ASEPTIC NECROSIS OF BONE IN CAISSON DISEASE

BY

JOHN D. THOMSON and ANDREW B. YOUNG

From the Royal Infirmary, Glasgow

(RECEIVED FOR PUBLICATION MAY 17, 1958)

Caisson disease, or decompression sickness, is encountered when a raised atmospheric pressure on the body is reduced too rapidly and excess gases which have been held in solution in the blood and tissue fluids are set free as their solubility decreases. Typically the condition is manifested by tingling and paraesthesia, itching of the skin, and by bone and joint pains. The symptoms vary within wide limits, the majority being of a mild nature and in most instances relief is obtained by prompt recompression. The fact that permanent changes can take place in bone has, however, been recognized for some time. Twynam (1888) reported a case of necrosis of the shaft of the femur. Bassoe (1913) and Kahlstrom, Burton, and Phemister (1939a) described degenerative joint lesions with adjacent bony infarcts.

Such lesions are not common. Ronald (1953) described infarcts involving the lower end of both femora, the upper end of both tibiae, and both humeral heads in a man who had been working as a diver for some 40 years. McCallum, Stanger, and Walder (1954) reported a case of necrosis of the femoral head in a caisson worker engaged in the construction of the Tyne tunnel. In reviewing the literature they found only two reports from Great Britain of bone infarction following decompression. One was in a caisson worker who died of acute cardiac ischaemia and at necropsy was found to have an avascular necrosis of both humeri (Swain, 1942). In the second report James (1945) gave an account of his findings in three of the five survivors of the ill-fated H.M. submarine *Poseidon* which sank after a collision in 120 ft. of water in 1931. The survivors escaped by use of the Davis apparatus and suffered in varying degree from "bends" immediately or shortly after surfacing. When examined 12 years later one had a lesion of the left humeral head, another had both femoral heads involved, while the third had an infarct of the left hip and left shoulder. In view of the rarity of this complication

of caisson disease the following case may be of interest.

Case Report

The patient first worked under increased atmospheric pressure in 1954. At that time he was employed in the building of a power station and in the course of five weeks had occasion to enter the pressure chamber about twice a day for periods of about 10 minutes each time. The pressure inside the chamber was 32 lb./sq. in. On each occasion the patient himself controlled the decompression, judging the rate by the feeling in his ears. He suffered no ill effects at this time.

In July, 1955, the patient was again employed in the caisson. He states that for five days he worked a shift of 12 hours at a pressure of 15 lb./sq. in., being allowed out of the chamber for two 20-minute periods each day. Decompression was controlled by the shift workers themselves. After a break of 48 hours the patient began work again on an eight-hour shift, the pressure having been increased to 20 lb./sq. in. Meals were taken inside the chamber. About three hours after this shift and while lying in bed, he was suddenly seized by severe pains in both upper arms, both hips, the right knee, and the right mastoid area. The pains came on instantaneously, were deep and boring in character and almost unbearable in their severity. With the aid of codeine tablets the pain became less severe and as his job at this time was not heavy, he was able to continue with his regular work in the caisson for two more days. By that time the pains had almost disappeared from everywhere except the right hip, but because of its crippling nature, he was forced to stop work. Over the next week the pains disappeared completely from the other areas but persisted in the right hip. During the next two years the patient was employed intermittently on a number of light jobs, but became increasingly crippled by the pain in the right hip, and the apparent weakness of the right leg.

Examination.—The patient is a strongly built man of 37 years of age. He walked with a slight protective limp on the right side, taking his weight on the ball of the foot. Posture was good and there was no spasm of the erector spinae. Forward flexion of the spine was limited by 25%, but other spinal movements were free. There was

obvious gluteal wasting on the right side and the Trendelenburg hip test was weakly positive on that side. Visible wasting of the right quadriceps was present and proved on measurement to be 1 inch. Movements of the right hip were all restricted; flexion 100°, internal rotation 15°, external rotation 15°, abduction 15°, adduction 15°. There was weakness of the right knee jerk, but no other abnormal neurological finding. General examination revealed no significant abnormality, and other joint movements were free.

Blood examination gave the following results:

Plasma inorganic phosphorus = 4.1 mg. per 100 ml. (2.4 mEq./litre), serum calcium = 11 mg. per 100 ml. (5.5 mEq./litre), plasma alkaline phosphatase = 1.2 Bodansky units. A radiograph of the right hip (Figs. 1 and 2) showed a sclerotic area involving the inferior aspect of the head and upper part of the neck of the right femur, consistent with the appearances of avascular necrosis. The radiographic appearances of the shoulders, knees, and left hip were normal.

