

## ORIGINAL ARTICLES

### UNDERWATER OXYGEN TREATMENT OF DECOMPRESSION SICKNESS

#### A REVIEW

Carl Edmonds

#### Abstract

The problem of decompression sickness (DCS) in remote areas is described with particular reference to the Indo-Pacific islands. The various approaches of medevac, surface and underwater oxygen (UW O<sub>2</sub>) are addressed and the techniques and equipment used in underwater oxygen are documented.

The favourable experience with the original UW O<sub>2</sub> tables are compared with the less conservative, more hazardous, oxygen decompressions used by abalone divers and the shorter but successful exposures of the pearl divers. The latter imply that, with very prompt treatment, the 9 m oxygen treatment may be reduced in duration.

#### Background

Treatment of decompression sickness (DCS) in the mid 1960s involved recompression with air, at a minimum depth of 30 m and more frequently at 50 m.

The first case on which I was consulted was another diving physician who had, that day, treated another diver in the chamber. The fact that the diver/patient got moderately better and the physician got "bent", did not inspire confidence in the treatment tables. Nor did a review of the success of other cases treated.

Australia had one established recompression chamber (RCC) capable of applying the conventional treatments, in Sydney, at the Royal Australian Navy (RAN). We were therefore committed to the diving medical cover for all civilians as well as service personnel.

Our catchment area covered a radius of about 5,000 km and included many excellent diving sites, but often with primitive diving and aviation facilities.

The air table failures were presumed to be a consequence of getting civilian divers many hours or days after the symptoms had developed and the pathology stabilised. This was possibly not the experience of other organisations, such as where commercial divers could be treated immediately.

#### Medevac

The RAN and RAAF accepted responsibility for treatment of civilians in 1965, in lieu of any alternative, from most of the surrounding Indo-Pacific region. From 1968, to reduce the delay if the diver was significantly injured, we were as likely to take all the equipment (chambers, oxygen, appliances, etc.) to him, as we were to bring him to the chamber. It all depended on which was the quickest. We preferred RAAF Hercules aircraft, pressurised to sea level (1 atmosphere), for transport.

Only serious cases warranted medevac from such distances. The SOS decompression meter contributed to the unacceptably heavy case load during the early 1970s.

#### Surface Oxygen

In 1968 we started using oxygen while awaiting recompression, during the inevitable delays. The diver would receive oxygen in transit to the chamber, or he would be placed on oxygen while we brought the chamber to him. This decision was based on the writings of Paul Bert<sup>1</sup>, and some unpublished experiments with guinea pigs. Most clinicians who used this first aid regime, in both Australia<sup>2</sup> and France,<sup>3</sup> seemed to be impressed with its success. It is now internationally accepted.<sup>4</sup>

#### Oxygen tables

Fortunately, in 1965, Goodman and Workman<sup>5</sup> produced their oxygen tables, allowing us to start treatment of almost all DCS cases at 18 m. These really only became used, with seriously ill divers, in about 1967. The oxygen treatments were also inadequate in many cases, possibly because of the delays and the development of complex pathophysiological changes only now being elucidated.<sup>6</sup>

That was when we decided to experiment. If a patient got worse during treatment, then the treatment was modified for that type of case. We capitalised on the beneficial effects of both pressure and oxygen without preconception. We took the usually severely ill diver to the shallowest depth that produced a satisfactory (but not necessarily complete) clinical response, i.e. one assessed as not to lead to permanent sequelae. We then decompressed with the maximum oxygen that would not produce convulsions. Each depth range had its own acceptable O<sub>2</sub> %, which was achieved by mixing air with 33% O<sub>2</sub>, 40% O<sub>2</sub>, 60% O<sub>2</sub> or 100% O<sub>2</sub>. Dramatic treatment for a serious illness.

Those were called the Australian tables<sup>7</sup> and I would still revert to them for serious cases (not the indefinite cases with “soft” signs that now seem to predominate). We even avoided air breaks as we saw little value in perpetuating a nitrogen problem; also it seemed some patients deteriorated at or soon after the air break. We later used heliox to reduce the occasional respiratory oxygen toxicity.

The UW O<sub>2</sub> tables introduced soon after this, were no more than the shallow part of these “Australian tables”, from 9 m to the surface.

This segment was frequently used in the RCC to treat;

- very recent cases (e.g. those who developed DCS from the navy chamber),
- minor cases of DCS,
- those in which we were not convinced of the diagnosis, and
- very delayed stable cases e.g. musculo-skeletal DCS, days after the dive (these responded equally well to surface oxygen).

