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EFFECTS OF INTERMITTENT EXPOSURE TO HYPEROXIA IN OPERATIONAL DIVING

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Two groups of six divers were exposed to hyperoxic mixtures during three no-stop dives of two hours duration per day, interrupted by one-hour surface intervals breathing air, five days per week. One of the three daily dives was an operational dive, while the other two were simulated in a dry compression chamber. Group I was exposed to 600 CPTD and group II to 800 CPTD per day. A control group of six divers went through a similar schedule in a dry and wet compression chamber on air at a pressure of 1.5 bar. Before and after each dive, tests were done to detect signs of oxygen toxicity.

We encountered a chronic oxygen toxicity syndrome consisting of symptoms as fatigue, headache, dizziness and paraesthesiae. Most characteristic was a feeling of numbness in fingers and toes. In group I this was prominent at the end of the second week, while in group II this started already at the second day.

It is uncertain whether these symptoms are of central or periferal origin. This is further investigated at present. Meanwhile, it is concluded that daily exposure to 600 CPTD or more can not be considered safe at a working week basis and a schedule as in our experiment.

INTRODUCTION

The value of pure oxygen or oxygen enriched mixtures in shortening decompression times, is obvious. Such procedures have been used extensively in the Eastern Scheldt, where a huge Storm Surge Barrier is under construction since 1976. The water depth in this area ranges up to 50 metres, so all diving is done on air. Many long exposure dives, with bottom times of approximately 4 hours, are and have been made with a diving bell and occasionally a habitat, for the purpose of subsea soil investigation or construction work.

To keep decompression times within practical limits after long exposure dives, we had to use oxygen as much as possible during decompression. After transfer under pressure to a dry chamber, oxygen was administered intermittently by mask from 2.5 bar up to the surface. Since little is known about the effects of intermittent hyperoxia when used on a

working week basis, an arbitrarily limit of 450 CPTD (Cumulative Pulmonary Toxicity Dose) per day was used for normal operational procedures. In more than 2000 man-dives, we had only a few cases of mild decompression sickness and no obvious complaints about oxygen related problems.

Last year in the Eastern Scheldt, a lot of work had to be done at depths around 20 metres, involving as many as 75 divers working day and night. It was considered that, particularly at these depths, the use of oxygen enriched air or nitrox could safe time by avoiding decompression. However, we stressed the importance of first doing research into the effects of hyperoxia on the long run, before these mixes to be used widely.

Most human research concerning oxygen toxicity is focussed on the effects on the lungs and the central nervous system, although it is known that oxygen can poison any living cell [Balentine (1977), Clark (1974)]. 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 [Clark et al. (1971a), Shilling et al. (1976)]. 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 [Young (1971)]. To avoid the risk of CNS oxygen toxicity in working divers entirely, we therefore chose 1.5 bar as a limit for oxygen partial pressure while diving with enriched air.

This left us with the possibility of pulmonary oxygen toxicity. Oxygen tolerance curves have been developed, which describe the "average" susceptibility of normal men to increased inspired oxygen tensions as a function of time [Clark et al. (1977), Clark et al. (1971a)]. Pulmonary tolerance curves are based on the decrease in vital capacity as an early sign of oxygen poisening. Clark and Lambertsen (1971b) found that the decrease in vital capacity began before symptoms became prominent, while others [Hendricks et al. (1977)] found symptoms well before vital capacity changes could be detected. Nevertheless, the measurement of vital capacity is still regarded as a useful index of pulmonary oxygen toxicity [Eckenhof (1983)].

Manifestations of oxygen poisening appear to be completely reversible in the early stages, once exposure to hyperoxia is terminated [Hamilton et al. (1982), Hendricks et al. (1977), Lambertsen (1965)]. Complete recovery, however, may take hours to even weeks [Clark et al. (1971b), Hendricks et al. (1977), Fisher et al. (1968)]. It is well known, that periodic interruption of oxygen exposure by normoxia extends CNS and pulmonary oxygen tolerance [Clark et al. (1977), Clark et al. (1971a), Hendricks et al. (1977). However, little quantitative data are available, particularly when longer normoxic interruptions are applied. Lambertsen (1955) observed that, during World War II operations, nearly daily exposures of divers to the limits of CNS oxygen tolerance over a period of more than a month did not result in evident cumulative toxic effects. Also Donald (1947), in a large series of subjects, found that multiple exposures to hyperoxia until the onset of CNS symptoms did not cause residual neurologic or pulmonary impairment.

