Avascular necrosis of the femoral heads in a compressed air worker / by R.I. McCallum [and others].

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AVASCULAR NECROSIS OF THE FEMORAL HEADS IN A COMPRESSED AIR WORKER

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Chronic joint disease from work in compressed air was first described by Bornstein and Plate (1911-12) and by Bassoe (1911) independently; and necrosis of the shaft of the femur was reported earlier by Twynam (1888). Bassoe (1913) was probably the first to distinguish between trophic bone changes secondary to the cord lesions of caisson disease, and chronic arthritis resulting from the local liberation of gas during decompression. The pathology of bone and joint lesions in compressed air workers was described nearly thirty years later by Kahlstrom and others (1939a), who reviewed the previously reported cases and added four of their own. Pathologically, there is multiple infarction of large or small parts of long bones, with new bone formation and calcification in weight-bearing areas, and later degenerative arthritis, sometimes with loose bodies. In parts of the bone away from the joints there is invasion with new bone formation and then encapsulation with a fibrous wall, which may become calcified and ossified. The bone lesions are usually multiple and have a typical radiographic appearance.

Since 1939 cases have been reported from various countries and it seems that the condition is a not uncommon sequel to work in compressed air, particularly if decompression has been too rapid (Twynam 1888, Gordon and Heacock 1940). Although pain from "bends" in joints subsequently affected by arthritis is common, this is not essential to the later onset of gross arthritis (Bell, Edson and Hornick 1942, Taylor 1944). Bone lesions are characteristically multiple (Coley and Moore 1940, Taylor 1944), and many are clinically silent, particularly those involving the shafts of bones, but if articular surfaces are involved there are usually symptoms (Swain 1942, Allan 1943). Long periods may elapse between exposure to high pressure and the onset of symptoms, and also between the onset of symptoms and diagnosis (Kahlstrom *et al.* 1939a, Walker 1940). The correct diagnosis depends on a complete occupational history that includes the whole working life, for exposure to compressed air may have occurred many years before and it is probable that the true origin of many of these cases is not recognised at all. Similar lesions are sometimes seen in patients who have never worked in compressed air (Kahlstrom *et al.* 1939b, Kahlstrom 1942, Taylor 1944), but these are less likely to be multiple. The pathogenesis of these cases is obscure and fat embolism or arteriosclerosis of nutrient vessels has been suggested as a possible cause. In compressed air workers it is suggested that injury plays a part in selecting the site for major lesions, and Gordon and Heacock (1940) asserted that the predilection for the hip and shoulder regions supports this. We cannot believe that the hip and shoulder are more often subjected to trauma than, for instance, the knee, but there can be no doubt that once infarction has occurred, trauma, or even normal weight-bearing, will influence the extent and nature of the destruction.

There is at present no agreement on the pathogenesis of avascular necrosis found in compressed air workers. Nitrogen emboli in the vessels of the epiphysis have been suggested, or gas formation in the intramedullary fat. Experimental rapid decompression of rabbits produces cavity formation in the bone marrow from nitrogen bubbles, and this may be an early stage in the process (Colonna and Jones 1948). Behnke (1951) thought that the casual

relationship between the lesions and embolic injury must be supported by further experiments and pointed out that in divers suffering repeatedly from experimental "bends" radiological bone changes have not been observed. In this instance the follow-up may not have been long enough.

Few of the reported cases have been treated except by symptomatic means. Arthrodesis and removal of part of the head of the femur has been performed (Walker 1940) and in another case a reconstructive operation on one hip (Rendich and Harrington 1940), but little detail is given.

There have been only two published reports from Great Britain of bone lesions after decompression, the first in a caisson worker who died from coronary thrombosis (Swain 1942). Necropsy was performed and he was found to have avascular necrosis of both humeri. There had been pain in the right hip in life but this was not examined. The second report described three survivors of a submarine disaster in whom radiographic bone changes were found twelve years later. In one man there was collapse of both femoral heads with osteoarthritis. These cases are of great interest because they all developed after a single decompression (James 1945).

