Avascular Necrosis of the First Metatarsal Head

A Different Perspective

ALAN S. BANKS, DPM*

Avascular necrosis of the first metatarsal head has been reported as a potential complication associated with osteotomies at the first metatarsal head for repair of hallux abducto valgus deformity. However, most if not all of the radiographic and clinical findings associated with avascular necrosis at this level may also be explained by other processes. A critical review of avascular necrosis of the first metatarsal head is presented in conjunction with a discussion of alternative etiologies for the radiographic and clinical findings that may be noted following capital osteotomies. (J Am Podiatr Med Assoc 89(9): 441-453, 1999)

Avascular or aseptic necrosis of the first metatarsal head has been reported as a complication associated with capital osteotomies of the first metatarsal. It has been generally presumed that the soft-tissue dissection required to release the deforming forces at the first metatarsophalangeal joint and to obtain clearance for the osteotomy disrupts the blood supply to the capital fragment.[1] Specifically, the release of the adductor tendon and other laterally contracted structures has been considered to cause vascular compromise.[2-5] Other authors believe that the osteotomy itself may transect the vessels supplying the blood to the metatarsal head.[3, 4] The proposed presence or absence of this complication is an integral part of many studies that evaluate the efficacy of distal metaphyseal osteotomies for the repair of hallux abducto valgus.[5-16]

The reported incidence of postoperative avascular necrosis varies greatly. This variability may be attributed, in part, to the number of clinical and radiographic findings thought to be representative of the process, including degenerative arthrosis, limited joint range of motion, subchondral cysts, and changes in radiodensity.[1-3, 6, 7, 9, 12, 17, 18] However, these findings could just as easily be the result of other factors having no relation to vascular insult of the first metatarsal head. The author proposes that there may be more reasonable explanations for these findings and that the interpretation of the images yielded by the radiographic and imaging modalities used in the past may be questioned. Moreover, other factors may influence the manner in which one views the clinical findings and imaging changes that may be encountered with distal metaphyseal osteotomies.

*Member, The Podiatry Institute, Tucker, GA; Director of Residency Training, Northlake Regional Medical Center, Tucker, GA. *Mailing address:* 2193 Northlake Pkwy, Tucker, GA 30084.

Definition of Avascular Necrosis

The anatomical site classically associated with avascular necrosis is the femoral head. Osteonecrosis at this site, with secondary involvement of the hip joint, is one of the clinically significant presentations of avascular necrosis. On examination, one may note profound morbidity because of the sequelae of the necrotic process at the femoral head. Some authors have used the more proximal hip joint as a model in attempting to explain certain findings that were noted following distal metaphyseal osteotomies.[17] Therefore, a brief discussion of the process as it occurs in the proximal femur is necessary to frame the subsequent discussion of postoperative changes seen in the foot.

The pathogenesis of avascular necrosis has been well documented.[19-23] Many conditions have been proposed as sources of osteonecrosis, including traumatic disruption of the blood supply, hemoglobinop-

athies such as sickle-cell disease, corticosteroid therapy, Cushing's disease, alcoholism, Gaucher's disease, dysbaric disorders, pregnancy, and irradiation.[22, 23] In general, each of these proposed sources of avascular necrosis features one of two pathologic mechanisms: a primary interruption of the blood supply to the femoral head, or an increase in the intramedullary pressure within the femoral head that creates venous occlusion with subsequent infarction of cellular elements of bone.

Avascular necrosis occurs as a sequence of events that can be divided into several phases. Initially, anoxia results in cell death. The surrounding viable tissue responds to the ischemic cells, leading to an increase in vascularity and inflammatory cell infiltration around the infarcted zone. As this process becomes organized, a reactive interface forms around the border of the necrotic region. At the outer margin of the ischemic zone and in the adjacent viable bone where there is adequate oxygenation to support cells, the ischemic fat cells are altered and become fibroblastic cells. There is a resultant resorption of bone and a disruption of the cancellous bone at this interface.[22] The hyperemia and inflammatory cell reaction can produce osteoporosis in the femoral head. When this happens, the infarcted zone, which remains unchanged by the hyperemia, may be slightly denser than the surrounding viable bone. The remaining vascular sites will undergo some osseous resorption. This process will continue until it ultimately involves the subchondral bone and the remainder of the infarcted zone.

Bone resorption in the subchondral region may cause a weakening of the underlying support structure for the cartilage. If sufficient weightbearing stress is applied, there may be collapse of the subchondral bone plate and depression of the articular surface. This may result in the classic subchondral fracture that is identified with avascular necrosis on conventional radiographs. This is a relatively late manifestation and is secondary to the response of the surrounding normal tissue to the necrotic zone and not necessarily a consequence of the infarction itself. In most cases, once the final phase of subchondral collapse has developed, secondary osteoarthrosis ensues.

The pathologic stages of avascular necrosis and the correlated imaging findings are listed in Table 1. For the purposes of this discussion, relative to the proximal femur, the most pertinent feature of the pathophysiology of osteonecrosis is that many changes occur in the femoral head and the underlying subchondral bone before there is any identifiable change in the joint space. From a radiographic standpoint, it is generally believed that the presence of joint-space narrowing or other osteoarthritic changes that precede the development of articular surface collapse implies that the primary process observed is osteoarthrosis or another arthritic process rather than primary osteonecrosis. Therefore, in order to make the radiographic diagnosis of primary osteonecrosis in the proximal femur with confidence, one must demonstrate changes in the femoral head prior to the development of articular-space narrowing or other evidence of osteoarthrosis.

It is certainly possible that an osteoarthrosis may progress and develop areas of necrotic bone secondary to subcapital collapse and other abnormalities that occur as a result of the arthritic process. However, if this occurs, the osteonecrosis is not the primary event leading to the abnormality. In true primary osteonecrosis, the infarction occurs first. The secondary response of the normal tissue to this necrotic zone will result in the changes previously described.

Extrapolating to other skeletal sites, it seems reasonable to follow a rather strict set of criteria for diagnosing osteonecrosis. In other words, if there are radiographic changes associated with joint-space disease such as osteoarthritis, one should be careful in attributing this finding to a primary osteonecrosis. One must demonstrate that abnormalities developed in the articular end of a bone after an initial preservation of normal joint space in order to make a diagnosis of primary avascular necrosis with confidence. Otherwise, subchondral sclerosis, subchondral cysts, and other phenomena secondary to osteoarthritis may be mistakenly described as osteonecrosis.

Unfortunately, in evaluating a possible case of osteonecrosis in the hip, if radiographs demonstrate a joint affected by end-stage arthritis, and there are no prior films available for review, it may be impossible to determine whether the primary process is osteonecrosis with secondary collapse and arthrosis or a primary osteoarthrosis with secondary subcapital collapse. In addition, because it is possible to identify pathologic necrotic bone in hips with end-stage primary osteoarthritis, the histologic findings may not help to differentiate between the two processes.