A means for quantifying pulmonary oxygen dose is the Unit Pulmonary Toxicity Dose, which can be accumulated into a Cumulative Pulmonary Toxicity Dose [Lambertsen (1965), Shilling et al. (1976), Wright (1972). For routine operations, a maximum dose of 615 units is recommended. Although not stated explicitly, this is usually taken as a daily dose. An acknowledged drawback of CPTD is, that it does not account for normoxic interruptions. Another lead for the estimation of a probable tolerable oxygen dose is the USN Oxygen Limit Table. The section for "exceptional

exposure" gives a tolerance time of 120 minutes for an oxygen tension of 1.5 bar. Again it is not clearly stated, whether this should be taken per dive, per 12 hours or per day. There is apparently a great need for more human data concerning oxygen tolerance.

METHODS

The work at the Eastern Scheldt provided us with a unique opportunity to collect such data in an operational setting. Led by the literature, our primary aim was to look for pulmonary oxygen toxicity. Two groups of six divers, all volunteers, where investigated thoroughly one week before the start of the experiment and trained in spirometry. Haematological data were collected before and after each week of the experiment. Chest x-rays were taken before and after the experiment.

Group I was exposed to three dives of two hours duration per day, breathing nitrox with an oxygen tension of about 1.5 bar, interrupted by one-hour surface intervals breathing air. One of the three dives was a working dive, to get the job done, at a depth of 15 + or - 1.5 metres, depending on the tide. The other two dives were simulated in a dry chamber at a depth of 18 metres. No decompression stops were necessary. This regime accounted for around 600 UPTD per day and was maintained five days per week. Before and after each dive spirometry was performed, as well as bookkeeping of subjective symptoms.

Since in the first week of the experiment no clear effects of oxygen poisening were encountered, we decided to start with the second group, in a similar fashion as group I, but expose them to 800 UPTD per day. To achieve this, the two chamber dives for group II were performed at 10 metres with pure oxygen for two hours, interrupted after one hour by ten minutes on air. The working dive was the same as for group I, with nitrox.

In a separate session, a control group of six divers was run at a same schedule as for group I, but on air in the compression chamber of the Diving Medical Centre at a pressure of 1.5 bar. The operational dive was simulated in the wet pot, where moderate work was done on a underwater ergometer. During the two simulated dives in the dry compartment, air was breathed by mask.

RESULTS

Due to the season, common cold interfered with the interpretation of the data on pulmonary oxygen toxicity. Nevertheless, clear symptoms of pulmonary poisening appeared in the nitrox divers, particularly in group II, but we did not find a decrease in vital capacity or other spirometric data. Also x-rays nor haematological data did show any significant change. Figure 1 shows some of the collected spirometric data. There is a slight rise in vital capacity for all groups, maybe due to a training effect, but definitely no decrease. The bulk of spirometric data, however is stil subject to further analysis.

To our surprise, however, we ran into other signs of oxygen toxicity, except for the control group. Figure 2 displays the frequency of possibly oxygen related symptoms for group I. After one week of little complaints,

	BEFORE		END WEEK I		START WEEK II		END WEEK II	
	VC	FEV1	VC	FEV1	VC	FEV1	VC	FEV1
CONTROL n=6	6.03 (0.50)	5.14 (0.43)	6.20 (0.51)	5.12 (0.41)	6.28 (0.56)	5.18 (0.44)	6.20 (0.45)	5.06 (0.46)
GROUP I n=6	5.85 (0.37)	4.30 (0.41)	6.14 (0.39)	4.57 (0.40)	6.02 (0.38)	4.43 (0.42)	6.22 (0.54)	4.65 (0.46)
GROUP II n=5	5.61 (0.71)	4.45 (0.48)	5.74 (0.73)	4.43 (0.60)	-	-	-	-

Fig. 1. Mean values in litres BTPS for vital capacity and FEV1, with standard deviations in parentheses.

there was a marked fatigue in all divers during the weekend, particularly while climbing stairs or when trying to do exercise like sports. There were also some complaints about mild muscle and joint pains, particularly in arms and legs. In the middle of the second week paraesthesiae started to occur, while at the end of this week all six divers were affected. The paraesthesiae existed of sensations of tingling and numbness in fingers and toes. Fatigue was marked in all.

