

MANAGEMENT OF OSTEONECROSIS OF THE FEMORAL HEAD

Kevin D. Plancher, MD, MS, and Afshin Razi, BA

Osteonecrosis or avascular necrosis of bone was first described by Munro in 1738. In 1794 Russell published a text on necrosis of bone, and Cruveillier recorded a gross deformity of the femoral head as a late complication from trauma.^{24, 38, 43, 68, 69} In 1930, Phemister increased our knowledge concerning the etiology, pathogenesis, and treatment of this condition.^{59, 60, 61}

Osteonecrosis of the femoral head is a pathologic process resulting from the death of living elements of bone. It is not a specific disease but rather the end result of various conditions, ultimately, with impairment of blood supply to the femoral head. Mechanical and biological factors contribute to osteonecrosis.

Osteonecrosis may affect young individuals, in whom the incidence of bilateral disease is surprisingly high. Osteonecrosis of the hip may progress through various stages and terminate with degenerative arthritis of the hip joint. This process may take 3 to 5 years from its onset or may never progress past an early stage.

Treatment options are numerous. Certain procedures that might have been performed years ago are no longer recommended. The consensus among clinicians is that treatment depends on the stage of the disorder at the time of diagnosis. The literature has been limited by too short of a follow-up or too small of a sample size. This article discusses the etiology, pathogenesis, radiologic staging, and treat-

ment regimens for osteonecrosis of the femoral head.

ANATOMY OF THE FEMORAL HEAD

The circulation of the femoral head has been well described.^{16, 19, 79, 94, 98} The medial femoral circumflex artery gives rise to the superior and inferior retinacular vessels (Fig. 1). The superior retinacular vessel branches into the superior metaphyseal and lateral epiphyseal, whereas the inferior retinacular branch becomes the inferior metaphyseal vessels. The obturator artery, through its acetabular branch, supplies the artery to the ligamentum teres, which terminates as the medial epiphyseal artery. Vessels that travel from the femoral shaft and trochanteric region to the femoral neck assume a subcortical position and terminate within the proximal portion of the neck. The lateral and medial epiphyseal arteries supply the lateral two thirds and medial one third of the femoral head, respectively, with the inferior metaphyseal artery supplying the femoral neck.

ETIOLOGY OF OSTEONECROSIS

Trauma

Many causes contribute to osteonecrosis of the femoral head (Table 1). Except for trauma,

From Albert Einstein College of Medicine, New York (KDP, AR); Montefiore Medical Center, New York; Monroe College, New York; Lehman College, New York; Blythedale Children Hospital, Valhalla, New York; Steadman Hawkins Clinic and Foundation, Vail, Colorado (KDP)

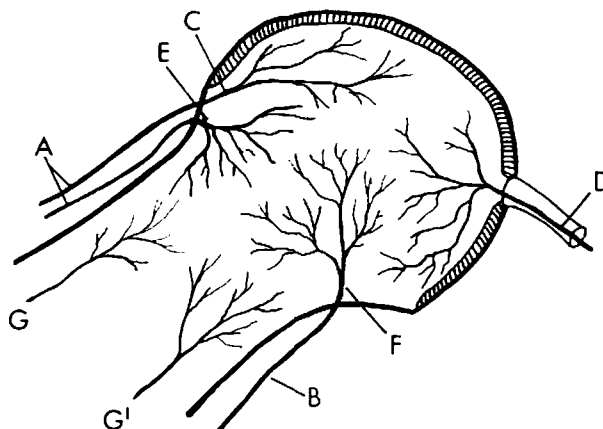


Figure 1. A = Blood supply of the femoral head; B = superior retinacular vessels; C = lateral epiphyseal artery; D = medial epiphyseal artery; E = superior metaphyseal artery; F = inferior metaphyseal artery; G and G' = intramaxillary vessels from the neck. (Courtesy of Kevin Plancher, MD, New York, NY.)

Table 1. ETIOLOGY OF OSTEONECROSIS OF THE FEMORAL HEAD

Traumatic	
	Intracapsular fracture of the neck
	Dislocation of the hip
	Crushing injury to the hip without fracture or dislocation
	iatrogenic
	Reconstructive hip surgery
	Cup arthroplasty
	Surface replacement arthroplasty
	Cuneiform osteotomy of the femoral neck
	Synovectomy
	Hip manipulation
	Treatment of congenital hip dislocation
	Treatment of slipped epiphysis
Nontraumatic	
	Sickle cell disease and its variants
	Legg-Calvé-Perthes disease
	Systemic steroid intake
	Renal transplant
	Lupus erythematosus and other collagen-vascular diseases
	Excessive alcohol intake
	Exposure to high altitude
	Caisson disease
	Decompression sickness
	Hemoglobinopathies
	Coagulopathies
	Pregnancy
	Gaucher's disease
	Hyperlipidemia
	Gout
	Pancreatitis
	Fibry's disease
	Burns
	Radiation
	Slipped capital femoral epiphysis
	Idiopathic

the biologic processes that initiate the pathologic phenomenon are unknown. Most cases of osteonecrosis of the femoral head are caused by trauma. Transcervical fractures, complete hip dislocations, and compression fractures of the femoral head are common culprits.^{11, 15, 18, 31, 37} Significant displacement of the femoral neck can interrupt the blood supply to the femoral head, superior retinacular, and lateral epiphyseal vessels, leaving the medial epiphyseal and, rarely, the inferior metaphyseal vessels intact. Osteonecrosis as a result of these interruptions may occur 8 hours from the time of injury.^{17, 75} In a review of 178 cases of traumatic femoral neck fractures, Claffey¹⁷ reported that 24 had evidence on radiographs of a linear line that crossed the lateral epiphyseal arteries in the subcapital region. All of the patients had evidence of osteonecrosis on clinical examination. Osteonecrosis developed in 10 of 20 cases where the fracture line involved the path of these arteries.

Osteonecrosis is reported as a complication in 85% of displaced fractures and in 15% to 25% of nondisplaced fractures of the femoral neck.^{75, 76} Segmental collapse as a result of mechanical factors occurs 6 months or more after the initial injury and can be seen radiographically. Two studies have reported that osteonecrosis was approximately five times as common in nonunited fractures.^{11, 12}

Osteonecrosis of the femoral head is seen following trauma to the hip without fracture. In a series of 125 cases in which osteonecrosis developed, 78% followed femoral neck frac-

tures, 6% followed dislocation, and 2% were related to trauma to the hip without dislocation or fracture.³¹ Other studies have reported the incidence of osteonecrosis to be 4% to 10% following hip dislocation.²²⁻³⁰ This incidence is related to a delay in effective reduction and to the degree of initial trauma to the hip.

Late segmental collapse of the femoral head has been reported following iatrogenic trauma.^{37, 66} Hip-nailing procedures for intertrochanteric fractures, prolonged or forceful manipulation with treatment of congenital hip dysplasia or slipped epiphysis, arthroplasties and synovectomies with dislocation and excision of the capsule and ligamentum teres, and the cuneiform osteotomy for the correction of slipped epiphysis or coxa vara have been reported as the cause of osteonecrosis.

Nontraumatic

Many drugs and diseases have been linked to osteonecrosis of the femoral head. Although the pathology in the various stages is known, the basic mechanisms initiating their pathogenesis are not fully understood. There is an increasing incidence of femoral head osteonecrosis associated with steroid intake for the treatment of medical illnesses (18% to 57%) or transplantation (13%).^{19, 24, 79} Explanations for the pathogenesis of steroid-induced osteonecrosis include fat embolization from a fatty liver, fat necrosis, steroid-induced vasculitis, compression fractures of osteoporotic bone, and increased coagulability and viscosity of the blood. The association of alcoholism with osteonecrosis of the femoral head is well known and is thought to be caused by fat emboli.^{9, 38, 49, 78} Bone changes owing to a hemoglobinopathy (particularly sickle cell disease) are caused by mechanical interruption to the blood supply of the superior weight-bearing portion of the femoral head. Kenzora and coworkers⁴⁰ proposed the accumulative cell stress theory in an attempt to explain an unidentified process that damages the osteocytes. Solomon⁷⁸ believed that protective pain responses and the anti-inflammatory state associated with analgesics, anti-inflammatory drugs, and alcohol combined with osteoporosis and stress weight-bearing led to subchondral bone collapse.

Several studies have shown that the major cause of osteonecrosis of the femoral head is an increase in the intraosseous pressure of the proximal femur. It is hypothesized that be-

cause the femoral head is a closed compartment, any ischemic episode produces venous obstruction that results in increased intraosseous pressure. Similar to a compartment syndrome, this increase in pressure produces a cycle of progressive ischemia that eventually leads to bone death.^{4, 26, 33} However, the segmental involvement of the anterosuperior weight-bearing portion of the head in osteonecrosis is difficult to explain with this hypothesis.

CLINICAL SYMPTOMS

The onset of symptoms is usually insidious, but an acute attack of pain may happen with minor traumatic injuries. The diagnosis is seen 70% of the time in men. D'Aubigne and Frain²⁰ reported that 75% of patients with osteonecrosis were between 30 and 60 years old. Pain is almost always unilateral; however, in approximately 55% of cases, the opposite hip becomes involved within 2 years. Range of motion is well preserved in the beginning of the disease but gradually deteriorates. Decreased motion and limp develop as degenerative changes progress.

PATHOGENESIS AND RADIOLOGIC CHANGES

Pathologic changes in osteonecrosis have been clinically studied and compared with the changes in experimental animal models.⁴¹ Clinical and pathologic changes may not appear until months or years after the insult; however, histologic and metabolic changes occur within hours. These two types of changes are divided into the early and late stages of osteonecrosis.

In the early stage, cells lose their viability 12 hours after a vascular insult. Histologic changes are not evident until 48 hours later, and radiologic changes are not seen before 2 months. The death of bony trabeculae is not evident until 2 or sometimes 4 weeks postinsult. Because of its nourishment from the synovial fluid, no changes occur in articular cartilage.

Growth of vascular fibrous tissue occurs during the first few weeks as a repair process occurs in the base of the femoral head.^{5, 15, 40, 59, 60, 76} The earliest sign of repair is an increase in density found on radiography in the superolateral area of the femoral head. Thickened trabeculae are noted, and, on occasion, diffuse

osteopenia may be the first radiographic abnormality.

In the late pathologic stage, repair is blocked because of the advancement of fibrous tissue, which becomes a dense avascular wall. Gross segmental collapse leads to increased density of mechanically compressed bone. Areas of radiolucency evolve within the head as the result of a reparative process. Frequently, a radiolucent line or crescent sign is seen beneath the subchondral bone of the superior portion of the femoral head in the anteroposterior or lateral view (Fig. 2A and B). This is a result of the collapse of dead cancellous bone that separates

from the articular cartilage. Ficat and Arlet²³ defined the appearance of the crescent sign as a transitional stage between a spherical and a flattened femoral head. Gradually, flattening of the weight-bearing area occurs, and the crescent sign gradually becomes obliterated with a radiograph that reveals dispersed regions of radiolucency. The acetabulum and articular cartilage remain grossly normal until the very late stage of the disease; however, some surface irregularities and cell injury may be seen histologically. Changes in the acetabulum, such as sclerosis, cystic degeneration, and osteophyte formation, begin late. Eventually, ra-



Figure 2. A, Plain radiograph of the crescent sign and subchondral collapse seen in a femoral head with osteonecrosis. B, MRI of the right hip demonstrating the crescent sign and subchondral collapse seen in a femoral head with osteonecrosis. (Courtesy of Kevin Plancher, MD, New York, NY.)

diography reveals degenerative arthritis of the hip.

CLASSIFICATION AND STAGING

There are several treatment modalities for osteonecrosis of the femoral head. Each treatment regimen may be helpful for a specific stage of the disease process. Evaluating the degree or stage of involvement in the femoral head is important. Staging the disease determines the prognosis as well as the optimal method of treatment.

Several staging systems have been described. The staging system recently reported by Steinberg and colleagues^{85, 86} (Table 2) is similar to that outlined by Marcus⁴⁸ (Table 3), Ficat and Arlet,²⁶ and Ficat (Table 4).²⁷ Steinberg's classification allows the physician to

Table 2. STEINBERG'S RADIOLOGIC STAGING OF OSTEONECROSIS OF THE FEMORAL HEAD

Stage	Criteria
0	Normal or equivocal radiograph, bone scan, and MR image
I	Normal radiograph, abnormal bone scan, and/or MR imaging Mild (<15% of head involvement as seen on MR imaging) Moderate (15% to 30%) Severe (>30%)
II	Abnormal radiograph showing cystic and sclerotic changes in the femoral head Mild (<15% of head involvement as seen on radiograph or quantitative CT) Moderate (15% to 30%) Severe (>30%)
III	Subchondral collapse producing a crescent sign without flattening Mild (subchondral collapse beneath <15% of articular surface) Moderate (crescent sign beneath 15% to 30%) Severe (crescent sign beneath >30%)
IV	Flattening of the femoral head without joint narrowing Mild (<15% of surface has collapsed, and depression is <2 mm) Moderate (15% to 30% collapsed or 2 to 4 mm depression) Severe (>30% collapsed or >4 mm depression)
V	Joint narrowing with or without acetabular involvement Mild (average of femoral head involvement, as Moderate determined in stage IV and estimated Severe acetabular involvement)
VI	Advanced degenerative changes

Table 3. MARCUS' RADIOLOGIC STAGING OF OSTEONECROSIS OF THE FEMORAL HEAD

Stage	Criteria
I	Normal or equivocal radiograph, MR image, or bone scan
II	Sclerotic or cystic lesions
III	Crescent sign
IV	Step-off in outline of subchondral bone
V	Narrowing of cartilage space with degenerative changes

quantify the extent of involvement of the femoral head in both early and late stages. The classification system involves bone scanning or quantitative MR imaging. Clinical records of pain and reduced range of motion are helpful in determining treatment and outcome, but are not part of the staging system.

The classification consists of seven stages. The extent of the disease is subdivided with each level of involvement recorded as mild (A), moderate (B), or severe (C). Correlation between outcome and size of the necrotic segment has been studied by several investigators.^{36, 41, 58, 70, 89} Each stage has a clinical correlation.

Stage 0

Plain films, imaging, and bone scans are either normal or equivocal. This stage includes patients with known risk factors and abnormal but not diagnostic MR imaging or bone scans, patients with hip pain, and patients with osteonecrosis in the contralateral femoral head.

Stage I

Plain films are within normal limits, but MR imaging or bone scanning indicates osteonecrosis. The percentage of abnormal femoral head can be approximated from the

Table 4. FICAT'S RADIOLOGIC STAGING OF OSTEONECROSIS OF THE FEMORAL HEAD

Stage	Criteria
I	Normal or equivocal radiograph, MR image, or bone scan
IIA	Diffuse porosis, sclerosis, or cyst
IIB	Flattening or crescent sign
III	Broken contour of head
IV	Decreased joint space, flattened contour, collapse of head

MR image or calculated by a computerized program.

Stage II

Plain radiographs show definite changes, with alternating areas of radiolucency and sclerosis sometimes distinguished from normal bone or by a sclerotic border. The abnormal area of the femoral head is measured on both the anteroposterior and frog lateral views by tracing the whole head and the involved area on paper. The percentages on the anteroposterior and lateral views are multiplied to obtain the total volume affected.

Stage III

This stage is defined by the emergence of the crescent sign on plain radiographs. This sign indicates collapse of the cancellous trabeculae without flattening of the articular surface. An anteroposterior or lateral view is obtained to measure the length of the crescent sign and the percentage of its length over the entire articular surface. This percentage is calculated with a map-reading planimeter or specially designed grids.⁸⁹

Stage IV

Flattening of the articular surface on either anteroposterior or lateral views is seen. The collapse is often apparent in the anterolateral or superior aspects of the femoral head. CT is helpful in detecting small amounts of segmental collapse if the patient has experienced more pain than anticipated. The acetabulum appears normal on radiographs. The extent measurement is performed as for stage III, and the maximum depression of the collapsed segment is also measured in millimeters using concentric circles or special grids.⁴⁶ This stage has serious prognostic and therapeutic implications because the changes are irreversible.

Stage V

Radiologic acetabular involvement is now present. Steinberg and others believe that this stage almost always follows the flattening of the femoral head because of secondary degenerative arthritis.⁸⁵ The changes evolve as pro-

gressive joint-line narrowing, cyst formation, and sclerosis above the acetabulum with marginal osteophyte formation. The extent and the amount of the collapsed femoral head is measured as in stage IV, and the degree of acetabulum involvement is estimated. The head involvement places a patient in a specific stage.

Stage VI

The hallmark of this stage is advanced degenerative changes.

DIAGNOSIS

Diagnosis in early stages provides the opportunity to prevent collapse of the femoral head. A thorough history must be taken, including past and present diseases, hip trauma, excessive drug intake, and exposure to sporting activities, such as diving. The complete physical examination should include an evaluation of the hip with documentation of the range of motion. The radiologic evaluation should include an anteroposterior view of the pelvis with the legs internally rotated and a lateral view of both hips. Frog leg lateral views can be substituted for a shoot through the lateral view if the patient can flex the hip to 90 degrees with some abduction.

The appearance of radiologic changes on plain staging systems lags several months from the vascular insult. Early diagnosis is important to prevent segmental collapse and to help select the best treatment modality. Special imaging techniques can be used to confirm the suspected clinical diagnosis when plain films are normal. Laminograms historically used in both anteroposterior and lateral views are now rarely obtained. Bone scanning with technetium-99m diphosphate is a sensitive but not specific test. CT is useful once structural changes have evolved in the bone. MR imaging is the best technique for the early diagnosis of osteonecrosis of the femoral head.

Dynamic bone studies and a core biopsy may provide diagnostic information in the early stages of disease. However, with the advent of MR imaging, the acquired information is more reliable than that obtained by dynamic studies. Evaluations, such as intraosseous pressure measurement and venography,^{26, 34, 35} bone-seeking isotope,⁵⁰ electrical determination of gas tensions, tetracycline fluorescence,¹⁰⁰ clearance of radioisotopes, thermal di-

lution, arteriography, and autoradiography have been used in the past but are rarely used today and have been replaced by MR imaging.

Subtle changes in the femoral head not consistent with stage II to VI disease require MR imaging or bone scanning. These modalities are recommended as routine imaging for the opposite hip once a diagnosis of osteonecrosis has been determined. Clinicians must maintain a high index of suspicion in the patient with risk factors for this disease, especially if the contralateral hip is already involved (Fig. 3A and B).

TREATMENT

Osteonecrosis of the femoral head remains a difficult therapeutic problem. The stage of disease at the time of diagnosis determines the best treatment options, although other factors, such as etiology, the duration of symptoms including pain and impediment, age, general health, and unilateral or bilateral involvement, are also important factors.

The ideal approach is to preserve rather than to replace the intact femoral head. The size of the devitalized bone will determine the treatment regimen. If the segment is not close to the weight-bearing surface and is small, it may remain silent, and the necrotic area may be replaced with living bone. Most intracapsular fractures have some associated osteonecrosis. This portion of the femoral head will often heal with proper open reduction and internal fixation. Nontraumatic causes of osteonecrosis can be reduced with adjusted steroid doses and optimal management of renal failure or sickle cell disease; proper precautionary measures should be taken by divers and caisson workers.

Increased force on the weight-bearing surface of the femoral head can affect the progression of segmental collapse. Nonoperative measures, such as observation, analgesics, and limited weight bearing, may be successful when the femoral head is minimally involved. Steinberg and others, however, found that 92% of their cases progressed in stage no matter what was done to avoid collapse.^{26, 41, 80, 82, 84} The authors believe that nonoperative measures should be recommended for pain relief, when the diagnosis is not confirmed, or for conditions that have not progressed to a point at which there is a need for reconstruction. In agreement with Steinberg, Musso and co-workers²⁷ showed no clinical improvement

with nonoperative treatment in 50 hips with Ficat Stage III disease, the rate of arthroplasty was 68% at an average of 16 months after diagnosis.

Nonoperative treatment has been used for many years. In a review of 2025 hips treated either by core decompression or nonoperative management, poor results were reported with nonoperative therapy when compared with core decompression; clinical success rates were 22.7% compared with 53% with a core decompression.⁵⁶ Ohzono⁵⁸ and Stulberg⁵⁷ and their colleagues⁵⁷ also reported a 68% rate of progression of collapse to the femoral head in 115 hips treated nonoperatively.

Preservation of the femoral head is the primary goal in surgery for osteonecrosis of the femoral head. Improved diagnostic techniques have made it possible to intervene before segmental collapse occurs, but no one technique has been completely satisfactory.⁸¹ The following is a discussion of the various surgical alternatives.

Core Decompression

Core decompression is performed through a lateral incision over the greater trochanter with a drill hole made in the distal end of the greater trochanter. Care must be taken to avoid a subtrochanteric fracture. A biopsy of the necrotic area is performed, and drilling is completed up to the subchondral surface and radiographic guidance through the center of the necrotic area. Patients are made non-weight bearing for 3 months postoperatively.

Arlet and Ficat⁴ analyzed osteonecrosis of the femoral head with the use of pressure measurement, venography, and core biopsy. This led them to use core decompression in treatment. Ficat realized that the goal of core decompression was to decrease the high intramedullary pressure in an attempt to relieve pain, improve venous drainage, and amplify vascular ingrowth.^{26, 27, 34, 35} Ficat reviewed the records of 133 patients with stage I and II disease and found a successful clinical result in 90% with no radiologic progression in 79%.²⁷ Hungerford and Lennox³³ reported on 204 hips followed up for 3 years, with no subsequent operation in 96%, 77%, and 60% of patients with Ficat Stage I, II, and III disease, respectively.

The popularity of this procedure has declined because of recent findings.^{14, 44} Smith and co-workers²⁷ reported the results in 114 hips



Figure 3. Plain radiograph (A) and MRI (B) of bilateral osteonecrosis of the hip seen with different Ficat stages. The left side is asymptomatic. (Courtesy of Kevin Plancher, MD, New York, NY.)

with an average follow-up of 3 years and 4 months. They concluded that core decompression should be used for Ficat stage I lesions, but an alternative procedure was recommended for stages above IIB. In a retrospective review of 54 hips, Markel and co-workers²⁶ reported a 35.2% overall success rate. Koo and co-workers³² found that there was no greater value of core decompression in comparison with nonoperative modalities in preventing

collapse in early osteonecrosis of the femoral head except for symptomatic relief. Fairbank and Hungerford²⁵ reported long-term results (11 years) of core decompression with satisfactory clinical results, although 56% of cases (128) had progressed radiologically by at least one additional Ficat stage. These investigators continue to recommend core decompression in young patients to delay the need for total hip replacement.

Core decompressions, when performed before femoral head collapse, has been advocated as an efficient technique to interrupt or delay the disease process in young individuals. However, conflicting clinical results continue to be reported because of the poorly defined natural history of the early stages in this disease.

Bone Grafting

Several different bone-grafting techniques have been advocated in the literature. These include the addition of autogenous or allograft cancellous bone to a core decompression, osteochondral grafts, muscle-pedicle bone grafts,^{7, 52} and free vascularized bone grafts with iliac⁵¹ or fibular bone. Phemister originally described the technique of using a bone graft from the tibia. This technique was subsequently modified by Bonfiglio and colleagues.^{9, 11, 61} The graft from tibia is placed in the canal after the drilling to provide mechanical support for the articular surface. Satisfactory results have been reported in 70% of cases.⁹

Meyers^{7, 52} uses a muscle-pedicle bone graft, inserting it (quadratus femoris muscle pedicle graft) into a posterior window at the femoral head-neck junction after curettage of the necrotic area and packing with cancellous bone. Because of poor results in postcollapsed heads, he modified the procedure to excise the collapsed area, fill the defect with cancellous bone, and then resurface it with fresh cadaveric osteochondral grafts. In a small series of five patients, there were three excellent and two good results at a 2-year follow-up.^{53, 54, 55}

Boettcher and co-workers¹⁰ reported good or fair results with autogenous grafting after a 6-year follow-up. Similar results were obtained by Buckley and co-workers¹³ using allograft. Steinberg and colleagues⁸⁶ reported the results of bone grafting from the trochanter and distal femoral neck. The core of bone is placed back into the hole in a reversed position. This method permits an adequate decompression and helps the repair process by the ingrowth of new vessels. A tibial donor defect is avoided. The authors prefer this approach for the treatment of a precollapsed head. In 1995 Steinberg reported long-term follow-up (2 to 12 years) of 300 hips treated with this approach. He concluded that core decompression with cancellous bone grafting is a safe and effective treatment for early stages of osteone-

crosis of the femoral head.⁸³ The authors agree with Rosenwasser and co-workers⁶⁷ that young patients with Ficat stage II and III disease should be treated with a core decompression and bone grafting.