The preceding discussion emphasizes the difficulties that may be experienced with the use of standard radiographs to make a diagnosis of avascular necrosis. Patients suspected of having avascular necrosis of the proximal femur often have normal radiographic findings. Nuclear medicine and MRI studies can be helpful in these situations. The MRI findings are often the most diagnostically specific findings because there is a characteristic MR pattern that has

Table 1. Radiographic and MRI Findings of Avascular Necrosis Correlated to Histologic Pattern

Histologic Pattern	Radiographic Findings	MRI Findings
Cellular death	Normal	Probably normal
Host response and hyperemia	Patchy osteoporosis and patchy sclerosis	Classic double-line sign or other MR patterns
Emergence of reactive interface	Patchy sclerosis and osteoporosis	Classic double-line sign (note that the double-line sign is not the subchondral fracture)
Remodeling of reactive interface	Patchy sclerosis and osteoporosis	Classic double-line sign
Crescent sign and articular collapse	Classic crescent sign	Classic double-line sign
Continued collapse and secondary loss of joint space	Secondary osteoarthritis	Secondary osteoarthritis

been associated with osteonecrosis. This pattern is often called the double-line sign[24] and is considered to indicate avascular necrosis regardless of the anatomical site where it is noted. However, avascular necrosis may sometimes be associated with other MR patterns as well. Making a diagnosis of avascular necrosis in these circumstances is more difficult.

One must also consider the fact that the presence of a specific MRI finding of avascular necrosis does not mean that the patient will definitely progress to clinically significant avascular necrosis. Patients at risk for avascular necrosis may have MRI findings consistent with this diagnosis yet never become symptomatic or develop collapse of bone.

Previous Methods of Evaluating the First Metatarsal

Standard Radiographs

Mann[1] discussed three types of complications involving the first metatarsal head that are purported to be the result of avascular necrosis: complete loss of the first metatarsal head, variable collapse of the metatarsal head, or a lesser degree of damage that manifests with cystic changes. His discussion was based on a retrospective evaluation of radiographs, but there was no discussion of the natural sequence of events leading to these findings.

Meier and Kenzora[17] proposed a radiographic criterion by which avascular necrosis of the first metatarsal head could be diagnosed and classified. Avascular necrosis was described as having three stages: precollapse, collapse, and osteoarthrosis. However, if a patient developed stage 1, or the precollapse phase of avascular necrosis, this did not imply that the patient would necessarily proceed to either of the next two stages. Therefore, a variety of radiographic findings could be noted, depending on the stage of avascular necrosis at the time of evaluation.

The precollapse or first stage may be subdivided further. Stage 1A develops immediately following the osteotomy with the interruption in blood supply to part of or to the entire metatarsal head. Radiographically, this stage appears normal, but it was proposed that, if radionuclide scans were sufficiently sensitive, a "cold" area corresponding to the area of devascularized bone would be evident. Stage 1B demonstrates an increased radiodensity within the metatarsal head as the surrounding bones undergo a disuse osteopenia relative to the devascularized capital fragment. Obviously, this is the same process seen following avascular necrosis of the talus. Stage 1C evolves as the metatarsal head heals, with a progressive increase in radiodensity developing as new bone is produced on the necrotic trabeculae.

Stage 2 avascular necrosis can also be subdivided according to the degree of collapse: stage 2A may present as an early or mild collapse of the articular surface, and stage 2B represents later or severe collapse. Meier and Kenzora[17] noted that not all metatarsals with avascular necrosis would collapse, but that there could be repair and arrest at stage 1C.

The final stage of avascular necrosis manifests as early or late degenerative arthrosis of the metatarsophalangeal joint (stages 3A and 3B, respectively). The signs and symptoms were proposed to be those experienced and evident with osteoarthrosis or hallux rigidus or limitus.

Clinical Findings

Several authors have described clinical findings associated with avascular necrosis. Jahss[2] stated that painful joint-space narrowing could occur even in relatively mild cases. Meier and Kenzora[17] noted that the average loss of joint motion increased from 17° to 28° when there was radiographic evidence of avascular necrosis. Patients in stage 2 could develop significant pain, tenderness, and stiffness. As noted earlier,

patients in stage 3 present with signs and symptoms similar to those of osteoarthrosis,[17] presumably pain and limitation of motion. Thomas et al[6] found that patients with radiographic evidence of mottling of the metatarsal head at 12 to 43 months following surgery had a mean total range of motion of 51°, compared with 67.5° in patients without these radiographic findings. Other authors, however, have noted no gross clinical differences between those patients with and without postoperative radiographic changes of the first metatarsal head.[3, 7, 8]

Specialized Imaging Techniques

Technetium Scans. Meier and Kenzora[17] proposed that if bone scanning techniques were sufficiently sensitive, avascular necrosis of the first metatarsal head should be able to be identified in stage 1A. Resch et al[7] undertook a study in which 36 patients (representing 39 feet) who underwent chevron procedures were investigated with ^{99m}Tc-methylene diphosphonate scintigraphy between 2 and 9 days postoperatively. Patients were assigned to one of two groups, with the distinction being whether or not an adductor tenotomy was performed. Fixation was employed in only one case. A clear central defect was visible in four feet, only one of which had undergone adductor release. Radiographs of all patients at 1 year after surgery demonstrated no evidence of major collapse, although radiographs of some patients demonstrated minor changes in radiodensity, cysts, and radiolucency. No significant difference in radiographic appearance was observed between those feet with "hot" and "cold" scans, or between those feet with and without adductor release. Furthermore, patients reported complete satisfaction with their results in 38 of 39 instances.

Resch et al[7] concluded that circulatory disturbances may develop immediately after surgery, but with limited involvement. The authors noted that if the changes observed were due to limited circulatory disturbance, the metatarsal head seemed to adapt quickly and heal adequately, with no discomfort experienced. In addition, the authors thought that adductor tenotomy could be performed without increasing the risk of avascular necrosis. Interestingly, the authors proposed that these potential vascular changes could be due to other temporary factors such as transient synovitis, osteophytes, and capsular stretching that may accompany realignment of the metatarsal head.

Magnetic Resonance Imaging. Wilkinson et al[3] reported on the first study in which MRI was used to evaluate patients undergoing capital osteotomies. Twenty patients underwent Austin procedures with adductor release and transfer. Five additional patients who underwent modified McBride procedures were used as controls. Magnetic resonance imaging was performed between the fifth and twelfth weeks following surgery. Positive findings indicative of avascular necrosis were listed as 1) on T1-weighted images, a decrease in signal intensity interpreted as a reduction in fat content of the bone secondary to necrosis; 2) on T2-weighted images, an increase in signal intensity interpreted as an increase in water content in the first metatarsal consistent with necrosis, hyperemia, and edema; and 3) alteration in the proton-density signal in comparison with similar areas of the contralateral metatarsal head.

Findings that were believed to represent avascular necrosis were noted in 10 of the 20 feet undergoing the modified Austin procedure, but in none of the feet of the control patients. Suspicious lesions were located in the four quadrants of the metatarsal head as follows: inferolateral, 0%; inferomedial, 30%; superomedial, 20%; and superolateral, 50%.

A year after the first study, a follow-up study was conducted involving an additional 12 consecutive patients who underwent Austin bunionectomy, but without adductor release.[4] Only one patient in this study demonstrated MRI findings that were interpreted as avascular necrosis. The authors concluded that the lateral dissection required to accomplish adductor release and transfer was a major contributing factor to avascular necrosis. However, a subsequent study utilizing gadolinium-enhanced MRI revealed normal perfusion of the first metatarsal head at 6 weeks following Austin bunionectomy, despite otherwise positive MRI findings that had been previously attributed to avascular necrosis.[25]

Discussion

The preceding review was intended to provide a background against which one can critically evaluate avascular necrosis and its relationship with capital osteotomies of the first metatarsal. However, one must question whether the imaging changes noted are really due to significant interruption of vascular perfusion to the metatarsal head. If alterations in radiodensity, joint-space narrowing, limited joint range of motion, and the described MRI and scintigraphic changes can be explained by processes other than avascular necrosis, the theory of avascular necrosis and its incidence need to be reevaluated.

