ORIGINAL ARTICLES

ATRIAL FIBRILLATION PRESENTING AS DECOMPRESSION ILLNESS (DCI)

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Summary

Early hyperbaric treatment to prevent long term sequelae of decompression illness (DCI) following scuba diving is established treatment. Diving related problems in general are now being increasingly recognised by both scuba divers and medical practitioners. However it should not be forgotten that divers may have non-diving related pathology which may manifest itself while scuba diving. The following case was referred to a hyperbaric facility as DCI but proved to be cardiac related. The significance of cardiac disease among divers is discussed.

Case Report

A previously well 47 year old male diver with 25 years of scuba diving experience was referred for hyperbaric treatment, with a presumed diagnosis of DCI, following the sudden onset of breathlessnesss, lethargy, unsteadiness, dizziness and chest discomfort following a scuba dive. The diver dropped his underwater camera while on the surface before his dive. Sea conditions were calm with a water temperature of around 17°C. Although wearing full scuba gear he performed a breath-hold dive to approximately 7 m and retrieved his camera. Almost immediately on surfacing he felt very short of breath and extremely unwell. This feeling resolved after several minutes and the diver started his scuba dive. He descended to 22 m, but again felt unwell and extremely tired almost immediately after descent. After 15 minutes at this depth he decided to surface due to this feeling of general unwellness. During the ascent, which was slow and controlled, he developed increasing shortness of breath associated with chest discomfort. This worsened while getting into the boat, where he also experienced a feeling of unsteadiness and lightheadedness. The diver at the time noted his heart to be racing, approximately 180 beats a minute. On admission to hospital at 1300 hours he was pale and sweaty. He had a radial pulse rate of 130 beats a minute with an apex rate of 170 beats a minute. Recumbent blood pressure was 110/80 mm Hg. Heart sounds were normal and no murmurs were present. His chest was clear with no evidence of aspiration or pulmonary oedema and there was no subcutaneous emphysema. Examination of the CNS showed the diver to be alert and orientated. Serial 7's were complete in 40s with one mistake. Three number recall and short term memory for phrases were normal. Cerebellar and cranial nerve examination was also normal. Sharpened Romberg test was normal, 50\60 right, 60\60 left. ECG showed atrial fibrillation with a ventricular rate of 170 beats a minute. Chest X-ray was normal.

Although the symptoms following the scuba dive suggested a diagnosis of DCI or cerebral arterial gas embolism (CAGE), the onset of symptoms after the initial breathhold dive and again at depth made such diagnoses unlikely. Following careful examination, it was judged that the cause of the diver's signs and symptoms were primarily myocardial, but precipitated by the initial breathhold dive. He was given 500 mcg of digoxin intravenously after which his heart rate slowed and he reverted to sinus rhythm.

Further questioning revealed that he had had two previous episodes of palpitations, one of which was associated with a breathhold dive 5 years earlier. On that occasion he had dived into a heated swimming pool to retrieve some coins from the bottom. On surfacing he felt excessively tired, slightly short of breath and had noted his heart to be racing. This was self limiting, lasting for approximately 45 minutes. A similar episode 2 years previously, not associated with diving, led his general practitioner to start him on a beta-adrenoceptor antagonist. This however the patient stopped taking it. Both the diver's parents had a history of ischaemic heart disease.

Following control of his atrial fibrillation he was reviewed by a cardiologist. Investigations included an echocardiogram, exercise ECG, complete blood count, biochemistry, thyroid function tests and cardiac enzymes, all of which were normal. A diagnosis of paroxysmal atrial fibrillation was made and he has been advised to refrain from scuba diving for 12 months, and then to have a full diving medical review by a specialist in diving medicine.

Discussion

Although the occurence of DCI and CAGE are well documented and have a high profile in the diving community, they account for only a small number of deaths associated with scuba diving. In a recent survey of causes of death of divers in Australia and New Zealand, 13% of fatalities were associated with pulmonary barotrauma, 12% of deaths were due to cardiac disease while there were none associated with decompression illness.[1] When the fatalities were examined for contributing causes, the incidence of cardiac disease among diving fatalities approached 21%. This group consisted of divers who had a mean age of 43.6 and died suddenly either at the beginning or end of a dive. Moreover, recent data from the Divers Alert Net-

work (DAN USA) has shown comparable figures for the incidence of cardiac disease and diving related mortality in the United States.[2]

The onset of our diver's symptoms was immediately following his first breath-hold dive. Although pulmonary barotrauma has been identified following breath-hold diving,[3] this is rare and was not identified in this case. The diver did not complain of a faulty regulator, and the lack of chest X-ray changes and of pyrexia make a diagnosis of salt water aspiration improbable. DCI is unlikely after breathhold diving, and does not usually present while at depth. Although pulmonary oedema following scuba diving has been described it usually presents with productive frothy sputum and abnormal radiological signs.[4]

