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Bone Necrosis Due to Decompression [and Discussion]

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Bone necrosis due to decompression

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[Plate 1]

Death of areas of long bone has been known to be associated with work in compressed air and diving for many years. Detection and diagnosis require specialist knowledge, but familiarity with the condition has increased markedly over the last 20 years. Most of it is symptomless and causes no disability, but if areas adjacent to the joint surfaces of the upper end of the humerus and the upper end of the femur are affected, the joint may be seriously damaged and disability ensue. This problem has been defined by surveys of groups of workers using compressed air and divers in an attempt to establish prevalence. In general the more diving undertaken and the deeper the dives, the more probability there is of acquiring bone damage. The commonest area to be affected is the lower end of the femur, but the knee joint is never involved.

The cause of bone damage is far from clear. None of the various explanations that have been offered are satisfactory, but research continues into the problem both in humans and in animals, particularly the miniature pig. At present it is difficult to quantify the risk taken by commercial divers in relation to changing technology either in terms of depth or duration of pressure.

Introduction

Bone necrosis, that is death of parts of the long bones related to hyperbaric exposure, has been recognized for 70 years. It was first reported in workers using compressed air, in whom it was linked with attacks of decompression sickness (the bends) in the same limb, and it has also been known in divers since the 1940s. The best description of the problem as a phenomenon in groups of men actively at work, as opposed to clinical reports of small numbers of disabled men, was given by Golding *et al.* (1960), who studied workers at the first Dartford Tunnel, which was built with the use of compressed air between 1952 and 1959. Investigations were later extended to a group of men involved in digging two tunnels under the River Clyde between 1958 and 1963. It was then possible to construct a classification of bone necrosis that has helped considerably in defining the condition and its prognosis. With the rapid expansion of the diving industry in the 1960s, attention turned to commercial divers, in whom the bone necrosis appeared to be essentially the same as that in workers using compressed air. Radiographs, medical examination results and data on diving experience of individual divers have been collected in the Decompression Sickness Central Registry at the University of Newcastle upon Tyne since 1974. It has therefore been possible to record bone necrosis in divers, to classify it according to type, and to relate the presence of bone damage to various factors in the diver and his diving record. Bone damage of this kind from causes other than exposure to raised ambient pressure is rare in young healthy men. This is important because it may, in theory, be difficult to distinguish bone necrosis due to hyperbaric exposure, either on radiological or histological grounds, from that due to a variety of other causes.

A comparison between radiographs of the long bones of 93 United States Navy divers aged 35 years or more, and a group of 177 matched controls (Hunter & Biersner 1982), showed that the divers had significantly more bone necrosis. What also emerged from this investigation was the poor reliability of the radiographic classification by experienced radiologists. It is our experience that familiarity with the type of bone change seen in radiographs of men exposed to hyperbaric environments is necessary, and even experienced radiologists may have difficulty with what are known as bone islands in deciding whether or not they are areas of necrosis. Quality of the radiographs is of great importance, as is the positioning of the bones, so that diagnosis can be reliable.

It is characteristic of the bone necrosis that symptomless opacities are found in the long bones on radiological examination. Sites usually affected are the lower end of a femur, the upper end of a tibia, the head of the humerus or its shaft, and the head of the femur (figure 1). However, lesions adjacent to a joint surface (*juxta articular*) may cause disability, probably in part because they are vulnerable to trauma from bearing weight, as in the hips; or from the mechanical stresses of heavy manual work or other exertion on the shoulder joints. So, symptoms of pain and stiffness, and disability in the hip or shoulder joint may occur in 10 to 40 % of *juxta articular* lesions.

