Avascular necrosis of bone: a review[1]

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'The power of reproduction which Nature possesses, displays itself in a great variety of morbid cases, but in none of them more remarkably than in a certain disease of bone termed necrosis', wrote James Russell, the first Professor of Clinical Surgery at the University of Edinburgh in 1794. This was the first description of bone necrosis which was clearly septic in origin. It was not until a century later that Axhausen (1928) realized that bone necrosis could occur in the absence of infection. The 20th century has seen an increase in the incidence of aseptic necrosis of bone in deep sea divers, tunnel workers, and in patients given corticosteroids for a variety of conditions including organ transplantation. The four most common causes of hip pain in the adult are now osteoarthritis, inflammatory arthritis, trauma, and osteonecrosis. The terms osteonecrosis, ischaemic necrosis, aseptic necrosis and avascular necrosis are synonymous.

Bone death appears to occur not as the result of a sudden occlusion or functional defect of the blood vessels to the bone or in the bone, but as a result of an imbalance between the metabolic requirements of the osteocyte and the ability of the circulation to meet those needs (Boettcher *et al.* 1970*b*).

It is now possible to diagnose avascular necrosis of bone before destructive changes become obvious radiographically, yet avascular necrosis remains a problem of management. It may affect patients who would generally be considered too young for total joint replacement, and so efforts have been made to treat avascular necrosis of bone more aggressively from an early stage in the hope of preserving joint congruity; results, however, remain unpredictable. The early diagnosis of avascular necrosis of bone is dependent upon an awareness of the conditions in which it can occur.

Conditions associated with the development of avascular necrosis of bone

Avascular necrosis of bone is known to occur in a variety of conditions, many of which are shown in Table 1. In the first four, i.e. fractured neck of femur, traumatic dislocation of the hip, slipping of the upper femoral epiphysis and following osteotomy, disruption of the blood supply to the femoral head is clearly beyond dispute. In most of the conditions described the exact cause of avascular necrosis remains uncertain, whilst in others – such as

Table 1. Conditions associated with the development of avascular necrosis of the femoral head

Fractures of the femoral neck	Radiation
Traumatic hip dislocation	Gaucher's disease
Slipped capital femoral epiphysis	Renal transplantation
Following cervical osteotomy	Caisson disease
Sickle cell anaemia	Pregnancy
Other anaemias	Tumours
Alcohol abuse	Others: Leriche syndrome
Corticosteroid administration	Coagulation deficiencies
Rheumatoid arthritis	Cretinoid epiphyseal dysgenesis
Systemic lupus erythematosus	Dyschondroplasia
Chronic pancreatitis	Charcot's arthropathy
Occlusive vascular disease	Idiopathic
Osteomyelitis	

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rheumatoid arthritis and systemic lupus erythematosus – it may be that the therapeutic regimen, such as corticosteroid administration, plays a part in bringing about avascular necrosis (Grigor *et al.* 1978).

Fractures of the neck of the femur

Damage to the retinacular vessels supplying the femoral head (Sevitt & Thompson 1965) and possibly tamponade due to bleeding into the confined joint space (Woodhouse 1964) account for the 16% incidence of late segmental collapse of the femoral head in undisplaced united fractures and a 27% incidence in displaced fractures (Barnes *et al.* 1976). The incidence increases with the degree of displacement of the head (Garden 1961); whilst there is probably no relationship with the number of manipulations or delay in treatment and open reduction, there is an increase in the incidence with extreme valgus reduction (Garden 1971, Woodhouse 1964) which may occlude the vessel of the ligamentum teres (Smith 1959). Fractures of the femoral neck remain the commonest cause of avascular necrosis of the femoral head.

Traumatic dislocation

The overall incidence of avascular necrosis is 3.2% following anterior dislocation and 13.4% following posterior dislocation (Epstein 1973). In 1962, Brav found that if the dislocated hip was reduced within two hours the incidence was 17.6%, whereas if greater delay occurred prior to reduction the incidence rose to 56.9%. Symptomatic avascular necrosis has been found to develop within two (Brav 1962) to three years (Pennsylvania Orthopaedic Society 1960).