Independently, the French developed their Comex tables,<sup>8</sup> which were a middle ground between the formal but very limited US Navy tables and the more flexible and thus complex Australian ones. The 9 m UW O<sub>2</sub> table differed little in effect from the subsequent COMEX 12 m RCC table

### Underwater oxygen treatments

This was developed in the late 1960s at the RAN, and by 1970 was employed through many parts of the Indo-Pacific,<sup>7</sup> where chambers were not readily available. The reason I attest no doubt regarding the origin of this treatment, is that no one else seemed to be prepared to share the flack when the knowledge of our techniques spread to the USA in about 1973 or when reported at an international conference in France, in 1978.<sup>9</sup>

The UW O<sub>2</sub> regime is still employed by many divers in remote areas, such as in the Pacific islands, the abalone fields of southern Australia, and the pearl fields of the Australian north. But variations in technique have developed. In Hawaii they have combined it with their UW air techniques, producing a deep air dip followed by the UW O<sub>2</sub> regime. I have no experience of this last modification, but I can elaborate on the others.

The UW O<sub>2</sub> treatment is now a part of many national diving manuals. It was included in the Royal Australian Navy manual as tables 81 and 82, but took 15 years and a some modifications, before it found its way into US Navy Diving Manual.

### Rationale

The value of substituting oxygen for air in the recompression chamber treatment of DCS, is now well established. The pioneering work of Behnke, Yarborough and Shaw,<sup>10,11</sup> over 50 years ago, eventuated in the oxygen tables produced 30 years ago.

The advantages of oxygen over air breathing include: increasing nitrogen elimination gradients, avoiding extra nitrogen loads, increasing oxygenation to tissues, decreasing the treatment depths and exposure time, reducing vascular/haematological damage, and improvement in overall therapeutic efficiency. The same arguments are applicable when one compares UW O<sub>2</sub> and UW air treatments.

Certain other advantages of UW O<sub>2</sub> over underwater air are obvious. Attendant divers are not subjected to the risk of DCS or nitrogen narcosis, and the affected diver is not going to be made worse by premature termination of the treatment, if this is required. Hypothermia is much less likely to develop, because of the greater efficiency of the wet suits at these depths.

The underwater site chosen can often be in a shallow protected area, reducing the influence of adverse weather on the patient, the diving attendants and the boats. Communications between the diver and the attendants are not difficult, and the situation is not as stressful as the deeper, longer, underwater air treatments or even as worrying as in some 3rd World recompression chambers.

### Technique

Whenever oxygen is given, the cylinder should be turned on and the flow commenced, before it is given to patients or divers to breathe.

Oxygen is supplied at maximum depth of 9 m (30 ft), from a surface supply. Ascent is commenced after 30 minutes in mild cases, or 60 minutes in severe cases if significant improvement has occurred (this time may be extended for another 30 minutes if there has been no improvement). The ascent is at the rate of 12 minutes per metre or 4 minutes/foot. After surfacing the patient should be given periods of oxygen breathing, interspersed with air breathing, usually on a one hour on, one hour off basis, with respiratory volume measurements and chest X-ray examination where possible.

### Equipment

No equipment should be used with oxygen if it is contaminated, dirty or oil lubricated.

The equipment required for this treatment includes the following: a large oxygen cylinder (e.g. 220 cubic feet (7,000 litres), G size). This is usually available from local hospitals, although in some cases industrial oxygen can be used from engineering workshops. Breathing this oxygen at a depth between 9 metres (30 ft) and the surface, for this duration, is usually insufficient to produce either neurological or respiratory oxygen toxicity. A 2 stage regulator, set at 550 kPa (80 psi) and fitted with a safety valve connects with 12 metres (40 ft) of supply line (HP hose). This allows for 9 m depth; 2 m from the surface of the water to the cylinder, and 1 metre around the diver.

A non-return valve is attached between the supply line and the full face mask (e.g. a Cressie-Sub). The latter is inexpensive and enables the system to be used with a semi-conscious or unwell patient. It reduces the risk of aspiration of sea water, allows the patient to speak to his attendants, and also permits vomiting without obstructing the respiratory gas supply. The supply line is marked in distances of 1 m from the surface to the diver, and is tucked under the weight belt, between the diver's legs, or is attached to a harness. The diver must be weighted to prevent drifting upwards.

