Revascularization of the Femoral Head in Osteonecrosis

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Abstract

Osteonecrosis of the femoral head accounts for as many as 18% of total hip arthroplasties performed in western countries. The young age of affected patients and the potentially poor outcome have sparked an interest in alternative treatment modalities. Extracapsular placement of a vascularized fibular graft in the subchondral region of the femoral head has shown great promise as a treatment option. The authors have used this procedure in the treatment of 646 symptomatic hips, of all grades of osteonecrosis, with a follow-up of 1 to 17 years. The resultant 10-year survival rate of greater than 80% suggests that this procedure may be preferable to total hip arthroplasty for the young patient with osteonecrosis of the femoral head.

J Am Acad Orthop Surg 1998;6:44-54

Osteonecrosis of the femoral head in the young patient, and the resultant debilitation affecting both daily activities and ability to meet occupational demands, is a growing concern. It has been estimated that 5% to 18% of total hip arthroplasties in western countries are performed for the primary diagnosis of osteonecrosis.[1-3] The incidence in developing countries with a high prevalence of sickle cell trait is higher; radiographic findings of osteonecrosis are seen in up to 50% of patients with sickle cell trait.[4]

A large proportion of patients with osteonecrosis are less than 50 years of age. Over the past 17 years, the average age of the 550 patients (822 hips) treated at our institution for osteonecrosis was 33 years. Many options for the treatment of osteonecrosis have been reported. However, the results have been less than ideal. No salvage method has been clearly shown to reliably halt the radiographic or clinical progression of osteonecrosis. Continuing concerns about cement disease, particulate matter, and stress-shielding changes in the femur make total hip arthroplasty a less desirable option for the younger patient (less than 50 years of age).

Early reports on the use of the free vascularized fibular graft have shown some promising results in select patients.[5-7] The success seen in these patients depends on careful attention to detail in the establishment of a patent vascular bone graft, both as a structural support of the weak subchondral area of the femoral head and as a source of osteoprogenitor cells and nutrients through the inherent vascular pedicle.

Natural History

It has been clearly established that the symptomatic hip with osteonecrosis warrants surgical treatment. The symptomatic hip almost uniformly progresses to further collapse without treatment; a collapse rate of more than 85% in the symptomatic hip has been reported, even if the patient was first seen in the earliest stages of the disease.[8-11]

The treatment plan for the asymptomatic, or "silent," hip is more controversial. It has been our experience that approximately 67% of silent hips will go on to collapse. We reviewed 56 asymptomatic hips with radiologic evidence of atraumatic osteonecrosis. Stage 2 or 3 osteonecrosis progressed in 36 of 49 patients, while stage 1 osteonecrosis progressed in only 2 of 7 patients. Of particular note, 95% of femoral heads with more than 50% involvement on biplane radiographs had collapsed by the time of follow-up. This finding of impending collapse in asymptomatic hips is also supported by Bradway and Morrey,[12] who found that all of a

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group of 15 presymptomatic hips eventually collapsed.

The earlier a treatment regimen is initiated, the better the outcome will be. The proponents of core decompression emphasize its benefits in the very early stages of osteonecrosis.[13-15] However, core decompression in the early stages often fails to halt the progression of disease.[8,16,17] The results of treatment of more advanced osteonecrosis have been mixed as well. Inconsistent results for the treatment of all stages of osteonecrosis probably reflect the lack of understanding of the natural history and prognostic indicators of the disease. For example, there is growing evidence that the size and location of the lesion in the femoral head may be as important as the stage in predicting the outcome for patients with osteonecrosis.[18,19]

Diagnosis

The patient is evaluated primarily with anteroposterior and frog-leg lateral plain radiographs. Magnetic resonance (MR) imaging can also be performed, but it is not absolutely necessary. Bone scanning and bone biopsy are not standard diagnostic tests; however, bone scanning is the most cost-effective test for assessing the status of multiple joints for the presence of osteonecrosis.

Pathognomonic changes of advancing osteonecrosis are a crescent sign and/or a wedge-shaped defect on a plain radiograph (best visualized on the frog-leg lateral view) or a serpiginous double line on a T2-weighted MR image. Other radiologic findings, clinical symptoms, and the history must be evaluated within the context of the individual case. The size and location of the lesion are also of value. The osteonecrosis is classified according to a modified Marcus-Enneking grading system[20] (Table 1).

Table 1
Staging of Osteonecrosis (After Marcus et al[20])

Stage 1	Normal radiograph; abnormal MR image or bone scan
Stage 2	Abnormal density or lucency within the femoral head
Stage 3	Subchondral fracture (crescent sign) without flattening of the head
Stage 4	Flattening of the femoral head with a normal joint space
Stage 5	Narrowing of joint space; loss of articular cartilage of the femoral head
Stage 6	Arthrosis involving both the femoral and acetabular sides of the joint

Etiology

In osteonecrosis, dead trabecular bone replaces the cancellous bone of the normal femoral head. Death of the bone elements and marrow in the femoral head can result from interruption of the vascular supply due to cyclical insults or one large crisis. The necrotic area can also include the osteochondral plate below the cartilage. The subchondral regions of bone in the body are particularly prone to osteonecrosis, which might be due to the microarchitecture of the blood vessels at these locations. There are no collateral vessels for the arterioles at these sites, which are isolated from the rest of the circulation by a cartilage boundary. Any deficiency in blood supply will not be accommodated due to the lack of another vascular supply.

