AVASCULAR BONE NECROSIS IN DIVERS

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INTRODUCTION

The place of acute decompression sickness as one of the major problems of diving has now been taken by a far more potentially crippling condition, that of avascular bone necrosis. There is no effective known treatment at present time and only avoidance may be used to prevent it. It is a condition that affects not only standard and self contained divers, but also caisson workers, hyperbaric chamber workers and, rarely, aviators. Significantly, it is now been found in increasing incidence in sports divers. Previously, only professional divers had been investigated for this condition.

HISTORY

In 1888 Konig described “Silent Necrosis of bone”. He was probably referring to ‘osteochondritis dissecans’, but he is credited as being the first to describe avascular necrosis of bone. Bornstein and Platt[1] in 1912 described a condition of bone affecting caisson workers, and in 1941 Grutszmacher[2] described necrosis of bone in standard divers. It was not until the late 1950’s and early 60’s that research workers started taking an immense interest in the bone necrosis affecting divers and from then on up until the present time, many surveys were conducted and are still being conducted, into bone necrosis affecting divers. The aetiology of avascular necrosis of bone is very varied as may be seen from Table 1.

The necrosis caused by increased corticosteroid administration for immunosuppression in organ transplantation and particularly that of kidney transplantation, has been causing an increased number of cases of bone necrosis to present. It has been known for a very long time that trauma such as fractures of the neck of femur, dislocations of the head of femur, fractures of the scaphoid, etc have been liable to cause avascular necrosis of bone but decompression is now being considered as one of the commonest causes of avascular necrosis of bone in the particular group of personnel that we have been considering and has merited the title of Dysbaric Osteonecrosis. There are a number of hyperbaric factors that have been associated with Dysbaric Osteonecrosis but none of them have been shown directly to be the cause of bone necrosis. Table 2 shows some of the factors concerned.

For example, in divers over the age of 35, necrosis has been found to be more frequent in those who have dived relatively deep or who have suffered some form of decompression sickness, but most of the evidence so far suggests relatively inadequate decompression as a precipitating factor whether or not that decompression lead to symptomatic acute decompression sickness[3]. In Table 3 some of the diving factors are shown, the lowest pressure as yet described as producing Dysbaric Osteonecrosis has been 38 feet of sea water. There have been cases reported of Osteonecrosis occurring after single exposures to pressure. There are, for example, the cases of Necrosis of the femoral and humeral heads in personnel escaping from the sunken HMS Poseidon in 1931[4] and there is well documented case occurring in a caisson worker who had only two exposures on different days at a pressure of 79 feet of sea water. It is undoubtedly true that there are many professional divers who have suffered attacks of decompression sickness, whose bones remain clear of any form of necrosis, and I would equate the onset of bone necrosis in many respects to the deadly game of Russian Roulette.

PATHOLOGY

There are many mechanisms that have been postulated for Dysbaric Osteonecrosis among these are vasculitis, coagulation defects, fat embolism, and gas embolism (Table 4).

As far as Dysbaric Osteonecrosis is concerned, there is no evidence for vasculitis or coagulation defects. However, the mechanism of avascular bone necrosis in steroid therapy has been shown to be due to fat embolism[5]. In these patients there is no evidence of osteoporosis and the histology of femoral heads and humeral heads removed at operation shows fat embolisation. No evidence of vasculitis or vascular obstruction is shown in these people although intravascular fat emboli have been demonstrated. In divers, I believe the pathology is related to the so-called silent bubbles that has now been demonstrated to occur in very many asymptomatic decompressions following pressurization[6]. The use of the Doppler by Spencer among others, has shown that micro-emboli of 20 micron diameter or greater can be demonstrated in the venous return through the heart. It can be shown that interruption of blood supply to the bone cortex for between six to nine hours results in an area of necrosis in that part of the bone. This, however, is not detectable by radiological means for some three to four months. I envisage the formation of venous gas emboli within the blood supply of bone. In cortical bone, capillaries are continuous with those of the medulla and flow is unidirectional and evidence points to this as being centrifugal. I suggest that venous gas bubbles form in the nedullary sinusoids, and even in the cortical sinusoids, thereby interrupting the flow of blood from the medulla through the cortex to the interfascial and intramuscular venous drainage system surrounding the bone. This results in an interrupted oxygenation of cortical bone and thereby paves the way for the necrotic process. The pathology of necrosis varies slightly as to whether the site is that of the head of the long bones (the femur and the humerus) or whether it occurs in the shaft of the long bones. Figures 1 and 2 illustrate a postulated pathological process.