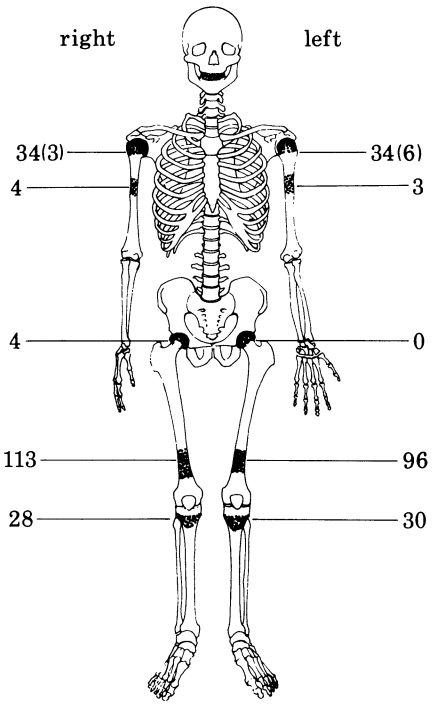

FIGURE 1. Sites of definite lesions of aseptic necrosis of bone in 207 commercial divers. The numbers in brackets indicate damage to joints.

The most recent information from the records in the Decompression Sickness Central Registry in Newcastle gives a total of 247 divers (4 %) with one or more sites of bone damage. This is based on the radiographs of over 6000 divers that have been scrutinized in the Registry. Disability due to collapse of a joint surface has been found in 11 men (0.2 %), nearly all of whom have sustained damage to a shoulder joint (table 1). More than 10 % of men that have been diving for 12 or more years have some bone necrosis.

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Table 1. Bone radiographs for commercial divers

radiograph	number of men
normal	2792
head, neck or shaft lesions	175 (69)
juxta articular lesions	72† (35)
irrelevant changes	2902
total	6045

† Disability in 11 men. The number of men with suspected lesions are given in brackets.

Relevant factors

There are a number of features of bone necrosis that have been identified but not necessarily explained. These are now discussed.

Repeated exposure to increased ambient gas pressure increases the probability of bone damage, but it is known that a single exposure can result in bone necrosis. One of the best documented examples of this is the Poseidon disaster of 1931. The submarine Poseidon sank in 36.5 m of water in the China Sea after a collision with a junk. Five men escaped from a chamber that they flooded over a period of 3.5 h and by using Davis apparatus they reached the surface of the sea in about 20 s. All of them had the bends on surfacing. Fourteen years later an orthopaedic surgeon examined three of the men and reported bone damage in all of them (James 1945). In one man, both femoral heads had been damaged and had subsequently collapsed, and the heads of both humeri were also the sites of bone necrosis; a second man had lesions in the head and neck of the left humerus, and the third man had a rounded dense area in the anatomical neck of the left humerus.

There are a number of other features of bone necrosis from hyperbaric exposure that still lack an explanation. Although single sites of necrosis are common, there is a striking tendency for the damage to appear symmetrically, and this can be marked in certain individuals. For example, bilateral lesions in the lower end of the femur or upper end of the tibia are very common, but bilateral lesions also occur in the femoral and humeral heads. This suggests that the cause of the necrosis involves the long bones in a much more widespread fashion than the radiological changes indicate. These in fact represent the healing process in dead bone and at the earliest take about three months to appear. At such sites resolution of the initial damage has apparently not been complete and death of bone occurs.

A striking feature of the condition is the large size of many of the lesions as shown by conventional radiography. This is particularly so in the lower end of the femur where the calcified lesions may measure up to 13 or 15 cm in length and 5 or 7.5 cm in width (figure 2, plate 1). Some men who do not admit to having had type I decompression sickness (bends) can be shown to have bone necrosis, while others with a history of frequent bends do not have any evidence of bone damage. Hypersusceptibility or resistance to bone necrosis has not been demonstrated and one cannot identify individuals that are more or less at risk from it. Bone necrosis becomes commoner with increasing experience in diving and with age; where it is possible to separate these two factors it is the experience that is the more important. Maximum depth dived is important and bone necrosis has not been found in the Newcastle centre in men who have never dived deeper than 30 m; but after that there is a progressive increase with

maximum depth dived. It is possible that saturation diving may involve greater risks of bone damage than other forms of diving, but at least in the past saturation divers have been the more experienced men and have therefore done a great many dives of all types.

Pathology

The pathology of the established lesion has been well described (Catto 1976). The histological changes are not peculiar to the hyperbaric condition but show features of aseptic necrosis from other causes. The picture is one of arrested incomplete repair to damaged bone in which in the *juxta articular* region dead bone is separated from the living by a dense collagenous band (figure 3, plate 1). Medullary lesions show marked calcification. Unfortunately the study of the pathology of the lesions has been disappointing in not pointing more clearly to the mechanisms involved.

Theories of causation

It has for long been assumed that bone necrosis is due to infarction from gas bubbles appearing during decompression and interfering with the blood supply to bone. It has been assumed also that this implies a faulty decompression and that bone damage should not occur if the decompression is made correctly. The belief that bends could give rise to bone necrosis in the same area has been held since the first description of bone necrosis in workers using compressed air.

For some time these views have appeared unconvincing. Infarction of such large areas of bone, if that were the case, would suggest that quite large gas bubbles, or large numbers of them, block the circulation in the bigger vessels, or several sizeable vessels, to the affected site. One would then expect infarction elsewhere, for example, in the liver, spleen or kidneys, but there is no evidence that this happens.

Gas bubbles are common if not invariable during decompression, and bone damage and indeed bends can occur even following a decompression made strictly according to accepted good procedures. There is a connection between the bends and bone necrosis as a statistical association but this does not necessarily imply a common cause.

A variety of theories have been put forward to explain the pathogenesis of bone necrosis; they include fat embolism, intravascular erythrocyte agglutination, platelet thrombi, and the osmotic effect of gas dissolved in the tissues. None of these theoretical mechanisms has yet been accepted as a likely cause of bone necrosis. Those that involve embolism in some form can be criticized in the same way as bubble embolism. Recent work in Newcastle upon Tyne has suggested another possible factor. Isolated fat cells have been shown to increase in size on exposure to compressed air, as a result of oxygen toxicity (Pooley & Walder 1981). It is suggested that within the rigid structure of bone this might interfere with blood supply by compressing vessels. Miniature swine that develop bone lesions comparable to those in man on exposure to compressed air, in a manner comparable to tunnellers or caisson workers (Gregg *et al.* 1980), have a statistically significant fall in femoral blood flow in these circumstances. This, it is suggested, might be the effect of an increase in size of marrow cells, which, though insufficient to obstruct blood flow completely, could be an important element in inhibiting the clearance of gas from the marrow during decompression, and contribute to the onset

