Section of Physical Medicine

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President's Address

Bone Necrosis in Joint Disease

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The term 'necrosis' is used in this title to cover aseptic or avascular necrosis involving destructive changes in joints although necrosis may not be the primary pathological process. Septic necrosis, neoplastic or other infiltrations may have to be considered in making the diagnosis, and indeed biopsy is often advisable. The various conditions may be classified as in Table 1.

Idiopathic Avascular Necrosis of the Femoral Head in Adults

This condition was in recent times defined by Chandler (1936) and has been more recently reviewed by Merle d'Aubigné *et al.* (1965). It has been discussed a good deal in European (Piguet 1956) and American literature (Phemister 1934, 1942). It has been called coronary artery disease of bone.

Typically, it occurs in middle-aged men and comes on fairly suddenly. The presenting symptom is pain in the groin; at first the attacks may be episodic but later the pain tends to be continuous. The opposite hip may be involved a few months later. Initially, the X-rays may be normal but, in time, first an area of subcortical porosis and then an area of opacity appears. This affects the superior lateral aspect of the head of the femur and varies from a large segment to a small crescent. During this time the joint space remains normal so that the changes are confined to the femoral head with no evidence of joint involvement. There is a tendency to repair and small areas of bone may be reformed, but with larger areas involved the head of the femur collapses, the joint space becomes involved and the end result is in appearance difficult to distinguish from a primary osteoarthrosis (Patterson *et al.* 1964). These changes are similar whether the cause of the avascular necrosis is primary or secondary. Since there is absence of joint involvement the condition is presumed to be a vascular disorder of the head of the femur, although arterial obstruction is often not demonstrable. Vascular factors are regarded as primary, but other precipitating causes have been described, including infection, trauma (either single or repetitive), venous obstruction and congenital abnormalities (Mankin & Brower 1962).

Table 1

Bone 'necrosis' in joint disease: classification

(1) Idiopathic avascular necrosis of the femoral head in adults
Perthes' disease
(2) Secondary avascular necrosis:
Diffuse lupus erythematosus
Hæmoglobinopathies
Decompression syndrome
Endocarditis
Local trauma (fracture, surgery)
Giant cell arteritis
Pancreatitis
Gaucher's disease
Multiple injuries, burns
Pregnancy
Hypercorticism
Angiokeratoma corporis diffusum
Radiation
Steroid arthropathy
Hyperuricæmia
Alcoholism
Arthropathies in which 'necrosis' may occur
(3) Rheumatoid arthritis, psoriatic arthritis
(4) Osteoarthrosis
(5) Charcot joints

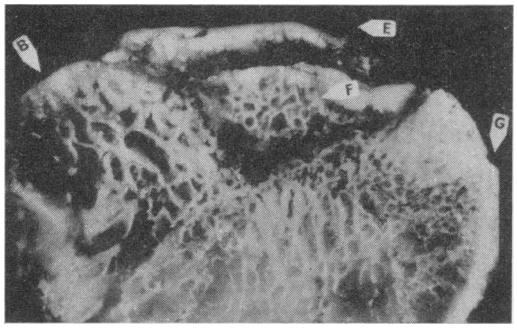

Fig 1 *Idiopathic avascular necrosis of right femoral head by ultraviolet light illumination (man aged 60).* B, *eburnated bone.* E, *detached fragment of articular cartilage, overlying* F, *detached fragment, mainly of dead bone but with a little cartilage underneath the label.* G, *edge of fibrillated osteoarthritic cartilage and eburnated reinforced bone; the thick struts under the detached fragments are also considerably reinforced*

The association with alcoholism now seems proved and this may be regarded as a systemic disorder causing avascular necrosis. Indeed, as more is learnt about avascular necrosis fewer cases will be regarded as idiopathic. The pathological changes are illustrated in Fig 1. This shows a triangular segment of bone destruction with the articular cartilage intact, but depressed into the necrosed area. On each side there is reactive bony thickening.

Secondary Avascular Necrosis

Diffuse lupus erythematosus: The underlying pathology in these cases is generally accepted as an arteritis (Dubois & Cozen 1960, Velayos *et al.* 1966). The condition is illustrated by the case of a woman aged 32 with a two-year history of pain in the shoulders and hips. She had no steroid therapy. X-rays showed typical changes of the late stages of avascular necrosis in the hips where the changes are similar to osteoarthrosis. In the right shoulder there was a radio-opaque strip overlying the area of avascular necrosis; such an appearance has also been described in decompression syndromes.

