno[2] believe that contractile fibroblasts called the myofibroblasts were present in the nodule. The significance of this theory is a confirming of the primary role of the hyperplastic nodule in Dupuytren's contracture and the secondary nature of the hypertrophic band. Microscopically one notes cellular proliferation with hyperchromatic nuclei. Mature fibroblasts with immature collagen is also seen. The nodules are usually highly cellular (Fig. 2).

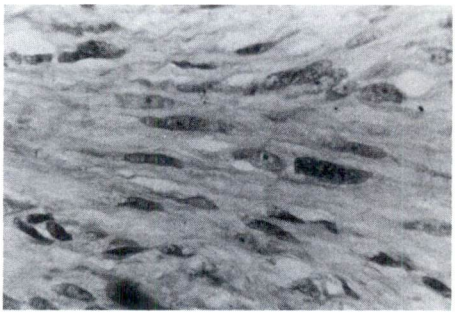

Fig. 2.

Histological changes revealing fibrotic connective tissue with fibroblastic proliferation.

A modification of staging Dupuytren's disease was set forth by Tubiana, Michon, and Thomine in 1968.[17].

Stage I: Nodule or band without contracture

Stage II: Overall contracture 1-45° (total contracture of all joints of the hand)

Stage III: Overall contracture 46-90°

Stage IV: Overall contracture 91-135°

Stage V: Overall contracture greater than 135°

CLINICAL ASPECT:

Once the digit begins to contract it is virtually impossible to extend the digit actively or passively, (Fig. 3-4). The skin is usually adherent to the nodules and in fact one may see dimpling of the skin in the palm. Patients will note increased disability from the inability to extend their digit. Activities such as washing their face, wearing gloves, even sporting activities such as bowling become difficult as the flexion contracture progresses. They also noted embarrassment when shaking hands with another person. These patients also have difficulty in taking care of the skin of the palm due to the collection of moisture that occurs from the dimpling in the skin surrounding the nodule, (Fig. 5).

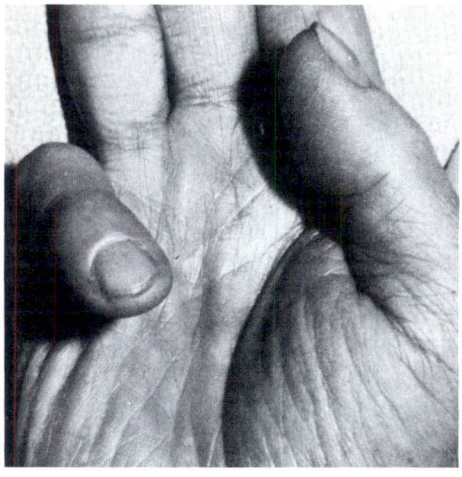

Fig. 3

Flexion contracture secondary to Dupuytren's contracture of the little finger. Note the inability to extend the digit prior to surgery. This produces increased disability by the patient when functions require the digit to extend.

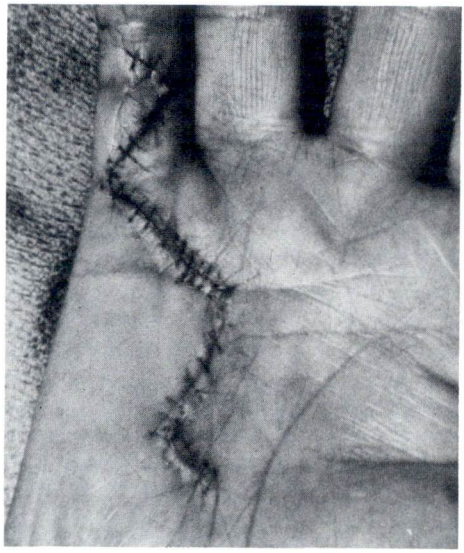

Fig. 4.

Same digit postoperatively. Note full extension of the little finger following surgical intervention, thus improving the functional use of the hand. Also note there was no difficulty with the skin closure even though there was marked flexion contracture of the digit prior to surgery.

TREATMENT OF DUPUYTREN'S DISEASE

There have been many non-surgical attempts at treating Dupuytren's from the injections of cortisone to applications of topical ointments, but they have not prevented the progression of the contracture. While the disease remains in the nodule stage, the treatment remains non-surgical. However, if the nodules become painful and disabling to the patient, then surgical intervention may be indicated to locally excise these nodules.

