MANAGEMENT AND TREATMENT OF OSTEONECROSIS IN DIVERS AND CAISSON WORKERS

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In the matter of treating patients with dysbarism-related osteonecrosis, the following questions arise:

—What advice should be given the individual diver or other compressed-air worker with a diagnosis of dysbaric osteonecrosis *1*) at a site where it is unlikely ever to cause symptoms; and 2) at a site where symptoms may arise, or have already arisen?

—What action may be necessary for a man with hip or shoulder-joint symptoms whose radiographs are normal?

—What is the best treatment for compressed-air workers who have painful and/or disabling lesions?

So little is known about the evolution of the bone changes in osteonecrosis that, at the moment, it is difficult to devise a realistic program of management and treatment. For this reason a central data-gathering organization — such as the Decompression Sickness Central Registry* — is particularly important. It must not be forgotten, as well, that valuable data can often be obtained from the postmortem examinations of compressed-air workers and divers who have died from conditions unrelated to their hyperbaric experience.

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Advice to Be Given to the Patient with Dysbaric Osteonecrosis

Osteonecrosis at a site not likely to cause symptoms. Lesions in the neck or shaft of a bone, or in its head well away from the articular surface, never (as far as we know) cause symptoms. Treatment is therefore not required, but a crucial question must be answered. Is it advisable for the patient to continue to dive or work in compressed air? No compelling evidence exists upon which to assess the probability that a man with radiographically demonstrable bone lesions will suffer additional bone damage, either in the form of new lesions or the worsening of existing ones, if he continues to work under hyperbaric conditions. The man should be told of the possible consequences of further compressed-air exposure so that he can share in the responsibility for the decision about his future work. It is probably reasonable to allow such a man to continue ordinary diving on air. But he should be excluded from unusual exposures, such as may occur in experimental diving.

Osteonecrosis at a site where symptoms may arise. In the case of a symptomless juxta-articular osteonecrotic lesion in the head of a femur or humerus, it seems reasonable to advise the patient to avoid any further potentially dangerous hyperbaric exposures. Practically, this would mean limiting caisson workers' exposure to pressures at which the risk of bone necrosis is believed to be virtually nil — *i.e.*, below 18 psig. A diver so afflicted should be restricted to oxygen diving, in which (so far as is known at present) there is no risk of bone damage. Because the possibility exists that a man with a juxta-articular hip or shoulder lesion will eventually develop symptoms, the risk of developing osteonecrosis in the other femoral or humeral head must be minimized. The consequences of having symptomatic osteonecrosis of both hip or shoulder joints would be catastrophic. Not only would the patient be unable to continue diving or tunnel work, but he would quite likely be incapable as well of performing other types of manual labor, or he might even become totally incapacitated.

When a juxta-articular lesion — particularly in the head of the femur — has progressed to the extent that it causes pain, the patient will probably be unable to work in any case, and will therefore readily accept the advice to give up compressed-air work or diving.

Action Necessary When Symptoms of Osteonecrosis Are Present but X-rays Are Normal

Occasionally a compressed-air worker or diver will complain of a painful hip or shoulder when nothing abnormal can be seen in the radiographs. In these circumstances special radiographic techniques, such as tomography or xeroradiography,

may be helpful. Some orthopedic surgeons believe that venography, biopsy, or radioactive scanning techniques may reveal the presence of osteonecrotic lesions not detectable in X-rays. Whatever technique is used, the primary objective must be to provide the patient with a definite answer about whether he has dysbarism-related osteonecrosis or not.

Treatment of Osteonecrosis

Juxta-articular lesions pose a particularly difficult therapeutic problem. They are usually symptomless until the articular surface is deformed by collapse or indentation, probably because of stress on the damaged area. Thereafter, pain upon movement of the limb is a prominent symptom. If the man is allowed to continue work without treatment, he will almost certainly develop arthritis in addition to the original lesion. The joint will then become so disorganized that it will not only be painful but also severely restricted in movement.

