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WHAT IS THE OPTIMAL TREATMENT "DOSE" FOR DECOMPRESSION ILLNESS?

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Key Words

Decompression illness, oxygen, treatment.

Background

The basis of any treatment of decompression illness (DCI) is an increase in pressure and oxygen content of the breathing gas. However, the optimal way to combine these has not been determined. Results of treatment vary considerably from centre to centre and there are few studies that actually compare the effect of different treatment algorithms in patients with similar symptomatology.

Even if many details regarding the pathophysiology of decompression sickness still are unknown, there is little doubt that the basic problem is the formation of bubbles in the living body. The formation of bubbles requires supersaturation, that the gas tensions in the tissue are above that of the environmental pressure. Treatment has, till now, been focused on this and is intended to reduce the volume of the gas phase and eliminate the excess inert gas load. There is, however, no practical way to measure this load. So the effect of treatment has been judged using clinical signs and symptoms as an end point. This has, however, diverted our attention from the fact that reducing the volume of the gas phase and eliminating inert gas are two different processes that have quite different time constants.

When pressure is applied, the volume of the bubble is immediately reduced according to Boyle's law. Thus, if the bubble itself has a mechanical effect on a tissue, increased pressure will immediately relieve the symptoms. The elimination of gas, however, is a much slower process, in particular if bubbles are present.[1] The bubbles trap gas and reduce the tissue gas tension, which increases the elimination time for the gas.[2] Furthermore, as gas has to be transported to the lungs by the blood, circulatory factors can play a significant role in determining the elimination time. Supersaturation and gas bubbles can be present for hours in the tissue, leading to secondary effects of bubbles that may eventually influence the final clinical outcome.

Different treatment procedures

When oxygen and pressure are used for treatment, there will be the following effects:

1 Increase in environmental pressure. This will reduce the size of the gas bubble and reduce the risk of ischaemic damage.

2 Increase in oxygen partial pressure in blood and tissue. This will increase the gradient for inert gas removal.

3 Increase in the oxygen content of arterial blood. This will increase the oxygenation of the tissue, thus reducing the risk of hypoxic damage.

4 Biochemical and reactive effects of oxygen. These effects, although the least understood, may be highly significant in the treatment of DCI.

Many different treatment regimens have been tried over the years. These procedures are usually based on clinical experience and few studies have been performed actually testing their effectiveness in different groups of patients.

Recommended treatment pressures vary from 200 to 780 kPa (2 to 7.8 bar or 10 to 68 m), while oxygen tensions vary from 180 to 300 kPa (kPa and bar are used in this paper as absolute, not gauge, measurements). However, as was pointed out in a recent workshop,[3] compression to 18 msw (280 kPa or 2.8 bar) breathing 100% oxygen is the only procedure where extensive clinical experience exists. Therefore this should probably be the basic treatment in all cases. In practice, this means the use of USN Table 6 (USN6). However, several studies have documented that both shorter tables at the same depth[4] and treatments at 200 kPa (2 bar or 10 m)[5] give equally good results. Recently, this last group published that 70% of the divers with neurological symptoms were symptom free after two to six hours at 200 kPa (2 bar or 10 m) and that 13% of these divers had persistent manifestations after one month.[6] Another point that is worth noting is that USN6 is used differently in different centres. The number of oxygen cycles vary, some centres use a short, deep pressure spike before commencing with this table and some centres use oxygen on the surface following the end of treatment.

There is little data to support higher treatment pressures. However, most people with experience in the field have case histories where a patient who showed no improvement at 280 kPa (2.8 bar or 18 m) improved on reaching 600 kPa (6 bar or 50 m), either breathing air or a nitrogen/oxygen mix.

Treatment at 600 kPa (6 bar or 50 m) used to be the recommended treatment for air embolism. The theoretical basis for this is that an increase in pressure will reduce bubble size. However, the reduction in bubble size is greatest at the first doubling of pressure (100-200 kPa, 1-2 bar or surface to 10 m). Indeed, Gorman et al. showed, in rabbits, that the vascular bubbles in the brain were cleared as effectively using 202 kPa (2.02 bar or 10.2 m) as using

