

IDIOPATHIC BONE NECROSIS OF THE FEMORAL HEAD

EARLY DIAGNOSIS AND TREATMENT

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Our understanding of idiopathic necrosis of the femoral head depends upon two fundamental concepts.

The first is that a *standard radiograph shows only the shadow of the mineralised portion of a bone*. The radiographic appearance of living bone is the same as that of dead bone of Egyptian mummies or prehistoric skeletons. Consequently, bone necrosis has no specific radiographic appearance and a normal radiograph does not necessarily mean a normal hip. A standard radiograph cannot help with early diagnosis, and every case of bone necrosis must pass through a preradiographic stage. When radiographic changes do appear, they are due to the reaction of living tissue to the ischaemia.

The second fundamental concept is that *bone necrosis is the end result of severe and prolonged ischaemia*. This again presupposes an initial stage in which vascular and medullary abnormality passes undetected by routine radiography.

These concepts point the need for other methods of investigation, especially in early cases. These methods include scintimetry and the study of the haemodynamics of the medullary circulation, termed by us the functional exploration of bone (FEB). These methods of diagnosis, together with a classification developed in association with Professor J. Arlet, are now presented (Ficat and Arlet 1977, 1980).

EARLY DIAGNOSIS

Scintimetry. A *bone scan* with ^{99m}Tc -MDP in early bone necrosis usually shows an increased uptake of the radionuclide; in the very earliest stage there may, rarely, be decreased uptake in the femoral head. These changes in uptake are pathological, but are not specific for bone necrosis. A *bone marrow scan* using ^{99m}Tc colloidal sulphur may provide more information and assist early diagnosis but, unfortunately, this material is considerably more expensive than ^{99m}Tc -MDP, and is no better than haemodynamic tests in making the early diagnosis.

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Functional exploration of bone (FEB). This simple and safe method is indispensable for diagnosis in the early stages when the patient has a painful hip with limitation of movement, but the radiograph is normal or near-normal. **Bone marrow pressure.** Measurement of bone marrow pressure is the first stage of FEB. It can be done through a cannula placed in the intertrochanteric area under local anaesthesia. The baseline pressure is usually about 20 mmHg, and 30 mmHg is regarded as the upper limit of normal.

If the baseline pressure is within normal limits, 5 ml of isotonic saline is injected into the bone and the pressure recorded five minutes after injection. This, the "stress test" pressure, is normally less than 10 mmHg above the baseline pressure.

If both tests are normal, they should be repeated using a cannula placed in the head of the femur.

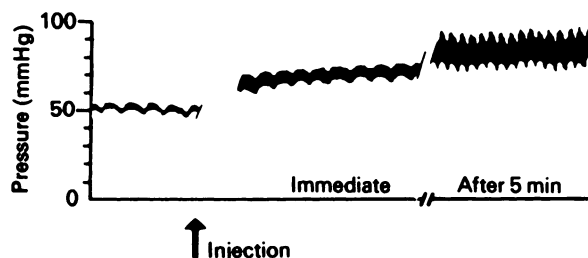


Fig. 1

Recording from a Functional Exploration of Bone. The bone marrow baseline pressure in the trochanteric area is 50 mmHg, which rises to 85 mmHg after the stress test.

In cases of bone necrosis, the baseline pressure is above 30 mmHg, and the stress test pressure is 10 mmHg or more above the baseline after five minutes (Fig. 1). The trocar in the trochanteric area also permits the collection of a specimen of blood for the measurement of *oxygen saturation*. A reading above 85% is indirect proof of anoxia in the head, strongly suggesting failure of circulation and of oxygen uptake ($P < 0.001$).

Intramedullary venography is the second stage of FEB. Ten millilitres of contrast medium is injected through the cannula used for the stress test. In a normal hip the radio-opaque material is easily and quickly cleared by normal efferent vessels, especially the ischial and circumflex veins, without any diaphysial reflux or stasis. In cases of bone



Fig. 2



Fig. 3

Figure 2—Venogram from injection into the trochanteric area. Diaphyseal reflux is seen, with stasis. Figure 3—A specimen of bone removed by core biopsy. The head portion above is dense and white, contrasting with the spongy bone from the neck of the femur.

necrosis the injection is difficult and painful, with reflux into the diaphysis and intramedullary stasis 15 minutes after injection (Fig. 2).

