OSTEONECROSIS OF THE FEMORAL HEAD: CURRENT CONCEPTS AND CONTROVERSIES

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INTRODUCTION

Osteonecrosis (ON) of the hip is a disease in which the living elements of bone in the femoral head die. Although it has been a well-known clinical entity for greater than 100 years, only in the last 20 years has a significant amount of research been devoted to this condition. The interest in ON has produced a recent explosion of literature concerning its pathogenesis, diagnosis, natural history, and treatment. However, this information has done little to clarify current concepts. In fact it has only heightened the controversy due to the variability in approaches to ON and the results of treatment. The purpose of this summary is to highlight past ideas, outline some current concepts, and explore recent controversies regarding osteonecrosis of the femoral head.

HISTORY

A German surgeon named Franz Konig is given credit for the first description of ON of the femoral head in 1888. The German pathologist George Auxhausen described the histology of bone necrosis and was the first to use the term aseptic necrosis to describe an anemic bone infarct. Dallas Phemister, influenced by Auxhausen's work, studied bone necrosis and bone grafting extensively in the 1930's and 1940's. He made tremendous strides in correlating the clinical findings, radiographic changes, and pathology of bone necrosis. Phemister believed in the concept of an ischemic bone infarct, and later reported on the treatment of necrosis by femoral head drilling and tibial bone grafting in 1949. In 1935, Chandler proposed that ON of the femoral head was analogous to a myocardial infarction and termed it "coronary artery disease of the hip"[30]. Based on work from these early pioneers, many investigations have been carried out to determine the pathogenesis, natural history, and treatment of ON of the femoral head. Although our knowledge of etiologic factors, stages, and the natural history in some cases has advanced, little concrete information regarding the actual pathogenesis or best form of treatment has been emerged since these early works.

Etiology and Pathogenesis

Many etiologies and associated factors have been identified in ON of the femoral head. Trauma such as an intracapsular femoral neck fracture or dislocation of the hip can interrupt the blood supply and result in ON. Osteonecrosis is a well-known complication of allograft organ transplantation with steroid administration used for a variety of conditions. Alcohol abuse is one of the most common causes of ON today. Dysbaric phenomena such as Caisson's disease, sickle cell anemia, and Gaucher's disease are well-known, although less common causes of ON of the femoral head. Other possible factors include gout and hyperuricemia, radiation, osteoporosis, hypophosphatemia, hyperparathyroidism, and connective tissue diseases. Often, many factors are present at one time, such as a patient with a connective tissue disease treated with corticosteroids who develops renal failure and undergoes renal transplantation. In addition, up to one-third of all cases may be truly idiopathic, without an identifiable cause, associated factor, or clear pathogenesis.

Much has been written, but little has been learned about the pathogenesis of most cases of ON of the femoral head. It seems plausible that the blood supply would be disrupted in displaced femoral neck fractures and hip dislocations, with subsequent development of ON. However, there are cases of certain complete circulatory disruption which do not lead to ON, and some cases where the blood supply should theoretically remain intact, yet ON develops.

In sickle cell disease and dysbaric phenomena, a thromboembolic mechanism is thought to occur. In sickle disease this would result from sludging of red cells, and in Caisson's disease this represents nitrogen bubbles. In infiltrative disorders such as Gaucher's disease, it is thought that the circulation is encroached with subsequent compromise of nutrition to the osteocytes and marrow.

Intravascular fat has been proposed as a cause of ON in steroid treated patients. This fat has been demonstrated histologically in animal experiments and in humans on steroids. However, no documentation of histologic necrosis has been found despite extensive fat emboli in these studies. Humans treated with steroids have been found to have fat emboli at autopsy, but this has not been correlated with necrosis[10,14]. Spencer, et al. suggested necrosis might occur from a direct cytotoxic affect of steroids on osteocytes or the interference with interosseous microcirculation in the subchondral bone. These conclusions were drawn from abnormalities in the microcirculation found in autopsies of patients on high dose corticosteroids[43,44]. Fat emboli have also been implicated as the cause of alcohol-associated ON.

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Arlot, et al. proposed that osteoporosis or osteomalacia may underlie ON in almost all patients, based on histomorphometry of iliac biopsies from patients with a variety of diagnoses[3]. Andresen and Nielsen believed that an abnormal status of the bone [i.e. osteoporosis] at the time of organ transplantation was important in the subsequent development of ON[2]. Elmstedt proposed that microfractures in osteopenic bone produced vascular changes and eventual ON[12]. Boettcher, et al. presented laboratory data supporting a coagulopathic state as the underlying cause in a series of patients with nontraumatic ON[6]. Saito, et al. supported a theory of single episodes of infarctions from the interruption of a segmental blood supply as the cause of necrosis in cases they called minimal osteonecrosis (MON)[40]. Finally, Ficat and Hungerford believe that intraosseous pressure elevation interferes with blood flow, analogous to a Starling resistor. They liken the femoral head to a compartment and the elevated pressure to a compartmental syndrome of bone[13,20].

