The references to the necessity for repetition and "overlearning" in the teaching situation are generally accepted principles which tend to lose out in the design of courses designed to meet strict financial constraints. Possibly there is need to define the essential skills necessary for survival and to concentrate on these in training, with a recognition that experience in situation management can never be delivered in a few dives using scuba equipment in a class. The training of Rodent E Lee has lessons for all who seek to train others in any complex task, diving included.

This book should prove a valuable resource to diving instructors who wish to improve their understanding of the sometimes unexpected and curious behaviour they will certainly encounter among their pupils. It should make them more alert to those requiring their special care in a dive group whose members have unknown personalities and whose relevant experience is unknown, or possibly thought to be suspect. The book should improve diver safety.

Douglas Walker

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DYSBARIC OSTEONECROSIS

David Elliott

Abstract

Sudden pain in a joint, unrelated to decompression, was the presenting manifestation of many of the first cases of bone necrosis to be reported. Collapse of dead sub-chondral bone led to surgical treatment which was less than satisfactory and many became crippled. Subsequent surveys of populations of divers revealed the presence of many lesions in the shafts of long bones as well as close to the weight-bearing surfaces.

A precise radiological procedure and agreed diagnostic criteria have led to valid comparisons of divers from different locations with different styles of diving. For once in occupational medicine, the boundary appears to have been clearly defined between the extreme of normality and the beginning of pathology. In spite of this, many individuals were medically disqualified from further diving for shaft lesions that had no potential for collapse.

Technetium MDP scanning has been introduced, but false positives are too common for reliable prognosis. Magnetic resonance imaging (MRI) shows some very early changes but it is expensive and, for prognosis, still lacks long-term follow up. A risk of neoplastic change exists, yet the providers of research funds favour other priorities for their resources.

Introduction

Bone necrosis is a relatively neglected hazard of diving. There was a time when dysbaric osteonecrosis was in the spotlight of media attention and was known as "bone rot disease of divers". Numerous cases have been published in the literature about sub-chondral collapse and its surgical treatment. Such cases in professional divers may not be uncommon in some parts of the world, but have become almost unknown in the North Sea. So the media spotlight has moved on. However, the condition has not gone away. Though not a problem for recreational divers who follow the decompression procedures of the worldwide sports diving agencies, I have been consulted in the past 12 months by two sports divers, one an instructor, with disabling osteonecrosis as a result of diving.

Not only is bone necrosis important as an occupational hazard of diving, it is also important because it is an example of how such occupational hazards should be investigated. The first cases of bone necrosis to be reported were those which had presented clinically due to pain in a collapsed joint. Some time later, surveys of populations at risk revealed asymptomatic lesions and, by the use of internationally agreed procedures, it was possible to acquire a reasonable understanding of the condition and its possible causes.

This story is in marked contrast to that of other possible long-term effects of diving in which no specific clinical condition has been described but which are now in the media spotlight. It is the purpose of this introductory paper on dysbaric osteonecrosis not merely to review an uncommon but troublesome diving disease but also, by tracing the history of its recognition and subsequent assessment, set an example against which the more recent "high-tech" conditions may be judged.

The early cases

At the turn of the century thousands of men were employed in the building of tunnels and bridges using compressed air to keep the workplace dry. It is from this population that Bassoe[1] in the United States and Bornstein and Plate[2] in Germany were the first to report disabling hip

and shoulder conditions which were associated with radiological evidence of joint degeneration. Perhaps because there are fewer divers and they work in smaller groups than compressed-air workers, the first case in a diver was not reported for another thirty years in 1941. This, too, presented as pain in a joint.[3] Around ten or more cases were reported in the literature during the next ten years and all were in divers who had sought treatment for persistent joint pain.

It is not possible to draw any valid conclusions from these cases because the X-rays are not always published and there was no agreed standard for radiological diagnosis. One can assume that of the 90 divers examined radiologically by Herget[4] possibly more had aseptic bone necrosis than the 29 which he reported. From the same location Alnor[5] found 72 cases of necrosis in 131 divers and of the 65 who had been kept under observation for more than 10 years, only 22 of them remained free from radiological lesions. Of the 43 with lesions 17 had symptoms and 7 were "totally unable to work". Bone necrosis was thus established as a significant occupational illness in divers at a time when the British Medical Research Council was already investigating this condition in great detail among the workers building the Clyde Tunnel.