The diagnosis of bone necrosis is probable even if only the baseline pressure, or the stress test or the venography is positive.

Core biopsy, the third stage of FEB, establishes the diagnosis with certainty. The technique is simple. A hollow trephine, 6 or 8 mm in diameter, is introduced into the neck of the femur from an opening in the greater trochanter and passed towards and into the head, stopping 5 mm short of the articular cartilage. This introduction is controlled in both the anteroposterior and lateral planes with an image intensifier. A second channel is made with a smaller trephine in a different direction.

The core specimens so obtained are sent for histological examination (Fig. 3) and the forage channels are left open, so providing decompression.

The histological lesions seen in these specimens have been classified by Arlet and Durroux (1973) into four types.

Type 1—disappearance of the haemopoietic marrow, separation of the lipocytes by oedema or haemorrhage, and the presence of foam cells (Fig. 4).

Type 2—necrosis of the fatty marrow, which is largely changed to an eosinophilic reticular pattern, sometimes with oil cysts and necrosis of the haemopoietic marrow of a granular type (Figs 5, 6 and 7).

Type 3—complete medullary and trabecular necrosis (Fig. 8).

Type 4—complete necrosis with dense medullary fibrosis and new bone formation in apposition to the dead trabeculae.

The interpretation of this histology requires comment. First, it should be stated that Type 1 histology is not diagnostic of bone necrosis. Secondly, all four types may be observed in the same specimen. Thirdly, it seems logical to suppose that the ischaemic process develops in three successive stages: circulatory (Type 1), medullary (Type 2), and osseous (Types 3 and 4). We are convinced that this evolution occurs but recognise that it may not be seen in all cases. Moreover, the evolutionary process may be reversible at any stage, as has been demonstrated experimentally by Rutishauser, Rhoner and Held (1960). Finally, it should be emphasised that there is little or no correlation between the histological typing and the radiographic or clinical features.

STAGING

Five successive stages of bone necrosis can be identified. **Stage 0.** This stage is both preclinical and preradiographic. The diagnosis is suspected in one hip when the other has definite disease, because it is known that there is a high incidence of bilateral involvement. This is the stage of the truly "silent hip".

Hungerford (1979) identified this stage and its evolution when he reported a series of 27 such silent hips. Of these, 17 had increased intramedullary pressure without symptoms or radiographic signs; 11 of the 17 developed disease, proved by biopsy, after an interval of one to five years. Thus bone necrosis developed in 64.7% of the hips with a positive FEB. One hip with a negative FEB later developed necrosis, so the total incidence in all 27 silent hips was 44.4%.

This reported experience is perhaps the best argument for early treatment of bone necrosis in order to avoid the complications of advanced disease.

Stage I. This is the earliest clinical manifestation of the syndrome. The most common symptom, seen in 50% of cases, is sudden pain in the groin. This may be progressive, and be associated with radiation to the thigh, which is often worse at night. This pattern of pain, which is typical of ischaemia, is often neglected in the literature. Pain may be aggravated by coughing, and there are sometimes intermittent vasomotor disturbances in the limb.

Examination of the hip reveals limited movement; this limitation may be in all directions, or only one, particularly medial rotation or abduction. Although this limitation is variable, it is of great importance, because it establishes that the pain is articular in origin. It is therefore essential to record the ranges of flexion, abduction and adduction in the supine position, and of extension, and both rotations in the prone position; and then to compare these with the corresponding ranges of movement in the opposite normal hip.

