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Lung diffusing capacity in a hyperbaric environment: assessment by a rebreathing technique

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Abstract

A rebreathing method was developed for measuring diffusing lung capacity for carbon monoxide (D_LCO) in a hyperbaric environment. Twenty two professional naval divers with normal lung function were included in the study. Significant correlations were found between rebreathing and single breath measurements for D_LCO ($r = 0{\cdot}94$; $p < 0{\cdot}001$; standard error of the estimate (SEE) = 0·66), alveolar volume (VA) ($r = 0{\cdot}79$; $p < 0{\cdot}005$; SEE = 0·51), and D_LCO/VA ($r = 0{\cdot}83$; $p < 0{\cdot}001$; SEE = 0·11). In 17 divers, rebreathing D_LCO (D_LCOrb) was also measured at 20 minutes pre-dive, during the first decompression stop of the dive to 45 m for 25 minutes, and at 10 minutes post-dive. Compressed air diving was performed in a dry walk-in chamber and the United States Navy decompression table was followed. The pressure induced decrease in the rate of CO binding to haemoglobin was adjusted to normobaric conditions using a theoretical approach. Also, the presence of venous bubbles post-dive was detected by precordial doppler monitoring. A biphasic change in D_LCO was noted: initially, D_LCO was increased during the dive ($p < 0{\cdot}005$); this was followed by a post-dive decrease; D_LCO/VA changed in a similar manner, as VA was only slightly altered. Only a small post-dive precordial doppler bubble grade was found. In conclusion, rebreathing D_LCO measurement is a useful respiratory function test in the hyperbaric environment. It appears that an increase in D_LCO during the compressed air dive is related predominantly to increased pulmonary capillary blood volume caused by increased negativity of the pleural pressure, hyperoxic pulmonary vasodilatation, and cardiorespiratory centralisation of the blood. The decrease in D_LCO post-dive was only partially related to the presence of the venous bubbles detectable by doppler.

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Diffusing lung capacity for carbon monoxide (D_LCO) is presently measured by three methods—namely, the standard single breath method and the less frequently used steady state and rebreathing methods. For steady state and single breath methods the subject has to be sufficiently mobile to attend the respiratory laboratory. Also, it is not possible for some patients to perform the single breath measurement because they cannot hold their breath for 10 seconds, or they have a vital capacity smaller than 1·3 l.[1] Clark *et al*[2] developed a bedside rebreathing technique predominantly for patients with pulmonary haemorrhagic oedema. Russell *et al*[3] assessed this technique in normal subjects and in patients with various lung diseases.

In the present study we used the rebreathing method in normal professional divers and compared the results with the single breath method. Then we carried out rebreathing D_LCO measurement in a hyperbaric environment and developed a theoretical adjustment of those measurements for the rate of CO binding to haemoglobin (Hb) under normobaric conditions.

Methods

Twenty two professional navy divers took part in the study. They had normal pulmonary function (table 1), electrocardiogram (ECG), and chest *x* ray films. Eight of these divers were current smokers (one heavy, three medium heavy, and four light).

The pulmonary function tests were composed of spirometry (forced vital capacity (FVC); forced

Table 1 Details of divers

	Mean (n = 22) (SD)	*Range*
Age (y)	28·6 (6·8)	20–44
Height (m)	1·77 (0·064)	1·70–191
Body mass (kg)	79·6 (8·3)	71–102
Years diving	9·2 (6·3)	3–23
FVC (l)	5·9 (0·8)	4·1–7·4
FEV_1 (l)	5·1 (1·1)	3·6–6·8
FEV_1/FVC (%)	87·2 (12·3)	74–112
PEF (l/s)	12·0 (3·3)	7·3–16·8
$MEF_{50\%}$ (l/s)	6·9 (2·7)	3·4–9·7
R_{tot} (kPa·l/s)	0·31 (0·11)	0·13–0·62
RV (l)	1·7 (0·7)	0·5–2·8
ITGV (l)	3·7 (0·7)	2·3–5·2
TLC (l)	7·8 (1·4)	6·5–9·6