The sites that have been shown to be affected in divers and compression workers are the shoulders, the hips, the lower end of the femur, the upper end of the tibia and very occasionally other sites. The problem sites are those immediately beneath the articular surfaces of the femoral head and the humeral head. It has been shown that it is in these situations where the joint cartilage may be maximally affected, resulting ultimately in secondary osteoarthritis. Where the lower end of the femur or upper end of the tibia are effected, it has only been shown in four cases throughout all world literature that the knee itself is affected. In surveys conducted by many research workers, there have been an interesting disparity between the major site affected. For example, in free swimming divers, such as surveyed by Ohta and Kawashima, 1973 it was found that in 450 divers, the vast majority of lesions occurred in the hips. However, other surveys conducted on professional divers who may use mainly their arms and also caisson workers, have suggested that the shoulders may be the more affected. I believe this is due to the increased usage of one joint over another with a corresponding increased blood supply, and therefore the increased risk of gaseous emboli forming in or around that joint. There has been a trend showing through one survey taken in Australia (Williams and Unsworth, 1976) that the dominant shoulder of professional diver is more often affected than the non-dominant shoulder.

RADIOLOGY

In 1966, well defined criteria for the radiological classification of bone necrosis was laid down by McCallum, Walder and others[7]. This divides the radiological appearances into types A1 to 5, (those lesions concerned with juxta articular areas) and types B1 to 4 (those lesions occurring in the head, neck and shafts of long bones). These criteria and classifications have been adopted internationally and certainly aid in the diagnosis and interpretation of bone lesions in divers. The original medical research council classification relied upon straight x-ray films. In more recent surveys[12], polytomography have been used with some substantial success, particularly in delineating cysts and spherical lesions.

The incidence of bone necrosis (Table 5) shows widely differing results. I believe this is due to the different type of diver concerned. Elliott and Harrison[8] for example dealt with 350 Royal Navy self-contained divers. The Medical Research Decompression Sickness Registry[9] dealt with over 1600 caisson workers, where Kawashima[10] dealt with 450 self contained Japanese shell divers. The US Navy[11] on the other hand restricted their survey to Naval divers alone and Williams and Unsworth[12] have started a survey in Australia looking at, not only professional, but also - and this is believed to be of some significance - sports divers. Figure 3 shows the break-up of Williams' survey taking 110 divers and compression workers. He showed the incidence of 24% with bone lesions, of which 7% were in the sinister position of juxta-articular. Of these juxta-articular, five affected the shoulder joint and only 3 the hip joint. It is believed that this ratio is due to the higher incidence of working divers who would be using their arms and shoulders more than their hips as in swimming. However, the increased incidence of femoral head, neck or shaft lesions over the lesions associated with the humerus must not be overlooked. It is interesting to compare the sites of bone necrosis among 238 cases of decompression sickness as opposed to 77 cases of steroid induced bone necrosis (Figure 4). As can be seen in the decompression sickness induced cases 52% occurred in the shoulders, whereas in the steroid-induced number, 80% occurred within the hip joints. This is very suggestive that it occurs in the dominant joint, dominant weight-bearing or active joint that may be involved. This significance of the active joint, of course, will apply, not to professional divers but equally to sports divers.

DIAGNOSIS

In the asymptomatic diver the only means of diagnosis are radiological and nuclear (Figure 5). Where a diver has reached the stage of joint disintegration, then clinical means of diagnosis enter the picture. The radiological techniques involve not only straight x-rays but as previously mentioned, polytomography also. The difficulty with nuclear medical isotopes uptake lies in the interpretation of the results[13]. The radiological classification and interpretation has been known for many years. where as it is only recently that nuclear medicine has been applied to avascular necrosis of bone in
Divers (Cox 1973), so for some while to come more emphasis will be laid upon the radiological diagnosis than upon the nuclear diagnosis.

MANAGEMENT

In the non-articular (the B classification), the recommended management of patients with bone disease is modifications to their diving technique. The individual is to restrict his diving to within the limiting line of the Royal Navy Table. This allows a maximum, for example, of 20 minutes at 180 feet. The hazardous decompression of experimental and oxyhelium diving would no longer be permitted. In articular asymptomatic lesions the individual if a professional should cease diving but there is no certainty that the disease even at this stage will regress. It is equally not known for certain whether continuing to dive will aggravate the histopathology, but it may be presumed that an area of disorganised bone vasculature mat invite further gaseous embolisation and degeneration.

Active measures in asymptomatic articular disease may involve drilling through the fibrous layers and inserting pegs or bony chips to improve the blood supply of the area and therefore reossification. These however, have not proved particularly successful. The management of symptomatic severe juxta articular lesions with pain and degeneration of the joint will involve well recognized orthopaedic procedures such as osteotomy or arthrodesis.

Elevation of the indented articular cartilage and bolstering with ships inserted beneath the articular cartilage have also been tried but with only limited success. When all other orthopaedic attempts have failed to improve the joint, then total hip replacement remains. The problem with total hip replacement is that in a young diver aged 25 to 30 and a hip replacement with an anticipated life of 10 to 15 years then one may anticipate perhaps two or three joint changes prior to that diver's demise and this is a particularly daunting thought. The surgery of established dysbaric osteonecrosis has up to this point in time, proved rather unsatisfactory. The predicament that we are in, is that an apparent unavoidable risk of diving is dysbaric osteonecrosis and that the only precaution is to avoid diving and not to expose the human body to changes in ambient pressure. But this not only involves considerable financial loss to professional divers but also involves giving up a sport that very many people not only enjoy at present but are likely to take up and enjoy in the future. We have therefore a potentially crippling condition about which we are not sure of its aetiology, the management of which is as yet unsatisfactory but of one thing we can be certain, that is of its existence - a most unfortunate situation and one to be resolved as soon as possible in the future.