Slipped capital femoral epiphysis

Slipping of the capital femoral epiphysis *per se* is less likely to produce avascular necrosis of the epiphysis than a forceful manipulation to reduce the slipped epiphysis (Lowe 1961). The capital epiphysis slips posteriorly and therefore does not damage the posteriorly-situated retinacular vessels, whereas reduction of the slip may do so: hence the current move towards pinning of a slipped epiphysis *in situ* (Griffith 1976). Again, the radiographic diagnosis of avascular necrosis may be made approximately six months following the slip. Acute cartilage necrosis is thought to occur in about one-third of patients with slipped upper femoral epiphyses (Maurer & Larson 1970). The incidence of avascular necrosis lies between 6 and 16% (Lowe 1961, Hall 1957). This is thought to be increased by excessive traction on the leg, an early proximal osteotomy, and forceful manipulations (Lowe 1961).

Cervical osteotomy

The more proximal the osteotomy, the greater the incidence of avascular necrosis of the femoral head, ranging from 21% for cuneiform osteotomy of the neck (Pearl *et al.* 1961) to a reported incidence of 0% following subtrochanteric osteotomies (Southwick 1973).

Sickle cell anaemia

It may be that sickling, which occurs in response to lower oxygen tensions, occurs in areas of bone such as the epiphyses where the collateral circulation is limited. It is most commonly found in the femoral head but also occurs in the humeral head and rarely in the vertebral bodies causing collapse. It occurs in all types of sickle cell disease, ranging from 4% in SS disease to 17% in SC disease (Barton & Cockshott 1962). Sickle cell trait is not known to be a factor in osteonecrosis unless associated with an abnormal form of haemoglobin (Allen & Jinkins 1978).

Other anaemias

Only two cases of avascular necrosis of the femoral head have been reported in other anaemias: one in a nine-year-old with haemolytic anaemia (Bozdech & Bozdechova 1961) and the other in aplastic anaemia (Jackson *et al.* 1962).

Alcohol abuse

Alcoholics with liver disease do develop fatty emboli (Lynch *et al.* 1959) which may concentrate in the metaphyses of proximal and distal femur and tibia (Jones *et al.* 1965). The incidence of avascular necrosis in alcoholics is uncertain, but of 268 consecutive autopsy reports on chronic alcoholics with liver disease, 77.5% had demonstrable fat emboli in the lung, brain and other tissues (Lynch *et al.* 1959). The patient does not necessarily need to be an alcoholic or have liver cirrhosis. It is possible that increased fat deposition in the femoral head reduces the intraosseous blood flow, leading to bone ischaemia and infarction. Alcoholics tend to deposit fat in a variety of areas including the femoral head and other bones (Solomon 1979).

Corticosteroid therapy

There have been two case reports of patients with Cushing's disease who developed avascular necrosis of the femoral heads, and in one patient the humeral head was also involved (Madell & Freeman 1964). Patients given steroids for conditions such as pemphigus erythema multiforme, purpura, sarcoid, rheumatic fever and eczema, which themselves do not produce avascular necrosis, have developed avascular necrosis following steroid therapy (Heimann & Freiberger 1960). Intra-articular injections of corticosteroids have been shown to predispose to Charcot's arthropathy which progressed to avascular necrosis.

Rheumatoid arthritis

Destruction of the hip joint is well known in rheumatoid arthritis and may be accelerated by the development of avascular necrosis. Edstrom (1961) found that 5% of patients with rheumatoid arthritis developed avascular necrosis of the femoral head whether or not they had received steroids. Most of the patients in his series who developed it had had rheumatoid arthritis for more than ten years. It may at times be difficult to distinguish between destruction of the joint by avascular necrosis and destruction by rheumatoid arthritis *per se*.

Systemic lupus erythematosus

Since steroids play a major role in the treatment of this disease, avascular necrosis has been attributed to the therapy rather than the disease (Grigor *et al.* 1978). However, isolated cases have been reported of patients with systemic lupus erythematosus who have never received steroids but who have developed avascular necrosis (Dubois & Crozen 1960, Siemsen *et al.* 1962). Histological studies, with the exception of those of Velayos *et al.* (1966), have shown evidence of vasculitis in the synovial tissue but not the intraosseous vessels.