Standard anteroposterior and lateral radiographs are usually normal in Stage I or, at most, show only minor changes such as subtle loss of clarity with poor definition or blurring of the trabecular pattern. There may be some

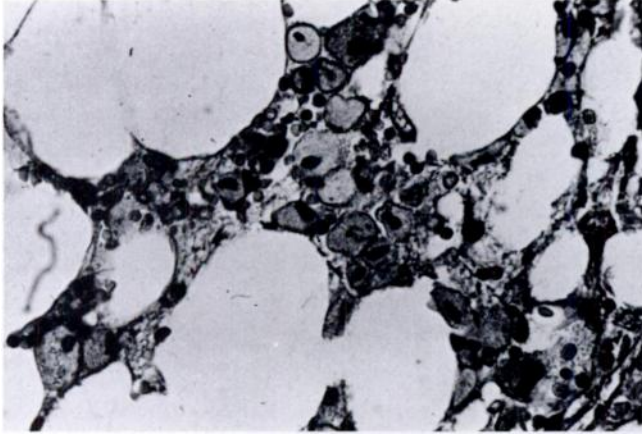


Fig. 4

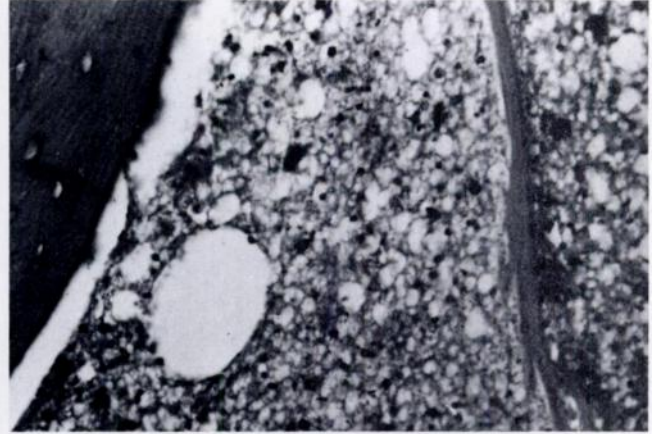


Fig. 5

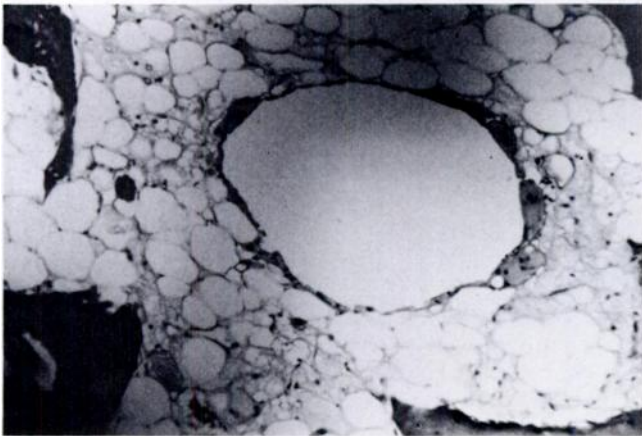


Fig. 6

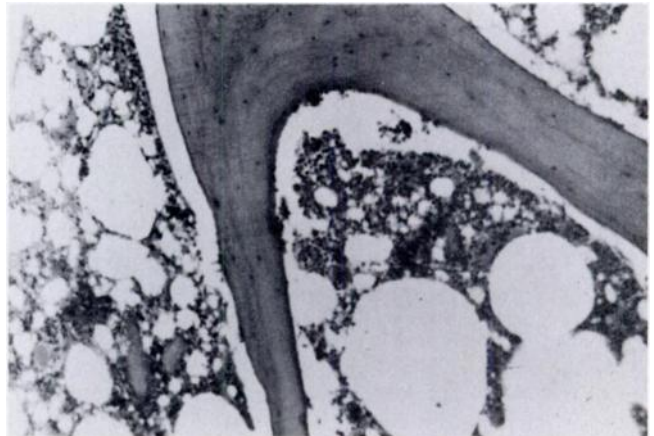


Fig. 7

Histology of various stages and types of ischaemic bone necrosis. Figure 4—Type 1, numerous foam cells are seen around the lipocytes. Figure 5—Type 2 changes with extensive eosinophilic reticular necrosis of the bone marrow. Figure 6—Type 2, showing necrosis of the bone marrow with an oil cyst. Figure 7—Type 2 with necrosis of the haemopoietic bone marrow. Figure 8—Type 3 showing necrosis of the bone marrow and of the bony trabeculae.

