## SURFACE OXYGEN IS AN ACCEPTABLE DEFINITIVE TREATMENT

#### Alf Brubakk

Key Words

Bubbles, decompression illness, oxygen, treatment.

#### Introduction

Richard Moon has presented why he believes that decompression illness (DCI) should always be treated by recompression to 18 m. We were ask to present two radically opposed views on what is required for the successful treatment of DCI. Here I will give a background on why I think there is a place for less than conventional and optimal therapy of decompression illness; optimal treatment still being the use of USN 6 at a well equipped and staffed treatment facility.<sup>1</sup>

Decompression illness can strike anytime and at any place, even with strict adherence to the decompression schedules. Many factors not associated with depth and bottom time can lead to this. For the divers in the audience it is worth citing something about the risk factors, written in 1876 by Snell,<sup>2</sup> who was in charge of people doing caisson work. "Fullness of habit; age; grey hair; exercise after decompression and alcohol abuse." I think the only risk factor that we do not have here in Layang Layang is exercise. The point about this is to make us remember that a large number of divers are at risk, often far away from any proper treatment facility.

There is probably general agreement that the symptoms of DCI are caused by the presence of free gas. The actual symptoms are of course dependent on where the bubbles are located. If the symptoms are minor, like skin itches or pain in a shoulder, this could be a sign that a severe problem might evolve or it may be a single symptom. If local bubbles in the shoulder is the only problem, then it probably is not very serious to have some small area of necrosis in the joint, if there are no other bubbles present. I want to point out here that, in my opinion, it is not likely that there will be bubbles only where there are symptoms. Bubbles can probably form in the venous system at any supersaturation<sup>3</sup> and several studies have shown that 85-90% of individuals with signs of musculo-skeletal decompression sickness also have other clinical signs, mostly from the central nervous system.<sup>4,5</sup>

However, the important question is, in my opinion:

What is the risk of serious sequelae after decompression illness following non-standard treatment of a single or a few incidents ?

## Sequelae after DCI

We do not know much about the natural course of DCI. However Snell said that pains in the limbs did not last more than 5 or 6 weeks and were not followed by any sequelae.<sup>2</sup> We know now that this is probably not correct, because there seems to be a connection between repeated cases of DCI and dysbaric osteonecrosis, whether treated or not.<sup>6,7</sup> Interestingly, DCI paralysis usually also passed off in from one to a few weeks. This was, of course, only when the individuals did not die, which would be not a very good end point for modern diving. The horrendous mortality and morbidity of the early series before adequate decompression and the recompression treatment of decompression sickness was introduced,<sup>8</sup> is not really relevant to modern recreational diving.

Using questionnaires, we studied the habits of Norwegian divers.<sup>9</sup> This study included sports divers, professional air divers as well as saturation divers from the North Sea. However we will here only present the data from the air divers, a total of 1,105 divers or about 63% of the diving population at that time. Figure 1 shows the incidence of treated and unreported decompression problems in these populations. Unreported decompression problems were defined as symptoms, which, had they been reported at the time, would have led to recompression treatment.



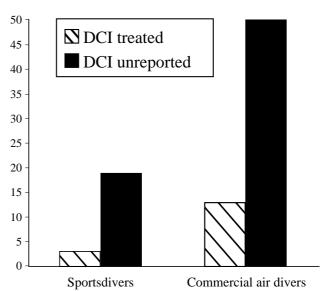
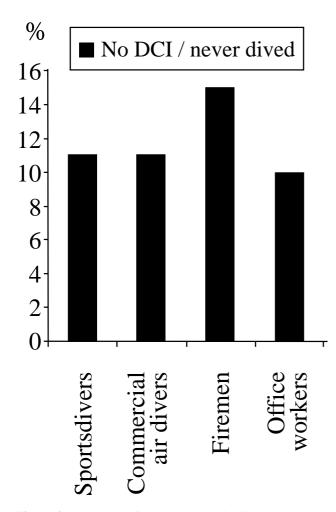


Figure 1. The incidence of treated and unreported DCI in sports- and air divers. Data from  $^9$ 

We furthermore used a standard set of questions that have been used for evaluating people who have had slight head trauma or who had been exposed to solvents. Using this questionnaire, approximately 15% of the population will have significant mental symptoms, such as short-term memory loss, irritability, lack of concentration, or periods of depression.  $^{10} \ensuremath{$ 

We will here only concentrate on the air divers, both commercial and sports divers. Our control group, which consisted of a large group of firemen and office workers, as well as the divers who had never had any decompression symptoms, all had approximately the same incidence of such mental problems as can be seen from Fig 2. We can see that the incidence of minor mental problems is similar in both diving groups and not significantly different from that seen in the control groups.



