REVIEW ARTICLE

Guiding principles in choosing a therapeutic table for DCI hyperbaric therapy

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ABSTRACT

Hyperbaric therapy is the basis of treatment for pervasive development disorders. For this reason, the choice of the right therapeutic table for each case is critical. Above all, the delay in recompression time with respect to the first symptoms and to the severity of the case must be considered. In our experience, the use of low-pressure oxygen tables resolves almost all cases if recompression takes place within a short time. When recompression is possible almost immediately, the mechanical effect of reduction on bubble volume due to pressure is of remarkable importance. In these cases, high-pressure tables can be considered. These tables can also be used in severe spinal-cord decompression sickness. The preferred breathing mixture is still disputed. Heliox seems to be favored because it causes fewer problems during the recompression of divers, and above all, because nitrox can cause narcosis and contributes nitrogen. Saturation treatment should be avoided or at least used only in special cases. In cases of arterial gas embolism cerebral injury, it is recommended to start with an initial 6 ATA recompression only if the time between symptom onset and the beginning of recompression is less than a few hours.

Key words: Hyperbaric oxygenation - Decompression - Tables.

In the therapeutic treatment of decompression illness (DCI), hyperbaric therapy has a prominent role; therefore, the choice of the correct therapeutic table for each case is very important.

Several recompression schedules (commonly referred to as "treatment tables") have been empirically developed, based on statistics or tests in healthy individuals (*e.g.*, Van Der Aue Tables of 1945). The initial experience of recompression with oxygen tables equivalent to 18 m deep had so much success that it became the basis of modern recompression therapy.[1] At 2.8 ATA pressure, 100% oxygen can be breathed with a low probability of oxygen toxicity. Thus, both treated divers and the staff who attend to them in the chamber can be quickly decompressed. The most widely used of these tables are USN5 and USN6 or a similar equivalent. These tables have algorithms that allow breathing oxygen at 2.8 ATA and at 1.9 ATA. The USN6 table can be extended to provide additional breathing cycles at both depths. An extreme example of this is the Catalina table, in which up to eight cycles of oxygen can be administered at 18 m, and 18 cycles can be administered at 9 m.

Many cases of DCI taking place on the surface can be compressed with satisfactory results using the equivalent 18 m deep pressure while the patient breathes 100% oxygen, using one of the principal treatment schemes available. U.S. Navy guidelines advise the use of table 5 only for mild DCI, and symptoms must resolve within 10 min after reaching 18 m. For all other situations, a different table must be used, usually USN6.

Most practitioners prefer to use the USN6 table for all instances of DCI. Most DCI cases in divers originating at the surface, therefore, can be managed using USN5 tables, USN6 tables or USN6 tables with extensions.[2] Oxygen cycles are administered at 18 m until the symptoms are relieved or until the patient is clinically stable, for a maximum time allowed by guidelines of the particular table. A single treatment is often sufficient to resolve symptoms completely; however, in the case of residual clinical signs and symptoms, supplementary treatments must be administered. An analysis report by the Divers Alert Network (DAN) suggested that this result was probably achieved with no more than seven treatments.[3] In divers with severe neurological DCI, however, a greater number of treatments may be required to reach maximum results on the treatment plateau. Multiple recompressions for isolated musculoskeletal (pain only) DCI are usually not recommended.

More severe cases can be managed using a deeper option, e.g., the Comex table 30 or 50, in which pressurization is 30 or 50 m depth, respectively, and breathed oxygen is mixed with helium or nitrogen. Animal studies [4] and a published case review [5] provide little evidence in favor of treatment at pressures >2.8 ATA. After bubbles have induced secondary pathophysiologic changes, reduction in bubble size is just one part of a complex therapy, which might include hyperoxygenation, rehydration and pharmacotherapy.

If the chamber complex and staff are capable of supporting saturation therapy, it should only be used when the severity of the symptoms are such that marked residual impairment or loss of life may result if decompression from 60 feet is undertaken.[6] These should not be used in the presence of numbness, tingling, decreased sensation of touch, hypesthesia, limb weakness or bladder problems without limb paralysis. It is not to be used in stabilized neurological conditions or residual painful symptoms.[6, 7] In this treatment, the chamber remains under pressure until clinical stabilization is reached, usually 12 h or longer. When the saturation treatment begins, decompression must be performed only when a large improvement is achieved. Other saturation depths have been proposed and may be compulsory for individuals decompressing from either deep bounce dives or saturation dives.[8] In such situations, the environmental pressure and the treatment gases pressure must be mixed so that the partial pressure of oxygen does not rise above the limits of 0.5 ATA (usually 0.35-4 ATA) and 2.8 ATA, respectively.