Experience shows that necrotic bone collapses where the greatest stress occurs. One might therefore suggest that, when the early signs of a lesion underlying the articular surface of a bone are found, the joint should be protected from load-bearing; there is always the possibility that lesions can heal spontaneously. However, since relief of load-bearing could mean putting the patient to bed for several months, this treatment cannot be undertaken lightly.

Figure 1 shows the radiographs of a shoulder joint with just a suspicion of a lesion, which becomes more definite in nine months, and then, six months later, is no longer visible. Apparently it has healed. Caution in the last diagnosis is necessary, however, because the radiological technique used in the three films may not have been consistent. If this humeral head were to be examined again by tomography, a lesion might still be seen.

Another difficulty is that, although serial radiographs can demonstrate a lesion growing progressively worse, it is not possible to forecast from a single film whether a particular lesion will progress or remain stationary. In Fig. 2*a*, for example, is the radiograph of a humeral head taken in February 1967, in which changes suggestive of osteonecrosis are very doubtful. But only four months later, the lesion has developed to such an extent that the whole humeral head has disintegrated (Fig. 2*b*). These films are a graphic demonstration of how rapidly some

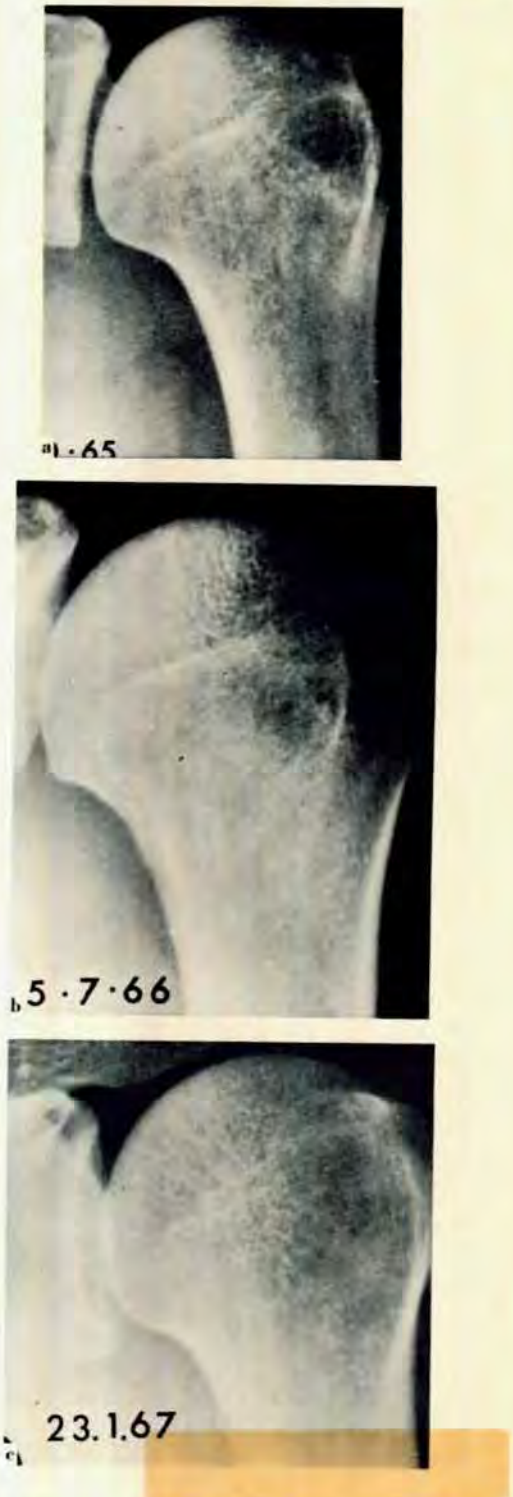

FIG. 1. Apparent healing of osteonecrotic lesion in humeral head of compressed-air worker, showing (*a*) very early lesion under cortical margin; (*b*) more definite lesion 9 months later; and (*c*) 6 months later, when lesion is no longer visible.