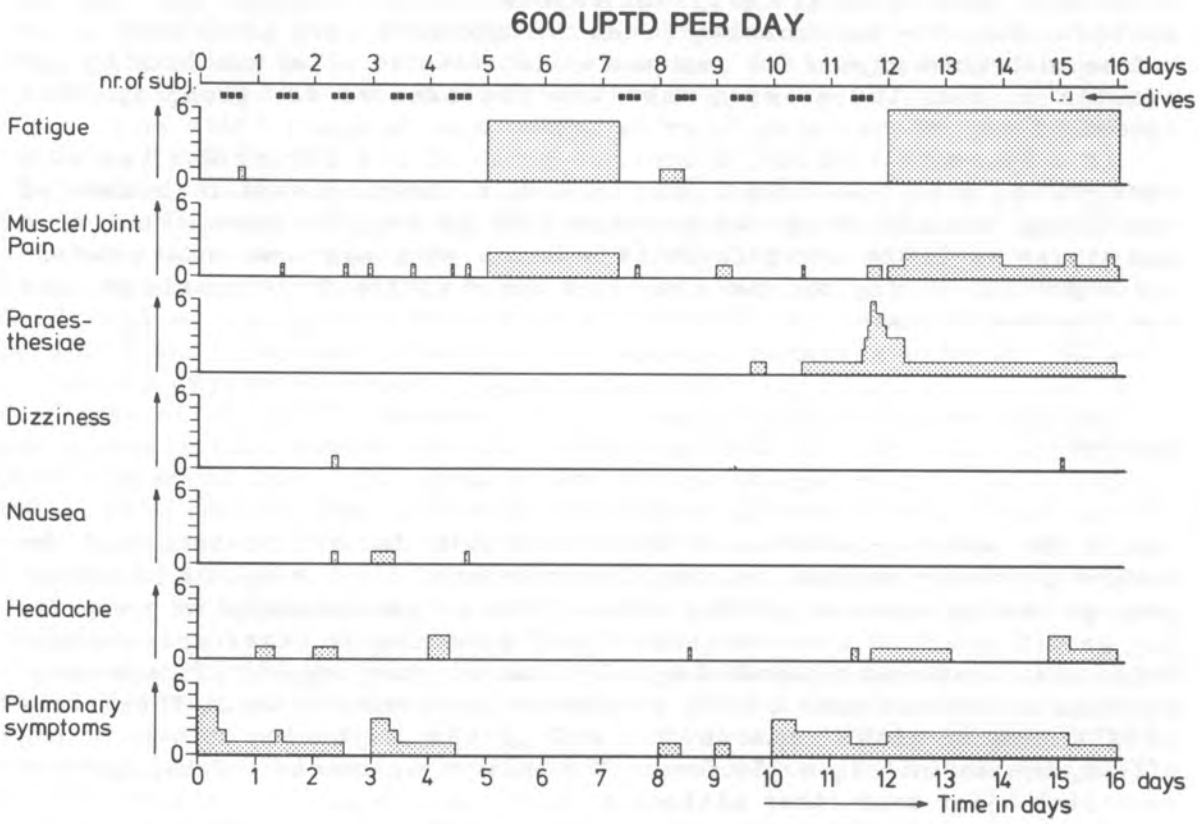

Fig. 2. Frequency of symptoms for group I.