It is clear that no theory on pathogenesis is widely accepted, nor can one theory explain all the causes of ON. Any theory must take into account the 80 percent incidence of bilateral disease in patients and the variety of clinical and radiographic presentations. It must explain the multitude of factors associated with the disease process, and the final common pathway of bone necrosis followed by repair and often collapse. Importantly, many surgical procedures have been adopted to address proposed pathogenetic mechanisms, often with little scientific foundation. Theoretically any intervention should address a proven pathogenesis to be successful.

Diagnosis

The diagnosis of ON is made in a patient at high risk who has typical radiographic findings. However, there is no "gold standard" in the diagnosis of early lesions. The history and physical exam are often non-specific, and radiographs are negative in early disease. Magnetic resonance imaging has recently emerged as the most sensitive, specific, and widely used diagnostic tool in these cases. In most reports, MRI can diagnose very early lesions with a greater than 90 percent specificity and sensitivity based on histology or eventual progression[4,5,18,33,34,38,44]. However, one study has not verified this degree of accuracy in early diagnosis, and debate exists regarding the usefulness of MRI in staging lesions, predicting progression, or following response to treatment[9,15,33,52,55]. With further experience, the accuracy should increase and the cost decrease. The added benefits of convenience and lack of ionizing radiation will solidify it as the diagnostic test of choice for suspected early ON.

Previously, plain radiographs, bone scans, CT scans, and tomograms have been used to diagnose osteonecrotic lesions. The accuracy of these tests done alone or in combination has not equaled MRI in comparative studies[33,34]. However, bone scans are useful in early diagnosis. Plain radiographs have a role in diagnosing later stages and in following progression. Other tests may have a role in selective cases and in preoperative planning. The functional exploration of bone (FEB) is a procedure that measures intraosseous pressure, employs venography, and includes a core biopsy (described later). Popularized and described as highly accurate by Ficat, its role in diagnosis has recently been questioned[33].

As part of a prospective protocol using a multimodality approach to the diagnosis in suspicious cases, Stulberg, et al. found MRI better than bone scans, SPECT scans or functional explorations of bone to diagnose asymptomatic lesions[49]. However, bone scanning was the most cost effective method for all cases. It had comparable sensitivity, specificity and predictive value and was recommended as the preferred test in symptomatic cases. The functional exploration of bone had the lowest sensitivity and specificity and was not well-tolerated by patients.

It is generally agreed that early diagnosis is essential in ON. Plain radiographs can be used to follow lesions once they appear radiographically, but this is relatively late in the course. Magnetic resonance imaging has superceded previous tests as the most accurate method of diagnosing lesions early and should be used in all symptomatic patients considered at high risk. Whether or not MRI should be used as a screening tool in all patients considered at high risk is debatable[9].

Classification

Three accepted classifications of ON have been described in the literature. These are shown in table form (see Tables I, II, and III). The Ficat system is the most widely accepted and seems to delineate the usual course of progression[13]. Marcus and Enneking's system does not include preradiographic phases with or without symptoms[28]. The Steinberg classification includes a stage 0 which he describes as a clinically suspicious hip without symptoms, MRI findings or radiographic changes[47].

No study has been done to compare the three staging systems. However, it appears that most patients go through an asymptomatic period followed by a symptomatic and preradiographic period. Therefore, the Ficat classification seems most appropriate.

Natural History

It is generally believed that ON of the femoral head will inexorably progress to collapse and advance to degenerative changes of the hip. This natural history has been demonstrated several times for hips in all radiographic stages[21,25,35,46]. Musso demonstrated that only three of

Table I
FICAT STAGING[28]

Stage	*Clinical*	*Radiographs*	*Bone Scan (MRI*)*
0+	Asymptomatic	Negative	Usually++
I	Pain	Negative	Positive
II	Pain	Mottling cysts	Positive
III	Pain	Collapse	Positive
IV	Pain	Degenerative changes	Positive

+Ficat originally described the four advanced Stages. Hungerford later identified Stage 0.
++Although Ficat believed Core Biopsy was the only method able to diagnose Stage 0, most would agree that the bone scan, and certainly the MRI, would be positive.
*MRI has superceded Bone Scan in the Early Detection of ON.

Table II
STEINBERG STAGING*[47]

Stage	*Criteria*
0	Normal or nondiagnostic radiographs, bone scan, and MRI
I	Normal radiographs, abnormal bone scan, and/or MRI
II	Abnormal radiographs (cystic, sclerotic changes without collapse)
III	Subchondral collapse
IV	Flattening of the femoral head without joint space narrowing or acetabular involvement.
V	Joint narrowing and/or acetabular involvement
VI	Advanced degenerative changes

*Steinberg also advises staging the extent of the lesion by letters A, B. and C depending on size by MRI or x-ray.

