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THE USE OF OXYGEN IN DECOMPRESSION

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Introduction

The beneficial effects of oxygen in decompression have since long been recognized. Oxygen can be used to lower the incidence of decompression sickness and/or to reduce decompression time. Breathing of oxygen or oxygen-enriched mixtures has been incorporated in many commercial and governmental decompression and diving procedures. It has also been advocated for use in decompression of caisson workers, for instance by Nashimoto (1).

Berghage et al. (2), in a brief review of the historical literature, starting of course with Paul Bert, conclude that two general concepts have developed to explain the beneficial effects of oxygen:
1) The decompression advantage gained is proportional to the amount of inert gas replaced (the basis of the Equivalent Air Depth theory); and
2) Oxygen breathing during decompression increases the gradient or partial pressure difference between the tissues and the alveolar air and therefore improves inert-gas elimination (Oxygen Window concept).
They emphasize that neither concept takes into account the physiological dynamics of the situation, since the body's response to elevated PO2 can alter gas exchange and can affect the effectiveness of oxygen.

Although Hills (3) states optimistically that "decompression sickness would be avoided completely if a subject could breathe pure oxygen for all exposures," it is not only the problem of oxygen toxicity that kills the joy. From the literature, as surveyed by Vann (4), it is clear that high PO2 can contribute significantly to the incidence of decompression sickness, possibly by behaving partly as "inert gas". The use of excessive oxygen may also affect filtration capability of the pulmonary circulation and could, during decompression, cause "silent" venous bubbles to become arterial emboli, with the risk of promoting neurologic decompression sickness (5).

Nevertheless, the benefits of oxygen in decompression do exist, as proven by daily practice. Despite all research, however, the optimum use of oxygen is still debatable, as can be seen from the different ways of application in the various decompression tables. Even the limits of hyperoxia, to prevent toxicity, are not firmly set.

Oxygen Toxicity

Monitoring of central nervous system and pulmonary toxicity risk

Ever since the work of Paul Bert (6) and Lorrain Smith (7), most concern has been focused on the toxic effects of oxygen on the central nervous system and the lungs, although it is known that oxygen can poison any living cell (8,9). Generally it is assumed that the lungs are the first to be affected when breathing oxygen at partial pressures between 0.5 and 2 bar, while at greater pressures the central nervous system effects precede (10,11). This may be true at rest, but CNS toxicity has been described in men breathing pure oxygen as shallow as 6 metres when working to exhaustion (12).

Oxygen tolerance curves have been developed, which describe the "average" susceptibility of normal men to increased inspired oxygen tensions as a function of time (10,13), for central nervous system as well as for pulmonary poisoning. These curves, however, suppose a continuous exposure to hyperoxia. Nevertheless, it is well known, that periodic interruption of oxygen exposure by normoxia extends CNS and pulmonary oxygen tolerance (10,13,14).

Hills (15) has proposed a cumulative oxygen toxicity index (COTi) to determine the likelihood of a toxic event. It permits the comparison of a complex oxygen exposure profile against a set of limits determined from single exposure levels and times. This index, however, has only been validated experimentally for CNS toxicity, although Hills suggested that it could be used also for pulmonary manifestations. This has been tried by Hamilton et al. (16) in the SHAD nitrox saturation dives, but they had to conclude that it did not forecast pulmonary toxicity under these circumstances.

A means for quantifying pulmonary oxygen dose is the Unit Pulmonary Toxicity Dose, which can be accumulated into a Cumulative Pulmonary Toxicity Dose (11,17,18). An acknowledged drawback of CPTD is that it does not account for normoxic interruptions, in contrast to the COTi. For routine operations, a maximum dose of 615 CPTD is recommended. Although not stated explicitly, this is usually taken as a daily dose.