Immediate recompression has the greatest success, and delays in treatment tend to worsen the prognosis.[9] The effect of delays on the long-term outcome in individual cases, however, is unpredictable. Although there is partial agreement in the international scientific community about the effect of delays, it is still a matter of debate. The points of contention are the pressure and the breathing gas. Pressure reduces bubble volume by a simple mechanical effect. According to Bergmann, the maximum bubble reduction is for pressure from 4 to 6 ATA. Beyond this depth, there is no further benefit.

Generally, the therapeutic pressures used do not allow the disappearance of intravascular gaseous sleeves formed by bubble confluence, but they cause fragmentation and re-direct the gaseous embolus into the circulation, releasing collateral vessels. Recompression acts on the primary disease cause, but not on its secondary effects. Furthermore, the recompression determines new inert gas tissue saturation and requires appropriate decompression.[10] In the past, the optimal depth was considered 3 ATA. On a USN initiative and as a consequence of animal testing conducted by Leitch et al., however, it was reduced to 18 m.[11]

The 18-m treatment may be considered too deep or too shallow. The initial studies of Behnke et al. seemed to indicate that 10 m was adequate for all cases.[12] On the other hand, the human trials conducted by Goodman et al. indicated that a shallow treatment depth was associated with a high treatment failure rate.[13] Wilson et al. reported 65 DCI cases treated on 100% oxygen at either 18

m or 14 m for three 30-min O_2 breathing periods. Only 16% of the patients treated at 18 m had recurrent symptoms, while 40% of the patient treated with the 14 m table had recurrences.[14] Depths >18 m appear to be beneficial, provided there are no hardware restrictions. Anecdotal evidence and isolated case reports describe the benefits of increased pressure, but there are no reported trials large enough to draw firm conclusions.[15] Some authors have also had experiences where there was rapid resolution of symptoms soon after compression to 50 m, which showed little or no resolution after several hours at 18 m. According to several authors, many cases of DCI resolve during compression to 18 m, especially if treated rapidly. The available evidence suggests that treatment at depths shallower than 18 m runs the risk of treatment failure and that treatments deeper than 18 m offer no particular benefit in the majority of cases.[16]

For spinal cord DCI treatment, we must consider that hyperbaric oxygen is thought to be able to improve reperfusion injury (symptom deterioration during compression), while it also has beneficial effects in cases of tissue ischemia or inflammatory phenomena. The effect of the therapy, therefore, results from an optimal relationship between the positive and negative effects of the hyperbaric oxygen. This requires optimization to obtain the best results. The initial studies of Leitch *et al.* seemed to show that the best results in cases of spinal cord DCI were obtained with a PiO_2 of 2 ATA. This suggested a reduction of the pressure in treatment tables, but it must be considered that in these experiments therapy took place 15 min after the onset of symptoms, when reperfusion injury was not so evident as in the usual situation when intervals are larger.[17] Further studies conducted by Leitch *et al.* indicated that the optimal oxygen partial pressure for treatment ranged from 2 to 2.5 ATA.[18] In another study, Leitch *et al.* showed that the beneficial effects of increasing PiO_2 stopped at 2.8 ATA. Human data suggest, however, that lowering the PiO_2 by breathing 100% O_2 at a lower treatment pressure leads to an increased incidence of symptom recurrence. Therefore, it appears that a PiO_2 of 2.8 ATA may be the optimal level.
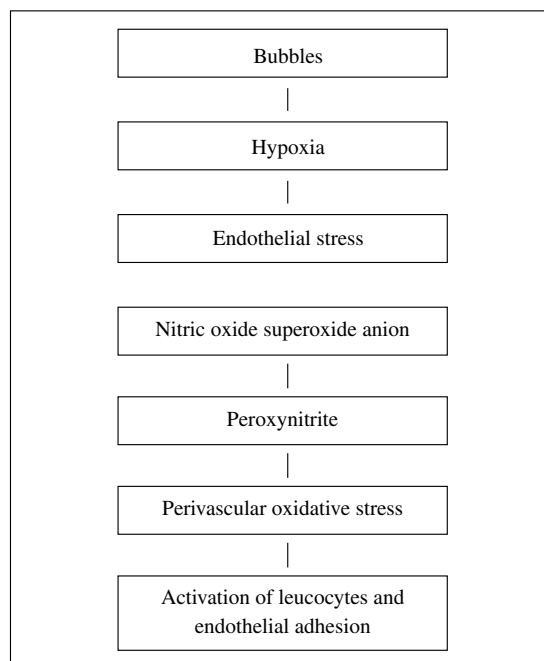
The use of oxygen as breathing gas is preferable

Figure 1.—Mechanism of activation of leucocytes and endothelial adhesion.

because it is metabolized by tissues and, therefore, does not accumulate like an inert gas does. This results in a reduction of the total gas pressure in the tissues surrounding the bubble, enhancing the rate of diffusion of inert gas from the bubble into the surrounding tissue.