THE TYNE TUNNEL

Between 1948 and 1950, while a tunnel was being constructed under the river Tyne between Howden and Jarrow, leakage of water through a porous river bed necessitated working in compressed air, at a pressure of about 35 pounds a square inch. Precautions as laid down in the Provisional Revised Draft (June 1948) of Regulations as to Safety, Health and Welfare in connection with Work of Engineering Construction (Factories Act, 1937) were taken to ensure proper decompression of all men leaving the tunnel. The incidence of "bends" was less than 2 per cent per week and a separate medical lock was used for its treatment. There were two serious cases of paraplegia, and the unusual circumstances in which they occurred are described elsewhere (McCallum and Walder 1953). Since then, one man has developed severe hip joint disease and this case is described here.

CASE REPORT

A caulker, aged 30 years, first attended in May 1950, complaining of aching pains from below the knees to the hips. They began gradually about six weeks before and were most severe in the front of the thighs, especially the right, with stiffness of the legs and difficulty in walking. Apart from these pains he was perfectly well, and his only previous illness of note was malaria. His previous employment had been that of a truck driver, and he had done six years of military service. He had also spent four months in the Battersea Tunnel at a pressure of 8 pounds a square inch, without ill effects. He stated that whilst employed on the construction of the Tyne Tunnel he had suffered from "the niggles*" on several occasions. In about ten months he worked 272 shifts, each of about six hours, followed by decompression lasting one hour and three-quarters. He had "bends" on three occasions, after 68, 114 and 126 decompressions respectively. These attacks occurred after work at pressures of 35-40 pounds a square inch, and they consisted of stabbing or aching pains in the thighs and knees, mainly on the right side, which came on up to nine hours after decompression and were relieved by recompression for five or ten minutes. On the last occasion pain recurred two hours after recompression and he had to return to the medical lock a second time. Shortly after he had ceased working in compressed air altogether, he had felt a weakness in the right knee with slight stiffness in the hips and thighs, and a sensation of loss of balance, but he thought little of it at the time.

On examination his general condition was good and there was no wasting of limbs or trunk. He walked with a stiff spastic gait, turning in the toes, and had difficulty in getting

* "The niggles" is a term used by compressed air workers for the pains of a mild attack of "bends."

