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STRATEGIES FOR TREATING DECOMPRESSION SICKNESS.

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Introduction

Decompression has generally been regarded as safe as long as it does not lead to clinical symptoms requiring treatment. Traditionally, the symptoms following decompression (dysbarism) has been distinguished according to where the main symptoms occur (Table 1).

This classification implies that the different categories are well defined disease entities and that there is reasonable agreement between doctors about the classification. Both the study of Smith et al.[1] and a study by Kemper et al.[2] demonstrate that there is considerable uncertainty between experts about classification. For instance, cerebral DCS cannot, in many cases, be distinguished from arterial gas embolism or vestibular barotrauma. Furthermore, several studies have shown that symptoms only from joints are quite rare, they are usually accompanied by central nervous symptoms,[3,4] Extreme fatigue can be classified as a harmless sign or be a sign of subclinical pulmonary embolism.[5] Francis et al.[6] therefore suggested the term decompression illness to include both decompression sickness and arterial gas embolism. They furthermore suggested that the disease should not classified as Type I and type II, but instead described according to clinical symptoms and their development. Using this classification scheme, a high degree of concordance between different doctors was reached .[7]

TABLE 1

CLASSIFICATION OF DECOMPRESSION DISORDERS (DYSBARISM)

Decompression sickness

Type I (mild)	Type II (serious)
Muscles and/or joints (bends, niggles)	Spinal
Skin	Cerebral
Lymph	Vestibular
Malaise/Fatigue ?	Cardiopulmonary (Chokes)

Arterial gas embolism
Barotrauma

Clinical diagnosis and reporting

"The major symptoms and signs of decompression sickness are pain (bends), asphyxia (chokes) and paralysis. Minor effects are rash and fatigue. The parts of the body chiefly involved are the extremities (bends), cardiorespiratory system (chokes) and the spinal cord".[8]

Even today, there is probably little to add to this description by Behnke in 1951, with the possible exception that we believe today that the brain may be more frequently involved and that extreme fatigue may be a more serious sign than previously thought.[5] However, it must be borne in mind that the symptoms can be slight and, as was described by one author, "as many as in syphilis and diabetes together".

In decompression disorders, the patients have to report their symptoms before treatment or investigations can be initiated. In many cases, the patients do not report their symptoms, either because they do not recognize them as being related to the dive or they feel reluctant to do so for many reasons.

There has been, for many years, anecdotal evidence that clinical symptoms of DCI are underreported to a considerable degree. We have recently asked a large group of Norwegian divers about this.[9] 19% of the sports divers, 50 % of the professional air divers and 63% of the saturation divers reported that they had symptoms that had not been treated, a majority of these symptoms were related to the CNS. Interestingly enough, there was a statistical relationship between this and later minor central nervous symptoms.

The incidence of decompression sickness.

There is probably little argument that severe violation of decompression procedures will lead to serious

symptoms and that these are caused by widespread gas bubble formation in many different organs. However, decompression illness requiring treatment is a rare disease. In commercial diving, the incidence of treated DCI is probably below 0.1%.[10] In recreational divers, the incidence is probably considerably below this. However, these general numbers hide the fact that some types of dives, even in commercial operations, have a much higher incidence of DCI. This was seen in the study by Shields and Lee,[11] where the majority of the incidents happened in the more stressful dives, as defined by a high $p\sqrt{t}$, where p (pressure) is the maximum depth of the dive in bar and t is the duration of the dive in minutes.

Even if decompression illness is quite rare, a large percentage of divers have been treated. In a survey among divers in an off-shore diving company in 1985, 38% of the divers with 1-9 years experience and 62% of those with 10-24 years of experience had been treated for decompression sickness.[12] A recent survey of a large population of Norwegian divers, showed that 3% of the recreational divers and 28% of the experienced professional divers had received treatment during their career.[13]

Table 2 shows an overview of symptoms of decompression sickness in several studies over a time period of 90 years.