**Figure 2.** Incidence of CNS problems in divers who never had experienced decompression problems and in controls, Data from<sup>9</sup>

However, as can be seen from Figure 3, there is a relationship between CNS symptoms and unreported decompression problems. Statistical analysis showed that unreported DCI was a significant risk factor for future central nervous problems. This does not mean that these people were seriously handicapped in any way. They were all working and all claimed that they felt healthy.

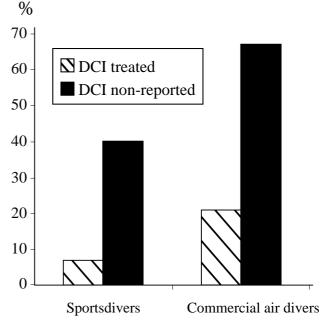


Figure 3. Percentage of divers with CNS problems who had experienced DCI, either treated or untreated. Data from<sup>9</sup>

The important message from this study is that it is important that all cases of decompression sickness are treated in some way, but perhaps also that even if some symptoms are ignored, the consequences are not major.

# Different types of DCI may have different treatment urgency.

The idea that there might by different types of decompression illness (sickness), with regard to delay to treatment, is presented in the US Navy Diving Manual,<sup>11</sup> where there are three categories of diving decompression emergencies.

Category A: Symptoms are severe, involve the inner ear, cardio-respiratory system and central nervous system; or are progressive or relapsing. Instituting treatment in these individuals should be considered an extreme emergency. An evaluation of the patient should not delay treatment or transport. These patients should preferably be treated immediately.

Category B: Urgent. The only severe symptom is pain. Symptoms are static, or have progressed slowly over the past few hours. Recompression is as soon as can be arranged, but there is time to conduct a full examination before beginning recompression. It is considered that you have time.

Category C: Symptoms are not severe and are not obvious without conducting a detailed examination. Any

organ system can be affected, but the patient is in no distress. Symptoms are static or progressing slowly over a period of hours. There is time for a complete workover before treatment is started. It is inappropriate to institute recompression without having done this.

This seems to confirm what is clinically well known; that there are many different categories of patients with decompression sickness. The general rule, however, is probably that early treatment will be beneficial in most cases. It is possible that oxygen at surface may be adequate for at least some of the cases in category C.

## **Remote Locations**

A remote location is a dive site that is at least four to six hours away from a chamber facility. Using this definition most dive sites can be considered remote.

In a place far away from a chamber, where it takes a long time to get help, the only medical advice you will get is through a telephone, if you are lucky. In such a situation we are talking about first aid, which would be rest, fluids and 100% oxygen if that is available. Treatment using pressure will be considered elsewhere.

## Oxygen

Oxygen at 100 kPa (1 bar) is safe. There is absolutely no data, as far as I know, that shows that oxygen at surface is not safe. Oxygen at pressure is clearly more effective for treating DCI. So what are the advantages of considering 1 bar oxygen as a definite treatment?

Studies have shown that oxygen has a positive effect on symptoms<sup>12</sup> and in many cases the divers presenting at the chamber have no symptoms. As far as I know, there are no studies where divers have been treated with oxygen alone, but anecdotal evidence tells us that a large number of divers have breathed oxygen on the surface for mild symptoms, without ever going on to chamber treatment. It must also be pointed out that there also is anecdotal evidence about divers who have breathed oxygen for some time, but then go on to develop symptoms once oxygen has stopped.

There is no doubt that as oxygen gets more and more common on dive sites, then a large number of individuals will use this as self treatment, whether we like it or not. However, if we insist that all divers breathing oxygen will have to go on to chamber treatment, this will probably mean that a lot of divers will not report their problems, as was clearly documented in the Norwegian study.<sup>9</sup> It is quite ironic that one of the reasons for not reporting problems is that we, as doctors, want perfection, we want to give them the best care possible. Unfortunately that also causes inconveniences for the diver. If he is a professional diver, treatment perhaps means the end of his career. For a sports diver, his holiday will be ruined as may be that of his friends. So there are strong incentives to suppress minor symptoms, or even major symptoms. Divers will deny their symptoms and will go diving the next day. Sometimes that works out alright. Sometimes he develops a problem which cannot be ignored. In our study, about 50% of the unreported symptoms could have originated in the central nervous system.