expiratory volume in one second (FEV_1); flow volume curve (peak expiratory flow (PEF); maximal expiratory flow rate when 50% FVC remains (MEF_{50}); body plethysmography (total airway resistance (Rtot); intrathoracic gas volume (ITGV); total lung capacity (TLC) (Jaeger MASTERLAB, Germany); single breath diffusing lung capacity for carbon monoxide (D_LCOsb); and rebreathing D_LCO (D_LCOrb) (Morgan MK-4, UK)). The best of triplicate manoeuvres for spirometry, flow volume, and body plethysmography were used. Measurement of D_LCOrb was by the modified method of Clark *et al.*[2] The instrumentation for this method is very simple. It is composed of an anaesthetic bag (1 litre) with a two way breathing valve connected to one end of the bag, and a two way tap connected to the other end. The bag was emptied with a vacuum pump (Morgan MK-4, UK) and filled with 0·75 l of the same gas mixture used in single breath measurements (14% He, 0·3% CO, 21% O_2, and balanced N_2). With applied nose clip, the patient breathed quietly through the open valve. At the end of normal expiration the two way valve was turned on and the subject started to breathe in and out of the bag, emptying it with each inspiration, at a rate of 10 breaths in 10 seconds, guided by a metronome. After the test was completed, the valve was turned off and the mixture in the bag was analysed for He, CO, and O_2 concentrations.

Krogh's equation for estimation of D_LCO/VA (mmol/min·kPa·1) was used.[4] For completeness and because of considerable ambiguity of published constants and units we first derived this basic relation for D_LCO determination by a rebreathing technique.

DERIVATION OF KROGH'S EQUATION

The quantity of CO in mmol present in the lungs at the time t is denoted by q(t). It decreases in time only due to the loss to the capillary sink by diffusion, which is guaranteed by the conditions of the experiment (the subject breathing in a closed system). The quantity of CO in mmol that crosses the alveocapillary membrane in 1 minute if the driving pressure gradient is 1 kPa is denoted as D_LCO (mmol/kPa·min). Then the quantity of CO removed from the lungs in the small time interval from t to t + Δ t, when the variable pressure gradient does not change appreciably from p(t), is approximately:

$$q = q(t + \Delta t) - q(t) = -\Delta t \cdot p(t) \cdot D_LCO \quad (1)$$

The equation (1) is valid exactly in the limit Δ t, Δ p → 0:

$$dq/dt = -p(t) \cdot D_LCO \quad (2)$$

Neglecting capillary CO back pressure, p is the alveolar pressure and, according to the ideal gas equation, it is given by:

$$p(t) = q(t) \cdot R \cdot T/VA \quad (3)$$

where R is the universal gas constant (= 0·008314 $kPa \cdot l \cdot K^{-1} \cdot mmol^{-1}$), T is the body temperature in degrees Kelvin, (310 K), and VA is the alveolar volume in litres under BTPS (body temperature and pressure saturated) conditions. Combining (2) and (3) gives:

$$dq/dt = -R \cdot T \cdot (D_LCO/VA) \cdot q(t) \quad (4)$$

The solution of the differential equation is:

$$q(t) = q(0) \cdot \exp(-R \cdot T \cdot (D_LCO/VA) \cdot t) \quad (5)$$

where the time 0 is the arbitrary onset of observation. After converting to logarithms and rearranging, (5) is written as:

$$D_LCO/VA = (1/R \cdot T) \cdot (1/t) \cdot \ln(q(0)/q(t)) \quad (6)$$

Specifying the units and inserting the numerical value for 1/R·T(= 0·388 mmol/kPa·1) the equation (6) reads:

$$D_LCO/VA = 0{\cdot}388\ (mmol/kPa \cdot 1) \cdot (1/t(min)) \cdot \ln(q(0)/q(t))\ (mmol/kPa \cdot min \cdot 1) \quad (7)$$