Avascular necrosis has become a diagnosis of default and convenience in the evaluation of changes revealed in images of the first metatarsal head fol-

lowing capital osteotomies. Clinicians have become comfortable making this diagnosis without considering other possible etiologies for the findings observed. In fact, however, there are a variety of alternative pathologies that could account just as well, if not better, for the findings associated with capital osteotomies that are widely considered to be indicative of avascular necrosis.

The basis for this discussion is the original classification provided by Meier and Kenzora in 1985.[17] Their theories and staging were well accepted and were embraced as offering an orderly explanation for a disparate set of complications and findings that could be encountered following capital osteotomies. However, in the ensuing years greater clinical experience has been attained, particularly with the Austin procedure. This has led to a better understanding of the procedure and the potential complications.

Dissection

In stage 1A avascular necrosis, it is presumed that any disruption in vascularity is unique to capital osteotomies, rather than being associated with osteotomy in general. Is there some process inherent in osteotomies in general that may temporarily interrupt blood flow between the two segments? On the surface, it is not logical that such a phenomenon would be restricted to the first metatarsal head. One might propose that the vascular supply to the first metatarsal head renders this site more susceptible to disruption of essential perfusion.

However, it is interesting to note the trend regarding the lateral release in conjunction with capital osteotomies. Initially, the adductor tenotomy was believed to be a primary influence in disruption of the blood supply to the metatarsal head[2, 17]; subsequent authors agreed.[3, 4] More recently, however, other authors have found little or no difference in the incidence of postoperative radiographic changes seen in the first metatarsal between those that did and those that did not undergo lateral release.[9, 10] In particular, it has been noted that the adductor tendon is not immediately adjacent to the primary lateral vascular structures of the first metatarsal head.[7, 11, 12] Ruch[18] has clearly demonstrated that adequate access can be achieved for the Austin osteotomy with good preservation of the soft tissue surrounding the metatarsal head.

Recent anatomical research indicates that the soft-tissue vessels supplying the first metatarsal head can be preserved during an Austin osteotomy.[11] This correlates with additional studies that have demonstrated minimal or no evidence of avascular necrosis at the time of follow-up evaluation in a large number of patients.[9, 10, 12-14] With better surgical technique and a better understanding of the proximity of the blood flow, soft-tissue dissection may not be as hazardous as some authors have suggested. However, one must still consider an alternative mechanism for the radiographic changes seen following osteotomy.

One must also account for the scintigraphic changes that have been reported following the Austin procedure. If completely accurate, these would seem to constitute more reliable evidence of some degree of vascular disruption with a capital osteotomy. In the study by Resch et al,[7] subsequent scans of the patients with imaging defects at 2 to 4 weeks postoperatively demonstrated "additional increase in activity." The authors took this to mean that any vascular insult was rapidly repaired. Under the current understanding of avascular necrosis, complete repair and revascularization at this point postoperatively would be inconsistent with this diagnosis.

It is interesting that fixation was employed in only one of the patients in the study by Resch et al.[7] The role of instability at the osteotomy site in the development of vascular disturbance has not been directly addressed by previous authors, but is certainly another area worthy of study. Also, any osteotomy that is not precise will result in the separation of segments of bone from either side of the osteotomy, thus posing the risk of necrosis.

Osteotomy and Bone Resection

Some authors have proposed that the Austin osteotomy itself may transect essential vessels supplying the metatarsal head.[3, 4] However, other investigators have noted that the cuts can be made in such a way that the dorsal and plantar blood supply to the metatarsal head is preserved.[11, 12] However, one cannot be certain what effects excessive medial resection from the metatarsal head may have on subsequent function and radiographic changes. Some authors have performed osseous resection that is sufficiently extensive that staking of the metatarsal head can be noted.[15, 17] In these instances, it seems that the aggressive bone resection would be a more likely explanation for the changes noted on postoperative radiographs than a primary bone anoxia created by osteotomy.

There is also the question of how bone responds once an overlying area has undergone decortication, as occurs following resection of the bunion. The cancellous bone of the metatarsal head is now exposed medially, and in some instances a varying degree of osseous resorption may be noted a few weeks or months following surgery. While clinical experience has not shown this to be a factor in the development

of subsequent symptoms or loss of motion, it may help to demonstrate that there may be imaging changes at the metatarsal head that are due to surgical intervention and not pathologic in nature. In fact, the bone resorption that may be seen at the medial metatarsal head may also create some of the cystic change noted on some radiographs or MRI scans. Peterson et al[12] have suggested that the resection of the medial eminence eliminates the blood supply to the medial aspect of the metatarsal head. If this is true, it could also account for some of the radiographic changes noted near this area. However, the radiographic changes associated with decortication of bone would be noted in patients undergoing repair of hallux abducto valgus deformity regardless of the type of procedure performed. Furthermore, the same scalloped appearance of the metatarsal head (Fig. 1) has also been encountered in other areas of the foot and ankle when cortical bone is resected and therefore is not likely to be due to a primary disruption of blood flow to the first metatarsal head.

Thermal Damage

If stage 1A avascular necrosis does develop, it is plausible that the vascular disruption is due to thermal damage sustained at the time of the osteotomy. However, one must presume that this factor alone would be sufficient to eliminate circulation to the metatarsal head. In other words, the blood supply provided by the soft tissues would also need to be disrupted; otherwise, nonunion would be the likely result, as opposed to avascular necrosis.

The amount of heat required to create thermal necrosis of bone is small. Eriksson and colleagues[26-28] have noted widespread tissue injury in bone that has been exposed to temperatures of 50°C for 1 min. At this temperature and exposure time, bone will not remain as functioning osseous tissue, but will be resorbed and replaced with fat cells. Exposure to temperatures of 42°C can cause vascular injury, but will not deter bone repair. Bone regeneration may be significantly impaired when bone is exposed to temperatures of 47°C for 1 min. Even very brief exposures to higher temperatures can result in more significant damage. Bone that has been exposed to a temperature of 90°C even briefly will exhibit overt necrosis.[27]

This evidence leads one to consider the effect of the amount of heat generated by a saw blade during a standard Austin osteotomy. To date, this question remains unanswered. In one study in which knee arthroplasties were performed, the median maximum temperature of the saw blade was measured at 68°C.[29] The median maximum temperatures measured in the bone at 2 and 3 mm from the cutting edge of the saw were 47°C and 42°C, respectively. In several cases, 50 to 100 mL of cool saline solution was applied to the area via a syringe as the osteotomy was performed; interestingly, this did not result in a significant reduction in the overall level of heat generated. In an earlier study using diaphyseal bone from an ox, irrigation with cooled saline solution at a rate of 600 mL/min resulted in a median maximum temperature of 42°C (±7°C). Using cooled saline solution from a syringe resulted in a median maximum temperature of 61°C (±18°C).[30]

It appears that there are several factors that may affect the temperature achieved during osteotomy with a saw blade. As already noted, cooling the saw blade by irrigating it does have some benefit; however, research in other anatomical areas indicates that, as an isolated measure, this may not be sufficient to prevent thermal damage.[29] Specific data regarding the first metatarsal are lacking. One would expect some variability in the heat generated during osteotomy with anatomical site and the type of power instrumentation and blades used.