Pathology

Osteonecrosis in divers is of two basic types: juxta-articular (i.e. sub-chondral) and shaft, a description which includes the neck and a portion of the head of a long bone. The shaft lesions are predominantly saponified fat. It is the juxta-articular lesions that have greater clinical significance. These lesions show areas of dead bone surrounded by a layer of collagen which forms a fibrous band and the formation of new bone. Beyond the area which can be detected radiologically is seen an area of creeping substitution and healing trabeculae.[6] The detailed pathology has been reviewed by McCallum and Harrison.[7]

Pathogenesis

The mechanism causing bone necrosis is not understood. There are many hypotheses.[7] These include lipid emboli, a condition which exists in many other illnesses which are also associated with aseptic bone necrosis; autochthonous bubbles, possibly arising from the disintegration of natural Uranium[238] and enlarged by decompression; an oxygen effect which leads to the swelling of fat and the possible compression of osteocytes; surface-activity effects possibly due to embolic bubbles; gas-induced osmosis; "stiff" red cells and raised intramedullary pressure. From these and other theories it can be concluded that only uncertainty prevails.

Prevalence

Perhaps the most important step towards the proper assessment of the significance of bone necrosis to the diving population was the standardisation of diagnostic techniques. With agreement on diagnosis it was possible to move away from purely clinical descriptions of the illness to surveys of the prevalence of pre-symptomatic lesions in the apparently healthy population. The credit for this must go to the members of the Medical Research Council's Decompression Sickness Panel in the late 1950s and early 1960s.

X-ray technique

The radiological diagnosis of bone necrosis depends upon the quality of the X-rays that are taken and the experience of those who read them. The X-ray remains the gold standard of diagnosis and although the disabling complications affect only the shoulders and the hips, extensive views of the lower femur and upper tibia are included in knee X-rays in order to find as many shaft lesions as possible. These are of value in confirming the diagnosis of a lesion seen in another view but, because the shaft lesions are relatively benign, can provide an alarming number of positives in a population who may otherwise seem to be perfectly healthy. The radiological technique to be used with each specific view has been described recently.[7] The X-rays should then be read by a radiologist who is experienced in reviewing the X-rays of tunnel workers or divers. In a survey, the X-rays are read independently by two radiologists who then meet to review any films about which there may be disagreement. From such consultations each individual can be described as having lesions according to the classification published by the MRC Decompression Sickness Panel and for each lesion whether that diagnosis is certain or doubtful. Gonad shielding must be used. The measured dose using dosimeters on a phantom was 1.6 rads which is less than half the maximum pubic dose.

A major fault is under-penetration of the radiographs and, because of this, trabecular detail is not always clear and small dense areas close to the joint surface may not be identified. This may be because during the process of bone repair, new bone is laid down on the trabeculae causing an increase in bone bulk. Therefore, unless the voltage is increased possibly by as much as 10 kv, a pale under-penetrated radiograph will result. This may be the commonest cause for misinterpretation and failure to identify osteonecrosis.

Tomography may be required to improve definition particularly in the femoral head, where detail is obscured by the overlying bone. In general however, good quality radiographs preclude the need for frequent use of tomography.

It was not easy to convince hospital radiologists who were unfamiliar with dysbaric osteonecrosis of the importance of such a meticulous approach to radiography and to diagnosis. Indeed, in the 1960s a number said that they "see those kinds of changes all the time". Such perceptions are of course based upon clinical judgement and not upon a scientific approach.

Differential diagnosis

As part of a survey of 383 naval divers[8] a comparable sample based on age and rank was selected from non-diving naval personnel (taking care to exclude those with high altitude flying experience). No lesions were detected in these 88 persons.

The majority of alternative causes of bone necrosis should be readily excluded in divers. They include chronic alcoholism, long-term steroid therapy and other conditions most of which are incompatible with fitness for diving.

Surveys

At the time of the Royal Navy's survey of 383 clearance divers, begun in 1966, in whom 19 were found to have positive evidence of bone necrosis[8] a similar survey was being conducted by Ohta[9] in Japan. He examined 301 diver fishermen and found 152 with lesions, a significantly greater incidence. The Japanese divers also had a greater proportion of juxta-articular lesions.

Some 15 years after the clearance divers had been X-rayed the MRC[10] published the results of their extensive review of commercial divers. They found that of 6,691 divers 4.2% had definite bone necrosis, a figure very similar to that found in the Royal Navy. This included 77 divers with juxta-articular lesions (1.1% of the total number of divers and 26% of those with osteonecrosis) of whom 12 (0.17% and 4%) had a pathological fracture.

Possible pathogenesis

The remarkable difference between the Japanese and UK results appears to have a relatively simple explanation. Whereas the European divers were diving in accordance with agreed decompression procedures, the Japanese had none. A typical diving day would be several hours at around 30 m to collect shellfish in the morning and then after lunch on deck, a similar dive in the afternoon. This technique may account for the diminishing number of divers found in each successive age group reported. Indeed, as one doctor said about the way in which the Japanese dived "this is why the flag of the factory ship is always at half mast". In the naval survey not only was there an association with age which is, of course not easily separated from diving experience but there was also a significant association with those who had undertaken deep dives which in those days were of a pioneering nature. A more analytical study by the MRC[10] found that bone necrosis is related to the type of diving exposure. There were no lesions in divers who never dived deeper than 30 m on air whereas at depths deeper than 50 m the proportion of men with positive lesions progressively increased with greater depth, rising to 15% for those with experience greater than 200 m. As with compressed air workers there is not a one-to-one relationship between acute decompression sickness and bone necrosis. Of those who said that they had never had acute decompression sickness, 1.7% had definite lesions. In contrast 40% of the population had had decompression sickness and in them the proportion was 10.7%. Of all but one of 9 men with a damaged joint reported that they had had decompression sickness. Men who had more than one episode of decompression sickness appeared to be at a greater risk than those who had had only one incident. It is important to remember that bone necrosis occurs in divers who have adhered to recognised decompression procedures.

Another important feature is that it only requires one decompression to cause necrosis. Six men survived for some hours, after the sinking of HM Submarine Poseidon in 1931, at depths between 24 and 36 m (80 and 120 ft) and then made emergency ascents. Twelve years later 3 were X-rayed and all had severe juxta-articular lesions.[11]

Other diagnostic techniques

Radiology is very slow to demonstrate the changes of osteonecrosis and although it is used as the diagnostic standard, other techniques are available and have some value in screening. MDP (^{99m} Technetium Methyl-di-polyphosphate) scans are very sensitive to local bone pathology. A "hot spot" indicates increased perfusion and metabolism and may be seen only hours after a dive. However in a Royal Navy survey[12] only 4 divers became radiologically positive some 2 to 3 years after 22 had demonstrated positive scans. Eleven still had abnormal scans and 7 had reverted to normality. Thus a positive scan is of little diagnostic significance, but may indicate a need for radiological follow-up.

Magnetic resonance imaging (MRI) has a remarkable power to detect early lesions but it is not generally available for routine screening of large populations.

Prognosis

The progression or possible regression of necrotic lesions is unpredictable. Any relationship between the lesion and the continuation of diving has yet to be studied.

The prognosis for an individual with a juxta-articular lesion has been studied in relation to the optimum time for surgery.[13]

However once a diver has got this far it is obvious that preventive occupational health has failed.

The management of shaft lesions is much easier. Since they do not cause joint collapse there seems to be no immediate indication for cessation of diving. However, the divers must be told of their lesion. Very rarely it may become malignant. The risk is considered by **** **** **** **** **** (AFIP) to be less than one in ten thousand and while a few such cases have occurred in compressed air workers, fibrous histiocytoma has been described in only one diver.[14]

Prevention

Although it seems that bone necrosis is in some way decompression related, and some improvements to compressed air workers decompression tables have resulted in a smaller incidence of this condition, not sufficient is known about the nature of the decompression insult to enable one to come to any conclusion, other than that a certain number of cases of bone necrosis in a diving population seems to be inevitable.

Routine Screening

After an initial base-line X-ray for trainee commercial divers, no X-rays are required subsequently provided that all diving is at less than 30 m and for durations less than 4 hours. Three yearly X-rays are recommended by the MRC for all commercial air divers diving deeper or longer than this. All other divers such as mixed-gas divers require annual X-rays. A recommendation has also been made that all divers should be X-rayed 3 years after an episode of decompression illness. At these follow-up examinations X-rays of only the shoulders and hips are required, a modification which halves the radiation dosage. If radiography is not judged necessary for other reasons, the Health & Safety Executive consider that it should be repeated at intervals of 5 years during the diver's career.