Hæmoglobinopathies: The bony changes in sickle cell disease have been described by Golding *et al.* (1959), Smith & Conley (1954), Tanaka *et al.* (1956) and Reich & Rosenberg (1953). They occur particularly in hæmoglobin S/C disease in contrast to hæmoglobin S/S disease which causes more severe systemic disturbances (Golding *et al.* 1959, Smith & Conley 1954). For example, a man aged 30 with hæmoglobin S/C disease showed radiological changes typical of this condition. He had a five-year history of pain in the right hip with a recent episode of acute pain involving the whole limb and in which localization was difficult. The X-rays showed deformity of the femoral head with preservation of the joint space. This seemed to indicate a late stage of a small lesion. In children the distortion of the femoral heads may simulate Perthes' disease and these changes may be reversible to a certain extent. The irregular patchy sclerosis described by Golding *et al.* (1959) may also affect the femoral heads and may lead to collapse.

Decompression syndromes: Nitrogen emboli seem to be the cause of avascular necrosis in this condition although some believe that intravascular thrombosis may also be responsible (McCallum & Walder 1966). The condition usually occurs on decompression from a high to a low pressure. The bone changes have been described by James (1945) and Bell *et al.* (1942). Recently Markham (1967) has drawn attention to the possibility of avascular necrosis having occurred in high flying airmen from World War II; he believed that the condition could be accounted for by exposure to a low pressure and that it may appear as a delayed effect.

Local trauma is well recognized as a cause of avascular necrosis resulting from direct interference with the blood supply to the head of the femur. The condition has been investigated by Catto (1965).

Giant cell arteritis is included in this classification as two patients in the series reported by Merle d'Aubigné *et al.* (1965) had arterial changes suggestive of temporal arteritis.

The cause of avascular necrosis in many conditions remains obscure. Jones & Engleman (1966) have reviewed many of them and they believe that fat emboli are often the underlying cause.

Pancreatitis, distant trauma, burns and pregnancy may produce effects in this way. Other views on the role of pancreatitis in the causation of necrosis have been given by Gerle *et al.* (1965) and Immelman *et al.* (1964).

Gaucher's disease may also cause fat emboli but here obstruction of vessels with histiocytes and infiltration with the abnormal cells may also occur.

Hypercorticism: One case has been described of avascular necrosis in adenoma of the suprarenal cortex (Madell & Freeman 1964). Moran (1962) has reported an abnormality in fat metabolism in Cushing's disease and it is suggested that this may give rise to fat emboli.

Angiokeratoma corporis diffusum: One case has been described in this condition (Pittelkow *et al.* 1955), presumably the result of the arteritic abnormality.

Steroid arthropathy: Jones & Engleman (1966) consider that fat embolism is the cause of avascular necrosis in steroid arthropathy. They claim to have found fat globules in the urine, and fat globules in the vessels of the femoral head in patients on steroid therapy, and Jones & Sakovich (1966) have demonstrated avascular necrosis of the femur both histologically and radiologically in rabbits injected with Lipiodol intra-arterially. They believe that this results from the disturbance of fat metabolism associated with steroid therapy (Moran 1962). Certainly, in one man who had had large doses of steroids for Behçet's syndrome, the radiological changes of the shoulders and hips were very similar to those demonstrated in other cases of avascular necrosis and support a systemic cause, such as fat emboli.

However, other views have been put forward and it is difficult to believe that local steroids exert their effect by a systemic disturbance. Velayos *et al.* (1966) studied by biopsy a small series of cases with a radiological diagnosis of avascular necrosis with steroid therapy. They found that in fact only half had pathological necrosis. In diffuse lupus erythematosus there was always arteritis and avascular necrosis, but in other steroid cases with rheumatoid arthritis there was no evidence of arteritis or large areas of avascular necrosis (although one case did have a small such area). They believe that the term steroid arthropathy should be retained until further information is available and they observe that avascular necrosis is a pathological diagnosis and that the radiological appearances may be deceptive. Other authors (Miller & Restifo 1966) have reported that bone tends to disappear at the site of injection of local steroids and the alternative view put forward is that steroids lead to a condition more related to the Charcot joint with loss of pain and inflammation in a diseased joint with osteoporosis so that collapse of the joint occurs.