Unfortunately there are no statistics on how many divers use surface oxygen as definite treatment today. But I believe that if we said that, under certain circumstances, surface oxygen could be regarded as definite treatment, many divers with minor symptoms would use it. If we define surface oxygen as a treatment which, in some cases, could be a definitive treatment, more oxygen would be carried on dive boats. Surface oxygen would be accepted by divers, especially if they think that by using oxygen they could save themselves a long trip to the chamber complex.

Clearly there are disadvantages. There is no doubt that pressure and oxygen is the standard treatment. Some divers will get sub-optimal treatment. There may be a higher incidence of sequelae. Perhaps there will be more divers who have some pathological changes in their body and, maybe, as a result of that, more long term effects, like osteonecrosis and minor cerebral changes. I will maintain, however, that these changes probably are minor. The Norwegian study showed that even without treatment, the consequences of ignoring symptoms may in many cases not be too serious.<sup>9</sup> And even if there are some case histories where osteonecrosis has been observed after a single decompression incident, this must be extremely rare.

#### Effects of oxygen at 1 bar

Oxygen has a number of effects that are beneficial in treating DCI. One major effect is that it replaces the inert gas in the blood, thereby increasing the gradient for inert gas elimination. Thus bubbles will shrink more rapidly than they would without oxygen.

We have tested this in an experiment where we measured gas bubbles in the pulmonary artery.<sup>13</sup> We dived the pigs to 500 kPa (40 m or 5 bar) for 40 minutes and decompressed them over 2 minutes. That produced a very large number of gas bubbles in most animals. In fact, the amount of bubbles produced proved to be rapidly lethal without treatment. We started treatment at the time of maximum bubble formation after the dive, which was 20 to 30 minutes after surfacing. We used many different treatment protocols; when using oxygen at 100 kPa (1 bar) we continued oxygen breathing until bubbles disappeared, gave a further 30 minutes on oxygen, then switched to air. Figure 4 shows the results.

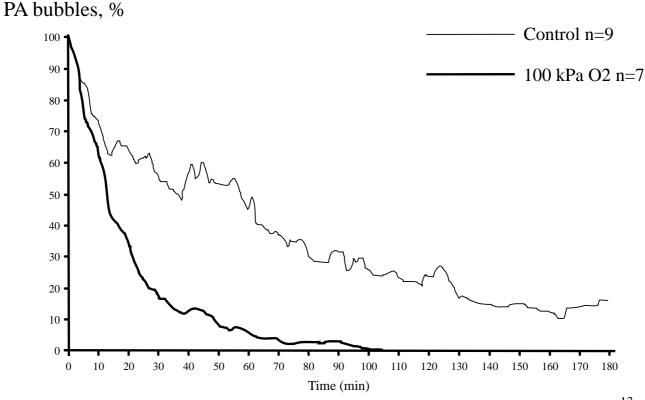
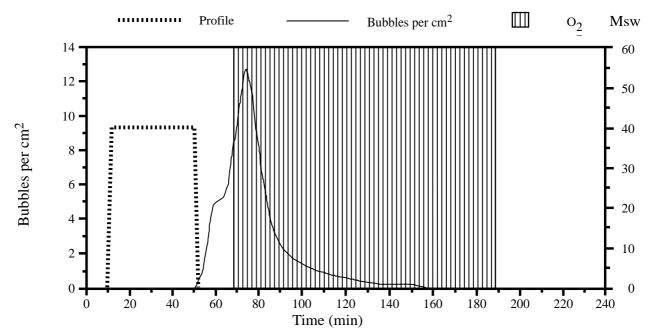


Figure 4. The effect of oxygen breathing on the elimination time of bubbles from the pulmonary artery. Data from<sup>13</sup>

Oxygen treatment at 100 kPa (1 bar), used immediately, is effective in removing bubbles. If no treatment is given, the extrapolation of the control curve will have the bubbles last for about eight hours, while oxygen made them disappear in an average of 74 minutes.<sup>13</sup>

When oxygen treatment was stopped, no further bubbles could be detected. This could indicate that the excess gas had been removed to a degree where no further bubbles could be formed, as can be seen from Fig 5.