Even given the possibility that there may be differences in reporting, there are remarkable differences in the symptomatology. Of particular interest is to note that pain is only present in about half of the cases in the amateur divers. Furthermore, that serious injuries of the spine and symptoms from the lungs are quite common in amateur divers. This might fit in with the observation that 17% of the amateurs had experienced extreme fatigue. This sign has been described as a sign of subclinical pulmonary embolism.[5] According to Lehner et al.[17] shallow and long or deep and short dives have a high incidence of chokes. The latter dives also have a high incidence of central nervous DCI. The main difference between these dives are the tissues that will be supersaturated. Thus, the change in symptomatology might indicate a different diving practice and that the decompression procedures are not adequate for the more stressful dives.

Pathophysiology of decompression illness

There seems to be no disagreement that the basic problem in decompression illness is the formation of gas bubbles in the organism. The studies of Boycott et al.[18] which form the basis of most decompression procedures, used the concept of allowable supersaturation, indicating that there was a level of supersaturation that could be tolerated without problems occurring. Many studies since then has shown that this level of allowable supersaturation is actually only related to clinical symptoms, not to bubble formation. Any supersaturation can lead to bubble formation. While many studies confirm this, there is a remarkable difference in the actual occurrence of bubbles between individuals and in one single individual at different times. We do not know the reason for this, but believe that there are significant differences in the number of nuclei present. These nuclei may be composed of small (approx. 1 micron) stable gas bubbles.[19] Furthermore, it is well known that the stress of the dive, including temperature and physical work, can increase the number of bubbles observed. It is important to be aware of the fact that the effect of environmental factors will be greater on the least stressful dives.

Gas bubbles have only been observed in a few locations, even after experimental and very stressful dives.

TABLE 2

INCIDENCE OF SYMPTOMS IN DCI

Subjects	**Caisson workers**	**US Navy divers**	**US Navy divers**	**Recreational divers**	**Recreational divers**	**Occupational divers**
Authors	**Keays**	**Behnke**	**Rivera**	**Kidd**	**DAN**	**Kelleher**
Year	1909 [13]	1947 [8]	1964 [14]	1969 [15]	1993 [16]	1994 [4]
Number	3,692	159	935		1.249	225
	%	%	%	%	%	%
Pain	89	72	92	70	57	67
Rash		14	15		4	5
Paralysis	0.9	0.6	6		6	
Fatigue			1		17	13
Visual disturbances		5	7		6	4
Chokes/Dyspnoea	1.6	4	2		9	8

These are the fat around the viscera, the white matter (myelin sheets) in the central nervous system, in the blood and in the fascia and capsules around joints.[20] There is little reason to doubt that the localized pain in a joint is caused by local gas formation. This has been elegantly demonstrated by Webb,[21] who showed that gas could be seen in periarticular and perivascular tissue spaces and that there was a correlation between the occurrence of gas and pain. Ferris and Engels further demonstrated that strain and muscular activity were correlated with pain at the site where the strain had been applied.[22] One further observations would tend to support this, namely the fact that local compression can in many cases remove the pain. Ferris and Engels claim that the pain can be eliminated by eliminating arterial inflow.

There is evidence from many studies that gas bubbles occur in the venous system during most decompressions.[23-26] Data from the study of Eckenhoff et al.[27] indicate that once the sum of the partial pressures of all gases exceeds the environmental pressure, gas formation occurs in the venous system.

Generally, the main focus on the lungs has been on its role as a filter, where the bubbles are eliminated before they can be transmitted to the arterial side, where their potential for damage is greater. However, if the gas load on the lungs is large, the filtering capabilities of the lungs will be exceeded and gas will enter the arterial circulation.[28] Furthermore, if an patent foramen ovale (PFO) is present, as it is in about 25-30% of the younger population,[29] gas bubbles will be transmitted to the arterial side at much lower pressures.