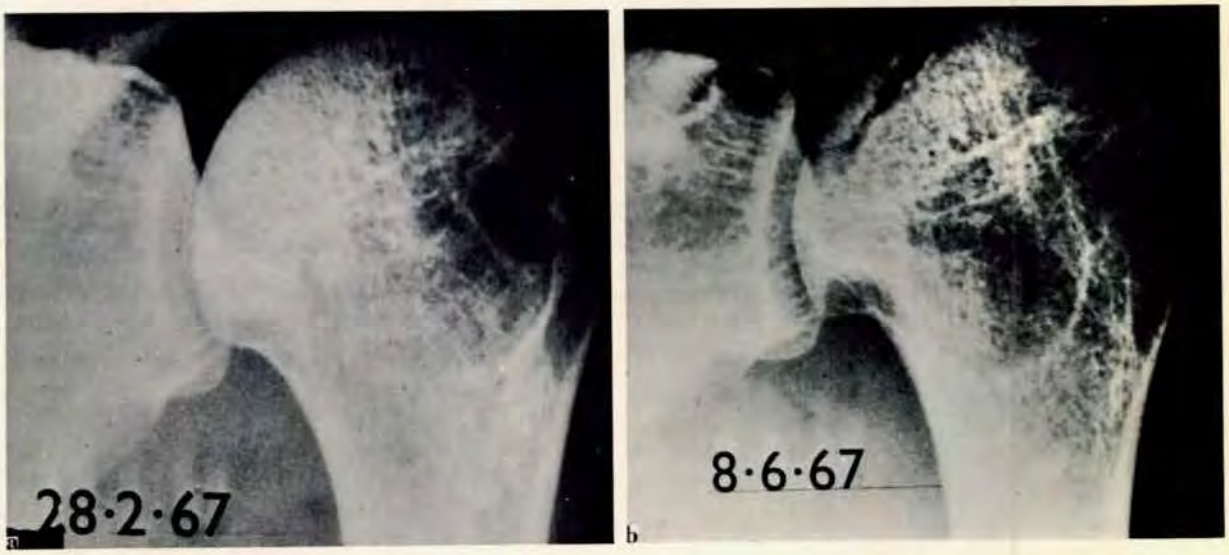

FIG. 2. Rapid progress of osteonecrotic lesion, in which there are (*a*) changes suggesting early lesion; and (*b*) disintegration of humeral head 4 months later.

comparatively mild-looking lesions can advance.

Some lesions, by contrast, remain static for many years. In Fig. 3*a*, a definite lesion can be seen in the head of a humerus; five years later (Fig. 3*b*), the lesion is virtually unchanged. Selection of those subjects who might benefit from relief of load-bearing is therefore not possible at present.

The radiographs shown in Fig. 4 illustrate the progression of a lesion to the point that operative treatment becomes essential. The first shows the humeral head of a man 3 months after he ceased

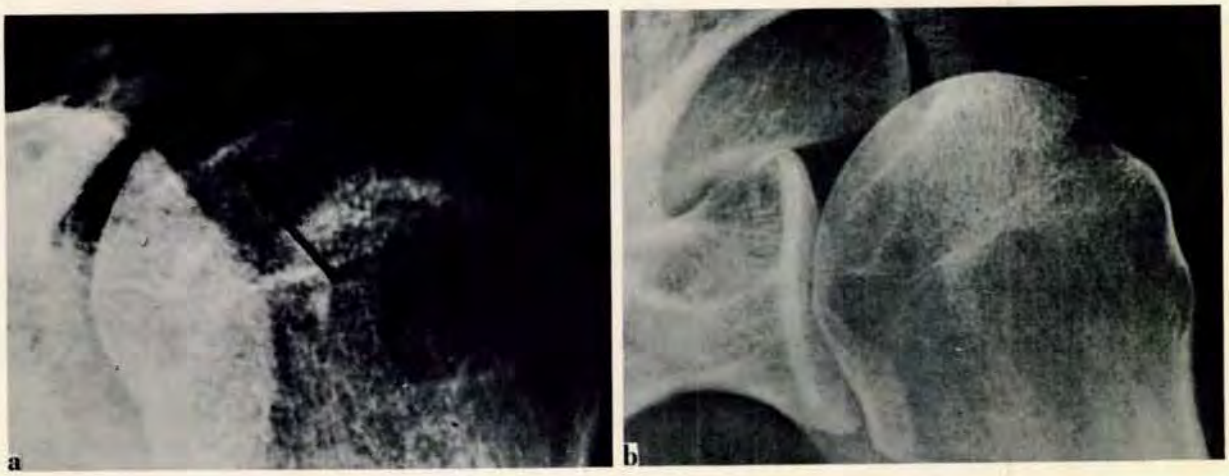

FIG. 3. Humeral head showing static osteonecrotic lesion (*a*) as diagnosed in 1962; and (*b*) 5 years later, in which no change is evident.

