Residual oxygen time model for oxygen partial pressure near 130 kPa (1.3 atm)

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ABSTRACT

A two-part residual oxygen time model predicts the probability of detectible pulmonary oxygen toxicity P(P[O_2tox]) after dives with oxygen partial pressure (PO_2) approximately 130 kPa, and provides a tool to plan dive series with selected risk of P[O_2tox]. Data suggest that pulmonary oxygen injury at this PO_2 is additive between dives. Recovery begins after a delay and continues during any following dive.

A logistic relation expresses P(P[O_2tox]) as a function of dive duration (T_{dur}) [hours]:

$$P(P[O_2tox]) = 100 / [1+exp\ (3.586–0.49 \cdot T_{dur})]$$

This expression maps T_{dur} to P(P[O_2tox]) or, in the linear mid-portion of the curve, P(P[O_2tox]) usefully to T_{dur}. For multiple dives or during recovery, it maps to an equivalent dive duration, T_{eq}.

T_{eq} was found after second dives of duration $T_{dur\ 2}$. Residual time from the first dive $t_r = T_{eq} – T_{dur\ 2}$. With known t_r, t and T_{dur} a recovery model was fitted.

$$t_r = T_{dur} \cdot exp\ [–k \cdot ((\boldsymbol{t}–5)/T_{dur})^2],$$ where

$\boldsymbol{t} = t – 5$ hours, k = 0.149 for resting, and 0.047 for exercising divers, and t represents time after surfacing. The fits were assessed for 1,352 man-dives. Standard deviations of the residuals were 8.5% and 18.3% probability for resting or exercise dives, respectively.

INTRODUCTION

The rebreather underwater breathing apparatus (UBA) is a self-contained breathing device that allows long-duration, bubble-free dives: Exhaled gas is inhaled again after carbon dioxide has been removed and oxygen has been added as needed. Some rebreather UBAs provide 100% oxygen, in part, to prevent inert gas partial pressure from increasing in body tissues, and thus, to eliminate the need for decompression time. This, unfortunately, is accompanied by a significant risk of central nervous system (CNS) oxygen toxicity at depths greater than about 6 meters of sea water; CNS oxygen tolerance is markedly lower in submerged subjects than in those in a dry environment [1]. Other rebreather UBAs control the partial pressure of oxygen (PO_2) at a constant value (a set point) for a range of depths to manage both CNS oxygen toxicity risk and decompression time.

The U.S. Navy has chosen PO_2 = 1.3 atm (130 kPa) as a set point at which evidence suggests that CNS oxygen toxicity is unlikely [2], and has developed appropriate decompression tables. However, long and repeated dives at PO_2 = 130 kPa introduce an additional risk – that of pulmonary oxygen toxicity (P[O_2tox]).

Two separate questions arise in planning repeated dives with PO_2 = 130 kPa:

(a) "What is the risk that signs or symptoms of pulmonary oxygen toxicity will be evident after an exposure?" and

(b) "How long after any exposure will pulmonary toxic effects, detectable or not, be additive with those of a subsequent dive?"

The first question can be addressed experimentally for single dives, but the two questions are interlinked when dives are repeated. To test exclusively for pulmonary effects or to measure them in conjunction with other testing, the U.S. Navy Experimental Diving Unit (NEDU) has performed 1,352 Ethics Committee-approved in-water man-dives with PO_2 = 130kPa to 140 kPa [3-15], for more than 5,500 man-hours of diving with PO_2 = 130 – 140 kPa. Despite the large numbers, the dives have covered very few combina-

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tions of dive duration and recovery time. Accordingly, we have addressed the questions of risk and recovery time by developing a residual oxygen time model (ROT). Previously published model formats based on data from NEDU and elsewhere [16-18] served as the starting point.

Any dive with PO_2 of 130 kPa to 140 kPa is assumed to begin a process in which pulmonary injury is proportional to exposure time. (The justification for this assumption is presented in "METHODS" below.) Symptoms or changes in pulmonary function, specifically in flow – volume parameters (ΔFV) indicate detectable pulmonary oxygen toxicity, but pulmonary oxygen toxicity can be present without signs or symptoms.

The probability of overt pulmonary oxygen toxicity is extremely low when oxygen injury is slight, close to unity if the injury is severe, and approximately proportional to injury for intermediate exposures. Incidence of symptoms and of ΔFV in the mid-range can thus be mapped to the exposure duration (T_{dur}). The incidence of symptoms or signs after a composite exposure to or during recovery from exposure to PO_2 = 130 kPa also can be mapped to an equivalent oxygen time (T_{eq}) defined as the single exposure duration with the same probability of pulmonary function changes or of symptoms.

Recovery from injury begins sometime after the start of exposure. Injury heals, symptoms or signs are resolved, and equivalent oxygen time decreases. The equivalent time remaining during recovery is the residual oxygen time (t_r). Later dives are assumed to add to any residual effects; after a second dive of duration $T_{dur\,2}$, $T_{eq} = t_r + T_{dur\,2}$.

In the development of ROT, t_r during recovery from exposures to PO_2 = 130 – 140 kPa was expressed as a function of recovery time and dive duration. Model parameters were determined from residual times fitted to incidence data in the range where incidence is proportional to T_{eq}, specifically, to data collected immediately after pairs of three- to six-hour dives [4, 6-8, 10-13] or single seven- to eight-hour dives [3, 5, 9, 15]. Resting and exercise exposures were fitted separately because experience shows a greater accumulation of pulmonary oxygen toxicity after repeated dives involving exercise than after repeated resting dives [8]. Several models were proposed, fitted to dive data and compared. This paper describes the development, testing of proposed models, and proposed use of the residual oxygen time model. In the description that follows, the term "dive" refers to similar exposures of multiple people.

METHODS

Injury proportional to exposure time

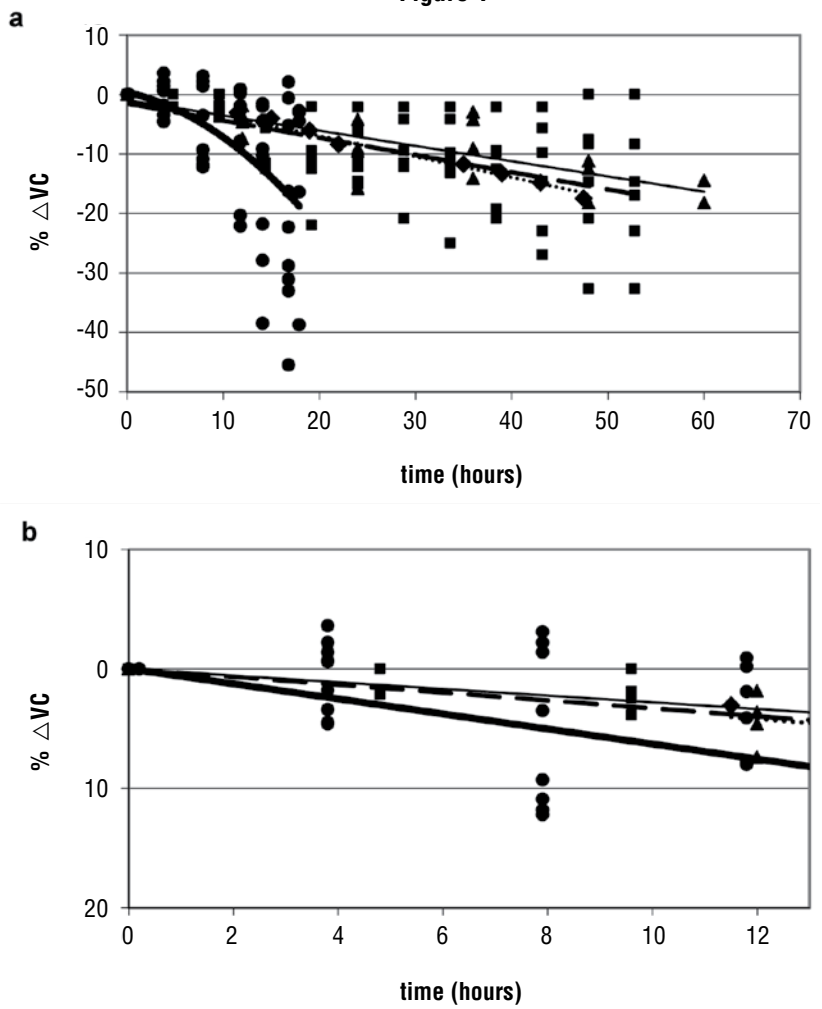
Pulmonary data collected during dry dives can be applied to in-water dives; the incidence and severity of reported symptoms and ΔFV indices were indistinguishable in 34 subjects after in-water or dry exposures to PO_2 = 160 kPa for 360 minutes [19]. (Data related to exposures to PO_2 = 160 kPa were not used to fit the model for PO_2 = 130 kPa.) The time course of changes in vital capacity (%ΔVC) during dry hyperbaric chamber exposures to several different elevated levels of PO_2 have been published by other investigators [20–23], and %ΔVC was considered to typify pulmonary oxygen injury.