Table III
MARCUS STAGING[13]

Stage	*Clinical*	*Radiographs*
I	Asymptomatic	Mottled densities
II	Asymptomatic	Infarcted demarcated by density
III	Pain—mild and intermittent	Crescent sign
IV	Pain with activity	Depression of infarct
V	Pain with activity	Flattening and compression
VI	Pain at rest	Degenerative arthritis

50 hips remained stable at an average follow-up of 16 months[35]. Despite bed rest, crutch walking, non-steroidal anti-inflammatory drugs (NSAID's), and analgesics, 47 out of 50 progressed. Thirty-eight out of 50 either underwent total hip arthroplasty (THA) or had it recommended. Steinberg, et al. presented similar data on 44 out of 48 lesions with radiographic progression[46]. Hungerford and Zizic reported 21 of 22 Ficat Stage II lesions progressed over time with conservative management[21]; all 11 Stage III lesions progressed. Glimcher and Kenzora reported that "following femoral head collapse, conservative measures will not provide symptomatic relief"[16], the majority will require THA. The only study apparently demonstrating resolution of radiographic changes in femoral head osteonecrosis was reported by Andresen and Nielsen. Regression occurred in seven out of 25 lesions, and stabilization in 10 out of 25 femoral heads following renal transplantation. These were all diagnosed in late stages by the radiographic density changes and/or collapse[2].

Osteonecrosis can now be diagnosed earlier with MRI and other studies. It has been assumed that the natural history in these lesions would be as unfavorable as those diagnosed in late radiographic stages. However, the minimal evidence that is available would suggest the contrary. Saito, et al. has described minimal osteonecrosis (MON) of the femoral head as a small and eccentrically localized lesion with clinically benign features[40]. They found MON in 19 hips out of 275 diagnosed with osteonecrosis. Of these 19 cases diagnosed by radiographs, scintigraphy and biopsy, none progressed to collapse or showed any increase in the size of the osteonecrotic lesion over a follow-up period of three to 13 years. Kopecky, et al. reported a prospective study using serial MRI's from the time of renal transplant to 24 months after transplant. Of the 25 hips with apparent ON by MRI, 11 showed regression or disappearance of the lesion without treatment, at an average follow-up of 16 months[23].

Although current recommendations are for early diagnosis and intervention, the actual natural history of early lesions is unknown and may be benign in the majority of cases. Several studies have demonstrated adverse effects of associated factors such as steroids or alcohol abuse[7,27,53]. Most recently, an increased size and location of a lesion in the weight-bearing portion of the femoral head has predicted a poor outcome in both the natural history and in treated cases[47].

TREATMENT

As described earlier, no form of conservative management such as bed rest, protective weight-bearing, analgesics, or NSAID's has proven effective in treating ON of the femoral head[35,46]. Most cases will lead to collapse and advanced degenerative changes; however, the natural history and response to conservative measures of early ON is not known.

The only universally accepted treatment of ON is THA, which is usually reserved for advanced stages. Because the disease most commonly affects young adults, who are

not good candidates for joint arthroplasty, the emphasis has been on procedures to halt progression of early stages. The following is a brief description and reported results of some of these procedures.

Structural Bone Grafting

Phemister first reported the technique of drilling holes and inserting rectangular bone pegs into the femoral head in 1949[37]. He believed that drilling removed dead bone, allowed rapid invasion of a healing response, and prevented fracture and collapse of the head. His anecdotal results on post-traumatic necrosis supported this technique which is still used today in some centers. At the University of Iowa, Bonfiglio and Boettcher modified Phemister's technique and demonstrated an 80 percent satisfactory "healing rate" in approximately 150 patients with traumatic and nontraumatic necrosis of the femoral head[6]. Poor results were attributed to poor technique in half their cases. Later follow-up in 1980 demonstrated continued good results if no or minimal femoral head collapse was present during the index operation[42]. Graft placement into the subchondral cortex was found to be critical to success. Nelson and Clark reviewed the more recent Phemister procedures done at the University of Iowa, with a minimum two year follow-up[36]. Fifty-two percent of patients required THR and between 82 and 95 percent progressed at least one Marcus stage.

At the University of Florida, Marcus, Springfield, and Enneking have supported cortical strut grafting in patients with Stages I and II (Table III)[28,45]. They reported a success rate of up to 90 percent in these early stages. Buckley, et al. recently reported on 20 operatively treated patients with a follow-up of greater than two years, again with a 90 percent success rate[7]. They also attributed failures to improper graft placement, poor surgical indications, continued steroid use, or alcohol abuse.