**Figure 5.** The effect of oxygen breathing on the elimination of gas bubbles from the pulmonary artery. The maximum bubble numbers seen is similar to a Grade 4+ on the Spencer scale. Data from 13

These animals were kept alive for a week and we closely observed them clinically. None of them developed any sign of decompression illness. At autopsy, no changes could be detected in the brain, the spinal cord, the lungs or the pulmonary endothelium in any of the animals, indicating that at least in this experimental model, the treatment had been remarkably effective.

That means that this treatment, instituted early, and continued for only about 100 minutes, was enough to prevent animals, with a <u>lethal</u> amount of gas bubbles, from dying. Not only did it save them, but it saved them without any sequelae that we could detect with any modern method of histology. This is quite astonishing, but perhaps demonstrates that early treatment, even with 100 kPa (1 bar) oxygen, seems to be quite effective. That does not say that it is just as effective in humans. We do not know that. But at least it gives an indication that the use of only surface oxygen is not totally irresponsible and may be effective even as a definite treatment in some cases.

We assume that tissue oxygen increases as the oxygen tension in inspired air goes up. There is not very much data on measured oxygen tension in tissue, particularly at increased pressure.<sup>14</sup> But if we look at the tension of oxygen in the brain (Figure 6), we see that it is considerably lower than what would be expected from the increase in inspired oxygen, probably due to numerous regulatory mechanisms.

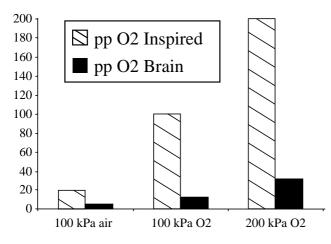
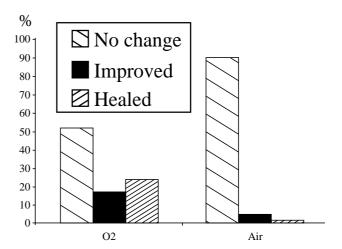


Figure 6. Oxygen tissue tension at various tensions of inspired oxygen. Data from.  $^{14}$ 

Another effect of oxygen, which is usually not considered, is the effect of increasing oxygen tension on shunt blood flow in the lung, blood that goes through the lung without have proper in contact with the alveoli, thus not being properly oxygenated. An increased shunt means that the oxygen tension in arterial blood will be lower than expected. We have demonstrated that the shunt is approximately 8% in a resting animal breathing air, increasing to something in the order of 15-20% when breathing 100% oxygen. Increasing oxygen tension further will further increase the shunt.<sup>15</sup> This means that the effect of increasing oxygen tension is smaller than would be expected from the changes in oxygen tension in the inspired air itself.

#### Results with oxygen at surface pressure

In Figure 7 you can see the results of treatment with oxygen from the DAN Europe database.<sup>16</sup> The study was published in 1996, and includes individuals who received oxygen before they got to a pressure chamber compared to those who did not get any treatment. Approximately 30% of the divers received some oxygen. On air, there was little change in symptoms, but in the oxygen treated group around 15% improved. But the interesting thing was that 20-25% "healed" during transport. The definition of healed was that the patients had no symptoms when they arrived at the chamber, it is therefore impossible to evaluate the result of the final recompression.



**Figure 7.** Clinical outcome of oxygen breathing prior to hyperbaric treatment. Data from DAN Europe 1994-95.<sup>16</sup>

#### What is an acceptable endpoint for treatment ?

An acceptable endpoint for treatment is not easy to define even when we talk about traditional treatment. Usually, treatment is continued until no more symptoms can be seen or there is no further improvement. However, there is virtually no data available on the long term effect of leaving minor symptoms.

What is the risk to the diver's future health if no residual symptoms can be detected following treatment with surface oxygen? If they say "I feel fine, I have no pain any more. I feel OK." after some hours of surface oxygen, I have a feeling that they probably will do alright. I do not know of any data to support the view that they will be at risk. I think that, in patients where no symptoms or signs were detectable after an oxygen treatment, no further improvement can be expected by subjecting them to pressure. Obviously, if symptoms reoccur, further treatment is indicated.