This is rarely performed in this stage of the disease. Surgery is indicated when the disease progresses to the contracture stage. Surgery can reverse the contracture by excision of the fibrotic diseased tissue. In treating this disease the surgeon must not allow permanent flexion contracture of the metacarpal phalangeal joint and especially the proximal interphalangeal joint. The goal of surgery is to correct the flexion contracture that has been produced by the fibrotic palmar fascia. It has been noted that the patients with the least amount of flexion contracture and the patients with the least skin changes will obtain the better surgical result. There have been many surgeries recommended to correct this condition. They are fasciotomy, partial palmar fasciectomy and radical fasciectomy.

Fasciotomy is a procedure that is performed by either the closed or open method. In the closed technique the facial cords are divided subcutaneously. The advantage of this procedure is that there is very little dissection, therefore there is little tissue disruption. Thus, complications such as hematoma are usually not a great factor with this procedure. However, if the fasciotomy is performed by the closed method then there is a good possibility of injury to the digital nerve or vessel and thus causing permanent sensory or vascular changes to the involved digit. This technique is indicated in the older patient who is unable to undergo extensive surgery. An open fasciotomy which was first recommended by Cooper and Dupuytren is another method of performing the surgery. The advantages of this procedure is that the nerve and vessels are visualized under vision prior to the dividing of the pathological fascial bands. Some believe that by performing only a fasciotomy and not resecting the diseased tissue, there is a greater risk for the contracture to recur.

A second type of surgery is the partial palmar fasciectomy. This can be performed by various types of skin incisions, including multiple transverse, zig-zag or "Z" plasty skin incisions (Fig. 6). The transverse palmar skin incision is parallel to the palmar crease but may not give good exposure into the digits unless you do extensive undermining of the skin flaps.

This may lead to postoperative hematoma or it may disrupt the blood supply to the skin flaps. The linear zig-zag incision may begin in the palm and extend distally to the involved digit. This does not require much undermining of the skin flaps and therefore there will be less soft tissue trauma. The ad-

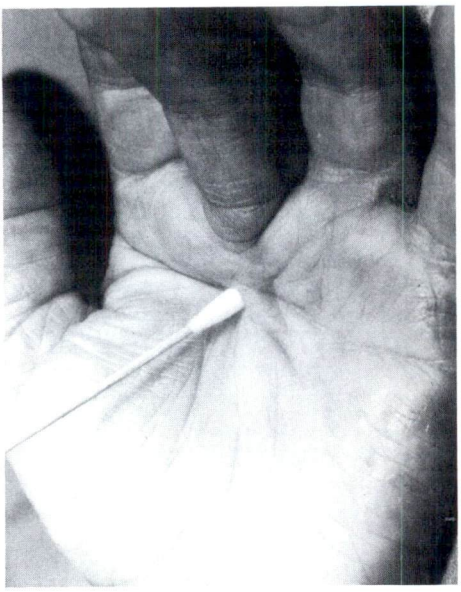

Fig. 5.

Skin dimpling produced by the fibrotic tissue contracture making it difficult to keep the skin dry.

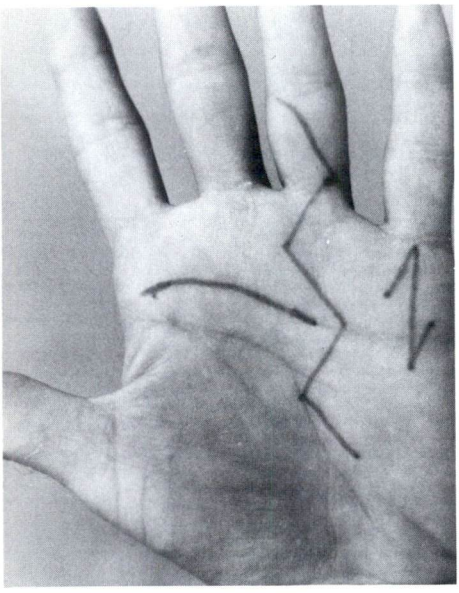

Fig. 6.

The transverse, zig-zag and "Z" plasty skin incisions are demonstrated above.

vantage of the zig-zag incision is its simplicty to closure of the wound. This skin incision allows for easy wound closure even when there is a contracture of the skin.

A third type of skin closure is the "Z" plasty which is also a linear skin incision but is then converted to a "Z" plasty to increase the amount of skin available for closure of the wound. Those who advocate the "Z" plasty do so because they feel the wound will not approximate due to the skin shortening from the contracture of the tissue.

Another method of performing this partial palmar fasciectomy has been advocated by McCash[11]. He recommends the Open Palm Technique which is resecting the pathological palmar fascia with the use of transverse skin incisions. McCash does not close the wound. He allows the wound to granulate in. He feels the wounds will heal by marginal epithelialisation in two to five weeks.