Sterk et al. (19), however, have drawn attention to a "chronic oxygen toxicity syndrome", developing in divers exposed to intermittent hyperoxia of 600 or 800 CPTD per day on a working week basis (five days per week), next to or apart from pulmonary oxygen toxicity. The most important symptoms of this "syndrome" were fatigue, paresthesias, headache and dizziness. Highly pathognomonic were sensations of numbness and tingling in fingers and toes. In the divers exposed to 600 CPTD per day, these symptoms became prominent in the second week, while in the divers exposed to 800 CPTD per day it started already on the second day. The symptoms appeared to be very persistent, when exposure continued after development of the first signs of this form of oxygen toxicity. It was concluded that exposure to 600 CPTD or more per day on a working week basis could not be considered safe. Defining a safe limit, however, was not possible from their study.

The USN oxygen limits

Other leads for the estimation of a probable tolerable oxygen dose can be found in the USN Diving Manual. Although unable to locate a reference, Hamilton et al. (16) hint at intensive investigations of oxygen toxicity conducted by the USN Experimental Diving Unit before WW II. It is possible that the guidelines on oxygen limits in this manual reflect the results of this research. The data of the USN Diving Manual, together with calculated CPTD, are presented in table 1.

The oxygen partial pressure limits table gives values for "normal" and "exceptional" exposures. In commercial diving, the "normal" exposure values are generally considered as too conservative and the limits given for "exceptional" exposure are followed, as in the Norwegian tables (20).

The section on oxygen diving gives also oxygen depth/time limits, for normal and exceptional operations, respectively shallower or deeper than 25 feet. For convenience, the depth has been presented in metres, taking 3 metres for 10 feet. With only slight inaccuracy, this can easily be transferred to oxygen pressure in bar.

The recently published new exposure limits for U.S. Navy closed-circuit oxygen scuba divers (21) are also included in table 1. There appears to be some inconsistency between the various limits, but particularly between the new oxygen depth/time limits and the oxygen partial pressure limits table. Interesting is, that the old limits allow a maximum CPTD of 355, while the new limits go up to 515 CPTD. This is probably permissible to prevent pulmonary problems, but does this also count for the chronic oxygen toxicity syndrome ?

The chronic oxygen toxicity syndrome

Symptoms as described by Sterk et al. (19), have been noted by others mainly in experiments concerning pulmonary oxygen toxicity after prolonged exposure to oxygen tensions up to 2 bar. They are usually reported incidentally as "other symptoms."

OXYGEN PARTIAL PRESSURE LIMITS TABLE

Normal Exposure			Exceptional Exposure		
PO2 (Bar)	Time (min)	CPTD	PO2 (Bar)	Time (min)	CPTD
1.6	30	58	2.0	30	75
1.5	40	71	1.9	40	94
1.4	50	82	1.8	60	133
1.3	60	89	1.7	80	166
1.2	80	106	1.6	100	193
1.1	120	140	1.5	120	214
1.0	240	240	1.4	180	294
			1.3	240	355

OXYGEN DEPTH/TIME LIMITS

	Old		New	
Depth (m)	Time (min)	CPTD	Time (min)	CPTD
15	-	-	10	32
12	10	28	15	42
10.5	25	64	25	64
9	45	106	80	189
7.5	75	161	240	515
6	110	212	240	463
4.5	150	256	240	410
3	240	355	240	355

Table 1. USN oxygen limits, with calculated CPTD.

Comroe et al. (22) exposed 55 men to pure oxygen at 1 atmosphere for 24 hours (1400 CPTD) and found such symptoms in 19 subjects, three of them having paresthesias such as tingling in the extremities "while breathing 100 per cent oxygen." They commented that these and other symptoms "were similar to those commonly noted in decompression sickness."

Becker-Freyseng (23) reported an experiment on 2 subjects, one being himself, exposed for 65 hours to 90 per cent oxygen at atmospheric pressure. Prickling and numb sensations of the fingertips started at the end of the second day (± 2000 CPTD) and intensified, amongst other symptoms, as the experiment continued. In his case, it took 2 weeks after the experiment for these paresthesias to disappear.

In their study of extension of pulmonary oxygen tolerance at 2 ATA by intermittent oxygen exposure, Hendricks et al. (16)

reported that all six of their subjects experienced "neurological symptoms of numbness and paresthesia." These subjects had been exposed to periods of 20 minutes oxygen and 5 minutes normoxic nitrox at 2 ATA until they showed clear signs of pulmonary toxicity, which took from 11.3 to 15.7 "oxygen hours" or 14.1 to 19.6 hours "real time" (1690-2350 CPTD). The above data, particularly referring to paresthesias, are summarized in table 2.

