Vol 2.4[Sept 2008]

Clinical Journal of Orthopaedic Surgery

Vasu Pai
Editor
Orthopaedic Surgeon

New Zealand

www.bonefix@co.nz

Contents

I Published Papers

1. Gastettner et al Acromio-clavicular joint dislocation. J Shoulder Elbow Surg 17:221

The best treatment for Rockwood type III injuries is still controversial. During a retrospective study, 24 patients who were treated surgically with a hook plate and 17 conservatively treated patients were examined with a mean follow-up of 34 months. The Oxford Shoulder Score, Simple Shoulder Test, and Constant score were assessed at the follow-up examination. Stress radiographs of both shoulders were taken, and the coracoclavicular distance, as well as the width of the acromioclavicular joint, was measured. The mean Constant score was 80.7 in the conservatively treated group and 90.4 in the group that underwent surgery. The mean coracoclavicular distance was 15.9 mm in the conservatively treated group and 12.1 mm in the surgically treated group. These differences were significant (P <.05, Mann-Whitney U test and Student t test). In this study, better results were achieved by surgical treatment with the hook plate than by conservative treatment

Operative Vs non-op :

The sound results achieved after conservative treatment that have been reported during recent years. Phillips et al12 ultimately advised against surgical treatment in their meta-analysis on this subject. A major flaw in the ongoing discussion of the treatment of AC dislocation is the lack of use of the Rockwood classification Different surgical techniques vary considerably with regard to the results reported, the concept, the advantages, and the drawbacks.

In our opinion, the advantage of the hook plate compared with other techniques, such as tension band wiring or the Bosworth screw, is the fact that, with the hook plate technique, no rigid fixation is done between the coracoid and the clavicle, as with the Bosworth screw, or between the coracoid and the acromion, as with tension-band wiring.

This fact allows the hook plate to be left in place for a longer period of time than other implants (3 months in our treatment regimen). Thus, the capsule and the coracoclavicular ligaments are given a longer time to heal sufficiently, whereas other implants have to be removed much earlier (eg, after 6 weeks).

In conclusion, a diagnosis via the Rockwood classification is indispensable for the appropriate treatment of AC dislocations. To detect a horizontal dislocation of the lateral clavicle, an axillary radiograph should always be obtained.

In our study, surgical treatment by use of a hook plate achieved better results than conservative therapy. Surgery, therefore, seems to be indicated in Rockwood type III injuries, especially when dealing with younger and physically active patients, whose demands often consist of not only the need for a free range of movement but also the capability for good power.

In comparison to other means of fixation, the advantage of the hook plate is that it can remain in the shoulder without problems for months, thus giving the coracoclavicular ligaments enough time to heal sufficiently..

2. Hanypsiak . ACL and Bone bruise. Am. J. Sports Med. 2008; 36; 671

Background: Although successful at restoring near normal laxity to the knee in the short term, anterior cruciate ligament reconstructions have not been shown to prevent the development of posttraumatic arthritis.
Hypothesis: Bone bruises and articular cartilage injuries sustained at the time of initial injury (1991) would not resolve. Our secondary hypothesis was that the presence of a bone bruise or articular cartilage injury originally identified on magnetic resonance imaging would not be associated with long-term outcomes after anterior cruciate ligament reconstruction evaluated by the International Knee Documentation Committee questionnaire.
Study Design: Cohort study (prognosis); Level of evidence, 1.
Methods: We attempted to contact all patients from an original cohort (N = 54) for follow-up evaluation, which included repeat radiographs, magnetic resonance images, physical examination, and International Knee Documentation Committee questionnaire more than a decade postoperatively.
Results: Forty-four patients (82% of the original cohort) returned for on-site follow-up. No patient with a bone bruise identified on original magnetic resonance imaging had one identified at 12-year follow-up. The mean (±SD) IKDC score at follow-up with no bone bruise originally present was 70.6 (±12.7) versus 70.0 (±8.1) when a bone bruise was observed (P > .05). No consistent association was observed between the presence of an initial articular cartilage lesion with a lesion on follow-up magnetic resonance images.
Conclusion: All bone bruises identified in our study with magnetic resonance imaging at the time of initial injury had resolved at 12-year follow-up. The presence of a bone bruise at the time of initial injury did not significantly alter the patient-oriented outcome by International Knee Documentation Committee after anterior cruciate ligament reconstruction. Additionally, articular cartilage abnormality on magnetic resonance imaging did not influence the International Knee Documentation Committee score.

Although the natural history of an ACL-deficient knee is well known, no single study has been able to link a successful ACL reconstruction with the prevention of osteoarthritis
Although the appearance and location of bone bruises on MRI in conjunction with ACL injuries has been well defined, the significance of this finding remains unclear.
The natural history of bone bruises and their ability to resolve remains controversial, in part because of the lack of prospective longitudinal cohort studies with 80% follow-up.
Bretlau et al3 found that only 12% of bone bruises identified on MRI in patients with acute knee injuries persisted at 12 months.

3.Mazzocca. Distal biceps repair. Orthop Clin N Am 39 (2008) 237–249

The treatment of distal biceps ruptures has recently received increased scrutiny
Newer treatment methods have the potential to decrease some of the complications noted with more traditional surgical techniques. Many of these newer techniques use single anterior incisions and apply hardware originally used in other areas of the body.
More recent literature noted an incidence of 1.2 per 100,000 patients.
The average age of the patient was 47 years.
A majority of male patients
Early authors suggested that a bony prominence may lead to damage at the tendon insertion
Microscopic studies of tendons that ruptured, including the biceps tendon, have shown intrinsic degenerative changes
The mechanism of injury is most often a single traumatic event characterized by an unexpected extension load applied to an elbow flexed to 90°.

Complete rupture

Most patients will experience functional improvements if a complete rupture of the distal biceps tendon is repaired back to the radial tuberosity.
Whereas those treated nonoperatively do not experience complete return of function, particularly with repetitive activities . particular supination strength is impaired.
Nonoperative management should be reserved for low-demand patients
Historically, repair techniques used an extensile anterior incision to reinsert the avulsed tendon.This method led to unacceptably high rates of radial nerve injury and prompted Boyd and Anderson to develop a two-incision technique designed to minimize risk to the neurovascular
In general,reported complications may be broadly grouped into heterotopic ossification leading to radioulnar synostosis and nerve injuries.

Suture anchor

Study reported excellent results for more than 60 patients treated with suture anchor repairs.
Benefits of suture anchor fixation include the easy availability of suture anchors in the surgical setting and surgeon familiarity with suture anchors for soft-tissue fixation.

Endobutton

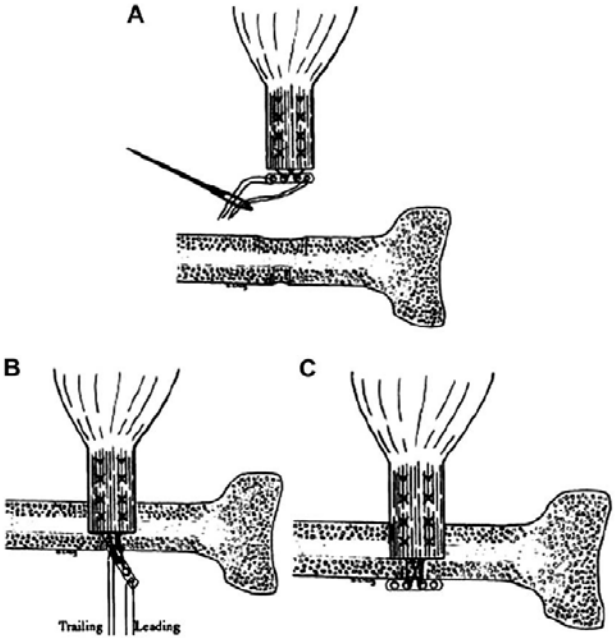
Endobutton and a bony trough to reattach the distal biceps tendon.

Interference screw

The tendon is connected to the cannulated screwdriver–screw construct by threading one suture attached to the
distal biceps tendon through the interference screw. The tendon is then guided into the trough and the interference screw is placed on the ulnar side of the tendon, pushing the repaired tendon toward the radial side. Once the screw is inserted, the suture passing through the screw is tied back to a second suture in the distal biceps tendon

Most complications reported in the largest clinical series from single anterior approach techniques involve nerve injury to either the posterior interosseous nerve (PIN) or the lateral antebrachial cutaneous nerve (LAC).
Biomechanical studies
Increasing interest in alternatives to the modified
Greenberg : compared bone tunnels, suture anchors, and Endobutton fixation in a nonanatomic model and found Endobutton to be significantly stronger.
In the most comprehensive test to date, Mazzocca and colleagues [61] compared four different methods of distal biceps repair biomechanically (bone tunnel, Endobutton, suture anchor,and interference screw), and the Endobutton had a significantly greater load to failure

than any other repair method. Failure loads ranged from 232 N (suture anchor) to 440 N (Endobutton).



4. Majid. Degenerative spine. J Am Acad Orthop Surg 2008;16:208-215

Degenerative spondylolisthesis with spinal stenosis is common in elderly patients. When symptomatic, the resultant neurogenic claudication often leads to a diminished quality of life. A nonsurgical approach is an appropriate first step. Maximizing the chance of a solid arthrodesis improves the possibility of a successful long-term outcome. Treatment of this pathology has evolved over the past twenty years with the publication of numerous prospective randomized trials assessing the influence of fusion and instrumentation following decompression. Current prospective trials have evaluated the use of bone morphogenetic proteins as a substitute for autogenous bone graft. Recently, soft tissue stabilization devices have been advocated as an alternative to fusion. Clinicians should critically evaluate these newer technologies and exercise caution regarding their use until controlled long-term trials are completed.

Treatment:

Mild to Moderate	Observation., NSAID, Epidural steroid, Physio
	Interspinous spacxer for claudication
Moderate to severe	Laminectomy +/- fusion

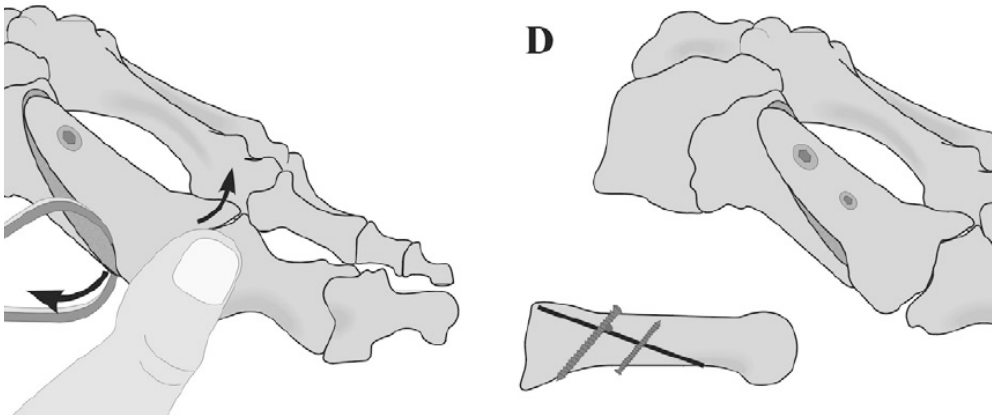
5.Ludloff. Hallux valgus. J Bone Joint Surg Am. 2008;90:531-539.

Background: The modified Ludloff proximal first metatarsal osteotomy is indicated for the surgical correction of moderate-to-severe hallux valgus deformity associated with metatarsus primus varus.
Methods: 99y-nine patients (111 feet) with a mean age of fifty-six years underwent a modified Ludloff proximal first metatarsal osteotomy and a distal soft-tissue procedure at two institutions for the treatment of a moderate-to-severe hallux valgus deformity. The American Orthopaedic Foot and Ankle Society score and weight-bearing radiographs of the foot were assessed preoperatively and after a mean duration of follow-up of thirty-four months. Clinical and radiographic outcome was also compared between younger and older patients, with the arbitrarily chosen age of sixty years dividing the two groups.
Results: The mean American Orthopaedic Foot and Ankle Society score improved significantly
The mean hallux valgus angle decreased significantly from 35_ preoperatively to 9_ at the time of the most recent follow-up ($p < 0.0001$), and the mean intermetatarsal angle decreased significantly from 17_ to 8_ ($p < 0.0001$). All osteotomy sites united without dorsiflexion malunion but with a mean first metatarsal shortening of 2.2 mm.
Conclusions: To our knowledge, the present report describes the largest cohort of patients undergoing a modified Ludloff osteotomy for the correction of hallux valgus deformity that has been reported in the literature. Our intermediate results demonstrate that the procedure achieves significant correction of moderate-to-severe hallux valgus deformity, significant reduction in forefoot pain, and significant improvement in functional outcome. Patients with an age of sixty years or less appear to have a more favourable outcome.
The Ludloff osteotomy The intermediate-term outcomes of the modified Ludloff osteotomy combined with a distal soft-tissue procedure are comparable with those reported for the proximal crescentic osteotomy
The recurrence rate in our series of Ludloff osteotomies was 4.5%
The 8% prevalence of hallux varus in the present investigation is comparable
The average first metatarsal shortening in the present series was 2.2 mm
As mentioned above, a dorsiflexed first metatarsal may lead to transfer metatarsalgia.
Ludloff osteotomies, transfer metatarsalgia developed in 4% of the feet, similar to the rates of 0% to 3% rest.
With regard to delayed union, we observed a delay in healing in 2%
Like other proximal metatarsal osteotomies that are performed from the medial aspect of the metatarsal, the modified Ludloff osteotomy has a low risk for dorsiflexion malunion.
We recommend careful radiographic evaluation in the immediate postoperative period, particularly for patients over the age of sixty years and those with suspected osteopenia. Such patients should be restricted from bearing weight on the forefoot for a longer period of time in comparison with younger patients who have satisfactory fixation of the osteotomy site

Procedure

Operative Technique
A dorsal 3-cm longitudinal incision is made over the first web space. The lateral joint capsule and the metatarsosesamoid ligament are divided immediately superior to the lateral sesamoid. The transverse and oblique tendons of the adductor hallucis are released. The lateral capsule is fenestrated at the first metatarsophalangeal joint, and a varus stress is applied to the hallux to complete the lateral release. The transverse intermetatarsal ligament is not routinely divided.
A midaxial skin incision is made over the medial aspect of the first metatarsophalangeal joint, and an L-shaped medial capsulotomy is performed. Next, the metatarsal shaft is exposed with

two Hohmann retractors. From the medial side, an oblique first metatarsal osteotomy is performed from the dorsal-proximal aspect of the first metatarsal (immediately distal to the first tarsometatarsal joint) to the plantar-distal aspect of the first metatarsal (immediately proximal to the sesamoid complex). The medial-to-lateral orientation of the saw blade limits the potential for dorsiflexion of the distal fragment, and, to further combat this risk, the osteotomy is routinely oriented 10° plantarward. Only the dorsal two-thirds of the osteotomy is initially performed. A guide-wire for a 3.0-mm cannulated screw is then inserted in the proximal aspect of the dorsal fragment, perpendicular to the osteotomy, and is overdrilled. The first 3.0-mmscrew is then inserted into the drill-hole without full compression, and the osteotomy is then completed. The plantar fragment is gently pulled medially with use of a towel clip, and the dorsal fragment is rotated laterally with gentle thumb pressure applied to the medial aspect of the first metatarsal head . After the desired correction is confirmed fluoroscopically, the first screw is tightened to secure the osteotomy site. A second screw is then inserted from plantar to dorsal across the distal aspect of the osteotomy site. With protection of the plantar soft tissues, the second screw must be directed as perpendicular to the first metatarsal as possible; if this second screw is placed obliquely with a lag technique, the correction of the first metatarsal angle may be lost. With correction of large intermetatarsal angles, some degree of oblique screw positioning is required to achieve adequate screw purchase. In these cases, we recommend that the osteotomy site be stabilized with a clamp when the second screw is tightened. Once appropriate correction has been achieved, the medial eminence of the first metatarsal head is excised in line with the metatarsal shaft. After skin closure, the foot is wrapped in a traditional, mildly compressive Bunion dressing



6.Mandalia. Painful TKR. Journal of Bone and Joint Surgery; Mar 2008; 90, 3

Problems: Pain, Stiffness, Instability, swelling
Causes: Loosening, Infection, PF problems, Instability, Osteolysis
Pain: Describe, where, difference between pre and post
Early or late onset
Start up or pain at rest or night TKR

Baker: 8000 patients. 19.8% may have painful

Causes:
1. Infection
2. Instability
3.Malalignment
4.Impingement: overhanging components, patellar clunk, fabelle and Popliteus impingement
5. Arthrofibrosis
6. Wear
7.Extenor mechanism
8. Repeatedly hemarthrosis

1. Radiological assessment
2. CT assessment
3. Check for extension and flexion stability [I.I}
4. Blood, bone scan [Bpne scan in THR may return by 12 months; in TKR it may not return indefinitely [III phase]
5. Mismatch Indium scan and Sulphur colloid bone scan
6. IL 6 (>10 pg/mL). This is elevated in infection and not in loosening
7. Joint aspiration: Gram stain is not useful; Cell count >2500 and >60% PMNC significant, C&S

7. Ritter. PCR TKA. J Bone Joint Surg Am. 2008;90:777-784

Methods: We retrospectively reviewed 5556 primary total knee arthroplasties performed with posterior cruciate retaining prostheses between 1983 and 2003. The relationship between postoperative range of motion and pain, walking ability, stair-climbing ability, and knee function scores was examined at three to five years postoperatively. The relationship between a postoperative flexion contracture or hyperextension and knee function was also ex°amined.