FIG. 4. Roentgenograms showing progression of osteonecrotic lesion severe enough that operative treatment is necessary: (*a*) bone island only, 3 months after patient ceased work in compressed air; (*b*) definite dysbarism-related osteonecrosis, in addition to bone island, 19 months after last compressed-air exposure; and (*c*) fragment of bone breaking away, with bone island still present, 22 months after last exposure.

working in compressed air. The small dense area just below the cortical margin is a bone island and is of no consequence. About 16 months later, however — 19 months after his last exposure — there is a definite osteonecrotic lesion, but the bone island has remained unchanged. Then, 22 months after his last compressed-air experience, a piece of necrotic bone is beginning to crack off. The bone island is still unchanged. The orthopedic surgeons ultimately reattached the fragment of bone with a screw and the result was fairly satisfactory. Unfortunately, however, the patient has now developed a lesion in the other shoulder.

(Incidentally, this series of X-rays illustrates the difference between a patch of osteonecrosis and a bone island. The bone island remains static over the years, whereas the lesion gradually progresses until the cortex of the bone collapses, giving rise to symptoms.)

Once operative treatment is indicated, the procedure to be adopted must be considered. But it must be borne in mind that surgical treatment in these cases is not always completely satisfactory.

In our recent experience, 12 patients with a total of 16 joints damaged by osteonecrosis have been operated on. They were all young — around 30 years of age. It was therefore felt that radical treatment, such as total hip-joint replacement (in which both the head of the bone and the articular cup into which it fits are removed and replaced by a prosthesis), was not justified. For the estimated life of an implanted joint prosthesis is only about 10 years.

Techniques less radical than hip replacement were therefore tried. In three patients the osteonecrotic lesion of the femoral head was drilled into from below. Pegs of bone were then inserted through the lesion to shore up the articular cortex (Phemister, 1949; Bonfiglio and Bardenstein, 1958). The results, however, have been unsatisfactory. In one patient it has already been necessary to remove the femoral head and insert a prosthesis. This same procedure will soon have to be followed in the other two patients.

In another two patients with lesions affecting the femoral head, drill holes have been made through the osteonecrotic area of bone into the underlying normal bone to provide channels along which revascularization can occur. Again the results have been very poor, as the patients have gained neither in freedom from pain nor in movement.

Two patients with sequestration of the humeral head have been treated by fixing the displaced necrotic fragment into place with a screw. These results have been encouraging; both men have remained symptom-free and have quite good mobility. Unfortunately, this surgical procedure is not advisable or, perhaps, even possible in the hip because of the problem of weight-bearing. A repair involving screws is not likely to be strong enough to resist the severe stresses encountered in the hip.

Another measure that has been tried in three patients in which sequestration has occurred was simply to remove the dead flake of bone. Although the one hip and two shoulders treated continue to give pain, their mobility is quite good.

The hips of four patients and the shoulder of one have been treated by partial replacement of the joint; only the necrotic head of the bone was replaced by a prosthesis. The results have been extremely satisfactory, although it appears that one hip will eventually need total replacement.

In one case arthrodesis was carried out. In this operation the humeral head was fixed to the shoulder blade (the bone with which it articulates), mobility thereafter depending on mobility of the shoulder blade. This form of treatment has proved satisfactory for the relief of pain and, at the same time, has allowed a surprising amount of movement.

It is clear from this account that, unfortunately, there are too many unknown factors at present to deal satisfactorily with the orthopedic problems posed by dysbaric osteonecrosis. More fundamental information about the initiation, development, and progression of osteonecrotic lesions is urgently required.

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