divers, making fewer dives, but at more risk than the NUADC rosy safety statistics would suggest. Since the publication of that analysis, "Underwater USA" has quoted new NUADC figures which greatly reduce their "active" diver population estimates, e.g. by over a million divers. Even PADI's own US diver estimates are substantially less than the NUADC figures. A recent Diagnostic Research Inc. (DRI) survey commissioned by the Diving Equipment Manufacturer's Association (DEMA) reported only 2 million US divers. My model estimates were for experienced divers only. What if one adds in resort course divers (400,000+ per NUADC) and student divers (500,000+) to my updated model estimates for experienced divers? You get a figure which is close to the survey results for all divers reported by DRI/DEMA. I would therefore argue that the inflated "active" diver population figures of NUADC should be rejected. Similarly, we have to abandon all those rosy US diving safety statistics based on NUADC figures.

Is the death rate among Japanese divers really lower than the death rate among Australian divers? I think not. The Japanese diving deaths are reported by an official government agency. However, PADI Australia has estimated an "active" diver population among Japanese divers by using a process similar to that employed by NUSADC. The result has been similar, namely, an inflation in the number of "active" Japanese divers. This leads to a substantial underestimate of the true Japanese diving death rate, just as happened with the NUADC figures. As further evidence, I point to the precipitous drop in Japanese diving death rates from over 50 to just 20 per 100,000 divers in just a few years. I doubt such a large drop could occur so rapidly among so many divers. Using a more reasonable dropout rate estimate, my models suggest the true Japanese diving death rate is substantially above that calculated for Australia here.

I must admit I was very glad to see these Australian diving statistics because of the support they lend to my models and estimates. My models estimated a minimum US diving death rate of 16.7 diving deaths per 100,000 divers. But I believe the true rate is considerably higher than this minimum figure. I further feel that the US and Australian diving death rates are roughly comparable, although I believe that both the Australian and especially the Japanese diving death rate are still higher than the US figures based on my models.

If PADI US wishes to continue to claim such a low death rate among US divers, then they need to explain the tenfold higher death rates among PADI Australia certified divers, and do something about it! PADI Japan, which dominates the Japanese market and is the world's fifth largest training agency, also has some work ahead of it. I presume both entities would prefer to abandon the low US death rates and accept my models and conclusions as reported here. I think it is past time that the US diving industry accepted the conclusion that diving is not as safe as the NUADC figures would suggest. My models and estimates suggest that diving is not getting any safer, either. I believe there is plenty of room for improvement in how we teach diving and in how we dive. That's my primary message here.

I am understandably envious of my Australian colleagues, with your co-ordinated chamber and rescue operations and your independent diving statistics collection efforts, particularly Project Stickybeak. I am hopeful that the US diving community will see fit to follow your example. I have called for an independent US entity to collect not just diving mortality data but also diving injury data as well. My viewpoint is that such an entity will need to be financially independent of interested parties in the diving industry, including diving equipment manufacturers, trade associations, and diver training agencies. I agree with your ideas that such a morbidity and mortality database will enable us to improve diving safety. Using it, we can determine where and how we can make effective safety improvements in diver training and diving operations. Thanks to your efforts, Australia enjoys what is undoubtedly the world's best program in this regard. Your efforts provide compelling proof that such a program is both possible and very worthwhile. Keep up the good work!

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THE EQUIVOCAL BEND, SHOULD WE TREAT WITH HYPERBARIC OXYGEN?

David Smart

SUMMARY

Three case histories of divers presenting to the Royal Hobart Hospital (RHH) with minor symptoms and signs after diving have been retrospectively examined to assess the effect that treatment with hyperbaric oxygen (HBO) had on their condition. Hyperbaric oxygen produced complete amelioration of almost all symptoms in each of the three cases. The problems in interpreting the data relating to

equivocal decompression sickness (DCS) were examined. A thorough neurological and psychometric assessment should be performed in all cases to detect possible neurological involvement which occurs in a significant proportion of these minor syndromes. Treatment of less serious decompression sickness with hyperbaric oxgyen is justified.

Introduction

Much has been written about decompression sickness (DCS) since the link between gas bubbles in the body and the clinical syndrome was first described by Paul Bert last century1. In recent years greater emphasis has been placed on the more serious syndromes which present to the diving physician. The efficacy of hyperbaric oxygen (HBO) in these situations is well established.