Chronic pancreatitis

Gerle *et al.* (1965) described 6 cases of avascular necrosis of the femoral head, the humeral head, femoral condyles and tibial plateau, but unfortunately described no histology. All their patients were chronic alcoholics. In a series of autopsies of patients with acute and subacute pancreatitis, 10.4% showed bone marrow fat necrosis with no relationship between the presence of bone lesions and the level of serum amylase or the duration of pancreatitis (Scarpelli 1956).

Occlusive vascular disease

Isolated cases of avascular necrosis have been reported affecting various bones of the skeleton in cases of polyarthritis nodosa and advanced atherosclerosis and subacute bacterial endocarditis (Bullough *et al.* 1965). An interesting case was reported by Hirsch in 1938 of avascular necrosis of the femoral head, with histological evidence of thromboangiitis obliterans of the vessel of the ligamentum teres.

Osteomyelitis
Numerous cases are reported of avascular necrosis of the femoral head following osteomyelitis. Clearly, bone necrosis is a common occurrence in chronic bone infection.

Radiation
Vascular damage of the femoral head following radiation was first described by Ewing (1926) who reported 3 cases. Histological study of fractures of the femoral neck following radiation (Goodman & Sherman 1963) has failed to demonstrate avascular necrosis of bone, but does demonstrate marrow fibrosis and subintimal fibrosis and a reduced number of osteocytes in the bone.

Gaucher's disease
The cerebroside-containing cells infiltrate bone and frequently give rise to avascular necrosis, most commonly in the femoral head and often the femoral shafts (Arkin & Schein 1948). This usually occurs bilaterally; between 40% (Arkin & Schein 1948) and 75% (Amstutz 1973) of affected patients develop this complication. The natural history is of alternating remission and relapse.

Renal transplantation
Often multiple areas are involved by avascular necrosis of bone. Cruess *et al.* (1968) reported 9 cases of avascular necrosis of the femoral head in 27 patients who survived more than six months. Nixon *et al.* (1980) reported the incidence as 7.5% in 181 patients who had received 210 renal allografts. Patients presented at an average of 30 months after transplantation, and those who developed this complication had on average four rejection episodes compared with one rejection episode in patients who failed to develop the condition. Patients who developed avascular necrosis of bone had on average received higher total doses of steroids during the first three and six months following transplantation. Following renal transplantation patients do tend to develop vague muscular aches and weakness, but the sudden onset of severe pain in one or more joints is very suggestive of the development of avascular necrosis of bone (Nixon *et al.* 1979). Children requiring transplantation tend to have severe pretransplantation renal osteodystrophy with growth retardation and sometimes epiphyseolysis as well as osteonecrosis (Stern & Watts 1979), whilst osteonecrosis can also occur in patients on chronic haemodialysis (Bailey *et al.* 1972).

Caisson disease
The incidence of this condition rises with the number of dives. Under 300 dives the incidence is 8.6% and over 900 dives it is 30.4% (McCallum & Walder 1966). Gregg & Walder (1980) have reported its occurrence in 3.5% of North Sea divers. The history of having had 'the bends' increases the risk of its development. Diving to more than 200 metres and suffering decompression sickness on the ascent appeared to lead to an increased incidence of bone lesions to as high as 16%, but radiology did not permit diagnosis until irreversible changes had occurred, nor did it give any indication of prognosis. There are two types: one is juxta-articular and is potentially disabling; the second type occurs in the neck or shaft and is never symptomatic but is indicative of a failure of protective measures taken (Walder *et al.* 1978). Bone isotope scans taken ten years after discontinuing work in high-pressure environments can still demonstrate 'hot spots', sometimes in the absence of radiological progression of the lesion or in the presence of radiological normality, suggesting a reactive or repair process which may be prolonged and not necessary beneficial as structural failure of the joint may subsequently occur (Gregg & Walder 1981).