One technique for avoiding thermal damage that may be of benefit regardless of the specifics of the procedure is the application of variable pressure to the bone as the osteotomy is performed. Wachter and Stoll[31] noted that the temperature during osteotomy steadily rose when constant pressure was applied to the saw blade. However, the maximum temperature dropped significantly—below the threshold for tissue damage—when variable pressure was applied as the cut was performed. Irrigation was used in each phase of the study. The authors proposed that changing the pressure during the procedure allowed the irrigation fluid to extend into the area of osteotomy, thereby reducing heat. It is also likely that the variable pressure allows debris to be loosened and disengaged from the teeth of the saw blade, resulting in a more efficient cut. Maximum temperatures during osteotomy are greater when debris is allowed to remain within the teeth of the saw blade.[32]

Hyperemia

On radiographs, stages 1B and 1C avascular necrosis show an increase in radiodensity of the metatarsal head. However, full evaluation of the radiodensity of the metatarsal head may be complicated by the presence of the sesamoids, especially considering the positional changes that may occur before and after surgical repair. Furthermore, one would expect some changes in radiodensity simply because an osteotomy had been performed in the area. It is possible that this

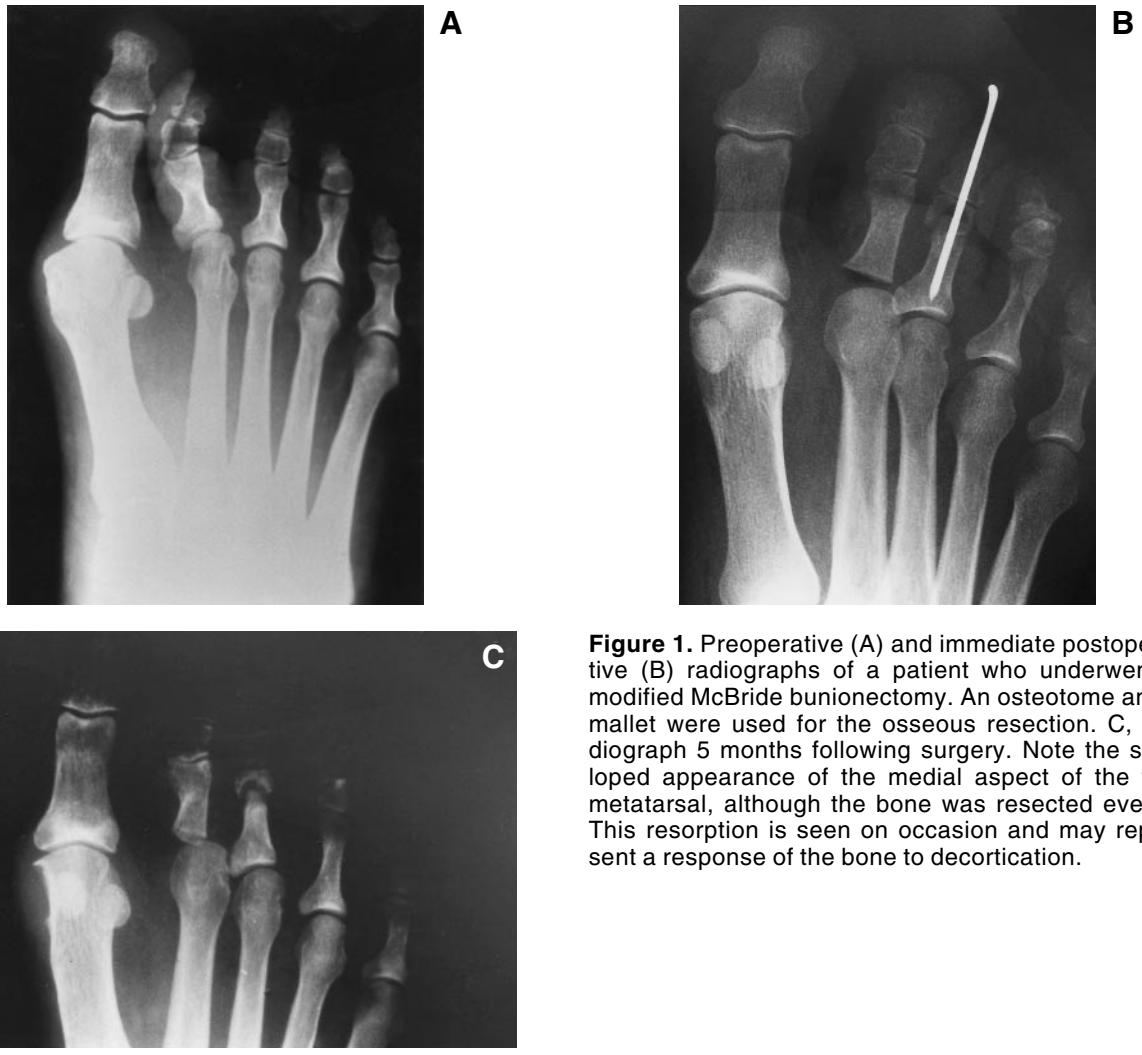

Figure 1. Preoperative (A) and immediate postoperative (B) radiographs of a patient who underwent a modified McBride bunionectomy. An osteotome and a mallet were used for the osseous resection. C, Radiograph 5 months following surgery. Note the scalloped appearance of the medial aspect of the first metatarsal, although the bone was resected evenly. This resorption is seen on occasion and may represent a response of the bone to decortication.

radiographic change purported to be pathologic may actually be a normal process associated with the osteotomy, transposition, and subsequent osseous healing, as opposed to indicating repair of vascular insult.

While vascular disruption has been emphasized as the event responsible for changes in radiodensity of the metatarsal head, there is no evidence that anyone has considered the influence of the hyperemia that follows osteotomy. As previously noted, Resch et al[7] found a significant increase in radionuclide uptake at the first metatarsal head following Austin osteotomy. Technetium scans may demonstrate increased uptake in the affected bone for 2 years or more. This author suggests that this major localized increase in blood supply, rather than anoxia, is a likely source of radiographic changes following capital osteotomies.

The findings of Thomas et al[6] are of particular interest in this regard. Crescent-shaped subchondral lucencies, "similar to Hawkins sign," were noted in 35% of the feet; these were usually noted 6 to 7 weeks postoperatively. The authors considered these lesions to indicate possible revascularization of dead bone. Follow-up examination revealed that normal bone density subsequently returned in each of these cases. However, one must question whether bone that is dead will resorb and revascularize in 6 weeks, or whether these changes are more likely indicative of a normal hyperemic response. Subchondral lucency seems more likely to represent a confirmation of vascular supply, much like the Hawkins sign following talar fractures. Therefore, these radiographic changes may represent a normal healing process.