Hyperbaric oxygen administration has other potential benefits such as oxygenation of ischemic tissue, reduction of central nervous system edema, and possibly inhibition of endothelial leukocyte accumulation.[19] In fact, the presence of gaseous bubbles in the venous vessels blocks the flow and induces hypoxia. This hypoxia causes endothelial stress followed by the release of nitric oxide (NO), which reacts with superoxide anion to form peroxynitrite. This, in turn, provokes oxidative perivascular stress and leads to the activation of leukocytes and their adhesion to the endothelium (Figure 1).

The adhesion of endothelial leukocytes at inflammation sites takes place in a multiphase process in which there is initially unsteady adhesion mediated by selectins, followed by a subsequent phase in which there is stable adhesion due to the activation of leukocytic β_2-integrins. These

integrins are induced by pro-inflammatory mediators that are produced by the endothelium or by inflamed surrounding tissues. The endothelial β_2-integrin ligands are represented by glycoprotein molecules, similar to immunoglobulin G, known as intercellular adhesion molecules 1 and 2 (ICAM-1 and ICAM 2).

When leukocytes adhere steadily to the endothelium, endothelin, xanthine-dehydrogenase, xanthine-oxygenase, and oxidants (O_2^-, H_2O_2, $ONOO^-$) are released and cause subsequent membrane lipoperoxidation and probable organ injury. At this point, the process can be self-maintained without the presence of bubbles. Thus, the action of the hyperbaric oxygen results in significant inhibition of β_2-integrins and interference with leukocyte adhesion, preventing the occurrence of subsequent processes that lead to cellular injury.

Oxygen can be administered safely in a dry hyperbaric chamber for a certain pressure and time, above which there is a significant oxygen toxicity risk to the central nervous system and lungs. It is widely accepted that an exposure >2.8 ATA should be avoided.

Oxygen treatment tables (*e.g.*, USN tables 5 and 6) were designed to allow 100% oxygen breathing at the highest practical ambient pressure, while avoiding oxygen toxicity.

According to several authors, at pressure >2.8 ATA, hyperoxygenated breathing mixtures must be used. The addition of an inert gas as a diluent allows the maintenance of a high partial oxygen pressure without the risk of oxygen toxicity. Breathing mixtures are composed of oxygen combined with helium or nitrogen. Recompression with such a mixture causes new inert gas saturation, leading to a longer and more difficult decompression. Nitrox (oxygen and nitrogen) is still largely used, but it is criticized because recompression with a breathing mixture including nitrogen contributes inert gas, which could theoretically increase the development of bubbles and cause the onset of new symptoms during subsequent decompression. Therefore, it is now more evident than ever that recompression with air should be avoided. The use of heliox (oxygen and helium), is characterized by greater diffusivity and is preferable. According to some authors, under some circumstances it may be preferable to use helium as the inert gas diluent, rather than nitrogen (particularly deeper than 50 m).[20]

Some diving physicians have suggested that heliox may be superior to oxygen as a breathing gas. The work of Hyldegaard *et al.* provided evidence that at 1 ATA, heliox breathing could result in faster bubble resolution than air or oxygen breathing.[21] There is some evidence that in divers who have breathed heliox and have gone through decompression all the way to the surface using this gas, nitrogen breathing can exacerbate the symptoms of DCI. Therefore, it seems logical that if a diver develops symptoms after surfacing from such a dive, he should be recompressed using either oxygen or heliox.[22]

The use of an He-O_2 breathing mixture instead of O_2 alone for the treatment of neurological decompression sickness has several theoretical advantages that have been confirmed by recent animal experiments. Although clinical reports on the use of He-O_2 in DCI are scarce and mostly anecdotal, studies by Shupak *et al.* have demonstrated a significant advantage of He-O_2 recompression over USN oxygen tables for severe neurologic DCI.[23]

According to Bergmann, isobaric counterdiffusion theory coined by Lambertsen *et al.* may point to the use of a heliox mixture (50/50) for high-pressure recompression. Indeed, when air bubbles are present inside a tissue with considerable lipid content (*e.g.*, the white matter of nervous tissue), helium diffuses into the bubble slower than nitrogen can diffuse in the opposite direction. This leads to a decrease in bubble size and facilitates nitrogen elimination.[24]

Hyldegaard *et al.* showed that 100% O_2 breathing produced a transient increase in bubble size before beginning to decrease.[25]

Even though neurological DCI symptoms may be due to extensive bilateral brain damage, in most cases they are due to spinal cord injuries only. It has been suggested that whenever the increased pressure caused by bubble accumulation in the white matter of the spinal cord exceeds the feeding arteriolar closing pressure, ischemia will develop in the spinal cord. Even temporary growth in bubble size might cause the critical increase in tissue pressure that is required for arteriolar occlusion, aggravating spinal cord ischemia and neurologic dys-

function. According to some authors, delayed elimination of the inert gas from the tissue and the presence of gas bubbles long after surfacing from a dive, even after recompression, might explain the clinical response to late heliox therapy that was observed in cases where treatment was delayed up to 7 days after the onset of DCI.