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TABLE 1

AVASCULAR NECROSIS OF BONE

AETIOLOGY

Endogenous Hypercortisonism
Corticosteroid Immuno-Suppression (renal transplation)
Trauma - fractures, dislocations
Polyarteritis Nodose
Chronic Alcoholism and Pancreatitis
Sickle Cell Anaemia
Alcaptonuria
Syphilis
Gaucher’s Disease
Schandler’s Disease
Decompression Sickness (dysbaric osteonecrosis)

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TABLE 2

HYPERBARIC FACTORS

Pressures > 17 psig (4 atm)
Frequent exposures
Long history of exposure
35+ years of age
Experimental diving
Inadequate decompression
Inadequate treatment of decompression sickness

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TABLE 3

DIVING FACTORS

Lowest pressure known - 38 feet sea water

Shortest time known - 2 exposures on different days (2.5 hours, 4.5 hours) in 79 feet sea water.

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TABLE 4

MECHANISMS

Vasculitis
Coagulation Defects
Fat Embolism
Gas Embolism

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TABLE 5

INCIDENCE

	No. Divers	% Lesions
Elliott and Harrison (1970)	350	<5
MRC D/C S reg (1972)	1694	19.7
Kawashima (1973)	450	59
USN (1973)	303	32
Williams and Unsworth (1976)	110	24

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FIGURE 1

SHAFT PATHOLOGY

ISCHAEMIA
Ø
OSTEOGENIC TISSUE SUBJECTED TO
Ø pH and pCO^2 ≠
Ø
THICKENED TRABECULAE AND INCREASED CALIFICATION

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FIGURE 2

HEAD PATHOLOGY

NECROSIS and OSTEOCYTE DEATH
↓
BONE RESORPTION
↓
PARTIAL REVASCULARIZATION
↓
THICKENED TRABECULAE
↓
ZONE OF COLLAGEN
↓
TRABECULAR BREAKDOWN
and
JUXTA-ARTICULAR COLLAPSE

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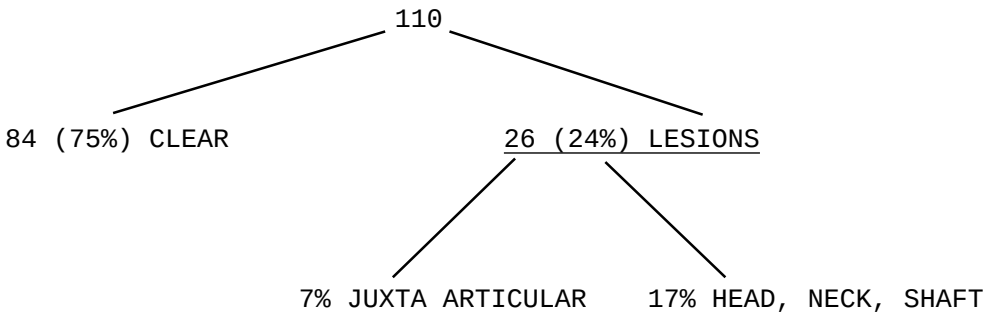
FIGURE 3

DIAGNOSIS

DIVERS AND COMPRESSION WORKERS



Williams and Unsworth (1976)

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FIGURE 4

SITES

Decompression	(238 cases)	Steroids	(77 cases)
Shoulders	249 (52%)		11 (7%)
Hips	94 (20%)		127 (80%)
Other	181		4

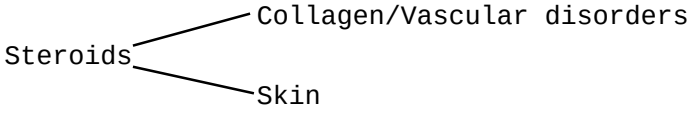

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FIGURE 5

DIAGNOSIS

Clinical	-	Orthopaedic
Radiology	-	Tomography
Nuclear	-	Skeltec

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A “red” Monster?

Soviet Oceanographic Society member A Pechersky told a reporter of the Komsomolskaya Pravda that he and his son saw a giant snakelike creature in Lak Kok-Kol in southern Kazakhstan. Water fowl fled the water in alarm as the monster reached the surface and ploughed through the water. Several sightings of a huge creature have been reported in the lake. Scottish scientific circles have refused to comment on the possibility that “Nessie” has defected.

The Australian, 31 January 1977

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The kiss of Mildred’s Life

Mildred the goldfish is alive and kicking today ... saved by the kiss of life. She had been found floating apparently lifeless in her tank by Rodney Griffiths, 8, at his home in Stevenage, Hertfordshire, England. his uncle, Michael Reed, lifted the fish from the water and blew into it’s mouth. “I was amazed,” said Mr Reed, “the fish responded immediately and looks fine now.”

6 April 1977

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