## When can surface oxygen be considered as definite treatment ?

The main indication will be divers who have minor symptoms that respond well to initial treatment with oxygen, where treatment is started immediately and where no recurrence of symptoms can be seen after treatment has stopped.

Other factors may also play a factor in making this decision. One reason would be if there is difficult or dangerous transport to the nearest chamber. There is no point risking the life of someone to get them to treatment. I do not think that is warranted unless you have someone who is dramatically sick or has serious symptoms. Even then they might improve considerably by having surface oxygen. I do not think transport time is significant, as studies indicate that if you do not treat immediately, then time to treatment will not seriously influence the outcome.

## Equipment for surface oxygen

If oxygen as a definite treatment is to be considered, then the development of further delivery systems is necessary. The main problem is probably to have an adequate supply of oxygen. If a valve with free flow is used, the oxygen percentage in the inspired air will probably rarely go above 65% and a lot of oxygen is used. A demand valve will reduce the amount of oxygen used considerably. Even better would be closed circuit rebreathers, where only about 50 litres of oxygen will be consumed per hour.

## Conclusions

Even in the absence of clinical data, I think there are enough other data to support the use of surface oxygen as a definitive treatment for DCI on a trial basis at remote locations. This can probably best be done by various training agencies and organisations like DAN. Initially this can be done by establishing a reporting routine for those that already practice this.

If we are going to encourage surface oxygen, we also have to consider training of the divers. In particular the people who run dive shops and are in charge of diving activities need to be able to recognise and evaluate symptoms better than they can do today. They have to be able to decide if further treatment is warranted. Today, many divers are not treated at all. Will they be better off with some surface oxygen?

### AUDIENCE PARTICIPATION

#### **Guy Williams**

To make things simple and to make a treatment regime easy to follow, would it not be a good idea to add yet another table. We have Tables 1, 2, 3, 4, 5, 6. Perhaps there should be a Table  $O_2$  which says go on 100% oxygen for 4-5 hours, then have an air break, then resume oxygen, air break, with perhaps written underneath, "This table should preferably be used under medical supervision or medical advice".

## Alf Brubakk

That would be an obvious thing. But at present I do not feel that what we are discussing is entirely acceptable. But I agree, we should have a procedure that tells people what to look for and what is acceptable. Some of the questions to be settled are: What is an acceptable endpoint? When does one say "enough is enough"? In what situation does one say "this is good enough"?

### **Cathy Meehan**

In Cairns we have a lot of tourists and the hyperbaric unit is in Townsville, which is 4 hours drive away. Sometimes we put affected tourists on 100% oxygen and use it as a diagnostic tool. If their symptoms do resolve, then we say it is likely to be decompression illness and they need to be recompressed. It would be nice to say they got better and so it is likely to be decompression illness, and that they do not need recompression. But if their symptoms have resolved, what do we say about flying?

#### Alf Brubakk

I think we should be even more conservative about flying, because, according to everything we think we know, this is a sub-optimal treatment. I think flying after an accident or surface oxygen should be restricted. One should wait longer than normal, perhaps double the time.

## References

- 1 Moon RE and Sheffield PJ. Eds. *Treatment of decompression sickness*. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1996
- 2 Snell E. Compressed Air Illness or so-called Caisson Disease. London: HK Lewis, 1896
- Eckenhoff RG, Olstad CS and Carrod G. Human doseresponse relationship for decompression and endogenous bubble formation. *JAppl Physiol* 1990; 69: 914-918
- 4 Denoble P, Vann RD and Dear GdeL. Describing decompression illness in recreational divers.