The theoretical benefit of heliox recompression treatment when the air bubbles are in a tissue with considerable lipid content (*e.g.*, the white matter of nervous tissue) might be attributable to the greater outflow of nitrogen under heliox than when breathing oxygen alone. When gas exchange is limited by tissue perfusion, the lower solubility of helium in blood and lipids compared with that of both nitrogen and oxygen would facilitate quicker bubble elimination. In cases of diffusion limitation, hyperbaric heliox breathing would still have an advantage in fatty tissues in which gas exchange is determined by the product of the solubility and diffusion coefficients; this product is lower for helium than it is for nitrogen and oxygen.

When gas exchange is diffusion-limited in aqueous tissue, however, bubbles would be expected to grow while breathing heliox, since the product of the solubility and diffusion coefficients in water is greater for helium than it is for nitrogen. Therefore, heliox might be not a good alternative to air-oxygen recompression when inner ear or pulmonary involvement in DCI is being considered.[23]

On the other hand, several animal studies have shown that there is no advantage, and perhaps even a disadvantage, to treating spinal cord DCI resulting from air dives with He-O$_2$ *vs* air.[26] A study using a Guinea pig model of severe DCI suggested that heliox treatment had no benefit.[27]

According to Bornmann, it is likely that the use of heliox has been recommended not because it produces beneficial effects, but simply because it seems have an advantage in decompression or because it reduces narcosis at maximum treatment pressures.[28]

Consensus Conferences guidelines

The Consensus Conferences recommended the following guidelines for therapeutic recompression in DCI:

—decompression accidents should receive the benefit of specialized treatment in dedicated centers only. A specialized center is considered to be a hospital-based recompression facility with permanent and adequately trained medical and paramedical staff;

—a decompression accident is a true medical emergency that should be treated with recompression as soon as possible; therefore, the victim should be immediately directed to the closest specialized center;

—in-water recompression should never be performed as the initial recompression;

—minor decompression accidents (pain only) can be treated with oxygen recompression tables at an 18 m depth maximum. (This is based on experience and the good results observed in commercial diving);

—regarding more serious decompression accidents (neurological and vestibular accidents), there are presently two acceptable protocols:

a) oxygen recompression tables at 2.8 ATA (with possible extensions);

b) hyperoxygenated breathing mixtures at 4 ATA.

No specific recommendations can be made regarding the optimal PiO$_2$ (maximum 2.8 ATA), and the preferred choice of diluent inert gas cannot be recommended based on scientific evidence at this stage.

Familiarity, availability and experience may affect decisions, but under no circumstances should the lack of availability of gas mixtures preclude or delay treatment by means of "low pressure oxygen tables".

—In case of cerebral arterial gas embolism (AGE), compression to 6 ATA should be performed using mixed gas (compressed air is to be absolutely avoided) when the delay to recompression is no more than a few hours.

—Actually, there are no data guiding:

a) the diluent inert gas or maximum PiO$_2$;

b) the maximum delay within which this therapy is still considered appropriate;

—in case of severe, persistent clinical signs during the initial recompression, the continuation of treatment with a therapeutic saturation table may be useful. This is in reference to AGE only.
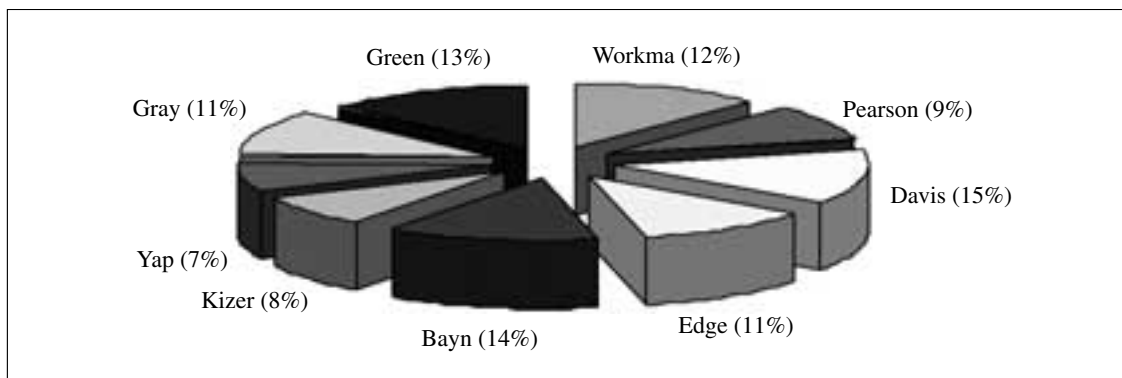
Figure 2.—Percentage of complete recovery with USN tables. USN tables complete success percentage.