REFERENCE	SUBJECTS	PO2	CPTD	PARESTHESIA
Becker-Freyseng (1950)	2	0.9	2000(3230)	2
Clark, et al. (1971)	13	2	1630	1
Comroe, et al. (1945)	55	1	1400	3
Hendricks, et al. (1977)	6	2/0.2	1690-2350	6

Table 2. Data from the literature. Reports on paresthesias during or after hyperoxia.

Tingling and numbness in fingers and toes have also been reported during air saturation dives. In SHAD II, storage depth 60 ft on air (PO2 0.58 bar), one diver reported numb fingertips and toes after about one week, having accumulated around 1600 CPTD. Since these symptoms "were not observed in conjunction with hyperoxia" and disappeared within 5 days, it was thought to be unlikely as a manifestation of oxygen toxicity (16).

Reports from SCORE, however, are more specific (25). Here, storage depth was also 60 ft on air, while excursions of one hour duration were made on air to 200, 250, and 300 ft. A total of 8 divers participated in this project, 2 of them having oxygen-related problems. Diver HT reported bilateral numbness of his fingers and backs of hands following the morning excursion to 250 ft. Onset of symptoms occurred when he had accumulated barely 790 CPTD. After thorough investigation, however, the final diagnosis was that the symptoms reported were indicative of oxygen toxicity.

The next morning, he was not allowed to participate in the scheduled excursion, since the tips of his fingers were still numb. Following a report that the symptoms had cleared, he was added to the afternoon excursion to 300 ft. At 54 minutes in this excursion, he went into convulsions. About 3 minutes later, he could be decompressed to storage depth. Ten minutes after return to storage depth, diver WM got a bend and was compressed to 100 ft. He was switched onto a 50/50 mix (first nitrox; after 10 minutes, heliox) during 25 minutes, which relieved his symptoms. Then he was put on 20 percent oxygen in helium and decompressed. At 70 ft, WM reported numbness of fingers, having accumulated a total of 1455 CPTD.

Long-exposure air dives in the Eastern Scheldt

Since 1976, a large number of long exposure air dives have been made in the Eastern Scheldt, with bottom times of approximately 4 hours, with a diving bell and occasionally a habitat. To shorten decompression time, oxygen is used intermittently from 15 metres up to the surface. For normal operational procedures, we allowed a diver to make 4 long-exposure dives per week and chose an arbitrary limit for exposure to hyperoxia of 450 CPTD per day. This CPTD limit was introduced to enable the use of oxygen for treatment, should decompression sickness occur, without getting trouble with pulmonary toxicity. Fortunately, we had very few cases of decompression sickness, nor did we encounter clear signs of oxygen toxicity.

DIVER	CPTD wk 1	CPTD wk 2	CPTD total
JO	671	1183	1854
ME	-	1476	1476
SL	639	1264	1903
TE	803	1274	2077
VE	971	2468*!	3439
ZE	436	1501	1937
ZW	971	1597*	2568

Table 3. Long-exposure air divers in the Eastern Scheldt, who accumulated more than 1200 CPTD in the second week and/or more than 1800 CPTD in total, during a two-week diving period in 1984.

Careful examination of our data, however, revealed an interesting case in the summer of 1984. At that time, 98 long exposure dives were made by 20 divers within two weeks to depths around 30 metres. Diving started in the middle of the first week. Some divers participated only in a few long exposures, while others went up to our limits. As shown in table 3, seven of these divers accumulated either more than 1200 CPTD in the second week and/or more than 1800 CPTD in total during the entire two-week period. Noteworthy are divers ZW and VE. Nearly 3 hours after the very last long exposure dive of the second week, ZW developed a bend in his left knee, which was treated on USN table 5. After that he was symptom free and no signs of oxygen related problems were reported. By then he had accumulated 749 CPTD during the last 24 hours, 1597 units in the last week and 2568 units in total.

