Use of oxygen for optimizing decompression

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Berghage, T. E., and T. M. McCracken. 1979. Use of oxygen for optimizing decompression. Undersea Biomed. Res. 6(3): 231–239.—For over 70 years, decompression has been facilitated by the use of elevated oxygen partial pressures. Oxygen has been administered even though little is known about the proper dosage or the way in which this benefit is derived. The historical literature indicates that there is an envelope or narrow range of oxygen partial pressures that can be used. If the oxygen is too low, the incidence of decompression sickness increases; if the oxygen is too high, oxygen poisoning becomes a problem. The present study was designed to explore this oxygen envelope and to define the relationships between oxygen partial pressure, exposure time, and pressure, and to delineate their effects on pressure-reduction limits. To define the ED_{50} (the effective dose that produced signs of decompression sickness in 50% of the animals), we exposed 820 female albino rats to 42 experimental conditions. Results suggest that the optimum oxygen level and the size of the oxygen envelope both depend on the ambient hydrostatic pressure and the exposure time. For short "shallow" exposures, the optimum oxygen level is high and the oxygen envelope is large; for long "deep" exposures, the optimum oxygen level is reduced and the envelope is restricted.

decompression
decompression theory
oxygen decompression
oxygen poisoning

The beneficial effects of breathing oxygen during a decompression have long been recognized. Paul Bert first discussed the topic in 1878; since then numerous studies have demonstrated the effect. By using oxygen before an altitude exposure, the aviation community made early use of the concept. A good review of this literature has been done by Bateman (1951). The construction industry has also made extensive operational use of oxygen. Papers by Nashimoto (Nashimoto 1967; Nashimoto and Mano 1972) have documented the benefits associated with the use of oxygen in decompressing caisson workers. These papers suggest that the benefits can either be in the form of a lower incidence of decompression sickness or a reduction in the required decompression time. Oxygen breathing has also been incorporated in decompression schedules presently used by many commercial and government diving organizations.

This widespread use of oxygen is occurring even though we know very little about the proper dosage or the way in which the benefit is derived. Paul Bert and many workers since have assumed that the beneficial effects of oxygen occur because oxygen replaces the inert gas in the tissues and is metabolized so rapidly that there is no danger of bubble formation during decompression. Von Schrötter (1898) suggested that divers and caisson workers should wash out the nitrogen absorbed during the dive by breathing pure oxygen under pressure for 5 min before decompressing. This was a good idea in theory, but it totally disregarded the toxic effects of oxygen under pressure. Ham and Hill (1905) correctly pointed out that there is a trade-off between oxygen poisoning and decompression and that the use of oxygen has its limitations.

Haldane was the first researcher who attempted to quantify the decompression process and who suggested that the partial pressure of oxygen could be totally disregarded. He proposed that the amount of oxygen in simple solution in the arterial blood will

> . . . increase in proportion to the rise in alveolar oxygen pressure; but as soon as the blood reaches the tissues this extra dissolved oxygen, which (except with exposures to enormous pressures) is only a small part of the total available oxygen in the arterial blood, will be used up, so that in the tissues and venous blood there will be at most only a very slight increase in the partial pressure of oxygen. For practical purposes therefore we need only take into consideration the saturation of the body with nitrogen (Boycott, Damant, and Haldane 1908, p. 345).

It is important to note that Haldane qualified his position by not considering grossly elevated oxygen levels, but it was not clearly stated which oxygen levels he considered high.

Two general concepts have developed out of this historical research:

1) The decompression advantage gained by using oxygen is proportional to the amount of inert gas replaced by oxygen (which is the basis of equivalent air-depth decompression procedures);

2) Oxygen breathing during decompression increases the gradient or partial pressure difference between the tissues and the alveolar air and therefore improves the inert gas elimination process. Behnke (1967) called this later concept the "oxygen window" method of decompression. Both of these ideas are based upon the chemical and physical properties of the gases (Fick's, Graham's, Dalton's, and Henry's Laws). The physiological dynamics of the situation are not taken into account. Few would deny that the body's response to elevated oxygen partial pressures can alter gas exchange and can affect the effectiveness of oxygen.

The present study set out first to describe the oxygen poisoning-decompression trade-off and then to define the envelope of usable oxygen partial pressures. Based upon what is known about oxygen, it seemed reasonable to expect that this trade-off relationship would vary according to exposure time and exposure pressure. Succinctly stated, the objective of this study was to define the relationship between oxygen partial pressure, time, and pressure, and their effects on pressure-reduction tolerance.