The functional form of injury onset is highly dependent on exposure PO_2 [17). A quadratic-type relation with time holds for %ΔVC when $150 \le PO_2 \le 250$ kPa, [17,18], but curvilinearity at 150 kPa is slight until exposure duration is longer than about 12 hours (Figure 1). Based on exposures to PO_2 = 85 [20], 94 [21], 106 kPa [22] and 150 kPa [23] (Figure 1), a linear injury onset was chosen as representative for PO_2 = 130 to 140 kPa for dives with T_{dur} of up to 12 hours. Thus, this model is not applicable to higher PO_2 or to exposure times longer than 12 hours. Note that a linear injury onset has previously been proposed without limitation on PO_2 or dive duration [24].

Probability of pulmonary oxygen toxicity

The focus of ROT is the probability of detectable pulmonary oxygen toxicity (P[O_2tox]). The presence of one or more symptoms or flow-volume deficits (ΔFV) after a dive was the indication considered. A flow-volume deficit was defined as a parameter value depressed from baseline beyond the 95% confidence bounds of day-to-day, non-diving variability previously measured at NEDU [3]. Specifically, decreases from baseline were called deficits if the average of three consistent measurements was below baseline (a three- to six-value average) by more than 7.7% for forced

Figure 1



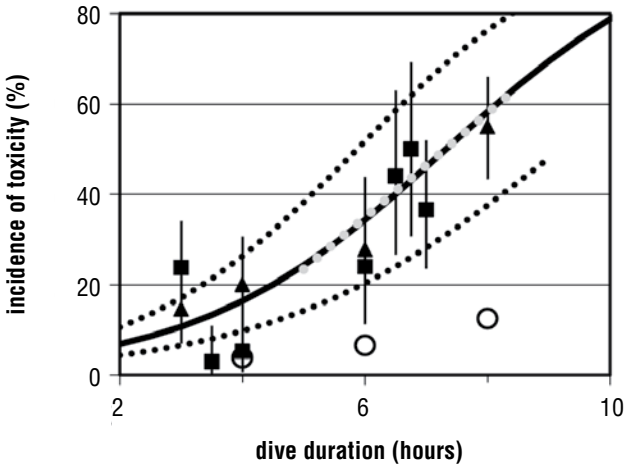
Changes from baseline in vital capacity as functions of time during hyperbaric chamber exposure, PO_2 85, 94, 106 and 150 kPa, individuals (symbols) and aggregate (regression lines). **Panel a:** all data available. **Panel b:** the first 12 hours.

PO_2 = 85 kPa [20]: I ■ and – – – ; PO_2 = 106 kPa [22]: ♦ and …… ;

PO_2 = 94 kPa [21]: ▲ and ——— ; PO_2 = 150 kPa [23]: ● and ━━━.

vital capacity (FVC), 8.4% for forced expiratory volume in one second (FEV_1), or 17% for forced expiratory flow from 25% to 75% of vital capacity $(FEF)_{25-75}$. Because three different indices were considered, each at the 95% confidence level, the probability that a pulmonary function decrease was falsely identified is approximately 15%; a false negative was considered to be a greater concern than a false positive. The symptoms included were inspiratory burning, cough, chest tightness and dyspnea. The estimate of $P[O_2tox]$ was the incidence – that is, the number of divers with symptoms and/or ΔFV – divided by the number of divers exposed. When different dive series provided information for the same dive profile, the incidences were considered to be the number of divers in all similar series who reported symptoms or showed ΔFV divided by the total number of divers exposed to the condition. The assumption that the populations could be pooled, that is, that the same binomial distribution applied for all groups, was tested using Tarone's Z [25].

Figure 2



Incidence-time model for any detectable pulmonary oxygen toxicity. The incidence of pulmonary oxygen toxicity at PO_2 = 130-140 kPa is presented as a function of single dive duration T_{dur}. Rest: ▲; exercise: ■, means and Agresti-Coull binomial 95% confidence intervals. Air dive: **O**. Air dive error bars are omitted for clarity, but confidence intervals are tabulated here.

Confidence intervals on incidences of evident pulmonary oxygen toxicity after dives with PO_2 approximately 30 kPa

duration	lower 95th CI	upper 95th CI
4 hours	0.92	11.3
6 hours	3.49	11.8
8 hours	0.50	49.5

The solid line represents the logistic regression, and the dashed lines show one standard error on the fitted parameters. The broken portion of the regression curve is the linear range from which T_{eq} was read from incidence to permit the determination of recovery parameters.

The incidences recorded after single dives are shown as a function of dive duration in Figure 2. The error bars are the Agresti-Coull binomial 95% confidence intervals on the measured values:

$$95\text{th \% CI} = \tilde{p} \pm 1.96 \cdot (\tilde{p}^{0.5}) \cdot (1-\tilde{p})^{0.5} \cdot \tilde{n}^{-0.5} \quad (1)$$

where $\tilde{p} = (X + 1.96) / \tilde{n}$, X = number of divers with $P[O_2tox]$/ total number of divers, and $\tilde{n} = n + 1.96^2$; similar in form to, but more reliable than, the commonly used Walsh formulation for binomial distributions [26]. They indicate the ranges of the true values of $P[O_2tox]$ for each dive duration.

Incidence of pulmonary oxygen toxicity as a function of exposure time (incidence – time model)

Logistic regression was used to determine the best fit relation between T_{dur} and the binomial measured incidences of pulmonary oxygen toxicity. The fitted curve (Equation 5, RESULTS) is shown with the data in Figure 2.

Resting and exercise dives were combined in the incidence-exposure time model; although the effects of repeated dives with exercise differ from those of repeated dives at rest [8], within two hours after surfacing, incidences after single four-hour resting dives were statistically indistinguishable from those after single four-hour dives with exercise [8]. Data were available from resting dives (3-7, 9, 12, unpublished]; from dives with alternating 30-minute periods of rest and moderate cycle ergometer exercise [8, 10, 11, 13, unpublished]; and from dives with some periods of weight-lifting exercise [14]. Moderate cycle ergo-meter exercise was defined as work sufficient for subjects to maintain heart rates of 90 to 110 beats/minute. Incidences after a total of 529 man-dives in eight distinct exposures were used to construct the incidence-time model; the number of dives for each condition is listed in Table 1.

Pulmonary signs and symptoms following open-circuit air dives were also available for four-, six- and eight-hour resting dives and four- and six-hour dives with exercise (Table 1). The air dives, with PO_2 approximately 30 kPa were identical to the open-circuit dives with 100% oxygen in all ways except for the breathing gas. They thus give the incidence of signs and symptoms that can largely be ascribed to effects of breathing underwater from a low-resistance demand regulator with approximately -30 cm H_2O of static load.

Recovery model parameter fitting

Recovery of vital capacity after exposure to PO_2 ranging from 150 kPa to 250 kPa has been seen to be exponential (16-18, 23, 24). Accordingly, the decrease in t_r during recovery was formulated as

$$t_r = T_{dur} \cdot \exp\,[-f(\boldsymbol{t}, T_{dur})], \quad \boldsymbol{t} > 0$$
$$t_r = T_{dur}, \quad \boldsymbol{t} \le 0 \quad (2)$$

where $\boldsymbol{t} = t - a$, a is delay, $\boldsymbol{t} = t - a$, t is the recovery time (t = 0 at the end of the exposure) and $f(\boldsymbol{t}, T_{dur})$ means a function of $\boldsymbol{t}$ and T_{dur}.