Undersea Hyper Med 1993; 20 (Suppl): 18

- 5 Kelleher PC, Francis TJR, Smith DJ and Hills RCP. INM diving accident database: analysis of cases reported in 1991 and 1992. Undersea Hyper Med 1993; 20 (Suppl): 18
- 6 Lehner CE, Adams WM, Dubielzig RR, Palta M and Lanphier EH. Dysbaric osteonecrosis in divers and caisson workers: an animal model. *Clin Orthop* 1997; 344: 330-332
- 7 Jones JP, Salbador GW, Lopez F, Ramirez S and Doty SB. High-risk diving and dysbaric osteonecrosis (Panel on diving physiology). In *Proceedings 14th Meeting US-Japan Cooperative Program in Natural Resources (UJNR)*. Smith EN. Ed. Silver Spring, Maryland: National Oceanic Atmospheric Administration (NOAA), 1998: 77-88
- 8 Blick G. Notes on diver's paralysis. *Brit Med J* 1909; ii: 1796-1798
- 9 Brubakk AO, Bolstad G and Jacobsen G. Helseeffekter av lufdykking. Yrkes og sportsdykkere. STF23A93053, Trondheim: SINTEF Unimed, 1993
- Ydreborg B, Bryngelson Y-L and Gustafsson C. Referansdata til ørebroformularen. 6. Ørebro, Sweden: Metodicum, 1988
- 11 US Navy Diving Manual. Best Publishing Co. 1980
- 12 Marroni A. Recreational diving to-day: risk evaluation and problem management. In EUBS Proceedings 1994. Cimcit M. Ed. Istanbul: EUBS, 1994; 121-131
- 13 Koteng S, Øernhagen H and Brubakk AO. Pressure and oxygen reduce elimination time for bubbles after diving. In *Diving and Hyperbaric Medicine Proceedings of XXIV EUBS*. Linnartson D. Ed. Stockholm, Sweden: EUBS, 1998
- 14 Camporesi E, Mascia M and Thom S. Physiological principles of hyperbaric oxygenation. In *Handbook* on Hyperbaric Medicine. Orriani G, Marroni A and Wattel F. Eds. Berlin: Springer, 1996: 35-58
- 15 Koteng S, Koteng Ø, Flook V, and Brubakk AO. Venous air embolism in swine: The effect of different oxygen partial pressures in the breathing gas on the shunt fraction. In *Proceedings XIX Ann Meet EUBS on diving and hyperbaric medicine*. Reinertsen, Brubakk AO and G. Bolstad. Eds. Trondheim: EUBS. 1993, 287-291
- 16 Marroni A. Recreational diving accidents in Europe. DAN Europe report 1994-1995. In *Proceedings of the International Joint Meeting on Hyperbaric and Underwater Medicine*. Marroni A, Oriani G and Wattel F. Eds. Milano: EUBS, 1996, 259-265

Professor Alf O Brubakk was a guest speaker at the 1999 SPUMS Annual Scientific Meeting. His address is Department of Physiology and Biomedical Engineering, Medical Faculty, Norwegian University of Science and Technology, Trondheim, Norway or Sandgt 1, N-7012 Trondheim, Norway. Telephone + 47-7359-8904 Fax + 47-7359-1005. E-mail <alf.o.brubakk@medisin.ntnu.no>

## RECOMPRESSION TREATMENT SHOULD ONLY BE ADMINISTERED IN A HOSPITAL-BASED FACILITY

Richard E Moon

#### **Key Words**

Decompression illness, hyperbaric facilities, hyperbaric oxygen, treatment

### Introduction

The five components of appropriate treatment of a diving casualty with decompression illness (DCI) are:

- 1 Availability of a skilled practitioner to assess the patient and make the diagnosis;
- 2 ability to administer initial therapy such as maintaining an airway with adequate ventilation and fluid resuscitation;
- 3 a treatment chamber in which 100% oxygen can be administered at increased ambient pressure;
- 4 appropriate procedures (i.e. treatment tables);
- 5 ability to assess and monitor the patient during treatment.

If all five components are available at the site of the diving accident then, since delay in treatment may involve clinical deterioration, immediate treatment is preferred. The present discussion, however, is in the context of hospital-based treatment where all components are available compared with on-site treatment, in which one or more components are not available.

Assessment requires ideally a physician but at least a person who has had specific training in assessment, treatment and monitoring of diving casualties. In addition to the trained individual, equipment is necessary. A stethoscope, sphygmomanometer, percussion hammer, otoscope, urinary catheter, equipment for administering intravenous fluids and for performing a tube thoracostomy. Ideally one would want a portable X-ray unit.

Therapeutic procedures include treatment tables that have been proven effective in the treatment of decompression illness. The US Navy tables 5 and 6, and their equivalents, have a long track record of efficacy. While shorter treatment tables designed for use in monoplace hyperbaric chambers have efficacy in treating mild or moderate bends, the available data suggest they are less effective in treating severe bends.<sup>1</sup>

Monitoring includes verbal assessment and objective measurement of the progress of treatment. In addition, blood pressure, heart rate and respirations must also be measured particularly in the critically ill individual: