Encouragement is the heart of the book and is well illustrated in the opening chapter on "Fitness for Diving". Serious diseases and chronic disorders disbar those divers who would be hazardous under water. Folk medicine and tales of woe are rebuked. The author should be complimented when he states "any ear can be made fit for diving".

Charles Finlay-Jones

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WHAT ARE THE LONG-TERM SEQUELAE OF DIVING?

David Elliott

Abstract

The effects of hydrostatic pressure and of the raised partial pressures of the respiratory gases are exerted on every cell in the body. Man at pressure is physiologically not the same animal that he is at the surface. With the additional hazards of decompression it seems rather surprising that, for many years, the only well recognised long-term sequelae of diving were those associated with neurological residua after an acute episode of decompression illness. Subsequently, bone necrosis was recognised as a hazard of raised environmental pressure. An assumption that this condition was also decompression-related was not supported by the lack of a one-to-one relationship with limb bends and a wealth of hypotheses remain, each trying to account for it. As new investigative techniques, with greater powers of resolution, became available for hospital use, they were applied by occupational physicians to apparently healthy workers. The presence of new "abnormalities" were detected and the absence of adequate control studies led to a number of erroneous conclusions.

Changes in lymphocytic chromosomes have been reported in divers but without significant sequelae. Changes of lung volumes and of carbon dioxide sensitivity have been recognised for some years, but, more recently, a reduction in pulmonary diffusion capacity (TLCO), with functional consequences, has been reported. With each new allegation of adverse diving sequelae, the implications to be considered include the validity of the diagnostic procedure used and the likely impact on the long-term quality of life for that individual.

Introduction

The person who takes up diving takes up a hazardous activity. Diving will always be hazardous and so the future level of activity for man in the sea will depend upon the balance between the benefits and the perceived risks. Any perception of risk is usually different from the actual risk and it seems important to estimate the actual costs, in terms of health and safety, of each type of activity underwater. The sea makes no distinction between the recreational diver and the professional, but the balance point between apparent benefit and perceived cost is not the same. In each diving group the benefits are relatively clear: pleasure for the sports diver and, for the employed diver, payment. Other than the consequences of a diving accident or of decompression illness, the perception of the recreational diver is probably that the risk of long-term health effects due to diving is around zero. This may not be quite so true for the professional air diver but, because his instruction has been confined to diving emergencies and his only knowledge about long-term risks may come from other divers and the media, he is not necessarily fully aware of the occupational hazards to his health.

It is the purpose of this review to look at some hazards of air diving and to assess the risks that these imply for the diver's continued good health.

The first part of this review will be devoted to hazards which may be at the cellular level, rather than the more obvious acute hazards to health, such as "running out of air". Bone necrosis has been described as a condition which, thanks to the standardisation of diagnostic techniques, is capable of being largely controlled by the adoption of appropriate decompression procedures and which can, for the individual diver, be monitored during his career. Other long-term effects for instance, of hearing, pulmonary function and chromosomes also provide important methodological background to a consideration of neurological deficits.

The Hazards

While at depth, every cell and each molecular process is subject to increased hydrostatic pressure. At great depths the molecular and cellular effects of hydrostatic pressure are relatively easy to demonstrate and some may have long-term consequences.

The effects of increased environmental pressure on the gas-containing spaces of the body are not confined to the various forms of acute barotrauma but may have other effects, for instance upon the pulmonary system.

The increased partial pressure of oxygen is perhaps the most prominent toxic agent in diving and its effects are not confined to the brain and the lungs.

Perhaps the most potent hazard to which the diver is exposed is that of the "silent bubble". The bubble can arise during accepted safe decompression procedures and is a powerful biological agent within the bloodstream.

Each of these four primary hazards is well known but the threshold of safe exposure to them is unknown because we have no means of defining “safe” in this context. Indeed, is there a “zero-threshold” effect?

HYDROSTATIC PRESSURE EFFECTS

While the vast majority of studies in hydrostatic pressure upon living cells and organisms have been conducted at extreme pressures,[1] the mechanism of these effects are simply related to changes of molecular volume and such changes need not be extreme. For instance the squid giant axon shows a reduction of threshold current and potential at a pressure of 7 bar.[2]

Under some circumstances dissolved gas can induce an osmotic effect, causing tissue water to be redistributed.[3] This is considered to be important pathologically only within a rigid structure such as bone and may lead to changes in intra-medullary pressure which may, or may not, be associated with dysbaric osteonecrosis.

Alterations of the stiffness of blood cell membranes have been described[4] as another possible cause of osteonecrosis and other diving-related conditions.

Increased pressure prior to decompression also induces platelet aggregation[5] and the effect upon platelet density of compression is related to the level of pressure achieved.[6]

There seems to be no evidence that the high pressure nervous syndrome (HPNS) is *per se* anything but a transient phenomenon although post-dive effects have been ascribed to it.

GAS-CONTAINING SPACES

In many cases, barotrauma is acute and any effect can be dated from a particular incident. But the effects of minor but multiple compression barotrauma upon the middle and inner ear can be responsible for changes which may well become long-term.