The ratio q(0)/q(t) in (7) can be substituted by the ratio of fractional CO concentrations in the bag at the start and the end of rebreathing respectively (F_{ICO}/F_{ECO}), multiplied by the dilution factor. The last is measured by the dilution of He as the ratio of initial and end fractional He concentrations in the bag ($F_{EHe}:F_{IHe}$). With the usual convention of expressing the rebreathing time t in seconds, equation (7) is transformed to:

$$D_LCO\ (mmol/kPa \cdot mm \cdot l) = \ln\left(\frac{F_{ICO} \cdot F_{EHe}}{F_{ECO} \cdot F_{IHe}}\right) \cdot \frac{23{\cdot}28\ mmol/kPa \cdot l}{t\ (s)} \quad (8)$$

Alveolar volume (VA) can be calculated by He dilution (at the start and at the end of the rebreathing period):

$$VA_{BTPS}(l) = \frac{F_{IHe} \cdot 0{\cdot}75\,l}{F_{EHe}} \cdot 1{\cdot}1 \quad (9)$$

where 0·75 l is the bag volume; 1·1 is a correction factor to convert ATPS (ambient temperature and pressure saturated) to BTPS, assuming an ambient

temperature of 20°C. It is equal to $V_{BAG} + V_{LUNG}$. D_LCO (mmol/min·kPa) was calculated as:

$$D_LCO = (D_LCO/VA) \cdot VA \quad (10)$$

All subjects performed duplicate D_LCOsb and D_LCOrb measurements. The mean of two tests for each method was used.

Adjustment to a standard Hb concentration of 146 g/l was done according to Cotes[5]:

$$D_LCO = \text{measured } D_LCO \cdot \frac{1{\cdot}022 + Hb\,(g/l)}{0{\cdot}17 \cdot Hb\,(g/l)} \quad (11)$$

ADJUSTMENT TO ATMOSPHERIC O_2 PRESSURE (CORRECTION FOR PRESSURE INDUCED DECREASES IN THE RATE OF CO BINDING TO Hb)

It is well known that the rate of CO binding to Hb depends on the partial pressure of arterial O_2 due to competitive inhibition and the saturation dependent nature of the reaction. This principle is utilised to calculate D_LCO membrane (Dm) and its vascular component. The vascular component is the product of the rate of CO binding to Hb (Θ) and the pulmonary capillary blood volume (Vc). As the inverse of D_LCO and its components Dm and $\Theta \cdot Vc$ are resistances to diffusion, their relation is governed by the Roughton-Forster equation:

$$1/D_LCO = 1/Dm + 1/\Theta \cdot Vc \quad (12)$$

The divers breathed the higher tension O_2 that produced higher Hb saturation, resulting in a decrease in the rate of CO binding to Hb and thus the decrease in D_LCO given in (12). To resolve other physiological effects of the hyperbaric environment that increase D_LCO, as discussed later, it is necessary to convert the D_LCO values obtained in the hyperbaric chamber to the ones that would have been obtained if nothing but Θ had changed from normobaric conditions. This means that D_LCO ($\Theta_{hyperbaric}$, Dm, Vc) and D_LCO ($\Theta_{normobaric}$, Dm, Vc) should be related. From (12) a simple algebraic manoeuvre gives the following ratio:

$$D_LCO\,(\Theta_{normobaric}, Dm, Vc) = D_LCO\,(\Theta_{hyperbaric}, Dm, Vc) \cdot \frac{\Theta_{normobaric}}{\Theta_{hyperbaric}} \cdot \frac{1 + (Vc/Dm) \cdot \Theta_{hyperbaric}}{1 + (Vc/Dm) \cdot \Theta_{normobaric}} \quad (13)$$

The rates $\Theta_{normobaric}$ and $\Theta_{hyperbaric}$ can be assessed from P_{AO_2} using the equation of Bates *et al*[11] (converting the original units: mm Hg to kPa; ml CO (STPD) to mmol):

$$1/\Theta_{normobaric}\,(kPa \cdot min \cdot ml/mmol) = 0{\cdot}13 \cdot P_{AO_2 normobaric}\,(kPa) + 2{\cdot}23$$
$$1/\Theta_{hyperbaric}\,(kPa \cdot min \cdot ml/mmol) = 0{\cdot}13 \cdot P_{AO_2 hyperbaric}\,(kPa) + 2{\cdot}23 \quad (14)$$