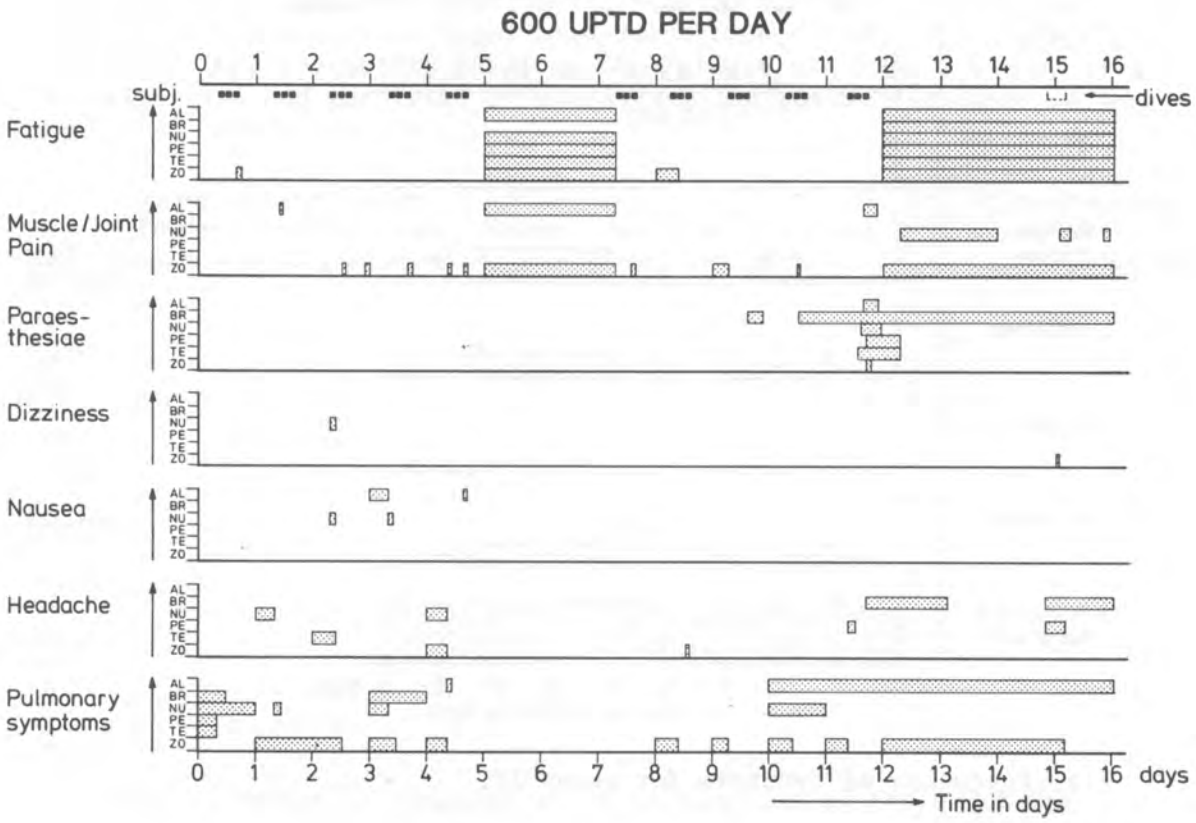

Fig. 3. Distribution of symptoms per diver for group I.

The distribution of the symptoms per diver is shown in figure 3. Remarkable is that the diver (BR), who started first with paraesthesiae, suffered longest. He had great difficulty in the handling of small objects.

The frequency of complaints for group II is depicted in figure 4. Here, paraesthesiae started already on the second day and unusual fatigue was also noted early. Headache and dizziness occurred more than in group I. Also pulmonary symptoms were more prominent, although the picture is clouded by the fact, that four divers started with a slight cold. Nevertheless, pulmonary symptoms became more severe as the experiment continued. On the third day, one diver (VO) had to be sent home because of a bad bronchitis. After day four the symptoms were so alarming, that we decided to bring the exposure down to 600 UPTD on day five. This was done by giving the oxygen sessions at a simulated depth of 4.5 meters. Figure 5 shows the distribution of the symptoms among the divers. Dizziness was very severe in diver VI. He suffered from spontaneous attacks of vertigo of about ten minutes duration, five to ten times per day.