Treatment

Any person who has a suspected juxta-articular lesion must avoid weight-bearing until his condition has been assessed by an orthopaedic surgeon. The further surgical management of such cases is beyond the scope of this review. The report of one case of aseptic bone necrosis in which hyperbaric oxygen therapy was associated with improvement is promising,[15] but a proper case control study is still required.

Conclusion

Bone necrosis thus illustrates the proper progression from isolated case reports to surveillance of asymptomatic individuals by the use of internationally accepted diagnostic criteria.

One consequence of this approach was to show that the potentially crippling lesions were present in only about 1% of the total diver population. This appears to have convinced the authorities responsible for funding research that bone necrosis is not a problem. Certainly, the MRC Registry had its funding stopped by the Health & Safety Executive some 10 years ago. As a result we still know nothing about the possible changing patterns of bone necrosis which may be consequent upon the changing patterns of diving techniques being used in the North Sea. In spite of this, the example of bone necrosis provides a practical model for the study of any diving-related medical condition.

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ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT

1/5/1992 - 30/4/1993

A Progress Report

John Williamson

Introduction

The Hyperbaric Medicine Unit (HMU), within the Department of Anaesthesia and Intensive Care at the Royal Adelaide Hospital (RAH) is now 9 years old. It was founded by Dr J (Fred) Gilligan, currently Director of Retrievals and Resuscitation at the RAH, and Dr Des Gorman, the inaugural Director (until 1990), currently Director of Medical Services, Royal New Zealand Navy. They were ably supported by the longstanding efforts of Dr Tony Swain, and Dr Robert Ritson, Member of the Legislative Council.

The medical staff consists of a full time Medical Director, a visiting diving medical senior consultant, and 4 part-time medical consultants with senior anaesthesia, resuscitation, and diving and hyperbaric medical qualifications and experience. Two have recreational diving instructor qualifications. Another two senior anaesthesia and intensive care consultants contribute upon request and an honorary senior visiting sports physician with hyperbaric expertise visits and contributes weekly. The Unit has a 6 months training appointment for a provisional year Fellow of the Australian and New Zealand College of Anaesthetists.

The nursing staff comprises a full time clinical nurse consultant with hyperbaric nursing qualifications and experience, a full time clinical nurse and one part time registered nurse with hyperbaric nursing training and experience. These three have intensive care (IC) training and qualifications; supplementing them is a pool of in-house-trained hyperbaric nurses (including some with IC qualifications).

There are two senior hyperbaric technical officers full time, and 1 casual on-call technician. They have extensive international off-shore and in-shore commercial diving, recompression chamber (RCC) operation, and diving supervision experience and qualifications. One is a former Royal Naval clearance diver and diving supervisor, and two are qualified open water diving instructors with commercial experience in recreational diving. A technical research officer participates part time in research trials.

A full time, highly skilled secretary handles correspondence, phone calls, manuscripts, visitors to the Unit, and coordinates advertising, applications and finances for the 3 diver medical technician (DMT) and 2 diving medical officer courses conducted each year. Assisted by one of the hyperbaric technicians she also co-ordinates the other diving and oxygen courses regularly conducted by the Unit.

The Unit fulfils the following general functions nationally and internationally in collaboration with other Australian and New Zealand HMUs; clinical patient care, 24 hour manning of DES/DAN (Divers Alert Network) Australia, research, teaching (including the 5 annual national courses), public education and voluntary work.

Clinical patient care

Ninety-six patients were treated in the recompression chamber (RCC) during the 12 month period from May 1st 1992 to April 30th 1993. The respective diagnoses affecting these patients are shown in Table 1.

Results

There were 2 deaths in the series (post-cardiopulmonary bypass in whom cerebral arterial gas embolism probably occurred during surgery, and post-traumatic cerebral oedema).

At least one of the carbon monoxide (CO) poisoned patients (who had a cardiac arrest at the CO exposure site) was left with severe neurological problems. Only 20 of the 34 CO poisoned patients presented for follow up at 6 to 12 weeks post-treatment. Of these 15 were classed as normal