Alcoholism may lead to pancreatitis and fatty changes in the liver and hence to fat emboli, but it may also cause a neuropathy and therefore lead to a type of Charcot joint; indeed, swollen crepitating painless knees are seen sometimes in alcoholics.

Hyperuricæmia: Recently McCollum *et al.* (1967), in 63 cases of 'idiopathic' avascular necrosis, found 23 instances of hyperuricæmia and therefore believe this is a possible ætiological factor.

ARTHROPATHIES IN WHICH 'NECROSIS' MAY OCCUR

Rheumatoid Arthritis

Collapsing joints have been described both with and without steroid therapy by Isdale (1962) and by Glick *et al.* (1963). These changes may be typical of avascular necrosis both radiologically and pathologically. Collapse of joints may occur, particularly in psoriatic arthritis, and this collapse may be painless. In the past psoriasis has been given as a cause of the Charcot joint.

Osteoarthrosis

Osteoarthrosis is usually regarded as a stable condition (Murray 1965) and reports of collapse of the femoral head are rare. Only Isdale (1962) has reported cases recently. It therefore seems worth while reporting a series of 15 cases in which collapse of the joint occurred in degenerative joint disease. The diagnosis was substantiated in most of the cases by synovial biopsy or at operation. Although mild inflammatory changes were found in some cases, in none were the changes those of rheumatoid arthritis. The latex fixation test was positive in one case, but the operative findings were not those of rheumatoid arthritis. None of these cases had evidence of rheumatoid arthritis in other joints, but many had evidence of generalized osteoarthrosis. Calcium and phosphorus metabolism has not been extensively studied but was normal in those cases investigated.

The age distribution was as follows: 50–60 years, 2 cases; 60–70 years, 7 cases; 70–80 years, 6 cases. There were 9 women and 6 men. In all the cases (except possibly one) the initial X-ray showed marked joint narrowing before any collapse of the femoral head so that avascular necrosis of the femoral head was not the primary event.

Clinical features: Usually, the onset of destruction of the femoral head could be recognized clinically by the marked increase in pain sometimes associated with general symptoms and a raised ESR. In some cases, destructive changes seemed to occur early while in others they occurred in an established osteoarthrosis. However, on occasions pain was not a marked feature and this has also been noted by Isdale (1962).

Case 1 Severe symptoms had occurred in a woman aged 66 who was first seen in August 1965; she had had a recent onset of pain in the left thigh. Examination showed some limitation of movement of the hip; X-ray (September 1965) showed evidence of an early arthropathy with narrow joint space and some possible osteoporosis (Fig 2). ESR 35 mm in 1 hour (Wintrobe); latex fixation test negative. Subsequently she complained bitterly of pain in the hip and general aches

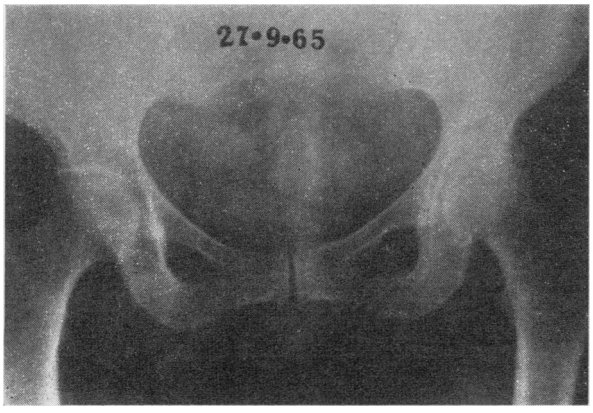

Fig 2 **Case 1** *September 1965: early changes of osteoarthrosis in left hip*

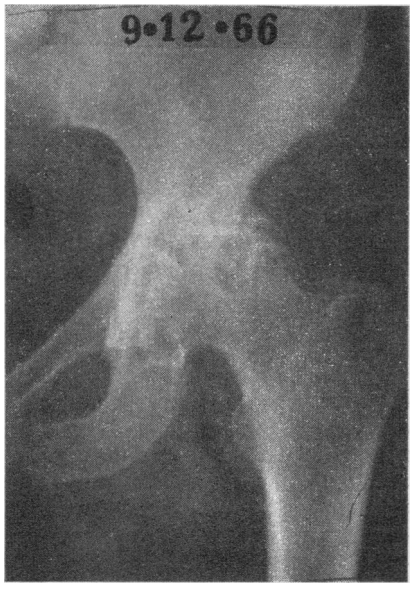

Fig 3 **Case 1** *December 1966: progressive development of osteoporotic area in upper femoral head which later collapsed*

and pains with tingling in the limbs and swelling of the face. A biopsy of the synovial membrane and related artery (to exclude giant cell arteritis) was undertaken and a psoas release was performed (November 1965). The biopsy showed dense fibrosis of the synovium and in the artery there was evidence of arteriosclerosis only.