Decompression barotrauma can lead to arterial gas embolism. The acute effects are well recognised but in some the effects can be sub-clinical. Extra-alveolar air was found in a number of submarine escape trainees after apparently successful ascents[7] and EEG changes found after successful ascents by submarine escape trainees implies the probability of some “silent” arterial embolism.[8]

Increased gas density may be related to some of the long-term pulmonary effects.

PARTIAL PRESSURE EFFECTS

The literature on oxygen toxicity is vast but has the threshold for relatively minor effects of long-term exposure yet been defined? Oxygen is well known to have effects throughout the body ranging from the endocrine system[9] to bone marrow in which Walder[10] proposed that a raised partial pressure of oxygen would enlarge the volume of fat cells.

The raised partial pressures of the other respiratory gases, such as carbon dioxide, also have possible long-term effects. There can be other gases in the breathing mixture. For the recreational diver failure of adequate air filtration can lead to contamination by oil and particulate matter, which may have health consequences, but for the commercial diver the list is long, and includes such factors as fumes in welding habitats.

DECOMPRESSION EFFECTS

The blood-gas interface induces a wide range of haematological and endothelial effects. The effects upon platelet aggregation, Hageman Factor activation, the clumping of blood cells, the release of lipid emboli, the activation of complement systems and kinins, the increased and decreased activity of some enzymes and other effects have been reviewed elsewhere.[11]

Respiratory function

One effect of diving is an increase in vital capacity with age in young professional divers, who then have difficulty in passing the FEV_1/FVC pulmonary function test at annual medical examinations.[12] The arbitrary minimum value of 75% should not be accepted without consideration of the implications of this long term effect. The general conclusion that divers tend to have larger vital capacities than non-divers was not confirmed by a study of 126 saturation divers by Thorsen[13] who suggests that there is a greater, belated, diminution. Any change of vital capacity probably has little effect upon the diver’s general health though recent studies have shown that divers do develop some degree of air flow obstruction due to airway narrowing.[14]

Perhaps a more significant long term change among divers is demonstrated by a minority of divers who can be identified as “carbon dioxide retainers”.[15] While it might seem an advantage for a diver to be able to tolerate a greater level of carbon dioxide, the synergistic effect of

increased carbon dioxide, oxygen and nitrogen is considered to make it more of a hazard than an advantage. The group can be identified by their inadequate increase in ventilation to a raised inspiratory carbon dioxide level.[16] Steady-state studies of end-tidal carbon dioxide levels showed that those divers who have high values continue to do so after they have finished diving.[17]

Pulmonary diffusion capacity is another physiological function which appears to deteriorate in divers with age. At the present time the work to assess this is incomplete, particularly in air divers. The early reports have all been confined to mixed gas divers,[18] which is in part due to the fact that divers are exhaustively examined before and after every deep dive, much more so than at the annual medical examination. The diminution of pulmonary diffusion post-dive may not be clinically significant and tends to improve during the next few weeks, but it is also associated with a diminution in exercise tolerance.[13] A number of explanations can be offered, the most favoured being an association with the possible cumulative dose of oxygen at tensions greater than 0.3 bar. In contrast, a study of 8 divers in a German deep dive showed no significant diminution of pulmonary carbon monoxide transfer capacity (TLCO),[19] though there were other variables. The study is now being repeated with a 450 m dive at the National Hyperbaric Centre in Aberdeen. The significance to recreational divers of findings after 28-day saturation exposures to maximum depths of 450 m may seem remote but, if the hypothesis of a lower threshold for pulmonary oxygen effects is correct, then one would expect to find this long-term effect in all saturation divers. It could also become a matter of concern for those who dive extensively each day at shallower depths.

Genetic Effects?

It would be difficult to think of a topic more likely to attract the spotlight of media attention than that of the possible genetic effects of diving. Although there may seem no reason why there should be any genetic changes, once such a question is raised it will not go away.

The discovery of triploid zygotes in the child of a diver was sufficient to initiate a pilot study in Aberdeen on chromosome aberrations in the cultured T-lymphocytes of divers. The results of that pilot study were sufficient to justify an enlarged study which was funded by the UK Department of Energy.[20] There are some positive features of this investigation which provide a useful lesson to other investigators.

Because of the emotive nature of this project, all who were concerned in it agreed to maintain strict confidentiality until it was complete. Blood was taken from more than 150 divers and an equal number of control subjects. Each person was fully informed about the purpose of this study and would be counselled in the event that any abnormalities were found.

Of 77 compressed air divers and 76 mixed gas divers, 6 had a few heavily damaged cells. The health risks imposed by these abnormal cells is unknown but the damage they contain is, in most cases, so extreme that they are likely to die at mitosis. No such cells were found in the controls.

This type of finding was unexpected and, because of such low numbers, no correlation was possible with the many associated occupational factors that were also studied.[20] The aberrations observed were typical of those induced by ionising radiation and were present in air divers as well as mixed gas divers. None of the affected divers admitted to using gamma-sources for examining welds at depth, whereas some of the divers who had normal chromosomes did use isotopes.