Change in Mechanical Forces

Another potentially important influence relative to capital osteotomies is the change in mechanical forces exerted on the metatarsal head following sur-

gery. A variety of studies have demonstrated that bone density and trabecular orientations are significantly influenced by the patterns of stress to which the osseous structure is subjected.[33-37] If a hallux that has been significantly abducted for years is realigned so that the joint is in a rectus position, it should be evident that significant changes in mechanical stress applied to the metatarsal head will result. A corresponding change in bone density and trabeculation would then be accepted as the norm rather than considered a pathologic entity.

Jahss[2] believed that avascular necrosis could be seen radiographically following Keller procedures. However, with Keller procedures, one would expect an even greater change in the mechanical stress applied to the metatarsal. Again, changes in loading would be a more likely explanation for this finding than avascular necrosis. This theory is readily confirmed when one considers the preoperative radiographic appearance of the first metatarsal head in some patients with hallux abducto valgus. A reduction in radiodensity is often noted at the medial joint or metatarsal head area, providing evidence of a reduction in loading caused by the abducted hallux (Figs. 2 and 3). However, most researchers appear to ignore this fact when evaluating radiographs.

Resch et al[7] noted no signs of major collapse in 41 feet evaluated radiographically at a minimum of 1 year following surgery. Some minor differences in radiodensity, cysts, and radiolucency were found, all in patients who had normal scintigraphic scans. However, the preoperative radiographs included in their article clearly demonstrate lucency in the medial aspect of the metatarsal head and a lack of uniform density. Complete visualization of the metatarsal head postoperatively is limited because of the improved alignment of the tibial sesamoid. The inconsistent density of the metatarsal head is not a new finding subsequent to surgery, but one that is present throughout the course of treatment.

Postoperative radiographs may show a radiolucency in the medial aspect of the first metatarsal head for a long period of time. The radiographic appearance may also be altered as a result of decortication at this area with resection of the prominent bunion. One must ask what effect this cortical loss may have on the cancellous bone over time and what will be the potential influences of the soft tissues that cover the area. As discussed earlier, the newly exposed cancellous bone of the metatarsal head in some patients may undergo some degree of resorption, which may also affect the radiographic appearance to a variable degree. This process should not be confused with avascular necrosis.

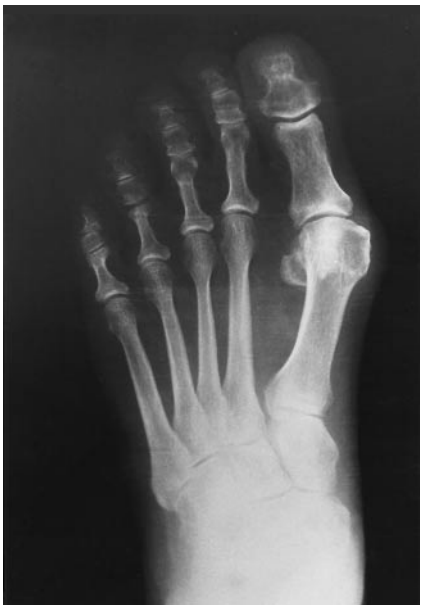

Figure 2. Radiograph of a patient with hallux abducto valgus deformity. Note the radiolucency in the first metatarsal head due to a change in the physiologic loading of the bone.

In addition to crescent-shaped subchondral lucencies, Thomas et al[6] found mottling of the metatarsal head in 50% of the feet in their study. Mottling was described as focal areas of increased bone density interspersed with focal areas of radiolucency. At the time of follow-up examination, this mottling was resolved in 32 of 40 feet. Focal radiolucencies were noted in 37 feet; in 8 cases, they were associated with the tip of the fixation pin. Some degree of lucency persisted in 13 of 29 feet at follow-up examination. Interestingly, these same authors concluded that these changes were not sufficient to prove that avascular necrosis had occurred, particularly if there was no fragmentation or collapse of the joint surface.

If radiographs do show mottling of the metatarsal head, it may be due to thermal damage sustained at the time of the osteotomy, without associated avascular necrosis. If bone tissue undergoes thermal damage, the subsequent repair will be altered. On a lateral radiographic view, one may note this as a delay in consolidation at the osteotomy site itself. Superimposition of the remaining metatarsals impedes better evaluation on this view. On the dorsoplantar radiographic view, this mottling may appear differently, evolving as a change in radiodensity within the metatarsal head. This is compounded by the orientation of the Austin osteotomy, as the dorsal and plantar wings overlap from this view. In this instance, it is

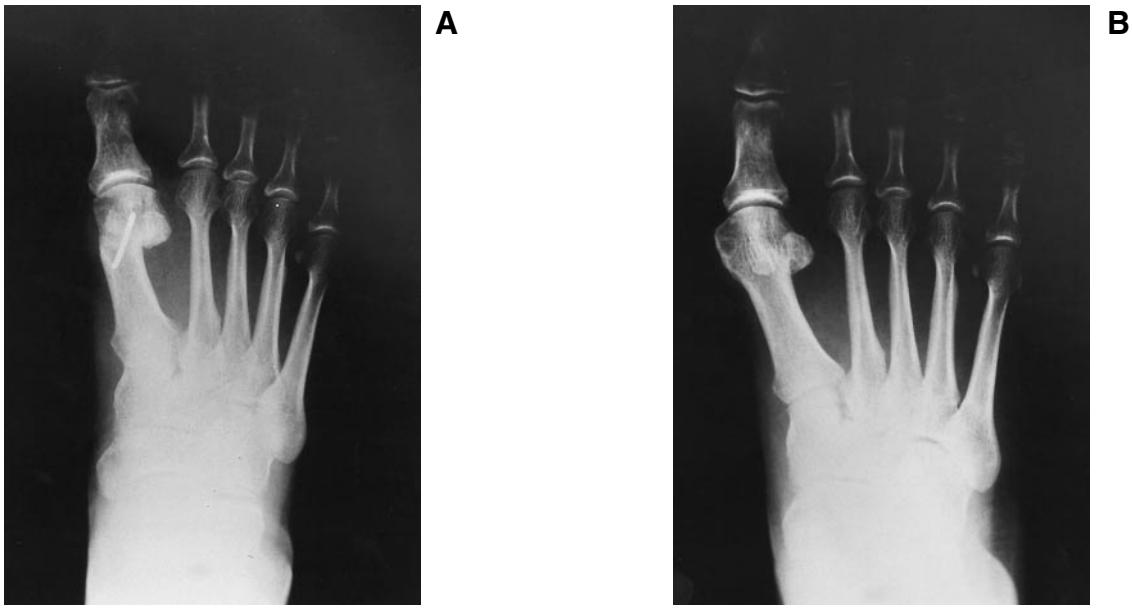

Figure 3. A, Postoperative radiograph of a patient who underwent hallux abducto valgus repair by means of an Austin osteotomy. Note the change in density of the first metatarsal head. Viewed alone, this change in density pattern would be considered representative of stage 1 avascular necrosis. However, this image was obtained immediately following surgery, before changes in bone density could occur as a consequence of the procedure. B, Radiograph of the same patient prior to surgery. Note the relative radiolucency in the first metatarsal head both medially and laterally. Also note how the lateral lucency has been somewhat obscured by the fibular sesamoid in Figure 3A. This demonstrates the importance of viewing sequential radiographs when attempting to determine whether avascular necrosis has developed.

more likely that what is being visualized is an irregularity in radiodensity.