Several studies have documented the relationship between the occurrence of many venous bubbles and the risk for clinical symptoms requiring treatment.[23,30,31] This, together with the fact that bubbles probably are present in the venous system during most decompressions, suggests that a diver complaining of pain in a joint may be suffering from two different conditions, namely tissue gas in and around the joint and pulmonary gas embolism.

It has been suggested that there is little relationship between gas bubbles detected in the pulmonary artery and clinical signs of decompression illness (DCI). The main reason for this is that gas bubbles have been detected in the absence of symptoms.[24] There seems, however, to be agreement that the risk of DCI increases with increasing number of bubbles. In our experience, having monitored many hundreds of air dives and numerous saturation dives, clinical symptoms do not occur in the absence of pulmonary artery gas bubbles. Nishi points out that for air dives, decompression illness was always accompanied by vascular bubbles.[24] Sawatzky[32] has shown that there is a 5-10% risk of decompression illness in individuals with a single observation of grade III - IV bubbles, using the grading system developed by Spencer and Johanson.[30]

Bubble formation is only the initial insult. The surface of the bubbles act as a foreign substance and will initiate numerous biochemical processes. In vitro studies have demonstrated that gas bubbles have an effect upon both formed elements and biochemical processes in the body. Using gas bubbles in vitro, Thorsen et al.[33] showed that gas bubbles lead to aggregation of thrombocytes.

Ward et al[34] demonstrated that gas bubbles could activate complement in-vitro. Using a different technique, Bergh et al.[3] were able to verify this. The importance of this mechanism in-vivo is still unclear. However, responses of the endothelium to gas bubbles seems to be important in decompression sickness. Chrysanteou et al. have shown that animals exposed to decompression will show breakage of the blood- brain-barrier.[36] Broman et al. have demonstrated that even very short contact between gas bubbles and endothelium (1-2 minutes) will lead to such breakage.[37] Furthermore, studies in rabbits indicate that such contact leads to endothelial damage and progressive reduction on cerebral blood flow and function.[38]

In the central nervous system, bubbles seem to form both in the vessels and in the myelin tissue.[39] Experimentally, it has been shown that after short, deep dives a significant number of individuals have significant hemorrhages in the spinal cord, these individuals are very refractory to treatment.[40]

Generally, decompression illness is considered mostly a "bubble disease". Even if bubbles most probably are the initiating event and the sometimes dramatic response to pressure increase show that the mechanical effect of bubbles certainly play a role, the biochemical reaction to the bubble surface must be considered. This leads to endothelial damage, aggregation of cells on the endothelial surface and an inflammatory response.[41,42] If bradykinin, a strong vasodilator that requires an intact endothelium for its effect,[43] is given after decompression, a dramatic increase in mortality occurs.[44] If however, the animals are treated with anti-inflammatory drugs before the dive, mortality and histological changes are significantly reduced.[44]

Is decompression illness a disease?

According to Webster's dictionary[45] a disease is:

"a condition of an organ, part, structure or system of the body in which there is incorrect function resulting from the effect of heredity, infection, diet or environment. A disease is a serious, active, prolonged and deep-rooted condition."

In contrast to this

"A disorder is usually a physical or mental derangement, frequently a slight or transitory one."

I think there is probably no disagreement when we say that DCI is potentially a disease if it is not treated properly, I will also claim that it can be a disorder if proper action is taken. The aim of all our effort must be to keep DCI as a disorder.

Even if acute clinical symptoms are not present, organic changes may occur. A recent consensus conference determined that such changes, even in individuals with few or no reported symptoms, have been found in the bones, central nervous system and the lungs.[46] The changes are, however, small and probably of little functional significance. They seem to by quite well documented for the lungs and much less defined for the central nervous system. A study of the spinal cord of 20 experienced divers, several with bone necrosis and with a history of decompression sickness, showed absolutely no changes.[47]