The partial palmar fasciectomy can be performed by one of the above described methods. The pathological palmar fascia is thus resected. The advantage of this surgery is that the diseased tissue is excised therefore, there is less chance of recurrence. The author prefers this surgical procedure using the zig-zag skin incision.

A third type of surgery is the radical palmar fasciectomy. In this method the pathological as well as the normal palmar fascia is resected. This requires a great amount of soft tissue dissection and thus increases the chance of complications. It also has increased morbidity with more difficulty in rehabilitation of the hand after surgery.

SURGICAL TECHNIQUE: PARTIAL PALMAR FASCIECTOMY USING THE ZIG-ZAG INCISION.

The extremity is prepped and draped in the usual sterile manner. The pneumatic tourniquet is inflated after the extremity is elevated and the Esmarsh applied. A sterile marking pen is used to diagram the zig-zag incision (Fig. 7). The skin incision begins at the base of the palm where the Dupuytren tissue begins. The incision is then continued distally to the involved digit making sure not to cross the skin creases at right angles. The skin is then incised down to the diseased tissue by sharp dissection. It is noted that in the base of the palm there

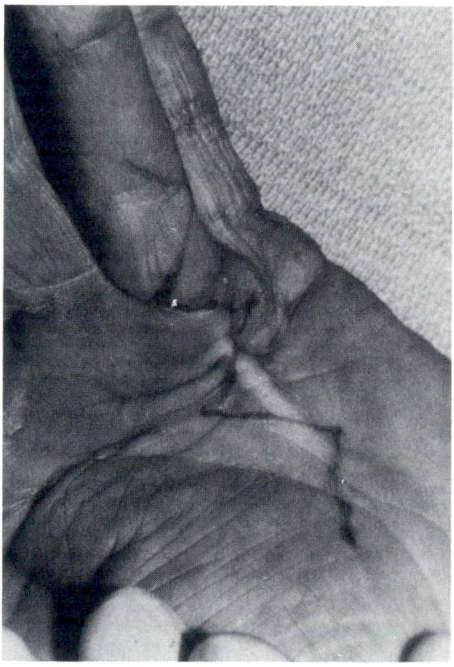

Fig. 7.

The zig-zag line over the fibrotic Dupuytren's band demonstrates the skin incision that will be used in the surgical procedure.

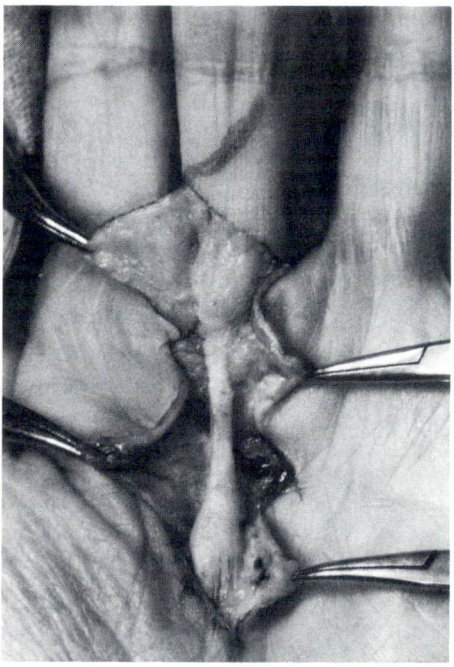

Fig. 8.

The fibrotic band is demonstrated by sharp surgical dissection.

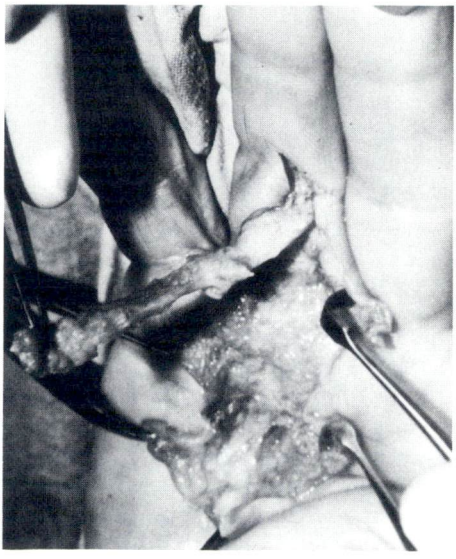

Fig. 9.

The fibrotic Dupuytren's band has been resected proximally as this is released, the surgeon will note the surgical release of the flexion contracture of the involved digit.