Core decompression without bone grafting has been the most popular and controversial treatment of ON of the femoral head. Described and popularized by Ficat, the procedure involves removing two separate 6-10 mm core tracks from the femoral head and neck. He believes that the pathogenesis of ON relates to interosseous hypertension, which causes impaired circulation and ischemia, similar to a compression neuropathy or compartment syndrome. As stated before, this is typically done as part of a three part procedure called the functional exploration of bone (FEB): 1) Interosseous pressure measurement (IOP)—a manometer is used to measure the pressure in the intratrochanteric region and femoral head. Resting pressure should be about 20 mm of mercury and greater than 30 mm of mercury is considered abnormal. Five cc's of saline is then injected, and the pressure is measured again. A greater than 10 mm of mercury difference after 5 minutes is considered abnormal. 2) Venography—contrast material is injected into the proximal femur and the venographic appearance assessed. Venostasis and reflux are considered characteristic for ON. 3) Core biopsy—the material removed during coring is assessed histologically for necrosis. Ficat and later Hungerford staged lesions based on results of the FEB, symptoms, and radiographic findings (Table I). They felt that the earliest treatment is best to avoid the complications of advanced disease, and recommend core decompression if the IOP or venography is abnormal[13,20,21]. Ficat has reported 94 percent good or excellent results in Stage I, and 82 percent good or excellent results in Stage II[13].

Ficat's early reports spawned great enthusiasm; however, several surgeons have reported extreme variability of both the success rate and complication rate of this procedure. Hungerford and Zizic reported on both alcoholic patients and patients with lupus treated with core decompression. They strongly supported the use of the FEB as the diagnostic procedure for ON and had results very similar to those of Ficat for halting progression of treated hips. Warner, et al. found that core decompression "prevented" collapse of femoral heads in 15 of 24 patients with Stage I and Stage IIA (which they called sclerotic predominant)[56]. They were unable to prevent progression in lesions beyond Stage II, and they failed to demonstrate a reliable association between elevated IOP, abnormal stress tests, and venography with ON. This and other reports have limited the enthusiasm for using core decompression beyond Stage II. Tooke, et al. reported that core decompression prevented progression of Stage I hips in all of 10 cases, and 15 of 26 Stage II hips with a short term follow-up[53]. They found much worse results if patients continued on steroids.

In the only known study to directly compare nonoperative and operative treatment (core decompression), Stulberg, et al. found a significantly better outcome in Ficat Stage II and III hips that underwent coring than prolonged protective weight-bearing[50]. This study is important for several reasons: 1) Patients were prospectively randomized to either nonoperative or operative treatment. 2) Each stage was randomized separately to allow direct comparison for a given stage. 3) A strict protocol for the operative procedure and method of nonoperative treatment was followed. 4) Results were analyzed with regard to clinical outcome, radiographic progression, and prevention of further procedures. Stage II and III hips that were treated operatively had statistically better results in all three of these categories. The results for Stage I patients were similar, but statistical significance was not achieved. The numbers of Stage 0 and Stage IV patients were not enough to allow statistical validity. Unlike many other reports, no subtrochanteric fractures or other operative complications were found. Although this study provides

the best evidence of efficacy in core decompression, criticisms can be made. The diagnosis of ON was based on a diagnostic protocol set forth by the same authors using a multimodality approach. Although they state the diagnosis of preradiographic stages required two positive tests, the numbers show that the results of these tests (bone scans, MRI, biopsy, etc.) often did not agree. Secondly, radiographic progression occurred in 70 percent of Stage II and all Stage III lesions in the surgically treated group versus 57 percent of Stage II and 30 percent of Stage III lesions in the conservatively treated group. The short minimum follow-up of 18 months (average 27 months) might indicate that the core decompression only temporarily relieves symptoms and delays the ultimate progression of disease. This discrepancy of clinical versus radiographic outcomes has been noted before[13]. Recently, Hungerford reported a 13 year follow-up of patients treated by core decompression with excellent results in the early stages[57]. However, it must be kept in mind that this and most reports of success using core decompression have focused on the earlier stages when the natural history of these lesions is not truly known. In addition, many recent reports have demonstrated poor results and high complication rates using core decompression.

Camp and Colwell showed a 60 percent rate of clinical and radiographic progression of Stage I and II hips followed for an average of 18 months after core decompression[8]. They found the FEB did not add to the accuracy of conventional diagnostic imaging. They also report the highest incidence of subtrochanteric fractures, ten percent. Hopson and Siverhus also found a low success rate in treating early lesions with core decompression[19]. In their series of 21 predominantly steroid-treated patients, all but one had histologic confirmation of necrosis and only 40 percent failed to progress or require a second operation. They had only one peri-operative fracture (4.7 percent). Learmonth, et al. found that clinical or radiographic progression occurred in 34 out of 41 hips in Stage I and Stage II[24]. Finally, Seiler, et al. had similar disappointing results in Stage I and Stage II disease with an average follow-up of only 12 months[41]. They concluded, as had the previous authors, that core decompression had an unexpectedly low success rate for halting progression, and in fact may not improve the natural history of the disease whatsoever. Additionally, these studies reported a significant morbidity, specifically peri-operative fracture through the core track.

ELECTRICAL STIMULATION

Steinberg, et al. has advocated the use of electrical stimulation in combination with core decompression and cancellous bone grafting. In their most recent article, they reported significantly improved results with coring, grafting, and electrical stimulation compared with coring and grafting alone or non-surgical treatment in unmatched, nonoperated controls[46-48]. Although the majority of surgically treated hips had radiographic progression, they did not clinically deteriorate. The follow-up of only 44 months may be too short to judge long-term success. Aaron, et al. reported that electrical stimulation was superior to the natural history or core decompression for both Ficat Stage II and III. Other studies have proposed a beneficial effect of electrical stimulation, but the data is confused by combinations of procedures and a lack of controls. A controlled, longer term follow-up is needed to assess the effectiveness of electrical stimulation in halting progression of ON.

Osteotomy

Osteotomies have many potential advantages in treating ON: 1) They preserve the hip joint by removing the necrotic segment from the weight-bearing forces. 2) They induce hypervascularity and may have a role in relieving interosseous hypertension.

In a retrospective review comparing "joint preservation operations", Saito found poor results with core decompression with or without cancellous bone grafting at average follow-up of four years[39]. Results were somewhat better with osteotomies, but only for those patients with localized lesions.

D'Aubigne' et al. reported using either varus or varus-rotational osteotomies in 56 patients, achieving satisfactory results in 47[11]. These authors recommended an osteotomy for the younger patient with minimal or no collapse and lesions "without marked extension". Maistrelli, et al. reported 106 osteotomies followed for a mean of 8.2 years (81 valgus and 25 varus), achieving a 58 percent good or excellent rating[27]. Better results were found in younger non-alcoholic patients with early necrosis that was limited in size. Sugioka devised and popularized the transtrochanteric rotational osteotomy that rotates the femoral head up to 90° (usually anteriorly)[51]. He found minimal complications and excellent clinical results. Other authors have found the technique very demanding, with high complication rates and only fair results[29,39,54].

Some general comments can be made concerning osteotomies for treating ON. The lesion must be small and rotated out of the weight bearing area. This treatment should be reserved for younger patients; and results have been fair to good with a tendency to deteriorate with time.[17]

Vascularized Bone Grafting

Meyers has reported excellent results in treating patients with Marcus Stage I and Stage II ON using a vascularized quadratus femoris muscle pedicle bone graft from the posterior femur[31,32]. Poor results followed

treated of Stage III or greater ON. Lee and Rehmatullah have reported on ten patients treated with a similar procedure. Seventy percent of patients with Marcus I and II stages had good results[26]. A long postoperative recovery period is required after this extensive procedure. Long-term follow-up will be necessary to determine if a vascularized graft offers an advantage over structural or cancellous grafting, or core decompression alone.

From the previous discussion, it seems clear that no surgical procedure is completely satisfactory in the treatment of ON. Core decompression has become the most popular and controversial procedure, but its current role in early treatment is uncertain due to the disparity of reported results. Structural bone, cancellous bone, and vascularized bone grafting procedures are generally advocated by those who report their use but confirmatory studies are lacking. Osteotomies have limited indications, and at best fair success. Total joint arthroplasty is indicated for advanced symptomatic lesions, but should be avoided as long as possible in young patients.

SUMMARY

Despite many investigations into ON of the femoral head, many issues remain unresolved. The pathogenesis in most cases is only speculative and may involve intravascular factors such as microemboli or extravascular factors such as increased interosseous pressure. MRI has emerged as the diagnostic test of choice for suspected early lesions, and radiographs should be used to diagnose and follow advanced lesions. Bone scanning can be useful for early diagnosis and CT scanning or tomography may help plan surgical procedures. The role of the functional exploration of bone is controversial. The natural history of early lesions is unknown; this makes it difficult to evaluate results of treatment. Radiographic ON will usually progress to collapse and arthrosis if treated nonoperatively. The role of core decompression or other joint preserving operations to prevent collapse is controversial, since the reports of success and complication rates have been extremely variable. These procedures are ineffective if used after radiographic collapse. Total hip replacement is the only satisfactory treatment for advanced symptomatic stages, but is relatively contraindicated in young active patients.

Further research is needed to assess the natural history of early ON and evaluate the role of surgery in preventing progression.

BIBLIOGRAPHY

1. Aaron, R., et al.: The Conservative Treatment of Osteonecrosis of the Femoral Head. Clin. Ortho. 249:209-218.

2. Andresen, J., Nielsen, H.: Osteonecrosis and Spontaneous Fractures Following Renal Transplantation. Acta Ortho. Scand. 52:475-479.

3. Arlot, M., et al.: Bone Histology in Adults with Aseptic Necrosis. JBJS 65A:1319-1327.

4. Bassett, L., et al.: MRI in the Early Diagnosis of Ischemic Necrosis of the Femoral Head. Clin. Ortho. 214:237-248.

5. Beltran, J., et al.: Femoral Head AVN: MR Imaging with Clinical-Pathological and Radionuclide Correlation. Radiology 166:215-220.

6. Boettcher, W., et al.: Non-Traumatic Necrosis of the Femoral Head. JBJS 52A:312-329.

7. Buckley, P., et al.: Structural Bone-Grafting for Early Atraumatic AVN of the Femoral Head. JBJS 73A:1367-1374.

8. Camp, J. and Colwell, C.: Core Decompression of the Femoral Head for Osteonecrosis. JBJS 68A:1313-1319.

9. Coleman, B., et al.: Radiologically Negative AVN: Detection with MRI: Radiology 168:525-528.

Cruess, R., et al.: The Etiology of Steroid Induced Avascular Necrosis of Bone. Clin. Ortho. 113:178-183.

11. D'Aubigne', R., et al.: Idiopathic Necrosis of the Femoral Head in Adults. JBJS 47B:612-633.

12. Elmstedt, E.: Avascular Bone Necrosis in the Renal Transplant Patient. Clin. Ortho. 158:149-157.

13. Ficat, R.: Idiopathic Bone Necrosis of the Femoral Head. JBJS 67B:3-9.

14. Fisher, D., et al.: Corticosteroid Induced Aseptic Necrosis. Clin. Ortho. 84:200-206.

15. Genet, B., et al.: Early Osteonecrosis of the Femoral Head: Detection in High Risk Patients with MR Imaging. Radiology 168:521-524.

16. Glimcher, M. and Kenzora, J.: The Biology of Osteonecrosis of the Human Femoral Head and Its Clinical Implications. Clin. Ortho. 139:283-312.

17. Gottschalk, F.: Indication and Results of Intertrochanteric Osteotomy in Osteonecrosis of the Femoral Head. Clin. Ortho. 249:219-222.

18. Hauzeur, J., et al.: The Diagnostic Value of MRI in Non-Traumatic Osteonecrosis of the Femoral Head. JBJS 71A:641-649.