Unfortunately the buddy of ZW from the last long exposure dive, diver VE, was put in the chamber as the tender. He did get oxygen during the last 30 minutes from 9

metres to the surface, but some 4 hours later developed a bend in both knees. At that time I was contacted and advised to start treatment on USN table 6. It was during this treatment that tingling fingers were reported. Figure 1 displays the dive and CPTD profile of diver VE during the entire two weeks. In the week before, he did not dive at all.

At the start of treatment on table 6, VE had accumulated a total of 2653 CPTD, of which 1682 units during the last week and 482 units during the last day. Since his DCS symptoms did not clear easily, I advised an extra period of oxygen at 18 metres. Due to a miscalculation of the supervisor in charge, he received even one more cycle on oxygen at that depth. In the meantime, his symptoms had disappeared. Then he was decompressed in accordance with the table to 9 metres and after 18 minutes at the second oxygen cycle of 60 minutes at that depth he started to complain about tingling fingers. By then he had received 636 CPTD during treatment, making 1118 units during the last 24 h, 2318 units during the last week and a grand total of 3289 CPTD.

Since I suspected oxygen related problems, I advised completion of Table 6 without further extension. On arrival at the surface he had received 786 CPTD during treatment, 1268 units during the last 24 hours and a grand total of 3439 units. Two hours later, the tingling in his fingers was almost gone. After six hours, he was totally symptom free.

The Use of Oxygen in Diving and Decompression

Setting the limits

Exposing a non-military diver in the water to the possible hazards of CNS oxygen toxicity is, in my opinion, not acceptable. The CNS toxicity risk seems to be completely ruled out by choosing 1.5 bar as a limit for oxygen partial pressure (26). Therefore, this should be the limit for commercial diving with oxygen enriched mixtures. There is no general agreement on this. The Norwegians, for instance, use the USN exceptional exposure table and go up to a PO2 of 2 bar in diving with nitrox (20). In a series of 2662 of such dives, one CNS oxygen hit was reported in a hard working diver at a depth of 43 metres (27) and the PO2 in his mix must have been around 1.7 bar, but the diving time was not mentioned.

The incidence of CNS oxygen toxicity in the Norwegian series seems low but can not be weighed as the relationship between PO2 and time is not reported. According to Vann (26), the risk should have been around 4 %. Such a risk can only be acceptable in a situation where all is under control, as during decompression in a bell or a chamber. In such a situation, the USN limits for exceptional exposure seem to be a valuable guidance to avoid problems.

The limits to prevent pulmonary and "chronic" oxygen toxicity are still difficult to assess from the available data. The wide variations in susceptibility to oxygen toxicity between and within individuals (28), makes it extra difficult. A sole exposure to a maximum of 615 CPTD seems safe enough for routine operations, compared to the data from experiments with continuous exposure, but not for repetitive exposures on a working week basis. In the air saturation dives discussed here, a daily dose of 300 to 400 CPTD was administered. As said before, a too high PO2 at storage depth could have prevented complete recovery from oxygen toxicity.

The "old" U.S. Navy limits show a maximum CPTD of 355. While this indeed reflects former research, this could well be the safe maximum. It is interesting to see that "new" U.S. Navy oxygen depth/time limits go up to 515 CPTD. Whether this has been weighed against the possible occurrence of a "chronic oxygen toxicity syndrome" remains questionable. The Norwegian tables, in setting limits for nitrox diving, go to a maximum of 419 CPTD, allowing 360 minutes at a PO2 of 1.1 bar (20).

In the Eastern Scheldt long exposure air dives, finally, the limit is set to 450 CPTD per day, up to 1800 CPTD per week. This seems to have been a lucky guess, since the only sign of oxygen toxicity was seen in diver VE, whose CPTD load is summarized in figure 4. Paresthesias started only towards the end of treatment on an extended USN table 6, after a two-week period of regular long exposure air diving. At the time of symptom onset, he had accumulated 1118 CPTD in the last 24 h, as displayed by the lower curve in Fig 2. Whether the development of his oxygen toxicity symptoms were influenced by the CPTD load of the last week (middle curve) or even the week before (upper curve), remains uncertain. Based on our previous experience (19), it is very likely that the CPTD history of the entire two weeks should be taken into account. In that respect, there is a great need for a better tool to monitor exposure to increased levels of oxygen than keeping track of CPTD load.