To fit the function $f(\boldsymbol{t}, T_{dur})$, values of t, a, T_{dur}, and corresponding t_r were needed. In principle, t_r corresponding to any incidence of detectable pulmonary

Table 1: Sources for single-dive data

A. "OXYGEN" : PO_2 = 130 to 140 kPa

dive duration	n (person dives)	breathing conditions	references
4 hours, rest	75	open circuit, test pool	3, 4, 7, 12
6 hours, rest	72	open circuit, test pool	3, 6, 12, u
8 hours, rest	71	n=23: open circuit, test pool n=31: open circuit, OSF n=17: set-point rebreather, tower	3, 5, 9
3 hours, exercise	38	n=23: open circuit n=15: O_2 rebreather	10, 11
3.5 hours, exercise	68	open circuit, test pool	15
4 hours, exercise	84	n=68 open circuit n=23: O_2 rebreather both: test pool	8, 11, 13
6 hours, exercise	25	open circuit, test pool	u
6, 6.5, and 7 hours, some exercise	88	set-point rebreather, O_2 rebreather at rest, OSF	14

B. AIR: PO_2 approximately 30 kPa

dive duration	n (person dives)	breathing conditions	references
4 hours, rest	8	open circuit, test pool	3
6 hours, rest	70	open circuit, test pool	3, u
8 hours, rest	8	open circuit, test pool	3
4 hours, exercise	70	open circuit, test pool	u
6 hours, exercise	83	open circuit, test pool	u

Reference "u" means "unpublished."

For single dives with oxygen, the counts include only single dives and the first of series of dives. With air, the counts comprise both single dives and dives repeated for up to five days with surface intervals of 18 hours (six-hour dives) or 20 hours (four-hour dives).

"Open circuit" dives used the MK 20 (Ocenco, Pleasant Prairie Wisconsin, U.S.) surface-supplied with humidified 100% O_2 or air. The "set-point rebreather" was the MK 16 Mod 1 (Carleton Technologies, Buffalo New York, U.S.) which controls at PO_2 = 130 kPa (1.3 atm) but overshoots somewhat on descent. The "O_2 rebreather" was the MK 25 (Lar V) (Draeger USA, Pittsburgh, Pennsylsvania, U.S.).

The test pool was 15 feet (4.6 m) deep. Divers at rest sat in chairs for a chest depth about 12 feet (3.7 meters), but sometimes moved about or even lay on the bottom to relieve boredom. Divers exercising were semi-prone on cycle ergometers, with chest depth similar to that when seated. They cycled against a brake load that kept heart rates between 95 and 110 beats/minute. Work periods of 30 minutes were alternated with 30-minute rest periods.

"Tower" refers to a 50-foot-deep training tower. For the 8-hour dives with the set-point rebreather, divers ascended once in the middle of the dive for a rig change-out.

"OSF" is the ocean simulation facility (OSF), a pressure chamber with a large wet-pot. The 6-, 6.5- and 7-hour dives included unknown PO_2 transients with change of depth.

For the "O_2 rebreather" dives, although depth was approximately constant during each dive, PO_2 varied because of nitrogen in divers' lungs at the start and tissue nitrogen washout during the dive. Depth and exercise in the 4-hour dives matched that for the open-circuit dives, and purge procedures gave at least 90% oxygen at the beginning of dives. Divers on ergometers were thus at PO_2 initially at least 1.3 atm. Purge procedures for the 6- to 7-hour dives gave about 80% O_2 and divers were at 20 fsw, for PO_2 initially 130 kPa.

Table 2: Dives used to find parameters of recovery

REST dive duration	n (person dives)	breathing conditions	references
3 hrs, SI = 2 hrs	12	open circuit, test pool	6, 12
3 hrs, SI = 4 hrs	24	open circuit, test pool	6, 12
3 hrs, SI = 6 hrs	12	open circuit, test pool	6, 12 r
4 hours, SI = 20 hrs	49	open circuit, test pool	4, 7, 12
6 hours, SI = 18 hrs	40	open circuit, test pool	6, 12 , u
6 hrs, SI = 42 hrs	14	open circuit, test pool	6, 12
6 hours, exercise	25	open circuit, test pool	u
8 hours, one dive	71	n=23: open circuit, test pool n=31: open-circuit OSF n=17: set-point rebreather, tower	3, 5, 9
EXERCISE **dive duration**	**n** **(person dives)**	**breathing conditions**	**references**
3 hrs, SI = 4 hrs	25	n=12: Open circuit, test pool n=13: O2 rebreather, test pool	11
4 hrs, SI = 15 hrs	28	open circuit, test pool	8, 13
4 hrs, SI = 20 hrs	16	open circuit, test pool	8, 13
6 hrs, SI = 18 hrs	12	open circuit, test pool	u
6 hrs, one dive	13	open circuit, test pool	u
6, 6.5, and 7 hours, some exercise, one dive	73	set-point rebreather, then O_2 rebreather only at rest, OSF	14

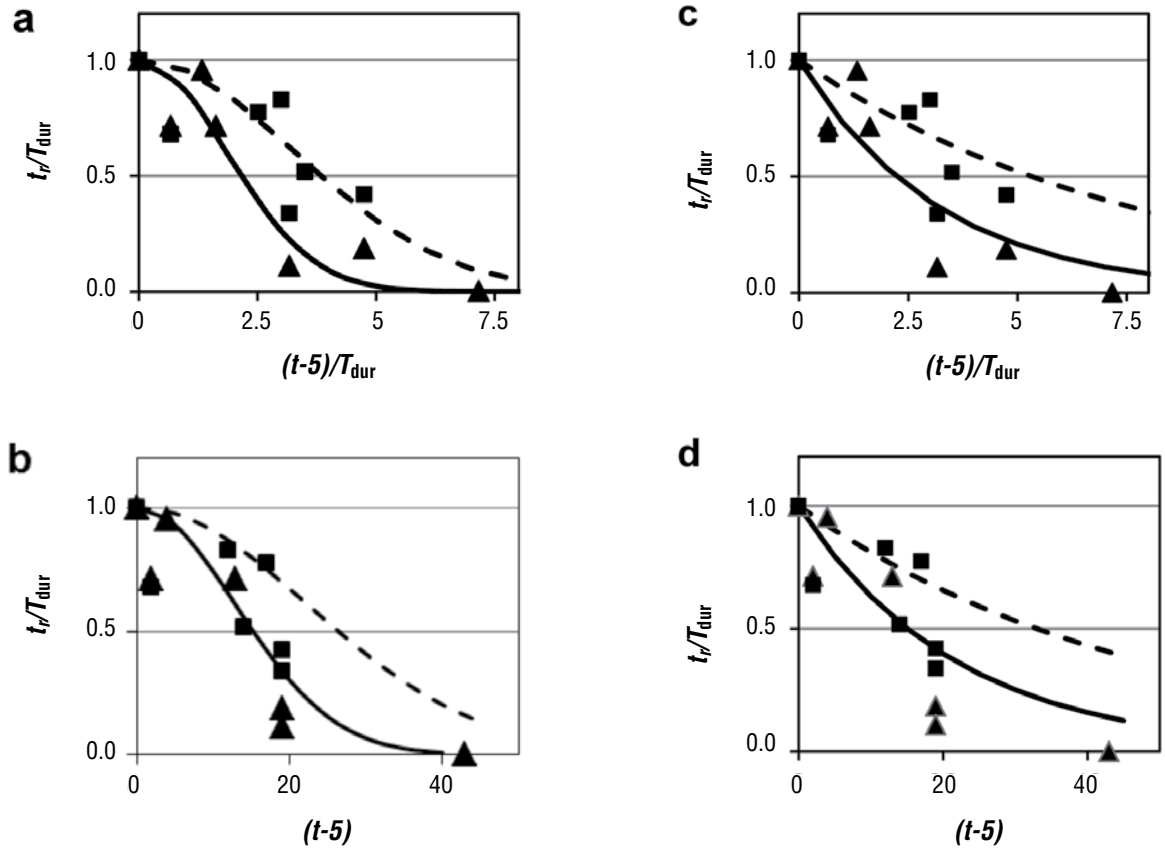
Dive conditions are listed under Table 1. Reference "u" means "unpublished."

oxygen toxicity can be read from the incidence-time graph (Figure 2), but with varying certainty depending on the value. In practice, incidences less than about 20% fall where the graph has low slope, thus where the inverse function is steep enough that a small difference in assessment of pulmonary oxygen toxicity causes a very large difference in estimated T_{eq}. Only in the mid-portion of the logistic regression is there a distinct one-to-one mapping of incidence of pulmonary oxygen toxicity to exposure or equivalent time (inverse mapping as the graph is presented). Thus only in that range of relatively high incidence could values of T_{eq} be selected for fitting of recovery functions. The incidence of pulmonary oxygen toxicity during recovery after most single dives lay outside of the usable range.