The edema associated with cell death and the normal reparative process causes further damage by increasing local compartmental pressure and decreasing vascular ingress. Repeated mechanical insult without the inherent healing mechanism of live bone results in propagated stress fractures and eventual femoral head collapse. This generally results in an anterolateral wedge-shaped area of necrosis with normal cartilage overlying the defect. Large defects do not heal spontaneously. Eventually, degenerative changes in the articular cartilage develop, and hip arthrosis occurs.

Several etiologic factors have been associated with osteonecrosis. These include trauma, interruption of the femoral-head blood supply, use of alcohol or corticosteroids, and various blood dyscrasias. Studies have implied that alcohol and steroid use may account for 90% to 100% of all nontraumatic cases.[19-22] Single boluses of steroids have not been shown to have an influence on osteonecrosis. The effects of both alcohol and corticosteroids seem to be dose-related over a longer period of usage.[23-25] Corticosteroid-induced osteonecrosis is presumably caused by interruption of the blood flow to the femoral head through intravascular coagulation and fat embolism due to alterations in lipid metabolism. Alcohol abuse is associated with hyperlipidemic states that can cause intravascular coagulation or fat emboli. Other intrinsic and extrinsic factors have also been suggested, such as clotting abnormalities, blood viscosity changes, and altered vessel structure.[4,24-28]

Certainly, there are many people in the population who fulfill the criteria for risk of development of osteonecrosis, but most never have the disease. A multifactorial model probably represents a better explanation for the disease. A combination of clotting factors and vascular abnormalities or pharmacologic influence may be needed for disease development. Of our patients, more than 50% had abnormalities in their clotting profiles, compared with 2% to 6% of the normal population. These findings suggest that a second insult due to an underlying hematologic abnormality may be necessary to initiate the development of osteonecrosis in at-risk patients.

Nonoperative Treatment

Nonoperative therapy seems to have little value in the treatment of the symptomatic hip. Mont and Hungerford[28] reviewed the nonoperative experience in the literature. Only 22% of the 819 hips in 21 pooled studies (with a relatively short average follow-up interval of 34 months) had a satisfactory result. It was noted that medial lesions had the best chance of a satisfactory outcome. There was no consensus regarding weight-bearing status or range of motion permitted.

Some authors have experimented with external application of pulsed electromagnetic fields to the hip area. Lack of control in the study design makes it difficult to assess the effectiveness of this treatment.[29]

Operative Options

Several operative approaches to the treatment of osteonecrosis have been advocated. In the early, or precollapse, stage of osteonecrosis, core decompression of the femoral head has been one of the most widely used methods of surgical management. The objective is to relieve pain by decompression and to stimulate vascular infiltration and bone regeneration. This procedure is favored by orthopaedic surgeons because it is not complex, the operative time is short, and it does not preclude subsequent arthroplasty.

There are very few prospective studies comparing core decompression with other treatments, although it has been shown by some authors to provide a reasonable clinical result.[13-15,30] Stulberg et al[14] have shown a significant ($P<0.05$) decrease in failures with core decompression. However, their patients all had precollapse-stage hip disease; therefore, the value of treatment of higher grades of osteonecrosis with decompression cannot be extrapolated from these data. Several other studies have questioned the efficacy of decompression,[8,16,18,31] particularly in later stages of the disease, and have concluded that core decompression does not prevent collapse or give long-term relief of pain. The addition of electrical stimulation to core decompression has not been shown to increase the survival of the hip.[32,33]

Osteotomies performed by an experienced surgeon have achieved good results in stage 2 and stage 3 disease,[34-36] but this is not universally accepted.[37,38] Inherent to osteotomy design is the risk of compromising the vascular supply to the femoral head or resulting in an abductor limp due to relative shortening. Some surgeons have noted an increase in complications when revising previously osteotomized hips to total hip arthroplasty.[39]

Bone grafting of the osteonecrotic defect has been performed by many surgeons. Phemister[40] and Bonfiglio and Voke[41] used tibial cortical grafts to the femoral neck with a success rate of nearly 80%. Buckley et al[42] reported a good success rate; however, all of their patients were asymptomatic before surgery. Grafting through a cortical window in the neck or a trapdoor through the cartilage surface is another option. Small studies in certain centers have had success with these methods.[43,44] Each of these options depends on intracapsular dissection of the femoral neck or head, which further increases the risk of vascular compromise and increases the rehabilitation period for the patient.