As long as no further data are available, a limit around 400 CPTD seems safe enough as a daily dose for intermittant hyperoxia in operational diving. This may also count for saturation diving, when PO2 at storage depth is lower than 0.58 bar.

Application in practice

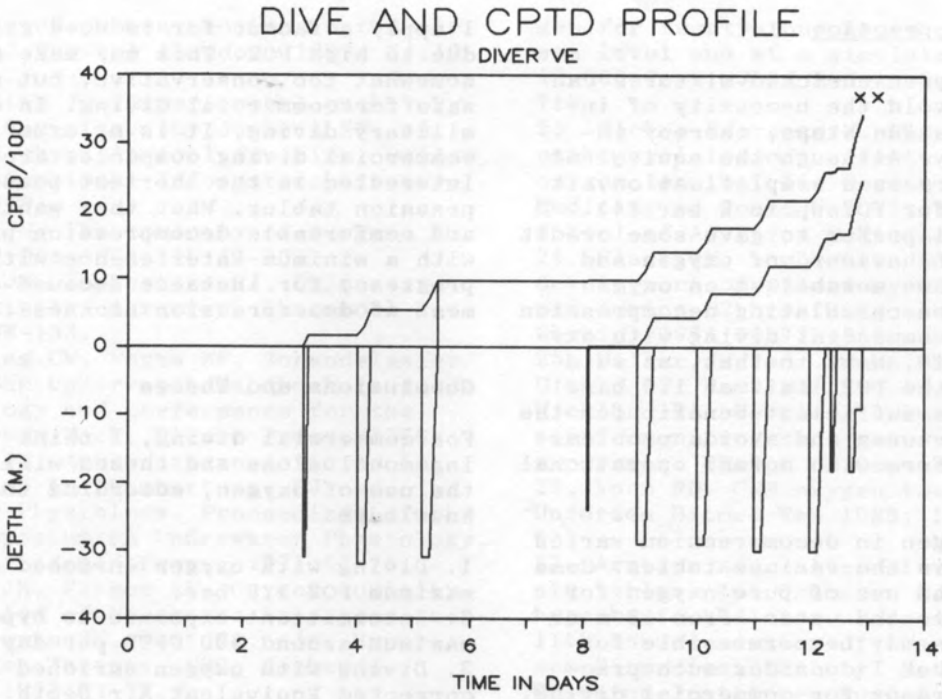

Figure 1. Dive and CPTD profile of long-exposure air diver VE. The duration of paresthesias is indicated by x x.