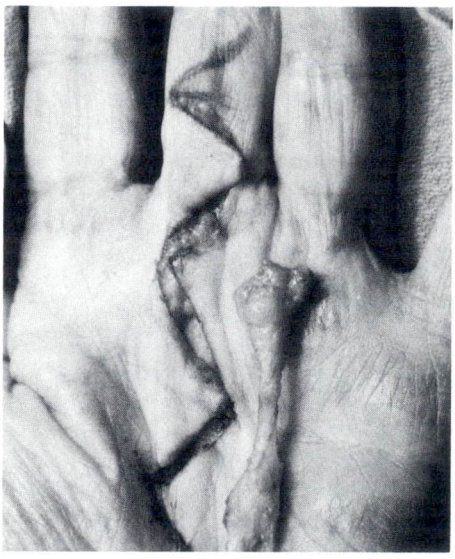

Fig. 10

The fibrotic pathologic band has been completely resected by performing a partial palmar fasciectomy via a zig-zag skin incision.

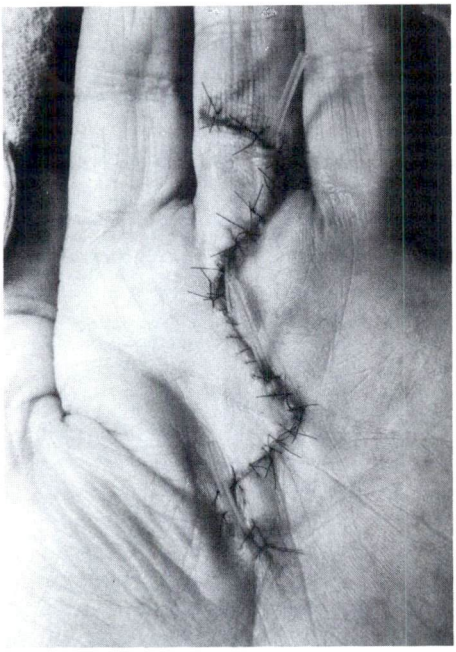

Fig. 11

The zig-zag skin incision has been approximated with 6-"0" Nylon suture with no tension on the skin closure.

may be normal subcutaneous tissue between the skin and the Dupuytren's tissue. However, as the dissection continues distally the subcutaneous tissue becomes more sparse. When a nodule is noted, there is often no subcutaneous tissue present, because the nodule is firmaly attached directly to the skin. By careful sharp dissection the fibrotic nodule can be separated from the skin at this level, as care is taken to maintain the integrity and the vasularity of the skin. Further dissection to the metacarpal phalangeal joint level, and in some cases to the proximal interphalangeal joint level is required for excision of the pathological tissue (Fig. 8). Care is taken not to injure the neurovascular structures which are often displaced at the metacarpal phalangeal joint level from their normal anatomical position by the Dupuytren tissue. Following the resection of this fibrotic tissue at the metacarpal phalangeal joint, one should note improvement and release of the contracted digit (Fig. 9-10). However, if the disease extends into the proximal interphalangeal joint and there is marked contracture of the proximal interphalangeal joint, then a volar arthrotomy of this joint may be used to release the volar plate. This will improve the motion at the proximal interphalangeal joint. After completing the resection of the Dupuytren's tissue, the tournquet is released to control any active bleeding. By doing this, the chances of developing a hematoma postoperatively is greatly diminished. After the wound has been irrigated with sterile solution, the zig-zag incision is then closed with 6-0 Nylon suture (Fig. 11). Silastic drains are left in the wounds and are removed at the first dressing change. A pressure hand dressing is applied followed by the application of a volar splint to the involved digit. This splint maintains the digit in extension for three to five days. The splint is then removed and the patient is then encouraged to begin active extension and flexion of the involved digit. The sutures are removed in two to three weeks following surgery.

RESULTS:

Thirty four surgical cases of Dupuytren's contracture performed at the Garden City Osteopathic Hospital were reviewed. The sex distribution revealed a male to female ratio of 10 to 1, (31 to 3). All patients were Caucasian. Seven patients had this disease occurring in both hands. Twenty-one patients had involvement of the little finger while the ring finger was involved in twenty-five patients. The middle finger revealed twelve cases and three patients had involvement of the thumb. Two patients had fibrotic nodules of the plantar fascia of the feet. A family history of Dupuytren's contracture was noted in five patients, while eight patients had a history of alcoholism. There was no history of seizures, however, six cases did have a history of diabetes mellitus. The average duration of the disease prior to surgical intervention was eight to ten years. Loss of extension of the digits at the metacarpal phalangeal joints prior