METHOD

The subjects for the experiment were 820 female albino rats (NMRI:0[SD]SV), Sprague-Dawley derived. The animals were housed in groups of five. Their mean and standard deviation free-feeding weight at the time of the experiment was 220.65 ± 24.30 g.

All pressure exposures were made in a Bethlehem Model 1836 10-HP chamber with a volume of approximately 170 liters. The chamber atmosphere was monitored with a Beckman Model F-3 paramagnetic oxygen analyzer; the oxygen percentage was maintained within

±0.2%. Carbon dioxide levels were checked and found to be 1% surface equivalent or less. Chamber pressure was monitored with a 0–2000 fsw Heise gauge and maintained within ±3 fsw (comparable to 0.09 ATA). The temperature in the chamber was maintained at 28 ± 2°C during the steady-state portions of the exposures.

During the observation period of each pressure exposure, the rats were exercised at a rate of 5 rpm (3.33 m/min) in a rotating cage. The 5-section cage, 63.5 cm long and 22.4 cm in diameter, is constructed of wire mesh over a Plexiglas frame. Each section is approximately 12 cm wide (Berghage, Woolley, and Keating 1974). The cage is rotated in the chamber by a sparkless, shaded-pole motor (Eberback Corporation, Con Torque 115-V, 60-cycle 1/10-HP).

An augmented 3 × 3 × 4 factorial experimental design was employed in the study; three levels of exposure pressure (10, 25, and 40 ATA), three exposure time intervals (30, 60, and 120 min), and four oxygen partial pressures (0.2, 1.2, 2.2, and 3.2 ATA) were used (Table 1). A fifth oxygen condition (4.2 ATA) was added in several instances to clarify the mathematical relationship between oxygen partial pressure and ED_{50} pressure-reduction values. These three independent variables were evaluated to determine their effect on a single dependent variable: the pressure reduction sufficient to produce manifest symptoms of decompression sickness in 50% of the subjects (the ED_{50}).

An ED_{50} value was determined by decompressing four groups of five animals each to four different observation pressure levels. The observation levels were selected to produce a decompression sickness incidence between zero and 100%. A least-squares best-fit line was applied to the four data points to establish the ED_{50} point.

The reliability of the ED_{50} value has been studied in our laboratory. Indications are that ED_{50} estimates are affected by the exposure pressure: the greater the exposure pressure, the greater the estimation error. The standard deviations of the ED_{50} estimates at the 3 pressures used (10, 25, and 40 ATA) are 0.12, 0.46, and 0.82 atm, respectively.

Procedures for all experimental pressure exposures were:

1. Compress 0,[1] 1, 2, 3, 4, or 5 atm to produce partial pressures of 0.2, 1.2, 2.2, 3.2, and 4.2 ATA.
2. Compress with helium to 10, 25, or 40 ATA at the rate of 1.82 atm/min.
3. Upon reaching the selected exposure pressure, check the oxygen percentage and correct as needed and adjust the temperature to 28°C.
4. Remain at the exposure pressure for 30, 60, or 120 min.
5. In the last 60 s of the bottom time, change the Po_2 to 0.51 ATA at the pressure level to which you are decompressing.
6. Abruptly decompress the animals to the preselected observation pressure level at the rate of 1 atm/s.[2]
7. Remain at the observation pressure with the exercise cage rotating for 20 min. At the end of this time, evaluate the behavior of the animals and classify them as to presence or absence of manifest signs of decompression sickness (DCS).[3]

RESULTS

The ED_{50} values obtained for the various combinations of exposure pressure, oxygen partial pressure, and exposure time are presented in Table 1. Also shown in this table are the number

[1]No compression with oxygen is necessary.

[2]The observation pressure level is varied for each pressure exposure to determine the ED_{50} value.

[3]A 20-min observation period was sufficient to observe 99% of the decompression sickness signs that occurred; these signs included paralysis, convulsions, and tumbling in the cage (Berghage, David, and Dyson 1979).