Expected outcome of the clinical scenario

The results of early treatment of DCI (within a time window not more than 4 h from the onset of symptoms) following conventional practices are usually very good. Some authors have reported 96% complete relief of symptoms. DAN (year 2000) reported symptom resolution in 75% of cases after treatment; furthermore, an additional 20% of cases showed symptom improvement after the completion of a treatment cycle when the complete relief was not obtained after the first recompression.[29] The tables used were not specified, but in the majority of cases, short oxygen tables have been applied.

Given the importance of immediate administration of normobaric oxygen and fluids, the main difference in expected outcome is not a consequence of the therapeutic table used but is primarily due to others factors, *e.g.*, the length of the delay in beginning recompression and the initial symptom severity.

Some significant predictors of poor outcome are:[30] amateur *vs* professional divers, severe symptoms, worsening of symptoms, increased age, and recurring symptoms after treatment.

There are some scoring systems to predict outcome.[31] One of these was devised by Mitchell and values every symptom according to its specificity for DCI, its natural development when untreated, its capacity to render unfit, and its correlation with others symptoms. For the above-stated reason, hyperbaric treatment may give various results in the treatment of DCI. The success rate, excluding the
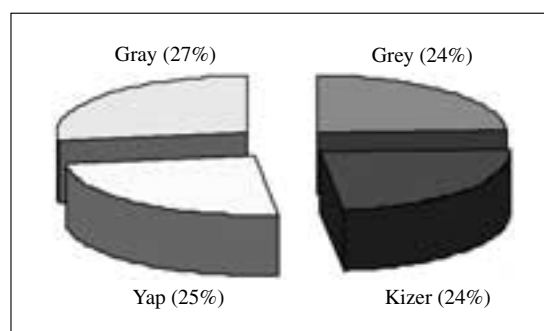

Figure 3.—Percentage of clinical improvement with USN tables. USN tables partial success percentage.

predictors of poor outcome mentioned above, depends on the severity of the early symptoms and the delay in recompression time. Typically, good results can be expected, provided that treatment begins no later than a few hours (generally 4 h) from symptom arousal. These results may be obtained by using both oxygen recompression tables at 2.8 ATA (PiO_2 100%) (with possible extensions) and hyperoxygenated mixtures at 4 ATA.

Thus, there is no advantage in the majority of cases to utilizing higher pressures or different breathing mixtures.

Only in the case of AGE cerebral injury can treatment begin with an initial 6 ATA recompression using mixed gas, given that the time between the onset of symptoms and recompression is less than a few hours. In exceptional circumstances, it may be necessary to use saturation tables, but only
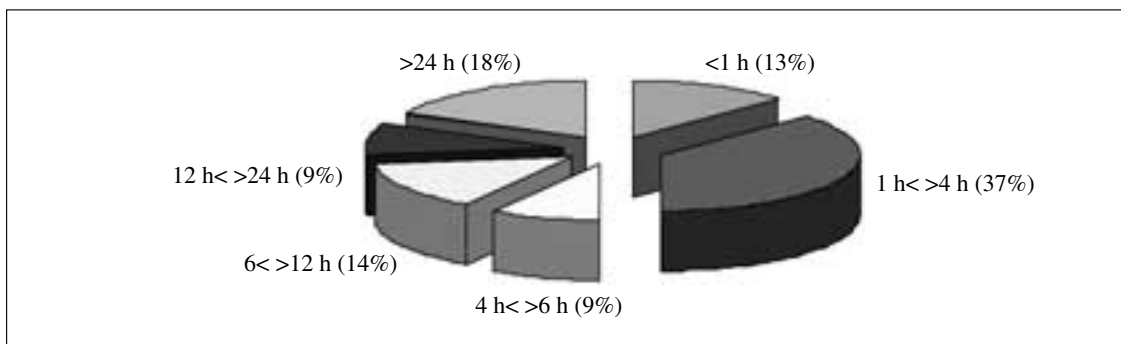
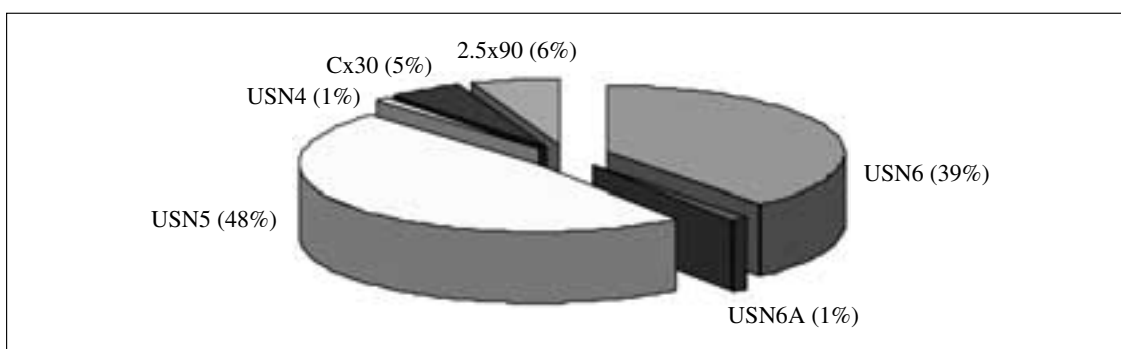Figure 4.—Italian cases: time from symptoms onset to recompression.