Compressed air workers have an 18% incidence of bone lesions of which 36% are juxta-articular and 12% in the femoral head (Medical Research Council 1971). United Kingdom commercial and naval divers have a 4% incidence of bone lesions of which 21% are juxta-articular. Japanese fishermen who dive to catch fish and who are often males over the age of 50 have an incidence of 75% where decompression methods are poor (Ohta 1974). The

greater the number of exposures, the longer the exposures, the greater the depth and the occurrence of decompression sickness, all increase the incidence of dysbaric osteonecrosis. There is no way to predict which lesions are going to remain static and which are going to progess (McCallum & Walder 1966).

Idiopathic

This was first described by Chandler (1948) as 'coronary artery disease of the hip' ('Chandler's disease'). Most of the patients described were alcoholics or had been treated with steroids in the past. The patients affected are usually middle-aged (range 45–60 years) with increasing pain in the hip. Males predominate by 4:1 and the disease is bilateral in 42–60% of cases (Mankin & Brower 1962, Patterson *et al.* 1964). It most frequently affects the femoral heads but has been described as affecting the femoral condyles also. When the area of femoral condyle involved exceeds 2.3 cm^2, the lesion tends to progress with the development of osteoarthritis (Rozing *et al.* 1980). When the hip is involved there is a sudden onset of pain in the groin, often with, initially, a normal radiograph. The other hip becomes involved in about 50% of patients within two years and hence provides excellent material for the study of the condition. Conditions associated with the development of idiopathic avascular necrosis of bone are gout and hyperuricaemia, diabetes, hyperglycaemia, minor congenital anomalies of the hip, obesity, the Leriche syndrome, coagulation deficiency, cretinoid epiphyseal dysgenesis, and dyschondroplasia. Charcot's disease and many other conditions have been described in association with avascular necrosis of bone.

Diagnosis

Idiopathic avascular necrosis of the femoral head is bilateral in 42–60% of cases (Mankin & Brower 1962, Patterson *et al.* 1964). The development of radiographic changes of avascular necrosis in the second hip often postdate the changes in the first hip by a matter of years, so making detailed study of changes in the second hip possible. A variety of techniques has been developed which allow the diagnosis of the very early stages of avascular necrosis before radiological changes become evident and well before collapse of the joint has occurred. This has redirected surgical efforts towards joint preservation in preference to joint replacement. Some patients remain asymptomatic throughout the early stages of avascular necrosis, but the majority of patients present with severe pain and limitation of movement of usually one joint and frequently the patient is known to have one or more of the associated conditions listed in Table 1. Symptoms can be difficult to interpret, since some patients with severe osteonecrosis and joint destruction may complain little of pain particularly if they are taking corticosteroids, whilst other patients – for instance those following renal transplantation – frequently complain of widespread arthralgia and muscle aches and weakness even in the absence of avascular necrosis (Calne 1968).

Radiological features of avascular necrosis of bone

It is now possible to identify four separate stages in the development of avascular necrosis. Stage 1 is asymptomatic with no radiological evidence of necrosis but can be diagnosed by osteomyelography or biopsy. Stage 2 represents early radiographic evidence of abnormal or irregular bone density with or without minimal collapse of the articular surface. A linear subchondral zone of fracturing may be evident, occurring in the dead bone approximately 2 mm from the joint surface and is often seen best on the lateral view of the hip (Doyle 1967). In stage 3, the intermediate stage, irregular bone density is obvious radiographically with collapse of the femoral head. Stage 4 is advanced, showing severe deformity of the femoral head, usually with associated secondary osteoarthritic change. The changes of patchy porosis and sclerosis and collapse may involve the whole or a segment of the femoral head.

Osteomyelography (osteomedullography)

The injection of a contast dye into the femoral head via a small drill hole laterally is

followed within 10 minutes by total clearance of the dye in a normal femoral head. If the dye is retained beyond 10 minutes there is delayed circulation to the femoral head and the diagnosis of stage 1 avascular necrosis is made (Matumoto & Mizuno 1966).

Intraosseous pressure measurements
Following the injection of dye it was noted that intraosseous pressures within the proximal femur were consistently raised three or four times above normal, suggesting intramedullary venous congestion in all 15 patients on high doses of steroids or with a history of excessive alcohol consumption, studied prospectively before radiological signs of necrosis appeared (Solomon 1979).