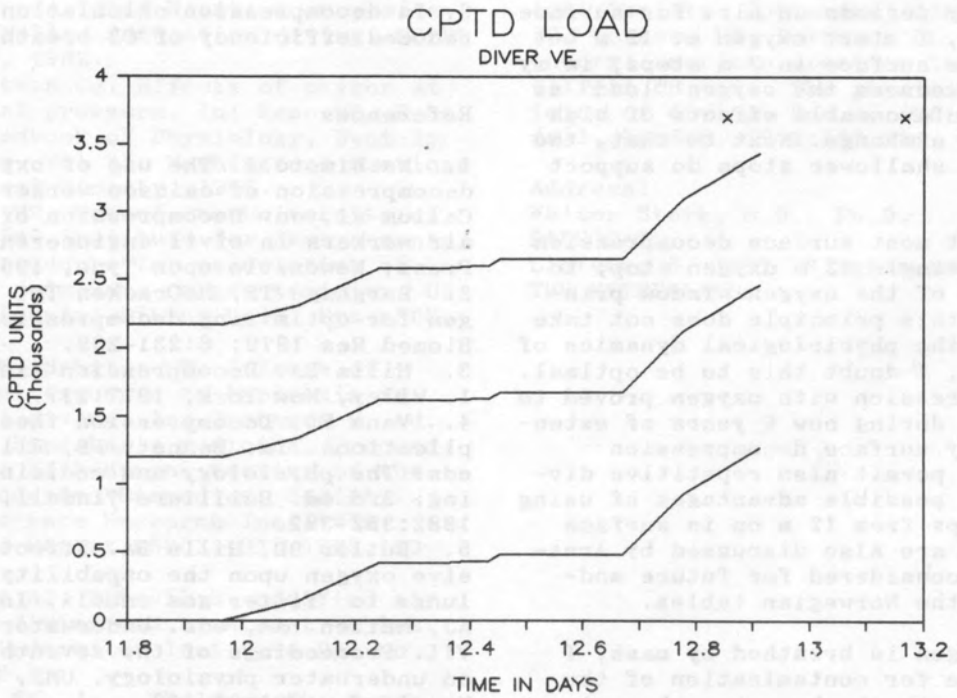

Figure 2. CPTD load of long-exposure air diver VE, in the last 24 hours (lower curve), the last week (middle curve) and for the entire two-week period (upper curve). The duration of paresthesias is indicated by x x.

Application in practice

Diving with oxygen enriched mixtures can be useful to avoid the necessity of in-water decompression stops, thereby increasing safety. Although the equivalent air depth theory is a simplification, it seems to hold for PO2 up to 2 bar (4). Nevertheless, I prefer to give some credit to "inert gas behaviour" of oxygen and effects of tissue metabolism on oxygen extraction, when calculating decompression schedules for commercial diving with oxygen enriched air. Next to that, as said before, I set the PO2 limit at 1.5 bar. All this leaves sufficient benefit for the use of such mixtures and avoids problems that can interfere with normal operational procedures.

The use of oxygen in decompression varies significantly in the various tables. Some tables allow the use of pure oxygen for decompression in the water, from 18 m or shallower. This may be permissible for military use, but I consider such practice too hazardous for commercial diving. Since no general agreement exists about how and when oxygen is to be used for decompression in commercial diving, over the years I have developed my own "rules", which are certainly debatable.

For decompression in a dry bell or a chamber, I allow the use of oxygen from 15 m on. In a wet bell, oxygen breathing is permitted from 9 m on. Conforming to general practice, the oxygen periods are interrupted by periods on air. For surface decompression, I start oxygen at 12 m but then go to the surface in 3 m steps; in my opinion this reduces the oxygen "load" as well as the unfavourable effects of high oxygen on gas exchange. Next to that, the air breaks at shallower stops do support "de-gassing".

I realize that most surface decompression tables use a single 12 m oxygen stop, to make full use of the oxygen window principle. Since this principle does not take into account the physiological dynamics of the situation, I doubt this to be optimal. Staged decompression with oxygen proved to be successful during now 6 years of extensive use of my surface decompression tables, which permit also repetitive diving (29). The possible advantages of using oxygen in stops from 12 m on in surface decompression are also discussed by Arntzen (27) and considered for future modification of the Norwegian tables.

When pure oxygen is breathed by mask, I make allowance for contamination of the inspired gas with chamber atmosphere in calculating the decompression schedule. This "habit" originates from measurements of the efficiency of various types of oxygen masks, in the beginnning of the 1970's at our Diving Medical Centre and is also practiced by others (20,30). Next to that, I apply a factor for reduced gas exchange due to high PO2. This may make my tables somewhat too conservative, but at least safe for commercial diving. In contrast to military diving, it is my experience that commercial diving companies are not really interested in the shortest possible decompression tables. What they want is safe and comfortable decompression procedures, with a minimum interference with work progress, for instance because of treatment of decompression sickness.

Conclusions and Theses

For commercial diving, I think the following conclusions and theses will hold for the use of oxygen, according to today's knowledge:

1. Diving with oxygen enriched air: maximum PO2 1.5 bar.
2. Intermittent exposure to hyperoxia: maximum around 400 CPTD per day.
3. Diving with oxygen enriched air: use corrected Equivalent Air Depth.
4. Oxygen during decompression: use regular air breaks.
5. No pure oxygen for in-water decompression.
6. In chamber or dry bell: oxygen from 15 m on.
7. In wet bell: oxygen from 9 metres on.
8. In surface decompression: staged decompression with oxygen. (may have advantages as reduced oxygen load and enhanced gas exchange)
9. In decompression calculation: allow for reduced efficiency of O2 breathing.

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