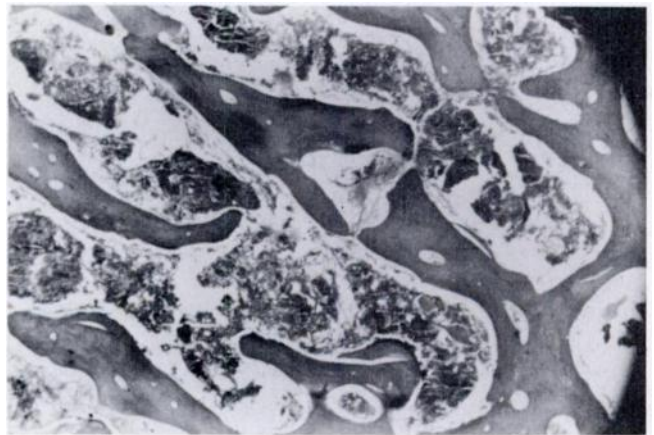
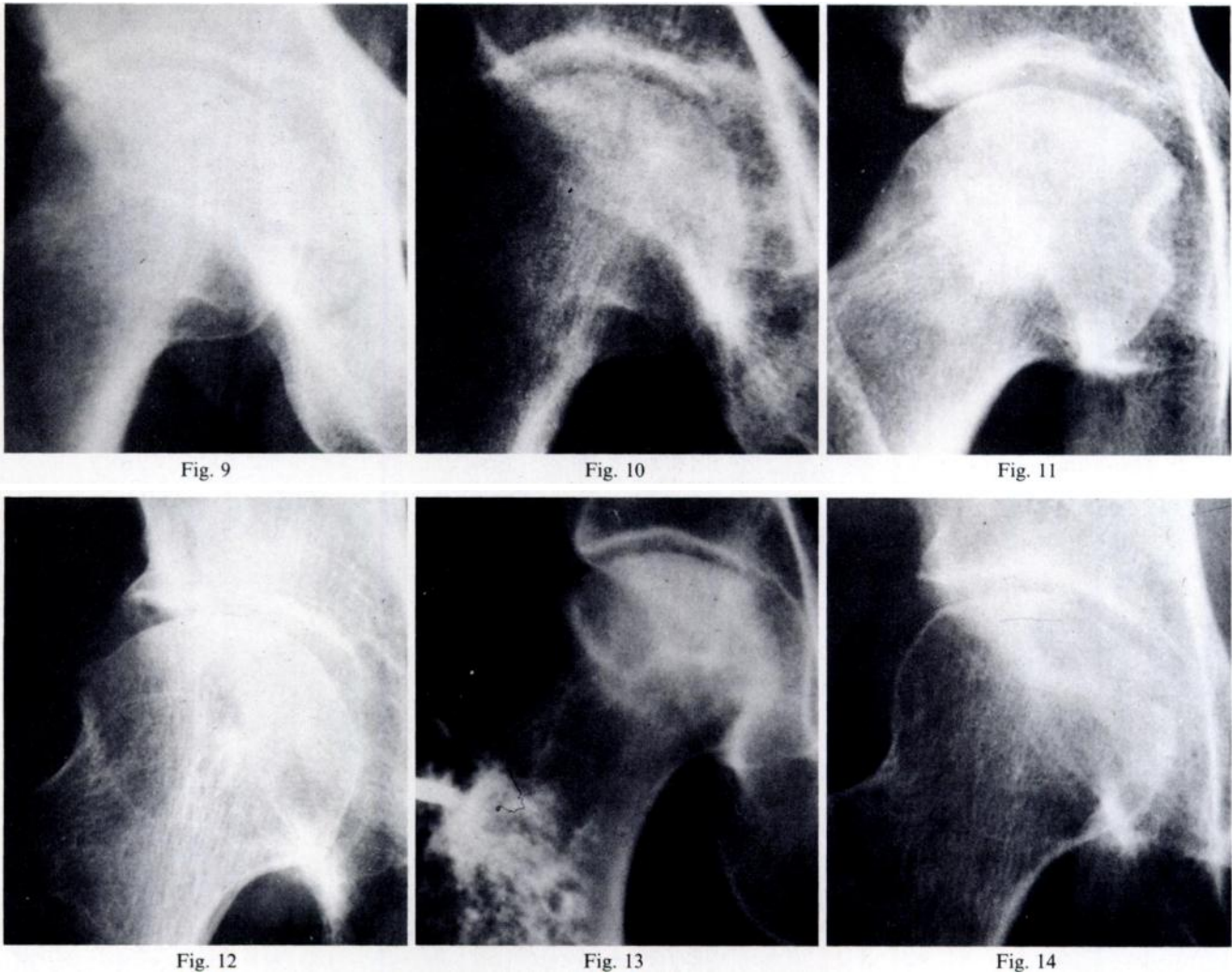