A diver attendant should always be present, and the ascent controlled by the surface tenders. The duration of the 3 designated tables is 2 hours 6 minutes, 2 hours 36 minutes and 3 hours 6 minutes.

The treatment can be repeated twice daily, if needed. Some experienced divers use an oxygen re-breathing system. Recreational divers tend to prefer oxygen from a (well marked) designated scuba.

### Experiences with UW O<sub>2</sub>

Apart from the original trials with UW O<sub>2</sub> (see Annexe A and references<sup>7,9,12</sup>) most of the cases known to me come from 3 very different diving communities:

#### TROPICAL ISLAND DIVERS

There is no way of knowing the number of cases treated on the tropical islands of the Indo-Pacific. Some areas, with which I am more personally associated, have advised me of dozens of such cases in each of the following localities; Solomon Islands, Papua New Guinea, Rabaul / Kimbi, Torres Strait Islands.

I am aware of other areas because of; the cases referred back to me, if they reside near Sydney, or where I have been directly involved in the treatments (Christmas Island, Lord Howe Island, the Cook Islands, Nauru, Truk Lagoon and other areas of Micronesia and the Great Barrier Reef). I am aware of only one accident during these treatments, but the aetiology is problematical. See Annexe B.<sup>13</sup>

#### AUSTRALIAN ABALONE DIVERS

In 1985, of the 200 or so registered professional abalone divers of Australia, 152 were submitted to a diving questionnaire, personal history taking, physical examination and various investigations.<sup>14</sup>

These divers were exposed to excessive diving durations, and 58% of them routinely employed a dive profile which required some form of decompression, according to the US Navy Tables, but which was omitted.

Although they employed repetitive diving, and some multi-level diving, this was frequently not in the manner usually recommended. On the contrary, the dives tended to be deeper as the day progressed, with deteriorating sea conditions. Also the water temperature was often cold (4-10 C).

At that time there was considerable ignorance in the field, as regards the UW O<sub>2</sub> techniques being employed by the RAN School of Underwater Medicine. Indeed, the few ex-RAN divers that were working as abalone divers at the time were usually a source of mis-information, having only been exposed to the conventional oxygen treatment tables used in the recompression chamber. A popular belief evolved that oxygen could be safely used at 18 m, as long as it was used for treatment.

Oxygen was rarely used for decompression per se, without decompression sickness, at that time. It had a poor reputation and the majority of the divers neither employed oxygen as a treatment nor had it on the boat. However, 8.6% had used oxygen for treatment on the surface, 7.9% also used it at a depth of 9 m or less underwater, 5.3% also used it at a depth greater than 9 m underwater. No diver used it in excess of 18 m.

Of the 625 cases of DCS that could be remembered by the 152 divers, 11% were treated in a recompression chamber, of which over half were neurological DCS, 15% were treated on the surface, with O<sub>2</sub>, 66% were treated underwater, on air and/or oxygen and 22% were not treated at all.

These figures are probably not accurate, because of the inevitable vagaries of memory and denial.

The DCS incidence is especially misleading, as many of the divers would complain of joint and other symptoms after diving that they would not attribute to DCS. As a general rule, they would ignore minor symptoms, without considering them to be DCS, or they classified them as "niggles" so not requiring any activity.

Problems with oxygen toxicity are documented in Annexe C. Note that all of these cases were using oxygen at much greater depths than recommended (>15 m).

An informal survey was undertaken by letter (it is not easy to obtain replies from this occupational group) to ascertain the current status of UW O<sub>2</sub>. After the 1985 Abalone Diver Survey, in which the UW O<sub>2</sub> regime was described, it would have been rewarding to report a safer oxygen use. Unfortunately this is not so. It has now superseded underwater air treatments, and is used frequently.

Although most of the deeper divers (18 m+) now routinely carry and use oxygen for treatment, and frequently for decompression, they have a large variety of protocols. Some use the UW O<sub>2</sub> for treatment, as prescribed. Others return to the depth of the dive (as deep as 30 m). Others routinely decompress on oxygen from variable depths, to avoid DCS. Re-education appears warranted.

#### PEARL DIVERS

This occupational group of about 100 divers off the North-West of Australia have had a horrendous diving history. Edwards<sup>15,16</sup> who writes on the history of these men, postulates that they have lost about 1,000 divers over the century of pearl diving.