19. Hopson, C. and Siverhus, S.: Ischemic Necrosis of the Femoral Head. JBJS 70A:1048-1051.

20. Hungerford, D. and Zizic, T.: Alcoholism Associated Ischemic Necrosis of the Femoral Head. Clin. Ortho. 130:144-153.

21. Hungerford, D. and Zizic, T.: The Treatment of Ischemic Necrosis of Bone in SLE. Medicine 59:143-149.

[22.] Kerboul, M., et al.: The Conservative Surgical Treatment of Idiopathic Aseptic Necrosis of the Femoral Head. JBJS 56B:291-296.
[23.] Kopecky, K., et al.: Apparent AVN of the Hip: Appearance and Spontaneous Resolution of MR Findings in Renal Allograft Recipients. Radiology 179:523-527.
[24.] Learmonth, I., et al.: Core Decompression for Early Atraumatic Osteonecrosis of the Femoral Head. JBJS 72B:387-390.
[25.] Lee, C., et al.: The "Silent Hip" of Idiopathic Ischemic Necrosis of the Femoral Head in Adults. JBJS 62A:795-800.
[26.] Lee, C. and Rehmetullah, N.: Muscle-Pedicle Bone Graft and Cancellous Bone Graft for the "Silent Hip" of Idiopathic Ischemic Necrosis of the Femoral Head in Adults. Clin. Ortho. 158:185-194.
[27.] Maistrelli, G., et al.: Osteonecrosis of the Hip Treated by Intertrochanteric Osteotomy. JBJS 70B:761-766.
[28.] Marcus, N., et al.: The Silent Hip in Idiopathic Aseptic Necrosis. JBJS 55A:1351-1368.
[29.] Masuda, T.: Results of Transtrochanteric Rotational Osteotomy for Osteonecrosis of the Femoral Head. Clin. Ortho. :69-74.
[30.] McCarthy, E.: Aseptic Necrosis of Bone. Clin. Ortho. 168:216-221.
[31.] Meyers, M.: Osteonecrosis of the Femoral Head. Clin. Ortho. 231:57-61.
[32.] Meyers, M.: Osteonecrosis of the Femoral Head Treated with the Muscle Pedicle Graft. OCNA 16, Vol.4:741-745.
[33.] Mitchell, M., et al.: Avascular Necrosis of the Hip: Comparison of MR, CT and Scintigraphy. AJR 147:67-71.
[34.] Mitchell, M., et al.: Femoral Head AVN: Correlation of MR Imaging, Radiographic Staging, Radionuclide Imaging, and Clinical Findings. Radiology 162:709-715.
[35.] Musso, J., et al.: Results of Conservative Management of Osteonecrosis of the Femoral Head. Clin. Ortho. 207:209-215.
[36.] Nelson, L. and Clark, C.: Efficacy of Phemister Bone Grafting in Non-Traumatic Aseptic Necrosis of the Femoral Head. Accepted for the Journal of Arthroplasty.
[37.] Phemister, D.: Treatment of the Necrotic Femoral Head in Adults. JBJS 31A:56-66.
[38.] Robinson, H., et al.: Evaluation of MRI in the Diagnosis of Osteonecrosis of the Femoral Head. JBJS 71A:650-663.
[39.] Saito, S., et al.: Joint-Preserving Operations for Idiopathic AVN of the Femoral Head. JBJS 70B:78-84.
[40.] Saito, S., et al.: Minimal Osteonecrosis as a Segmental Infarct within the Femoral Head. Clin. Ortho. 231:35-50.
[41.] Sieler, J., et al.: Correlation of Findings of MRI with Those of Bone Biopsy in Patients Who Have stage-I or II Ischemic Necrosis of the Femoral Head. JBJS 71A:28-32.
[42.] Smith, K., et al.: Non-Traumatic Necrosis of the Femoral Head Treated with Tibial Bone Grafting. JBJS 62A:845-847.
[43.] Spencer, J.D., et al.: Early Avascular Necrosis of the Femoral Head. JBJS 68B:414-417.
[44.] Spencer, J.D. and Brooks, M.: Avascular Necrosis and the Blood Supply of the Femoral Head. Clin. Ortho. 235:127-139.
[45.] Springfield, D. and Enneking, W.: Surgery for Aseptic Necrosis of the Femoral Head. Clin. Ortho. 130:175-185.
[46.] Steinberg, M., et al.: Treatment of AVN of the Femoral Head by a Combination of Bone Grafting, Decompression and Electrical Stimulation. Clin. Ortho. 186:137-153.
[47.] Steinberg, M., et al.: Osteonecrosis of the Femoral Head. Clin. Ortho. 249:199-209.
[48.] Steinberg, M., et al.: Early Results in the Treatment of AVN of the Femoral Head with Electrical Stimulation. OCNA 15:163-175.
[49.] Stulberg, B., et al.: Multimodality Approach to Osteonecrosis of the Femoral Head. Clin. Ortho. 240:181-193.
[50.] Stulberg, B., et al.: Osteonecrosis of the Femoral Head. Clin. Ortho. 268:140-151.
[51.] Sugioka, Y.: Transtrochanteric Anterior Rotational Osteotomy of the Femoral Head in the Treatment of Osteonecrosis Affecting the Hip. Clin. Ortho. 130:191-201.
[52.] Thickman, D., et al.: MRI of AVN of the Femoral Head. Skel. Radiology 15:133-140.
[53.] Tooke, S., et al.: Results of Core Decompression for Femoral Head Osteonecrosis. Clin. Ortho. 228:99-104.
[54.] Tooke, S., et al.: Results of Transtrochanteric Rotational Osteotomy for Femoral Head Osteonecrosis. Clin. Ortho. 224:150-157.
[55.] Totty, W., et al.: MRI of the Normal and Ischemic Femoral Head. AJR 140:1273-1280.
[56.] Warner, J., et al.: Studies of Non-Traumatic Osteonecrosis. Clin. Ortho. 225:104-127.
[57.] Jinnah, R., Bhatia, D., Hungerford, D.: Long-Term Results of Core Decompression of the Hip: A Prospective Study. Presented at the 60th Annual Meeting of the AAOS, February, 1993.

Joyce Cahalan

From: Rachel Wilkins
Sent: Wednesday, April 22, 2020 2:39 PM
To: Joyce Cahalan
Subject: RE: Publications

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From: Joyce Cahalan <jcahalan@towsonortho.com>
Sent: Wednesday, April 22, 2020 2:26 PM
To: Rachel Wilkins <rwilkins@towsonortho.com>
Subject: RE: Publications

We got this!

From: Rachel Wilkins <rwilkins@towsonortho.com>
Sent: Wednesday, April 22, 2020 2:20 PM
To: Joyce Cahalan <jcahalan@towsonortho.com>
Subject: Publications

PUBLICATIONS

1. Mulliken, BD: *"Osteonecrosis of the Femoral Head. Current Concepts and Controversies."* The Iowa Orthopaedic Journal, Volume 13, pp. 160-166.