The articular cartilage, as well as the osseous tissue, is influenced by changes in mechanical loading following surgery involving the first metatarsal head. Intraoperatively, when performing a hallux valgus repair, one may witness a loss of cartilage on the medial aspect of the joint. Reduced loading of a joint, or part of a joint, may result in a number of biochemical and histologic changes, including the advancement of the subchondral plate.[38] When one considers that hyperemia, bone remodeling, and changes in bone density are normal processes that follow capital osteotomies, the argument that the changes in radiodensity are benign and, furthermore, not representative of avascular necrosis becomes more compelling. One may wonder why radiographic changes of this nature are not routinely visualized when first metatarsal osteotomies are performed in other anatomical areas, such as in a closing base wedge osteotomy, especially if there is a similar redirection of mechanical force exerted on the metatarsal head. It would appear that the Austin and similar capital osteotomies result in a more significant localized hyperemia that may accelerate this remodeling process, as compared with a more limited bunionectomy that may accompany a base osteotomy.

Hallux Limitus

Another postoperative factor is the potential role of joint limitus in the development of subchondral cysts or degenerative changes of the first metatarsal head. Some authors have noted reduced range of motion in the first metatarsophalangeal joint following capital osteotomy, and attributed this to avascular necrosis.[2,6] However, joint surgery may result in some subsequent limitation of motion. The capital osteotomies may require more soft-tissue disruption at the metatarsal head to allow surgical access. Therefore, there may be a greater likelihood of some type of postoperative adhesions or capsulodesis, which would have the potential to limit joint mobility. Despite this fact, the loss of motion at the first metatarsophalangeal joint has been viewed in a different light.

If some surgeons hesitate to immobilize other joints following surgery for fear of loss of motion, that concern has apparently not been applied to hallux valgus repair. Joint immobilization, even without surgery, has been noted to create some changes in

joint cartilage.[39] In some studies it was noted that patients were bandaged and even maintained nonweightbearing for several weeks following Austin osteotomies.[3, 4, 15] Theoretically, this may allow some degree of capsulodesis to occur, possibly limiting later joint motion. Loss of motion can ultimately cause progressive degenerative changes in the joint, regardless of the site of osteotomy or the etiology of the hallux limitus. In one experimental study, there was a reduction of between 18% and 27% in bone density in the metaphyseal area of an immobilized rabbit limb 3 weeks postoperatively.[38]

Immobilization also reactivates the subchondral growth front, which then advances toward the joint. This may lead to overt arthritic change over time, with changes in the radiographic appearance of the joint and metatarsal head. It has been the author's experience that early postoperative range-of-motion exercises may ultimately make a significant difference in the quality and extent of motion in many patients following hallux valgus repair.

In patients with hallux limitus, the degenerative process may be accelerated by the redirection of forces at the first metatarsophalangeal joint in combination with hyperemia following surgery, particularly if the joint is not rectus. Asymmetric joint loading, particularly in an adductus position, appears to be the more destructive component in these instances (Fig. 4).

The formation of subchondral cysts in the first metatarsal head following an Austin procedure has been described by earlier authors, most of whom have indicated that this finding was a sign of avascular necrosis.[1, 6, 17] In some instances, the cystic change in the metatarsal head was demonstrated in patients with Silastic implants (Dow Corning Wright, Arlington, Tennessee) in the base of the proximal phalanx of the hallux.[2] However, subchondral cysts associated with hemi-implant arthroplasty is a well-recognized finding caused by detritic synovitis, not by vascular insult associated with surgery.[40] This is an example of how advances in knowledge have affected the understanding of the radiographic findings, but without a full reevaluation of the theory of avascular necrosis. Joint-space narrowing and associated subchondral cysts are classic findings consistent with osteoarthrosis. Osteoarthrosis may occur as a result of avascular necrosis, but collapse of the subchondral bone will occur prior to loss of joint space. Therefore, most of these cases seem to involve primary osteoarthrosis, which could easily be mitigated by restricting motion of the first metatarsophalangeal joint following surgery.

It is stage 3 avascular necrosis that generates the most controversy. Why would degenerative joint disease following capital osteotomy be attributed wholely to avascular necrosis, especially on retrospective review and without intervening radiographs? Avascular necrosis is obviously not the only possible explanation, but the literature would not lead one to believe otherwise. In most if not all reports on this topic, no other potential etiology for the degenerative change within the joint is ever mentioned. Furthermore, in most instances, no sequence of radiographs is shown demonstrating the pathologic progression of these patients. As noted in the earlier discussion of avascular necrosis of the hip, isolated radiographs viewed after joint collapse or degenerative joint change are insufficient when attempting to identify the primary etiology of the destructive process. Unfortunately, many clinicians conclude that these degenerative changes are a result of avascular necrosis on the basis of the surgical procedure performed, or on the type of surgeon who performed the procedure, rather than conducting a thorough sequential review of the radiographs.

Surprisingly, few researchers have voiced any objection to this diagnostic approach. Resch et al[7] noted that, short of a major joint collapse, all of the aforementioned radiographic findings could be accounted for in other ways, including unstable fixation, bone compaction with subsequent sclerosis, and remodeling of the metatarsal head.

Green et al[10] reviewed the radiographs of 164 feet that had undergone Austin osteotomies between 12 and 94 months previously. In six feet, the follow-up radiographs demonstrated mild-to-moderate degenerative change at the metatarsal head. Specifically, these changes consisted of irregular subchondral margins, small cysts, and asymmetric joint-space narrowing. However, comparison with preoperative radiographs revealed no significant changes. Thus surgeons must be careful in diagnosing avascular necrosis if a sequential review of radiographs cannot be done.

One might argue that the etiology of degenerative change does not matter if the consequence is the same. However, if avascular necrosis is not responsible for the change, the surgeon may be able to exert some influence on the ultimate appearance and function of the joint. If joint limitus is a potential factor in some of these clinically and radiographically observed problems, a strong case can be made for instituting early range-of-motion exercises following surgery. Different methods of handling the soft tissues at the time of surgery may also reduce subsequent adhesions. In short, if the author's propositions are correct, proactive measures may make capital osteotomies even more viable and successful.