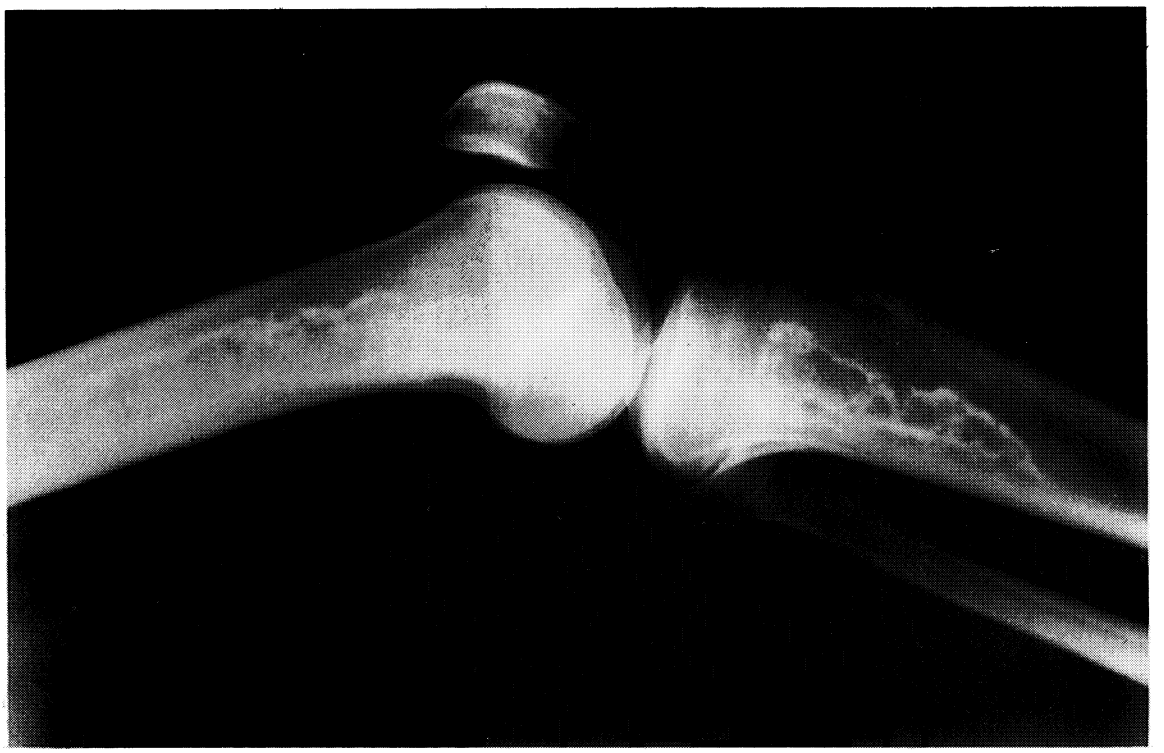

FIGURE 2. Massive symptomless medullary lesions in the lower femur and upper tibia of a professional diver aged 38 years. He had made about 500 air dives, 100 Heliox dives and a saturation dive to 200 m.

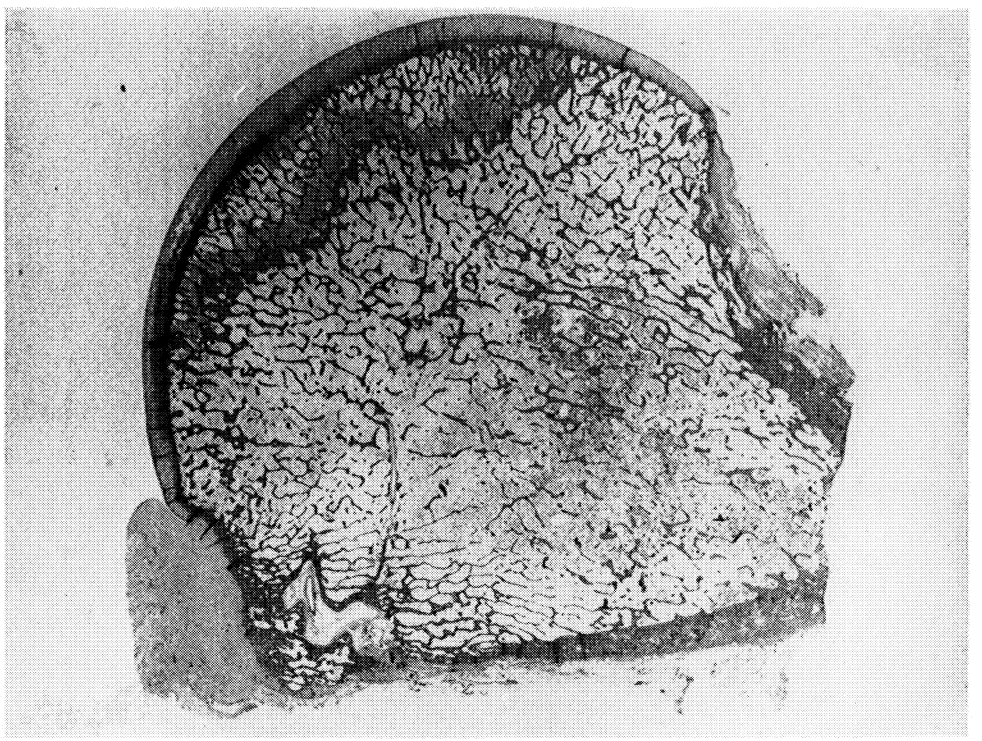

FIGURE 3. Section of head of a humerus of a worker who used compressed air. It shows bone necrosis. The necrotic area lies to the right of the irregular band extending from the top to the bottom of the articular surface. (Courtesy of Dr M. Catto.)

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of necrosis (Thomas *et al.* 1982). Further evidence as to the likely operation of this mechanism is required and an evaluation of other factors that might be contributing. But the hypothesis has the advantage of locating the primary damage anatomically in the bone itself, thus taking into some account the unique quality of bony tissue; its rigidity.