While dysrhthymias have been reported during breath-hold diving, these are usually bradycardias or junctional rhythms.[5,6] Indeed the combination of facial immersion in cold water with maximal inspiratory breath-hold have been implicated in the augmentation of the diving reflex in man.[7] This has been used in clinical practice to terminate supraventricular tachycardia. Data is limited in humans regarding the rhythm changes seen during recreational scuba diving. McDonough reported self-limiting supraventricular tachycardias in divers during recreational dives in cold waters,[8] while asymptomatic ventricular fibrillation has been reported in a commercial diver during a training dive.[9] Factors important in initiating these dysrhythmias may include water temperature, anxiety level, effort during the dive and pre-existing cardiac pathology. Increased central blood volume secondary to body immersion causing raised right sided filling pressures resulting in stretching of atrial stretch receptors. This may be of importance in the initiation of such supraventricular rhythm problems. A further possibility is that the diver had in fact been in atrial fibrillation before the initial breath-hold dive. The combination of tachycardia combined with a sustained Valsalva manoeuvre impeding venous return could have led to a significant decrease in cardiac output, resulting in the diver's symptoms of extreme tiredness and malaise.

A study which looked at forearm vascular resistance in divers with a history of diving induced pulmonary oedema concluded that a pathological increase in vascular resistance occurred in an idiosyncratic fashion following exposure to cold.[10] As most of the divers with abnormal vascular reactivity developed hypertension later in life, this may be an important aetiological factor in its development. However studies which look at the prevalence of cardiac disease in the diving community and how diving affects divers under certain situations, e.g. deep diving, wreck, cave diving and during training, are lacking . It is known that novice parachutists have a dramatic cardiovascular response to their first jump, with heart rates of around 170 beats a minute occurring,[11] however there is no data on this aspect of scuba diving in the literature. This may have significance in those novice divers with known cardiovascular pathology or those at high risk e.g. strong family history of ischaemic heart disease, smokers and those with increased serum cholesterol.

As the Australian and New Zealand Study has indicated, at least 25% of divers who died were medically unfit.[1] This emphasises the need for physicians qualified in diving medicine to perform medicals on all prospective diving candidates and also raises the question of the need for certified recreational divers to have regular medical examinations to ascertain continued fitness to dive, similar to those involved in commercial diving.

Finally the question whether to allow this diver to return to scuba diving is debatable. The various investigations have shown no identifiable structural or metabolic reason for his dysrhythmia. The conseqences of this were mild and on one ocasion self terminating. However he has now demonstrated repeated episodes of cardiovascular instability, and these may not necessarily be as easily terminated in the future. Moreover by the nature of diving, urgent medical attention may not be possible for many hours or even days.

Conclusions

Cardiac disease may be more common in the recreational diving community than is currently appreciated. This factor, along with the potential for provocation during a dive, makes primary cardiac pathology high on the list of differential diagnoses in the diver who presents with chest pain, shortness of breath and extreme fatigue, symptoms which occur in DCI and CAGE. The diagnosis of a primary diving related pathology can be made only after exclusion of all other causes of the symptoms by a careful history taking and full clinical examination. Further studies are needed to identify more accurately the incidence of known or covert ischaemic heart disease among divers and the effects of scuba diving.

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THE WORLD AS IT IS

LONG TERM HEALTH OF PROFESSIONAL DIVERS

International Consensus Conference
held at
Godøysund, Norway, 6-10 June 1993

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Ten years ago an international workshop entitled "Long Term Neurological Consequences of Deep Diving", organised by the European Undersea Biomedical Society (EUBS) and the Norwegian Petroleum Directorate (NPD), took place in Stavanger, Norway. At that time preliminary results from a newly started Norwegian research program on possible long term effects of deep diving on the divers' health caused enough concern to arrange that workshop.

That time, U.S.A.,U.K., France, Sweden and Norway were represented with 38 invited participants. The views on the subject varied widely, and any attempt made to reach consensus failed.

In his introduction to the proceedings[1] of the conference, the chairman, Professor R.I. McCallum concluded as follows: "In view of the lack of hard evidence of actual neurological damage from deep diving and the need for more data, is there a case for limiting deep diving now? Many feel that deep diving, when properly carried out, is safe and I think it is fair to say that there was little support for limiting such activity at the present time, but rather for intensifying the monitoring of those taking part in it."

And the monitoring was intensified, including also the pulmonary and auditory functions. The results of our research have been published internationally, and met with scepticism. Nevertheless, this time we optimistically called the convention a ***consensus*** conference in our invitational leaflet, although we knew the subject was controversial.

Now the scope was widened from possible ***neurological*** effects of ***deep*** diving to possible effects on the diver's ***health*** of professional diving in a broader sense, and in addition to the nations participating in 1983, representatives from Australia, Switzerland and Ukraine were included. Of the 36 persons invited, only two did not show up at all. In addition, the authorities and the oil and diving companies had observers in the conference.

The discussions were lively, to say the least, but since the conference was held in a small island on the Norwegian west coast, no one could escape until some sort of consensus was reached. Naturally, the final statements had to be rather general:

"There is evidence that changes in bone, the central nervous system and the lung can be demonstrated in some divers who have not experienced a diving accident or other established environmental hazard. The changes are in most cases minor and do not influence the diver's quality of life. However, the changes are of a nature that may influence the diver's future health.

The scientific evidence is limited and further research is required to obtain more definite answers to the long term effects of diving."