pressures up to 1,010 kPa (10 bar or 90 m).[7] Kunkle and Beckman showed that bubble resolution time would decrease by a factor of two if oxygen at 280 kPa (2.8 bar or 18 m) was used instead of oxygen at the surface and that further increase in pressure would not decrease resolution time further.[8] Monitored bubbles in the pulmonary artery we were also able to show this and further noted that there was no difference in bubble elimination time in the pressure range from 200-400 kPa (2-4 bar or 10-30 m).[9] In performing these studies, we were impressed by the effectiveness of recompression to 200 kPa (2 bar or 10 m) even using air. Animals with a large numbers of gas bubbles, with hardly any heart beat and no respiration, recovered immediately on arrival at pressure, demonstrating clearly, that in these cases, compression of the bubbles was lifesaving. Furthermore, histological studies of these animals central nervous systems showed that only one out of seven animals had any damage (Brubakk et al. unpublished).

One important point that is often disregarded in evaluating treatment procedures is the ability of the procedure itself to produce supersaturation and gas bubbles. Oxygen in excess of metabolic needs will increase total gas tension and procedures using inert gas mixes can add to the inert gas load. Thus it is conceivable that a procedure that initially removes the gas bubbles, actually may produce new gas bubbles during ascent to the surface.

During recent years, there has been considerable discussion about the use of helium/oxygen mixtures, mostly the use of 50/50 heliox at 400 kPa (4 bar or 30 m) (COMEX 30).[10] This procedure was developed by a French diving company (Comex) who claim to have excellent results with this approach. There are several differences between this approach and USN6, namely a higher environmental pressure (400 vs 280 kPa), a reduced oxygen tension (200 vs 280 kPa), longer oxygen breathing times at greater depths and the use of helium. It is very difficult to judge which of these factors play a significant role for the treatment outcome.

The dose of oxygen has only been considered to a limited degree when evaluating treatment procedures. In general, there is a belief that more oxygen is better and that the only limitation is oxygen toxicity. Oxygen is a vasoconstrictor and, at oxygen tensions of about 200-280 kPa (2-2.8 bar or 10-18 m), blood flow to all organs will be reduced by approximately 20-25%.[11] Furthermore, as oxygen tensions increase, the shunt fraction through the lung will increase, thus reducing the effect of higher oxygen tensions.[12] The use of lower oxygen tensions may actually also be of benefit. Leitch and Hallenbeck, in 1985, showed that oxygen at 200 kPa (2 bar or 10 m) was the optimal treatment gas in spinal cord decompression sickness.[13]

More importantly, oxygen at pressure has numerous biochemical effects which may be of importance when judging the optimal dose of oxygen. If indeed vascular obstruction and endothelial damage plays an important role in decompression illness, decompression illness may be compared to reperfusion injury. Blocking leucocyte adhesion[14] and C5a activation[15] by monoclonal antibodies significantly reduce the injury after ischaemia and reperfusion. In these situations reactive oxygen species[16] play a significant role and it is reasonable to assume that the correct dose of oxygen is important for successful treatment. For example, it has been demonstrated that the glucose metabolism in the injured brain improve after 35-40 minutes at 150 kPa (1.5 bar or 5 m) oxygen, but deteriorated after 15 minutes exposed to 200 kPa (2 bar or 10 m).[17] Timing of treatment as well as the tissue at risk probably also plays a role.

Thom et al. have shown that a single 45 minute exposure to an oxygen tensions of 280 kPa completely blocks activation of leucocytes, a mechanism of central importance in tissue injury and endothelial damage,[18] and this effect lasts for up to 8-10 hours.

End point of treatment

In most situations, the end point of treatment is the elimination of clinical symptoms. Sometimes this can be pretty obvious, as in a patient with a severe paralysis who is able to move his legs. In other cases, it may be much less clear and the treatment results may be influenced by the skill and thoroughness of the examining doctor. It is also well documented that even severe damage to the spinal cord can leave few symptoms.[19]

What is "treatment dose" ?

This is not an easy question to answer. As is pointed out above, there are numerous treatment variations, which make it difficult to compare different treatment algorithms. One simple way of defining this would be to simply integrate the treatment profile. For a single treatment this could be

ppO2 * Environmental Pressure * time (Bar^2 * min).

Using this formula a single USN6 treatment would have a dose of approximately 1,133, while a Comex 30 will give a dose of 2,187, or about 90% higher.

This calculation is an oversimplification and does not, in any way, take into consideration differences in the effects of pressure and oxygen, the possible importance of using different inert gases or the effect of air breathing intervals or ascent rates. It does, however, illustrate nicely the considerable differences between two treatment procedures. For example, breathing oxygen at surface for 120 minutes, will give a dose of 120. This treatment, when initiated at the time of maximum bubble formation, removed

all gas bubbles and was able to prevent serious decompression sickness and CNS changes in a study in pigs (Brubakk et al. in preparation).

Where do we go from here ?

As is pointed out above, USN6 is considered the standard treatment for all cases of decompression sickness. It is however worth asking if this is a correct approach. The three most important questions that can be raised here are probably.

1 Will time to treatment influence the treatment procedure ?
2 Will the type of dive that initiated the symptoms influence the treatment used ?
3 Will symptomatology influence the choice of treatment ?

Recently, studies have claimed that time to treatment is not of importance.[20] The authors argue, based on their results, that patients should be transported to a large facility with sufficient medical resources. However all the patients in these studies had several hours between exposure and treatment.

There is sufficient data to support the assumption that very rapid treatment can reduce the effect of even serious decompression accidents. Surface decompression using oxygen is a well established diving technique, that apparently has few decompression problems.[21] In this procedure, the diver returns rapidly to the surface and is recompressed within 5 minutes to 12 msw (220 kPa or 2.2 bar). We have demonstrated in animals that this procedure produces a significant number of gas bubbles in the surface interval, which disappear during treatment. In many cases however, the bubbles reappear after treatment is ended, indicating that the treatment is sufficient for removing the initial bubbles, but that it is not adequate for eliminating all excess gas.[22]

A large number of successful in-water decompression treatments have been performed, using air, by going to 9 m deeper than the dive. This is remarkable and is perhaps due to the fact that treatment is performed shortly after symptoms appear.[23] We were able to demonstrate experimentally that short (70 minute) treatment at 200 kPa (2 bar or 10 m) using air was effective in animals, if treatment is initiated at the time of maximum bubble formation. The same study demonstrated that 100% oxygen at surface was equally effective under these circumstances. (Brubakk et al. in preparation).

Nearly all decompression tables have been tested by using decompression sickness as an endpoint. When decompression sickness occurs, the diver is immediately treated and it is generally assumed that he thus can escape serious injury. This argument has been used repeatedly by researchers seeking approval for their experiment from the ethical committees.

Rapid recompression will be effective in reducing the size of the gas bubbles and thus limit their direct, mechanical effects. However, considerably more time is needed for eliminating all excess gas. Some treatment procedures recompress the diver considerably deeper than the standard 18 msw (280 kPa or 2.8 bar). If reduction of bubble size is the aim, this does not make much sense as the relative size reduction of additional pressure increase is small.[7] We have shown that the elimination time for bubbles in the pulmonary artery is similar in the pressure range of 200–400 kPa (2-4 bar or 10-30 m).[9]

In a deep dive, particularly if helium is used, a considerable gas load will accumulate. To eliminate this gas load will require time. Thus it is quite conceivable that the length of the treatment, but possibly not treatment depth, may be dependent upon the primary dive.

Due to the difference in the speed of uptake and elimination of gas in the different tissues, it is likely that the gas load and thus the degree of bubble formation will be different in different tissues. A short, deep dive will produce bubbles in quite different tissues than will a long, shallow dive. This is in accordance with what was pointed out by Lanphier and Lehner,[24] that different dives produce different symptomatology. Central nervous symptoms are more common in deep, short dives, while long, shallow dives give predominantly symptoms from joints and muscle. Computer simulations support this and also indicate that bubbles from such deep dives disappear more quickly using pressures of 400 kPa (4 bar or 30 m) with 50% oxygen than when using USN6.[25]

The approach used by Comex for many years, where they treat minor symptoms at 220 kPa (2.2 bar or 12 m) and go to 400 kPa (4 bar or 30 m) for more serious symptoms, may actually have considerable merit.

I think, however, that the time to treatment is important and may influence the choice of table.

Conclusion

At present we do not have sufficient information to make adequate decisions about the optimal treatment "doses" of pressure and oxygen, but such information is urgently needed. This is especially important as we can expect new treatment challenges as divers, using rebreathers, are now able to go deeper, stay longer and use a number of gas mixtures.

Both clinical and experimental data indicate that even compression to 200 kPa (2 bar or 10 m) using air can be

effective if treatment is started early. This has to be further explored as it will have significant impact on the acute management of decompression accidents.

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