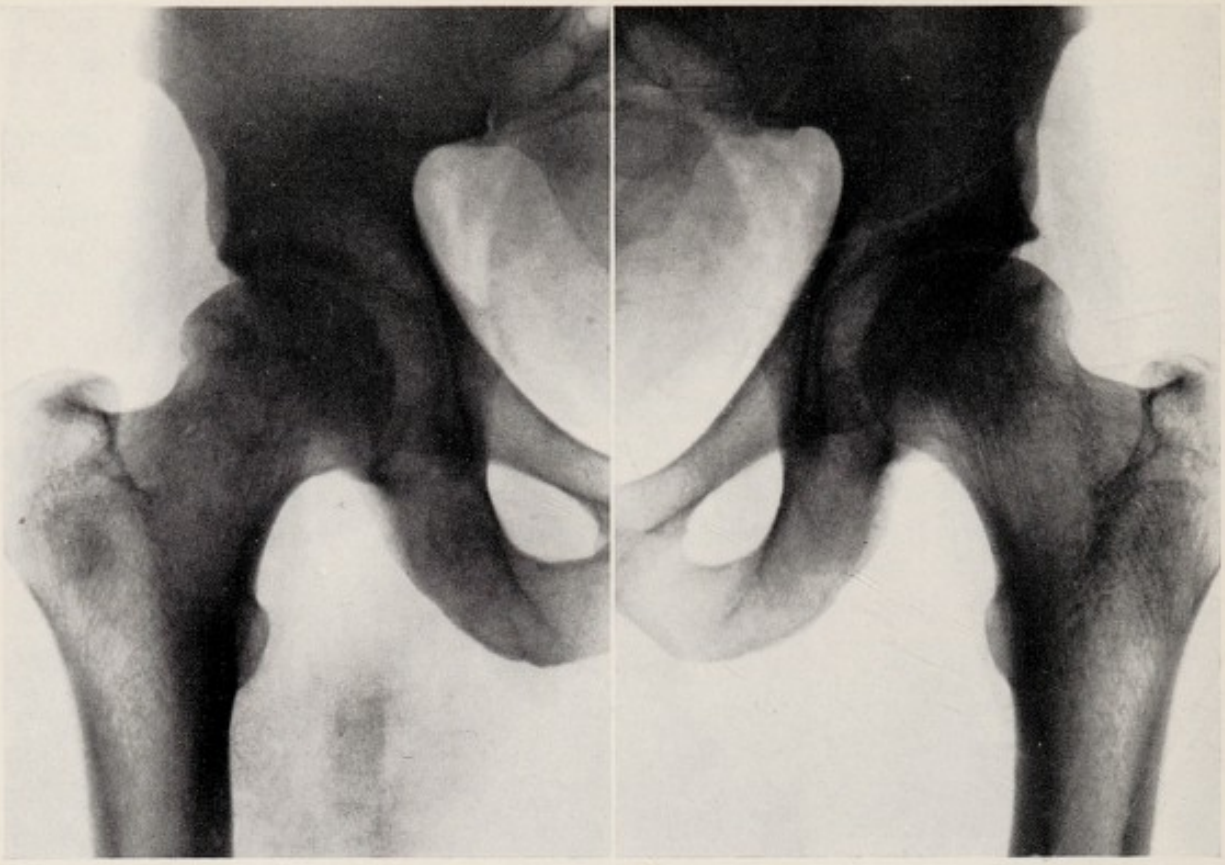

FIG. 1

Initial radiograph of hips, taken six months after the patient had ceased working in compressed air.