Since we ended up with all divers having more or less serious symptoms of oxygen poisening by the end of the second week, we decided to stop further exposure in the chamber and have them perform only one working dive per day. On the second day of the third week, however, all divers had to be sent home because of persistence of serious disabling symptoms.

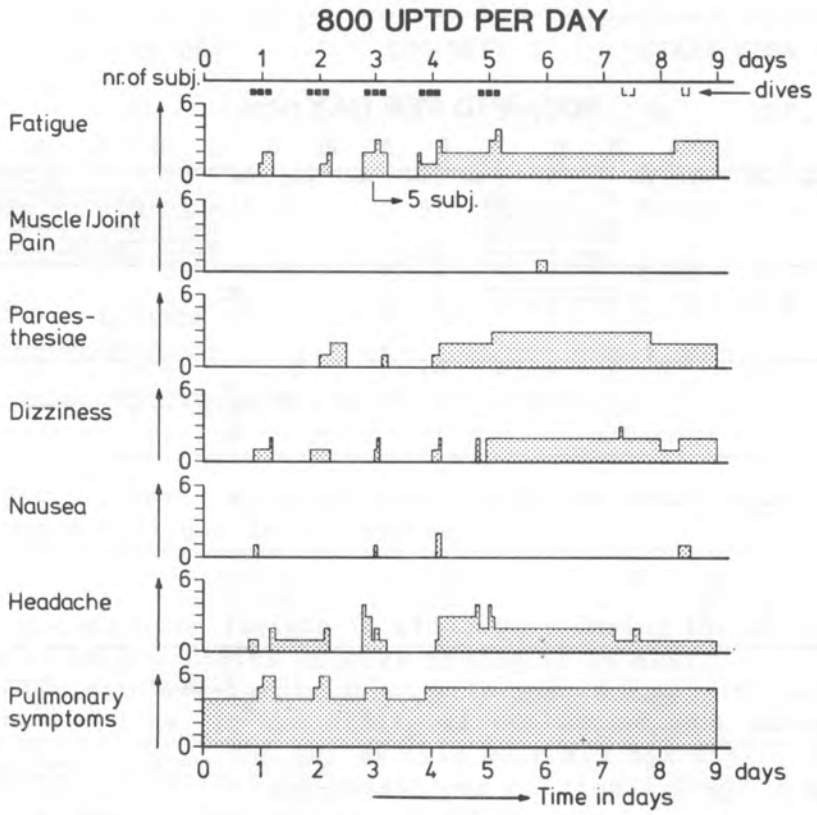

Fig.4 Frequency of symptoms for group II.