This certainly raises the question of how to regard vascular gas bubbles (as detected by ultrasound) without any clinical symptoms, the so-called "silent bubbles" described by Behnke.[8] Most will probably not regard this as DCI. However, the fact that such bubbles are present during most decompressions is similar to the situation in many infectious diseases with detectable pathological flora and few or no symptoms. The question still remains whether these bubbles can have an effect on the organism. We have recently been able to show in the pig that the degree of endothelial damage in the pulmonary artery is dependent upon the number of gas bubbles, if few bubbles were present (less than Grade III on the Spencer scale) no damage could be found.[48]

Initial treatment of DCI

The basis for any treatment is a correct diagnosis. As is pointed out above, this is not easy. Furthermore, many of the treatments are initiated by individuals with little medical and clinical training. If the diver is treated immediately after the onset of symptoms, then treatment is mostly successful and the particular procedure used is probably not very important. However, there is no clear definition of prompt treatment, even a delay of a few hours may reduce the chances of full resolution. If treatment is not prompt, the treatment results are usually less favourable, with residual clinical symptoms being seen in about 50% of the cases.[4] This is particularly the case when there is a long delay between injury and treatment, if this is more than 12 hours, about 70% of the individuals have residual symptoms.[49]

The initial treatment, if a pressure chamber is not available, is breathing oxygen. DAN data, both from Europe and the USA, has shown that this significantly reduces clinical symptoms and reduces the number of sequelae. Preferably a demand valve should be used with a well fitting mask. In more severe cases, fluid may be given i.v. The successful use of other drugs, even if theoretically advantageous, has not been documented.

The basis of any definite treatment of decompression illness is pressure and oxygen. There are four effects of this treatment.

- Increase in environmental pressure. This will reduce the size of the gas bubble and thus reduce the risk of ischemic damage.
- Increase in oxygen partial pressure in blood and tissue. This will increase the gradient for inert gas removal.
- Increase in the oxygen content of arterial blood. This will increase the oxygenation of the tissue, thus reducing the risk of hypoxic damage.
- Biochemical and reactive effects of oxygen. These effects, although the least understood, may be highly significant in the treatment of DCI.

The treatment of decompression sickness has till now been based on mostly empirical data, where a standard treatment has been applied to every case of decompression sickness. The only exception to this has been that in some serious cases has one tried treatments using higher pressures, other gases or saturation. There has been no clear criteria for choosing one over the other.

Recommended treatment pressures vary from 200 to 780 kPa, while oxygen tensions vary from 220 to 300 kPa. However, as was pointed out in a recent workshop,[50] compression to 18 msw (280 kPa) breathing 100% oxygen is the only procedure where extensive clinical experience exist. This treatment should therefore probably be the basic treatment in all cases. In most cases this means the use of USN Table 6. However, several studies have documented that both shorter tables at the same depth[51] as well as treatments at 200 kPa[52] 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 and that 13% of these divers had persistent manifestations after one month.[53]

There is very little data to support higher treatment pressures. However, most people with experience in the field have case histories where a patient that show no improvement at 280 kPa improved on reaching 600 kPa either breathing air or a nitrogen/oxygen mix. Treatment at 600 kPa 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 the largest at the first doubling of pressure (100 - 200 kPa). Indeed, Gorman et al. showed in the rabbits that the vascular bubbles in the brain were cleared as effectively using 202 kPa as using pressures up to 1010 kPa.[54] This is also supported by the study of Kunkle and Beckman,[55] who showed that bubble resolution time would decrease by a factor of two if oxygen at 280 kPa was

compared to the use of oxygen at surface and that further increase in pressure would not decrease this time further.

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 (COMEX 30). This procedure was developed by a diving company who claim to have excellent results with this approach. There are three difference between this approach and the USN Table 6, namely a higher environmental pressure (400 vs 280 kPa), a reduced oxygen tension (150 vs 280 kPa) and the use of helium.