Finally, the ratio Vc/Dm in (13) may be measured outside the chamber, or, in normal subjects, approximated as the constant value 4·5 ml·kPa·min/mmol. In our calculations we measured the ratios Vc/Dm in all divers, as detailed further. $P_{AO_2 normobaric}$ was measured pre-dive by multiplying the fractional O_2 concentration by atmospheric pressure (101·3 kPa), and $P_{AO_2 hyperbaric}$ was obtained by multiplying fractional O_2, measured during the first decompression stop, by the corresponding pressure (1·6·101·3 kPa). Because fractional concentrations were involved in the last, measurements outside the chamber apply equally to intrachamber values.

In the first part of the study, correlation between the single breath and rebreathing methods was assessed in the sample of 22 divers. In all subjects the duplicate D_LCOrb test was performed, followed by the duplicate D_LCOsb test one hour later.

The second part of the study had the following protocol: the subjects were exposed to absolute air pressure of 5·5 bar for 25 minutes in a three compartment recompression chamber (Draeger, Germany) and the standard United States Navy decompression table was used;[6] decompression was carried out with air at two decompression stops (first decompression stop for four minutes at 6 m and the second decompression stop for 17 minutes at 3 m). The D_LCOrb was measured three times: the first measurement, which included duplicate D_LCO, Dm, and Vc measurements, was performed 20 minutes pre-dive, followed by the second one during the first decompression stop as the single measurement and the third one in duplicate at 10 minutes post-dive. During dives the subjects were seated upright. As our recompression chamber has three compartments, the person performing the rebreathing test was able to enter and leave the main compartment during the first decompression stop.

Venous bubbles were detected precordially with a doppler bubble detector (Sodelec SA, France) for one minute at rest and during 10 consecutive cardiac cycles with the diver performing deep knee bends (squatting down slowly and then rising to an upright position (repeated twice), using the Kisman-Masurel code.[7] Precordial bubble grade was monitored at 10 minutes post-dive.

For comparison of the pre-dive, dive, and post-dive D_LCOrb, $D_LCO/VArb$, and VArb values paired *t* tests were used. For correlation of the rebreathing and the single breath values least squares linear regression analysis was applied. All data were expressed as the mean (standard deviation (SD)) and range.

Results

Table 2 shows the mean values of D_LCO, D_LCO/VA, and VA measured with rebreathing and single breath methods in 22 professional navy divers. On average D_LCOrb was reduced by 28·3% in comparison with D_LCOsb due to decreased VArb (27·0%).

Significant correlations were found between

Table 2 Mean values of D_LCO, D_LCO/VA, and VA measured with rebreathing or single breath methods in 22 divers

	Mean (n = 22) (SD)	*Range*
D_LCOsb (mmol/min·kPa)	12·08 (1·75)	8·4–15·6
$D_LCO/VAsb$ (mmol/min·kPa·l)	1·87 (0·19)	1·65–2·45
VAsb (l)	6·45 (0·81)	4·8–8·3
D_LCOrb (mmol/min·kPa)	8·66 (1·28)	6·1–10·8
$D_LCO/VArb$ (mmol/min·kPa·l)	1·85 (0·18)	1·62–2·32
VArb (l)	4·70 (0·69)	3·4–5·9

D_LCOsb and D_LCOrb ($r = 0{\cdot}94$; $p < 0{\cdot}001$; standard error of the estimate (SEE) = 0·66 mmol/kPa·min) (table 3 and fig 1), $D_LCO/VAsb$ and $D_LCO/VArb$ ($r = 0{\cdot}83$; $p < 0{\cdot}001$; SEE = 0·11 mmol/kPa·min) (table 3 and fig 2), and VAsb and VArb ($r = 0{\cdot}79$; $p < 0{\cdot}005$; SEE = 0·51 mmol/kPa·min) (table 3).