Fig. 8



Radiographs of bone necrosis of the femoral head at various stages. Figure 9—Normal appearance in early necrosis (Stage I). Figure 10—Same hip three months later showing diffuse osteoporosis (Stages I-II); core biopsy showed an extensive eosinophilic reticular pattern. Figure 11—Sclerotic changes in Stage II. Figure 12—Cystic changes in Stage II. Figure 13—Mixed sclerotic and cystic change in Stage II, with an arcuate line of increased density, concave upwards. Venography has been done. Figure 14—Segmental flattening above the fovea, seen in the transition between Stages II and III.

slight patchy osteoporosis in comparison with the opposite side, but the changes are not really significant (Figs. 9 and 10).

Stage I osteonecrosis must be suspected in every painful hip with limitation of movement and a normal radiograph; the special FEB tests should be carried out as soon as possible. These tests are the key to the differential diagnosis of all hip disorders which present with this clinical picture.

Stage II. This extends over several months and sometimes much longer. The clinical signs persist, or worsen, and the radiographs show changes in the trabecular pattern of the femoral head. Sclerosis may be diffuse, in localised areas, or in a linear arc which is concave superiorly (Fig. 11). Decalcification may also be generalised or in the form of small cysts in the head, usually at some distance from the joint space (Fig. 12). A mixed form including both sclerosis and cysts is sometimes seen (Fig. 13). The diagnosis of osteonecrosis of the femoral head is suggested by these appearances, but it can be confirmed with

certainly only by FEB tests.

Stage III. This stage is characterised by the pathognomonic appearance of a sequestrum on the radiograph. This appearance is often preceded by changes representing a transition between Stages II and III; these are a *crescentic line* due to a subchondral fracture, and *segmental flattening* of the femoral head giving the so-called "out of round" appearance (Fig. 14).

The sequestrum later becomes manifest by a break in the articular margin extending from one end of the affected area to the other, followed by collapse of the sequestered area into the femoral head. Paradoxically, and because of the limited size of the sequestrum, the joint space is preserved or even increased.

The clinical picture is now of increasing and more constant pain, limitation of movement in all directions, progressive functional incapacity, and a limp which usually requires the use of a stick.

Stage IV. This is the terminal phase of the necrotic process, and is characterised by progressive loss of articular

Table I. The stages of bone necrosis of the femoral head

Stage	Clinical features	Radiographic signs	Haemodynamics	Scintigram	Diagnosis without core biopsy	
Early	0 Preclinical	0	0	+	Reduced uptake?	Impossible
	I Preradiographic	+	0	++	Increased uptake	Impossible
	II Before flattening of head or sequestrum formation	+	Diffuse porosis, sclerosis, or cysts	++	+	Probable
Transition		Flattening Crescent sign				
Late	III Collapse	++	Broken contour of head Sequestrum Joint space normal	+ or normal	+	Certain
	IV Osteoarthritis	+++	Flattened contour Decreased joint space Collapse of head	+	+	Arthritis