At this stage it was necessary to release the results. Before publication, a letter was written by doctors associated with the project to each participant and to each diving company, explaining the significance of the findings. In spite of the uncertainties of the study and of the concerns that could easily have been created, this briefing was sufficient to assure the diving population that the problem had been investigated meticulously and that, although the cells studied were only lymphocytes, there was no evidence that would indicate a significant effect in the germ line. This extensive briefing meant that when the newspapers became aware of this project, there was no angle to the story which they wanted to print. All occupational health surveys should be confidential to the investigators until the results are known. This shows that it can be done.

Hearing Loss

It is well known that old divers are deaf. Indeed a number of audiometric studies have shown that a population of divers demonstrate hearing losses greater than in age-matched controls.[22, 23] If the deafness is insidious and without obvious cause then it falls within the remit of "long-term effect".

Noise-induced hearing loss is a likely cause for deafness in professional divers. A temporary threshold shift can occur whenever one is exposed to a loud noise. In professional divers these hazards are more common and include the rush of gas entering a chamber during compression, the circulation of gas in diving helmets, the use of noisy underwater tools and the occasional underwater explosion. Repeated exposures will cause a permanent threshold shift in both ears.

There are many other causes of hearing loss in divers. Even if those for which there are obvious acute

causes, such as decompression sickness or inner ear barotrauma, are excluded, repetitive sub-clinical episodes of these same conditions must be considered.

Susbielle[24] suggested that repetitive minor barotrauma to the middle ear can lead to changes of pressure within the inner ear. He also suggested that inert gas dynamics could lead to osmotic dysbarism in the endolymph and perilymph. In a study of 116 divers, Molvær and Albrektsen[22] concluded that much of the loss of hearing acuity in these men was due to high noise levels at work but, in addition, residual damage from acute inner ear injury caused by barotrauma was also found. In a study of the effects of compression and decompression upon a small series of mini-pigs[25] it was shown that inner ear changes occurred in these animals on a pressure schedule that is usually regarded as safe for man but, of course, the mini-pig may not be very good at ear-clearing. An important feature of this study was the loss of hair cells throughout the cochlear in all the compressed animals.

Conclusion

This review is far from comprehensive but each of these long-term health effects has been well documented and is of potential concern to air divers. A wide range of possible pathogenetic pathways has been mentioned, some of them specific to one condition, others common to several. There are also many other possible long-term effects such as subfertility in animals[26] and in man effects on such organs as the liver,[27] the heart[28] and even the hand.[29] We have a random collection of observations in which no common thread can be discerned. How is one to assess the clinical significance of these conditions? An effect upon the quality of life is probably the ultimate test, but is not one that can be quantified.

One way forward was demonstrated in the MRC study of bone necrosis in tunnellers and divers by the creation of the Decompression Sickness Registry in Newcastle. The collection of thousands of individual X-rays, annual medical examinations and, for the tunnel workers, the depth time profile of each shift's exposure, was a monumental piece of work. From it has emerged many invaluable observations encapsulated in a number of papers. Even so, many questions about bone necrosis remain unanswered. That the research is incomplete can be attributed to the failure of national funding authorities to recognise the importance of a central registry to the health surveillance of a relatively small numbe of high-risk workers, who are travelling the world in search of employment. A broad data base was established before the Health and Safety Executive (H&SE) withdrew funding nearly 10 years ago. Since then no further data has been collected.

Cross-sectional studies may be useful in the definition of an occupational problem but longitudinal studies provide much better scope for analysis. In the 1960's and early 1970's many supported the concept of a central European diving registry, not only for the collection of data related to occupational health but also because of a need for centralised medical records internationally for the North Sea.

Perhaps a breakthrough could still happen. For commercial divers in the UK, at least, it has now been agreed between the oil industry, the diving companies and the Offshore Division of the Health & Safety Executive that all air-range dives will be monitored on-line. The pressure profile, together with breathing gas composition and thermal status, will provide a complete diving record for each diver. One immediate benefit of on-line dive data recording is for the retrospective analysis of any diving accident. A second benefit is not merely relating a bend to the causative dive but in time, by providing tens of thousands of such dive records, providing the opportunity for precise statistical analysis. This would lead to the generation of safer decompression tables. Now (May 1993) start-up funding seems assured. Given the co-operation of those responsible for recording annual diver fitness, one can begin to appreciate the great potential of a new preferably international registry, in which each diver's lifetime diving history is recorded and retrievable.

Such registry could allow identification of the contributory factors that may lead to long-term health effects, particularly in those who never have been recompressed for decompression illness. The data are now being recorded on each and every dive. The divers are examined medically each year. To turn this process into a reliable epidemiological survey of the relatively limited population of occupational divers worldwide no longer seems to be a dream. If bureaucratic hurdles can be removed, it might become possible not only to recognise any potential harmful effects at the earliest stage, but also perhaps to prevent them.

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UNDERWATER ESCAPE FROM DITCHED HELICOPTERS

David Elliott and Michael Tipton

Abstract

Some people fail to escape when a helicopter ditches into the sea. Protective clothing has for years focussed on hypothermia whereas almost no attention has been given to the immediate effects of cold immersion. Sudden immersion in water as warm as 15°C causes physiological effects which may jeopardise through-water egress from an in-