Figure 3 shows the effect of a single air dive to 45 m for 25 minutes on D_LCO measured during the first decompression stop at 6 m ($n = 17$). By comparison with the pre-dive values intrachamber D_LCO increased significantly ($p < 0{\cdot}005$), whereas it decreased post-dive. The ratio $D_LCO/VArb$ was also increased during the first decompression stop ($p < 0{\cdot}005$), because VArb was unchanged. All of these statements apply equally to the raw D_LCO data as well as to D_LCO adjusted to standard Hb and rate of CO binding to Hb (for individual data see table 4). Mean precordial doppler bubble grades at rest and after deep knee bends were 0·1 and 0·7 (grading scale 0–4) respectively.

Discussion

Measurement of diffusing lung capacity is an important clinical test of respiratory function in evaluating the exchange of O_2 and CO_2 across the blood gas barrier. Because the most frequently used single breath method has certain limitations (patients have to be mobile to attend the respiratory laboratory, to be able to hold their breath for at least 10 seconds, and to have a vital capacity above 1·3 l),[1] recent modification of the rebreathing method by Clark *et al*[2] expanded its use to virtually all patients. Certain assumptions apply to rebreathing D_LCO measurement—namely, a negligibly small dead space of subject and equipment; insignificant time spent at end expiration in relation to the time spent at full inspiration making the bag extension of the lung so that alveolar volume is calculated as $V_{BAG} + V_{LUNG}$; and instantaneous and uniform mixing of CO and He.[3] Despite this, surprisingly good correlations between D_LCOsb and D_LCOrb, $D_LCO/VAsb$ and

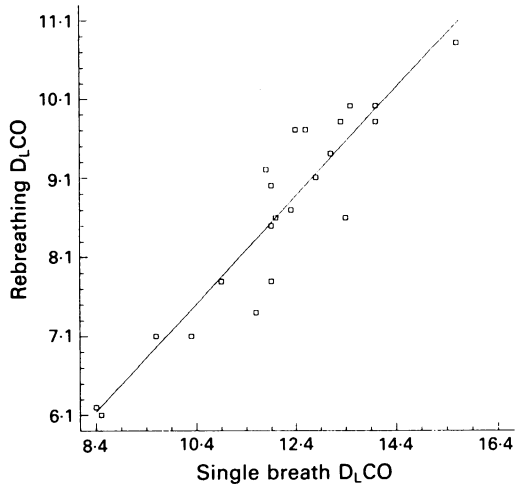

Figure 1 Relation between D_LCO values measured by single breath and rebreathing methods ($y = 1{\cdot}27x + 1{\cdot}11$; $r = 0{\cdot}94$; $p < 0{\cdot}001$; SEE = 0·66) in 22 divers.

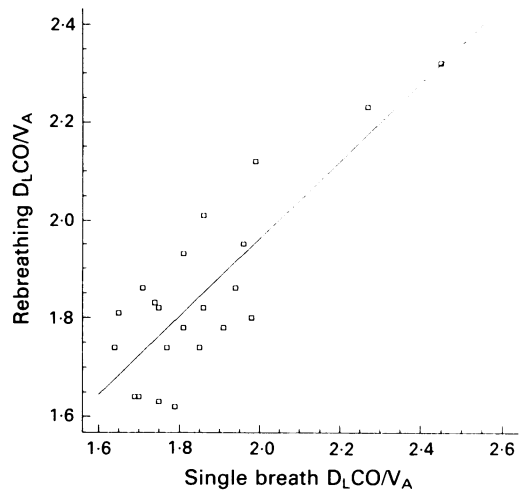

Figure 2 Relation between D_LCO/VA values measured by single breath and rebreathing methods ($y = 0{\cdot}84x + 0{\cdot}32$; $r = 0{\cdot}83$; $p < 0{\cdot}001$; SEE = 0·11) in 22 divers.