Less has been written about the patient who presents with more diffuse symptoms after diving, not classically identifiable as DCS. Many of these symptoms are without signs and could represent a multitude of ills, the only common factor in their aetoiology being that the patient has recently dived and claims that the symptoms were not present before diving, or that diving made the symptoms worse.

Should all patients with suspected DCS be treated aggressively with hyperbaric oxygen? Traditionally only those with definite DCS (e.g. localised pain or neurological bends) have been treated with HBO.

In this study the case histories of three patients with atypical symptoms after diving were examined retrospectively to assess the effects that HBO treatment had on their condition. The patients were selected from 27 total divers treated at the Royal Hobart Hospital Hyperbaric Unit since August 1984. These cases are representative of six divers presenting with similar syndromes during the same period.

Case reports

Case 1

A 30 year old male abalone diver with 4 years' experience as a professional diver and a past history of an arthroscopy of his right knee for a meniscal lesion. No surgery was performed during the arthoscropy. He also had similar problems with his left knee but no orthopaedic intervention.

Three days before presentation he dived for six hours to 18-24 m of sea water (msw) then "decompressed" for one hour on the anchor line at 3.5-4.5 msw. After leaving the water at 4 p.m. and feeling slightly lethargic he cleaned his gear and went home. At 12 midnight (8 hours post dive) he noticed the onset of pain in his left calf, left knee and general fatigue with headache. His symptoms worsened over one hour then gradually improved. He was almost pain free when he saw his local doctor the following day. He had full range of movement both knees with neurological examination normal. Detailed sensation testing was not performed. He was prescribed oral fluids and 12 hours of 100% oxygen. By day three all of his pain had resolved except for a dull ache in his left knee exacerbated by movement. He still felt generally lethargic and his headache had not improved.

He was transferred to the Royal Hobart Hospital 82 hours post dive still complaining of a dull ache in his ledt knee despite full range of movement. Neurological examination was normal. He was recompressed using USN table 5, 84 hours after initial symptoms began. At 18 msw the patient was symptom free and remained so for the remainder of the treatment. After decompression a slight ache (less in intensity than before HBO treatment) had returned in his left knee. His constitutional symptoms had disappeared. After further 100% oxygen at one atmosphere absolute he returned home much improved. By one week he was symptom free.

Case 2

A 36 year old abalone diver had an influenza-like illness with cough, coryza, myalgia and vague fleeting polyarthralgia for a number of weeks. His general practitioner treated the productive cough with antibiotics and terbutaline with no response. The worst affected joints were his knees, elbows and shoulders.

He had dived six and five days prior to presentation for 8 hours each day to approximately 9 msw, however he did not use a depth guage. He did not decompress. The day after these dives the pains in his knees, elbows and shoulders became more severe. He presented 110 hours after his last dive because the pains had not improved. Examination revealed a neurologically normal man with full range of movement and power in all joints which had pain, the elbows, knees and shoulders.

He was recompressed using USN table 6 and during the second oxygen period at 18 msw noted that all symptoms had gone apart from slight left elbow and shoulder pain. This pain persisted when he was decompressed to the surface. After overnight oxygen, symptoms were still present and the patient was further recompressed for 60 minutes at 18 msw breathing 100% oxygen, with resolution of his symptoms. However, on decompression he complained of pain in his right knee. This had gone after further 100% oxygen at one atmosphere absolute for 12 hours but he had more pain in his elbows and upper arms.

Investigations performed were as follows:

FBC. Hb 15.1. WCC 6.8, normal differential. Platelets normal. ESR 1.

CT scan Brain normal

ASOT - Negative

Antinuclear antibodies positive 1:20 litre (non specific equivocal result).

After a further 24 hours, the patient was discharged home with fleeting minor symptoms, far less than at presentation and free of systemic symptoms. Subjectively he felt better. At follow up at one month he was asymptomatic.

Case 3

A newly qualified 21 year old female scuba diver with two months' experience had recently recovered from a viral illness. She had completed 12 open water dives since starting her course and the two days prior to her presentation dived the following profiles:

Day 1 15 msw for 60 minutes exit 1730

Day 2 15 msw for 60 minutes exit 1130
20 msw for 30 minutes entrance 1500

She had a rapid ascent at the end of this dive due to buoyancy control problems.

No decompression stop was performed after the second dive. No symptoms were noticed after the final dive. Three hours afterwards, whilst travelling home over hills at an altitude of 300 metres, she noticed the onset of headache, nausea, vomiting and vertigo.

These symptoms continued overnight with further vomiting. She presented 18 hours after her last dive with general fatigue, headache, myalgic pains in her neck, back and knees, and vertigo.