Donor sites for vascularized bone grafts have included the ilium, the fibula, and local pedicle grafts. Leung[45] reported a 67% success rate with an iliac-crest transfer to a trough in the femoral neck. A few surgeons have had success with pedicle grafts. Meyers[46] reported a success rate of only 57%. Baksi[47] obtained good results in 93% of his patients. Other researchers have reported successful results in small groups. Yoo et al,[6] from Korea, were able to achieve a 91% success rate at a follow-up interval of 3 to 10 years. Brunelli and Brunelli[7] achieved 78% good to excellent results with a follow-up period of more than 5 years.

Many surgeons would consider total hip arthroplasty for all cases of osteonecrosis graded advanced stage 3 or above. This is certainly an option, but in the young patient it is desirable to save the patient's own hip if a good functional and relatively pain-free outcome can be predicted.

Free Vascularized Fibular Grafting

We have recently reported on a cohort of 103 consecutive hips treated with a vascularized fibular graft with a minimum follow-up of 5 years (median, 7 years).[5] We were able to delay total hip replacement by more than 5 years in 70% of hips that already had some collapse (stages 4 and 5). The procedure was successful in more than 80% of precollapse hips (stages 2 and 3).

Since this study, we have looked at our experience with 646 consecutive grafts with a follow-up of 1 to 17 years (Fig. 1). The expected survival or time to conversion to total hip arthroplasty of the vascularized fibular graft for all stages of osteonecrosis (including stage 5) was over 10 years (as determined by the Kaplan-Meier method of survivorship analysis). The overall survival in this series (all stages included) was 82.7%. There was, however, a greater failure rate for stage 5 osteonecrosis compared with all other grades. Overall, excluding hips with stage 5 disease, the failure rate was 16.1% (93/579). The median time to failure, if it occurred, was 2.3 years. These relatively long-term results suggest that free vascularized fibular grafting is a viable option for treatment of advanced osteonecrosis of the femoral head (Figs. 2 and 3).

Indications

We have established relative indications for the selection of patients at our institution. Extracapsular free vascularized fibular grafting is used for symptomatic hips with osteonecrosis that has been documented radiologically (with MR imaging or plain radiography). Patients less than 20 years of age with stage 2 through stage 5 osteonecrosis are candidates for the procedure. Patients aged less than 50 with stage 2 through stage 4 disease are also candidates.

The better results now being obtained with total hip arthroplasty in the young patient indicate that free vascularized fibular grafting should be offered to patients over 40 years of age. If the patient has advanced stage 4 disease, with involvement of more than 50% of the femoral head, and is over 40 years of age, total hip arthroplasty is recommended over use of a free vascularized fibular graft.

We currently do not operate on asymptomatic hips. Instead, we closely follow them and recommend free vascularized fibular graft placement when pain is first noted. However, as recent data suggest a high rate of collapse for treated patients, perhaps new guidelines should be formulated.

Technique

The historical complaints against using free vascularized fibular grafting are the technical demands and the time required for the procedure.[27] Careful planning, which includes patient and radiographic positioning as well as certain technical shortcuts, has overcome some of these objections. Previously, this operation required two teams working for more than 6 hours. Currently, this operation is generally performed with two surgeons and one scrub nurse, with an average operating time of less than 3 hours.