There was no improvement in her symptoms and an X-ray (December 1966) (Fig 3) showed the development of a radiotranslucent area in the upper and outer aspect of the femoral head. By February 1967 this area was beginning to collapse and an intertrochanteric osteotomy with internal fixation was performed. Her symptoms improved after this, but an X-ray in May 1967 showed further collapse of the radiotranslucent area.

Case 2 A similar course of events was seen in another woman, aged 75. At first (June 1964) the X-ray was similar to that of Case 1 with a triangular area of translucency in the upper and lateral aspect of the right femoral head. Later (July 1965) the X-ray showed more complete collapse of the right hip. ESR 24 mm in 1 hour (Wintrobe); latex fixation test positive. A Girdlestone procedure was carried out. The synovial membrane showed some inflammatory changes, but not sufficient to diagnose rheumatoid arthritis. Later, her left hip became affected and showed partial collapse with protrusio acetabuli.

Case 3 Man aged 75. He had been known to suffer from osteoarthrosis of the left hip since 1957 (with X-ray confirmation). One week before admission in November 1966 he had a severe pain in the left leg and was unable to walk. Examination was difficult as he held the whole leg rigid and the site of the pain was difficult to locate. X-ray showed joint space narrowing of the left hip with a small crescentic area off the upper surface. ESR 26 mm in 1 hour (Wintrobe); latex fixation test negative. A biopsy of the synovial membrane was undertaken and a psoas release was performed (December 1966) with improvement of symptoms. Biopsy showed chronic nonspecific inflammatory changes.

Case 4 Pain was minimal in the case of a man aged 66 who in 1957 had a fall on the right hip; the X-ray taken to exclude a fracture showed no abnormality. Following this he had some pain in the hip, but his main complaint was that his leg was getting shorter. An X-ray of his pelvis in 1965 showed some destructive changes in the right femoral head, but he did not complain of his hip until 1967 by which time marked destructive changes in the right femoral head had occurred. In this case trauma seemed to be the initiating factor and it may be that avascular necrosis of the femoral head was the primary lesion.

Two other cases appeared to have precipitating factors, the changes in one (Case 6) following hernia operation:

Case 5 A man aged 69 had been diagnosed radiologically in 1964 as having osteoarthrosis of the right hip. In spite of this he continued his job as a window cleaner until he was forced to give it up because of pain and was seen again in October 1966. An X-ray of the hip now showed marked destructive changes of the femoral head and acetabulum with osteoporotic and

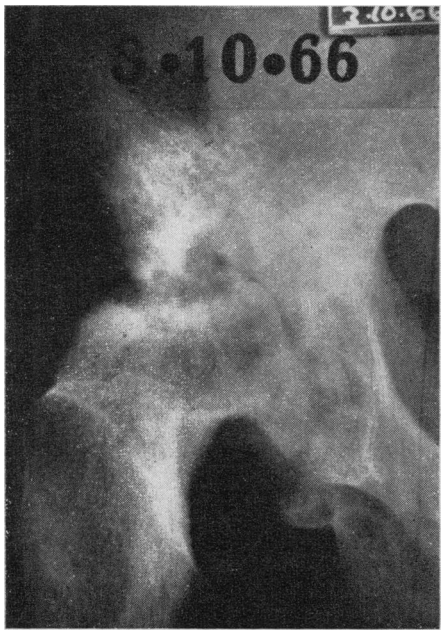

Fig 4 **Case 5** *October 1966: severe destructive changes in femoral head and acetabulum in osteoarthrosis of right hip*

sclerotic areas (Fig 4). ESR 3 mm in 1 hour (Wintrobe); latex fixation test negative. A Girdlestone procedure was carried out and the pathological findings in the femoral head were only degenerative in nature.