Some animal studies performed by Hyldegaard[56-58] seem to support the use of heliox over 100% oxygen, in particular if the bubbles are located in fatty tissue. However, the advantage of using helium is largely lost if helium is introduced at 280 kPa.[59] A further problem is the location of the bubbles. If they are located in the white matter of the brain, which contains only about 20% fat and where the elimination of the bubbles is largely diffusion limited, a nitrogen bubble will grow. We have demonstrated the growth of such bubbles in aqueous gels for over a week.[60]

The increased pressure may be of benefit. We have shown in pigs that the gas bubbles in the pulmonary artery disappear significantly quicker when recompression is performed according to Comex 30 compared to USN Table 6.[61] Recent extensions of this study demonstrate that bubbles in the pulmonary artery disappear at the same rate for compression pressures from 200 - 400 kPa.[62] In performing these studies, we were impressed by the effectiveness of recompression to 200 kPa even using air. Animals with a large number of gas bubbles, with hardly any heart beat and no respiration recovered immediately on arrival at pressure. This would be a strong support for recompression even in divers who are terminally ill.

The use of lower oxygen tensions may actually also be of benefit. Leitch and Hallenbeck showed that oxygen at 200 kPa was the optimal treatment gas in spinal cord decompression sickness in dogs.[63]

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, at oxygen tensions of about 200-280 kPa, blood flow to all organs will be reduced by approximately 20-25%.[64] Furthermore, as oxygen tensions increase, the shunt fraction in the lung will increase, thus reducing the effect of higher oxygen tensions.[65]

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 leukocyte adhesion[66] and C5a activation[67] by monoclonal antibodies significantly reduce the injury after ischemia and reperfusion. In these situations reactive oxygen species play a significant role[68] 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 oxygen, but deteriorated after 15 minutes exposed to 200 kPa.[69] 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 tension of 280 kPa will completely block activation of leucocytes, a mechanism of central importance in tissue injury and endothelial damage, this effect lasts up to 8-10 hours.[70]

The use of drugs is at present largely experimental and no definite recommendations can be made. In France, aspirin is regularly used,[53] although in-vitro studies have demonstrated that acetylsalicylic acid has no effect on platelet aggregation induced by gas bubbles.[71] If further studies demonstrate that endothelial damage plays a significant role, this will open exciting possibilities for drug treatment.

An adequate circulatory volume should be maintained, but it is important to keep in mind that overhydration may lead to the risk of cerebral edema, particularly if the blood-brain barrier has been damaged. If fluids are used, it is important to keep in mind that there is growing evidence that hyperglycaemia will significantly increase the injury of the central nervous system.[72] If treatment is performed in a hot climate it is also important to be aware of the fact that hyperthermia also will lead to an increased injury.

Treatments if initial treatments are not successful

The definition of a non-successful treatment is not easy. In many cases, the patient improves under pressure, but some symptoms are still present. This can be handled either by keeping the patient under pressure and performing more oxygen cycles or by going deeper using some of the options available or by decompressing to the surface and performing follow up treatments. There is not enough data to support any one approach. Data from DAN indicates that more than 5 follow-up treatments may not give any additional benefit.[73]

The future

There is still much that is unknown in the treatment of decompression illness. One particularly interesting point

is the question whether a standard treatment should be used for all cases of decompression illness. Due to the difference in the speed of uptake and elimination of gas in the different tissues, it is likely that the gas load in the different tissues, and thus the degree of bubble formation, will be different in different tissues. A short, deep dive will produce bubbles in quite different tissues from a long shallow dive. This is in accordance with what was pointed out by Lehner, that different dives produce different symptomatology.[74] Central nervous symptoms are more common in deep, short dives, while long, shallow dives produce predominantly symptoms from joints and muscle. Computer simulations support this and also indicates that bubbles from such dives disappear more quickly using pressures at 400 kPa with 50% oxygen than when using USN Table 6.[57] Thus an approach like the one used by Comex for many years may actually have considerable merit, where they treat minor symptoms at 220 kPa and go to 400kPa for more serious symptoms.