To circumvent this problem, pairs of dives grouped by duration and surface interval were used to provide most of the recovery the data. For inclusion in the calibration set, the incidence of toxicity after the second dive was required to be between approximately 25% and 75%, for T_{eq} greater than about five hours. The value of t_r was found as $T_{eq} - T_{dur\,2}$ with no restrictions on the magnitude of t_r except that it had to be greater than zero. Thus, the use of dive pairs allowed t_r after the most common, low-incidence dives to be represented in the recovery model as long as the combined T_{eq} was sufficiently high.

T_{dur} for the first dive was known, as was the pertinent recovery time t, the time between the end of the first dive and the measurement session after the second

Figure 3



Values of t_r/T_{dur} used in parameter fitting for the recovery models. The symbols indicate values obtained from the incidence of pulmonary oxygen toxicity.

After resting dives: ▲; after dives with exercise: ■.

The lines are the best fit single-parameter models fitted to the data: —— for resting dives; – – – for dives with exercise.

Panels a) & **b):** sigmoidal recovery (exponent a function of t^2).

Panels c) & **d):** exponential recovery (exponent a function of t).

a) t_r/T_{dur} plotted against t/T_{dur}, with lines $t_r/T_{dur} = \exp[-k\,(t/T_{dur})^2]$, k = 0.149, 0.047;

b) t_r/T_{dur} plotted against t, with lines $t_r/T_{dur} = \exp(-h\,t)2$, h = 0.003, 0.002;

c) t_r/T_{dur} plotted against t/T_{dur}, with lines $t_r/T_{dur} = \exp(-a\,t/T_{dur})$, a = 0.313, 0.179; and

d) t_r/T_{dur} plotted against t, with lines $t_r/T_{dur} = \exp(-b\,t)$, b = 0.046, 0.028.

Curves shown in Panel a) indicate the data fit selected.

dive, in other words, the sum of the surface interval and the duration of the second dive. (After the second dive of a pair of dives, t_r from the first dive was calculated at the end of the second dive.) Recovery from the first dive continued during the second dive and also continued for t_r smaller than the values on the linear portion of the incidence-time curves.

From a total of 620 man-dives, seven combinations of dive duration, recovery time and t_r were available for parameter-fitting after resting dives and six after dives with exercise (Table 2). For fits of the exercise data, an extra value, $t_r = T_{dur}$ when $t = 0$, was included to anchor the start of the fit.

Two forms of the recovery function $f(t, T_{dur})$ for Equation 2, were considered, one to yield exponential recovery:

$$f(t, T_{dur}) = -(c + g / T_{dur}) \cdot t \qquad (3)$$

and, after inspection of the data (Figure 3), one that gives sigmoidal recovery:

$$f(t, T_{dur}) = -[(h \cdot t^2 + k \cdot (t / T_{dur})^2]. \qquad (4)$$

A set of six models, specifically, three exponential (Equation 3) and three sigmoidal formats (Equation 4), in each case with dependence on t only, on t/T_{dur} only, or on both t and t/T_{dur}, was considered for each of rest and exercise. Figure 3 shows the data and functions of either t or t/T_{dur} for each of Equations 3 and 4.

Although the incidence data are binomial, they are transformed to the continuous variable t_r by inverting the logisitic regression equation. The probability distribution of t_r is inverse logit, but logit and probit functions are similar in their middle (approximately linear) ranges, the portion of the curve from which the t_r data were drawn for parameter fitting. The inverse of the probit function is the normal distribution. To confirm that the selected values of t_r could be assumed to be normal variates, quartile plots were inspected. They showed no significant deviations from a normal distribution.

For normally distributed errors, weighted non-linear least squared error fitting is equivalent to maximum likelihood estimation [27]. For fitting, each value of t_r was weighted by its inverse variance multiplied by the number of dives for the specific condition and divided by the total number of dives. (The extra starting point exercise datum was arbitrary weighted one.) The variance for each value of t_r was estimated as the square of the corresponding standard deviation of the logistic regression.

Parameters of Equation 2 with the recovery function given by Equations 3 or 4 were fitted by non-linear regression using the Gauss-Newton algorithm (SYSTAT10, SPSS Inc., Chicago, Illinois, U.S.).

For rest and exercise each, the six fitted models were compared using Akaike's Information Criteria with small-sample bias adjustment (AICc) [28], and the model with highest Akaike weight was selected. Fits of the selected recovery models for rest and exercise were then examined in more detail using all available data. For each dive in the data set t_r was calculated, values of T_{eq} were computed from t_r and T_{dur} as appropriate, and the incidence-time model was used to translate from the continuous T_{eq} value to the binomial predicted incidence. Predicted and measured incidence values were then compared.

RESULTS

Probability distribution of proportions

None of the combined data sets showed dispersion significantly greater than that expected for a binomial. The combination of results for similar dive trials was justified.

Incidence-exposure time model

The logistic regression yielded

$$P(P[O_2\text{tox}]\ (\%) = 100 / [1+\exp(\ 3.586–0.490 \cdot T_{dur})] \quad (5)$$

for any symptoms or ΔFV measured within two hours of surfacing. The regression fit is shown with the data in Figure 2. The dotted curves indicate ±1 standard error for each parameter. The incidence-time model predicts that with $T_{dur} = 0$, that is, that with no exposure to elevated PO_2, symptoms or signs normally considered to represent pulmonary oxygen toxicity will be apparent in 2.7% of divers. The 95% confidence limits for this extrapolation to an exposure of zero length extend from 0.8% to 4.9%.

Air dives

After a total of 239 shallow air dives (PO_2 = 30 kPa) with rest and with exercise, of durations four, six and eight hours, (unpublished data) overall incidence of symptoms or ΔFV immediately after diving was 5.9%. With PO_2 approximately 30 kPa, there was no significant effect of duration from four to eight hours (Figure 2).

Recovery model parameter fitting

Data for estimation of the delay were thin. The value of five hours was chosen somewhat arbitrarily, with the choice motivated by the results after two three-hour dives separated by a two-hour surface interval. For resting data, r^2 between fitted t_r and estimated t_r (the square of the correlation coefficient) increases as delay is increased from zero to five hours but changes very little between five and 5.5 hours. Fitted parameters for all of the candidate models with delay a = 5 hours are listed in the Appendix (Table A1). The $(\boldsymbol{t}/T_{dur})^2$ models were chosen for both rest and exercise (Appendix, Table A2):

$$\text{resting } t_r = T_{dur} \cdot \exp\,[– 0.149 \cdot (\boldsymbol{t}/T_{dur})^2],\ r^2=0.90 \quad (6);$$

and

$$\text{exercise } t_r = T_{dur} \cdot \exp\,[–0.047 \cdot (\boldsymbol{t}/T_{dur})^2],\ r^2=0.59; \quad (7)$$

where r^2 is the square of the correlation coefficient between fitted t_r and estimated t_r, $\boldsymbol{t} = t – 5$ hours, and "resting" or "exercise" refer to the conditions of exposure, not of recovery time. (Activity levels during surface intervals were neither controlled nor monitored.)

To calculate recovery times necessary to reduce t_r to a preselected value, the equations were rearranged. After resting exposures, recovery time $\boldsymbol{t}$ for a chosen t_r is given by

Table 3: Sample calculations of recovery times

A. Resting dives (times in fractional hours and hours:minutes)

1	2	3	4	5
t_r/T_{dur}	$A=\boldsymbol{t}/T_{dur}$	If T_{dur} =	$t = A\ T_{dur} + 5$	SI to add t_r to 2nd dive
	($\boldsymbol{t} = t-5$)		[decimal hours] hours after surfacing	$t - T_{dur\,2}$
0.5	2.16	2	[9.32] 9:19	7:19
		4	[13.64] 13:38	9:38
		6	[17.96] 17:58	11:58
0.25	3.05	2	[11.10] 11:06	11:06
		4	[17.20] 17:12	13:12
		6	[23.30] 23:18	17:18

B) Dives with exercise (times in fractional hours and hours:minutes)

1	2	3	4	5
t_r/T_{dur}	$A=\boldsymbol{t}/T_{dur}$	If T_{dur} =	$t = A\ T_{dur} + 5$	SI to add t_r to 2nd dive
0.5	3.84	2	[12.68] 12:41	10:41
		4	[20.36] 20:22	16:22
		6	[28.04] 28:02	22:02
0.25	5.43	2	[15.86] 15:52	13:52
		4	[26.76] 26:43	22:43
		6	[37.58] 37:35	31:35

Column 1: Residual time expressed as a fraction of dive duration.