Radiography

Diagnosis is based on conventional radiographic examination of the long bones but because of the delay in the appearance of an abnormal opacity, the study of the circumstances from which the bone damage arose is limited.

Bone scanning or scintigraphy, which is now being used on an experimental basis in divers, promises more precise information. This technique involves the injection of a labelled bone seeking compound (technetium, ^{99m}Tc) whose uptake is increased in active bone lesions. Bone scans made within a few days of decompression from dives to depths varying from 15 to 660 m have shown the presence of bone damage (Pearson *et al.* 1982). Not all the changes detected in this way progress to opacities on conventional radiographs although when they do, the changes recorded are more limited than those shown by the earlier bone scan. Bone scans have not shown any firm relation between symptoms of decompression sickness or the bends and the site of the bone lesion. Nor has there been any good correlation with length of dives, or with any of the many physiological and biochemical measurements that have been made, and the bone scan changes. Some workers have not been confident about the use of bone scanning because of the poor correlation between radiographic changes and the results of scanning. However, a refinement of the bone scan technique using parametric or functional imaging now makes it possible to identify the lesions that will in fact progress to radiographic changes on the basis of diminished medullary vascular perfusion (MacLeod *et al.* 1982). This method involves a gamma camera and computer assisted dynamic scintigraphy to separate two rate constants; one related to amplitude, which measures osteoblastic activity in the lesion; the other related to accretion rates. Where the latter are decreased or absent, implying reduced local microvasculature, changes characteristic of the well defined radiographic criteria will occur in due course. So it is claimed that one can, by this technique, now predict which of the lesions diagnosed soon after their appearance on scintigraphy will progress to necrosis. While this would be an advance on the present diagnostic methods it remains to be worked out how the technique might be generally applied and whether the early knowledge so gained can be used to prevent progress of the lesion to necrosis. For the moment the method seems best suited to monitoring experimental dives rather than for routine use.

Conclusions

Bone necrosis in divers is important in two ways. First, to the individual in whom even a small risk of disability causes concern and second as a general problem in the conduct of compression and decompression in that the occurrence of even a small proportion of bone necrosis, which affects about 4 % of divers, raises doubts about the effectiveness and correctness of current decompression procedures. The usual criterion on which to judge decompression tables has been their ability to prevent acute decompression sickness (types I and II) in the great majority of dives, a proportion of acute decompression sickness being accepted as

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inevitable. A specific attempt to reduce the prevalence of bone damage in workers using compressed air, by redesigning their decompression tables, began in 1966, but it is now evident that cases of bone damage are still occurring. Although in the diving population disability is fortunately rare by comparison and lesions in the head of the femur are unusual, an understanding of the aetiology and pathogenesis of aseptic bone necrosis related to hyperbaric exposure is clearly of great importance not only in preventing possible disability but in increasing our knowledge of the pathophysiology of compression and decompression.

I am indebted to colleagues in the Decompression Sickness Central Registry in the University of Newcastle upon Tyne and to the Health and Safety Executive, which supports it; and to members of the Medical Research Council Decompression Sickness and Diving Panel, for much of the data quoted. Opinions are my own. Figure 1 is reprinted by courtesy of the *Lancet*.

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Discussion

D. H. Elliott (*Shell U.K. Ltd, London, U.K.*). As reported in divers, the only consequences of dysbaric osteonecrosis are those related to sub-chondral collapse of the shoulder or hip joint. Since four of the eight X-rays taken on each diver are of the knees, and since only shaft lesions can be detected in the knees, is it justifiable to continue this investigation? The films are expensive to the diver or his employer, the radiological dosage to the man should not be ignored, and the diagnostic effort required by the radiologists is significant. If the results from X-rays of the knees are ignored, how are the relations affected between the presence of necrosis (*juxta articular* or shaft) and factors such as age, experience, maximum depth and previous decompression sickness?

R. I. McCallum. As the cause of bone necrosis is still unknown it is essential to continue research into the connection between diving exposure and bone damage. In this context presence of *any* bone lesion is important and those around the knee joint are by far the commonest to occur. This, I think, fully justifies the investigation in the wider interests of the whole diving profession in spite of some extra expense.