Radioisotope studies
Technetium-99m sulphur colloid is normally taken up in the bone marrow. Solomon (1979) noted a markedly diminished uptake in a number of patients before any radiographic signs of necrosis appeared. Numerous authors have demonstrated that an increased uptake of radioactively-labelled technetium diphosphate occurs in stage 1 avascular necrosis of bone (Nixon *et al.* 1979); this probably represents an attempt at revascularization of the dead bone.
Autoradiography: The injection into the nutrient arteries of rabbits' bones of 5 micron-diameter glass spheres to produce bone infarction, followed by the administration of technetium-99m methylene diphosphonate, shows concentration of this radioisotope in areas of new bone formation and this coincides with areas of increased uptake by bone cells (Stothard & Walder 1978).

It is therefore now possible to diagnose avascular necrosis of bone before irreversible destruction of the joint occurs. This clearly alters the whole emphasis of management of these patients in favour of a biological means of preventing joint destruction instead of waiting until destruction is severe enough to justify total joint replacement.

Pathology
With increasing age the number of viable osteocytes contained within bone lacunae diminishes. The gradual loss of osteocytes from bone with increasing age is similar to the gradual loss of neurones from cerebral tissue with age. This may contribute to the reduced healing capabilities of bone with increasing age. The development of avascular necrosis can be conveniently divided into three phases: (*a*) the pathological phase of bone infarction; (*b*) the physiological phase when the body attempts to repair the infarcted area; (*c*) the mechanical phase when collapse of the partially revascularized bone occurs.

Pathological phase
It is well known that bone can be removed from its blood supply, sterilized in the autoclave and then reintroduced into the body, and it will still act as a framework across which new bone will form. The pathological phase of osteonecrosis consists of the loss of all the osteocytes from the bone because the metabolic needs of the osteocytes are no longer met from the blood supply. It has been suggested that the blood vessels are occluded by fatty emboli in alcoholics who develop osteonecrosis. Velayos *et al.* (1966) have noted the development of vasculitis in patients with systemic lupus erythematosus who develop osteonecrosis. There is no alteration in the mechanical properties of bone simply because the lacunae are devoid of osteocytes; bone may continue to function for a matter of months or sometimes years after infarction. The pathological phase of osteonecrosis therefore does not of itself lead to fracture or collapse of the bone.

Physiological stage
This is essentially a repair process whereby dead bone is resorbed following the invasion by new vessels and the arrival of osteoclasts and leads to the laying down of woven bone, which

is mechanically weak, in apposition to the dead trabeculae. Over a period of months the woven bone is modified and re-modelled so that it becomes capable of carrying out the functional requirements imposed upon it. During the period of removal and replacement a point is reached where sufficient old, strong bone has been resorbed to weaken the bone, yet insufficient new bone has been formed and remodelled to take over its function: this is a temporary phase through which the bone must pass to be restored to its former strength. It is at this point that the bone is particularly vulnerable to deforming forces and may fracture. Two types of fracture occur following the ischaemic process. One is the linear subchondral fracture, which is propagated in dead bone at the point of greatest sheer stress where the compact subchondral plate of bone meets the spongy coarse cancellous bone. This process probably occurs because the dead bone is unable to make a cellular response to changing loads imposed on the trabecula, leading to the eventual development of a fatigue fracture. Once initiated, the fatigue fracture propagates rapidly by placing additional loads on the adjacent intact trabecula. The second type of fracture develops and propagates centrally into the head at the junction of the dead bone and the living repairing bone, where there is a difference in elastic modulae between the two areas.

Dead bone may function mechanically for months or years without gross structural failure and is weakened by the revascularization process, so one approach to treatment is to prevent revascularization by replacing the dead bone with plastic or by chemically modifying the bone matrix. This is termed a 'living endoprosthesis' since the articular cartilage covering it remains viable, nourished by the synovial fluid.

Osteonecrosis or death of bone tissue *per se* causes no change in the radiographic appearances of bone as an organ, tissue or substance. There is little substantive evidence to support the concept that ischaemia due to blood vessel wall disease or blockage of blood flow by thrombus or emboli is the underlying cause of idiopathic osteonecrosis. Even in dysbaric osteonecrosis there is little evidence to implicate emboli (fat or gaseous) as the principal cause of the presumed decrease in microvascular blood flow (Glimcher & Kenzora 1979).