Figure 5.—Italian cases: recompression treatment.

in the case of a patient with a truly life threatening condition that is becoming worse during recompression at 18 m.

Case evaluation

Upon analyzing cases from the DAN for 1996 with regard to recompression therapy for DCI (1331 cases), we can evaluate the frequency of usage of various tables. It is evident that the most used tables were the low-pressure (2.8 ATA) short tables with 100% oxygen breathing gas. The USN6 was applied in more than 70% of cases among the 1331 treatments considered.

From analysis of Figures 2 and 3, it is possible to estimate the outcomes that some authors have achieved using the USN oxygen treatment tables. A total of 1614 treatments have been carried out, and there were positive outcomes in the vast majority of cases. In particular, there was complete success, on average, in 79% of treated cases (minimum 50% and maximum 98%), and substantial success, on average, in 86% (minimum 83% and maximum 94%). Figures 4-7 show a comparison of therapeutic methods and the outcomes achieved on an international scale with outcomes based on the analysis of 129 DCI cases treated in hyperbaric centers scattered throughout Italy in 2004.

From the previous graphics, we report the following findings. The reporting of DCI symptoms in a hospital often takes place in autonomous manner, and consequently, there is a substantial delay to treatment. Only 50% of cases are recompressed within 4 h of the accident, despite the fact that in Italy, the diving sites are not far from hyperbaric centers. With regard to the recompression treatment used, short oxygen tables have been used in most of cases (87%) with the following percentages: USN5 (48%) and USN6 (39%).

The results obtained after the first treatment, according to the Della Torre classification, have
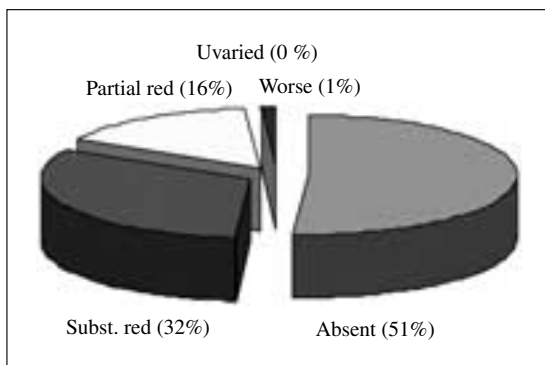
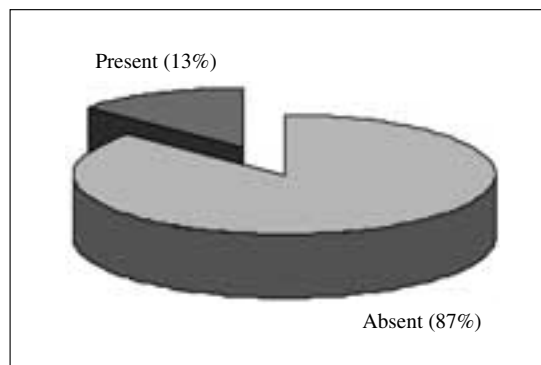Figure 6.—Italian cases: symptoms after the first treatment.

Figure 7.—Italian cases: symptoms after one week.

been the following: 0) symptoms absent: 51%; 1) symptoms substantially reduced: 32%; 2) symptoms partially reduced: 16%; 3) symptoms unchanged: 0%; 4) symptoms worse: 1%.