Results: Patients with 128° to 132° of motion obtained the highest scores for pain, walking, and knee function and the highest Knee Society scores. The outcomes became substantially compromised with motion of <118°. Patients with 133° to 150° of motion had the highest scores for stair-climbing.

A postoperative flexion contracture and hyperextension were associated with lower scores for pain, walking, stair-climbing, and knee function.

Conclusions: The best functional results following total knee arthroplasty are achieved with 128° to 132° of motion. A postoperative flexion contracture and hyperextension of ‡10° are associated with a poorer outcome except that stair climbing is improved with more motion.

Discussion

Knee range of motion has long been recognized as an important outcome measure of TKA. This study is the first one, as far as we know, that has been designed to define optimal ranges of motion after total knee arthroplasty. The results were not completely uniform, but the trends in the data agree with past studies. Range of motion has been positively associated with walking ability, stair-climbing, and knee function scores1.

This study also found that pain scores are positively associated with range of motion. A new finding is that 128° to 132° was the ROM that had the highest average scores for every outcome measure tested, although it was not always significantly different from the immediately surrounding groups. Patients in this group even performed better than those in the group with higher range of motion. This suggests that there may be a limit to the benefits gained from increased range of motion, at least
for the parameters we measured. This study also showed that outcomes became substantially compromised with motion of <118°.

In addition to the range-of-motion data, flexion contracture and hyperextension were also shown to play a role in the success of total knee arthroplasty. A flexion contracture or hyperextension of ‡10° postoperatively led to lower scores for pain, walking, stair-climbing, and function. Genu recurvatum is most commonly seen in patients with extensor mechanism weakness, valgus deformity, rheumatoid arthritis, or paralysis, and this deformity is associated with negative arthroplasty outcomes23-25.We observed that flexion contractures were most prevalent in the patients with low ranges of motion, and hyperextension was most prevalent in patients with high ranges of motion. This is because a flexion contracture, or a loss of extension, decreases the range-of-motion calculation while hyperextension increases the RMO

The limitations of this study relate to the measurement of range of motion, to its retrospective nature, and to the evaluation of patients only from a Western culture. There is some question as to the reproducibility of goniometric measurements of knee flexion and extension. One study has found that 22% of goniometric measurements differ by >5° from radiographic measurements.

We found that a patient with 128° to 132° of motion and without a flexion contracture or without a hyperextension deformity appears to obtain the best functional results following a posterior cruciate-retaining total knee arthroplasty. This is true whether the patient had unilateral or bilateral knee
replacement. We are not sure that in a different culture, in which there is a need for a greater range of motion, the results would be the same.

8.Seymortier. Proximal tibiofibular dislocation.Am. J. Sports Med. 2008; 36; 793

Traumatic proximal tibiofibular dislocation is a rare injury that is often unrecognized or misdiagnosed at the initial presentation because of a lack of clinical suspicion. When diagnosed, the injury should be promptly reduced. Missed injuries or late presentations are a potential source of chronic morbidity.

The authors stress the importance of evaluating the distal syndesmotic ligaments and the interosseous membrane because the mechanism of trauma can also cause a disruption of the distal tibiofibular syndesmosis. In the presence of syndesmotic instability, prompt stabilization is advocated.

The main stabilizers of the proximal tibiofibular joint are the anterosuperior and the posterosuperior tibiofibular ligaments, which represent thickenings of the joint capsule. The joint capsule is much thicker anteriorly, being reinforced by both the anterosuperior tibiofibular ligaments and an extension of the biceps femoris tendon.

Additional stabilizers are the lateral collateral ligament, the biceps femoris tendon, the popliteal tendon, the arcuate ligament, the fabellofibular ligament, and the popliteofibular ligament. With knee extension, the fibular head is pulled posteriorly because the lateral collateral ligament and the biceps femoris tendon are tightened. With knee flexion, the fibular head migrates anteriorly due to relaxation of the lateral collateral ligament and the biceps femoris tendon.

Classification [Ogden]

Type I is an excessive AP motion without dislocation. It occurs before skeletal maturity and is often a self-limiting condition.

Type II represents an anterolateral dislocation. It is the most common form, accounting for 85% of proximal tibiofibular dislocations The mechanism of injury of the type II dislocation is a sudden internal rotation and plantar flexion of the foot combined with an external rotation of the leg and flexion of the knee1

Type III is a posteromedial dislocation often resulting from a direct blow to the fibular head (eg, a car bumper or gate post while horseback riding).9 This type accounts for 10% of proximal tibiofibular dislocations.

Type IV is a superior dislocation following a high-energy trauma often associated with a fracture of the fibular head, a superior dislocation of the lateral malleolus, and a rupture of the interosseous membrane.. An associated transient peroneal palsy is commonly seen in types II, III, and IV

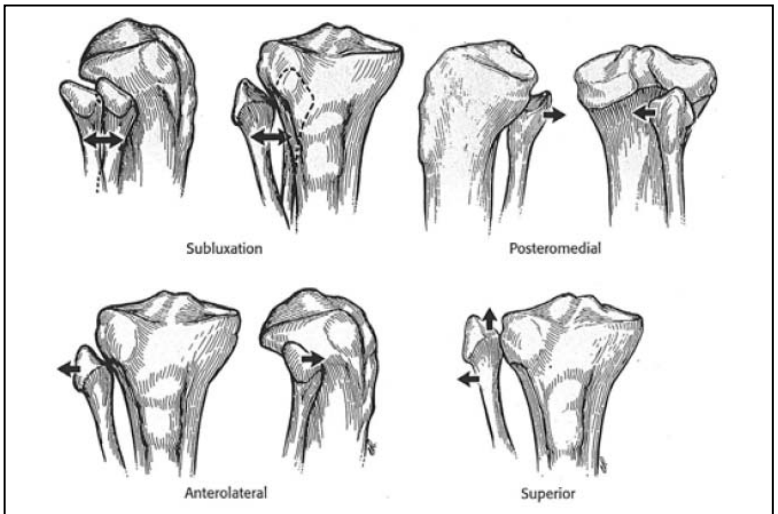


Figure 2. Classification of proximal tibiofibular dislocations as described by Ogden: subluxation (type I), anterolateral dislocation (type II), posteromedial dislocation (type III), and superior dislocation (type IV). (Reproduced with permission

The diagnosis of acute proximal tibiofibular dislocation is primarily a clinical one based on a thorough physical examination and a high level of clinical suspicion. It should be considered in any patient presenting with acute-onset lateral knee pain after an aggressive torsion trauma to a flexed knee (eg, soccer, parachuting, snowboarding, horseback riding, long-jumping, or direct-impact trauma).9

Clinical features may include lateral knee pain aggravated by palpation of the fibular head (often misdiagnosed as a lateral meniscal tear), a visible bony prominence in the region of the fibular head, and locking and popping of the knee when mobilized. Usually the knee has a full range of motion, but limitation due to pain is possible. Many patients are unable to bear weight because of pain. Knee effusion is usually absent. Ankle movement may exacerbate knee pain.21 Transient peroneal palsy has been described in anterolateral, posteromedial, and superior dislocations (types II, III, and IV).

Clinical examination of the ankle joint is mandatory to detect disruption of the syndesmotic ligaments and the interosseous membrane.

Clinical features include acute tenderness over the anterolateral joint line.

Plain radiographs (anteroposterior and true lateral views) are useful first-line investigations but are considered less accurate.10 Comparison anteroposterior and lateral plain radiographs of both knees are necessary to identify the subtle radiographic changes

CT is the investigation of choice.

Chronic Proximal Tibiofibular Joint Instability/Subluxation

Missed injuries or late presentations are a potential source of chronic morbidity. The diagnosis of chronic proximal tibiofibular instability is primarily a clinical one based on a thorough physical examination and a high level of clinical suspicion.

Treatment

Acute Proximal Tibiofibular Dislocation

Treatment options include closed reduction without postoperative immobilization, closed reduction with postoperative immobilization, and open reduction with temporary internal fixation.

As an initial treatment, closed reduction is advocated in all 4 types of acute proximal tibiofibular joint dislocation.

After reduction, reassessment of knee stability and the lateral collateral ligament is mandatory. Reduction of a type II injury is usually easy. Reduction of a type III or IV injury can prove more difficult. Closed reduction may fail when the fibular head is perched on the lateral tibial ridge by the lateral collateral ligament.

We prefer immobilization in an above-knee cast for a total of 3 weeks in combination with nonweight bearing.

Open reduction is indicated for failed closed reduction of an anterolateral dislocation, unstable reduction of an anterolateral dislocation

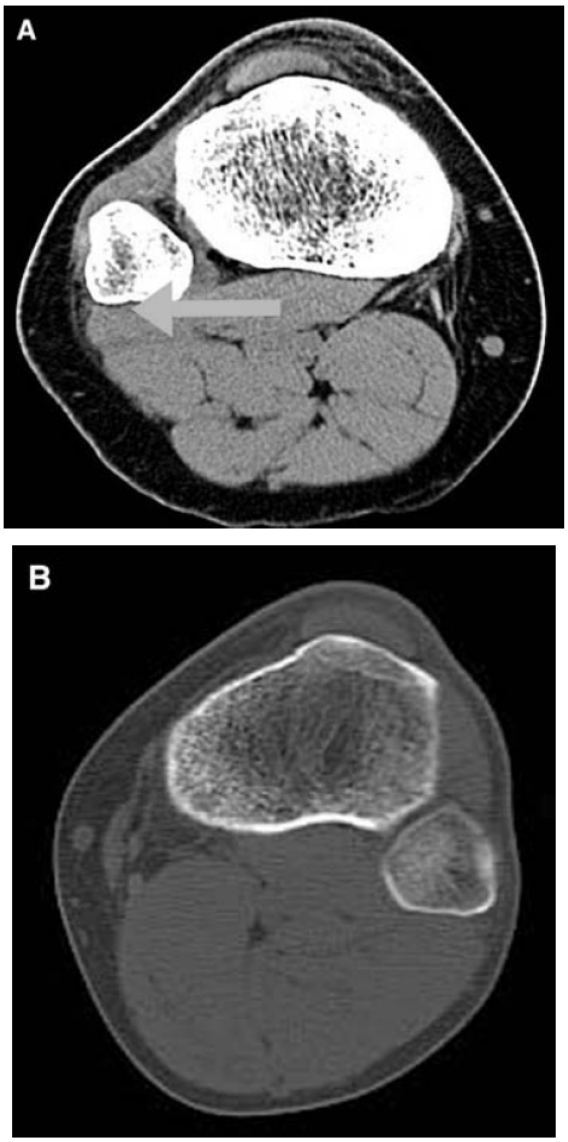
After open reduction, temporary internal fixation:: Kirschner wires, bioabsorbable pins, or a cancellous screw

The internal fixation devices should be removed after 6 to 12 weeks.

In case of a documented syndesmotic injury, proper fixation of the syndesmosis is mandatory.

Chronic Proximal Tibiofibular Joint

Surgical treatment options include resection of the fibular head, permanent arthrodesis of the proximal tibiofibular joint, soft-tissue reconstruction of the proximal tibiofibular joint, and temporary fixation of the fibular head.. `



e 5. A, axial view illustrating the anterolateral disloca-
f the tibiofibular joint (arrow). B, axial view showing the
al alignment of the proximal tibiofibular joint.

9.Lonner. What's new in TKA. J Bone Joint Surg Am. 2007;89:2828-2837

Epidemiology
The age and gender-adjusted incidence per 100,000 person-years increased over time, from 29in 1971 to 1975 to 157 in 2000 to 2003, representing a >400% increase in the incidence of total knee arthroplasty
The largest percentage increase was in patients less than fifty years old.
The volume of revision total hip arthroplasties is projected to grow from 40,800 in 2005 to 96,700 in 2030 (a 137% increase), and the volume of revision total knee arthroplasties is projected to grow from 38,300 in 2005 to 268,200 in 2030 (a 600% increase).

UKO

The shifting demographics of patients with Unicompartmental knee arthritis, including younger, more active patients, is a major impetus for the growing interest in conservative surgical alternatives
In a prospective study of 142 Unicompartmental arthroplasties performed with the same minimally invasive
approach, Fisher et al. found better knee scores and pain scores at one year in association with metal-backed tibial components as compared with all-polyethylene tibial components, although these differences were eliminated by three years.
The cost-effectiveness of Unicompartmental arthroplasty can be impacted by survival rates, implant selection, hospital costs, perioperative mortality, and utility values achieved with each procedure.
Patellofemoral arthroplasty is gaining popularity as a treatment method for isolated patellofemoral arthritis. Sisto and Sarin5 reported on twenty-five patellofemoral arthroplasties that had been performed with use of a custom implant and found that, after a mean duration of follow-up of 73 M; no patient required additional surgery and no knee had component loosening.
Lonner et al.6 reported on a series of twelve failed PFA in ten patients who had a revision to total knee arthroplasty. At the time of the most recent follow-up, there was no clinical or radiographic evidence of patellofemoral maltracking, loosening, or wear. It appears, therefore, that the results of total knee arthroplasty after a previous patellofemoral arthroplasty are not compromised.

MIS TKA

Despite the success of minimally invasive TKA as reported by many authors and the enthusiasm for the technique as reported in publications from 2005 until recently, not all studies have demonstrated clear advantages of minimally invasive techniques
Laskin et al.,TKA that had been performed without patellar eversion were compared with TKA that had been performed with patellar eversion. The authors found that an acquired patella Baja occurred in 37% of patients with patellar eversion during surgery, compared with 12% of those without patella eversion during surgery. The presence of patella baja resulted in reduced flexion and increased pain.
Agleitti: did not find any early benefit derived from the quadriceps-sparing approach.