It may be that the blood supply is compromised to a point where cells are just able to remain viable, but the administration of a cytotoxic agent such as ethanol or pharmacological agents such as cortisone may tip the balance to cause cell death both in bone and endothelial lining cells. The rate and extent of the repair process (which is often incomplete) may be modified by: (1) cause of cell death; (2) spatial extent of the necrosis within the femoral head; (3) the administration of drugs such as cortisone.

Mechanical phase

Most bone infarcts which occur away from the articular surface are small and relatively isolated, and even a cumulative effect is generally insufficient to result in a fracture of bone. Osteonecrosis adjacent to an articular surface is well recognized as being a major contributor to painful, crippling joint destruction. The articular surface collapses during the repair process and joint congruity is lost permanently; secondary degenerative changes then follow. The necrotic zone does not correspond to the total area of bone supplied by the lateral epiphyseal artery, which is thought to be the principal source of blood for the femoral head, nor does it conform with other arteries in the femoral head except in some cases of displaced subcapital fractures of the femoral neck.

Whether or not the articular surface collapses will be determined by (*a*) the extent of osteonecrosis; (*b*) the stresses applied to the joint which may be weight-bearing or non-weight-bearing; and (*c*) the shape of the articular surface. Clearly shear stress is concentrated on the convex side of the joint such as the femoral condyle or femoral head, whereas the sheer stresses are dissipated on the concave side of the joint such as the tibial plateau or the acetabulum. Naturally if only a small area of bone is undergoing repair, the surrounding bone will be able to provide buttress support. If a joint in the upper limb is involved it will be subjected to less stress than in the lower limb and consequently there will be less chance of collapse of the articular surface.

The surface area of the joint through which the body weight is transmitted is less at the hip than at the knee and there is greater concentration of stress at the hip joint. This stress concentration is increased by the moment of force created about the femoral head by the presence of the neck shaft angle of the femur in comparison with an almost linear transmission of force across the knee joint. Catto, however, has stated that the pathological changes of avascular necrosis are not seen in the acetabulum or the glenoid side of the joint when they are present in the femoral head or humeral head (M Catto 1982, personal communication).

Despite the fact that the necrotic zone does not correspond to the area of bone supplied by any particular vessel supplying the head, Catto has noted that more than 50% of femoral heads have some viability around the fovea following fractures of the femoral neck. The vessels of the ligamentum teres do anastomose with other vessels in many patients and revascularization can occur from the artery of the ligamentum teres. The heads which revascularize are those with a good foveal anastomosis. Despite bone death in the femoral head, the articular cartilage often remains viable although frequently detached from the femoral head. This is because articular cartilage is an avascular structure and the chondrocytes derive their nutrition largely from the circulating synovial fluid. At a later stage granulation tissue arising from the healing collapsed head can take on the characteristics of pannus and develop over the articular cartilage, eroding the cartilage in the process.

The pathogenesis of avascular necrosis can be divided into six stages: (1) sinusoidal occlusion and venous stasis; (2) marrow necrosis and partial osteocyte death; (3) frank bone necrosis and early osteoblast response (these first three stages can be diagnosed by histological biopsy of the femoral head or by measurement of an increased intramedullary pressure in the femoral head); (4) early bone repair and increased bone density (the first four stages in the pathogenesis of osteonecrosis if treated early by decompression may progress no further); (5) late repair and distortion of the femoral head; (6) subchondral fragmentation and articular breakdown. Stages 4, 5 and 6 are clearly evident on plain radiographs. In stage 5, if the subchondral necrosis is localized, an appropriately designed osteotomy might unload the threatened area.

Treatment

Idiopathic ischaemic necrosis of the femoral head in adults appears to have a natural history of relentless progression once the disease has begun (Lee *et al.* 1980). Non-surgical methods of treatment of affected femoral heads have been uniformly unsuccessful. Numerous surgical methods have been employed in an attempt to preserve the femoral head and natural function of the hip joint.