Asymptomatic patients after one week were the majority of cases (87%), while others still had over all paresthesia, vestibular syndromes and weakness. From these data, we can conclude that in Italian hyperbaric centers and according to international experience, it is preferable to use short 2.8 ATA oxygen tables in DCI recompression therapy to obtain good results in the majority of treated cases.

We might recommend greater sensitivity of divers (and some practitioners) to avoid the habit of undervaluing the first symptoms so that the injured patients comes to the attention of a hyperbaric practitioner and begins recompression treatment as soon as possible. Statistical analysis is reported in Table I.

Discussion

There are several parameters to consider in the choice of a therapeutic table. Among these, the delay in recompression time from the onset of symptoms is critical.

It is established that although recompression should never be performed in water, therapeutic recompression must be initiated as soon as possible. When recompression is possible almost immediately, the mechanical effect of bubble volume reduction due to pressure is of great importance. In these cases, high-pressure tables (4 ATA) can be considered.

In a hyperbaric medicine center, later accidents after the bubbles have initiated secondary pathophysiological processes require that bubble volume reduction be only a component of a multifactorial therapy that includes rehydration, pharmacological therapy, and above all, hyperoxygenation. Therefore, the role of oxygen assumes a greater importance and is essential to avoid iatrogenic damage due to recompression that is too deep. We prefer the use of low pressure hyperoxygenated tables.

The choice of a therapeutic table also greatly depends on illness severity. Minor decompression accidents (pain only) can be treated with oxygen recompression tables: USN5 and eventually USN6 if the symptoms do not resolve completely within 10 min of reaching pressure. With initial recompressive treatment for more serious decompression accidents (*e.g.*, neurologic, cerebral, medullary and vestibular accidents), there are no scientific data to reach to a definitive conclusion. At present, there are two acceptable protocols: oxygen recompression tables at 2.8 ATA (with possible extensions) or hyperoxygenated breathing mixtures (50:50 Heliox or Nitrox) at 4 ATA. No specific recommendations can be made regarding the optimal PiO$_2$ (maximum 2.8 ATA) and preferred choice of diluent inert gas based on scientific evidence at this stage.

It has been acknowledged, however, that recompression with a breathing mixture containing nitrogen would result in additional uptake of inert gas, which theoretically could enhance bubble growth and result in new symptoms during subsequent

TABLE I.—*Statistical analysis of results obtained with USN treatment Table 5 and 6 (Casuistry SIMSI 2004).*

Variable	Table V N.=71 (53%)	Table IV	P	OR	Cl 95%
Age (y)	37.4±8.6	38±9.4	0.69		
Sex [M (%)]	62 (87%)	48 (76.2 %)	0.07	0.47	0.19-1.2
Total dive time (min)	34.9±23.0	36.9±27.3	0.65		
Depth of dive (m)	38.0±18.2	44.5±24.6	0.1		
Expert [N. (%)]	47 (66%)	40 (s63.5%)	0.44	0.89	0.44-1.8
Previous DCI [N. (%)]	17 (23.9%)	12 (19.7%)	0.32	0.75	0.5-1.7
Repetitive 8 [N. (%)]	20 (28.2%)	15 (23.8%)	0.35	0.8	0.5-1.7
AIR [N. (%)]	69 (97.2%)	37 (90.5%)	0.1	0.3	0.12-1.4
Error of decompression [N. (%)]	41 (57.7%)	21 (33.3%)	0.004	2.7	1.35-5.5
Symptomatic at onset [N. (%)]	59 (83.1%)	60 (95.2%)	0.02	4.07	1.1-15.2
General symptoms [N. (%)]	7 (9.9%)	11 (7.5%)	0.15	1.9	0.7-5.3
Symptoms osteo-muscolar [N. (%)]	30 (42.3%)	33 (52.4%)	0.16	1.5	0.8-3
Symptoms neurological [n. (%)]	29 (40.8%)	45 (71.8%)	<0.001	3.6	1.8-7.5
Symptoms ENT [N. (%)]	0 (0%)	1 (1.6%)	0.47		
Therapy during transfer [N. (%)]	30 (42.3%)	33 (52.4%)	0.16	1.5	0.8-3
Antiplatelet drugs [N. (%)]	5 (7%)	9 (14.3%)	0.14	2.2	0.7-6.9
Steroids [N. (%)]	6 (8.5%)	22 (34.9%)	<0.001	5.8	2.1-15.5
Analgesic drugs [N. (%)]	6 (8.5%)	2 (3.2%)	0.18	0.4	0.07-1.8
Symptoms at treatment start [N. (%)]	49 (69%)	62 (98.4%)	<0.001	27.8	3.7-213
General [N. (%)]	1 (1.4%)	5 (7.9%)	0.08	6	0.7-53
Osteo-muscular [N. (%)]	29 (40.8%)	21 (33.3%)	0.24	0.7	0.36-1.5
Neurological [N. (%)]	25 (35.2%)	52 (82.5%)	<0.001	8.7	3.9-19.6
ENT [N. (%)]	0 (0%)	1 (1.6%)	0.47		
Outcome					
Symptoms cured [N. (%)]	28 (53.1%)	21 (33.9%)	0.033	2.2	1-4.76
Symptoms reduced [N. (%)]	20 (40.8%)	37 (59.7%)	0.037	2.15	1-4.6
Symptoms unchanged/increased [N. (%)]	3 (6.1%)	3 (4.8%)	0.54	0.78	0.15-4
Symptoms after 1 week [N. (%)]	3 (6.1%)	13 (21%)	0.023	4.1	1-15.2