The very optimistic and atypical "official" figures for 1993 claim no deaths and only 3 (later upgraded to 12) cases of DCS, all successfully treated by UW O<sub>2</sub>, amongst the 74 divers in Broome. They conducted: 21,452 dives, about 15,000 hours underwater, averaging 290 dives per diver

The following information is pertinent:<sup>16-18</sup> They dive daily for 6-10 days, at the neap tides each month, for 5-6 months each year, 5-10 times per day, usually to depths varying from 15-45 m, but they do extend this range. The consecutive dives are as often deeper, as shallower. They usually use O<sub>2</sub> for decompression for some dives 13 m to 21 m and all dives over 23 m.

I carried out a survey by inspection of the diving logs (representing about 10% of the dives out of Broome and Darwin) over a 4 year period, 1988-1991, which is less reassuring. DCS was the commonest medical disorder recorded (45%). The existence of a DCS diagnosis in the diving logs was verified by the recorded extra O<sub>2</sub> decompression time. This involved a administration or extension of O<sub>2</sub> at 9 m for 30-45 minutes.

The incidence of DCS from a diving day increases progressively from: 0.2 % at 10-14 m depths to 13.6 % at 45-54 m depths.

Of the 1,834 diver days worked (11,776 dives), there were 56 cases of DCS. 1 required medevac. Fifty five were treated successfully on the U W O<sub>2</sub> regime.

By extrapolation to the remainder of the Broome and Darwin fleets, we can calculate a DCS case load of

about 500 treated underwater on oxygen over those four seasons of diving.

Provisos must be noted.

- 1 All cases occurred at sea, and treatments were usually given within 30 minutes.
- 2 Irrespective of the symptomatology, the illness was always referred to as "a niggle". This, according to their regulations, permitted the diver to resume diving and thus not lose any days diving (and therefore money). Clinically, they were very obvious DCS cases.
- 3 Except for the diver who required medevac, most divers continued diving on that or the next day without any more problems (49/55).
- 4 We have no idea of how this treatment influences the propensity to dysbaric osteonecrosis.

Like the abalone divers, the pearl divers have modified the treatment regime, but not in the same manner. Their consistent routine is to employ oxygen for 30 minutes at 9 m, extendible if any symptoms persist, and then ascend at a relatively fast rate, 3 m per minute.

The rapid exposure to effective treatment may explain an apparent discrepancy, as in many of our delayed-treatment RCC cases, attempts to reduce the ascent rate from a very slow 12 minutes/m to 9 minutes/m, occasionally resulted in recurrence of symptoms.

As regards oxygen exposure, the 1988-91 pearl divers survey<sup>20</sup> disclosed a great deal of oxygen exposure, for both decompression and recompression therapy. Based on a 10% sample, there was a total of 10,064 days diving with oxygen. It averaged 70 minutes usage per day (range 10-150 minutes), spread over 1-5 dives with increasing durations and depending on the original dive profiles. There were no oxygen convulsions or toxicity's noted during this period. Nor have there been any since (personal communication, Dr Robert Wong, 1995).

#### Discussion

The physiological principles on which UW O<sub>2</sub> is based are well known and not contentious, although the indications for treatment may be.

It was originally hoped that the UW O<sub>2</sub> treatment would be sufficient for the management of minor cases of DCS and so avoid medevac requirements, and to prevent deterioration of the more severe cases while suitable transport was being arranged. When the regime is applied early, even in the serious cases, the transport was rarely required.

It is a common observation that improvement continues throughout the ascent, at 12 minutes per metre. Presumably the resolution of the bubble is usually more

rapid at this ascent rate than its expansion due to Boyle's Law.

The UW O<sub>2</sub> recompression treatment is not applicable to all cases, especially when the patient is unable or unwilling to return to the underwater environment. It is presumably of less value in the cases where gross decompression staging has been omitted, or where a coagulopathy has developed. I would be reluctant to administer this regime where the patient has epileptic convulsions or is unconscious. Reference to the case reports in Annexe A reveals that others are less conservative.

One of the common reservations in Australia<sup>19</sup> is that this underwater treatment regime is applicable to the semi-tropical and tropical areas (where it was first used), but not to the southern parts of the continent, where water temperatures may be as low as 4°C. There are certain inconsistencies with this belief. Firstly, if the diver developed DCS while diving in these waters, then he is most likely to already have effective thermal protection available to him. Also, the duration for the UW O<sub>2</sub> treatment is not excessive, at a depth in which his wet suit is far more effective than at his original diving depth. If he is wearing a dry suit the argument is even less applicable. The most effective argument is that it is used, and often very successfully, in the cold southern waters of Australia.