At present we do not have sufficient information to make adequate decisions about optimal treatment strategies. Such information is urgently needed, considering the large number of individuals who are left with sequelae after treatment with today's procedures. This is even more important as we can expect new challenges as recreational divers will be able to go deeper, stay longer and use a number of gas mixes.

A possible future strategy for treatment

1 Oxygen as soon as possible after the insult.

2 Initial treatment regardless of symptoms apply pressure and oxygen, 200 kPa ?

3 If not immediate response of treatment or more than 2 hours between insult and treatment, evaluation of case:

what is the gas load in different tissues ?
where are the bubbles located ?
are there many bubbles in vessels ?
are there bubbles in the brain / spinal cord tissue ?
will the resolution of the bubbles be dependent upon diffusion or perfusion limitation ?
what role does an inflammatory responses play ?
is hypoxia an important part of the picture ?

4 The evaluation above shall result in a treatment algorithm that will define optimal pressure, breathing gas and decompression procedure as well as the optimal use of drugs.

It must be pointed out that much of this information is not available today. It can, however, give us a framework for what information may be of importance and the need for the development of the necessary methods for obtaining that information.

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RECOMPRESSION THERAPY FOR DECOMPRESSION ILLNESS

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Key Words

Decompression illness, oxygen, treatment.

Abstract

Recompression therapy for decompression illness was developed empirically based on observations by compressed air workers. The rationale that was developed fit the evidence that the disease was caused by bubbles, and it has been presumed that the major mechanism of action is related to physical reduction of bubble size. Oxygen was later added to increase the gradient for diffusion of nitrogen from bubbles, and to relieve tissue hypoxia. Definitive treatment of decompression illness (DCI) includes the administration of oxygen under pressure. Current recommendations include initial recompression to 2.8 bar, using USN, RN or closely related commercial procedures. A review of experimental data and experience with recompression tables is discussed. Expeditious application of recompression using oxygen along with standard resuscitative measures is usually successful in treating decompression illness. Recent evidence suggests that pharmacological effects of hyperbaric oxygen, in addition to the physical effects on bubble size, gas diffusion and oxygenation, may be important in resolving the disease.

Introduction

Recompression therapy dates back to the 19th century. The bridge across the Mississippi River at St. Louis, completed in 1874, at was an engineering milestone in the United States, because the bottom of the Mississippi is covered in mud and it was impossible until that time to construct piers using traditional bridge building techniques. In order to excavate down to bedrock the engineers used what resembles an upside-down cup (caisson), into which was pumped compressed air to maintain the internal pressure equal to that of the hydrostatic pressure outside. As the caisson rested on the bottom, the air pressure prevented the ingress of water and mud, allowing workers inside to facilitate pumping of the mud to the surface. The caisson gradually sank by its own weight, aided by the mass of the bridge pier being constructed atop the caisson. Once on bedrock, the caisson was filled with concrete, locking the bridge pier permanently into place. This was the first major use of caisson construction work in the United States.

At the end of a shift the men decompressed in an independently pressurised lock. As the depth (and hence the ambient pressure in the caisson) increased, the men were subjected to progressively increasing decompression stress. Many of the men developed neurological decompression sickness (DCS) and 14 of them died. It is perhaps of note that as a result the engineer, James Eads, hired a local doctor, Dr Alphonse Jaminet, who then became the first occupational physician in the United States concerned with the welfare of men working under pressure, to take care of the men. This man, although not knowing the pathophysiology of decompression sickness, elucidated several procedures and principles for the prevention of this illness that are still believed correct to this day.[1]

One of Dr Jaminet's contributions is an account of an episode of spinal cord bends that he experienced after leaving the caisson following a visit to the work site. With no definitive treatment available, other than tincture of time, he went home, drank some wine and gradually got better. Unfortunately this was not the fate of Washington Roebling, the engineer of the Brooklyn Bridge, built a few years afterward using the same technology, who became permanently disabled by spinal cord DCS after helping to fight a fire in the caisson.