Column 2: The recovery time after the delay required to reach the residual time expressed in Column 1, with recovery time expressed as a fraction of dive duration, where $t = 0$ at the end of the dive.

Column 3: Sample dive durations.

Column 4: For the dive duration in Column 3, the elapsed time from the end of the dive for the t_r in Column 1.

Column 5: For two dives of the duration in Column 3, the surface interval necessary for an equivalent time at the end of Dive 2, that is the sum of Column 1 time and dive duration.

$$\boldsymbol{t}/T_{dur} = [6.71 \cdot ln\,(T_{dur}/t_r)]^{0.5} \quad (8)$$

and after exposures with exercise, by

$$\boldsymbol{t}/T_{dur} = [21.3 \cdot ln\,(T_{dur}/t_r)]^{0.5}. \quad (9)$$

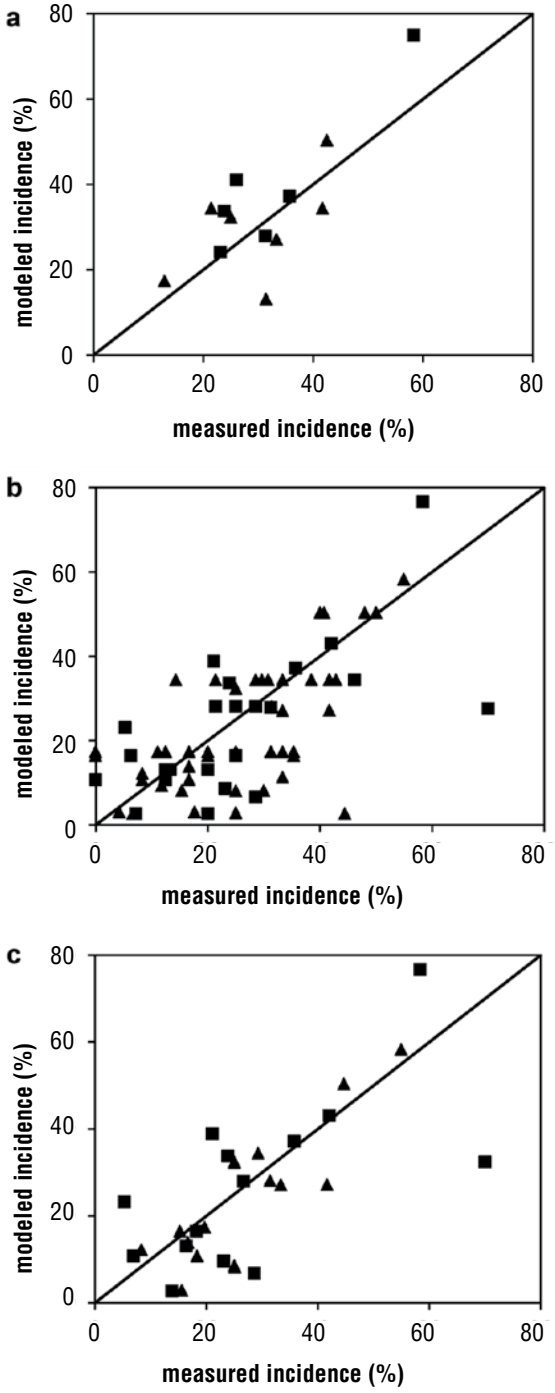
Here, *ln* represents the natural logarithm.

Recall that $\boldsymbol{t} = t - 5$ hours, that $t = 0$ at the end of the dive, and that t_r after a second dive is assessed at the end of that dive. Let $A = \boldsymbol{t}/T_{dur}$ for a selected t_r/T_{dur}. The elapsed time from the end of the dive that is necessary to recover to the chosen t_r is thus $t = A \cdot T_{dur} + 5$ hours. However, the surface interval (SI) needed to reach A at the end of the second dive is $(A-1) \cdot T_{dur} + 5$. The t_r can be chosen by choosing an acceptable P[O_2tox] for the end of the second dive, finding T_{eq} from the incidence-time model, and subtracting T_{dur2} from it. Table 3 shows some sample values.

Model comparison with NEDU data

The composite model using residual oxygen time and incidence-time calculations for rest and exercise were applied to all of NEDU's available pulmonary oxygen toxicity dive data for PO_2 of 130 kPa to 140 kPa [3-156 and unpublished]. The data comprise single dives, pairs of dives, and series of four, five, and 10 dives. The observed incidences of detected pulmonary oxygen toxicity were compared to the model predictions dive by dive. When more than one dive had the same model prediction, the observed incidences often differed. Thus, as a separate comparison, the observations from all dives of matching T_{eq} were pooled, assuming, in effect, that the model was correct and that the observed differences represented random variation. In those cases, the observed incidences of pulmonary oxygen toxicity at constant model value were calculated as total number of occurrences of signs or symptoms in all of those dives, divided by the total number of man-dives for the group; dives were treated as independent even though they were often dives by the same individuals on successive days. Correspondence of model and data can be assessed in Figures 4 and 5. In Figure 4, fitted values are plotted against observed incidences, while in Figure 5, the difference between the two estimates of probability are plotted against the average of the two (Bland-Altman plots) [30]. Panels "a" show only the "calibration" data, the data used to fit the recovery coefficients. Panels "b" represent all available dives, with each dive represented individually. Panels "c" include all the data with the dives of matching modeled incidence pooled as was described above.

Figure 4



Incidences of pulmonary oxygen toxicity after dives with PO_2 = 130 kPa, modeled vs. measured.

Resting dives: ▲; dives with exercise: ■. The identity line is shown.

a) Data used to fit the recovery models. Best fit slopes through the origin are 0.97 for resting dives and 1.04 for dives with exercise.

b) All available data. Best fit slopes through the origin are 0.84 for resting dives and 1.00 for dives with exercise. Data with identical modeled values but different observed incidence are evident as horizontal lines.

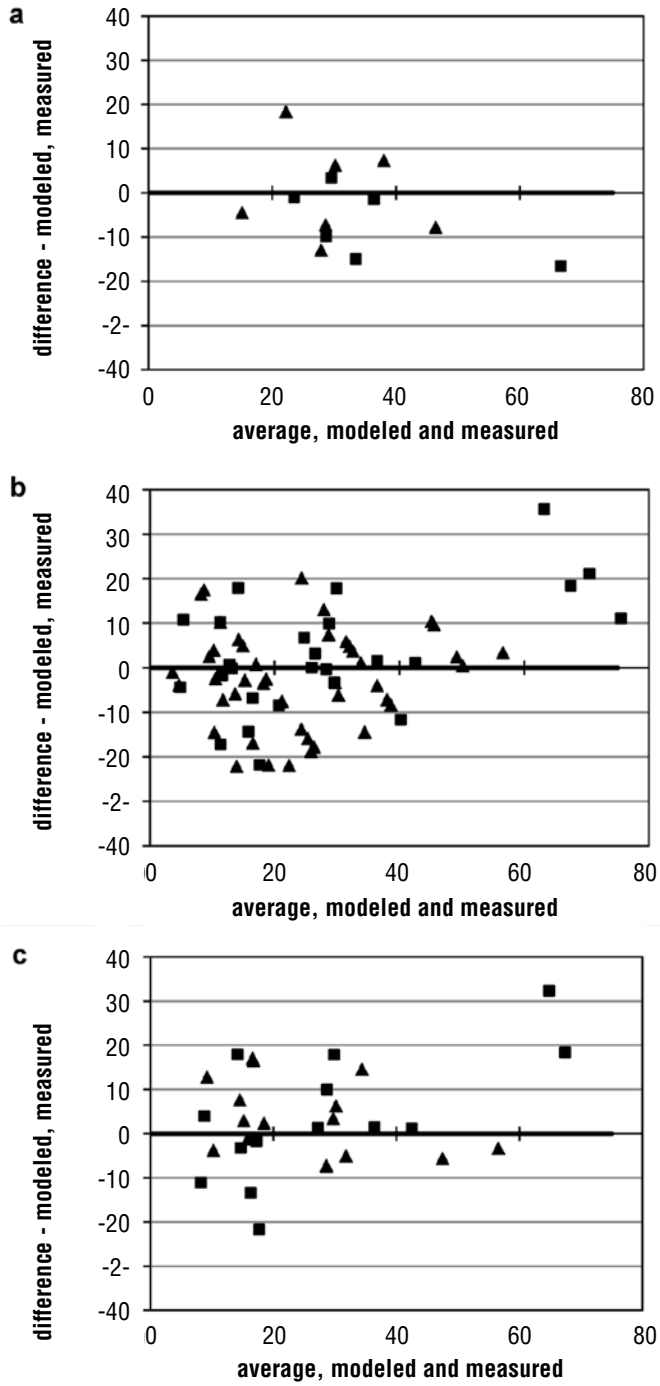
c) All available data, but the dives with identical modeled incidence have been pooled. Best fit slopes through the origin are 0.91 for resting dives and 0.88 for dives with exercise.