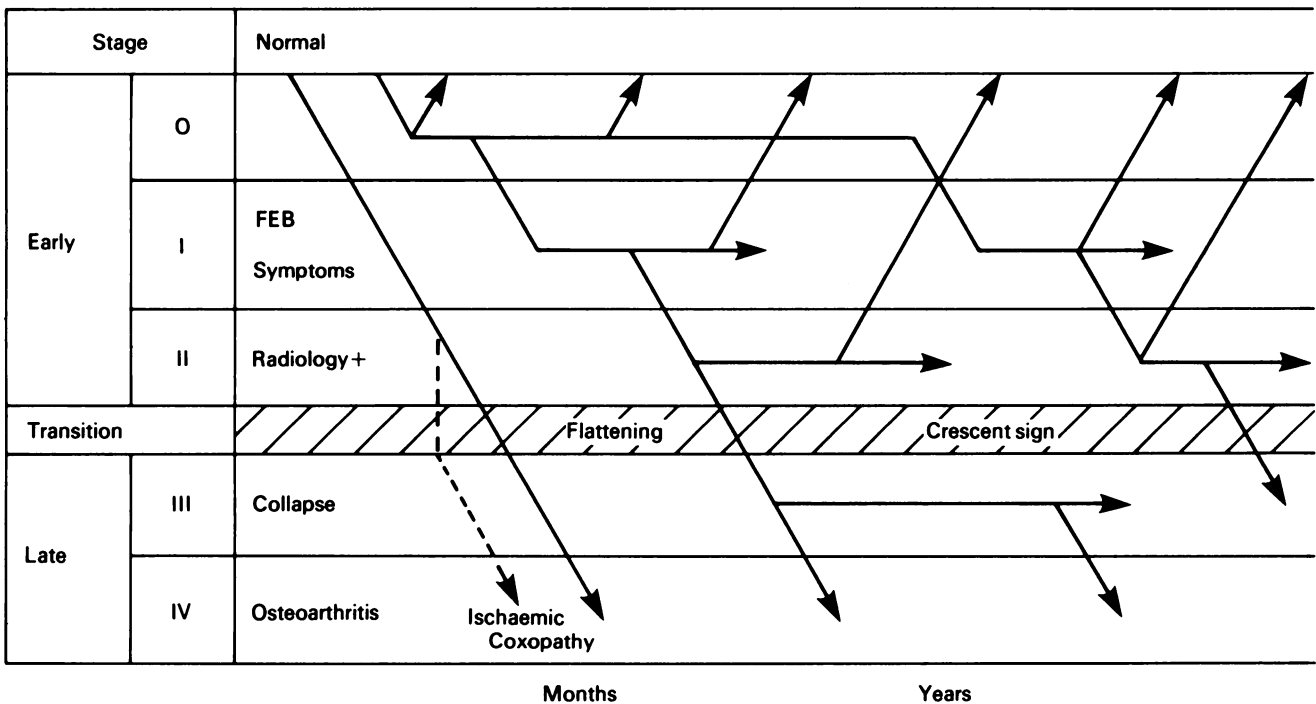


Fig. 15

Diagram of the evolution of osteonecrosis.

cartilage and the development of acetabular osteophytes; the radiographic picture is of osteoarthritis superimposed on a deformed femoral head. Movement is progressively diminished until only a small range of flexion remains.

Two variations from this typical form of the condition are recognised.

Ischaemic coxopathy. This is characterised by narrowing of the joint space in the early stages of the disease; it occurred in 17.6% of 108 cases in our series of osteoarthritic hips.

If the special tests (FEB) are not done and there is no obvious sequestrum in the radiographs, an incorrect diagnosis of primary osteoarthritis may be made. The clinical characteristics of ischaemic coxopathy are that it is commonest in the sixth decade; that the onset is sudden with pain at night; and that there is joint space narrowing, either generalised or confined to the superomedial quadrant, with little osteophyte formation. It can occur not only in morphologically normal hips, but also in dysplastic hips in which articular degeneration can be expected.

Table II. Period of follow-up of 133 cases of bone necrosis in Stages I and II

Stage	Number of hips	Follow-up (years)													
		5	6	7	8	9	10	11	12	13	14	15	16	17	
I	82	7	4	1	17	14	10	5	4	11	3	1	3	1	
II	51	7	6	—	8	5	12	3	6	3	2	—	—	—	
I+II	133	14	10	1	25	19	22	8	10	14	5	1	3	1	

Bilateral involvement. This occurs in 50% of cases of idiopathic bone necrosis and in as many as 80% of cases of steroid-associated necrosis. The condition is frequently asymmetrical, with Stage III changes on one side and Stage I (radiographically normal) on the other. The Stage I hip may be absolutely "silent" and clinically normal, and therefore the FEB tests are clearly indicated. The prognosis is usually bad.