She was febrile (38.1 ^{0}C). There was no skin rash. Cardiovascular system, respiratory and gastro intestinal tract examinations were normal, as was neurological examination apart from falling to the left on sharpened Romberg test (standing with the feet one behind the other, arms folded and with eyes closed). There was no nystagmus and detailed sensation testing was normal.

Treatment consisted of recompression using USN table 6. This resulted in a marked improvement in her headache and lethargy. Her nausea still remained. After a night on 100% oxygen she was symptom free and her sharpened Romberg test was normal. Her fever had also gone. Electronystagmogram just prior to discharge was normal.

Discussion

The above cases illustrate some of the problems encountered by the diving physician when assessing divers who present with atypical symptoms after diving.

Common to the cases were:

1. Delayed onset in symptoms up to 8 hours post dive.
2. Minor non-specific symptoms.
3. Intercurrent illness or pre-existing complaints which clouded the presenting picture.
4. Delays in treatment of up to 110 hours.
5. All responded to HBO therapy with a significant reduction in constitutional symptoms.

It may be easy in retrospect to attribute all of the symptoms described in the case studies above to DCS, however the diagnosis was by no means clear at presentation. Attitudes of divers towards DCS play a role in clouding the picture at presentation and result in delays in seeking treatment. There is a tendency to rationalise symptoms and attribute them to a recent event or illness (e.g. a hangover, diver fatigure, or influenza). There is also a real fear amongst divers that to get bent will result in them being prevented from diving again or is a reflection of their inadequacy as a diver. Fear of losing their livelihood is understandable with professional divers, but should not be the case with sport divers. Injuries in other sports are only regarded as a temporary setback in their training and playing schedule, and perhaps DCS in divers should be regarded the same way.

These fears combined with the minor nature of the symptoms described in the case histories above probably accounted for the delay in seeking medical advice. It was only the persistent nature of the symptoms that led to the divers seeking medical advice.

The pattern of DCS resembling a viral illness with lethargy, nausea, light headedness and malaise has been described before alone or in association with more serious DCS2,[4]. In particular, headache is now recognised a very common finding in divers with DCS and occurred in 19 out of 27 divers treated at RHH since 1984. Fever has also been described[2]. In case 3, this fever apparently responded to HBO treatment.

Case 1 demonstrates the recurrence of symptoms in an old injury following diving, which was an isolated event on this occasion. Exacerbation of pain in a pre-existing injury after diving has been documented in the literature, possibly due to local changes in tissue perfusion and vascularity[2].

The patient in case 2 had significant symptoms for a number of weeks prior to diving. If these symptoms had not been documented prior to his dives there would be no hesitation in attributing the post-dive picture to DCS. In retrospect his earlier symptoms may well have been pain due to DCS. He responded to treatment with HBO but each time he returned to the surface symptoms recurred in different parts of the body. Due to the systemic nature of DCS, the pains can be migratory.

Case 3 demonstrated a similar systemic pattern of illness to case 2 with two important differences. Firstly, there was the description of vertigo, dizziness and vomiting and slightly abnormal findings on neurological examination. Secondly, the patient developed her symptoms on ascent to altitude with its concomitant changes in ambient pressure. The resultant effects on bubble formation within

the body are well known. Many cases of DCS precipitated by flying in aircraft after diving have been documented. Two patients treated for DCS in the unit at the Royal Hobart Hospital and discharged symptom free actually relapsed on ascent to 200 metres when travelling home and required further recompression.

In all cases above, treatment with HBO resulted in clinical improvement or complete abolition of symptoms. This provides further strong evidence that the symptoms were due to mild DCS. The milder DCS syndrome correlates with later onset of the symptoms. The earlier the onset of symptoms after diving, the more severe is the clinical picture2,6.

SHOULD THESE PATIENTS RECEIVE HBO ?

If it is assumed that symptoms result from bubble formation, then treatment can be justified to reduce bubble size, to increase the pressure gradient pushing nitrogen back into solution from the gas phase, and to reduce distal tissue hypoxia. Venous gas emboli have been demonstrated by doppler techniques in a majority of patients being exposed to shallow air saturation at 7.7 msw and 8.9 msw for extended periods of time7. The duration that bubbles were present in veins correlated with symptoms. Symptoms noticed by these patients included fatigue, limb and joint pain, headache, myalgias and pruritis. Except for pruritis, these symptoms occurred in the divers described in this paper. Hence treatment with HBO to reduce the bubble load may be justified even in minor cases of DCS. It results in improvement in up to 95% of cases[8]. In many studies less severe cases of DCS have been found with more detailed assessment to have neurological involvement[9]. This provides additional weight in favour of treatment of the more 'minor' syndromes.