Some claim that the UW O<sub>2</sub> treatment is of more value when there are no transport facilities available. Initially this was also our own teaching, but with the logic that comes with hindsight, only a 3 hour gap is needed between the instituting of UW O<sub>2</sub> therapy and the arrival of transport, to be able to utilise this system. It is probably more important to treat the serious cases early, even if full recovery is not achieved, than to allow the progression of pathology during those hours.

There is no doubt, especially in serious cases, that transport should be sought while the underwater treatment is being utilised.

There has been a concern that if this technique is available for treatment of DCS, other divers may misuse it to decompress on oxygen underwater, and perhaps run into subsequent problems. This is more an argument in favour of educating divers, than depriving them of potentially valuable treatment facilities. One could use this illogical argument to totally prohibit all safety equipment, including recompression chambers, and thereby hope to circumvent all diving related problems.

It has been claimed that UW O<sub>2</sub> treatment is unlikely to be of any value for those patients suffering from pulmonary barotrauma. It may well be so in some cases. The treatment was not proposed for this. It is, however,

possible that the treatment may be of value for mediastinal emphysema, and perhaps even a small pneumothorax.

When hyperbaric chambers are used in remote localities, often with inadequate equipment and insufficiently trained personnel, there is an appreciable danger from both fire and explosion. There is the added difficulty in dealing with inexperienced medical personnel not ensuring an adequate face seal for the mask. These problems are not encountered in underwater treatment. Medevac's aggravate these difficulties and also introduce appreciable hazards of their own.

The UW O<sub>2</sub> treatment table is an application, and a modification, of current regimes. It is not meant to replace the formal treatment techniques of recompression therapy in chambers. It is an emergency procedure, able to be applied with equipment usually found in remote localities and is designed to reduce the many hazards associated with the conventional underwater air treatments.

The customary supportive and pharmacological adjuncts to the treatment of recompression sickness are in no way superseded, and the superiority of experienced personnel and comprehensive hyperbaric facilities is not being challenged. The UW O<sub>2</sub> regime, as described, is considered as a first aid regime, not superior to portable recompression chambers, but sometimes surprisingly effective and rarely, if ever, detrimental.

The relative value of current first aid regimes (the various UW O<sub>2</sub> procedures, including an additional deep air dip, and surface oxygen administration) needs to be clarified.

Whether we approve of the concept or not, it will continue to be used for as long as it is needed. The various diving communities are widening the UW O<sub>2</sub> protocol, and this may reflect the different types of cases encountered and the speed of its application.

Until we understand DCS better, the divers are more likely to research this field than medical experts, and they are unlikely to abide by our preconceived but well-intentioned restrictions.

The most effective way that I can envisage us contributing to diving medical first aid for DCS in remote areas, is by demonstrating a safer but equally effective UW or surface treatment e.g., with a helium/oxygen mixture that can be stored and used in emergencies, as oxygen is now.

#### **ANNEXE A CASE REPORTS**

Because this treatment is often applied in remote localities, many cases are not well documented. Twenty

five cases were well supervised before this technique increased suddenly in popularity. Two such cases are described.<sup>12</sup>

#### Case 1.

A 68 year old male salvage diver did two dives to 30 m (100 feet) for 20 minutes each with a surface interval of one and a half hours, while searching for the wreck of HMAS PANDORA about 100 miles from Thursday Island in the Torres Strait.

No decompression staging was possible, allegedly because of the increasing attentions of a tiger shark. A few minutes after surfacing, the diver developed paraesthesia, back pain, progressively increasing in coordination and paresis of the lower limbs.

Two attempts at underwater air recompression had been unsuccessful when the diving boat returned to its base moorings. The National Marine Operations Centre was finally contacted for assistance.

It was about 36 hours after the last dive, before the patient was finally flown to the regional hospital on Thursday Island. Both the Air Force and the Navy had been involved in the organisation, but because of very hazardous air and sea conditions, and very primitive air strip facilities, another 12 hours would have been required before the patient could have reached an established recompression centre 3,000 km (2,000 miles) away.

On examination at Thursday Island, the patient was unable to walk, having evidence of both cerebral and spinal involvement. He had marked ataxia, slow slurred speech, intention tremor, severe back pain, generalised weakness, difficulty in micturition, severe weakness of his lower limbs with impaired sensation, increased tendon reflexes and equivocal plantar responses.