Free Vascularized Bone Grafting

Vascularized fibular grafting has been advocated to promote healing of the femoral head by decompression, excision of the sequestrum, and examination of the spontaneous repair process through replacement of the affected bone with a viable cortical graft and cancellous bone, all to prevent the progression of disease. Urbaniak reported on his initial series of 80 cases treated by curettage and placement of autogenous iliac bone⁷ and a vascularized fibular graft. A total of 26 patients had Ficat Stage I and II disease and 54 patients Ficat Stage III and IV disease. A Watson-Jones surgical approach was used. A drill hole is made into the femoral head as performed in routine decompression. The diameter of the hole is made large enough to fit the graft without compression. Cancellous bone is taken from the drill hole and pushed back into the cavity, followed by insertion of the fibular graft. The fibular graft is harvested according to the method described by Gilbert and colleagues.²⁹ Fixation of the graft is accomplished with a small Steinmann pin. An end-to-end vascular anastomosis is performed. At 3-year follow-up, Urbaniak reported a failure rate of 7.5%.^{50, 95}

In 1995 Urbaniak and co-workers⁸⁶ reported their long-term follow-up study of 103 hips treated with free vascularized fibular grafting. There was an 81% patient satisfaction, with 86% of patients reporting a decreased need for pain medication. Several other investigators have reported similar results and suggest that this is an excellent method to salvage patients with osteonecrosis.^{45, 102} Urbaniak⁹⁷ recently reported that the removal of a vascularized fibular segment was associated with some motor weakness and sensory deficit in the foot in 11.8% of patients at 5 years. The prevalence of pain in the ankle and foot increased with time from 1.6% at 3 months to 11.5% at 5 years. It was concluded that although this procedure remains ideal for young patients, the morbidity must be weighed against the benefit. The authors consider free vascularized bone grafting to be a reliable alternative method for the management of femoral head osteonecrosis in young patients (Fig. 4A and E).

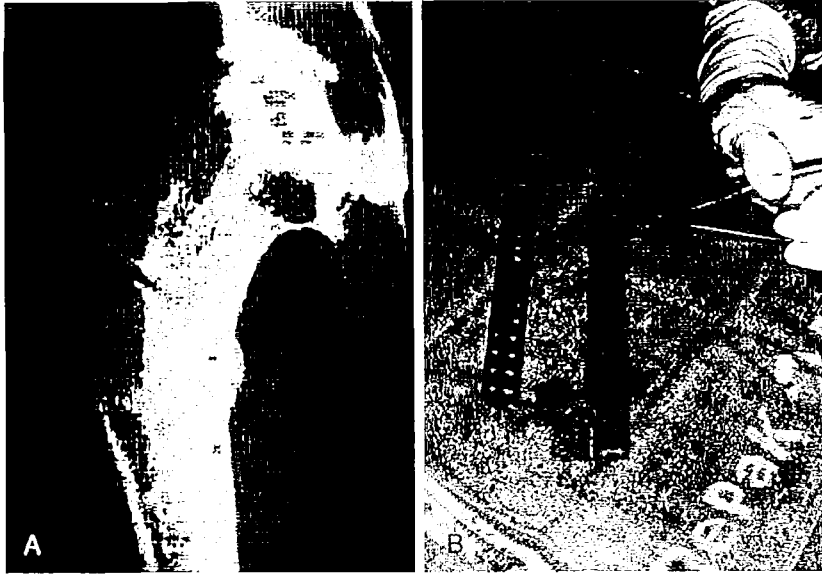


Figure 4. A, Plain radiograph of osteonecrosis of hip; Ficat stage II. B, Free vascularized fibular graft harvested.

Illustration continues on opposite page

Some centers have performed other types of vascularized bone grafts in conjunction with an osteotomy. Ganz and Buchler have described a technique used by Flori. A distally ligated arteriovenous pedicle, arteriovenous fistula loop, or other vascularized bone graft is placed in the slot of decompression and a realignment osteotomy performed.²⁸

Electrical Stimulation

Electrical stimulation of a femoral neck nonunion is associated with an 80% to 85% union rate.⁶ Electrical stimulation (pulsed electromagnetic field) has been advocated for the treatment of osteonecrosis of the femoral head because its histologic appearance is similar to that of nonunion.^{5, 15} In 1989 Steinberg and co-workers² reported that the addition of direct current stimulation to decompression and grafting seemed to improve the results. Aaron and co-workers¹ compared the results in 50 hips treated by decompression with the results in 56 hips treated with pulsed electromagnetic fields with an average follow-up of 2.3 years. The outcome was successful in hips with Ficat Stage II and III disease treated with pulsed electromagnetic fields. In 1990 Trancik and co-workers⁹³ evaluated 11 hips with Ficat stage II disease treated with core decompression and placement of an electrical stimulating coil. There was no extra benefit in the prevention

of further femoral head destruction. Similar findings were obtained with capacitive coupling as an adjunctive treatment with decompression and grafting.⁸¹ Currently, most clinicians do not use electrical stimulation for the treatment of femoral head osteonecrosis.

Osteotomy

Various types of femoral osteotomies have been performed to preserve the femoral head in young individuals with stage III disease. Pathophysiologic events of osteonecrosis of the femoral head, as studied by Kenzora, suggest that subchondral resorption exceeds repair. This results in the loss of structural integrity with collapse. Osteotomy helps to avoid this phenomenon by redirecting the forces on the femoral head.

Flexion/extension with a varus osteotomy was advocated by D'Aubigne and Frain²⁰ in 1972. This procedure was performed on 92 subjects with a 1- to 13-year follow-up. Forty percent of the patients progressed to degenerative arthritis. It was suggested that this procedure should be performed only when the necrotic segment is small and can be completely removed. Kerboul and co-workers⁴¹ recommended a varus osteotomy when at least a 20-degree arc of the lateral head was intact. Saito and colleagues⁷⁰ supported this procedure