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With regard to radiation dose, we have looked at this in considerable detail and are satisfied that it is not excessive provided a proper technique is employed and we have published what this should be. It includes gonad protection, which, surprisingly, is still omitted in some X-ray departments, or is badly done.

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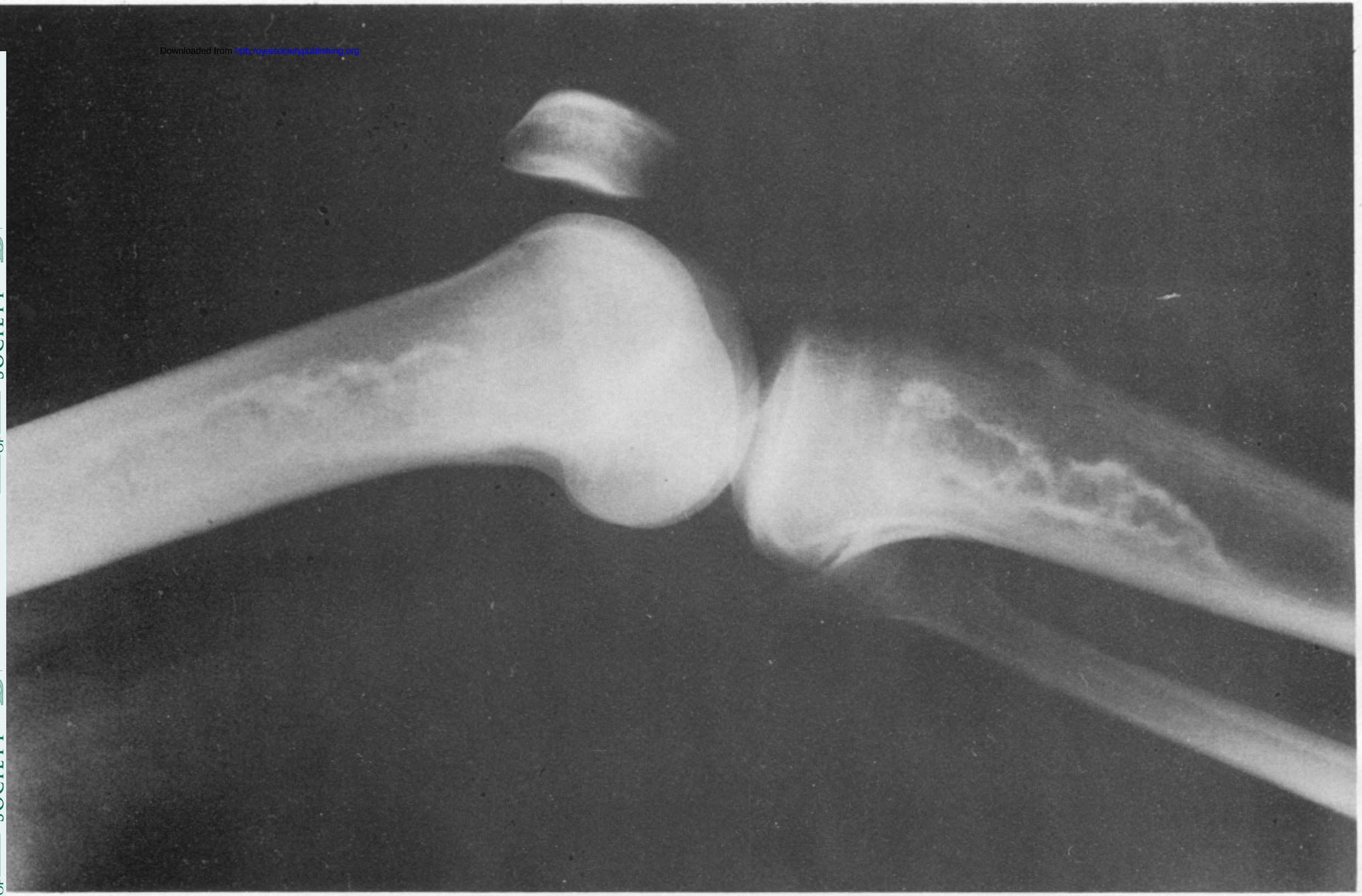