Navigation

Many centers are harnessing the power of computers in an attempt to increase the precision of total knee arthroplasty component implantation and soft-tissue tensioning.
Most studies to date have focused on attempts to demonstrate a significant improvement in implant positioning, whereas few have aimed at identifying a clinical advantage.
In a case-control study by Stulberg et al.9, seventy-eight patients underwent total knee arthroplasty with use of either a conventional or a computer-assisted approach. There were no significant differences between the conventional and computer-assisted groups with regard to

limb alignment or component positioning. At one and six months, there were no significant differences between the groups with regard to clinical, functional, or pain scores. The surgical time was increased by twenty-seven minutes in the computer-assisted group, and the quantity of blood transfused was slightly higher.

DVT

Remains a controversial topic

Burnett et al. performed a prospective study to investigate the use of LMWH [10 day] for THA & TKA

The prevalence of symptomatic DVT was 3.8% and the prevalence of non-fatal pulmonary emboli was 1.3%. The rates of surgical site complications necessitating readmission, irrigation and debridement of a hematoma and the wound, or prolonged hospitalization for wound drainage were 4.7%, 3.4%, and 5.1%, respectively. Wound drainage occurred for four to seven days after 9.3% of the procedures.

Parvizi et al. found that patients who have development of a postoperative hematoma, wound drainage, or both had a significantly higher risk of development of a deep periprosthetic infection than those who did not.

Lonner et al.18 found no significant difference in the prevalence of post thrombotic syndrome in patients with asymptomatic deep-vein thrombosis after total knee arthroplasty as compared with those without venographically proven deep-vein thrombosis.

Westrich performed a randomized study of 275 patients undergoing TKA with spinal epidural anaesthesia, pneumatic compression devices, and administration of either enoxaparin or aspirin postoperatively for four weeks. The overall prevalence of deep-vein thrombosis was not significantly different between the drug treatment groups.

In the study by Gelfer et al. pneumatic compression combined with low-dose aspirin was compared with enoxaparin. The study included patients undergoing total hip arthroplasty as well as those undergoing TKA. Venographic evidence of DVT was observed in 28.3% of the patients in the enoxaparin group, compared with 6.6% of those in the pneumatic compression/aspirin group. While this difference was significant, the prevalence of adverse events was similar in both groups.

Complications

Patellofemoral complications after total knee arthroplasty have been mitigated by a greater appreciation of component rotation and position as well as design improvements; however, they remain a potential source of problems.

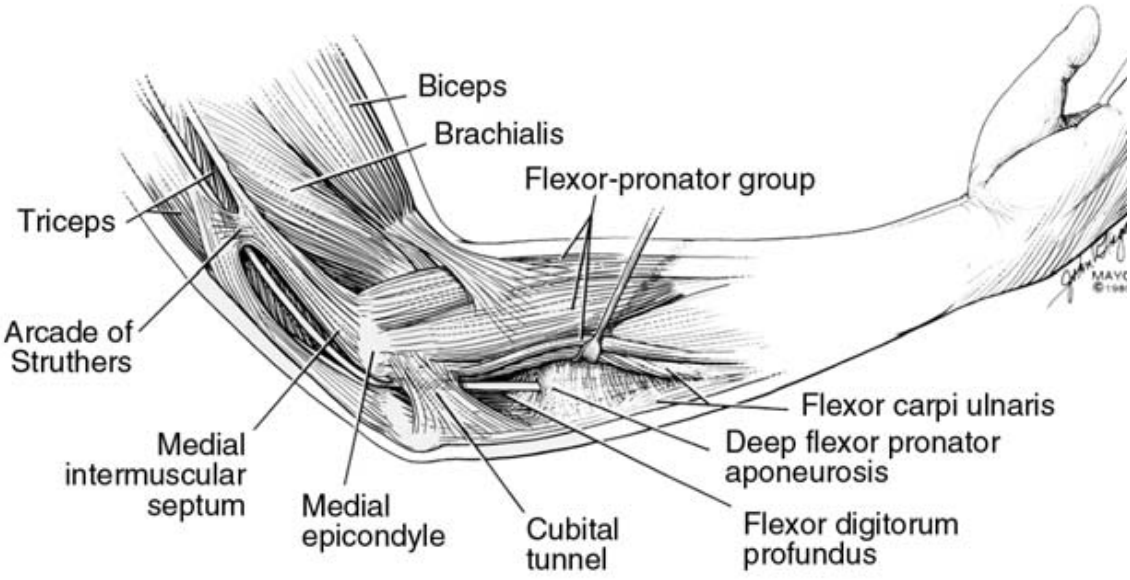
Gandhi et al.32 described the risk factors for the development of postoperative stiffness (de-fined as flexion of <90_) in an analysis of 1216 TKA.. At one year, they found a 3.7% rate of stiffness. On the average, for every increase of 2 mm in patellar thickness, a decrease of 3° in maximum passive flexion resulted.

Namba presented the results of early and late manipulation for the treatment of stiffness in flexion. One hundred and two patients underwent early manipulation (less than ninety days postoperatively) and ninety-three patients underwent late manipulation (more than ninety days postoperatively). In the early manipulation group, the average flexion improved from 68° to 101°. In the late manipulation group, the average flexion increased from 81° to 98°. All patients had a decrease in pain

Barrack reported the results for patients who had an unexpected positive bacterial culture at the time of revision TKA. Of 692 patients who had a revision for the treatment of aseptic complications, 5.3% had an unexpected positive intraoperative culture. They were managed with six weeks of intravenous antibiotics.

10. Elhassan. Entrapment Ulnar Neuropathy JOAAS 2007;15:672-6811

Ulnar nerve entrapment is the second most common nerve entrapment syndrome of the upper extremity. Although it may occur at any location along the length of the nerve, it is most common in the cubital tunnel. Ulnar nerve entrapment produces numbness in the ring and little fingers and weakness of the intrinsic muscles in the hand. Patient presentation and symptoms vary according to the site of entrapment. Treatment options are often determined by the site of pathology. Many patients benefit from nonsurgical treatment (eg, physical therapy, bracing,injection). When these methods fail or when sensory or motor impairment progresses, surgical release of the nerve at the site of entrapment should be considered. Surgical release may be done alone or with nerve transposition at the elbow. Most patients report symptomatic relief following surgery.



The five sites of potential ulnar nerve entrapment around the elbow: arcade of Struthers, medial intermuscular septum, medial epicondyle, cubital tunnel, and deep flexor pronator aponeurosis

Decompression

With in situ decompression, all sites of compressions are relieved, keeping the nerve in its bed. Wilson and Krout27 reported on 16 consecutive patients (17 elbows) treated with simple decompression. They reported eight excellent, five good, and four fair outcomes, as well as one revision. The authors concluded that the best outcome of simple decompression is obtained in the patient with mild weakness, recent onset of symptoms, and mild abnormality of sensory action potentials.

Transposition

Relieves the biomechanical mechanism of cyclic traction and compression on the ulnar nerve by placing the nerve anterior to the axis of elbow motion. In subcutaneous transposition, the nerve is placed superficial to the flexor-pronator origin, and a fascial sling is created to prevent nerve migration. Results: Good in (83%), satisfactory in 1 (6%),and unsatisfactory in 2 (11%). Subcutaneous ulnar nerve transposition is technically easier to perform than either submuscular or intramuscular transposition. However, its disadvantages include inadequate decompression of all areas of compression and, especially in the thin patient, the vulnerability of the nerve to repeated trauma because of its position in the subcutaneous tissue.

For moderate to severe compression, anterior submuscular transposition offered the best results with the lowest recurrence rate, whereas anterior intramuscular transposition gave the fewest excellent results and the most clinical presentations of recurrence of ulnar nerve symptoms.

Nabhan et al37 compared simple decompression with anterior subcutaneous transposition in 66 patients with cubital tunnel syndrome. Thirty-two patients underwent simple decompression, and 34 underwent anterior subcutaneous transposition.No significant difference in pain, motor and sensory deficits, or nerve conduction velocity studies was found between the two groups at 3- to 9-month follow-up. The authors recommended simple decompression of the ulnar nerve.

II Free Paper

Acute Periarticular calcification [HADD] – an unusual cause of carpal tunnel syndrome

Vasu Pai MS, D[orth], National board [Orth], FICMR, FRACS[Orth], MCh[Orth]
Vishal Pai
Midlemore Hospital
New Zealand

INTRODUCTION

Acute carpal tunnel syndrome is an uncommon condition. When present, it usually follows a carpal injury or fracture of the lower end of the radius.

We describe a case of acute carpal tunnel syndrome in a 64 year old woman caused by periarticular calcific deposits around the wrist joint. Emergent surgical decompression of the carpal tunnel with removal of the calcified material was required and has led to remission of pain and recovery of median nerve function. The biopsy material is reported to be hydroxyl apatite crystals consistent with Hydroxyl apatite deposition disease [HADD]

CASE REPORT

A 64 year old woman presented to the emergency department with severe pain in her right wrist of two days duration. This had been preceded by 6 weeks of mild discomfort but no loss of function following a mild 'jarring' while gardening. She reported that this acute severe pain was worst at night (10 out of 10) and exacerbated by wrist movements and extension of the fingers. It had not responded to paracetomol and NSAIDs prescribed by her general practitioner the preceding day. There was a sensation of 'pins and needles' in her hand. This progressively got worst. Her past medical history was not suggestive of gout or hyperparathyroidism.

On examination the patient was afebrile with heart rate and blood pressure within normal limits. She was in obvious discomfort and held the fingers of her right hand in a flexed position. A diffuse swelling of the volar aspect of the right wrist was noted. Attempts at active and passive movement of the wrist and extension of the fingers were markedly limited as they caused excruciating pain. A reduction to sensation of light touch was noted in the distribution of the median nerve. There was objective sensory loss (2-point discrimination greater than 20 mm) in the median nerve distribution. The muscles of the forearm were neither tense nor tender to palpation. Radial pulse and capillary refill appeared to be normal.

Due to the degree of pain she experienced on movement it was difficult to elicit Phalen's or Tinel's sign, nor to test power in the muscles supplied by the median nerve

Full blood count, urea, electrolytes, uric acid, calcium, phosphate, alkaline phosphotase and thyroid function were all within normal ranges and rheumatoid factor was negative. Plain radiographs **[Fig 1]** of the wrist showed a 2 cm x 2cm large, dense shadow near the ulnar and volar aspect of the right radiocarpal joint with no other bony abnormality or joint lesion. An urgent CT scan of the same region showed lobulated calcification in the wrist joint close to the volar capsule **[Fig 2]**.

The patient was admitted for pain relief and emergent surgical decompression of the carpal tunnel. This was performed within three hours of her presentation to the emergency room

under general anaesthesia. An extended carpal tunnel incision was used to decompress the median nerve. There was an evidence of significant tenosynovitis of common flexors **[Fig 3].** The median nerve appeared congested. Tenosynovectomy of flexor tendons was performed. On retracting flexor tendons, there were two areas of nodular swelling **[Fig 4]** in the capsule which on exploration revealed chalky material with "tooth paste like consistency".

The biopsy of the nodule reported to be acute calcification around the joint which was histologically consistent with acute periarticular calcification [hydroxyl apatite crystals]. The tenosynovium revealed nonspecific inflammatory cells. Culture was negative and polarising light did not reveal negative birefringent crystals. With this clinical, radiological, histological findings were suggestive HADD [periarticular calcification]

There was dramatic relief after surgery. Wrist was held in a slab for a week and was given a course of NSAID for two weeks. After removal of slab active mobilization is commenced. At one year, patient was symptom free.

DISCUSSION

The syndrome of acute compression of the median nerve has only recently been reported (Bauman et al 1981). Although fractures of the distal radius are the most common underlying injury [2,3] the syndrome may also be seen with carpal injuries [4,5,6] . Nontraumatic acute carpal tunnel syndrome been described secondary to: infective tenosynovities [7], coagulopathies [8], false aneurysm [9] , gout or rheumatologic disorders[3]

Local pressure from haematoma or thickened synovium may contribute to fibrosis, probably by causing ischemic damage within the nerve. It is important, however, for medical staff to recognise the symptoms of acute carpal tunnel syndrome, as there is potential for long-term median neuropathy if treatment is delayed[3,10,11]. Regardless of the underlying cause, operative decompression is indicated as soon as possible when the diagnosis of acute carpal tunnel syndrome is suspected. Delay in treatment may result in incomplete recovery and may be responsible for long term sequelae ..

Calcific periarthritis is an uncommonly reported condition characterised by periarticular deposition of calcium hydroxyapatite crystals in bursae, tendons and ligaments. It was first described in 1870 in the shoulder and this remains the most commonly affected joint, but there is the potential for deposition around any joint. Crystal deposits are often asymptomatic and detected as incidental findings, but may cause acute calcific periarthritis (thought to be due to rupture of the deposit into surrounding soft tissues leading to an inflammatory response) with localised pain, tenderness and loss of function. These acute episodes typically resolve gradually over two to three weeks without treatment, only rarely causing chronic pain or dysfunction.

One of the confusing aspects of periarticular calcification is the associated nomenclature. A variety of terms have been used. A very characteristic homogeneous cloudlike appearance distinguishes HADD [as the present case] from most other disorders. Finding calcifications in the specific sites favoured by HADD without an underlying disorder should separate this entity from others in the differential diagnosis.[12]

HADD should not be confused with the more linear and diffuse CPPD crystal calcifications [Table 1]. Gouty tophi are more faintly calcified and are associated with elevated urate levels. Heterotopic bone and myositis ossificans have a trabecular pattern with a cortical rim that can be distinguished from HADD and CPPD calcifications. Tumoral calcinosis, either primary

idiopathic or secondary to renal disease, may mimic HADD if it presents in small amounts. One should look for a metabolic disorder in the latter situation to distinguish this cause. Collagen vascular disease such as scleroderma or dermatomyositis can also produce calcifications that can mimic HADD. These calcifications are usually more widespread, can involve the subcutaneous tissues, and are associated with a known underlying disease. Periarticular metastatic calcification may be seen in association with sarcoidosis, hypervitaminosis D, hypoparathyroidism, and milk–alkali syndrome.

The carpal joints are infrequently affected by acute calcific periarthritis and reports in the literature of acute carpal tunnel syndrome caused by calcific deposits are very rare, consisting only of case reports[13-18]. Most of these reports[13-17] describe cases where small calcifications within the confines of the carpal tunnel led to symptoms of carpal tunnel syndrome which responded well to conservative treatment with immobilisation and NSAIDs. In most cases the symptoms resolved completely within 3 weeks with gradual resorption of the calcifications. 2 cases[18,19] such as the one we present here in which a large apatite deposit produced severe pain deficit and acutely threatened the median nerve such that immediate surgical decompression was indicated is very rare.

The reason for the development of periarticular calcific deposits remains unclear, but a local trauma (as is the case in our patient) may precipitate calcific lesions. Recurrence appears to be uncommon and our patient continues to have good function of her hand and wrist with no further pain.

In conclusion, this rare case should remind orthopaedic surgeons and radiologist to consider periarticular calcifications when abnormal deposits seen in the vicinity of the joint within the carpal tunnel when evaluating for acute carpal tunnel syndrome,.

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LEGENDS

Fig 1
A plain anteroposterior radiograph of the right wrist, showing a amorphous opacity at the wrist level anteriorly

Fig 2
CT demonstrating an amorphous deposit of high signal intensity suggestive of calcified mass anterior to the carpal bones

Fig 3
Intraoperative photograph of the wrist, showing congested median nerve with tenosynovitis of the flexor tendons.