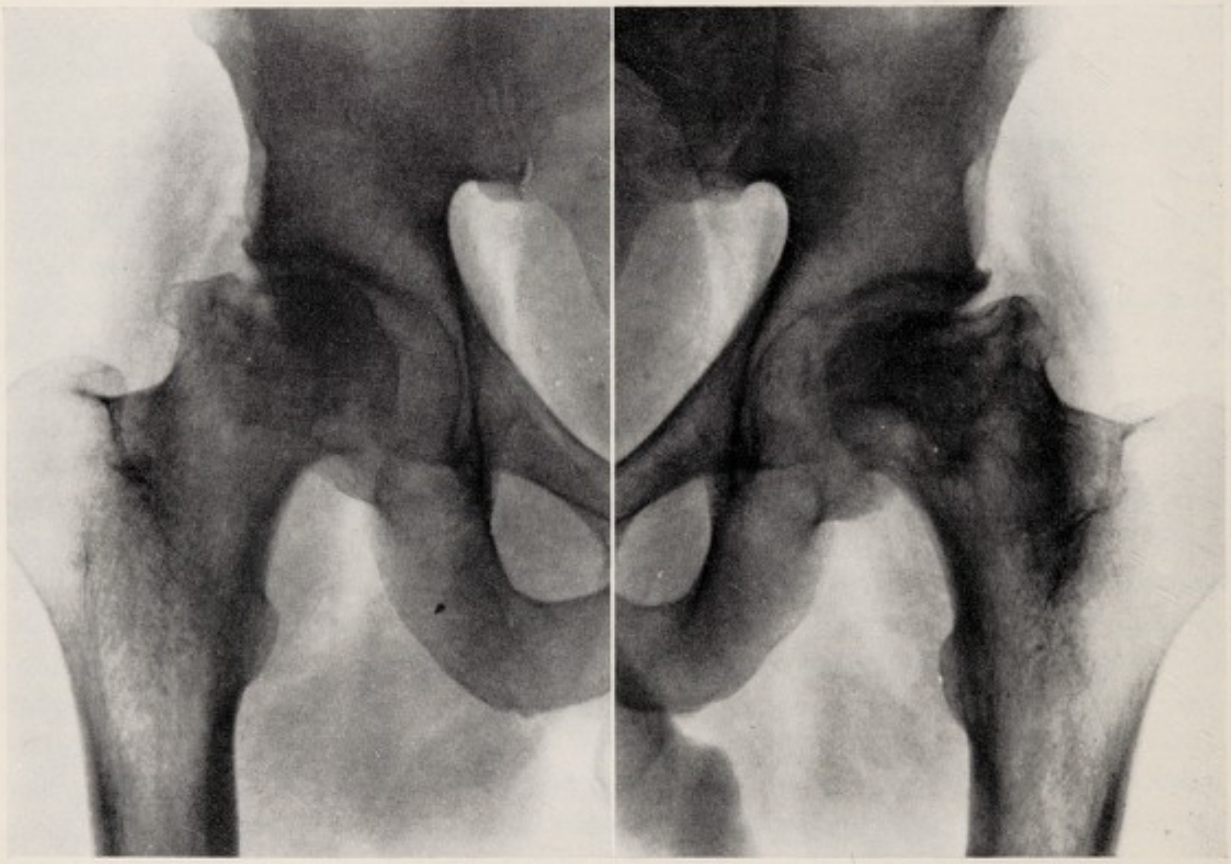

FIG. 2

Condition twenty-one months later.

on or off a couch, or in sitting up from the lying position. Passive movements of the lower limbs produced pain in the groins and knees and the limbs felt stiff. No abnormality was found in the lungs, heart, or alimentary system; the blood pressure was 135/70 and the central nervous system was normal, with brisk but equal limb reflexes. There was no fever, and the blood sedimentation rate was only 3 millimetres (Westergren). On radiography six months after he had left compressed air work the femoral heads were thought to be normal (Fig. 1). No definite diagnosis was made at that time and he was given symptomatic treatment with massage and exercises to the lower limbs.

There seemed to be some temporary improvement after this, but later the pain and disability became worse until his legs became so weak that he could not flex them. On one occasion he fell down and could not get up. In February 1952, twenty months after the onset of symptoms, further radiographs showed marked arthritis of both hips, and orthopaedic advice was sought. His general condition was still good, but the man was severely disabled. He could just manage to move about with the aid of crutches, but had to be helped into and out of a chair.

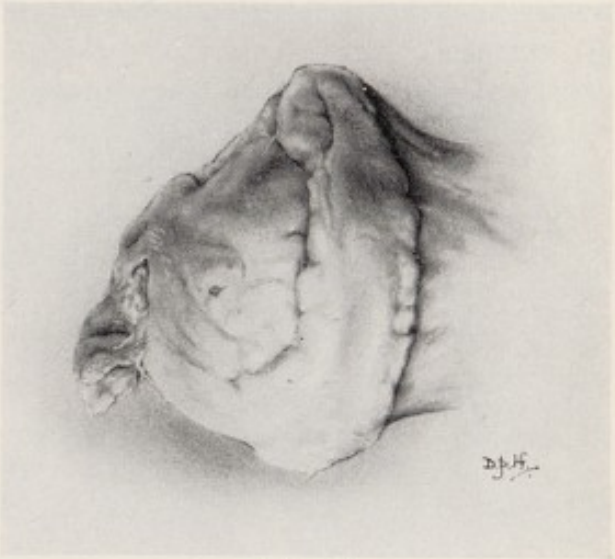

FIG. 3
Drawing of the left femoral head removed at arthroplasty.