For the calibration data, the slopes through the origin of modeled vs. observed incidence are 1.03 and 1.16 for rest and exercise data, respectively, and the overall correlation between modeled and measured probabilities is 0.86 (Figure 4a). The modeled values are slightly lower than those observed, (Figure 5a), with a small increase in bias (offset from zero) for greater incidence ("slope vs. average," Table 4), though elimination of a single outlier would decrease the differences.

For all available dives considered individually, the slopes of the best fit lines through the origin are 0.84 for resting data and 1.01 for exercise data, and the overall correlation between measured and modeled incidences of pulmonary oxygen toxicity (either sign or symptom) is 0.71 (Figure 4b). The differences between modeled and measured values (Figure 5b) scatter generally about zero except for the four exercise-dive outliers at high incidence. Bias and slope of the residuals vs. the average are low, but the standard deviation of the residuals is high (Table 4). The multiple dives where several observed values correspond to a single model prediction are evident as horizontal lines on Figure 4b and as linear trends in Figure 5b. The constant (horizontal) lines of Figure 4b become the diagonal lines on Figure 5b as follows: if the modeled value is called k and the measured values m_i, the graph shows k-m_i against $0.5(k+m_i)$, a line with slope –2. The lines disappeared with pooling of those results (Figures 4c, 5c).

For the grouped dives, the slopes through the origin are 0.91 and 0.96 for rest and exercise data, respectively, (Figures 4c) while the overall correlation between modeled and measured probabilities remains 0.71. The Bland-Altman plot of the grouped data (Figure 5c) has similar bias to that of the ungrouped data, and the standard deviation of the differences is not reduced (Table 4).

Figure 5



Bland-Altman plots of probability for any evidence of pulmonary oxygen toxicity. The difference between model and measured incidences is plotted against the mean of the two estimates.

Resting dives: ▲; dives with exercise: ■.

a) Data used to fit the recovery models.

b) All available data. Dives with identical modeled values but different observed incidence are evident as linear runs with slopes of –2 (see text.)

c) All available data, but the dives with identical modeled incidence have been pooled.

DISCUSSION

ROT provides a method to plan repeated dives at PO_2 of 130 kPa to 140 kPa with a personally acceptable risk of pulmonary oxygen toxicity. The model:

1) describes the progress of recovery after exposure to his PO_2 (Equations 6 and 7) to answer the question of how long after a dive deleterious effects remain;
2) determines the minimum necessary surface interval for a chosen residual oxygen time (Equations 8 and 9); and
3) describes the risk of signs and symptoms of pulmonary oxygen toxicity associated with any oxygen exposure time, residual oxygen time, or the sum of the two (Equation 5).

Thus, the model predicts how likely it is that someone will have noticeable pulmonary oxygen toxicity after a particular combination of dives and surface intervals.

Unlike a number of other pulmonary oxygen toxicity models [18, 24, 30, 31], ROT attempts no association between exposure duration and either the magnitude of ΔFV or the severity of symptoms. It is worth noting, however, that in the extensive NEDU data set of exposure to PO_2 = 130 kPa to 140 kPa, almost all measured changes or reported symptoms have been mild to moderate. Severe symptoms were reported only in conjunction with more than two dives with exercise in series of four-hour dives with 20-hour SIs [8,14] or six-hour dives with 18-hour SIs [unpublished]. The experiments cited in this work also have shown that either symptoms or ΔFV may be present without the other, but that symptoms are more common than ΔFV.

The model treats recovery from one exposure as continuing even as injury from the next dive accumulates. This is based on experimental evidence: divers beginning a dive with symptoms or mild ΔFV are often without symptoms or ΔFV when they leave the water [4, 7, 13], and symptoms or measurable ΔFV early in a dive series may clear by later in the week [13, 14]. Recovery is modeled to begin five hours after surfacing from a dive, and thus not to begin during the dive when the injury is incurred. This is consistent with a

Table 4: Statistics for residuals of ROT, sigmoidal, t/T_{dur}

residuals model - measured	calibration rest	calibration exercise	all data rest	all data exercise	all data, pooled rest	all data, pooled exercise
mean	–2.3	–4	–2.7	0.8	3	0.7
standard deviation	8	8	12	15	9	18
minimum	–13	–18	–42	–42	–7	–42
maximum	7	3	20	36	17	32
correlation, residuals to measured	0.46	–0.71	–0.38	–0.04	–0.10	–0.12
fraction of residuals ≥0	43%	50%	52%	52%	60%	60%
slope, residuals vs. measured	0.34	–0.45	–0.33	0.04	–0.07	–0.19
slope, residuals vs. avg. (measured, modeled)	0.09	–0.42	0.04	0.26	0.09	–0.42

two-phase process; for example, a primary oxidative injury followed by fluid incursion and inflammation that take time to resolve and that can continue to heal even as new oxidative damage may be occurring. A better delay term would be timed from the start of the oxygen exposure, but our data cannot support a more detailed analysis of the time at which recovery begins.

The five-hour delay time may be shorter than optimal; the change observed in r^2 was a plateau, not a maximum, and it was seen only in the resting data. Further work could better define this parameter, but more data from pairs of dives with short surface intervals might be needed.

The incidence-time model and air dives

Dives with PO_2 = 30 kPa resulted in non-zero incidences of signs and symptoms, as did the incidence-time model with T_{dur} = 0. Although the signs and symptoms considered are associated with pulmonary oxygen toxicity, at low incidence they also can be associated with underwater breathing in general.

Calculation method

The effective exposure time at the end of a second or later dive in a series is the duration of the dive just completed plus the sum of residual times from any previous dives. Residual time is calculated at the end of the latest dive, the same time at which the exposure to the new dive is assessed. Each component of residual time must be computed with its own T_{dur}, and from the end of the dive to which it relates, as described in Equation 11, below. Even with identical dive durations and conditions, the calculation is

$$\boldsymbol{t} = t - 5 \text{ (hours)}$$

$$t_{r\,tot} = T_{dur} \cdot \{\exp[-k \cdot \boldsymbol{t}^2/T_{dur}^2] + \exp[-k \cdot (t-t_e)^2/T_{dur}^2]\} \quad (10)$$

because the functions of time are squared. Here t_e is the time at the end of the second dive, and $t = 0$ at the end of the first dive.

Recovery from a long dive is slower than from a short dive, as indicated by the dependence of the recovery exponent on T_{dur}. Additionally, the initial insult from a long dive is greater than that from a shorter one; severity of injury is approximately proportional to T_{dur}. Thus, assuming that recovery time is essentially unlimited after the completion of the entire job, the more efficient use of time for the same pulmonary risk is to complete a shorter dive before a longer one.

For two dives of the same duration, one with exercise and one at rest, the initial injury after the dives does not differ, but recovery is slower after the dive with exercise; for recovery rate alone, an exercise dive is equivalent to a resting dive with the duration multiplied by a factor, $[0.149/0.047]^{0.5} = 1.8$. In other words, recovery from a four-hour dive with exercise takes approximately as long as does recovery from a seven-hour resting dive. The more time-efficient order of two dives of the same length is rest before exercise.