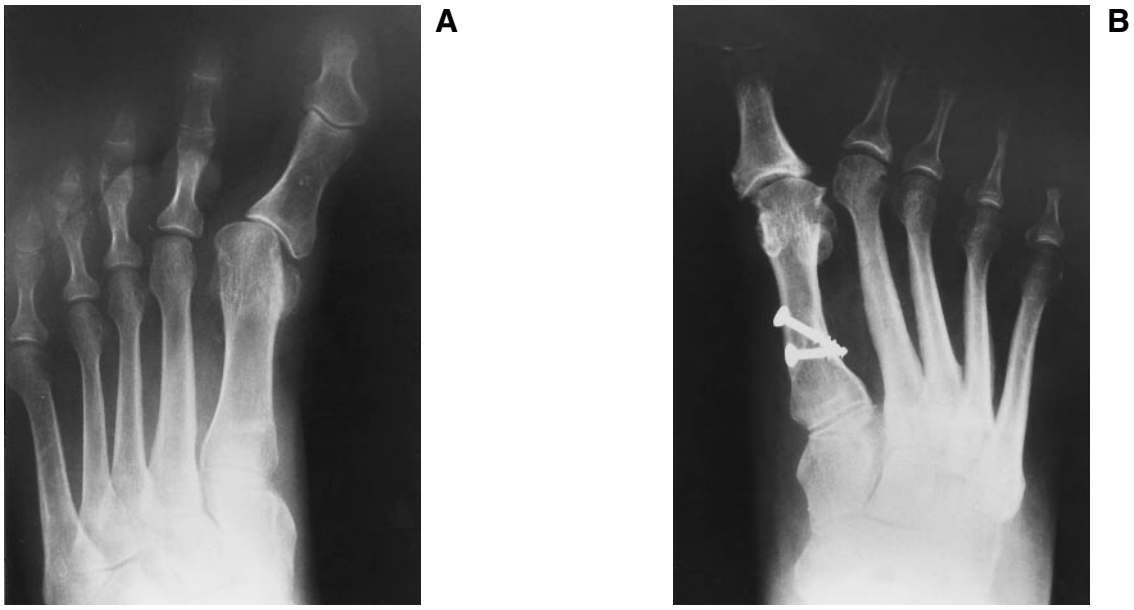

Figure 4. Radiographs of two patients with postoperative hallux varus following base wedge osteotomy. A, This patient underwent surgery several years previously and, despite significant deformity and a long first metatarsal, does not have joint symptoms or osteophyte formation. The joint is very flexible, and there is no limitation of motion. B, One-year postoperative radiograph of a second patient; early osteophytic formation may be seen laterally. The joint is stiff and painful. The primary difference between these two patients is the limitation of motion noted in the second patient. Hallux limitus may lead to degenerative changes in the joint and is one possible cause of radiographic changes seen following capital osteotomies. The limitation of motion is the primary event; it is not secondary to avascular necrosis.

Magnetic Resonance Imaging

Magnetic resonance imaging would appear to be more useful than radiography in the evaluation of possible avascular necrosis of the first metatarsal head. Initial studies using this modality revealed changes thought to represent avascular necrosis of the first metatarsal head.[3, 4] The authors of a subsequent study argued that the MRI changes noted were nonspecific and could have been related to hemorrhage, edema, and inflammation associated with the osteotomy itself.[9] This is certainly feasible, as the findings described on MRI scans could be seen in a number of different scenarios, including fractures, osteotomies, tumors, infection, reflex sympathetic dystrophy, arthritis, and neuroarthropathy, as well as avascular necrosis.

A later MRI study using gadolinium-enhanced imaging confirmed that the metatarsal head was well perfused despite the presence of cysts.[25] However, there was no attempt to explain the combination of cystic change and the absence of apparent avascular necrosis. Another critical point is that no preoperative scans were performed on any of these patients to determine what, if any, changes were present prior to surgery. Of interest is the orientation of the pin used for the internal fixation in the initial study. The Kirschner wire was directed from the dorsolateral articular surface proximally to the dorsomedial diaphysis.

Earlier the author discussed the potential adverse effects of thermal damage that could be sustained with use of a saw blade during surgery. These same thermal effects may also be seen with the insertion of wires. A study measuring the temperatures sustained when a 1.1-mm Kirschner wire is driven into the phalanges of a human finger demonstrated that the mean temperature was 118°C (±29°C).[41] This is far in excess of the heat required to inflict significant thermal damage on bone; this may explain some of the cystic changes seen on MRI scans. Confirmation of this concept may be derived from a follow-up study in which 8 of 37 lucencies noted in the first metatarsal head on standard radiographs were attributed to the tip of the fixation pin.[6]

Osteotomy Stability

The means by which osteotomy stability may affect the radiographic appearance of the first metatarsal head is uncertain. Instability has been linked to compromise of the vascular supply, but there was no ex-

planation of the mechanism involved.[7] Theoretically, if the osteotomy is not stable, one may note a greater degree of hyperemia in the area, as well as greater bone resorption, which may result in increased radiolucency in the metatarsal head. The patient may experience more symptoms and swelling during the healing phase, which may result in a reduced range of motion at the joint. However, because an osteotomy is inherently stable, and internal fixation is not mandatory for healing, it would be difficult to determine what other findings would be noted.

Conclusions

Avascular necrosis of the first metatarsal head has been discussed for years, with a variety of findings noted to be representative of the process. In fact, it is surprising to see how quickly some authors have come to the conclusion that some of the postoperative imaging changes observed constitute avascular necrosis without critically assessing whether alternative entities or processes may be responsible for these changes. Of particular interest is the general change in philosophy concerning avascular necrosis over the past decade. Early critics of the Austin procedure believed that it was risky and warned of dire consequences that would inevitably ensue.[2] Some believed that the incidence of complications, including avascular necrosis, had been understated.[1] Interestingly, one of these early critics appears to have subsequently performed the Austin procedure with favorable results, and without findings indicative of avascular necrosis.[16] As time passed, most authors concluded that even though avascular necrosis was evident to some degree in varying numbers of patients, it made no difference in the long-term clinical results.[3, 7, 8, 12]

Several different factors may account for this change in philosophy. It seems that the Austin procedure is now well accepted by the majority of clinicians who are performing hallux valgus repair. This was not always the case. Some clinicians may have been premature in their criticisms, basing them more on the opinions of their peers than on personal experience with the Austin procedure. As the utility and efficacy of the procedure became more widely known, it became more difficult for surgeons to view the findings of "avascular necrosis" so negatively.

In spite of the change in philosophy, most authors have not viewed the imaging findings as representing any process other than vascular disruption of the metatarsal head. The current author has described a number of different means by which these radiographic changes may develop that are more reasonable explanations than avascular necrosis. A significant problem is that many of the studies that have been conducted to date concentrate on postoperative radiographs and do not include a sequential review of all of the films taken throughout the patient's treatment process.

On the basis of the preceding discussion, one may consider the following proposals relative to the performance of osteotomies:

1) Judicious soft-tissue dissection will allow adequate exposure without vascular compromise at the metatarsal head. The adductor tendon can be safely released without disruption of the major vascular elements supplying the metatarsal head and without fear of avascular necrosis.

2) Reducing the heat generated at the time of the osteotomy may enhance the healing potential of the bone. Methods of heat reduction include irrigation of the saw blade and variation of the pressure applied with the saw blade during the osteotomy.

3) Early postoperative range-of-motion exercises for the first metatarsophalangeal joint may enhance motion and improve the quality of later function for some patients. The release of lateral soft-tissue contracture may also aid in providing better range of motion for the joint.

A final question remains: Does true avascular necrosis of the first metatarsal head develop following capital osteotomies? There are probably rare instances in which other mechanisms do not adequately explain the process that is witnessed clinically and radiographically. However, the true incidence of this complication is most likely extremely low.

Acknowledgment. Richard Stiles, MD, who assisted in the development of the thoughts regarding MR patterns of avascular necrosis and the classic pathology involved in this process.