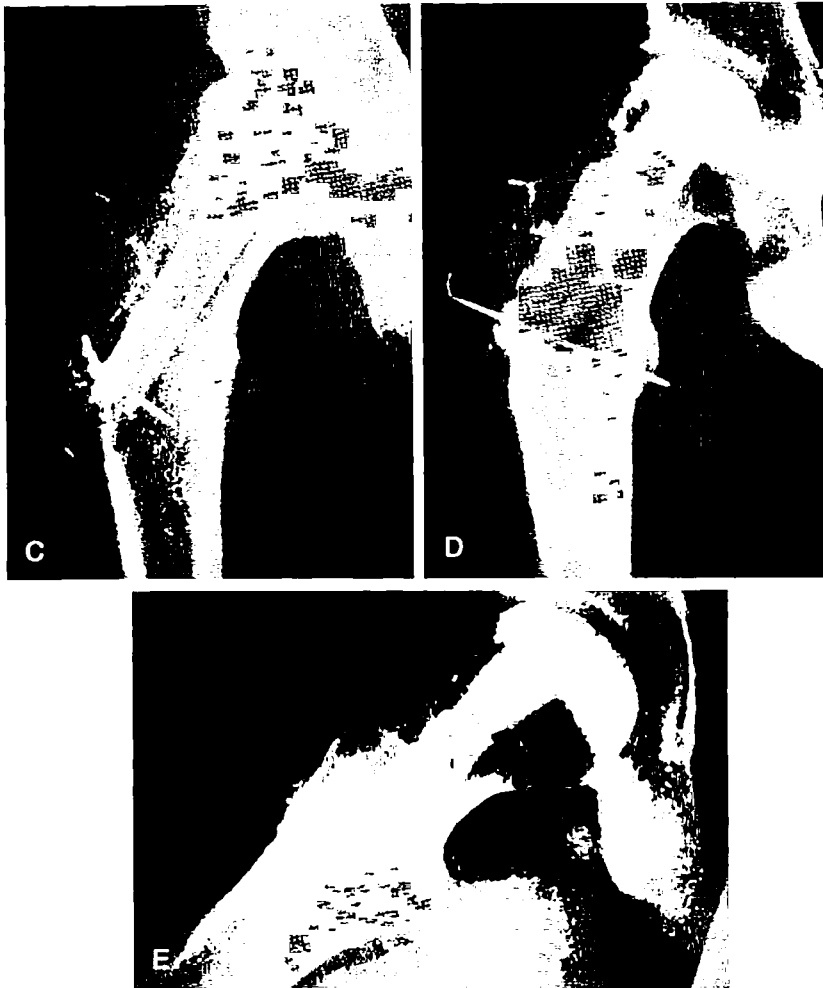


Figure 4. (Continued). C. AP radiograph 1 month postoperatively from a free vascularized fibula transfer. D. AP radiograph 6 months postoperatively in the same patient. E. Lateral view 6 months postoperatively. (Courtesy of Kevin Plancher, MD, New York, NY.)

when more than one third of the femoral head was intact laterally (Fig. 5A and B).

A valgus/flexion osteotomy is recommended by Wagner when the total angle of necrosis is 200 degrees or less, the lesion is primarily anterolateral, and the patient is young and active (Fig. 6A and B). The procedure is performed with the hip flexed 30 to 60 degrees with valgus of no more than 15 to 20 degrees. If the lesion is more central, a varus or varus/extension osteotomy is preferred.³⁹ Scher and Jakim⁷ conducted a prospective evaluation of 45 hips with Ficat stage III disease treated with valgus/flexion intertrochanteric osteotomy and autogenous bone grafting. The success rate was 87%. The authors recommend this osteotomy in patients with osteonecrosis that involves the anterosuperior segment of the femoral head, in patients with Ficat

stage III disease, and in male patients less than 45 years old or young female patients.

Sugioka⁴⁰ advocated another type of osteotomy to relieve the pressure from a large area of necrosis in the weight-bearing zone. The femoral head and neck are rotated 60 degrees or more on the long axis of the neck. Good joint surface preservation was found in a 10-year follow-up. In 1992 Sugioka introduced a transtrochanteric anterior rotational osteotomy.⁴⁰ It was possible to obtain transposition of the articular cartilage to the weight-bearing area in the sagittal plane. The rate of excellent results was 89% in 98 hips with Ficat Stage II disease, 73% in 134 hips with stage III disease, and 70% in 63 hips with Stage IV disease. Femoral head collapse was prevented in 93% of cases followed up for 3 to 16 years, even in patients receiving steroids. This procedure has

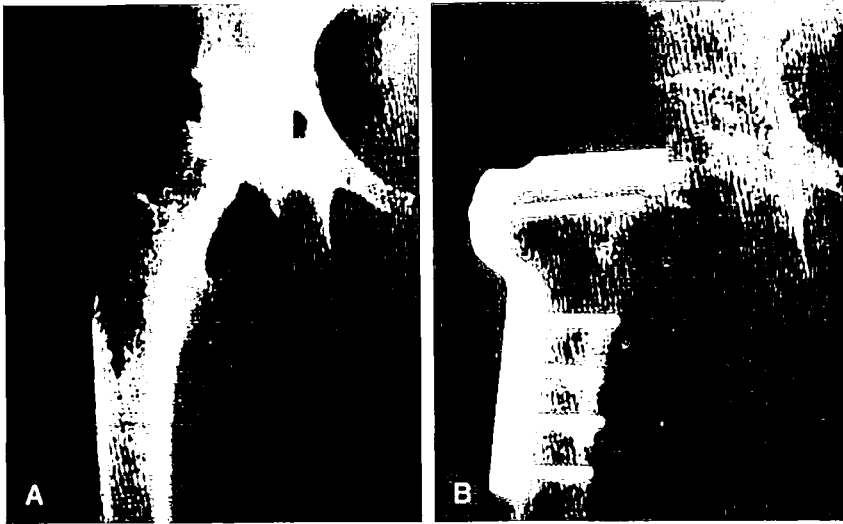


Figure 5. AP radiographs preoperatively (A) and postoperatively (B) of a hip with osteonecrosis treated with a varus/extension osteotomy. (Courtesy of T. Thornhill, MD, Boston, MA.)

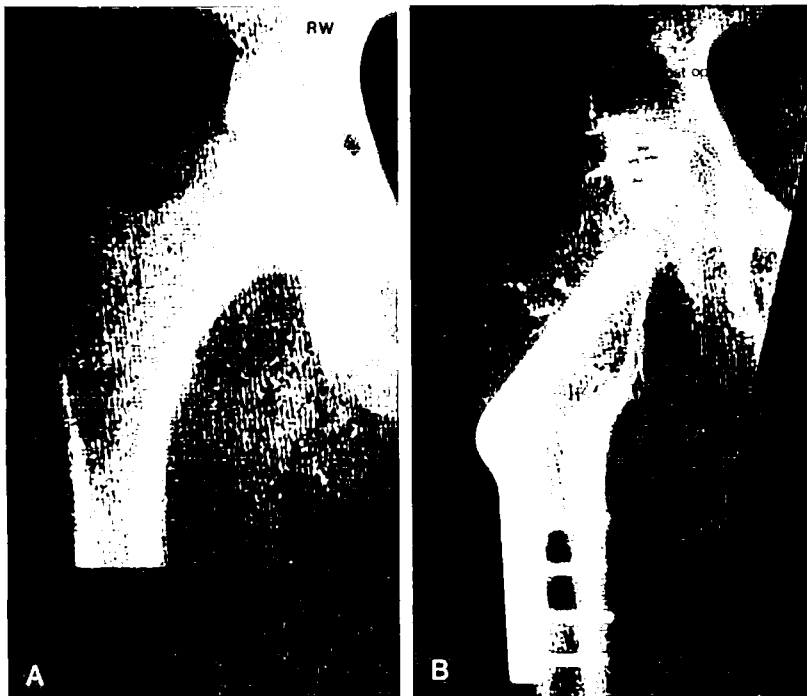


Figure 6. AP radiographs preoperatively (A) and postoperatively (B) of a hip with osteonecrosis treated with a valgus/extension osteotomy. (Courtesy of T. Thornhill, MD, Boston, MA.)

not been recommended by other surgeons because it is technically difficult to perform, and most surgeons are unable to reproduce Sugioka's results.⁸⁹⁻⁹¹ In 1993 Dean and Cabanela²¹ reported that 83% of their cases (15 of 18) had further collapse following this procedure. Isotope scans have shown that this type of osteotomy may impair the residual blood supply of the femoral head. Dean and his colleagues subsequently abandoned the use of this procedure at their center.