Case 6 A man aged 76 who had a left inguinal hernia operation in July 1965. Until this time he had had no pain in the hip. After operation he complained of pain in the groin and was found to have limitation of hip movement. An X-ray in August 1965 showed fluffy sclerotic changes with narrowing of the joint space. This man had quite marked general symptoms. He looked ill and complained of a lot of pain. ESR 46 mm in 1 hour (Wintrobe); latex fixation test negative. An X-ray in April 1966 showed some remodelling of the head of the femur with a sharper outline. A psoas release and biopsy were performed (September 1966) with marked improvement of symptoms. Biopsy showed some nonspecific inflammation of the synovial membrane. It appeared that local surgery initiated the collapse of a previously arthritic hip, perhaps through venous congestion.

Isdale (1962) mentions a patient who developed avascular necrosis of the femoral head after a prostatectomy.

In this series both hips were involved in 6 cases and one hip only in 9 cases. It was not always possible to follow the course of events, but in at least 2 cases of bilateral involvement one hip was first involved, to be followed after a few months by the other hip:

Case 7 A man aged 56 had had bilateral osteoarthrosis for some years. X-rays in 1964 showed evidence of early osteoarthrosis with cystic changes in both hips. Following this there was fairly rapid increase in symptoms. The X-ray in 1965 (Fig 5) showed deterioration, particularly in the right hip. A low-friction arthroplasty was performed on this hip with relief of symptoms. However, there was now an increase in symptoms on the left side and an X-ray of the hip (1967) showed an increase in cyst formations and commencing collapse of the superior lateral aspect of the femoral head (Fig 6). A similar operation on the left side relieved his symptoms and he has now returned to work. The pathological process appeared to be collapse of bone cysts.

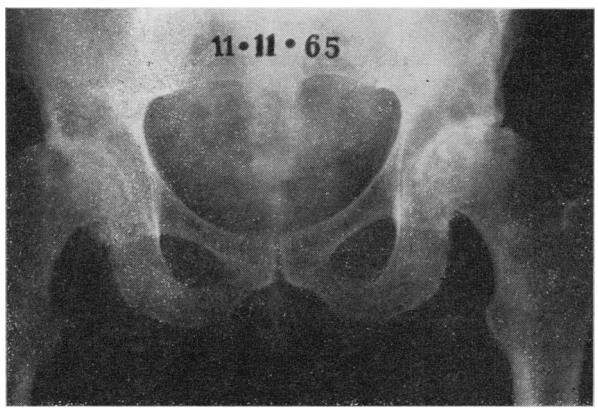

Fig 5 **Case 7** *November 1965: development of bone cysts in osteoarthritic hips*