An UW O<sub>2</sub> unit was available on Thursday Island for use by the pearl divers. The patient was immersed to 8 m depth (the maximum depth off the wharf). Two hours were allowed at that lesser depth and the patient was then decompressed. There was total remission of all symptoms and signs, except for small areas of hypoaesthesia on both legs.

#### Case 2.

A 23 year old female sports diver had been diving with a 2,000 litres (72 cu ft) scuba cylinder in the Solomon Islands. The nearest recompression chamber was 3,500 km. away and prompt air transport was unavailable. The dive was to 34 m (114 ft) for approximately 20 minutes, with 8 minutes decompression. Within 15 minutes of surfacing she developed respiratory distress, then numbness and paraesthesia, very severe headaches, involuntary extensor spasms, clouding of consciousness, muscular pains and weakness, pains in both knees and

abdominal cramps. The involuntary extensor spasms recurred every 10 minutes or so.

The patient was transferred to the hospital, where neurological DCS was diagnosed, and she was given oxygen via a face mask for three hours without significant change. During that time an UW O<sub>2</sub> unit was prepared and the patient was accompanied to a depth of 9 m (30 feet) off the wharf. Within 15 minutes she was much improved, and after 1 hour she was asymptomatic. Decompression at 12 minutes per metre was uneventful and the patient was subsequently flown by commercial aircraft to Australia.

### ANNEXE B UNDERWATER OXYGEN CONVULSION (Heron Is)

The only complication amongst "recreational" divers that I am aware of, is in a diver who sustained a tethering of his air line, followed by an emergency ascent, during which he may or may not have sustained pulmonary barotrauma, but which he definitely did inhale a considerable amount of salt water into his lungs.

Because of the rapid ascent and the fear of the development of DCS, he was given oxygen through a full face mask at a depth of 8 m. Within a few minutes he began to show clonic movements of his limbs and appeared to be losing consciousness. He was surfaced, and treated for his salt water aspiration, which cleared up over the next 24 hours. There were no sequelae but the provisional diagnosis of oxygen toxicity was made.

I find it difficult to understand how one can become toxic to oxygen, having sustained a salt water aspiration that produces an appreciable drop in arterial oxygen levels. It is not really known whether the problems were due to oxygen excess from the treatment or hypoxia from salt water aspiration. He did not have a typical epileptic fit.<sup>13</sup>

The actual events were clarified only after I attempted to follow up the case, and found that the movements were definitely clonic, the "unconsciousness" was only a possible impairment of consciousness and he was apparently cyanotic on the surface. Unfortunately the clinical data in this particular case is extremely unreliable, making differential diagnosis difficult. The heading of the article was misleading, to say the least.

### ANNEXE C ABALONE DIVER PROBLEMS WITH UNDER WATER OXYGEN BREATHING

There were a few cases of problems, usually associated with using oxygen in excess of 9 m depth, and often using it while continuing to catch abalone, thereby

employing their oxygen decompression time in a more valuable manner. The cases were as follows:

#### Case 1

Breathing oxygen at 12 m caused his lips to “go funny” and he noted a tingling and numbness over the whole of his body.

#### Case 2

Used oxygen mainly because of his navy training and therefore his experience with this. The maximum depth and duration would be 1 hour at 15 m. He would continue collecting abalone during that time and sometimes noted his right arm twitching and jerking, a loss of sight, appearance of star light objects underwater, twitching of the mouth and body. He claimed never to have lost consciousness underwater, however other abalone divers state that this is not so and that he had been rescued at least once by his boatman.

#### Case 3

Lost consciousness after a few minutes (it must have been more than this as he had half filled his abalone bag) at about 18 m.

#### Case 4

Never used oxygen in excess of 18 m, and always tried to reduce the duration even at shallow depths, to less than 3 hours.

#### Case 5

After breathing oxygen for more than 10 minutes at 18 m, his eyes went swimmy and fuzzy and he started to twitch. These symptoms indicated to him that it was time to quit.

It can be seen by the above case reports that some basic training in the use of oxygen underwater was required and this was given during the 1985 Australian Abalone Diver Survey.

It is believed that, since that time, most of the abalone divers have been using the underwater oxygen, but not always as proposed in reference.<sup>7</sup> One was particularly worrying. The diver dives to 30 m regularly, and uses oxygen for both decompression and treatment from that depth. He frequently notices visual symptoms, such as “mini stick figures running around the edges of my vision”. He will not alter this regime as he “feels better with it”.

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