TABLE 1
EXPERIMENTAL DESIGN MATRIX

Exposure Pressure, ATA	Exposure Time, min		Oxygen Partial Pressure, ATA				
			0.2	*1.2*	*2.2*	*3.2*	*4.2*
		n	20	20	20	15	20
	30	*r*	.95	.85	.99	1.00	.88
		ΔPED_{50}	7.8	7.6	7.9	8.6	8.3
		n	20	15	20	20	20
10	60	*r*	.99	.99	.98	.96	.80
		ΔPED_{50}	7.7	7.9	7.9	7.9	7.8
		n	20	20	20	20	25
	120	*r*	.95	.94	.98	.87	.73
		ΔPED_{50}	7.8	8.1	8.1	8.2	7.1
		n	20	20	20	20	15
	30	*r*	.96	.99	.68	.90	.99
		ΔPED_{50}	13.9	14.7	16.6	17.3	15.9
		n	20	20	20	25	20
25	60	*r*	.96	.99	.99	.76	.94
		ΔPED_{50}	13.6	14.7	15.4	16.0	14.5
		n	20	20	20	20	—
	120	*r*	.94	.95	.96	.99	—
		ΔPED_{50}	13.0	16.0	16.4	14.3	—
		n	20	20	20	20	—
	30	*r*	.95	.98	.94	.98	—
		ΔPED_{50}	22.3	23.4	24.7	23.0	—
		n	20	20	20	20	25
40	60	*r*	.87	.95	.94	.95	.81
		ΔPED_{50}	23.0	23.8	23.6	22.0	17.8
		n	20	15	20	25	—
	120	*r*	.92	.99	.94	.83	—
		ΔPED_{50}	22.4	23.9	22.7	20.1	—

of animals used in each condition and the correlation coefficients for the least-squares best-fit line used to determine the ED_{50} values. These ED_{50} values are further interpreted in Tables 2 and 3 and Figs. 1 and 2.

Figure 1 graphically displays the relationship between the three independent variables, as well as the single dependent measure (pressure reduction sufficient to produce signs of decompression sickness in 50% of the animals). The relationships shown in Fig. 1 are least-squares best-fit polynomials between oxygen partial pressure and ED_{50} pressure-reduction values (raw data are given in Table 1). The polynomial equations and their respective standard errors and correlation coefficients are presented in Table 2. Also shown in Table 2 are the

TABLE 2
BEST-FIT CURVES FOR DETERMINING OPTIMUM OXYGEN MIXTURES

Exposure Pressure, ATA	Exposure Time, min	Least-Squares Best-fit Polynomial	Standard Error of the Estimate, atm	Correlation Coefficient	Optimum Oxygen Partial Pressure, ATA
10	30	$y = 7.64 + .14x + .014x^2$	.35	.79	4.89
10	60	$y = 7.56 + .42x - .079x^2$	.28	.68	2.65
10	120	$y = 7.60 + .72x - .193x^2$	.25	.92	1.86
25	30	$y = 13.09 + 2.42x - .40x^2$	.73	.93	3.03
25	60	$y = 13.08 + 1.98x - .38x^2$	.43	.94	2.61
25	120	$y = 12.10 + 4.77x - 1.28x^2$	.02	.99	1.87
40	30	$y = 21.62 + 2.72x - .70x^2$	.72	.91	1.94
40	60	$y = 22.41 + 2.36x - .81x^2$	.36	.99	1.46
40	120	$y = 21.97 + 2.68x - 1.03x^2$	.29	.99	1.30

y = ED_{50} pressure change in atm; x = oxygen partial pressure.

optimal oxygen levels for each of the nine exposure pressure-time combinations. These oxygen levels are optimal in the sense that they maximize the ED_{50} pressure-reduction values and the extent of the decompression that can be tolerated.

The results shown in Fig. 1 and enumerated in Table 2 indicate that the optimal oxygen level is affected by both the exposure pressure and exposure time. The intercorrelations among these variables are given in Table 3 and the relationships are graphically shown in Fig. 2. In addition to the individual effects of these two variables, it appears that they interact synergistically to affect the optimal oxygen level. The combined effects of exposure pressure and exposure time have been incorporated into a linear model for estimating the optimum oxygen level for rats. The equation is:

$$y = 6.29 - 0.11x_1 - 0.037x_2 + 0.0008x_1x_2 \qquad (1)$$

where y = optimum oxygen level in ATA; x_1 = exposure pressure in ATA; x_2 = exposure time in minutes; SEE = 0.48 atm; $r = 0.94$.