The maximum rates of recovery, the inflection points

of Equations 6 and 7, occur when $(t\text{-}5)/T_{dur} = (2 \cdot k)^{-0.5}$, where t, in hours, is measured from the end of the second dive. The duration of the second dive must be subtracted from t to find the SI. Thus, for resting dives, SI of at least 8.3 and 10.0 hours for four- and six-hour dives, respectively, would be more efficient than any shorter SI. Similarly, for dives with exercise, SI of at least 14.0 and 18.6 hours for four- and six-hour dives, respectively, reach the time of fastest recovery.

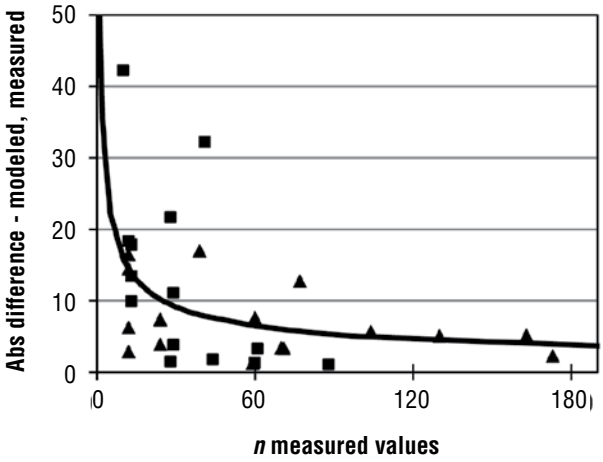
The sigmoidal recovery pattern is a departure from the functional form of other published recovery models [17, 18, 31], models in which physical healing or chemical clearance were expected to follow first-order reaction kinetics despite the cascade of probable processes. The sigmoidal pattern was proposed based on observed values of P(*P*[O2tox]) rather than on putative mechanisms. The format was selected instead of an exponential because the Akaike criterion method and the magnitude of the residuals indicated that it better fit the data (Appendix). Subjectively, the sigmoidal fit is tighter than the exponential during the initial slow recovery and during the steep recovery in the middle time range of the calibration data (Figure 3).

Model comparison with NEDU data

Pairs of dives were used to develop the model, and later dives in series having four, five, or 10 dives provided a check of goodness of fit. However, the measured data, low-incidence samples from a binomial process, have inherently high variance. Thus, not all of the differences between model and data should be ascribed to the model; the uncertainty of the differences is the sum of the uncertainties of the two estimates.

Correspondence between model and observation improved when dives of identical modeled outcomes were pooled, and the magnitudes of the differences model to observed incidence (the residuals) were approximately proportional to $n^{-0.5}$, where n is the number of man-dives for each point (Figure 6). Note that uncertainty in a binomial estimation of probability also is proportional to $n^{-0.5}$ (Equation 1). Most of the large deviations from the line of identity in Figure 4 represent cohorts of 9 to 13 divers, where one diver with a different outcome would change the incidence by close to 10%, that is, from dives where the true probability of detectable pulmonary toxicity lies in a wide band about the measured incidence. All of the large deviations represent dives with n less than or equal to 28, the median number of divers in the grouped data sets. The variability appears related to the binomial process.

Figure 5



Magnitudes of the differences, model to observation, of incidences of observable pulmonary oxygen toxicity vs. the number of observations. Data are represented after like dives have been pooled (see text).

Model comparison with data published by others

Average dive durations from a series of working rebreather dives have been published [32] in conjunction with a report of symptomatic pulmonary oxygen toxicity after the fourth day of diving. Six divers made dives nine to 15 times across eight days with a nominal PO_2 of 130 kPa. ROT calculations based on the average values for each dive, assuming that the entire dive time (bottom and decompression time) was at the nominal PO_2, predict pulmonary symptoms after diving in a range of 3% to 11% of divers, with the maximum probability after the second dive on the fourth day of the series. Three of the six divers reported pulmonary symptoms after the fourth day, a higher incidence than anticipated based on the average dives, but possibly partly attributable to unequal exposure within the group. Previous reports for occurrence of substernal distress as quoted by another author [33] indicate that half of subjects exposed to PO_2 = 1.4 atm will be symptomatic after approximately eight hours. Equation 5

gives a similar prediction: For values obtained within two hours after a dive, the dive duration after which 50% of subjects are expected to experience pulmonary oxygen toxicity is seven hours.

Model comparison with other models

In general, comparisons between published models and ROT are difficult because of the mismatch of purpose or of appropriate PO_2 among the models. Further, ROT can be compared only with models that include recovery terms. Both Arieli [18] and Vann [31] have published models predicting changes in vital capacity and the time course of recovery from those changes. Because ROT predicts probability rather than magnitude of change, values of %ΔVC were calculated from Arieli's model and Vann's optimized model for the dives in our data set, and the probability that %ΔVC > 7.7% was computed for each dive; %ΔVC > 7.7% was the number used with the experimental data to define the presence of reduced vital capacity (VC) [3]. The data were assumed to be normally distributed with standard deviation 5%, the standard deviation reported for a set of 312 baseline measurements of VC [31].

Arieli's model [19] was derived from exposures to PO_2 from 150 kPa to 250 kPa, and Vann's [32] from exposures to PO_2 from 80 kPa to 200 kPa. Neither model predicted well the incidences of reduced flow-volume parameters measured with PO_2 of 130 kPa to 140 kPa in the NEDU data set. Thus, neither corresponded well to the model derived from those data.

The REPEX model [35, 36] calculates oxygen exposure as oxygen toxicity units based on the University of Pennsylvania's unit pulmonary toxic dose (UPTD) model [31]. ROT and REPEX models are very different: REPEX attempts to control the risk of pulmonary oxygen toxicity in divers by limiting exposure time depending on the number of planned days of diving, while ROT is non-prescriptive. ROT requires that PO_2 is approximately 130 kPa, while REPEX applies the UPTD model across PO_2 values between 50 kPa and 150 kPa, and up to 175 kPa with some restrictions. ROT incorporates recovery after each dive, while REPEX lumps all exposures within a 24-hour period together as the daily exposure. ROT predicts low risk for REPEX exposures to PO_2 = 130 kPa repeated across many (e.g., 12 or 13) days, but as the number of days becomes fewer and the individual dives allowed by REPEX are longer, ROT anticipates that increasing numbers of divers will have symptoms or show ΔFV after the maximum exposures permitted by the REPEX tables.

Limitations of ROT and its development

The model developed here assumes simple additive effects of dives on the factors driving probability of symptoms and signs. The varied recovery rates after dives of differing duration are assumed not to affect the recovery rate from previous dives. No physiological mechanism is proposed. Rather, the acceptability of the assumption is based on model correspondence to data.

The incidence-time relation does not show zero incidence at zero duration. The (extrapolated) end-point was allowed to float during fitting. In support of the non-zero incidence with no oxygen exposure, we note exposures to PO_2 = 30 kPa with reported symptoms.

After dives, ΔFV and symptoms of pulmonary oxygen toxicity are sometimes delayed in onset. This model has no explicit injury onset delays, though the sigmoidal recovery function provides for slow initial healing after the somewhat arbitrary five-hour recovery delay. Data were unavailable for true fitting of the recovery delay. Further, the evidence of pulmonary oxygen toxicity used in model building was only that which manifested within two hours after pairs of dives or that manifested a day after single long dives. In other words, the sampling interval was coarse.

The data from third and later dives of series that were used to check the model were serially correlated with the data used to fit the model. In other words, they came from the same individuals, included the same individual confounding factors, and were compared to the same baselines. Even though errors of measurement after one dive could not affect values after any other dive, any errors in baseline (consisting in general of two sets of three reproducible values) would affect all the data from that diver.

Many of the NEDU data are from dives with PO_2 that varies around 135 kPa because it was set by water depth with divers free to move near the bottom of the pool. This is a good representation of diving, where rebreather PO_2 varies around the set-point. Those variations of PO_2 are within the level of precision of the data.

Exercise was treated as an all-or-nothing condition. Most dives with exercise involved moderate aerobic work on cycle ergometers for half the dive (alternating 30 minutes of work, 30 minutes of rest). Resting dives had divers either stationary or moving about freely in the pool, but without organized work. Clearly, open-water dives represent a continuum of effort, from fish watching or scooter riding to swimming hard against a current.

Residual oxygen times developed here are based only on pulmonary data. Very few data exist for non-pulmonary, "whole body" symptoms – for example, finger numbness [36, 37] or hyperoxic myopia [6, 32]. Those symptoms may have different residual times than those for pulmonary effects. Nevertheless, an estimate of pulmonary residual time is a step forward in planning.