Discussion

At present there does not appear to be any agree-

ment on the precise pathogenesis of avascular necrosis in caisson disease. Of the gases released on decompression, oxygen is readily utilized and carbon dioxide is exhaled through the lungs. The excess nitrogen is not disposed of so readily; it tends to accumulate in the tissues of the central nervous system or in the marrow of bone where the lipoid and fat content are high. Nitrogen emboli in the vessels of the epiphysis have been held by some (Colonna and Jones, 1948) to be the cause, but others (Kahlstrom *et al.*, 1939a) doubt if this is so. Kahlstrom *et al.* (1939b) were unable to reproduce the lesion experimentally in animals. It seems reasonable that the infarct may be due to an accumulation of nitrogen at a particular site in bone causing local ischaemia by pressure on vessels. An additional factor suggested by End (1938) is that external pressure tends to increase the intravascular agglutination of erythrocytes, thereby leading to a local thrombosis of the smaller vessels. The bone lesions, by their nature, may be silent, especially if they

FIG. 1

FIG. 2

Figs. 1 and 2.—Radiographs of right hip, showing extensive destruction of the head and upper inferior aspect of the neck of the femur, with surrounding sclerosis.

occur in the shafts (Coley and Moore, 1940). In our case it is interesting to note that the pain of "bends" was most noticeable in the region of the right hip and it remained at that site after recompression and persisted after the other pains had gone. It seems probable that more frequent radiographs of the bony parts involved would lead to an earlier recognition of the lesion and to a more accurate assessment of its incidence.

We should like to express our thanks to Mr. P. Kelly for his reproduction of the radiographs.

REFERENCES

- Bassoe, P. (1913). *Amer. J. med. Sci.*, **145**, 526.
 Coley, B. L., and Moore, M. (1940). *Ann. Surg.*, **111**, 1065.
 Colonna, P. C., and Jones, E. D. (1948). *Arch. Surg. (Chicago)*, **56**, 161.
 End, E. (1938). *J. industr. Hyg.*, **20**, 511.
 James, C. C. M. (1945). *Lancet*, **2**, 6.
 Kahlstrom, S. C., Burton, C. C., and Phemister, D. B. (1939a). *Surg. Gynec. Obstet.*, **68**, 129.
 ———, ——— (1939b). *Ibid.*, **68**, 631.
 McCallum, R. I., Stanger, J. K., and Walder, D. N. (1954). *J. Bone Jt Surg.*, **36b**, 606.
 Ronald, J. (1953). *Lancet*, **2**, 855.
 Swain, V. A. J. (1942). *Brit. J. Surg.*, **29**, 365.
 Twynam, G. E. (1888). *Brit. med. J.*, **1**, 190.

THE JULY (1958) ISSUE

The July (1958) issue contains the following papers:—

The Accuracy of Occupational Vital Statistics. By M. A. Heasman, F. D. K. Liddell, and D. D. Reid.

The Influence of Clothing on the Pattern and Severity of Burns Studied in Some Recent Colliery Fire-damp Ignitions. By M. N. Tempest and J. B. Atkins.

The Toxic Properties of Demeton-methyl ("Metasystox") and Some Related Compounds. By M. Vandekar.

Tissue Reactions Produced by Calcium Fluoride in the Lungs of Rats. By E. J. King, M. Yoganathan, and G. Nagelschmidt.

The Tissue Reaction in the Lungs of Rats after the Inhalation of Coal Dust Containing 2% of Quartz. By E. J. King, S. Zaidi, C. V. Harrison, and G. Nagelschmidt.

Pneumoconiosis of Coal-miners in Three Northumbrian Collieries. By R. I. McCallum and D. J. Newell.

Industrial Dermatitis in the Coal-miner. By O. P. Edmonds.

Lesions of the Skin in Process Workers Caused by Contact with Butyl Tin Compounds. By W. H. Lyle.

Climate and Health in the Royal Navy. By F. E. Smith.

The Emotional Stability of Miners with Nystagmus. By Judith S. Lion.

A Case Illustrating the Effect of Calcium Disodium Versenate (CaNa₂E.D.T.A.) on Chronic Mercury Poisoning. By S. M. Woodcock.

A Case of Beryllium Disease. By C. H. Wood, K. P. Ball, and N. Donald Teare.

Book Reviews.

A number of copies are still available and may be obtained from the Publishing Manager, British Medical Association, Tavistock Square, W.C.1, price 17s. 6d.