Fig 4
Intraoperative photograph of the wrist, showing chalky, granular deposits in the volar capsule of the wrist consistent with hydroxyapatite crystals

Fig 1

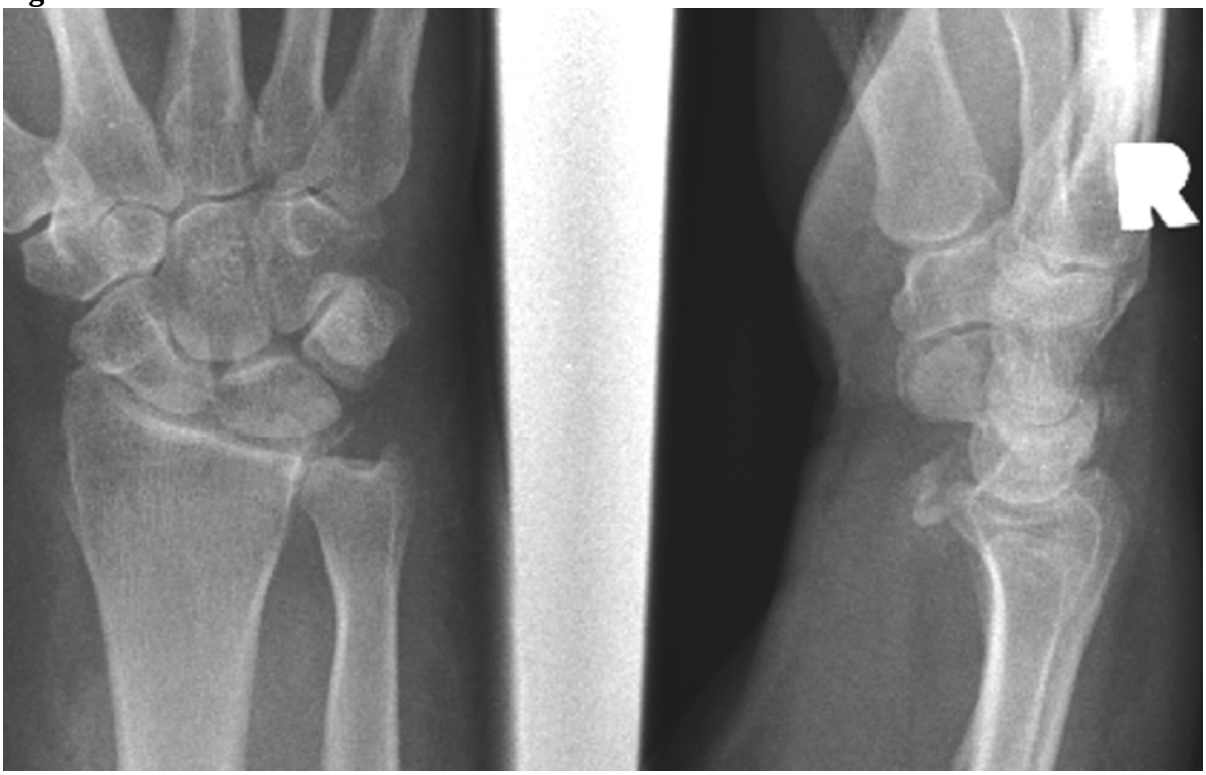


Fig 2

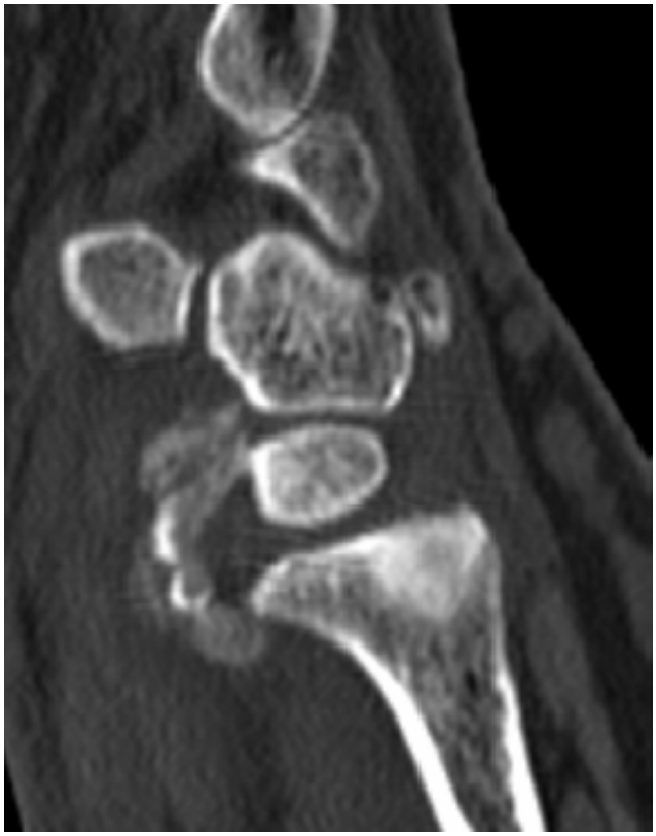


Fig 2

Fig 3

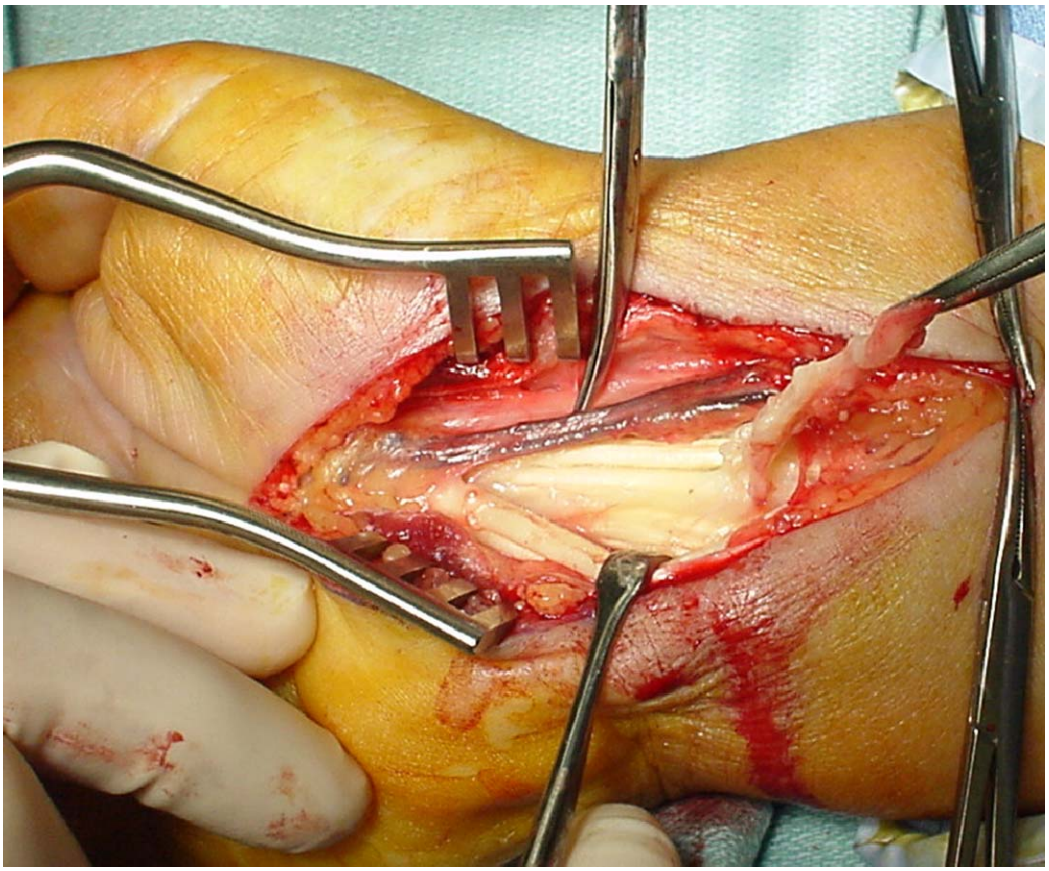


Fig 4

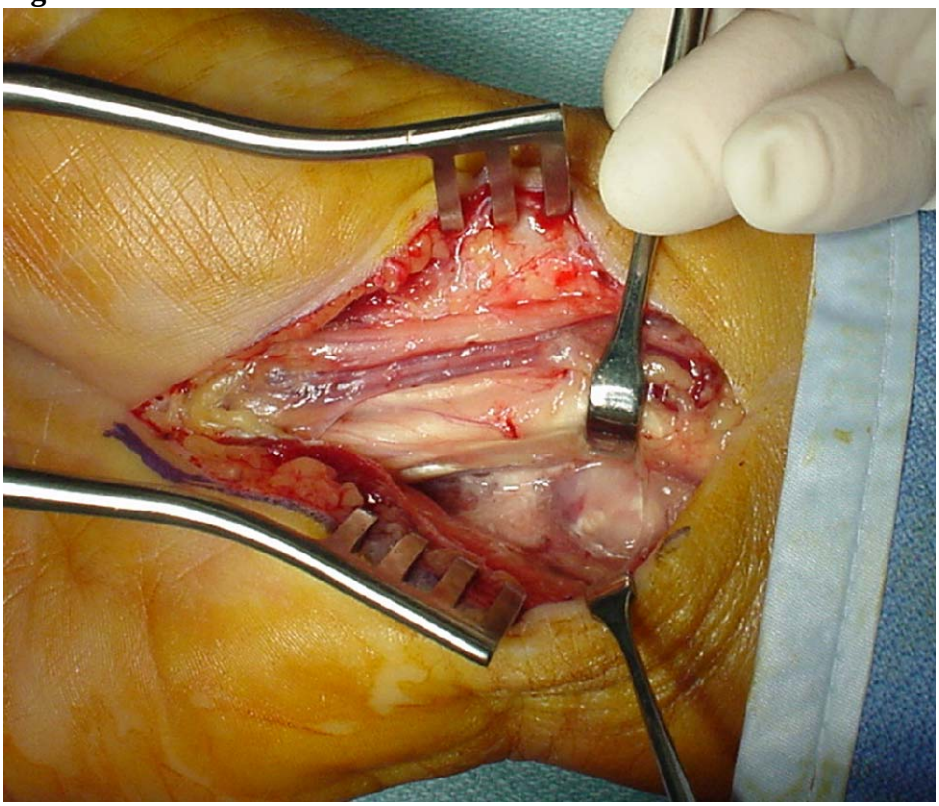


Table 1

Calcium pyrophosphate [CPPD]	Calcium hydroxyapatite crystal [HADD]

The most common crystalline arthropathy,	Less common
Usually polyarticular . Sites: knee >hip,>shoulder , elbow	Usually monoarticular and most commonly presents: Shoulder> elbow. MPJ, Wrist
Tendon: The sites more frequently involved include: the supraspinatus , triceps, quadriceps, Achilles	The most common site of deposition is in the flexor carpi ulnaris tendon near its attachment to the pisiform
Acute/subacute/chronic and usually self limiting	Acute/subacute/chronic and usually self limiting
X ray: Prominent linear or punctuate deposits and parallel the subjacent subchondral bone	X ray: Calcifications in HADD are more homogeneous or cloudlike,

III Notes:. CONGENITAL UPPER LIMB DEFORMITIES

SWANSON'S CLASSIFICATION

1. Failure of Part formation

Transverse absence: Congenital amputation

Longitudinal absence: Radial Club hand
Ulnar Club hand
Cleft hand

2. Failure of Part Differentiation
Radioulnar synostosis
Camptodactyly
Trigger thumb
Syndactyly

3. Duplication
Polydactyly: Pre-axial, Post-axial

4. Overgrowth
Macrodactyly

5. Undergrowth
Thumb hypoplasia (Blauth)

6. Miscellaneous

Constriction ring syndrome

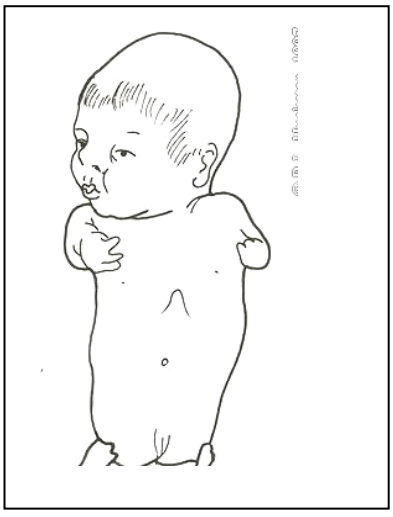
Congenital dislocation of radial head

Madelung's deformity

FACTS

Congenital anomalies affect 1% to 2% of newborns

Approximately 10% of those children have upper-extremity



Genetics

Trisomy of 21, 18, 13 Polydactyly, Syndactyly:

Apert's syndrome AD

Environmental

Drugs: Talidomide, Dilantin, Warfarin

ASSESSMENT

Initial visit: an emotion filled event.
Underlying guilt.
Parents have difficult for families to accept.
Risk for Future Pregnancies: Genetic counseling

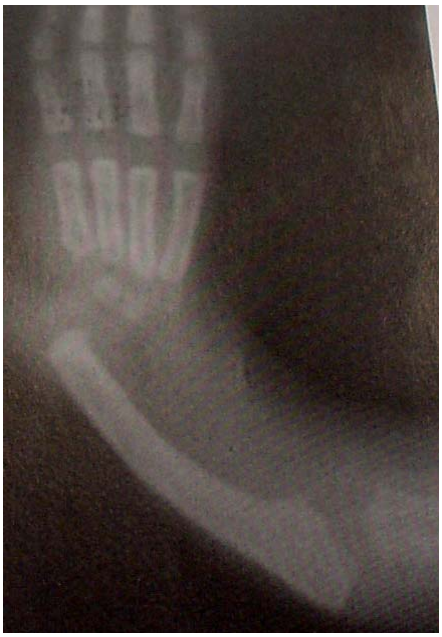
TRANSVERSE AMPUTATION

Part distal is missing.
Rare
Eg: Absence hand at wrist

INTERCALARY AMPUTATION

Segment in between is absent.
More common with Talidomide Intake during pregnancy

RADIAL CLUB HAND



1: 30,000

Bilateral in 60%

Clinical

Absence of radius and radial rays

Bowing of the ulna

Associated Anal atresia

Tracheo- oesopharyngeal fistula

Radial bone absence

Renal anomalies

Cardiac anomalies

Treatment

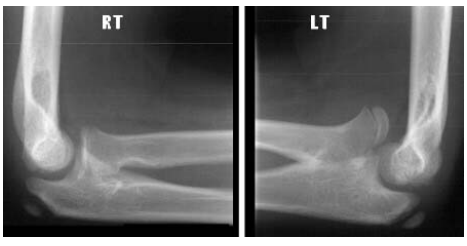
Use of serial casts

Realignment and stabilization: Lower end of ulna is transferred radially.

Tendon transfers - Ulnar side tendons transferred radial side

Correction of the ulnar bowing – osteotomy

RADIO-ULNAR SYNOSTOSES



AD;

Bilateral in 60%

Site: Proximal 1/3 of forearm

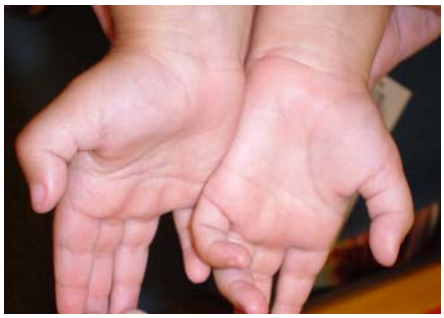
Deformity: Forearm pronation

Rx: Leave it alone.