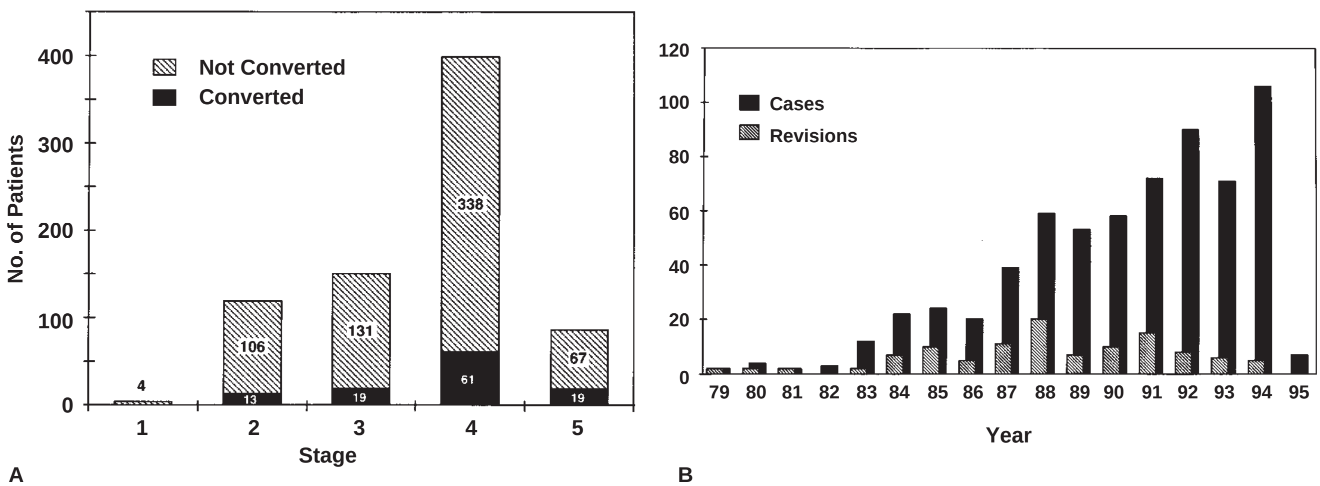


Fig. 1 Free vascularized fibular grafting was performed in 646 hips with symptomatic osteonecrosis of the femoral head with a 1- to 17-year follow-up. **A,** The total number of hips in each stage is shown in the hatched area, and the number of hips converted to hip arthroplasty is shown in the black area. (Reproduced with permission from Coogan PG, Urbaniak JR: Multicenter experience with free vascularized fibular grafts for osteonecrosis of the femoral head, in Urbaniak JR, Jones JP Jr [eds]: *Osteonecrosis: Etiology, Diagnosis, and Treatment.* Rosemont, Ill: American Academy of Orthopaedic Surgeons, 1997, p 330.) **B,** The number of procedures each year and those converted to total hip arthroplasty.

A B

Fig. 2 Anteroposterior radiographs of the hip of a 28-year-old man who had stage 3 disease. **A,** Preoperative film. **B,** Image obtained 10 years after the operation. The patient was free of pain.

We obtained an arteriogram on all of the initial 400 patients to confirm that they had three vessels (anterior tibial, posterior tibial, and peroneal) distal to the trifurcation of the femoral vessel. We discovered a deficiency in the vascular tree in only 2 patients. In the past few years, we have treated an additional 400 patients. In that group, if the patient had both dorsalis pedis and posterior tibial pulses by palpation or Doppler examination, an arteriogram was not obtained. If either pulse was not detected, an arteriogram was ordered. Fewer than 1% of patients required preoperative arteriography with this protocol.

Operative Procedure on the Hip

Ideally, the hip and the fibula are approached simultaneously to diminish operative time. The approach to the proximal femur is through a curved 20-cm anterolateral incision, splitting the interval between the tensor fascia lata and the gluteus medius (Fig. 4). A large self-retaining hip retractor is used throughout the procedure for the fascial retraction. The origin of the vastus lateralis is then reflected to expose the lateral femur. The fascia is firmly grasped and pulled distally. With use of a knife, the lateralis is removed in the avascular plane from the vastus ridge. Approximately 5 cm of posterior lateralis is reflected from the linea aspera. Anteriorly, the origin of the vastus intermedialis is carefully detached with a right-angle dissector and a knife to fashion a trough that provides a shorter route for the ascending vessels and eliminates tension on the anastomosis. To avoid damaging the vessels and the femoral nerve, the dissection must be halted as soon as the fat layer medial to the vastus is encountered.

The deep plane of dissection is between the rectus femoris and the vastus intermedialis. This plane is held open with claw retractors attached to three or four sides of the retractor. The donor vessels originate from the lateral circumflex vessel and course laterally between the two muscles. Three branches run forward into the wound: ascending, transverse, and descending (Fig. 5).

A B

Fig. 3 Anteroposterior radiographs of the hip of a 33-year-old man who had stage 5 disease. **A,** Preoperative film. **B,** On image obtained 10 years after the operation, the joint space appears to have increased. The patient had occasional mild pain.

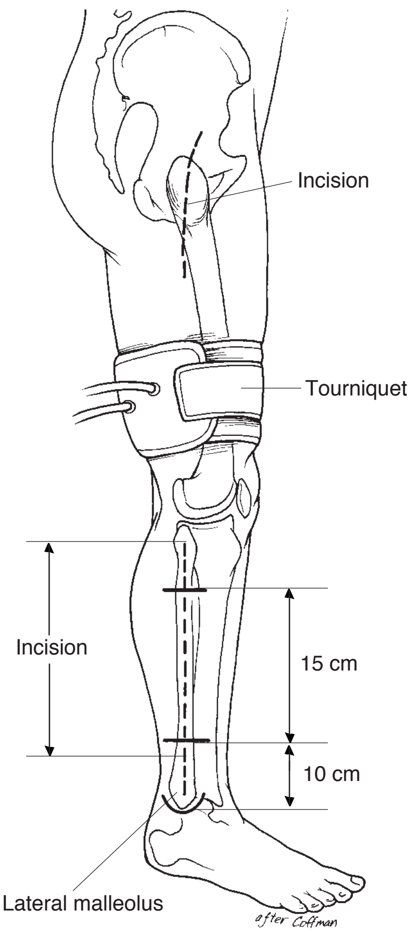


Fig. 4 The location of the incisions to approach the hip and fibula.