CONCLUSIONS

The residual oxygen time model presented here corresponds well with available measurements of pulmonary effects after multiple dives with PO_2 near 130 kPa. It can assist in planning the length of successive rebreather dives when a surface interval is predetermined or in determining the necessary surface interval if a dive duration is fixed. To remain within the scope of the data used for model construction, this model is recommended for planning any number of resting or exercise dives with PO_2 = 130 kPa to 140 kPa and duration of six hours or less, but no more than two consecutive dives of more than six hours' duration unless the surface interval is long enough that t_r is negligible.

Residual oxygen time is specific to PO_2 = 130 kPa to 140 kPa and exposures of eight hours or less, the exposures for which there are data. It cannot be used to determine recovery time between hyperbaric oxygen treatments, or for dives with oxygen-accelerated decompression or other exposures to higher PO_2, where evidence suggests that effective dive durations are not simply additive [37]. Because no multiple dives longer than six hours were used to develop the model, it also should not be used for multiple exposures of that duration.

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Conflict of interest

The author reports no conflict of interest with this submission. ■

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Appendix: Details of the model fit

The fitted coefficients and some relevant statistics are listed in Table A1.

Table A1: Coefficients of model fits from the calibration set, the dives listed in Table 2

Equation	$t_r = T_{dur} \cdot \exp(c + g / T_{dur}) \cdot t$				$t_r = T_{dur} \cdot \exp[(h \cdot t^2 + k \cdot (t/T_{dur})^2]$			
MEAN	**c**	**g**	r^2 **RSS**	**No recovery after**	**h**	**k**	r^2 **RSS**	**No recovery after**
REST		–0.313	0.85			**–0.149**	0.90	
n=7 $r^2 = 0$		0.062	0.374			0.024	0.159	
mean = 0.50	–0.046		0.66		–0.003		0.81	
RSS = 1.527	0.012		0.668		0.001		0.348	
	0.115	–1.148	0.86		0.003	–0.333	0.93	
	0.072	0.559	0.227	10 hrs	0.003	0.167	0.126	10.5 hrs
EXERCISE		–0.131	0.48			**–0.047**	0.59	
n = 7 $r^2 = 0$		0.025	0.194			0.008	0.155	
mean = 0.73	–0.021		0.17		–0.001		0.18	
RSS = 0.370	0.005		0.318		0.000		0.324	
	0.037	–0.361	0.66		0.0002	–0.056	0.61	
	0.025	0.162	0.128	9.8 hrs	0.001	0.029	0.593	

For parameters *c, g, h,* and *k,* the second-row entry is the asymptotic standard error of the estimate. Time is expressed in hours. For rest, seven dive profiles were used. For exercise, six dive profiles were used, augmented with a value of $t_r = T_{dur}$ at $t = 0$. RSS represents residual sum of squares, and r^2 is the square of the correlation between observed and predicted values. The parameters selected are shown in bold.

The best fit for each of rest and exercise was selected by applying Akaike's information criterion with small sample adjustment, AIC_c. For normally distributed error and models fitted by minimizing the sum of squares, AIC_c can be written

$$AIC_c = n \cdot \ln(RSS/n) + 2 \cdot m + (2 \cdot m \cdot [m+1])/(n-m-1), \quad \text{(A1)}$$

where RSS is the residual sum of squares from the regression fit, n indicates the number of data in the fit and m is the number of parameters in the model. If $AIC_{c\,min}$ denotes the smallest value of AIC_c obtained among the models under consideration for a data set, and $\Delta_i = AIC_{c\,i} - AIC_{c\,min}$, then, given the data, the relative likelihood of model i is proportional to $\exp(-0.5 \cdot \Delta_i)$. That relative likelihood divided by the sum of the relative likelihoods for all the candidate models gives the probability that, given the data, the specific model is the best of the candidates [29].

For each of rest and exercise, the six fitted models and the null model (the mean of the data) were compared and weighted. For resting dives the Akaike criteria strongly favored the $(t/T_{dur})^2$ model; its probability (Akaike weight) was 70%, that of the two-term sigmoidal recovery model, was 19%, and that for all others was less than 5% (Table A2). For exercise dives, the model selection was not as clear as it was for resting dives; the most likely $(t/T_{dur})^2$ model had an Akaike weight of 46%, only slightly more than twice that for the (t/T_{dur}) model (Table A2). However, the choice of the $(t/T_{dur})^2$ model for recovery after dives with exercise was bolstered by analogy to the resting dive condition.

When Akaike criteria are used for model selection, models with Akaike weight greater than 5% are often combined [28]. However, for the resting data here, the second most likely model was the combination of the most likely and the third most likely (Table A2). In other words, addition of the third most likely model to the most likely one reduced its probability. Further combination of the resting models seemed illogical, and the pattern was carried over to the exercise models.

Comparison of sigmoidal and exponential recovery

The models were selected based on the quality of fit to the calibration data. The relative performance of the sigmoidal and the best fitting of the simple exponential models,

Table A2: Akaike criteria for model selection. Models are arranged in order from smallest to largest AIC_c.

a) Resting data (n=7)

model	m	RSS	AIC_c	Δ_i	relative likelihood	Akaike weight
squared, t/T_{dur}	1	0.159	-23.693	0.000	1.000	0.697
squared, both	2	0.126	-21.122	2.572	0.276	0.193
squared, t	1	0.348	-18.210	5.483	0.064	0.045
linear, t/T_{dur}	1	0.374	-17.706	5.987	0.050	0.035
linear, both	2	0.227	-17.001	6.692	0.035	0.025
linear, t	1	0.668	-13.646	10.048	0.007	0.005
average (null)	0	1.527	-10.658	13.035	0.001	0.001

b) Exercise data (n=7)
six dive sets plus one arbitrary anchor point: ($t_r = T_{dur}$ at **t** = 0, weight = 1)

model	m	RSS	AIC_c	Δ_i	relative likelihood	Akaike weight
squared, t/T_{dur}	1	0.155	-23.872	0.000	1.000	0.460
linear, t/T_{dur}	1	0.194	-22.301	1.571	0.456	0.210
linear, both	2	0.128	-21.011	2.860	0.239	0.110
average (null)	0	0.37	-20.581	3.291	0.193	0.089
squared, both	2	0.152	-19.808	4.063	0.131	0.060
linear, t	1	0.318	-18.841	5.030	0.081	0.037
squared, t	1	0.324	-18.710	5.161	0.076	0.035

Grayed-out cells are for models with relative likelihood ≤ 5%. Those are unlikely to be useful to explain the data.

n is the number of data (number of sets of second or long dives) used in fits;

m is the number of parameters in the model;

RSS is the residual sum of squares from the regression fits;

AIC_c is the "Akaike Information Criterion" corrected for small sample size;

Δ_i is the difference of the AICc of the model from the smallest AIC_c found;

relative likelihood = $\exp(-0.5\ \Delta_i)$ is the relative likelihood, given the models and the data, that a particular model is the best of the set; and the

Akaike weight is the probability that the model is the best in the set, given the data.

functions of t/T_{dur} only (Table A2) was checked also for the entire available data set (Table A3).

The standard deviations of the sigmoidal residuals for resting and exercise dives, respectively were 31% and 29% smaller than those of the exponential fits. The mean bias of the sigmoidal fit was -3% for resting dives and 1% for exercise dives, in contrast to 7% and 13% for the exponential. Finally, the increases in bias with increasing incidence for the sigmoidal fit were 0.04 and 0.26 for rest and exercise dives, respectively, while for the exponential fit, bias increased with incidence with slopes of 0.43 and 0.53 for rest and exercise respectively.

■

Table A3: Comparison of residuals of sigmoidal and exponential fits with parameter $t/T_{dur,}$ all available data

residuals	sigmoidal, t/T_{dur}		exponential, t/T_{dur}	
model – measured	rest	exercise	rest	exercise
mean	–2.7	0.8	7	13
standard deviation	11.6	15.3	16	22
median	–2.6	0.2	5	6
minimum	–42	–42	–42	–17
maximum	20	36	45	65
correlation, residuals to measured	–0.40	–0.04	–0.20	0.08
fraction of residuals >0	52%	52%	64%	74%
slope, residuals vs. measured	–0.33	0.04	–0.23	0.09
slope, residuals vs. average(measured, modeled)	0.04	0.26	0.43	0.53