When bilateral: rotational osteotomy in one forearm to a

Functional position

TRIGGER THUMB



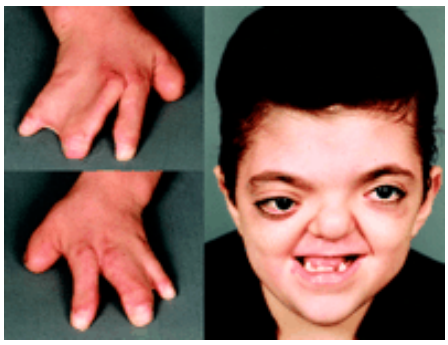
Often bilateral, with fixed flexion of thumb

If present >1 yrs Rarely recover

Treatment: - Surgery

Release A1 pulley [base of the tendon sheat

SYNDACTYLY



Syndactyly: Syn = together in Greek

Mnemonic 5,15,50,30:[Thumb and Index 5%; Ring and lit

More common in white males, AD

Present bilaterally in 50%

Types: I Isolated

II Complex Trisomy 13, 14, 21

Apart syndrome [clinodactyly; facies]

Holt Oram Syndrome [ASD, Radial club hand]

Poland's [Absence of Pectoralis major]

Treatment

Surgery: At 18 months old.

Full-thickness skin grafting is almost always required for soft tissue coverage.

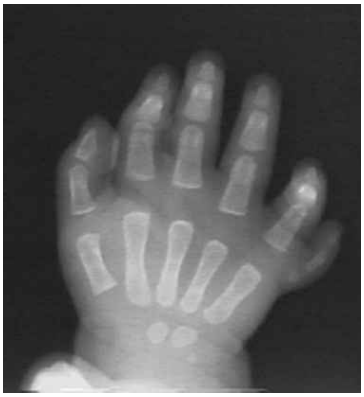
Technique

Mark the skin flaps Z plasty

Dorsal butterfly flap, to make the web

Both sides of the same digit : never separated on the same day (>6 wks)

POLYDACTYLY



POST AXIAL POLYDACTYLY

Post axial [next to the little finger]

Is a common hand anomaly

10 times more in Blacks

AD

Nonsyndromal

PRE AXIAL POLYDACTYLY

Preaxial [next to the thumb]

Is common duplication in whites

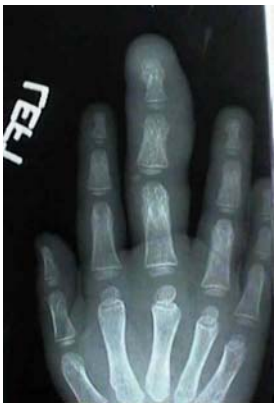
AR [17 chromosome]

Syndromal

Treatment

Excise the rudimentary finger

MACRODACTYLY



Hamartomatous enlargement of soft tissue and underlying bone

Associated with neurofibromatosis

When hemi-hypertrophy, suspect

Wilm's tumor

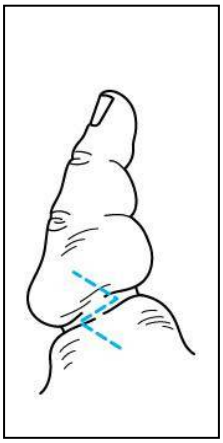
Adrenal carcinoma

Hepatoblastoma

Treatment Debulking, Osteotomy

Epiphysiodesis

CONSTRICTION RING SYNDROME [CRS]



Also called: Streeter's syndrome

1 in 15,000 live births

The etiology of CRS is unclear

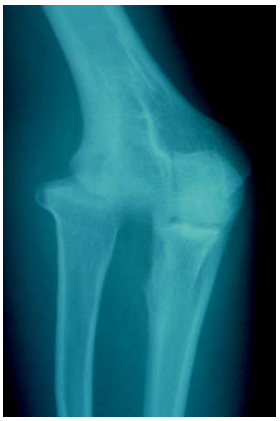
It is not genetic

CRS frequently affects both arms and all four limbs

Commonly associated with clubfoot

Surgical treatment: Z-plasty of constriction

CONGENITAL DISLOCATION OF THE ELBOW



Incidence: 0.16%

D/D

Old Monteggia

Multiple endochondromatosis or exostosis

Radio-ulnar synostosis

Clinical: Family history; Bilateral; anomaly

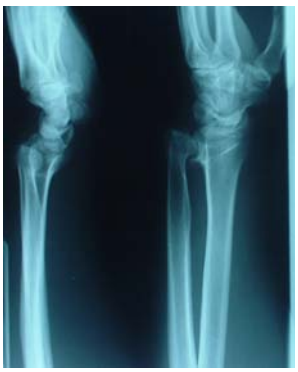
Loss of supination

Radiological: Hypoplasia of the Capitullum

Dome shaped Head

Treatment: If painful, excision after maturity

MADELUNG'S SYNDROME



Epiphyseal arrest on ulnar and volar half of the distal radius

Joint is directed ulnarward and volarward

AD variable expressivity; Females (4:1)

Wrist motion: Extension and supination are limited

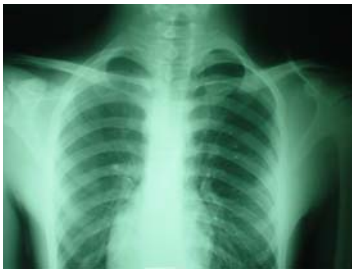
X ray may show: Classical deformity

Inferior Radio-ulnar dislocation

Treatment Corrective osteotomy

Physeal resection and fat graft`

SPRENGEL'S SYNDROME



Failure of the normal caudal migration of the scapula

Associated with Congenital scoliosis

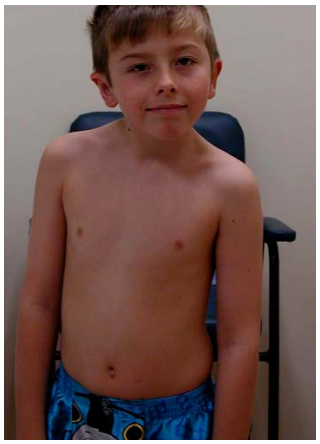
Klippel Fail syndrome

The superomedial scapula is connected to the proximal cervical spinous process through an omovertebral

connection (bone, cartilage) in 30%

Treatment: Surgical correction

TORTICOLLES



Associated with other "Molding disorders"

Hip dysplasia

Metatarsus adductus

Plagiocephaly

Treatment Stretching exercise 90% good results

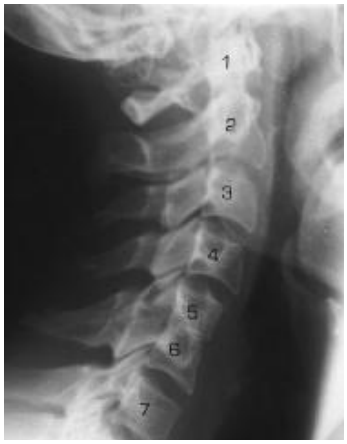
. Surgery if necessary at one yr (Distal or proximal release or both)

Look for Upper cranial anomaly

Ophthalmologic disorder

Posterior fossa brain tumor

KLIPPEL FAIL SYNDROME



Low hair line

Limited ROM of the neck

Can be associated with Sprengle's shoulder,

Renal or cardiac anomaly

Avoid contact sports

IV. Current concept: Osteoporosis: Management and Treatment Strategies for Orthopaedic Surgeons. Laura Gehrig, MD[1], Joseph Lane, MD[2] and Mary I. O'Connor, MD[3]
J Bone Joint Surg Am. 2008;90:1362-1374.

Introduction

The purpose of this lecture is to provide orthopaedic surgeons with a guide for osteoporosis management and treatment that may be used in the practice setting. Fracture prevention is the key efficacy end point in the medical management of osteoporosis for any patient. Enhancement of bone mass and improvement of bone quality are achieved by a combination of lifestyle modification, dietary supplementation with calcium and vitamin D, and pharmacologic treatment. This strategy has proved effective for the prevention and treatment of osteoporosis.

The orthopaedic surgeon is in a unique position to identify patients with osteoporosis. As the orthopaedic surgeon is often the only physician to see a patient who has sustained a fracture, he or she must make every effort to determine if the injury is a fragility fracture so that the patient can be treated to prevent future fractures.

Treatment

Nonpharmacologic Treatment

A multidisciplinary approach is essential in the treatment of osteoporosis. Nonpharmacologic treatments are used to complement pharmacologic therapy and thus optimize fracture risk reduction. Commonly used nonpharmacologic interventions include calcium and vitamin-D supplementation, fall prevention, hip protectors, and balance and exercise programs.

Calcium Supplementation

Optimal bone health depends on adequate calcium. A normal calcium status is defined as a corrected serum calcium level of 9.5 to 10.5 mg/dL (2.4 to 2.6 mmol/L). The National Osteoporosis Foundation recommends a daily calcium intake of 1000 mg/day for men and women under the age of fifty years and 1200 mg/day for men and women over the age of fifty years[1]. Since a typical American woman consumes approximately 600 mg of calcium through diet alone, supplementation is indicated for the majority of patients. Supplementary calcium is available in two forms, calcium carbonate and calcium citrate. Calcium citrate is the preferred form. The use of calcium carbonate by individuals with a physiologic or pharmacologically induced reduction in acid production results in suboptimal calcium absorption, as calcium carbonate requires a low pH for salt dissociation[2]. The incidence of kidney stones is decreased in patients taking supplemental calcium citrate instead of calcium carbonate as citrate binds to oxalate, reducing its intestinal absorption. In an effort to optimize absorption, total daily calcium supplementation should be divided throughout the day with individual doses limited to <500 mg[3].

Vitamin D

Orthopaedic surgeons know that vitamin D plays a critical role in promoting absorption of calcium from the gut and that insufficient absorption results in lower serum calcium levels. These lower levels trigger the release of parathyroid hormone, which mobilizes calcium from bone (secondary hyperparathyroidism), ultimately resulting in osteopenia and eventually osteoporosis. Recent studies have also indicated that patients with osteoarthritis can have osteoporosis as well as vitamin-D deficiency[4].

Vitamin-D deficiency has been shown to increase the risk of falls by the elderly[5-7]. In a recent randomized controlled trial, the impact of a high dose of vitamin D on nursing home residents' risk of falling was compared with that of a placebo over a five-month period[6]. The researchers found a 72% reduction in the risk of falls for individuals given 800 IU of vitamin D2 plus calcium compared with those who received a placebo. Moreover, severe vitamin-D deficiency is associated with persistent, nonspecific musculoskeletal pain[8].

Beyond the musculoskeletal system, vitamin D influences many other organ systems (the brain, heart, gut, skin, pancreas, and immune system). These organs have cells with vitamin-D receptors and may even express the enzyme to convert vitamin D to its active form[9]. Furthermore, insufficient vitamin D has been associated with type-1 diabetes, multiple sclerosis, Crohn disease, hypertension, cardiovascular disease, schizophrenia, depression, rheumatoid arthritis, and osteoarthritis[10]. With insufficient vitamin D, the serum level of calcium is also at risk of being insufficient. This couples the physiologic state of low calcium and vitamin D to these diseases and to the skeleton of those who have these diseases. The skeletons of those with these diseases are at risk for low bone density, osteoporosis, and fracture.

Sources of Vitamin D

Vitamin D can be obtained from three sources: exposure of skin to sunlight of adequate ultraviolet strength, diet (such as salmon, tuna, sardines, and cod liver oil) including fortified foods (breakfast cereals, milk, some orange juices, and yogurts), and dietary supplements. Synthesis of vitamin D from the skin occurs with exposure of 7-dehydrocholesterol, a lipid in the dermis, to pre-vitamin D3. Approximately one to fifteen minutes of sun exposure to the hands and arms two or three days per week is thought to be adequate. However, the intensity of the sunlight is critical. In northern latitudes such as Boston and Seattle, there is no vitamin production from November through February regardless of the length of sun exposure[11]. In Los Angeles and Atlanta, vitamin-D3 synthesis is adequate throughout the year. Use of sunscreen dramatically reduces vitamin-D3 synthesis, with 99% eliminated with the use of a sunscreen with a sun protection factor (SPF) of 15 and 92.5% eliminated with use of a SPF-8 sunscreen[9,12]. Synthesis is decreased, potentially by as much as 99%[12], in individuals with dark skin pigmentation. Furthermore, the epidermis thins with aging. Lipid content is lost with a resultant estimated 75% reduction in vitamin-D synthesis in a person who is seventy years old[9].

Dietary supplements, therefore, are a very important source of vitamin D. Both vitamin D2 (usually labeled as calciferol or ergocalciferol) and vitamin D3 (usually labeled as cholecalciferol) are used in over-the-counter supplements, but the form available by prescription in the United States is vitamin D2[10]. Vitamin D3 is the preferred form, as vitamin D2 is only approximately 30% as effective in maintaining serum 25-hydroxyvitamin-D levels[13,14]. If vitamin D2 is used, up to three times as much of the vitamin may be required[10].

Vitamin-D Supplements and Risk of Fracture

It is well established that many patients with osteoporosis or a history of a fragility fracture have suboptimal levels of vitamin D. Furthermore, the prevalence of low vitamin-D levels is greater in individuals in nursing homes than in those living in the community. A meta-analysis of studies in which individuals were given 400 IU of vitamin D3 per day showed little benefit in terms of reduction of hip or vertebral fractures. However, higher doses of vitamin D have been found to have benefits. In individuals with inadequate vitamin-D levels of 17 ng/mL (42.4 nmol/L), 700 to 800 IU of vitamin D per day resulted in a mean increase in

vitamin-D levels of approximately 40 ng/mL (99.8 nmol/L) and a reduction in the prevalence of both nonvertebral and vertebral fractures[5].

Ethnic differences have also been observed relative to vitamin D and fragility fractures. In a series of eighty-five patients with acute fragility fractures, black and Hispanic patients were significantly younger than whites ($p < 0.001$) and more likely to have serious comorbidities such as diabetes or hypertension. Perhaps of even greater interest is the fact that, despite significantly higher bone mineral density values ($p < 0.01$), blacks had the highest rate of vitamin-D deficiency and secondary hyperparathyroidism[15].

Recommendations for Vitamin-D Supplementation

The current recommendations from the Institute of Medicine are 200 IU daily from birth to the age of fifty years, 400 IU daily for adults fifty-one to seventy years of age, and 600 IU daily for those seventy-one years of age and older[16]. Many experts in the field consider these recommendations to be too low and believe that the minimum adult intake should be 800 to 1000 IU daily.

Higher doses of vitamin D are required to replenish depleted total body stores. Fifty thousand international units of ergocalciferol (vitamin D2) can be taken orally twice a week for six to eight weeks, followed by a maintenance dose of 1000 IU per day. Toxicity, even with these higher doses, is very rare. Doses of up to 10,000 IU per day for up to five months have not caused toxicity[17].

Evaluation of the vitamin-D level followed by treatment if a deficiency is found is now part of the management of osteoporosis. This is essential as vitamin-D deficiency is completely preventable and reversible.

Life style

Lifestyle evaluation is an important component of the comprehensive treatment for osteoporosis. In addition to encouraging smoking cessation and moderation of alcohol consumption, physicians should also counsel patients about fall prevention and appropriate exercise training to further reduce the risk of fracture.

Fall Prevention and Hip Protectors

The evaluation of osteoporotic patients' risk of falling and the initiation of appropriate intervention are important for fracture prevention. Fracture prevention is most effective when both intrinsic and environmental risk factors for a fall are taken into consideration. Physicians should limit sedative medications when possible, recommend regular weight-bearing exercise, consider physical and occupational therapy for fall prevention, and facilitate environment modification such as the installation of assistive devices in the home. In addition, clinicians may encourage their patients to wear hip protectors, which effectively attenuate force from a fall and are associated with >50% reductions in the risk of hip fracture as well as improvement in the patient's self-confidence that they can avoid a fracture if they fall[2,18]. However, compliance is low as many patients find hip protectors difficult to manipulate when they dress and undress[2].