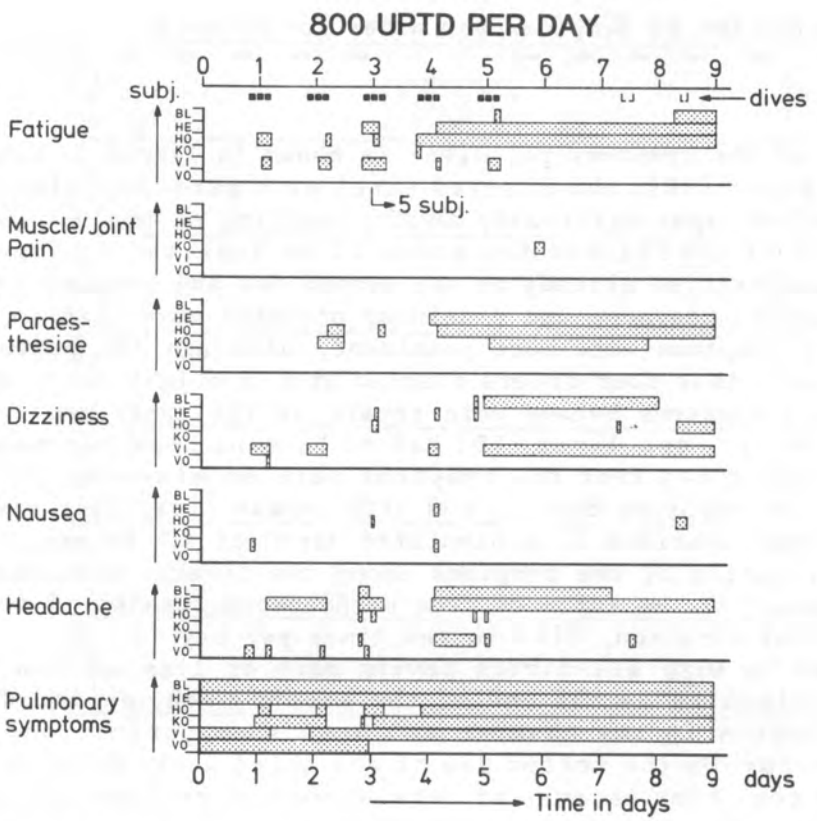

Fig. 5. Distribution of symptoms per diver for group II.

After the third week, all divers were thoroughly examined. As shown in figure 6, four divers of group I had suffered from fatigue, while two had experienced vague muscle or joint pain. One diver had still serious complaints of paraesthesiae, while he also had developed headache and dizziness, expressing as light headedness when bending over, after termination of the experiment. In group II, fatigue was also a marked symptom. Paraesthesiae had persisted in two divers and severe dizziness in one.

	Symptoms 1 week after experiment							
DIVERS	Pulmonary	Headache	Nausea	Dizziness	Paraesthesiae	Muscle/Joint Pain	Fatique	Nr. of days after experiment for all symptoms to disappear
600 UPTD/DAY								
AL	-	-	-	-	-	+	+	7
BR	-	+	-	+	+	-	+	37
NU	-	-	-	-	-	-	+	7
PE	-	-	-	-	-	-	+	7
TE	-	-	-	-	-	-	-	-
ZO	+	-	-	-	-	+	-	?
800 UPTD/DAY								
BL	+	-	-	-	-	-	+	14
HE	-	+	-	-	+	-	+	33
HO	-	+	-	-	+	-	+	26
KO	-	-	-	-	-	-	-	-
VI	-	+	-	+	-	-	+	24
VO	-	-	-	-	-	-	-	-

Fig. 6. Course of symptoms after termination of the experiment.

Neurological examination revealed only disturbed pin point discrimination in the divers with paraesthesiae. EEG and EMG were normal. The vertigo in diver VI seemed to be of vestibular origin; his symptoms disappeared, however, before this could be further evaluated. In both divers complaining about dizziness, the consulted neurologist had the impression of vasovegetative instability since, amongst other symptoms, profuse sweating was noted. As shown in the table, it took quite some time for all symptoms to disappear. Particularly paraesthesiae were very persistent.

DISCUSSION AND CONCLUSIONS

Symptoms like we found in our experiment, are usually regarded as early signs of CNS oxygen toxicity, preceding an insult. Nevertheless, such symptoms have been noted in experiments concerning pulmonary toxicity, after prolonged exposure to oxygen tensions up to 2 bar [Clark (1971b), Comroe et al. (1945), Hendricks et al. (1977), Fisher et al. (1968), Shilling et al. (1976)]. Similar symptoms have also been encountered in SCORE [Miller et al. (1976)] and SHAD [Hamilton et al.(1982)] nitrox saturation dives. They are mostly considered as mild neurological effects of hyperoxia, that disappear within a few hours. In our experiment these symptoms appeared to be very persistent, particularly when exposure continues once signs of oxygen toxicity have developed. We were unable,

though, to find a clear-cut neurological substrate. More research into this is certainly needed.

Remarkable is, that in group I signs of oxygen toxicity became only general at the last working day of the second week. A cumulative effect is apparently present even after long normoxic intervals. This should be borne in mind when hyperoxic mixtures are to be used intensively. It could also have implications for saturation diving, as the possibility exists that long exposure to an oxygen tension of around 0.5 bar sensitizes the subjects to oxygen poisening, when exposed to hyperoxia during excursions or decompression.

Our final conclusion is, that exposure to hyperoxia of 600 UPTD per day or higher, can not be considered as safe, when used on a working week basis at a schedule as in our experiment. To find a safe limit, however, requires more research.

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