TABLE 3
INTERCORRELATION MATRIX

Variables	1	2	3	4
1. Exposure Pressure (P)	—			
2. Exposure Time (T)	.00	—		
3. P × T	.64	.69	—	
4. Optimum Oxygen Level	−.62	−.60	−.69	—

$(r = .61) = (P < 0.05)$ $(r = .78) = (P < 0.01)$

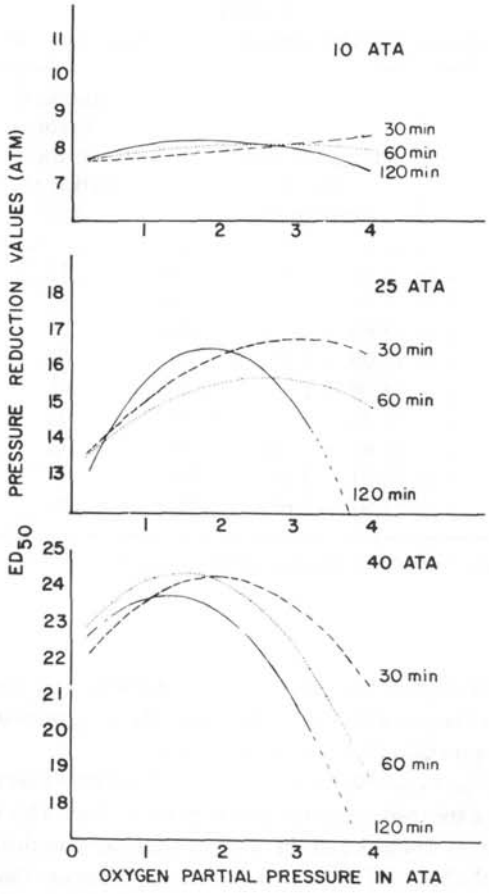

Fig. 1. Relationship between exposure pressure, exposure time, and oxygen partial pressure, and their effects on pressure-reduction tolerance. ED_{50} = pressure reduction that will produce signs of decompression sickness in 50% of the animals.

DISCUSSION

There is ample evidence that oxygen can be used to reduce the incidence of decompression sickness, but there is little in the literature to help in deciding what level of oxygen to use or in determining what other variables need to be considered. Our results support the idea that there is an optimal level of oxygen to be breathed to maximize pressure-reduction tolerance. By increasing the oxygen partial pressure above 0.2 ATA, some initial improvement in decompression is obtained. As the oxygen is further increased, the pressure-reduction tolerance improves to some maximum value and then starts to decrease. The optimal oxygen level appears to be affected by the exposure pressure, the exposure time, and an interaction between the two. The greater the exposure pressure, the lower the optimum oxygen level; the longer the exposure time, the lower the ideal oxygen level. The optimum oxygen level for a long exposure to a high pressure is less than would be expected when the individual effects of the variables are considered together.

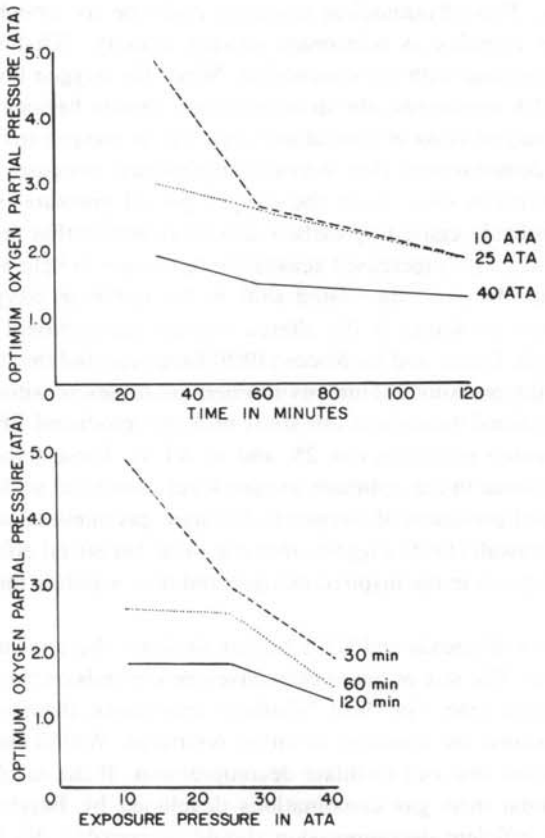

Fig. 2. Changes in optimum oxygen level associated with exposure time and exposure pressure.

This study does not elucidate the mechanisms involved in oxygen's initial beneficial and subsequent detrimental effects. It is assumed that the benefits derived from higher oxygen levels are the result of the two principles previously mentioned, i.e., replacement of the inert gas, and gas-exchange facilitation; however, neither of these concepts can explain the obtained results fully. For both principles, one would expect to find a linear relationship between the amount of oxygen added and the amount of increase in pressure-reduction tolerance. When one examines Fig. 1, it appears that one could expect a linear relationship if exposure pressure and exposure time were held constant. But across experimental conditions, there is no systematic change in pressure-reduction tolerance associated with elevated oxygen.