Balance, Posture, and Exercise Training

Osteoporotic patients are likely to benefit from programs that target balance, posture, and strength. Balance training programs are ass-ociated with an approximate 50% reduction in the

incidence of falls. Postural exercise programs have been shown to increase back extensor strength. Activities such as tai chi may be particularly helpful, with intense training programs reducing the risk of falls in elderly populations by as much as 47%[19]. Careful attention must be paid to identifying appropriate weight-bearing activities, as fragile patients with severe osteoporosis are known to sustain new fractures during routine activities such as bending over and turning in bed[18].

Pharmacologic Treatment

The pharmacologic agents currently available are commonly divided into two classes, antiresorptive and anabolic. Antiresorptive agents such as the bisphosphonates limit bone resorption through inhibition of osteoclast activity. The anabolic agent parathyroid hormone promotes active building of bone mass. Both antiresorptive and anabolic agents have demonstrated antifracture efficacy in randomized clinical trials[18].

Antiresorptive Agents

The antiresorptive agents currently approved for use in patients with osteoporosis include calcitonin, hormone replacement therapy, selective estrogen receptor modulators, and bisphosphonates.

Calcitonin

Calcitonin effectively inhibits bone resorption by decreasing osteoclast formation and activity[20,21]. Calcitonin acts quickly. Its effects are reversible and transient. This is likely due to its rapid clearance from the body and desensitization and internalization of the calcitonin receptor with prolonged exposure[21,22]. Calcitonin has been approved by the U.S. Food and Drug Administration for treatment of established osteoporosis but not for prevention of postmenopausal osteoporosis. It is available as both a parenteral injection and nasal spray. The intranasal formulation of calcitonin is the most widely prescribed because of its ease of use and superior tolerability[21].

Nasal calcitonin has proven to decrease bone turnover and modestly increase bone mineral density over one to five years[20,21]. Despite this increase, gains in bone mineral density are not maintained after discontinuation of treatment[21]. The efficacy of calcitonin in reducing the risk of vertebral fractures was best examined in the PROOF (Prevent Recurrence of Osteoporotic Fractures) study[20]. This five-year, double-blind, randomized, placebo-controlled study of 1255 postmenopausal osteoporotic women showed that treatment with 200 IU of nasal calcitonin daily reduced the risk of new vertebral fractures by 33% as compared with the risk in individuals taking a placebo. The effects of calcitonin treatment on the risks of hip and other nonvertebral fractures remain uncertain[20,21]. The fact that calcitonin substantially reduces the risk of vertebral fracture with only modest increases in bone mineral density suggests that yet to be elucidated calcitonin-mediated enhancement of bone quality may contribute to fracture risk reduction[22]. In addition to its antiresorptive action, patients with painful new vertebral compression fractures who were treated with calcitonin had, by two weeks, reduced pain, consumed fewer traditional analgesic medications, and regained mobility sooner, which may reduce bone loss secondary to prolonged bed rest[21,23]. Calcitonin-induced analgesia may be mediated by increases in plasma β-endorphins. This implicates involvement of the endogenous opiate system, while animal studies demonstrating calcitonin-binding sites in brain areas involved in pain perception suggest that calcitonin may directly modulate nociception in the central nervous system[23].

Hormone Replacement Therapy

Estrogen formulations were approved by the U.S. Food and Drug Administration for use in prevention of osteoporosis, but not for treatment of osteoporosis. Estrogen, both with and without progestin, has consistently been shown to not only maintain, but also increase, bone mineral density[24,25]. The Women's Health Initiative (WHI) clinical trials of hormone replacement therapy showed that long-term therapy with estrogen alone reduced the rate of hip, clinical vertebral, and total osteoporotic fractures by 30% to 39% as compared with the rates in patients taking a placebo[26]. Fracture reduction rates of a similar magnitude were found among participants randomized to receive long-term treatment with estrogen plus progestin. Hip and clinical vertebral fracture rates were reduced by 34%. Total osteoporotic fracture rates were reduced by 24% when compared with the rates in patients taking a placebo[25-27]. While the majority of studies and meta-analyses support the bone health benefits of hormone replacement therapy, some studies, most notably the Heart and Estrogen/Progestin Replacement Study (HERS), have not demonstrated evidence of fracture risk reduction in women similarly treated with hormone replacement therapy[25,28,29]. However, HERS had limited power to detect fracture risk reduction; it was able to detect only large reductions of at least 80%[25].

While improvements in bone mineral density and reductions in the rate of fracture occur, the associated risks of treatment preclude the use of estrogen formulations as primary agents in the treatment of osteoporosis. Women treated with estrogen alone have no change in the incidence of coronary heart disease; however, they have been found to have increased rates of stroke and deep vein thrombosis[27,29,30]. In addition, estrogen plus progestin increases the risk of breast cancer, dementia, and gallbladder disease[27,29,31]. The risks for cardiovascular disease, breast cancer, and dementia far exceed the benefits of estrogen and estrogen plus progestin therapy with respect to osteoporosis. This is true even for women at greatest risk for osteoporotic fracture[26]. This unfavorable safety profile restricts use of hormone replacement therapy for osteoporotic patients. However, women receiving short-term hormone replacement therapy for menopausal symptoms are likely to reap additional benefits with regard to bone health. Referral to a primary care physician or a gynecologist is the safest approach if hormone replacement therapy is planned.

Selective Estrogen Receptor Modulators

Selective estrogen receptor modulators are a class of compounds that bind estrogen receptors. They act as estrogen receptor agonists in some tissues and as estrogen receptor antagonists in others. Of the selective estrogen receptor modulators currently approved for clinical use, only raloxifene has been approved for the prevention and treatment of osteoporosis[32]. The effects of raloxifene on bone are known. Raloxifene has consistently proven to increase bone mineral density in the lumbar spine and femoral neck by 2% to 3% and to moderately decrease levels of bone-turnover markers by 30% to 40% (levels comparable with mean levels found in premenopausal women), suggesting an antiresorptive effect on bone tissue[33-35]. More importantly, raloxifene has also been shown to reduce the risk of vertebral fracture[34,35]. However, reductions in the overall risk of nonvertebral fractures did not reach significance[34-36]. The effect of raloxifene on fracture reduction is greater than what would be expected in light of the modest increases in bone mineral density. This suggests that raloxifene may also contribute to improvements in other components of bone quality[34].

As a result of raloxifene's selective estrogen receptor antagonist properties in breast tissue, women treated with raloxifene also benefit from a 62% reduction in the incidence of all types of breast cancer, with a 72% reduction in the risk of invasive breast cancer and an 84%

reduction in the risk of invasive estrogen receptor-positive breast cancer[28]. Additionally, raloxifene is not associated with an increased risk of endometrial cancer[28]. The risk of venous thromboembolic events is increased threefold, which is comparable with the elevated risk seen with hormone replacement therapy. Use of raloxifene also increases the incidence of vasomotor symptoms and may increase the risk of fatal stroke[34,37]. Clinicians must weigh the benefits of the reduced risks of vertebral fracture and invasive breast cancer against the increased risks of venous thromboembolism and fatal stroke when considering osteoporosis management.

Tamoxifen, a selective estrogen receptor modulator approved for use for the prevention and treatment of breast cancer, has been associated with reductions in the risk of vertebral fracture of a magnitude similar to those seen with raloxifene[38-40]. However, the greater risk of venous thromboembolism imparted by tamoxifen, as compared with that associated with raloxifene, and its association with an increased risk of endometrial cancer preclude its use in the treatment of postmenopausal osteoporosis[32,38-40].

Bisphosphonates

The bisphosphonates, a class of antiresorptive agents, are the current cornerstone of osteoporosis treatment and prevention. These nitrogen-containing compounds bind to the bone surface. There they exert their effect on the bone reabsorbing osteoclasts, decreasing osteoclastic activity and reducing cellular life span. Treatment with bisphosphonates reduces the rate of bone resorption, increases bone mineral density, and improves trabecular connectivity. These resultant effects serve to improve bone strength and reduce fracture risk. Both oral and intravenous forms of the treatment exist.

Currently, four bisphosphonates have been approved by the U.S. Food and Drug Administration for the treatment of postmenopausal osteoporosis: alendronate (Fosamax), risedronate (Actonel), ibandronate (Boniva), and zoledronic acid (Reclast). These drugs differ in their potency, dosing schedules, and mode of administration. All have been shown to possess antifracture efficacy. Placebo-controlled trials involving postmenopausal women treated with one of these agents have demonstrated reductions in the risk of vertebral fractures, ranging from 45% to 70%, relative to the risks for patients taking a placebo.

Alendronate, an oral bisphosphonate currently given in doses of 70 mg/wk for the treatment of osteoporosis, has been shown to increase bone mineral density in the spine, hip, and femur as well as to reduce the risk of fracture by an average of 50%[41]. Women with low bone mineral density and a history of vertebral fracture treated with daily alendronate for three years had a 47% reduction in the risk of vertebral fracture compared with the risk for those treated with a placebo[41]. Participants without a prior vertebral fracture had a reduction in the risk of a future vertebral fracture of 44%[42]. A meta-analysis of studies involving the effect of alendronate on the risk of hip fracture demonstrated an overall risk reduction of 45%[43]. Alendronate therapy has proven efficacious in the treatment of osteoporosis in men. Bone mineral density in the hip, spine, and total body is increased. The risk of vertebral fracture is decreased[44]. These antifracture effects of alendronate have been observed as early as one year after the initiation of therapy and have persisted ten years into the treatment period. Concerns regarding prolonged treatment are beginning to arise, as described below[45]. In a twelve-month head-to-head trial comparing two bisphosphonates, alendronate and risedronate (discussed below), patients in the alendronate group were found to have greater gains in bone mineral density and reductions in bone-turnover markers[46]. However, another study comparing the two agents failed to show any significant difference[47,48].

Risedronate, an oral bisphosphonate given in doses of 35 mg/wk, has also been shown to increase bone mineral density and reduce the risk of vertebral, nonvertebral, and hip fractures in osteoporotic women. In the placebo-controlled Vertebral Efficacy with Risedronate Therapy (VERT) study, daily treatment of postmenopausal women with osteoporosis (as defined by the previous occurrence of a vertebral insufficiency fracture) with 5 mg of risedronate decreased the cumulative incidence of new vertebral fractures by 41% and reduced the incidence of nonvertebral fractures by 39%[49]. A reduction of vertebral fracture risk of up to 61% has been found after only one year of treatment[50]. In another study, specifically assessing the effect of risedronate on the risk of hip fracture, that risk was found to be reduced by 30% compared with that associated with a placebo[51].

Ibandronate, one of the newer bisphosphonates made popular by its monthly (150-mg) oral dosing schedule and monthly intravenous (3-mg) formulation option, confers similar antiosteoporotic effects. As with alendronate and risedronate, patients treated with ibandronate have substantial increases in bone mineral density at all sites. In addition, they have decreases in vertebral fracture risk. However, ibandronate's anti-hip-fracture efficacy is still to be shown[52,53]. If compliance is an issue, ibandronate may be a useful option in certain patient groups. Patient compliance with weekly dosing regimens remains suboptimal, with rates ranging from 58% to 76% at one year[54]. If patient compliance is increased, treatment with ibandronate may improve therapeutic outcome.

The side effects of the oral bisphosphonates are similar and are due to their inherent toxicity to epithelial cells lining the gastrointestinal tract. The result may be gastrointestinal irritation and ulceration. Therefore, it is recommended that patients take the medication first thing in the morning on an empty stomach along with 8 oz (0.2 L) of water and then remain upright for thirty minutes. Osteonecrosis of the jaw, defined as exposed bone in the maxillofacial region that fails to heal within eight weeks after identification by a health-care provider, is a troubling potential complication of bisphosphonate use[55]. It has been reported that the patients who are at greatest risk are those with multiple myeloma or metastatic carcinoma of the skeleton who are being treated with relatively high doses of the intravenous bisphosphonates zoledronic acid and pamidronate. This patient population has included 94% of the reported cases[56]. In a recent report, the American Society for Bone and Mineral Research estimated the risk of osteonecrosis of the jaw in patients taking oral bisphosphonates for the treatment of osteoporosis to be between one in 10,000 and less than one in 100,000 patient-treatment years[55]. This is lower than the estimated incidence of one to ten cases per 100 patients with cancer receiving intravenous treatment[55]. Sixty percent of the cases of osteonecrosis that do occur are preceded by a surgical dental procedure. Theories regarding potential mechanisms for the development of osteonecrosis of the jaw include oversuppression of bone turnover and bisphosphonate toxicity of the soft tissues overlying the jaw[56-58]. Limited data exist regarding prevention and management of the condition. It is recommended that patients in need of a dental procedure establish meticulous oral hygiene and consider completing dental work prior to starting bisphosphonate treatment[58,59]. No evidence supports the discontinuation of established bisphosphonate therapy prior to a dental procedure[60].

Zoledronic acid is available in an intravenous formulation given once yearly as an infusion. It has demonstrated efficacy in increasing bone mineral density and reducing fracture risk[61,62]. In the multinational, multicenter, placebo-controlled HORIZON (Health Outcomes and Reduced Incidence with Zoledronic Acid Once Yearly) Pivotal Fracture Trial, women who received an infusion of 5 mg of zoledronic acid once yearly had a 70% reduction in the risk of new spine fractures ($p < 0.0001$) and a 41% reduction in the risk of hip fractures ($p = 0.0032$)

over three years compared with the risks for women taking a placebo[62]. In a group of osteoporotic patients who had received an infusion of zoledronic acid within ninety days after a hip fracture repair, the risk of any fracture decreased by 35% and mortality from any cause decreased by 28% compared with the rates for patients given a placebo[63]. Patients being treated with weekly oral alendronate can switch to zoledronic acid and maintain the beneficial bone effects for twelve months after a single infusion. The most common side effects associated with use of zoledronic acid include influenza-like post-infusion symptoms of fever, muscle pain, headache, and bone pain. The majority of symptoms resolve within three days. Osteonecrosis of the jaw was not seen in any of the trials investigating the use of zoledronic acid in postmenopausal women with osteoporosis[61-63]. Atrial fibrillation has also been seen. This association is yet to be defined[62,63]. Given the convenience of a yearly dosing schedule, zoledronic acid may be a suitable option for osteoporotic patients in need of bisphosphonate treatment for whom gastrointestinal toxicity is a problem.

Once a decision has been made to begin treatment with bisphosphonates, optimization of the mineral environment and monitoring of the bone turnover state ensure that the best possible result is achieved. The importance of adequate vitamin-D and calcium status is highlighted by case reports of bisphosphonate-induced hypocalcemia in patients with unrecognized vitamin-D deficiency. Animal studies have also demonstrated a blunting of the bisphosphonate response in the setting of vitamin-D deficiency[64,65]. All patients should receive 1500 mg of calcium citrate and 800 IU of vitamin D3. Those found to have deficiencies (a serum calcium level of <9.5 mg/dL [<2.4 mmol/L] and/or a serum 25-hydroxyvitamin-D level of <32 ng/mL [<79.9 nmol/L]) may require greater doses for a short time until they are considered calcium and/or vitamin-D-replete.

Measures of bone mineral density may clinically diagnose osteoporosis but are of limited value for assessing a patient's response to bisphosphonate treatment[66]. Fractures are a key efficacy end point in bisphosphonate trials. Studies have demonstrated an inconsistent relationship between changes in bone density and fracture risk[67]. Data relating changes in bone turnover to subsequent fracture outcomes suggest that high turnover itself may be an independent risk factor for fracture[68]. Thus, markers of bone turnover may be useful for assessing a patient's response to treatment. The markers most commonly used in clinical practice include the markers of bone formation, bone-specific alkaline phosphatase and osteocalcin; and the markers of bone resorption, urine N-telopeptide of collagen cross links (NTx) and serum C-telopeptide of collagen cross links. In the Fracture Intervention Trial (FIT), greater reductions in bone turnover with alendronate therapy were associated with fewer hip, non-spine, and vertebral fractures[68]. Despite these results, controversy remains regarding the use of bone turnover markers in monitoring response to treatment. For patients taking bisphosphonates, the ideal therapeutic range of urine levels of NTx is 20 to 40 nmol BCE (bone collagen equivalents)/mmol of creatinine.