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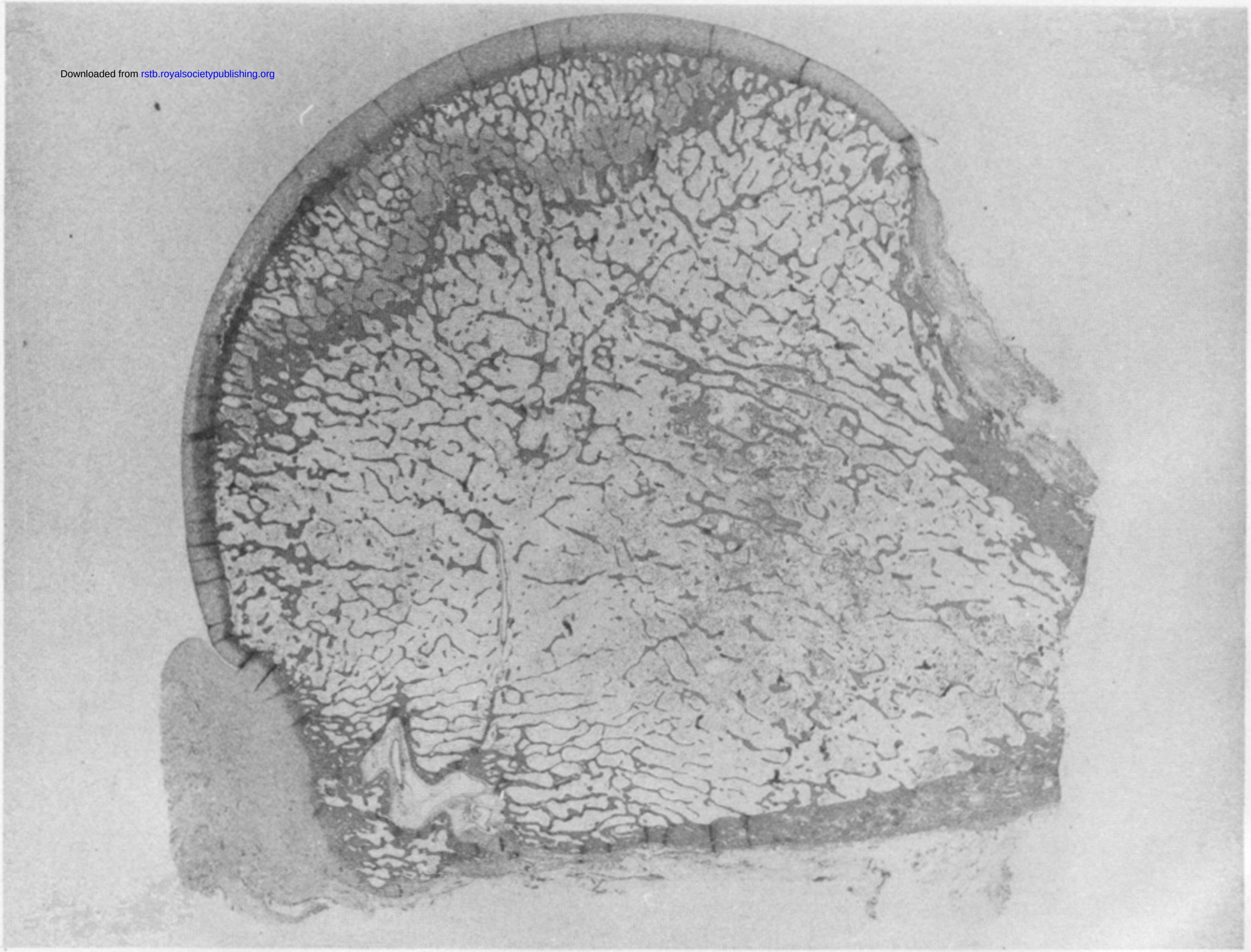

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