A drilling procedure was first advocated by Kleinberg (1939) and Bozsan (1941). These methods were designed to replace dead bone by channels in which revascularization could occur as far as the necrotic areas and there bring about revascularization. More recently, Hungerford & Zizic (1978) have claimed that drilling is beneficial by decompressing the high intraosseous pressure in the necrotic femoral head, thus relieving pain and also allowing the occurrence of creeping substitution to the necrotic area through the drill channels. Introduction of a peg of bone into a drill channel was first described by Albee in 1915; this method was used to obtain bony union following femoral neck fractures, and was later advocated by Scheurmann and Phemister for the treatment of post-traumatic aseptic necrosis of the femoral head (Phemister 1949). They postulated that the bone pegs, in addition to acting as channels for revascularization, acted as a mechanical support to the weakened necrotic head and subchondral bone and prevented collapse of the head.

Osteotomy in the trochanteric region was advocated by Bonfiglio & Voke (1968) and more recently by Sugioka (1978), who recommends a trans-trochanteric anterior rotational osteotomy. The osteotomy is recommended for patients with segmental collapse; it is possible to rotate a relatively uninvolved area of the femoral head into a weight-bearing position. This is usually the healthy posterior cartilage of the femoral head.

In an earlier attempt at revascularization of the femoral head, Judet (1962) described the muscle-pedicle bone graft. This procedure was modified by Meyers (1978) by detaching the insertion of quadratus femoris into the posterior intertrochanteric line with a block of bone protecting the medial femoral circumflex artery, and then inserting this vascularized bone peg into the femoral head after removal of the necrotic area, thereby supporting the cartilage surface and encouraging revascularization and creeping substitution to occur. Lee & Rehmatullah (1981) followed up 10 patients with 'the silent hip' – that is radiological stage 1 and 2 necrosis – following muscle-pedicle bone grafting, and 7 of them showed radiological signs of healing and revascularization and remained symptom free. Three patients progressed, possibly due to removal of excess bone from the head followed by subsequent collapse of the articular surface. Meyers (1978) reviewed 24 patients with muscle-pedicle graft procedures; he recommended the procedure for early asymptomatic stage 2 avascular necrosis but found poor results from stages 3 and 4, which is only to be expected. For stage 3 avascular necrosis with a collapsed segment, Meyers recommended osteochondral allografts and described an increased range of movement and reduced pain in all 5 patients on which he performed this procedure.

Judet *et al.* (1981) took this a stage further with 19 patients with avascular necrosis of the femoral head and used a free fibular graft and a microvascular technique to anastomose its nutrient vessel to the anterior circumflex vessel, placing the fibula alongside the anterior surface of the femoral neck and into the femoral head. The authors felt this procedure was suitable for young patients with advanced avascular necrosis in whom the only alternative would be total hip replacement, but in their own series it was still too early to report the long-term results. Barnes (1980) reported that bone grafting in Caisson disease was usually unsuccessful with an established lesion, but if the diagnosis could be made before joint collapse occurred, bone grafting might be of assistance. Smith *et al.* (1980) reviewed 38 patients with avascular necrosis of 56 hips managed with tibial bone grafting. They noted that the proper placing of the graft in the subchondral cortex of the anterior superior quadrant of the head was vital to this operation, and that ideally patients should be young with a good life expectancy. This rules out many of the patients with necrotic lesions of the femoral head who will die prematurely from the disease which initially caused the avascular necrosis (Boettcher *et al.* 1970*a*). Smith's ideal patient would also have minimal collapse and degenerative change and a satisfactory preoperative hip rating equal to or greater than 80 on the Iowa hip rating scale (Larson 1963).

Osteotomy

The avascular lesion in the femoral head is quite often located in the anterior lateral aspect of the head and, by rotating the head and neck anteriorly on a longitudinal axis of the neck by between 45–90°, this damaged area is removed from the weight-bearing area and replaced by intact cartilage and bone. Clearly this procedure must be done before the head collapses completely. A trans-trochanteric anterior rotation osteotomy was described in 41 patients by Sugioka (1978), who stated that it prevented progression of the collapsed segment and preserved the joint surfaces. He recommended a rotation osteotomy for patients with involvement of less than two-thirds of the diameter of the head at its junction with the neck.