Joint Reconstruction

Reconstructive measures are preferred once the femoral head has collapsed or acetabulum degenerative changes have occurred. Several reconstructive procedures have been reported for different stages of osteonecrosis. Prosthetic replacement is frequently an unappealing option for young patients. Risk factors associated with osteonecrosis accompany many of the complications of these reconstructive modalities.

Cup Arthroplasty

Two types of cup arthroplasty have been advocated for the treatment of osteonecrosis of the femoral head. Kerboul and co-workers⁴¹ described the use of adjusted cup arthroplasty when the results from the standard technique were not satisfactory. The cup was impacted onto the reamed femoral head. Good results were obtained in 66% of cases, fair results in 16%, and failure in 13% regarding function and pain relief. Sedel and co-workers⁷³ reported the results of spherocylindric cup arthroplasty in 38 hips with an average follow-up of 6 years. They recommended this procedure as a justifiable alternative to total hip replacement in young adults because some patients remain stable even after 10 years. Sellman and Froimson⁷⁴ reported long-term follow-up of a young child with Gaucher's disease. Resurfacing arthroplasty was performed instead of cup arthroplasty. Currently, the authors are not aware of any centers in the United States that continue to perform cup arthroplasty.

Femoral Head Replacement

It is logical to change the femoral head without replacing the acetabulum because the ini-

tial pathology of osteonecrosis is in the femoral head. The Thompson and Austin-Moor single-component and the Bateman bipolar prosthetic devices have been advocated. Long-term unsatisfactory results have been reported with both cemented and noncemented fixed-head prostheses. Complications include femoral loosening (distal subsidence or angular shift), deterioration of acetabulum cartilage, and protrusio acetabuli.^{23, 101} Steinberg and colleagues reported that for patients under 50 years of age, the revision rate was 47% with a mean of 5.4 years to revision. Multicomponent devices are associated with a better outcome than single-component prostheses. In 1992 Takaoka and co-workers⁹⁷ compared a series of 83 hips treated with bipolar prostheses with or without cement with a series of 19 hips treated with Austin-Moor single-component prostheses. The bipolar prostheses were the preferred method of treatment for advanced osteonecrosis.

Surface Replacement

Surface replacement has some advantages over total hip replacement because the former procedure leads to preservation of the femoral head and neck and allows for a future total hip replacement if necessary. Several investigators have suggested that this procedure is effective in cases in which the femoral head is not extensively involved, permitting the prostheses to be fit on structurally viable bone.² A total of 100 porous surface replacements were performed by Amstutz and colleagues in patients with a mean age of 53 years with 1 to 4 years of follow-up. The porous replacement avoids the major disadvantage of an acrylic-fixed surface replacement, namely, the excessive acetabular reaming. The method of surface replacement has been abandoned in many centers because of its high failure rate.

Total Hip Replacement

In 1981, approximately 10% of the more than 500,000 patients undergoing total hip replacement in the United States had a diagnosis of osteonecrosis of the femoral head.⁴⁷ When there is involvement of both the acetabulum and femoral head, the best and most frequent reconstructive therapy is total hip replace-

ment^{25, 80} (Fig. 7). Several reports indicate that this method is not used in young individuals who are heavy or physically active. These studies reveal a 15% to 20% 5-year failure rate in cemented total hip replacements in patients younger than 40 years.^{47, 80} Several studies of cemented total hip replacements suggest that osteonecrosis is a bad prognostic indicator. In a report of 103 total hip arthroplasties, 12 hips failed; 11 of these 12 hips had a diagnosis of osteonecrosis.⁷⁴ Saito and co-workers⁷¹ reported that patients with a diagnosis of osteonecrosis had a poorer outcome after cemented total hip replacement than age-matched patients with osteoarthritis. Moreover, several reports have demonstrated that patients with idiopathic osteonecrosis or with trauma have better outcomes than patients with a systemic disease that requires the use of steroids. When arthroplasty is considered for the treatment of young patients, the use of total hip implants without cement, which facilitates bone growth, is a better alternative than arthroplasty with cement.⁶² Piston and co-

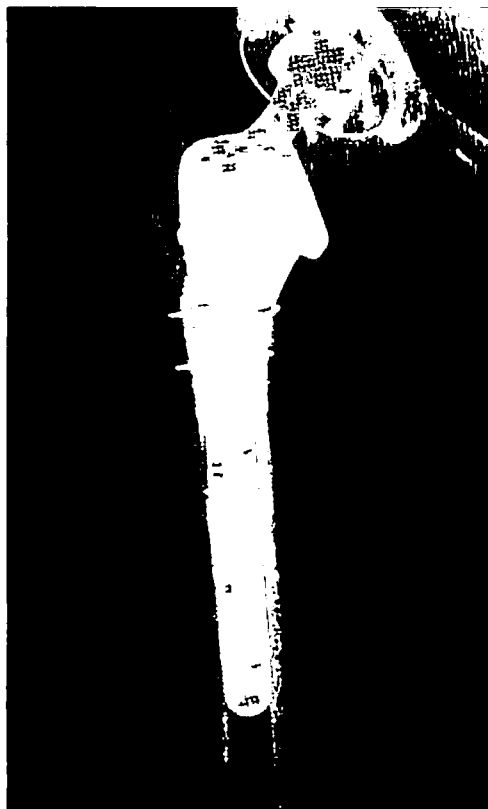


Figure 7. Total hip arthroplasty used for treatment of osteonecrosis of the hip. (Courtesy of Kevin Plancher, MD, New York, NY.)

workers⁶³ recommended caution when performing arthroplasty without cement because of its high rate of complications and the potential of failure owing to osteolysis. Reports citing high incidences of thigh pain in cementless total hip arthroplasties are well known. Katz and co-workers⁵⁷ reported a 29% prevalence of this complication. Additional studies with longer follow-up and larger sample size need to be performed to evaluate cementless versus cemented total hip arthroplasties.