For better visualization, the bridge of aponeurosis between the anterolateral femur and the rectus femoris is dissected. This bridge, or falx, is detached from the anterolateral corner of the femur at the junction of the deep quadriceps muscles. The area beneath the falx is covered with a fat pad, which is swept away to reveal the ascending vessels. The artery and the two veins are visualized on the inferior (caudal) surface of the falx 8 to 10 cm distal to the anterosuperior iliac crest. They are mobilized with careful attention to length; 4 cm of length from the origin is usually easily obtained. The first major division of the ascending branch that is visualized as the vessels are approached from a superficial direction is a good starting point to isolate the vessels and provide adequate length. Small hemostatic clips are placed on any small branches before dividing to facilitate the harvesting of the vessels. A hemostatic clip is placed on the end of each of the three vessels, which are left deep in the wound for later anastomosis. All the retraction devices are then removed from the wound.

A sterile, draped C-arm fluoroscopic unit is positioned over the table. With the patient in the lateral position, the C-arm of the fluoroscopic unit is positioned so as to obtain anteroposterior and frog-leg lateral images of the femoral head and neck during the procedure. Beginning about 20 mm distal to the vastus ridge and at the junction of the middle and posterior thirds of the exposed lateral femur, a 3-mm guide pin is directed into the center of the necrotic area. Care must be taken, particularly on the lateral fluoroscopic view, that there is adequate room in the neck on both sides of the guide pin to pass a 19-mm reamer if needed. The leg can be manipulated easily for positioning between the anteroposterior and lateral views at this time because the fibular graft should have already been removed. The guide pin is oriented somewhat vertically in an effort to position the graft so as to support the subchondral area of the defect.

Cannulated reamers are progressively used over the guide pin, starting with the 10-mm reamer. The average female and male patients require a final reaming diameter of 16 mm and 19 mm, respectively. The size depends on the largest diameter of the fibular graft. The reaming extends to within 3 to 5 mm from the articular surface of the femoral head. It is safer to do the final distal reaming under fluoroscopy. The healthy bone from the reamers is saved for bone grafting. The obviously necrotic bone from the femoral head is discarded.

Once the optimal reaming size has been obtained, any additional necrotic bone is removed under fluoroscopic control with a ball reamer. The ball reamer is seldom used if the osteonecrotic lesion is 25% or less of the femoral head or if the grade is less than stage 3. It is used to remove any cyst that can be seen on fluoroscopy. Diatrizoate sodium meglumine contrast medium is instilled into the cavity to document the completeness of the removal of necrotic tissue.

Cancellous bone is obtained from the lateral femur with a large curette through the fenestration made for the reamer. During the

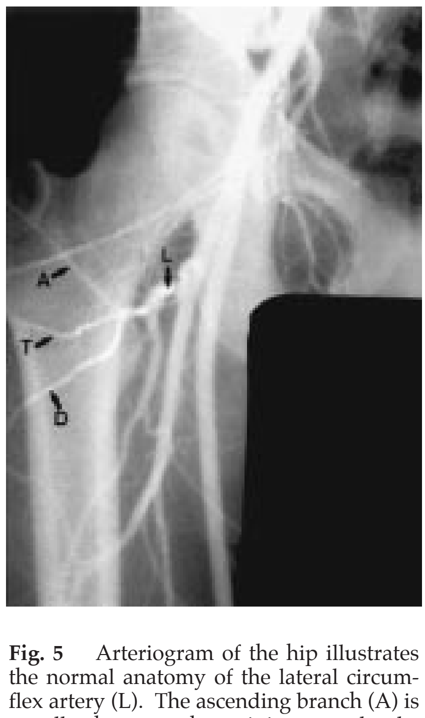


Fig. 5 Arteriogram of the hip illustrates the normal anatomy of the lateral circumflex artery (L). The ascending branch (A) is usually chosen as the recipient vessel to be anastomosed to the peroneal artery of the fibular graft. T = transverse branch; D = descending branch.

entire reaming procedure, a filtered suction tip (KAM Super Sucker [Anspach, Palm Beach Gardens, Fla]) is used to remove the loose fragments and blood from the cavity. Periodically during the procedure, the contents of the suction tip are emptied into the bone graft dish. A large amount of free bone can be procured in this manner.

Cancellous bone graft can then be placed into the cavity with the use of a custom-made impaction device (Fig. 6). A scale on the surface of the custom impacter gives the exact depth of the cavity, so that the length of the fibular graft can be determined. The impacter has side windows at the area of the subchondral bone. This window can be adjusted to place the cancellous graft in the desired position. For pediatric patients, this impacter can be fashioned from a 10-cm^3 syringe with a hole cut in the distal side. Contrast material is used to document that the cavity has been adequately impacted by the cancellous bone graft.

Operative Procedure on the Fibula

The fibula is approached through a lateral incision simultaneous to the hip approach. The surgeon stands on the posterior side. The incision is planned on a line directly over the fibula. The fibular bone cuts should be 15 cm apart, with the distal cut 10 cm proximal from the distal fibular tip (Fig. 4).