This classification is summarised in Table I and the evolutionary possibilities in Figure 15. The importance of detecting the disease in the early stages must be emphasised, since treatment given before the point of no return can prevent the irreversible lesions seen in Stages III and IV.

RESULTS OF CORE DECOMPRESSION

From our total experience of more than 600 cases of bone necrosis, 144 patients with 156 hips in the early stages of the disease have been reviewed. All were treated by core decompression and have been followed up for over five years. Of the 144 patients, 23 were not available at five years; five had died of unrelated conditions, and 18 were lost to late follow-up although when reviewed at between one and three years there were 15 good results and 3 failures.

In the remaining 121 patients there were 133 involved hips; 82 hips were in Stage I (61.6%), and 51 in Stage II (38.4%). There were 69 men and 52 women, with about equal involvement of right (68) and left (65) hips. The age range was from 17 to 80 years with a peak incidence between 40 and 50 years (30.5%). The patients were followed up for from 5 to 17 years with an average of 9 years 6 months (Table II). The criteria used to record the results are set out in Table III.

The clinical results of core decompression are given in Table IV; good results were obtained in 93.9% of Stage I hips and in 82.3% of Stage II hips. The radiographic results (Table V) show 86.6% good results in Stage I, and 66.7% good in Stage II. Of the 28 cases in which there was radiographic failure, 13 also had clinical failure.

Two kinds of radiographic failure could be recognised. *Joint space narrowing* occurred in 18 cases, 8 of which were treated in Stage I (9.8%), and 10 in Stage II (19.6%). Most of these cases remained asymptomatic, but the fact that the changes took place confirm the possible evolution of bone necrosis of the femoral head into ischaemic coxopathy as discussed above. The appearance

Table III. Criteria for the clinical and radiographic grading of results

Pain	Range of movement (degrees)	Radiograph	Result
Nil or slight	Slight or no loss Flexion over 100 Abduction 45–60	Normal	Very good
Slight intermittent	Flexion 80–100 Abduction 30–45	Patchy sclerosis Small cysts	Good
Severe	Flexion under 80 Abduction under 30	Crescent sign Flattening Narrowing of the joint space	Fair
Permanent	Stiffness +	Sequestration Collapse Arthritis	Failure

Table IV. Clinical results after core decompression

Stage	Number of hips	Results			
		Very good and good		Failure	
		Number	Per cent	Number	Per cent
I	82	77	93.9	5	6.1
II	51	42	82.4	9	17.6
Both	133	119	89.5	14	10.5

Table V. Radiographic results after core decompression

Stage	Number of hips	Results			
		Very good and good		Failure	
		Number	Per cent	Number	Per cent
I	82	71	86.6	11	13.4
II	51	34	66.7	17	33.3
Both	133	105	78.9	28	21.1

of a *sequestrum* was the second mode of failure. Ten hips showed a sequestrum with collapse of the head, three of which were originally treated in Stage I and seven in Stage II.

DISCUSSION

The important question is—how does core decompression work? The answer must be approached through the aetiology and pathogenesis of the disease.

The factors which cause osteonecrosis. Some causes are well established: major trauma, dysbarism, sickle-cell anaemia

mia, Gaucher's disease, postirradiation, steroid therapy and arterial disease. Other causes may be multifactorial and include: alcoholism, dyslipoproteinaemia, fatty degeneration of the liver, pancreatitis, hyperuricaemia, minor trauma, dysplasia, phlebitis, arthritis, osteoporosis, osteomalacia, and connective tissue disease. Nevertheless, there remain many cases in which no specific cause or association can be found.

The pathogenesis of the condition may involve a variety of mechanisms at different sites. *Extra-osseous vessels* may be involved; arterial obstruction could be due to thrombosis, embolism, rupture, stenosis, or compression, while venous block could be produced by phlebitis or compression. *Intra-osseous vessels*, either arterial or venous, might be obstructed by gas or lipid embolism, thrombosis, or vasospasm. Capillary obstruction could be by compression from *extravascular factors* such as hypertrophy of trabeculae or of adipocytes, invasion by abnormal cells, or haemorrhage. The *cell population* could be affected by cytotoxic factors, such as irradiation or chemical substances.