OTHER TECHNIQUES

Girdlestone Pseudarthrosis

Girdlestone pseudarthrosis is used as a salvage procedure for special circumstances. A painful hip with superimposed sepsis, a failed total hip replacement with sepsis, or a femur without good bone stock are all indications for a girdlestone procedure. Pain relief and good range of motion can be achieved if the procedure is performed properly. Conversion to a total hip replacement can be performed at a later date.

Hip Fusion

Hip fusion is infrequently recommended for osteonecrosis because of its high failure rate. Patients are not usually satisfied with a fused hip. Consideration should always be given to the opposite hip because there is a high incidence of bilateral involvement. Roberts and Felto⁶⁵ believe that this procedure could serve as an alternative to cemented total hip arthroplasty until cementless total hip replacement is proven to be successful in younger patients.

TREATMENT RECOMMENDATIONS ACCORDING TO STAGING

Stages 0 through II

Prophylactic measures should be taken before collapse of the femoral head. An aggressive approach is recommended to halt the progression of disease. Core decompression and grafting are advised in these stages. An adjunctive pulsed electromagnetic field could be added to this regimen. Free vascularized grafts

have shown good outcomes. Osteotomies are suggested in very late stage II disease.

Stage III through IVA

As long as there is no or minimal flattening of the femoral head, it is still ideal to preserve the head. This is possible using osteotomy procedures. However, once collapse occurs, the process will inevitably progress.

Stage IVB through IVC

At this point, there are no therapeutic measures to preserve the head; the only nonreconstructive therapy is the Sugioka rotational osteotomy. A femoral head arthroplasty using a single-polar but preferably bipolar prosthesis is advised. Nevertheless, most of these patients will need total hip replacement long term.

Stages V through VI

The acetabular cartilage has already been distracted in this stage, and total hip replacement is the only modality left. In special situations, other techniques may be utilized.

CONCLUSION

Osteonecrosis is a complex entity that requires a specific treatment regimen after accurate diagnosis. Early diagnosis is crucial. MR imaging is the gold standard diagnostic tool. As is true in all areas of orthopedics, attention to a detailed history and physical examination is important. Preservation of the femoral head is the primary goal of any intervention modality. Improved diagnostic techniques have made it possible to intervene before segmental collapse occurs, but no one technique has been completely satisfactory.

References

- Aaron RK, Lennox D, Bunce GE, et al: The conservative treatment of osteonecrosis of the femoral head: A comparison of core decompression and pulsing electromagnetic fields. *Clin Orthop* 249:209, 1989
- Amstutz HC, Christie J, Mensch JS: Treatment of osteonecrosis of the hip. *In* The Hip: Proceedings of the Third Open Scientific Meeting of the Hip Society. St. Louis, Mosby, 1975, pp 19-34
- Amstutz H, Kilgus D, Kabo M, et al: Porous surface replacement of the hip with chamfered-cylinder component. *Arch Orthop Trauma Surg* 107:73, 1988
- Arlet J, Ficat P: Biopsy drilling as a means of early diagnosis. *In* Zinn WM (ed): Idiopathic Ischemic Necrosis of the Femoral Head in Adult. Baltimore, University Park Press, 1971, pp 74-80
- Bassett CAL, Schink-Ascani MM, Lewes SN: Treatment of femoral head osteonecrosis with pulsed electromagnetic fields (PEMFs). *In* Arlet J, Ficat RP, Hungerford DA (eds): Bone Circulation. Baltimore, Williams & Wilkins, 1983, pp 343-354
- Bassett CAL, Valdes MG, Hernandez E: Modification of fracture repaired with selected pulsing electromagnetic fields. *J Bone Joint Surg* 64[A]:888, 1982
- Baksi DP: Treatment of post-traumatic avascular necrosis of the femoral head by multiple drilling and muscle bone grafting. *J Bone Joint Surg* 65[B]:268, 1983
- Bobechko WP, Harris WR: The radiographic density of avascular bone. *J Bone Joint Surg* 42[B]:626, 1960
- Boettcher WG, Bonfiglio M, Hamilton HR, et al: Nontraumatic necrosis of the femoral head. *J Bone Joint Surg* 52[A]:322, 1970
- Boettcher WG, Bonfiglio M, Smith K: Nontraumatic necrosis of the femoral head: II. Experiences in treatment. *J Bone Joint Surg* 52[A]:322, 1970
- Bonfiglio M, Voke EEM: Septic necrosis of the femoral head and non-union of the femoral neck. *J Bone Joint Surg* 50[A]:48, 1968
- Boyd HB, George IL: Complication of fractures of the neck of the femur. *J Bone Joint Surg* 29[B]:13, 1947
- Buckley PD, Gearen PF, Petty RW: Structural bone-grafting for early traumatic avascular necrosis of the femoral head. *J Bone Joint Surg* 73[A]:1357, 1991
- Camp JF, Colwell CW: Core decompression of the femoral head for osteonecrosis. *J Bone Joint Surg* 68[A]:1313, 1986
- Catto M: A Histological appearances of late segmental collapse of the femoral head after transcervical fracture. *J Bone Joint Surg* 47[B]:777, 1965
- Chung SMK, Alavi A, Russell MO: Management of osteonecrosis in sickle-cell anemia and its genetic variants. *Clin Orthop* 130:158, 1978
- Claffey TJ: Avascular necrosis of the femoral head. *J Bone Joint Surg* 42[B]:802, 1960
- Coleman SS, Compere CL: Femoral neck fractures: Pathogenesis of avascular necrosis, nonunion and late degenerative changes. *Clin Orthop* 20:247, 1961
- Cruess RL, Blennerhassett J, MacDonald FR, et al: Aseptic necrosis following renal transplantation. *J Bone Joint Surg* 50[A]:1577, 1968
- D'Aubigne RM, Frain PG: Theory of osteotomies. *Rev Chir Orthop Reparatrice Appar Mot* 58:159, 1972
- Dean MT, Cabanela ME: Transtrochanteric anterior rotational osteotomy for avascular necrosis of the femoral head: Long term results. *J Bone Joint Surg* 75[B]:597, 1993
- Donalson WE Jr, Rodriguez EE, Skovron M, et al: Traumatic dislocation of the hip joint in children. *J Bone Joint Surg* 50[A]:79, 1968
- Drinker H: The universal proximal endoprostheses: A short term comparison with conventional hemiarthroplasty. *J Bone Joint Surg* 61[A]:1167, 1979
- Dubois EL, Cozen L: Avascular (aseptic) bone necrosis associated with systemic lupus erythematosus. *JAMA* 174:108, 1960
- Enneking WF: The choice of surgical procedures in idiopathic aseptic necrosis. *In* The Hip: Proceedings