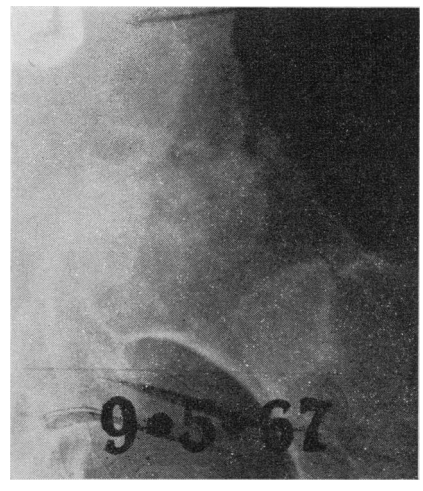

Fig 6 **Case 7** *May 1967: collapse of bone cyst in left hip*

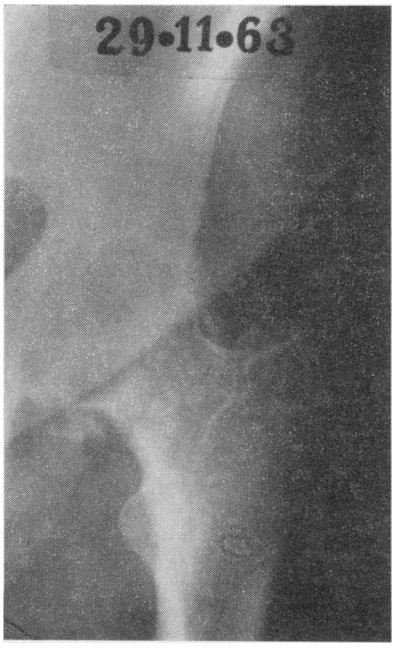

Fig 7 **Case 9** *November 1963: early changes of osteoarthrosis in left hip*

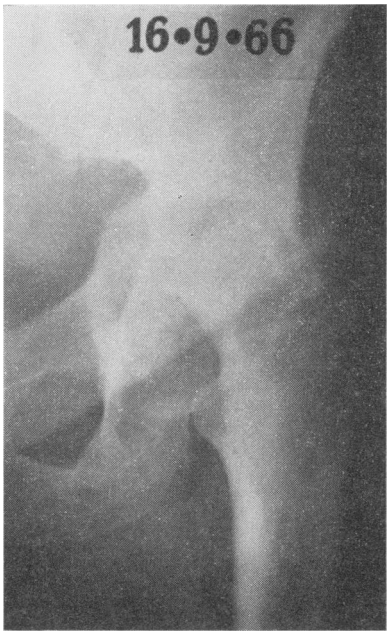

Fig 8 **Case 9** *September 1966: progressive attrition of upper surface of femoral head with development of protrusio acetabuli*

The course of disease extended over a few years in Case 7 but in Case 8, a woman of 77, collapse of the femoral head appeared to occur in a period of four months.

Radiological changes: Many of these have been described in the preceding cases.

Collapse of the femoral head may result from local change occurring at the superior lateral aspect of the head of the femur (Cases 1 and 2), or the whole upper surface of the head of the femur may be destroyed. This was so in Case 9.

Case 9 A woman of 66 was seen in November 1963 after four months' pain in the left hip. X-ray showed a general narrowing of joint space with buttressing of the femoral head (Fig 7). She continued to have pain in the hip and this became worse with pain at night. When seen again in September 1966 the upper part of the femoral head had been destroyed and she had developed protrusio acetabuli (Fig 8). Resection of the femoral head with subtrochanteric osteotomy was carried out and the femoral head is shown in Fig 9 by ultraviolet light illumination. This shows attrition of the upper surface with granulation tissue and some fibrocartilage overlying this. There was no evidence of avascular necrosis in the specimen.

Involvement of acetabulum:

(1) Protrusio acetabuli occurred in 4 cases (Figs 8 and 10). It has been described as a feature of rheumatoid arthritis but in 2 of the cases histological evidence did not support this diagnosis.

(2) Marked destructive changes were seen in one case (Case 10, Fig 11) associated with collapse of the femoral head.

(3) In one case at least collapse of a bone cyst of the acetabulum was a marked feature.

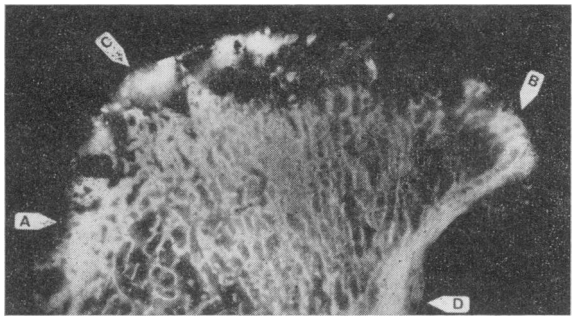

Fig 9 **Case 9** *Femoral head by ultraviolet light illumination.* A, *eroded, rather thickened, bone trabeculæ with a little light granulation tissue over them.* B, *eburnated reinforced bone.* C, *new fibrocartilage.* D, *new bone reinforcement under the femoral neck*