Long-term use of bisphosphonates may suppress bone turnover to such an extent that a paradoxical decrease in bone strength and resilience develops; this is referred to as adynamic bone. In this state of oversuppression, microfractures generated through the wear and tear of normal daily life begin to accumulate and coalesce, leading to spontaneous nonspinal fractures[69]. Accumulation of microdamage is associated with a reduction in bone toughness, defined in animal studies as the ability of the bone to sustain deformation before breaking[70]. In another study, this decrease in toughness was found to be offset by an increase in bone volume and mineralization, the combination of which resulted in no significant impairment in bone mechanical properties[71]. Odvina et al. reported on nine women treated with high-dose

alendronate who presented with a spontaneous fracture of a long bone[72]. Six of these women also displayed evidence of delayed or absent fracture-healing during alendronate therapy. Histomorphometric analysis of bone biopsy specimens from these patients revealed marked suppression of bone turnover, demonstrated by a reduced or absent osteoblastic surface, a diminished osteoclastic surface, and minimal matrix synthesis. For these patients, changes in therapy such as a rest period from bisphosphonates or the use of an anabolic agent such as teriparatide (as discussed below) should be considered. In a study comparing women who stopped taking alendronate after an average of five years of use with those who continued to use the drug, those who stopped did not have accelerated bone loss or a marked increase in bone turnover[73]. These results indicate a persistence of alendronate's effect on bone after therapy is stopped[73]. Currently, it is unknown whether long-term treatment with bisphosphonates beyond five years is indicated. More studies are needed to investigate the potential positive and negative impact that prolonged bone suppression can have on fracture risk.

Anabolic Agents

Parathyroid Hormone

Approved by the U.S. Food and Drug Administration in 2002, teriparatide (parathyroid hormone [PTH1-34]) is the only anabolic agent available for the treatment of postmenopausal osteoporosis. Self-administered subcutaneously with use of a pen-like device, daily teriparatide injection is the most effective therapy for restoring bone quality[74,75]. The effects of parathyroid hormone are mediated by enhancement of bone turnover. When administered intermittently, the anabolic effects predominate, increasing bone mass up to 13% over two years of therapy. This increase is greater than that achieved with bisphosphonate therapy[76]. The antifracture efficacy of teriparatide is similar to that seen with bisphosphonates. After treatment of postmenopausal women with osteoporosis (as defined by bone mineral density) with daily 20-µg injections of parathyroid hormone, the risk of vertebral fracture and nonvertebral fracture was reduced by 65% and 53%, respectively[76]. The antifracture efficacy of parathyroid hormone may be related to more than just increases in bone mineral density. Microcomputer tomographic analysis has demonstrated an increase in trabecular number as well as trabecular thickness[77].

Although it has been proven to be efficacious across the spectrum of osteoporosis disease severity, the use of parathyroid hormone has been limited[78], most likely as a result of the combination of high cost, relative inconvenience, and potential adverse reactions associated with use of the drug. Evidence of osteosarcoma in rodents exposed to prolonged high doses of teriparatide led the U.S. Food and Drug Administration to prohibit its use in patients at high risk for skeletal cancer[79,80]. The use of teriparatide is contraindicated in patients with active Paget disease of bone, metastatic cancer in the skeleton, or a history of skeletal irradiation, and in children with open epiphyses. In an estimated more than 300,000 exposures to teriparatide for the treatment of postmenopausal osteoporosis, a single case of osteosarcoma was recently reported[81], and the existence of a causal relationship between teriparatide use and osteosarcoma in humans remains uncertain. Additional adverse reactions associated with teriparatide include nausea, swelling, pain, weakness, erythema around the injection site, and elevation in plasma calcium levels. Plasma calcium may be adjusted, and vitamin-D supplementation may be needed[82,83]. Hypercalcemia may be monitored by measuring serum calcium levels at one month following the initiation of treatment[84].

Antiresorptive therapies have long been, and continue to be, the mainstay of osteoporosis treatment. Patients who have been previously treated with antiresorptive therapy constitute a

large group in whom parathyroid hormone treatment may be indicated. Data suggest that previous treatment with potent inhibitors of bone turnover, such as alendronate, appears to diminish the initial response to teriparatide[85]. It also appears that the degree of the initial teriparatide effect depends on the potency of the previously used antiresorptive agent, and this effect has not been demonstrated in association with less potent agents such as raloxifene[86]. Many practitioners advocate a brief (six-month) rest period between the discontinuation of the antiresorptive agent and the start of teriparatide treatment.

Combination Therapy

Despite an initial attractiveness of the combined use of anabolic and anticatabolic therapy, a synergistic effect between teriparatide and the bisphosphonates has not been seen. On the contrary, concurrent use of a bisphosphonate has been shown to blunt the bone-building potential of parathyroid hormone[87,88]. However, in a recent trial by Deal et al., concurrent administration of raloxifene was found to enhance the bone-forming effects of teriparatide[89]. Postmenopausal women who received a combination of teriparatide and raloxifene over a period of six months had a greater increase in bone mineral density in the hip compared with groups that received raloxifene or teriparatide alone. A similar synergistic effect has been seen following coadministration of teriparatide and hormone replacement therapy[90]. Additional studies that include the assessment of fracture outcome as well are needed.

The bisphosphonates, while not recommended during teriparatide treatment, can play a valuable role after completion of teriparatide therapy. Soon after discontinuation of teriparatide treatment, gains in bone mineral density begin to regress rapidly. Declines in bone mineral density begin as early as eighteen months after the last dose of teriparatide is given[91]. The immediate use of bisphosphonates or other antiresorptive therapy has been shown to optimize valuable gains in bone mineral density. The use of bisphosphonates not only prevents a decline in bone mineral density but also enhances additional densitometric gains[92,93]. Subsequent treatment with bisphosphonates facilitates the mineralization of osteoid laid down during the previous period of increased metabolic activity. In an effort to "lock in" and "protect" the valuable gains in bone mineral density achieved during the two years of teriparatide treatment, many practitioners advocate starting or restarting antiresorptive therapy on completion of the anabolic therapy.

Future Directions

The treatment of osteoporosis is currently associated with numerous problems ranging from adverse drug reactions to suboptimal patient compliance[94-98]. Better drugs with more specific targets will reduce the adverse effects and improve the outcome of therapy. The understanding of cellular mechanisms regulating bone formation and remodeling has improved substantially in the last few years. In the arena of antiresorptive agents, denosumab,

a human monoclonal antibody against receptor activator of nuclear factor- B ligand (RANKL), has been shown in preclinical trials to increase bone mineral density and decrease bone resorption in postmenopausal women with osteoporosis. Used commonly in patients with multiple myeloma and metastatic disease of the skeleton, denosumab exerts its action through inhibition of RANKL, a key mediator in osteoclast activation[99,100]. Denosumab is now awaiting approval for entry into the market. Cathepsin-K inhibitors are another group of novel antiresorptive agents. It is hoped that these drugs, which were designed to reduce the activity of cathepsin K (a powerful osteoclast protease), can limit the enzymatic degradation of bone matrix proteins[101]. The efficacy of cathepsin-K inhibitors in the treatment of postmenopausal osteoporosis is still under investigation in clinical trials.

New anabolic agents are currently on the treatment horizon. Strontium ranelate, used routinely in Europe but unavailable in the United States, is considered to be the only agent to have a dual mechanism of action, acting as both an antiresorptive and an anabolic agent. Treatment of postmenopausal osteoporotic women with strontium ranelate has been shown to decrease fracture risk and increase bone mineral density. While the long-term effects remain unknown, strontium may prove to be an attractive option for patients unwilling or unable to use parathyroid hormone[102,103]. The development of alternative forms of parathyroid hormone, including noninjectable forms (oral, nasal, sublingual, and transdermal modes), is also under way. These new analogs of parathyroid hormone appear to possess longer half-lives, allowing sustained exposure in the setting of less frequent dosing[94,96-98,104-106].

Treatment of osteoprosi in orthopaedic Situation

Calcium and Vitamin-D Supplementation for Patients with a Fracture

Calcium and vitamin-D supplementation is a baseline critical component of any fracture treatment therapy. The increased bone turnover stimulated by fracture repair and remodeling leads to an increased metabolism and demand for calcium and vitamin D. The estimated daily intake required for fracture-healing is 1500 to 2500 mg of calcium and 1000 to 2000 IU of vitamin D.

Use of Bisphosphonates for Patients with a Fracture

Healing of both stabilized and unstabilized fractures involves stages of osteoclastic activity[107]. The limited data currently available indicate that the use of bisphosphonates does not impair, and may actually enhance, fracture-healing[108]. Studies have shown that, while development and remodeling of the fracture callus is delayed in the setting of bisphosphonate use, the overall mechanical strength of the callus is either unchanged or increased[109,110]. Concern regarding a bisphosphonate-driven increase in the rate of nonunion also continues to be unsupported in the literature[108-110]. Timing may play a role in the effect of bisphosphonates on fracture-healing. The administration of zoledronic acid to rats two weeks after creation of a fracture resulted in a greater increase in the mechanical strength of callus compared with what was seen with administration prior to the fracture[111]. Given the development of the primary callus during the first two weeks of fracture-healing, some studies support initiation or continuation of bisphosphonate treatment after this time[108]. Animal fracture data combined with the known efficacy of bisphosphonates in preventing future fractures are compelling enough to support initiation of treatment in a "timely fashion" for all patients with an osteoporotic fragility fracture[112].

Since bisphosphonates inhibit osteoclastic resorption and osteoclastic activity is involved in fracture repair, a patient who is already being treated with bisphosphonates may have an initial delay in the early stages of the fracture repair process. Some animal studies have shown interference with fracture repair and the mechanical strength of the fracture site dependent on the chemical structure, dosage potency, and duration of the treatment with the bisphosphonate. Additional studies of humans are needed to determine the ultimate effect on union and on the restoration of mechanical strength and anatomic architecture after fracture-healing. Physicians may choose to stop the bisphosphonate treatment for two weeks—i.e., until the initial fracture-repair period has passed.

Use of Teriparatide for Patients with a Fracture

Recent animal studies have suggested that teriparatide may also play a valuable role in the treatment of fractures. An acceleration of fracture-healing has been demonstrated in animals treated with intermittent doses of parathyroid hormone[113-116]. Stimulation of proliferation and

differentiation of chondrocytes and osteoprogenitor cells, leading to an increase in the production of bone matrix proteins, is believed to be the mechanism[113,117]. The fracture callus in parathyroid-hormone-treated animals forms more rapidly, remodels more quickly, and possesses superior biomechanical properties when compared with that of controls[114,115]. Parathyroid hormone (PTH1-34) may prove to be an attractive agent to enhance healing and limit the risk of nonunion of poorly healing or high-risk fractures when human trials on fracture-healing have been performed.

Use of Bisphosphonates for Patients Who Have Undergone Arthroplasty

Aseptic loosening and osteolysis are the most common causes of failure of total joint arthroplasty. Osteolysis is caused by wear-debris-mediated stimulation of the osteoclast. This leads to subsequent bone resorption. Drugs used to treat osteoporosis inhibit the osteoclast and also increase endosteal bone formation[118]. They have therefore been used experimentally[119-126] as possible therapies to improve the life of prostheses; however, additional animal and human studies are needed.

Most studies have shown that patients being treated with bisphosphonates maintain more periprosthetic bone mineral density and have less periprosthetic bone loss[127-131]. Bisphosphonates have a larger effect on bone loss following arthroplasties with cement, and especially knee arthroplasties with cement[132]. With anabolic bone therapy, uncemented prostheses may have the potential for better ingrowth and survival. However, future human studies may demonstrate better prosthetic survival in patients using drugs for reduced bone mass. Nevertheless, patients undergoing total joint replacement should be evaluated and treated for decreased bone mass if they have a number of risk factors for osteoporosis

V.MCQ

ACL and Bone bruise. [Am. J. Sports Med. 2008; 36; 671]
1. No consistent association was observed between the presence of an initial articular cartilage lesion with a lesion on follow-up magnetic resonance images.
2. All bone bruises identified in our study with magnetic resonance imaging at the time of IVinitial injury had resolved at 12-year follow-up.
3.The presence of a bone bruise at the time of initial injury did not significantly alter the patient-oriented outcome by International Knee Documentation Committee after anterior cruciate ligament reconstruction.
4.The natural history of bone bruises and their ability to resolve remains controversial, in part because of the lack of prospective longitudinal cohort studies with 80% follow-up.
5. Bretlau et al3 found that only 12% of bone bruises identified on MRI in patients with acute knee injuries persisted at 12 months.

2. **Distal Biceps Repair**.[OCNA 39 (2008) 237]
1. Newer treatment methods [single anterior incision] have the potential to decrease some of the complications noted with more traditional surgical techniques.
2. 1.2 per 100,000 patients; The average age was 47 years. .Majority are males
3. Most complications reported: nerve injury to either the posterior interosseous nerve (PIN) or the lateral antebrachial cutaneous nerve (LAC).
4. Biomechanical studies: Endobutton, suture anchor,and interference screw), and the Endobutton had a significantly greater load to failure than any other repair method. Failure loads ranged from 232 N (suture anchor) to 440 N (Endobutton).

3.Painful TKR
1. Rest pain Vs start off pain
2.Radiological assessment
3. Check for extension and flexion stability [I.I}
4.Blood, bone scan [Bpne scan in THR may return by 12 months; in TKR it may not return indefinitely [III phase]
5. Mismatch Indium scan and Sulphur colloid bone scan
6. CT assessment
7.IL 6 (>10 pg/mL). This is elevated in infection and not in loosening
8. Joint aspiration: Gram stain is not useful; Cell count >2500 and >60% PMNC

4 What's new in TKA.[J Bone Joint Surg Am. 2007;89:2828]
1.UKO: The shifting demographics of patients with Unicompartmental knee arthritis, including younger, more active patients
2. Minimally invasive TKA as reported by many authors and the enthusiasm for the technique as reported in publications from 2005 until recently, not all studies have demonstrated clear advantages of minimally invasive techniques
3. An acquired patella Baja occurred in 37% of patients with patellar eversion during surgery, compared with 12% of those without patella eversion during surgery. The presence of patella baja resulted in reduced flexion and increased pain.
4.Many centers are harnessing the power of computers in an attempt to increase the precision of total knee arthroplasty component implantation and soft-tissue tensioning.
There were no significant differences between the conventional and computer-assisted groups with regard to limb alignment or component positioning.

5. The use of LMWH [10 day] for THA & TKA The prevalence of symptomatic DVT was 3.8% and the prevalence of non-fatal pulmonary emboli was 1.3%. The rates of surgical site complications necessitating readmission, irrigation and debridement of a hematoma and the wound, or prolonged hospitalization for wound drainage were 4.7%, 3.4%, and 5.1%, respectively. Wound drainage occurred for four to seven days after 9.3% of the procedures.
6.Westrich performed a randomized study of 275 patients undergoing TKA with spinal epidural anesthesia, pneumatic compression devices, and administration of either enoxaparin or aspirin postoperatively for four weeks. The overall prevalence of deep-vein thrombosis was not significantly different between the drug treatment groups.
7. Postoperative stiffness (de- fined as flexion of <90_) in an analysis of 1216 TKA.. At one year, they found a 3.7% rate of stiffness. On the average, for every increase of 2 mm in patellar thickness, a decrease of 3° in maximum passive flexion resulted.
8.Barrack: An unexpected positive bacterial culture at the time of revision TKA. Of 692 patients who had a revision for the treatment of aseptic complications, 5.3% had an unexpected positive intraoperative culture. They were managed with six weeks of intravenous antibiotics.