Whatever the cause and the starting point, and whatever the pathological process, *blockage of the osseous microcirculation* with intramedullary stasis appears to be the common denominator. It is the start of the vicious circle which leads to increasing pressure, metabolic disturbance, and then to anoxia and necrosis.

The pathophysiology is explained by the *compartmental nature of bone*; not only do the walls of the trabecular cavities and the cortical foramina behave like a multitude of tunnels, but the entire bony system acts as a rigid closed cavity. Such a system is particularly sensitive to an increase of pressure, especially when this is above 30 mmHg, as is the case in other compartment syndromes. The first effects of raised pressure are on the sinusoids and the small capillaries of the marrow, and then on the venous out-flow; even the nutrient arteries to the bone may be blocked by reflex spasm before they enter the cortex.

The effect of a core biopsy is similar to that of a decompression operation for a nerve tunnel syndrome or a fascial release for a muscle compartment syndrome. In osteonecrosis the vicious circle is broken at its origin by the reduction of medullary hypertension. The pressure drops

immediately the cortex is broached and the actual core decompression adds very little. Pain is relieved, often immediately and dramatically. Venous drainage is improved, relieving the congestion of tissues threatened by the necrosis. Revascularisation of the femoral head is promoted through a complex series of reactions.

Soon after the decompression operation there is hypervascularity of the entire joint, with vasodilatation and decalcification; this is similar to the early vascular effect of an osteotomy. After this the thousands of vessels opened by the trephine form a source for the formation of new vessels which invade the femoral head. Most importantly, the cortical foramina and the intramedullary sinusoids are decompressed and arterial spasm is relieved.

Once obstruction has been relieved, the microcirculation is restored, and metabolic processes return to normal. In this way decompression can function at several levels. Its application at the correct stage of the disease can be remarkably effective.

CONCLUSIONS

All hips which develop osteonecrosis pass through a stage in which the joint is radiographically and clinically normal (Stage 0, the "silent hip"); later the hip becomes painful but the radiographs remain normal (Stage I). Consequently, a normal radiograph does not necessarily imply a normal hip and in these circumstances diagnosis is possible only by functional exploration of bone (FEB).

Despite the complex pathogenesis, the basic cause, a disturbance of intra-osseous circulation, determines the evolution of the disease. The stages of this evolution account for the considerable variation in the clinical and radiographic signs.

Prevention of osteonecrosis must be based on treatment of the cause. *Curative treatment* can be achieved by forage-biopsy in the preradiographic Stage 0 and in Stages I and II. Early diagnosis is therefore essential; this is agreed by other authors (Marcus, Enneking and Massam 1973; Hungerford and Zizic 1983). *Prognosis* depends entirely on early diagnosis and effective treatment; the clinical results are much better in Stage I than in Stage II.

REFERENCES

- Arlet J, Durroux R. Diagnostic histologique précoce de l'ostéonécrose aseptique de la tête fémorale par le forage biopsie. In: *Premier symposium international de circulation osseuse*. Toulouse, Paris: INSERM, 1973.
- Ficat P, Arlet J. *Ischémie et nécrose osseuses*. Paris: Masson, 1977:224.
- Ficat RP, Arlet J. *Ischemia and necrosis of bone*. Baltimore, London: Williams & Wilkins, 1980:196.
- Hungerford DS. Bone marrow pressure, venography and core decompression in ischemic necrosis of the femoral head. In: *The hip: proceedings of the seventh open scientific meeting of The Hip Society*. St Louis: CV Mosby, 1979:218-37.
- Hungerford DS, Zizic TM. Pathogenesis of ischemic necrosis of the femoral head. In: Hungerford DS, ed. *The hip: proceedings of the eleventh open scientific meeting of The Hip Society*. St Louis, Toronto: CV Mosby, 1983:249-62.
- Marcus ND, Enneking WF, Massam RA. The silent hip in idiopathic aseptic necrosis: treatment by bone grafting. *J Bone Joint Surg [Am]* 1973;55-A:1351-66.
- Rutishauser E, Rhoner A, Held D. Experimentelle Untersuchungen über die Wirkung der ischämie auf den Knochen und das Mark. *Virchows Arch [Pathol Anat]* 1960;333:101.