Table 3 Correlations between D_LCO, D_LCO/VA, and VA measured with rebreathing or single breath methods in 22 divers

Variable	*Equation*	*r*	*p Value*	*SEE*
D_LCO	$D_LCOrb = 1{\cdot}27 \cdot D_LCOsb + 1{\cdot}11$	0·94	< 0·001	0·66
D_LCO/VA	$D_LCO/VArb = 0{\cdot}84 \cdot D_LCO/VAsb + 0{\cdot}32$	0·83	< 0·001	0·11
VA	$VArb = 0{\cdot}93 \cdot VAsb + 2{\cdot}09$	0·79	< 0·005	0·51

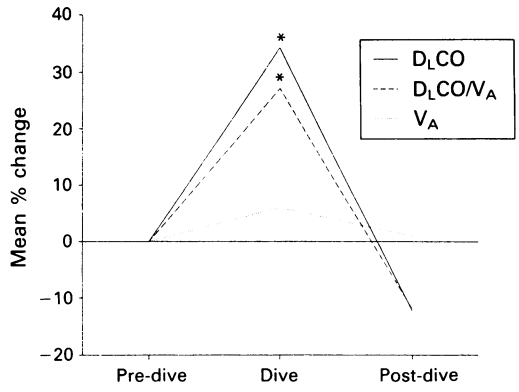

*Figure 3 Mean % change of D_LCO, D_LCO/VA, and VA measured with rebreathing technique before, during, and after a single air dive to 45 m for 25 minutes in 17 divers. *$p < 0.005$.*

D_LCO/VArb, and VAsb and VArb were found in the present study, in accord with reports of other investigators.[2 3]

The simplicity of the method and mobility of the rebreathing equipment allowed us to measure D_LCO in the hyperbaric environment, to our knowledge, for the first time. It was measured before, during, and after the compressed air dive to 45 m for 25 minutes in the walk-in recompression chamber. A significant increase in $D_LCO$rb was found during the dive at a depth of 6 m, followed by a post-dive decrease (fig 3). Possible explanations for this effect of the hyperbaric environment on D_LCO are: (1) a pressure proportional increase of air density and consequent increased airway resistance increases respiratory muscle activity. This results in increased negativity of the pleural pressure that facilitates the venous return, finally causing enlargement of the pulmonary capillary blood volume; (2) breathing hyperbaric air increases the total amount of O_2 predominantly through solutions in plasma and the increased amount of oxygen is available to the peripheral tissues. By autoregulation this causes local vasoconstriction[8] and redistribution of the blood to the central cardiorespiratory region. Quantitative support for this idea is given by Fagraeus *et al.*[9] They reported that during compressed air diving the mean arterial pressure was unchanged in the presence of bradycardia. To maintain the mean arterial pressure the stroke volume should have been increased. The sole reason for this must be increased diastolic filling, accomplished by centralisation of the blood caused by peripheral vasoconstriction. The last was not caused by sympathetic nervous system activity, as bradycardia occurred and blood noradrenaline concentration was decreased.[9] Therefore it appears that the centralisation of hyperoxic blood volume augments diastolic filling and the capillary blood volume considerably; (3) pulmonary blood vessels responded to hyperoxia with vasodilatation; (4) alveolar volume was found to be increased in the present study during the single air dive compared with pre-dive values, although this was a minor contribution; and (5) decrease in the rate of CO binding to Hb (Θ) in a hyperoxic environment has the opposite effect of decreasing D_LCO. This effect can be assessed by in vitro measurement as the sole function of PO_2 and thus presumably does not exhibit intraindividual variations. Therefore we found it advantageous to adjust intrachamber D_LCO values to normobaric Θ.