5.Entrapment Ulnar Neuropathy [JOAAS 2007;15:672]
1. Ulnar nerve entrapment is the second most common nerve entrapment syndrome of the upper extremity.
2. The five sites of potential ulnar nerve entrapment around the elbow: arcade of Struthers, medial intermuscular septum, medial epicondyle, cubital tunnel, and deep flexor pronator aponeurosis
3. A simple decompression is obtained in the patient with mild weakness, recent onset of symptoms, and mild abnormality of sensory action potentials.
4. For moderate to severe compression, anterior submuscular transposition offered the best results with the lowest recurrence rate
5. A comparative study: simple decompression with anterior subcutaneous transposition in 66 patients with cubital tunnel syndrome. No significant difference in pain, motor and sensory deficits, or nerve conduction velocity studies was found between the two groups

6. 11. Charcot Marie Tooth Syndrome. Beals Foot Ankle Clin N Am 13 (2008) 259–274
1. CMT is typically a progressive condition
2.Muscle forces demonstrate very strong tibialis posterior and peroneus longus. Conversely, the peroneus brevis and tibialis anterior muscle are generally weak.
3. To assess the degree of flexibility of the hindfoot in a patient who has a cavus deformity. The ‘‘Coleman block test’’ requires a patient to stand on a block of wood with the heel and the lateral forefoot supported by the block, which allows the plantar-flexed first ray to drop.
4. Characteristically, muscle involvement in CMT progresses from distal to proximal. However, the described patterns of muscle function have limitations. Relative sparing of the extensor hallucis longus can be observed
5..Despite the relative weakness of the tibialis anterior, it can continue to be a powerful deforming force because of its static resistance to eversion in a patient who has a varus hindfoot.
6. A common soft tissue technique in this population is a plantar fascial release.
16. Key principles appear to be to understand the specific features of a given patient in detail when creating a customized surgical plan that uses bony reconstruction procedures to create a mechanically balanced foot, and then to apply soft tissue releases and tendon transfers as

necessary to maintain functional control of the foot and ankle, while appreciating the likelihood of progression of the neuromotor imbalance..

7. Radiological angles in CMT

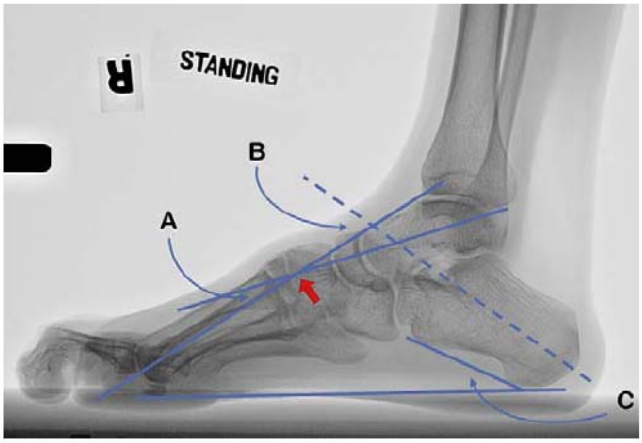


Fig. 4. Radiographic angles as measured on a standing lateral radiograph of an adolescent boy with Charcot-Marie-tooth. (A) Meary's angle, formed by the intersection of lines drawn through the longitudinal axes of the first metatarsal and talus (normal is between 0° and 5°, cavus greater than 5°). (B) Hibb's angle, formed by the intersection of lines drawn through the longitudinal axes of the first metatarsal and the calcaneus (normal less than 45°, cavus greater than 45° and typically 90°). (C) Calcaneal pitch angle, formed by the intersection of a line parallel to the floor and another line drawn along the plantar surface of the calcaneus from the tuber to the anterior process (calcaneocavus greater than 30°, forefoot cavus less than 30°).

8.Frozen shoulder [J Shoulder Elbow Surg 2008:231]

1. The mean age at symptom onset was 53.4 years; with women affected more commonly than men (1.6:1.0).
2.20% of patients reported bilateral symptoms, but there were no recurrent cases.
3.In the long term, 59% of patients had normal or near normal shoulders and 41% reported some ongoing symptoms.
4.The majority of these persistent symptoms were mild (94%), with pain being the most common complaint. Only 6% had severe symptoms with pain and functional loss.
5.Primary frozen shoulder is a common:prevalence is reported as 2-5%
6.Arm dominance has been suggested as affecting the prognosis, but no evidence of a ifference was found

9.CPDD Vs HADD

Calcium pyrophosphate [CPPD]	Calcium hydroxyapatite crystal [HADD]
The most common crystalline arthropathy,	Less common
Usually polyarticular . Sites: knee >hip,>shoulder , elbow	Usually monoarticular and most commonly presents: Shoulder> elbow. MPJ, Wrist
Tendon: The sites more frequently involved include: the supraspinatus , triceps, quadriceps, Achilles	The most common site of deposition is in the flexor carpi ulnaris tendon near its attachment to the pisiform
Acute/subacute/chronic and usually self limiting	Acute/subacute/chronic and usually self limiting
X ray: Prominent linear or punctuate deposits and parallel the subjacent subchondral bone	X ray: Calcifications in HADD are more homogeneous or cloudlike,

10. Anteromedialisation for PFA [.J K nee Surg. 2008;21:101-105]
1. Anteromedialization of the tibial tubercle is a definitive treatment option for isolated patellofemoral arthritis in active older patients.
2. Isolated patellofemoral arthritis is present in 5% to 8% of individuals with symptomatic osteoarthritis of the knee.
3. Anteromedial tibial tubercle transfer produces good long-term results in patients with patellofemoral arthritis who have healthy cartilage onto which the patellar tracking may be transferred.
4. The tibial **tuberosity** is **transfer**red both medially and anteriorly, with the relative proportion of each **transfer** being determined by the slope of the cut. A 45° cut leads to equal medial and vertical displacements. Fixed with 2 fully threaded 4.5 cortical screws.
5.If the tibial **tuberosity** were displaced 11 mm along a 45° degree plane, which is a substantial displacement, this would lead to 8 mm of medial displacement and 8 mm of anterior displacement.
6. Ateshian and Hung[7] calculated that this would result in only a 10% reduction in stress. This would explain why the procedure is not well suited for the treatment of global arthritis of the patellofemoral joint
7.Fulkerson: Based on radiographs alone, previous reports have found malalignment present in 40% to 86% of patients with isolated patellofemoral arthritis. We found this number increases with a careful clinical examination and history.

VI.Case Study: A 28-year-old man with a history of knee mass diagnosed as osteochondroma

presented with acute pain and enlarging swelling overlying this region.

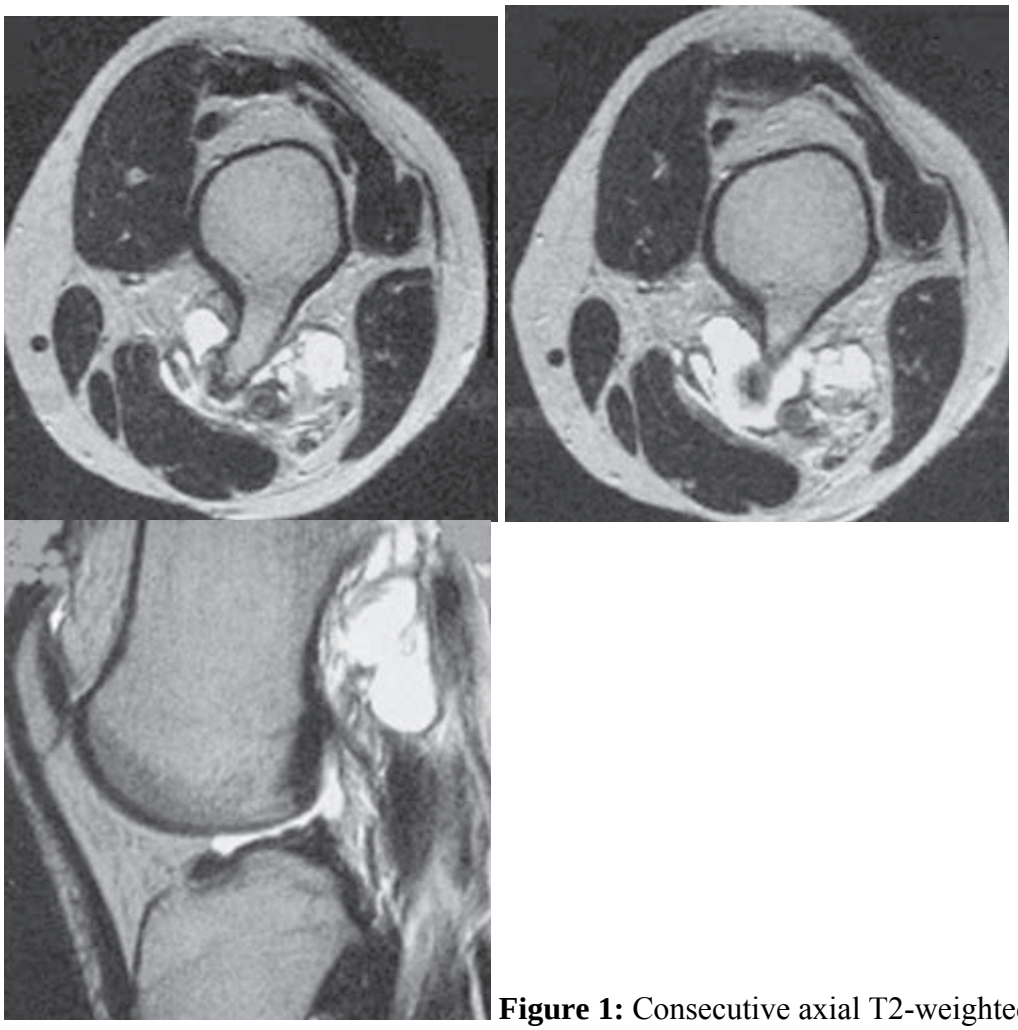


Figure 1: Consecutive axial T2-weighted MRIs of the left knee (A-D).

Figure 2: Sagittal T2-weighted MRI

The patient presented with acute symptoms of pain and enlarging swelling, with a known history of solitary osteochondroma that had previously been quiescent. Malignant transformation of the osteochondroma was suspected.

Magnetic resonance imaging (MRI) demonstrated the known femoral osteochondroma with an overlying fluid filled sac. Rapid growth following a period of quiescence is a possible indicator of malignant transformation.

In this case, bursal formation was demonstrated on MRI, a rare complication of osteochondroma that mimics chondrosarcoma at clinical presentation.

DISCUSSION

Osteochondroma represents approximately 35% of all benign bone tumors.1 Although it is often referred to as a tumor, it is felt to be a developmental lesion rather than a true neoplasm.1 The lesion contains a hyaline cartilage cap overlying a bony exostosis, which consists of cortical and medullary bone that are in direct continuity with the underlying native bone (Figure1). This radiographic appearance is diagnostic of osteochondroma.

The majority of osteochondromas are solitary lesions, with approximately 65% located around the knee.1 The solitary form of osteochondroma is suspected to occur by developmental separation of a section of growth plate cartilage, with continual growth of this fragment.

Other causes, including trauma and surgically induced separation, as well as osteochondroma arising in irradiated bone, have been described.2 Multiple osteochondromas, most often the sessile rather than pedunculated type,may occur in patients with hereditary multiple exostoses, an autosomal dominant disorder that linkage analysis has traced to the exostoses genes.3 In the Western world, an estimated 1:50,000 to 1:100,000 prevalence of this disorder usually manifests itself in the first decade of life..

In individuals with hereditary multiple exostoses, the size, number, and location of osteochondromas may vary, even among members of the same family. These patients frequently report pain and cosmetic concerns as common deformities associated with hereditary multiple exostoses include short stature,discrepancies in limb length,and valgus deformity of both knees and ankles.

Although osteochondromas are benign, several complications may arise. These include malignant degeneration,bursitis, vascular complications,nerve palsies and fracture.

Malignant degeneration of the cartilaginous portion of the osteochondroma into a sarcoma, which has previously been reported to be as high as 25%, is believed to occur in 1%-2% of solitary osteochondromas, and in 3%-5% of patients with hereditary multiple exostoses.2,3 Clinical presentation often includes symptoms of pain, other neurologic symptoms such as numbness, local swelling,and continued growth of the osteochondroma after skeletal maturity. Radiographic clues that indicate malignancy include irregularity of the osteochondroma surface, regions within the osteochondroma containing lucency or heterogeneous mineralization, and a surrounding soft-tissue mass that may contain punctuate calcification within it. Another clue to malignancy is a thick cartilage cap as seen with computed tomography, MRI, or ultrasound, which has variously been reported as mean thickness of 1.5 and 2.0 cm.2,3

The majority of sarcomas arising in osteochondromas tend to be well differentiated,low grade chondrosarcomas,with a better prognosis as compared to primary chondrosarcoma.

Other, rarer sarcomas with osteochondroma have also been described, and include the development of secondary osteosarcoma, although it has been debated as to whether it arises in the base of the osteochondroma stalk or the cartilage cap. The most feared complication of osteochondroma is malignant degeneration, with formation of soft-tissue mass overlying the lesion. A complication that may mimic this is prominent bursal formation overlying the lesion that clinically would present as an enlarging mass indistinguishable from the clinical presentation of sarcomatous transformation.

The true incidence of bursitis associated with osteochondroma is unknown,as only a few case reports describing this complication have appeared in the literature. Bursa formation most often occurs in osteochondromas with overlying moving tissues that cause friction,and most common sites include the scapula, hip, and shoulder Both ultrasound and MRI can demonstrate the fluid-filled sac overlying the osteochondroma, indicating its benign nature.6 Other complications that may arise as a result of bursal formation include infection or infl ammation of the synovium that would show enhancement on contrast enhanced MRI.

The MRI characteristics of bursal sac formation include low T1-signal intensity and high T2-signal intensity. Filling defects in the overlying fl uid-filled cavity may represent granulomatous tissue and cartilaginous debris that should not be mistaken for the heterogeneity that may be seen in a sarcoma.

TREATMENT

Asymptomatic: These require no further workup or treatment, unless complications arise. In bursitis associated with an osteochondroma, this may initially be treated conservatively, with surgical resection contemplated for such complications as entrapment of vessels, nerves and tendons, and persistent pain.

In the case of vascular compromise such as pseudoaneurysm formation, surgical management also is indicated. Simple surgical resection of the osteochondroma may provide relief of claudication. Further management for pseudoaneurysm formation includes thrombectomy, evacuation, and direct suture repair, either primary repair or with the aid of patch grafts or interposition grafts.

Fracture treatment of the osteochondroma may include both nonoperative therapy, mainly involving restriction of activity until symptoms resolve, or surgical excision. A shorter recovery period, with quicker resumption of physical activity, has been observed with surgical resection, and is advocated in physically active patients. The treatment of chondrosarcoma involves wide surgical resection, which owing to the low grade nature of the secondary chondrosarcoma,is usually curative. This tumor is not sensitive to chemotherapy and radiation. Distant metastases are uncommon due to the low grade nature, and instead, local recurrence is more closely related to mortality than distant metastasis.

SUMMARY

Osteochondroma is a common benign entity. However, symptomatic complications may arise that raise the question of malignant degeneration. Bursa formation about an osteochondroma may clinically and symptomatically mimic malignant degeneration. Magnetic resonance imaging usually permits unequivocal diagnosis distinguishing between secondary bursitis and malignant degeneration. Secondary bursitis can most often be managed conservatively. Magnetic resonance imaging also can differentiate other less common benign complications such as pseudoaneurysm formation caused by the exostosis and fracture at the base of theexostosis.