The sterile tourniquet is inflated to 300 mm Hg. The incision is extended through the skin and lateral fascia but not through the muscle-fascia layer. Self-retaining retractors are placed at either end of the wound and are repositioned as needed during the case. A scalpel is used to reflect the muscles off the lateral fibula. The periosteum should not be broached during dissection. A thin layer of muscle, 1 to 2 mm in thickness, is left on the fibula, such that there is a slight marbling effect of the muscle on the periosteum.

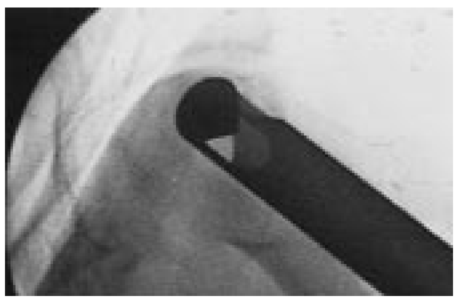
Anteriorly and posteriorly, the exposed fascia is incised with a knife. The thick fascial layer at the distal tip posteriorly is cut on the bone at this time to afford exposure of the distal pedicle. Anteriorly, a right-angle dissector is run directly on the bone, removing all muscle from the fibula. As there are no feeding vessels to the fibula on the anterior surface, there is no vascular danger with this maneuver. Deep in the wound, the interosseous membrane can be visualized and is incised along its length with a scalpel.

Posteriorly, after incision of the deep fascial layer, the flexor hallucis longus is visible. As the pedicle is directly under this muscle, care must be taken in its release. The posterior tibial bundle will also be seen and can be mistaken for the peroneal pedicle.

The fibula is cut at this stage. A right-angle clamp is used to create a tunnel where the bone is to be cut distally and proximally. It is important to stay directly on the bone. With the clamp under the fibula and the pedicle protected, a malleable ribbon retractor is placed between the clamp and the bone; from the opposite direction, a second malleable retractor is placed between the first retractor and the bone. A Gigli saw is used to cut the fibula. Care must be taken to hold the fibula in the wound during the second cut; otherwise, the vascular pedicle may be detached from the bone. A bone clamp is placed around the freed fibula to be used as a handle for dissection.

In the distal wound, the pedicle is dissected free from the muscle and divided with the application of medium hemostatic clips. There is often branching of the pedicle

A



B

Fig. 6 **A,** Custom-made cancellous bone impacter is inserted into the core in the femoral neck and head. Autogenous cancellous bone is inserted into the tube, and the motorized drill extrudes the bone out of the three portals on the left into the prepared cavity in the femoral head. The impacter is calibrated to measure the core depth for accurate sizing of the fibula. **B,** Fluoroscopic image shows the impacter in position in the prepared cavity of the femoral head.

proximal to the bone cut; if both branches are not clipped during dissection, the pedicle can be torn from the fibula.

Incising the interosseous membrane through the length of the transected fibula frees the cut fibula for mobilization, which greatly facilitates the exposure. The fibula is rotated anteriorly and posteriorly in the wound during dissection so that no perforators are missed. In a high percentage of cases, a large soleal perforator will be seen at the proximal cut; this is too large a vessel for cautery and must be clipped.

A small branch of the nerve to the flexor hallucis longus often runs with the pedicle. The sacrifice of this nerve can usually be avoided, but severance does not seem to have a clinical effect on patient outcome. The self-retaining retractors are removed, and the assistant uses two thyroid retractors—one on the posterior fascia and a second pulling anteriorly on the proximal stump of the fibula to expose the origin of the pedicle. A 5-cm-long pedicle should be freed before placing two large clips across the pedicle just distal to the bifurcation from the posterior tibial vessel. After the pedicle has been transected, the fibular graft is freed from the leg. The tourniquet is deflated with the retractors in place, and the leg is examined for bleeding. The fascial layers are left open, and the subcutaneous layer and skin are closed over a drain. With this method, the fibular harvest averages about 45 minutes at our institution. The leg is then put into a bulky dressing.

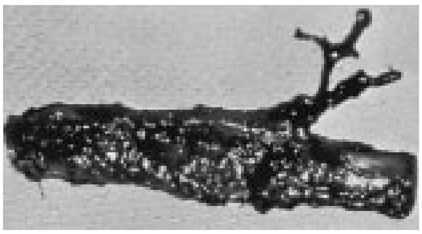


Fig. 7 The donor fibula, with peroneal artery and veins, is harvested from the ipsilateral leg.

Final preparation of the fibula occurs on the back table. Forty milliliters of heparinized lactated Ringer's solution is immediately injected into both veins and the artery of the pedicle to inspect for leaks. The pedicle is reflected from the proximal fibula until a large nutrient vessel is found entering the cortex (Fig. 7). If the pedicle is not at least 5 cm long, the bundle is dissected farther from the fibula. If the pedicle branches before 5 cm of pedicle can be mobilized, both branches are preserved with subperiosteal dissection. When the feeder vessel is located, the fibula is cut at that point with a reciprocating saw and the use of copious irrigation. The end of the pedicle is prepared with microsurgical dissection. One vein is chosen as the recipient, and a small hemostatic clip is placed on the end of the other. The final measurement from the calibrated bone impacter is used to determine the second distal bone cut. Absorbable suture is used to bind the pedicle to the distal end of the fibula in order to prevent stripping of the pedicle during insertion into the core in the femoral neck and head.

Placement of Fibular Graft Into the Femoral Head

The contrast material must be completely removed from the cavity before inserting the fibular graft, so that the final resting position of the fibula can be visualized on fluoroscopy. The fibula is inserted with the pedicle located superiorly and anteriorly on the fibula (Fig. 8). The pedicle should be positioned into the fibular sulcus for better protection from the walls of the femoral tunnel. Placement of the fibular graft at the posterior border of the core allows the pedicle to be free from compression. The fit should be snug, but not tight, to ensure that the vessels are not being compromised. The location is checked with fluoroscopy; if the graft is not seated, it is tamped into position. A 0.62-mm wire is used to hold the graft in place; it crosses both cortices of the fibula and inserts into the medial cortex of the lesser trochanter. Careful protection of the pedicle is necessary during this step. The wire is bent over after cutting. The fluoroscopic unit is then removed.

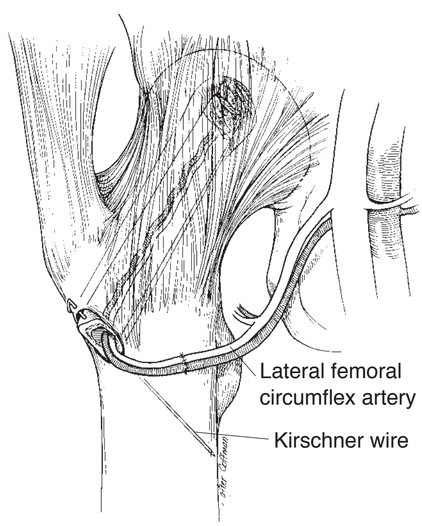
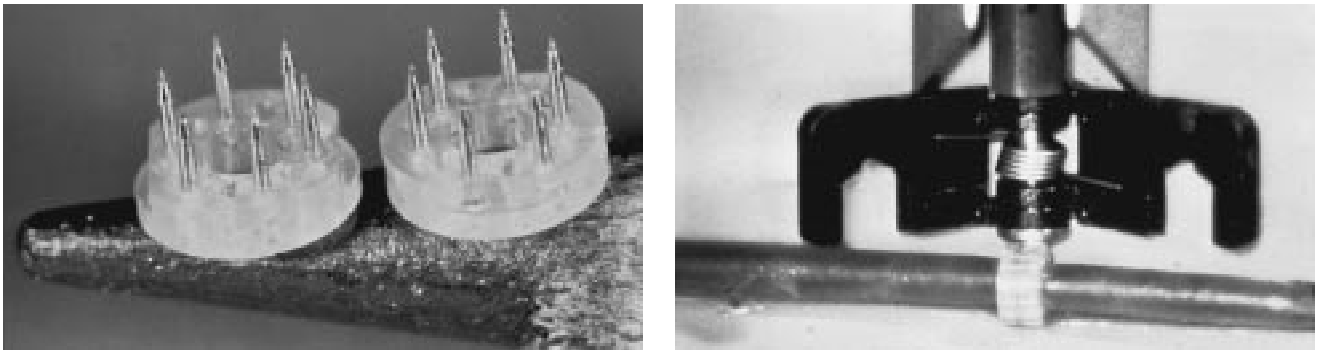


Fig. 8 Cancellous bone chips and the fibular graft are inserted into the core in the femoral neck and head and stabilized with a Kirschner wire. The peroneal vessels are anastomosed to the ascending branch of the lateral femoral circumflex artery.

Exposure of the donor vessels is optimized by appropriate placement of claw retractors attached to the four sides of the self-retaining hip retractor. The microscope is positioned to perform the anastomosis. The vein is coupled first to diminish bleeding, which could obscure the field if the artery were anastomosed first. We generally use a coupling device (Fig. 9) for



A B

Fig. 9 The vein anastomosis is usually performed with a coupler device. **A,** The free ends of the vein are pulled through the center of the plastic rings and secured over the small spikes. **B,** The rings are then coupled with an approximating device below the completed anastomosis.

the vein anastomosis to diminish operating time. The coupler may be inadequate for the artery because of the thickness and stiffness of the arterial wall. Because we have observed intimal cracking when the artery is stretched over the coupling device, we prefer to anastomose the artery with interrupted 8-0 or 9-0 nylon sutures. A disposable microsurgical suction mat of contrasting color is helpful in performing the microsurgery.

After the vessels have been anastomosed, endosteal bleeding must be observed from the fibula to document vascularization of the fibula. Postoperative angiograms have shown good blood flow in the anastomosed vessels (Fig. 10).