Table 4 Individual data for 17 divers

	D_LCO (mmol/kPa·min)			D_LCO/VA (mmol/kPa·min·l)			VA (l)		
Diver	*Pre-dive*	*Intrachamber*	*Post-dive*	*Pre-dive*	*Intrachamber*	*Post-dive*	*Pre-dive*	*Intrachamber*	*Post-dive*
1	5·8	8·6 (7·6)	5·4	1·35	2·26 (2·00)	1·32	4·3	3·8	4·1
2	6·3	10·6 (9·3)	5·7	1·44	2·12 (1·86)	1·21	4·4	5·0	4·7
3	5·9	8·0 (7·2)	5·5	1·41	1·60 (1·43)	1·17	4·2	5·0	4·7
4	6·1	8·3 (7·4)	5·1	1·48	1·66 (1·48)	1·21	4·1	5·0	4·2
5	4·8	6·9 (6·1)	4·5	1·56	1·97 (1·74)	1·55	3·1	3·5	2·9
6	6·2	9·4 (8·4)	5·7	1·40	2·23 (2·01)	1·04	4·4	4·2	5·5
7	7·0	10·5 (9·2)	5·9	1·50	2·06 (1·81)	1·09	4·7	5·1	5·4
8	8·8	11·1 (9·8)	8·0	1·73	2·41 (2·13)	1·86	5·1	4·6	4·3
9	7·0	11·3 (10·2)	6·5	1·70	2·51 (2·26)	1·48	4·1	4·5	4·4
10	8·2	9·6 (8·7)	5·7	1·49	1·88 (1·70)	1·19	5·5	5·1	4·8
11	7·3	11·0 (9·8)	6·3	1·40	1·86 (1·66)	1·43	5·2	5·9	4·4
12	9·0	11·1 (10·1)	6·6	2·00	2·22 (2·02)	1·65	4·5	5·0	4·0
13	9·2	12·1 (10·7)	7·9	1·74	2·09 (1·85)	1·46	5·3	5·8	5·4
14	7·0	8·4 (7·5)	6·4	2·12	2·10 (1·85)	2·00	3·3	4·0	3·2
15	7·4	9·0 (8·1)	5·9	1·96	1·96 (1·78)	1·31	3·8	4·6	4·5
16	7·4	8·2 (7·1)	6·7	1·61	2·05 (1·77)	1·56	4·6	4·0	4·3
17	9·9	12·8 (11·3)	10·0	2·32	2·84 (2·50)	2·22	4·3	4·5	4·5
$\bar{x}$	7·3	9·8	6·4	1·66	2·11	1·46	4·4	4·7	4·4
SD	1·4	1·6	1·3	0·27	0·30	0·33	0·6	0·7	0·7

D_LCO and D_LCO/VA intrachamber values are reported adjusted to standard Hb and for standard rate of CO binding for Hb. The raw data are given in parentheses; pre-dive and post-dive values are reported adjusted to standard Hb.

By the removal of this calculable variable, it is possible to appreciate more thoroughly the impact of other physiological parameters on changes of D_LCO in hyperbaria. In our conditions (6 m depth) measured D_LCO values were adjusted to normobaric Θ by multiplying by 1·12. At greater depths, however, this correction may become quite large (for instance, at 50 m it equals 1·84).

Diffusing lung capacity is dependent on the perfusion of the pulmonary capillary bed, the thickness of the alveolocapillary membrane, the surface area of the membrane, and the diffusion coefficient of the gas in the membrane. It appears that the observed increase of D_LCO (+ 34%) in the hyperbaric environment is probably a result of greatly increased pulmonary capillary blood volume, which expands the capillaries and increases the surface through which gases can diffuse in the blood. Another minor contribution is an increase in the alveolar volume (+ 6%), which expands the surface area of the respiratory membrane.

In the present study a decrease in D_LCO was found after the single air dive to 45 m for 25 minutes (fig 3). At the same time the precordial doppler bubble grade was small. The possible explanations for this discrepancy between D_LCO (quantitative parameter) and precordial bubble grade (semiquantitative parameter) are (1) doppler has limited sensitivity to venous bubbles with radius smaller than 50 μm,[10] whereas D_LCO is a method which measures the gas exchange in the pulmonary capillaries with radius of about 8 μm; (2) bubble grade is an index of an instant rate of production of the bubbles, whereas D_LCO shows the net effect of integrated production and elimination, up to the time considered; and (3) bubble grade may not be at its maximum at that time (10 minutes post-dive). The mechanism of post-dive D_LCO reduction is that trapped venous bubbles in the pulmonary circulation decrease the effective vascular surface area and volume with resulting impaired gas exchange.

In conclusion, we applied the rebreathing method for measurement of diffusing lung capacity in the hyperbaric environment. It appears that the increase in diffusing lung capacity during the compressed air dive was predominantly caused by increased pulmonary capillary blood volume, and the change in alveolar volume was not important. The decrease in D_LCO after the dive was only partially caused by the venous bubbles detected by precordial doppler monitoring.

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