Some interesting work recently completed by Gruenau and Folker (1978) suggests that one of the beneficial effects of oxygen is its effect on the blood-brain barrier. The presence of vascular gas emboli after decompression increases the permeability of the blood-brain barrier and exposes the brain to blood chemistry changes. By increasing the Po_2, the authors were able to decrease the permeability of the blood-brain barrier and thereby increase survivorship. The relationship between these observed changes and decompression tolerance are not fully understood, but there appears to be an important connection.

The lowered pressure-reduction tolerance associated with high oxygen partial pressures is probably due to some physiological response of the body rather than to the operation of any of

the physical gas laws. This physiological response could be as straightforward as simple vasoconstriction or as complex as pulmonary oxygen toxicity. Whatever the mechanism, there is probably interference with gas elimination. When the oxygen level was raised to 4.2 ATA during the 40-ATA exposures, the decompression results became erratic and on two occasions animals displayed signs of central nervous system oxygen toxicity.

Bennett (1967) has demonstrated that increased hydrostatic pressure reduces the time to onset of oxygen convulsions even when the oxygen partial pressure is held constant. He suggests that this is probably caused by carbon dioxide retention that results from breathing gases of increased density. This increased sensitivity to oxygen in helium at raised pressures may be responsible for the pressure-related shift in the optimum oxygen levels. Another possible explanation can be found in the altered thermal environment associated with increased pressure. Giretti, Rucci, and La Rocca (1969) have reported that the low core temperature produced by a high-pressure helium environment increases sensitivity to oxygen toxicity. The 28 ± 2°C maintained throughout this study probably produced different core temperatures at the three exposure pressures (10, 25, and 40 ATA). These temperature differences may help explain the change in the optimum oxygen level associated with exposure pressure.

The use of high partial pressures of oxygen to facilitate gas elimination may not be necessary. The study by Kindwall (1975) suggests that the same beneficial effects can be obtained by alternating the inert gases in the inspired mixture and thus avoiding the problem of oxygen toxicity.

Between the extremes of anoxia and CNS oxygen toxicity, there is an envelope of usable oxygen partial pressures. The size of the oxygen envelope depends on the ambient hydrostatic pressure and the exposure time. For short "shallow" exposures, the envelope is quite large; for long "deep" exposures, the envelope is rather restricted. Within each oxygen envelope there is an optimum level that can facilitate decompression. If the results of this study are coupled with the optimal inert gas combinations developed by Berghage, Donelson, and Gomez (1978), a more efficient decompression should be possible. By keeping the oxygen close to optimal but below toxic levels, and by facilitating gas elimination through the careful shifting of inert gases, it may be possible to expedite the decompression process.

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The experiments reported herein were conducted according to the principles set forth in the "Guide for the Care and Use of Laboratory Animals," Institute of Laboratory Resources, National Research Council, DHEW, Pub. No. (NIH) 78-23.

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Berghage, T. E., and T. M. McCracken. 1979. L'usage de l'oxygène pour l'optimisation de la décompression. Undersea Biomed. Res. 6(3):231–239.—Pendant plus que soixante-dix ans, la décompression a été facilitée par l'usage des pressions partiales d'oxygène élevé. L'oxygène a été administré même si peu est connu du dosage correct ou de la façon de laquelle ce bienfait est derivé. La littérature historique indique qu'il y a une enveloppe ou une portée étroite des pressions partiales d'oxygène qui peut être utiliser. Si le niveau d'oxygène est déficient, l'incidence de la maladie de décompression augmente; si le niveau d'oxygène est excessif, l'empoisonnement d'oxygène devient un problème. Cette étude a été projetée pour explorer cette enveloppe d'oxygène et pour determiner qui sont les relations entre les pressions partiales d'oxygène, la durée de l'éxposition, et la pression, et aussi pour tracer leur effets sur les limites du réduction de pression. Pour determiner le DE_{50} (le dosage effectif qui a produit des indications de la maladie de

décompression chez 50% des animaux), nous avons éxposé 820 rates albinoes aux 42 conditions experimentales. Les résultats suggèrent que le niveau optimum d'oxygène et la taille de cette enveloppe dependent, tous les deux, de la pression ambiente hydrostatique et de la durée de l'éxposition. Pour les éxpositions courtes "superficielles," le niveau optimum d'oxygène est haut et l'enveloppe est grande; pour les éxpositions prolongées "profondes," le niveau optimum d'oxygène est diminué et la taille de l'enveloppe est reduite.

décompression
théorie de décompression
décompression d'oxygène
empoisonnement d'oxygène

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