2. Mulliken, BD; Bourne, RB: *"Advantages and Disadvantages of Cemented Primary Total Hip Arthroplasty."* Current Opinions in Orthopaedics, 1994, pp. 11-15.

3. Mulliken, BD; Renfrew, DL; Brand, RA; Whitten, CF: *"The Prevalence and Natural History of Early Osteonecrosis of the Femoral Head."* The Iowa Orthopaedic Journal, Volume 14, pp. 115-119.

4. Mulliken, BD; Renfrew, DL; Brand, RA: *"The Prevalence of Previously Undetected Osteonecrosis of the Femoral Head in Renal Transplant Recipients."* Radiology, Volume 192, pp. 831-834.

5. Mulliken, BD; Rorabeck, CH; Bourne, RB; Nayak, NK: *"The Surgical Approach to Total Hip Arthroplasty. Complications and Utility of a Modified Direct Lateral Approach."* The Iowa Orthopaedic Journal, Volume 15, pp. 48-61.

6. Nayak, NK; Bourne, RD; Rorabeck, CH; Mulliken, BD; Robinson, EJ: *"Techniques for Exposure of the Stiff Total Knee."* The Knee, Volume 2, No. 4, pp. 189-194.

7. Mulliken, BD; Nayak, NK; Bourne, RB; Rorabeck, CH; Bullas, R: *"Early Radiographic Results Comparing Cemented and Cementless Total Hip Arthroplasty."* Journal of Arthroplasty, Volume 11, No. 1, pp. 24-33.

8. Nayak, NK; Mulliken, BD; Rorabeck, CH; Bourne, RB; Robinson, EJ: *"Osteolysis in Cemented Versus Cementless Acetabular Components."* Journal of Arthroplasty, Volume 11, No. 2, pp. 135-140.

9. Robinson, E; Mulliken, BD: *"Catastrophic Osteolysis in Total Knee Replacement."* Clinical Orthopaedics, December 1995.

10. Mulliken, BD; Rorabeck, CH; Bourne, RB: *"Uncemented Revision Total Hip Arthroplasty: A 4 to 6-Year Review."* Clinical Orthopaedics and Related Research, No. 325, pp. 156-162.

11. Mulliken, BD; Bourne, RB; Rorabeck, CH; Nayak, NK: *"The Results of the Cementless Mallory-Head Primary Total Hip Arthroplasty: A 5 to 7-year Review."* The Iowa Orthopaedic Journal, Volume 16, pp. 20-34.

12. Mulliken, BD; Bourne, RB; Rorabeck, CH; Nayak, NK: *"A Tapered Titanium Femoral Stem Inserted Without Cement in Total Hip Arthroplasty: Radiographic Evaluation and Stability."* JBJS, 78A, pp. 1214-1225.

13. Nayak, NK; Rorabeck, CH; Bourne, RB; Mulliken, BD; Robinson, EJ: *"Interpretation of Radiologists of Orthopaedic Total Joint Radiographs: Is It Necessary or Cost-Effective?"* The Canadian Journal of Surgery, Volume 39, No. 5, pp.393-396.

14. Rorabeck, CH; Bourne, RB; Mulliken, BD; Nayak, NK; Laupacis, A; Tugwell, P; Feeney, D: *"The Nicholas Andry Award. Comparative Results of Cemented and Cementless Total Hip Arthroplasty."* Clinical Orthopaedics and Related Research, No. 325, pp. 330-344.

15. Mulliken, BD; Rorabeck, CH; Bourne, RB: *"Total Hip Arthroplasty in the Management of CDH in Adults."* Orthopaedics Today, August 1997, pp. 18-21.

16. Mulliken, BD; Rorabeck, CH; Bourne, RB; Nayak, NK: *"A Modified Direct Lateral Approach in Total Hip Arthroplasty: A Comprehensive Review."* Journal of Arthroplasty, Volume 13, pp. 737-747.

17. Nayak, NK; Mulliken, BD; Rorabeck, CH; Bourne, RB; Woolfrey, MR: *"The Prevalence of Heterotopic Ossification in Patients with Osteoarthritis: A Randomized Clinical Trial."* The Canadian Journal of Surgery, No., 40, Volume 5, pp. 368-374.

18. Kelley TC, Adams MJ, Mulliken BD, Dalury DF. Efficacy of Multimodal Perioperative Analgesia Protocol With Periarticular Medication Injection in Total Knee Arthroplasty:A Randomized, Double-Blinded Study. Journal of Arthroplasty. 2013;28(8):1274-1277.

EXPOSURE PAPER

Dalury, DF; Mulliken, BD: *"A Prospective, Randomized Study Comparing Extensor Mechanism Management Exposures for Minimally Invasive Total Knee Arthroplasty."*