Movement of the hips caused severe pain. The range on the right side was: flexion 30 degrees, abduction nil, adduction 5 degrees, rotation nil. On the left side there was flexion 70 degrees, abduction 50 degrees, adduction 30 degrees, rotation nil. Radiographs showed gross destructive changes in the heads of both femora. The right acetabulum was normal, and the only change in the left was a minor degree of lipping of the upper margin. Though the changes extended throughout the femoral head, the greatest deformity of the articular surface was on the superior aspect, which is normally subjected to the greatest weight-bearing pressure. The radiological findings resembled those seen in avascular necrosis from severe damage to the blood supply to the femoral head (Fig. 2), and were not at that time characteristic of osteoarthritis. This, together with the history of work in compressed air, suggested a diagnosis of caisson disease of the hips. Radiographs of the rest of the skeleton showed no other lesions.

Treatment—In May 1952 a Judet acrylic arthroplasty was performed on the left hip, through an anterior Smith-Petersen approach. The capsule was found to be greatly thickened and the synovial membrane was villous. The joint cavity contained an excess of synovial fluid and several small cartilaginous loose bodies. The femoral head (Fig. 3) was distorted, the articular cartilage degenerate and, in parts, necrotic, and there was marginal lipping, but the acetabulum appeared to be little affected. The capsular and synovial tissues were excised as completely as possible, and after replacement of the femoral head with the prosthesis the joint seemed stable (Fig. 4). The patient made an uneventful recovery. In July 1952 a similar operation was performed on the right hip, with similar findings. When his rehabilitation was completed, in December 1952, the patient had no pain in either hip, and flexion was limited by only 20 degrees. Since then he has worked regularly as a bus driver.

Pathological examination—The right femoral head was flattened, with a central shallow depressed area of irregular outline covered by thin cartilage. There was considerable osteoarthritic lipping. Section showed an area of avascular necrosis corresponding to the surface depression; it had the characteristic wedge-shape of an infarction, and this was confirmed microscopically. There were no gross changes in the arteries of the ligamentum

teres. The appearances were those of avascular necrosis such as may be found in caisson disease.

DISCUSSION

Avascular necrosis of bone may occur from time to time without known cause, but the onset of severe destructive changes in the femoral heads of a man of 30 within twenty-one months of work in compressed air suggests that this was the direct cause. Pain in the hips began only five months after his work in the tunnel had finished, and while working in the pressure he had " bends " affecting both lower limbs, though not to a degree that could be considered unusual in a compressed air worker.

The prevention of bone disease in compressed air workers depends partly on a fuller understanding of the pathogenesis. Some cases have resulted from too rapid compression