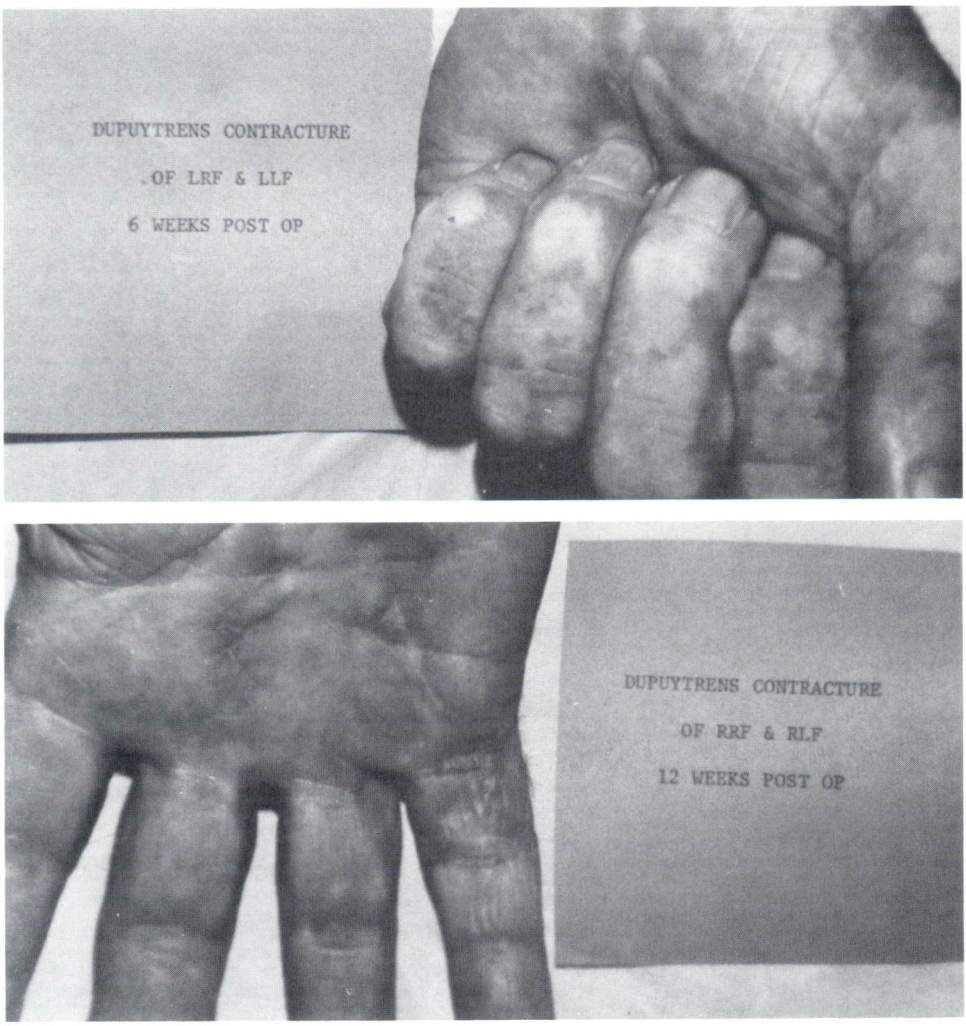

Fig. 12-13.

Demonstrates full functional return of Dupuytren's contracture of the ring and little fingers six and twelve weeks postoperatively.

to surgery ranged from -10° to -75° with a range of 35°. All cases that had involvement of the metacarpal phalangeal joint returned to normal extension following surgery. Cases that involved the proximal interphalangeal joint and did require an arthrotomy and release of the volar plate did reveal slight limitation of full extension at the PIPJ following surgery. The return to full extension of the digit depended on how severe the proximal interphalangeal joint was involved. If the disease only involved the metacarpal phalangeal joint the results were good to excellent, (Fig. 12-13). The indications for surgery in this study was progressive flexion contracture of the involved digit. Palmar nodules without flexion contracture was not considered an indication for surgery. There were no cases of nerve damage and no cases of soft tissue infection or hematoma, following surgery. Skin closure using the zig-zag incision revealed no evidence of skin loss and no evidence in difficulty in closing the wound. There were three cases that did reveal a minor skin necrosis in the corner of the skin flaps, but they healed well with no further surgical intervention required. There were no scar contractures up to two years postoperatively. Three cases did reveal minimal recur-

rences in the area operated but none were sufficient to warrant further surgical treatment.

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