Kotz (1981) reviewed 17 patients with idiopathic avascular necrosis, with an average follow up of 25 months, and found that the anterior trans-trochanteric osteotomy of Sugioka (1978), combined with a total circumcision of the capsule near the acetabular rim, was suitable for small- to medium-sized areas of necrosis provided there was no osteoarthritis present. When applied to lesions of stage 3 severity the results were poor, and when applied to extensive areas of necrosis again the results were poor. The postoperative result correlated with the size of the involved area. If the area of necrosis was greater than 120°, a joint-sparing procedure never resulted in improvements. The Sugioka method gave better results than an intertrochanteric osteotomy with muscle release, but an angle of involvement greater than 120° and stage 3 changes with established osteoarthritis were contraindications. Kotz (1981) added that this was a difficult operation.

The subtrochanteric abduction or adduction osteotomy is similarly not effective for extensive lateral lesions (d'Aubigne *et al.* 1965, Patterson *et al.* 1964). In 61 patients with 80 avascular femoral heads, the results in early necrosis revealed complete relief of pain in 17 patients and appeared to arrest further joint collapse, but there was no increase in the range of movement of the joint (Patterson *et al.* 1964). In addition, rather surprisingly, 11 patients with stage 3 and 4 necrosis derived complete or fairly complete pain relief following the subtrochanteric osteotomy, but developed a diminished range of movement. Osteotomy may have a beneficial effect on the vascularity of the femoral head.

In the treatment of spontaneous osteonecrosis at the knee, a high tibial osteotomy – often with drilling of the collapsed defect and packing with bone chips – is well described as both mechanically correcting the varus deformity and stimulating the formation of new bone in the avascular segment.

Koshino (1982) described 36 patients with avascular necrosis of the femoral condyles in 37 knees which had resulted in a varus deformity: in 23 knees surgery was performed which consisted of drilling and grafting of the avascular segment of the femoral condyle. The necrotic lesion became radiographically undetectable in 13 knees, and improved in 17. Improved results with the medial femoral condyle occurred when the valgus angle was restored to between 7° and 16°. Radiological improvement was greater when the tibial osteotomy was combined with drilling and grafting than when a tibial osteotomy was performed alone. These results are similar to those described by Rozing *et al.* (1980)

Total joint replacement

Clearly some patients present too late for joint-preserving biological procedures to be undertaken. In these instances the only surgically satisfactory procedure is total joint replacement. This is extremely effective for hip disease, and 2 patients have now been described with total hip arthroplasty following cardiac transplantation (Burton *et al.* 1978). Here the pathological changes found in the femoral heads were identical to those following renal transplantation. This suggests that the immunosuppressive regimen with high doses of corticosteroids is probably responsible, due to fatty microemboli (Jones & Sakovich 1966) or alterations in the coagulability of the blood (Danzig *et al.* 1976). Numerous series have been reported of total hip replacements in patients following renal transplantation (Murray 1973, Nixon *et al.* 1980). The results, as measured by relief of pain and restoration of some movement to the joint, certainly justify this procedure being undertaken in young patients with advanced necrosis. The operation is no more difficult in patients following transplantation than in patients with advanced rheumatoid arthritis who are on steroids. The bone is extremely soft and bleeds profusely but good results can be expected. The underlying cause of avascular necrosis of bone does not appear to interfere with the cement-bone bond.

Conclusion

The development of new investigative techniques designed to detect the very early changes of avascular necrosis of bone has stimulated interest in biologically orientated joint-sparing surgical procedures. Without surgical intervention in the very early stages of the disease, it seems that the joint is destined to collapse and then develop secondary degenerative osteoarthritis. The initial results of pedicle grafting, trochanteric osteotomy and decompression procedures, when performed in the early stages of necrosis, appear promising. Some patients will still go on to require total joint replacement at a later stage, or may present too late for a conservative surgical approach to be adopted. What remains unclear, however, is the sequence of events which lead ultimately to bone death in a wide variety of differing medical conditions; and it is only when these biochemical and cellular processes are better understood that it may be possible to adopt non-surgical measures to protect the joint.

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