DCI: decompression illness; OR: odds ratio; CI: confidence interval.

decompression. Therefore, the use of helium is preferable. Evidence in the literature has shown that heliox seems to have an advantage in cases of neurological DCI, but it does not seem to be effective in vestibular or pulmonary injuries. A prerequisite for saturation treatment is the availability of appropriate structures, stock of gas, trained personnel and adequate facilities. In addiction, this form of treatment should be reserved for patients for whom the potential improvement in outcome warrants the increased complexity of the treatment. The significantly higher cost and possible interference with other necessary care (*e.g.* physical therapy), as well as treatment of other patients, must also be considered.

In cases of cerebral AGE, compression to 6 ATA is proposed using mixed gas (and no compressed air) when the delay to recompression is no more than a few hours. There are no data guiding the maximum PiO$_2$, the breathing mixture or the maximum delay within which this therapy is still considered appropriate.

Conclusions

As recommended in previous workshops and in the Consensus Conferences, definitive treatment of diving related DCI is compression and administration of breathing gas with elevated partial pressure of oxygen. It is very important to minimize the delay in pressurization and oxygen administration. Therefore, divers should be better educated to reduce the delay in recognizing the first symptoms of DCI; 100% oxygen administration should begin during transportation; and there should be definitive treatment with recompression. A wide variety of initial hyperbaric regimens have been described. Current treatment

options differ in the level of treatment pressure, time under pressure, partial pressure of oxygen, and the diluent gas. At present, the apparent differences in protocol and procedures between different hyperbaric centers may be the result of local circumstances (*e.g.*, the availability of trained staff), the local style of diving (*e.g.*, deep occasional or shallow multi-day) leading to different presentations, and perhaps most importantly, the delay before recompression (delays >12 h in some locations compared to some naval and commercial diving sites that are required to have a chamber on the site). There are no human outcome data obtained in prospective, randomized studies comparing the various regiments; however, the following principles are agreed upon:

— complete resolution is most likely to result from early hyperbaric treatment;

— since their introduction in 1965, the US Navy oxygen treatment tables with initial recompression to 18 m have been the most widely used and studied recompression procedures for DCI treatment beginning at the surface. Other procedures such as those used by the Royal Navy and Comex follow the same general principles of pressure and oxygen breathing. A review of the effectiveness of USN oxygen treatment tables shows a high degree of success in resolving symptoms if the delay to treatment is not excessive;

— saturation treatment can be only applied in the case of a critically ill patient with a long delay to recompression therapy or if there is deterioration of symptoms during the 18 m compression. It remains an extreme and impractical option, and there is no scientific evidence of its real utility;

— the use of tables with a more shallow or deeper initial treatment depth should be reserved for facilities and personnel with the experience, expertise and hardware necessary to deal with unexpected responses. Of note, the chamber to be working continuously for at least 48 h;

— while there is an inverse relationship between the delay to treatment and complete resolution of symptoms, the data currently available have not established a maximum time (hours or days) after which recompression is ineffective. In the absence of an altitude exposure, the onset of symptoms >24 h after a dive is unlikely to be caused by DCI.

— although administration of surface oxygen often resolves symptoms, they frequently recur after cessation of oxygen breathing. For diving related DCI, surface oxygen breathing is not a substitute for hyperbaric treatment;

— under some circumstances, when recompression is required to depths at which 100% oxygen cannot safely be administered, it may be preferable to use helium as the inert gas diluent rather than nitrogen. There is no evidence to specify exactly the kind of hyperoxygenated mixture or oxygen partial pressure.

Only prospective randomized trials will permit the proper evaluation of the benefits that each therapeutic option can give. At present, much research is focused in this direction. Other approaches besides hyperbaric therapy should not be ruled out, especially in the study of cytotoxic phenomena.

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