The tensor fascia lata is not reattached during closure to prevent compromise of the vascular pedicle. The gluteal fascia is closed over a drain.

Postoperative Care

The suction drains are removed 24 hours postoperatively, and the bulky dressing for the leg is removed on the second day after surgery. Early motion, particularly of the ankle and toes, is encouraged. Passive extension of the great toe is encouraged to avoid a flexion contracture of the toe, which sometimes occurs due to scarring of the dissected flexor hallucis longus muscle. The patient is out of bed in a chair on the first postoperative day. On the second postoperative day, non-weight-bearing ambulation is begun with crutches or a walker.

Non-weight-bearing ambulation is continued for 6 weeks, and then partial weight bearing of 20 to 25 lb is commenced, progressing to full weight bearing by 6 months after surgery. If both sides are being treated in staged procedures, the second operation is done 3 months after the first. Weight bearing is accelerated on the first side after 6 weeks of non-weight-bearing status.

Complications

We have reported on donor-site morbidity in 247 consecutive grafts in 198 patients.[48] At the 5-year follow-up, an abnormality was noted in 24% of lower limbs. A sensory deficit was found in 11.8% of limbs, and 2.7% of patients had some motor weakness. Pain at the ankle itself was a complaint in 11.5% of limbs; pain at other sites was reported by 8.9% of patients. Contracture of the flexor hallucis longus was present in 2% of patients due to the intramuscular plane used to protect the pedicle of the graft; this complication is avoidable with careful stretching of the toes in extension in the first few days after surgery.

In the 822 vascularized graft procedures we have done, there have been three thromboembolic complications. Two patients had deep venous thrombosis, which

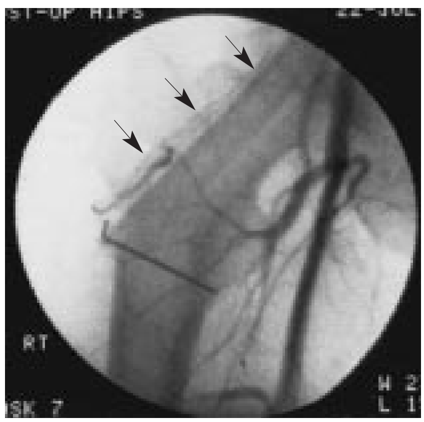


Fig. 10 A digital arteriogram obtained 5 days postoperatively shows a patent vessel (arrows) along the course of the fibular graft.

was treated successfully with anticoagulation. One patient had a massive pulmonary embolus 6 weeks postoperatively and died. Two other patients had arterial anomalies in the leg that required harvesting of the contralateral fibula in one patient and a reverse saphenous jump graft in the other. Two patients with superficial infection were treated with irrigation and antibiotics. There was ulceration along the suture line in the initial healing phase in four cases. One patient had transient paralysis in the distribution of the deep peroneal nerve. A branch of the superficial peroneal nerve was injured in another patient.

Summary

Various methods have been advocated for the treatment of osteonecrosis of the hip in the young patient. The goal should be to preserve the hip whenever possible. The literature suggests that operative treatment for the symptomatic hip is the conservative method of management. The relative efficacy of core decompression, osteotomies, electrical stimulation, and bone grafting is difficult to evaluate because there are few prospective controlled studies in the literature.

Vascularized bone grafting to the femoral head has provided the most consistently successful results. Morbidity of the donor site is minimal, and the operative time for an experienced team is comparable to that for a total hip arthroplasty. The graft entails a longer rehabilitation time and generally does not afford as complete pain relief as a total hip arthroplasty. However, when compared with a total hip arthroplasty, the vascularized fibular graft has several advantages. The femoral head is preserved, and no femoral implant is inserted. The presence of a fibular graft does not preclude later conversion to a total hip arthroplasty. Such a conversion is easier than after hip osteotomy and is certainly a much more desirable operation than an early hip revision. There is no increased risk of infection. If the procedure is done before collapse, there is a greater than 80% chance that the graft will be viable for at least 10 years.

Extracapsular free vascularized fibular grafting is the treatment of choice for osteonecrosis of stages 1 through 4 in the young patient at our institution. To date, we have reserved the procedure for symptomatic patients. Obviously, the results would be improved if we operated on asymptomatic patients. This may be justified because in our experience silent osteonecrosis of the femoral head progressed to more advanced disease in 67% of patients.

Theoretically, the graft treats many of the ongoing processes of osteonecrosis. It allows decompression of the femoral head to halt the ischemia due to increased interosseous pressure. Necrotic bone, which can inhibit healing, is removed and replaced with cancellous bone, which has osteoinductive and conductive factors. The cortical strut supplies a basis of reinforcement for the subchondral bone, and the vascular pedicle guarantees a supply of nutrients and blood to the healing femoral head. Our long-term follow-up of more than 10 years has indicated that the procedure has proved its efficacy in the treatment of osteonecrosis of the hip with no collapse, even when early collapse has preceded degenerative arthritis.

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