SHOULD LESS SEVERE CASES OF DCS WITH DELAYED PRESENTATION BE TREATED ?

The cases presented here had delays of up to 110 hours before treatment was initiated. Some authors regard the potential seriousness of DCS to be so significant that they advocated treating even factitious cases until full particulars were obtained[10]. There is considerable evidence in favour of the efficacy of delayed treatment of DCS with hyperbaric oxygen in all degrees of severity[2,4,5,11,12,13,14,15]. The persistence of symptoms is thought to result from continued presence of gas bubbles in the tissues, and activation of haemostatic mechanisms[15,16].

There are many difficulties in interpreting data from retrospective case analyses when patients are treated with HBO.

Firstly, the placebo effect of HBO is not known. No data exists on the effect of HBO on symptoms described above in patients who have not dived. It is not possible to generate a control treatment when constructing a clinical trial because the pressure effects of hyperbaric therapy are difficult to mimic. In generating a controlled study, the ethical problems of not treating patients with DCS are also encountered. Non diving controls in such a trial would be at greater risk of barotrauma if subjected to HBO.

A high percentage of divers with cutaneous and musculoskeletal DCS have been shown to have detectable deficits when given more thorough neurological, EEG and psychometric testing[9]. A recent report from the Royal Navy demonstrated frequent frontal lobe perfusion defects in patients with DCS[17]. Considering this evidence and data presented in Eckenhoff's study[7], it is apparent that this form of DCS is not as benign as was previously thought. A strong argument can be made in favour of HBO treatment of divers with more minor DCS syndromes. More detailed assessment including psychometric testing and single photon emission computerised tomography (SPECT) scanning (if available) should occur at presentation. The natural history of DCS is for gradual remission, however the long term effects of not treating minor cases are not known.

Conclusions

Presented here are three case histories of divers who developed minor or non-specific symptoms after diving and which initially caused some difficulty in diagnosis. All were treated with hyperbaric oxygen and all responded to treatment. It is likely, in retrospect, that each had symptoms which could be attributed to DCS. In view of the relative safety of treatment, and the fact that HBO acts partially to correct the underlying initial pathological event, treatment with HBO is justified in "equivocal" cases of DCS even after delayed presentation. Further studies combined with psychometric testing, electroencephalography and SPECT scanning are needed to ascertain the natural history of the syndrome of myalgias, fatigue, headache and non-specific symptoms, with and without HBO treatment.

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MINUTES OF THE MEETING OF THE EXECUTIVE COMMITTEE OF SPUMS HELD AT MANA ISLAND ON 11TH JUNE 1988

PRESENT

Drs A.Slark, C.Acott, G.Barry, J.Knight, A.Sutherland (NZ Representative), and (by invitation) J.Williamson .

APOLOGIES:

Drs D.Davies, C.Lourey, P.McCartney, and D.Walker.

1. The meeting of the Executive Committee was declared open at 1200 hours.

2. MINUTES OF THE LAST MEETING:

The minutes were read and are to be amended to include discussion of the article in "Chest".

3. MATTERS ARISING:

3.1 The President will write to the various registration bodies in Australia and New Zealand informing them of the existence of the Diploma but specifically pointing out that SPUMS is not seeking registration of the Diploma.

3.2 The next AGM will be held in Vila as previously decided in Adelaide. It should be timed to finish prior to the 5th of June, 1989 so that members can also attend the Undersea and Hyperbaric Medical Society (UHMS) meeting in Honolulu from the 7th to the 11th of June, 1989. It is hoped that UHMS members will attend the SPUMS meeting. Insurance arrangements should be made such that a refund would be available if the meeting should be cancelled for any reason.

4. NEW BUSINESS:

4.1 Dr Williamson raised the matter of insurance to cover sickness, diving accidents and retrieval. Allways Travel had informed him that there was a Group Insurance Policy with GRE Insurance, however, GRE (Brisbane) could not confirm the existence of this policy. The policy issued by GRE to Dr Williamson did cover the costs of retrieval but the company was surprised at the suggested costs that would be involved. Dr Acott will investigate further.

4.2 Dr Peter McCartney is holding a seminar in Hobart in early November. Several Executive members will be giving papers. It is suggested that the next Executive meeting is held at that time.

5. The meeting closed at 13.15 hours.