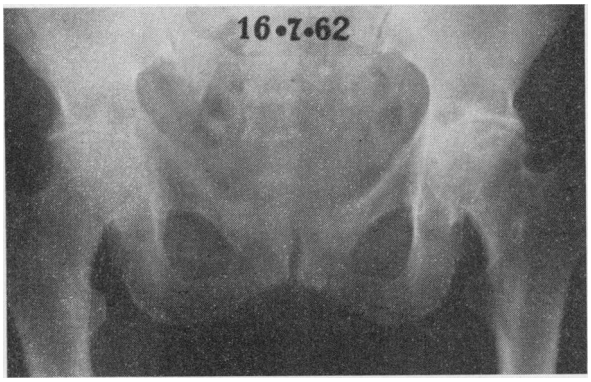

Fig 10 **Case 10** *July 1962: bilateral change of early osteoarthrosis*

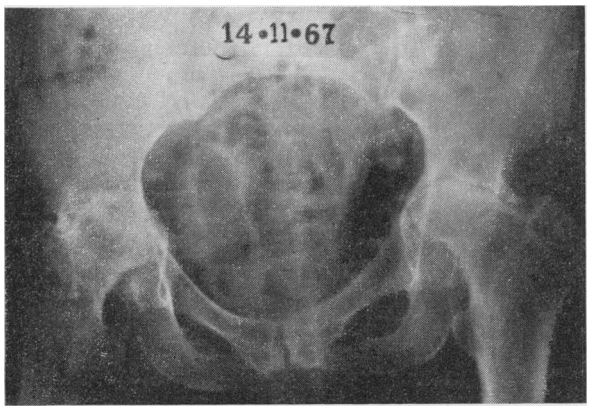

Fig 11 **Case 10** *November 1967: late changes with bilateral protrusio acetabuli, destruction of femoral heads and some reformation of joint space on the left*

It seems possible to trace the course of events radiologically, the early change being osteoporosis with sometimes areas of fluffy osteosclerosis and cystic changes, while later the femoral head takes on a firmer outline and the joint space seems to reform (possibly occupied by fibrocartilage); but it is necessary to emphasize that care must be taken in interpreting X-rays in terms of pathology.

Case 10 The first and end stages were seen in the case of a woman aged 66 who in 1962 had had two years' pain in the groins. She had evidence of bilateral arthropathy of the hips (Fig 10). Pain was not very marked, but she had progressive difficulty in walking and has been mainly in a wheel chair since 1965. In 1967 she was seen again and the X-rays now showed collapse of both hips with evidence in the right hip of reformation of the femoral head and reappearance of the joint space. The changes in the left did not appear to have reached this stage (Fig 11). ESR 5 mm in 1 hour; latex fixation test negative.

(4) The radiological appearances alone, however, may be misleading as some patients who show early changes do not proceed to destruction of the femoral head. There seems no way of predicting the extent of destruction in the early cases. This contrasts with idiopathic avascular necrosis (Merle d'Aubigné *et al.* 1965).

Pathology: All but 4 patients have either had synovial biopsy or have been examined at operation. Nonspecific inflammatory changes were frequently found in the synovial membrane, but these were more consistent with those sometimes found in osteoarthrosis than with rheumatoid arthritis. The gross changes in the femoral head of one case have already been described (Case 9, Fig 9). In another case (a woman aged 56) the pathological findings were similar to those in Case 9 except that the superficial layer of fibrous tissue and fibrocartilage were absent. The head of the femur seemed to have been worn away. There were no areas of necrosis in this case either.

Although the findings in these cases were not those of rheumatoid arthritis the course of events seem to be similar to the collapse of joints which may occur in this condition (Glick *et al.* 1963).

Treatment: Medical treatment during the phase of collapse is not very effective and surgery is usually indicated. Rest does not seem to arrest progress and may aggravate it by increasing osteoporosis. Excessive exercise may be an important initiating

factor. It has been advocated that vigorous non-weight-bearing exercise is the most logical line of treatment. Analgesics, of course, are necessary but Murray (1965) believes that phenylbutazone may be a factor responsible for initiating the collapse. Indeed, any effective pain reliever may increase collapse of the femoral head as in the case of formation of a Charcot joint.

Surgery seems the treatment of choice. There may well be some uncertainty about the diagnosis and a biopsy will usually be necessary. As some of the patients may be elderly, major operations may be hazardous and in such cases minor procedures such as psoas release may be indicated in the first instance. The latter was successful in 2 patients although it failed in one. However, more extensive measures may be necessary. Clearly, all cases differ and I cannot discuss here the relative merits of the procedures which are available. If surgery is thought to be too hazardous it must be remembered that, as in rheumatoid arthritis, there are indications that the process is self-limiting and stabilization may occur though this is likely to be a lengthy process.

Discussion: Collapse of joints in osteoarthrosis may occur in one of three ways: (1) Avascular necrosis presumably the result of arteriosclerosis. (2) Attrition or pressure necrosis. (3) Collapse of bone cysts; this may be regarded as a variant of attrition collapse.