References

1. Mann RA: Letter to the editor. Foot Ankle **3:** 125, 1982.
2. Jahss MH: Hallux valgus: further considerations—the first metatarsal head. Foot Ankle **2:** 1, 1981.
3. Wilkinson SV, Jones RO, Sisk LE, et al: Austin bunionectomy: postoperative MRI evaluation for avascular necrosis. J Foot Surg **31:** 469, 1992.
4. Neary MT, Jones RO, Sunshein K, et al: Avascular necrosis of the first metatarsal head following Austin osteotomy: a follow-up study. J Foot Ankle Surg **32:** 530, 1993.
5. Rossi WR, Ferreira JC: Chevron osteotomy for hallux valgus. Foot Ankle **13:** 378, 1992.
6. Thomas RL, Espinosa FJ, Richardson EG: Radiographic changes in the first metatarsal head after distal chevron osteotomy combined with lateral release through a plantar approach. Foot Ankle **15:** 285, 1994.

7. Resch S, Stenstrom A, Gustafson T: Circulatory disturbance of the first metatarsal head after Chevron osteotomy as shown by bone scintigraphy. Foot Ankle **13:** 137, 1992.
8. Horne G, Tanzer T, Ford M: Chevron osteotomy for the treatment of hallux valgus. Clin Orthop **183:** 32, 1984.
9. Pochatko DJ, Schlehr FJ, Murphey MD, et al: Distal chevron osteotomy with lateral release for treatment of hallux valgus deformity. Foot Ankle Int **15:** 457, 1994.
10. Green MA, Dorris MF, Baessler TP, et al: Avascular necrosis following distal Chevron osteotomy of the first metatarsal. J Foot Ankle Surg **32:** 617, 1993.
11. Jones KJ, Feiwell LA, Freedman EL, et al: The effect of chevron osteotomy with lateral capsular release on the blood supply to the first metatarsal head. J Bone Joint Surg Am **77:** 197, 1995.
12. Peterson DA, Zilberfarb JL, Greene MA, et al: Avascular necrosis of the first metatarsal head: incidence in distal osteotomy combined with lateral soft tissue release. Foot Ankle Int **15:** 59, 1994.
13. Loretz L, DeValentine S, Yamaguchi K: The first metatarsal bicorrectional head osteotomy (distal "L" Reverdin-Laird procedure) for correction of hallux abducto valgus: a retrospective study. J Foot Ankle Surg **32:** 554, 1993.
14. Hattrup SJ, Johnson KA: Chevron osteotomy: analysis of factors in patients' dissatisfaction. Foot Ankle **5:** 327, 1985.
15. Courtman NH, Weighill FJ: Distal first metatarsal osteotomy and adductor release as a treatment of hallux valgus. J R Coll Surg Edinb **40:** 133, 1995.
16. Mann R, Donatto K: The Chevron osteotomy: a clinical and radiographic analysis. Foot Ankle **18:** 255, 1997.
17. Meier PJ, Kenzora JE: The risks and benefits of distal first metatarsal osteotomies. Foot Ankle **6:** 7, 1985.
18. Ruch J: "Anatomical Dissection of the First Metatarsophalangeal Joint," in *Comprehensive Textbook of Foot Surgery*, ed by ED McGlamry, AS Banks, MS Downey, p 469, Williams & Wilkins, Baltimore, 1992.
19. Glimcher MJ, Kenzora JE: The biology of osteonecrosis of the human femoral head and its clinical implications: I. tissue biology. Clin Orthop **138:** 284, 1979.
20. Glimcher MJ, Kenzora JE: The biology of osteonecrosis of the human femoral head and its clinical implications: II. the pathological changes in the femoral head as an organ and in the hip joint. Clin Orthop **139:** 283, 1979.
21. Glimcher MJ, Kenzora JE: The biology of osteonecrosis of the human femoral head and its clinical implications: III. discussion of the etiology and genesis of the pathological sequelae; comments on treatment. Clin Orthop **140:** 273, 1979.
22. Sweet D, Madewell J: "Osteonecrosis: Pathogenesis," in *Diagnosis of Bone and Joint Disorders*, 3rd Ed, ed by D Resnick, p 3445, WB Saunders, Philadelphia, 1995.
23. Resnick D, Niwayama G: "Osteonecrosis: Diagnostic Techniques, Specific Situations, and Complications," in *Diagnosis of Bone and Joint Disorders*, 3rd Ed, ed by D Resnick, p 3495, WB Saunders, Philadelphia, 1995.
24. Mitchell D, Rao V, Dalinka M, et al: Femoral head avascular necrosis: correlation of MR imaging, radiographic staging, radionuclide imaging, and clinical findings. Radiology **162:** 709, 1987.
25. Chen JB, Youngberg RA, Jones RO: Enhanced magnetic resonance imaging of the first metatarsal after a bunionectomy. JAPMA **84:** 406, 1994.
26. Eriksson R: *Heat-Induced Bone Tissue Injury: An In Vivo Investigation of Heat Tolerance of Bone Tissue and Temperature Rise in the Drilling of Cortical Bone* [dissertation], Goteburg, Sweden, University of Goteburg, 1984.
27. Eriksson AR, Albrektsson T: Temperature threshold levels for heat induced bone tissue injury: a vital microscopic study in the rabbit. J Prosthet Dent **50:** 101, 1983.
28. Eriksson A, Albrektsson T, Grane B, et al: Thermal injury to bone. Int J Oral Surg **11:** 115, 1982.
29. Toksvig-Larsen S, Ryd L: Temperature elevation during knee arthroplasty. Acta Orthop Scand **60:** 439, 1989.
30. Toksvig-Larsen S, Ryd L, Lindstrand A: On the problem of heat generation in bone cutting. J Bone Joint Surg Br **73:** 13, 1991.
31. Wachter R, Stoll P: Increase of temperature during osteotomy: in vitro and in vivo investigations. Int J Oral Maxillofac Surg **20:** 245, 1991.
32. Toksvig-Larsen S, Ryd L, Lindstrand A: Temperature influence in different orthopaedic saw blades. J Arthroplasty **7:** 21, 1992.
33. Beaupre GS, Orr TE, Carter DR: An approach for time-dependent bone modeling and remodeling-application: a preliminary remodeling simulation. J Orthop Res **8:** 662, 1990.
34. Carter DR: Mechanical loading history and skeletal biology. J Biomech **20:** 1095, 1987.
35. Carter DR, Orr TE: Skeletal development and bone functional adaptation. J Bone Miner Res **7** (suppl 2)**:** S389, 1992.
36. Carter DR, Van Der Meulen MC, Beaupre GS: Mechanical factors in bone growth and development. Bone **18** (1 suppl)**:** 5S, 1996.
37. Lanyon LE: Functional strain as a determinant for bone remodeling. Calcif Tissue Int **36:** S56, 1984.
38. Smith RL, Thomas KD, Schurman DJ, et al: Rabbit knee immobilization: bone remodeling precedes cartilage degradation. J Orthop Res **10:** 88, 1992.
39. Finsterbush A, Friedman B: The effect of sensory denervation on rabbits' knee joints. J Bone Joint Surg Am **57:** 949, 1975.
40. Vanore J, O'Keefe R, Bidney M, et al: "First Metatarsophalangeal Joint Implant Arthroplasty," in *Comprehensive Textbook of Foot Surgery*, ed by ED McGlamry, AS Banks, MS Downey, p 617, Williams & Wilkins, Baltimore, 1992.
41. van Egmond DB, Hovius SE, van der Meulen JC, et al: Heat recordings at tips of Kirschner wires during drilling through human phalanges. J Hand Surg [Am] **19:** 648, 1994.