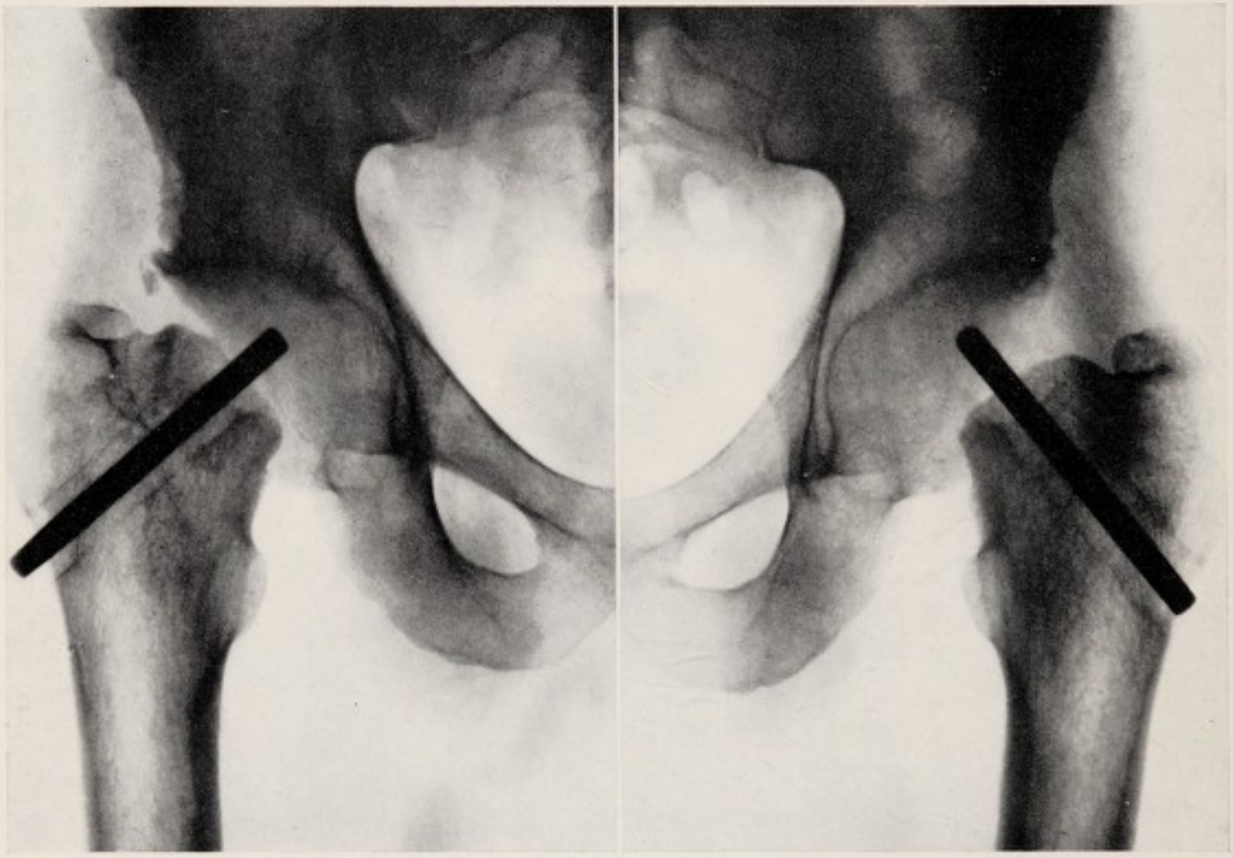

FIG. 4
Radiograph of hips after bilateral Judet arthroplasty.

and they emphasise the need for strict control of decompression procedures and for a high standard of discipline among the workers. Prevention is made more difficult because bone changes can occur without previous " bends " or after a single rapid decompression. Decompression of our patient was carried out according to regulation, but it is probable that bone lesions, like other forms of decompression sickness, cannot be prevented entirely by decompression according to standard tables, and that individual susceptibility plays a part. It is possible that bone disease occurs more frequently than is recognised; and, in cases of unexplained avascular necrosis, particularly in the humerus or femur, a history of work in compressed air should be enquired into.

It has been suggested (Milne 1948) that if all joints in which an attack of " bends " had occurred were always examined radiologically eight or ten weeks afterwards, the condition could be detected, and that immobilisation of the joint might lead to revascularisation of the sequestrum with complete cure. Brailsford (1944) stated that radiographs might show

evidence of disturbance of the vascularity of a fragment of bone within six weeks of the causal incident, but one of us (J. K. S.) has recognised such evidence at three weeks.

It is noteworthy that six months after this patient ceased work in the tunnel, at a time when he was already suffering severe pain, the radiograph of the hips (Fig. 1) showed no abnormality, but twenty-one months later gross changes in both femoral heads were apparent (Fig. 2). From our experience of avascular necrosis in bone from other causes (for instance, osteochondritis dissecans), immobilisation of the joint and protection from weight bearing is not a practical possibility, because complete revascularisation, if it ever occurs, would probably take years rather than months.

Judet arthroplasty is of particular value in a case of this type because the femoral head has been destroyed, and therefore cup arthroplasty is impracticable. Furthermore, it is undesirable to sacrifice the articular cartilage of the acetabulum which, in the early stages of this condition, is little affected. The Judet arthroplasty permits complete study of the damaged femoral head, previously impossible. However, although arthroplasty is undoubtedly the operation of choice in bilateral disease, arthrodesis might still be the correct procedure in a unilateral case.

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