Some of the features in the cases described suggest at least a relationship with avascular necrosis. There was one case where it certainly occurred in a case of rheumatoid arthritis with psoriasis. Some of the associated factors described in avascular necrosis were present in these cases. Inflammation was seen in the synovial membrane in many, but those changes were consistent with an inflammatory phase of osteoarthrosis. There was evidence of trauma, both single and repeated, and venous obstruction may have been a factor in the post-operative cases. There was no suggestion of alcoholism in these cases. However, in at least 2 cases there was no evidence of avascular necrosis and the presence of protrusio acetabuli in 4 cases suggests a bony abnormality. Further investigation of calcium and phosphorus metabolism might yield information, but an ageing change seems likely – possibly related to senile osteoporosis. There may have been some local osteoporosis in our cases, but this was not general and there were some younger patients in the group. The changes of rheumatoid arthritis seem to be similar and perhaps the occurrence of spontaneous fractures in older rheumatoid arthritic patients is a related event (Haider & Storey 1962). The speed of events may be an important factor. If the cartilage gives way rapidly the underlying bone may not have time to respond and may disintegrate. The process of attrition may follow the course described by Harrison *et al.* (1953) although they regarded this as a late event in osteoarthrosis and seem to suggest that it takes place gradually. They describe how, as the result of pressure at the weight-bearing point, small areas of necrosis develop and these are rubbed off. They attribute this necrosis to ischæmia, presumably due to pressure and different from the primary vascular type.

It has been suggested that the two processes, attrition and avascular necrosis, may coexist. It is postulated that during attrition cracks and stresses are set up in the femoral head and these may rupture vessels deep in the head and hence give rise to larger areas of avascular necrosis, but the relationship between these two types of necrosis remains to be elucidated. Certainly, the formation of bone cysts seems to result from local trauma to the bone cortex so that synovial fluid is squeezed into the femoral head and becomes encysted (Landells 1953, Golding 1966). Thus collapse of bone cysts may be regarded as a slowed down form of attrition. It should be possible to answer some of these problems now that the removal of complete joints surgically is more frequently undertaken.

Charcot Joints

The collapse of joints is here seen in its most florid form. In some cases joints seem to melt away while in others sclerosis is a very early change before even the joint is involved. This change has been attributed to avascular necrosis, caused by stress in the bone, obstructing the blood supply; the bone is therefore friable and easily disintegrates. However, here too there is a tendency to stabilization and the bone ends may reform in an abnormal shape (Storey 1964).

Conclusion

The group of cases described in which collapse of the hip occurs appears to represent a variation or complication of degenerative joint disease, although similar changes may result from rheumatoid arthritis. The collapse results either from avascular necrosis or from a condition related to senile osteoporosis causing attrition necrosis. At the same time there are also similarities to the Charcot joint.

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Investigation of Joint Fluids [*Abridged*]

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Examination of Joint Fluids for Crystals

The central role of uric acid in gout was discovered over a hundred years ago (Garrod 1848), but it was not until recently that Hollander (1960) first showed that urate crystals could be identified in synovial fluid removed from acute gouty joints. Shortly afterwards, McCarty *et al.* (1962) noted a different type of crystal, now known to be calcium pyrophosphate dihydrate (CPPD), in synovial fluid from the acutely inflamed joints of patients showing radiological evidence of calcification of articular cartilage. It is now clear that these patients with chondrocalcinosis and 'pseudo-gout' were suffering from a not uncommon condition for which McCarty proposes the name 'crystal deposition disease – type calcium pyrophosphate', to distinguish it from 'crystal deposition disease – type urate' – or gout. Common to these two conditions are episodes of acute crystal synovitis, during the course of which the responsible type of crystal may be identified in the synovial fluid.

Examination of joint fluids for crystals involves the use of the polarizing light microscope in which, essentially, the specimen is placed on a rotatable mechanical stage, with polarizing filters (polars) above (the analyser) and below (the polarizer). The orientation of the plane of polarization of these two polars can be varied independently, the most useful arrangement being that in which they are at right angles to each other ('crossed polars').

Crystals of urate and CPPD both have asymmetrical internal molecular structures such that light traversing the crystal may travel at one of three different speeds, depending on its plane of polarization. These crystals thus have three different refractive indices. This gives them the optical property of birefringence, enabling their presence to be easily detected in biological material. It is also possible for the two types of crystal to be differentiated.

Synovial fluid for examination is collected without added anticoagulant. Any clot which forms will entangle both cells and crystals, and part of such a clot must therefore be included in the drop of fluid placed on a slide, on to which a coverslip is then placed. Specimens often remain