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Respiratory effects of a single saturation dive to 300 m

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ABSTRACT Lung function and the response to exercise were monitored in seven diver/welders who took part in a test saturation dive to 300 m for an average duration of 12 days; decompression took an average of nine days. Immediately after the dive the forced vital capacity was increased above base line by on average 0·5 l, the forced expiratory volume by 0·28 l and peak expiratory flow rate by 0·7 l s^{-1}. There was no change in flow rate at small lung volumes ($FEF_{75\%FVC}$). Recovery was complete and appeared to have a half time of 28 days. Transfer factor of the lungs for carbon monoxide (*Tl*CO) was reduced by on average 9·6% after the dive but while partial recovery occurred, the values at one year were on average lower than those observed initially. The reason is unclear. One subject developed transient oxygen toxicity with stiff lungs and increased ventilation and cardiac frequency during submaximal exercise; a second subject developed similar changes but without accompanying symptoms. There is need for detailed physiological surveillance of people undertaking deep dives; this should be undertaken in circumstances that permit accurate measurements and full subject cooperation.

In commercial divers the forced vital capacity has been found to vary from year to year, reflecting changes in diving exposure.[1] Over any one year the diving effect is the result of all the individual dives undertaken during that year but the changes associated with a single dive are likely to be small except when the dive is unusually deep or the workload much above average. These circumstances were met during a recent demonstration dive to 300 m; it enabled an estimation to be made of the time course of the changes in vital capacity and provided information on other aspects of lung function, including flow rates at small lung volumes and the physiological response to exercise. A brief account of the work has been given to the Medical Research Society.[2]

Subjects and methods

The subjects were seven commercial diver/welders who were referred for assessment on four occasions (designated one to four), before the dive (I), during the first week after return from saturation (II), and at one month and one year after the dive (III and IV respectively). In the interim the divers had resumed their normal occupations. The assessments were based on those required by the Norwegian diving authority[3] but with additional tests.

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Diving exposure was obtained from the divers' log books for the year before and after the dive. These were used to obtain maximal depth and the number and duration of dives. Time in saturation was subdivided into chamber time and excursion time.

Respiratory symptoms were assessed using the MRC questionnaire (Medical Research Council 1976). Venous blood was drawn for measurement of haemoglobin concentration. Forced expiratory volume and vital capacity (FEV_1 and FVC) and indices from the flow volume and volume time curves were measured using a McDermott digital bellows spirometer.[4] The flow rate indices were peak expiratory flow rate (PEFR) and flow rate when 50% and 75% of vital capacity had been expired (respectively $FEF_{50\%FVC}$ and $FEF_{75\%FVC}$). Flow rates on different occasions were compared after being standardised to a common lung volume (see discussion). Subdivisions of total lung capacity including residual volume by helium dilution and transfer factor (diffusing capacity) of the lung for carbon monoxide by the single breath method were measured using transfer test apparatus (Morgan). Static volume/pressure curves for estimation of lung compliance and recoil pressure were obtained from measurements of pressure difference between mouth and oesophageal balloon during slow inspiratory and expiratory vital capacity manoeuvres with multiple interruptions; pressures were recorded after the airway had been interrupted for 0·4 s. Maximal inspiratory and expiratory force was measured as pressure at the mouth when the subject performed maximal inspiratory and expiratory manoeuvres against a closed airway, respectively from residual volume (also functional residual capacity) and total lung capacity. A small leak was used to avoid error from movement of the cheeks. The largest of three pressure determinations at each volume was recorded. Progressive exercise was performed at a single session on a motor driven treadmill. The initial belt speed was 3 kmh^{-1}; this was increased to 7 kmh^{-1} after two minutes and the subjects asked to start running. The incline was then raised to 4° for one minute and thereafter speed and incline were increased by 1 kmh^{-1} and 1° on alternate minutes. The endpoint was when the subject stopped or when oxygen uptake failed to increase by 4·5 mmol min^{-1} despite an increase in workload. During each minute of exercise measurements were made of inspiratory ventilation minute volume and respiratory frequency using a vane anemometer (ventilometer, Morgan); concentrations of carbon dioxide and oxygen in mixed dried expired gas were measured using respectively infrared and paramagnetic gas analysers in series. Water vapour was absorbed before passage through the oxygen analyser. Cardiac frequency was obtained using a memory cardioscope (Rigel model SM801R); the frequency was checked against a separately recorded electrocardiogram. These data were used to obtain by interpolation the ventilation and cardiac frequency at an oxygen uptake of 67 mmol min^{-1} ($\dot{V}_E67$ and fC_{67} respectively), the tidal volume at a minute volume of 30 l min^{-1} (Vt_{30}), and the oxygen uptake at both a respiratory exchange ratio of unity ($\dot{n}O_2$ at $R_{1\cdot0}$) and the breaking point of exercise ($\dot{n}O_2$ max).

Maximal exercise was not performed on occasion II. Instead maximal oxygen uptake was estimated from $\dot{n}O_2$ at $R_{1\cdot0}$, cardiac frequency during submaximal exercise and other variables using a prediction equation previously validated for shipyard workers.[5]

For all the measurements, standard methods, calibration procedures, and reference values were used: these are described in detail elsewhere.[6] Changes in lung function indices between occasions were expressed in the form $\Delta x \times 100 \div \bar{x}$ to avoid error associated with taking the initial value as the reference point.[7] The statistical methods included paired *t* tests and cross correlation analysis. The 5% level of probability was accepted as significant.

Table 1 *Personal details of subjects at first attendance*

	Mean	*Range*
Age (years)	34·1	29–37
Stature (m)	1·78	1·67–1·85
Body mass (kg)	87·0	79–99

Results

Personal details of the subjects are given in table 1 and their recent diving exposure in table 2. For the year before the deep dive one man (No 3) had no log book entries though previously he had been diving regularly; the other had spent an average of 65 days in saturation at a maximal depth in the range 140–160 m. For the year after the dive an average of 78 days was spent in saturation at a maximal depth in the range 72–175 m. The dive itself was to a storage depth of 290 m with excursions to 300 m. The average time spent at these depths was 12 days (including 2·1 days on excursion) and the average decompression time was nine days. The total time under pressure was on average 21 days (range 10–30 days). The gas mixture breathed was reported to comprise 1·8% oxygen in helium. The mean results for the lung function indices are given in table 3.

On occasion I the subjects were free from notable respiratory symptoms despite three being current smokers and three ex-smokers. The initial lung function was also within normal limits including flow

Table 2 *Details of subjects' diving exposure*

	1	*2*	*3*	*4*	*5*	*6*	*7*
Years diving	10	9	8	13	8	12	7
12 months before dive:							
No of dives	40	27	0	14	54	45	36
Maximal depth (m)	160	145	0	143	160	156	140
Saturation time (days)	NI	46	0	19	100	68	92
Details of deep dive:							
Bell time (days)	26·5	19·7	18·4	18·7	18·8	7·8	25·0
No of excursions	12	4	6	7	8	7	11
Excursion time (days)	3·4	1·3	1·5	2·3	2·2	2·1	2·0
12 months after dive:							
No of dives	45	37	37	51	46	24	60
Maximal depth (m)	72	175	109	132	110	109	150
Saturation time (days)	82	52	73	76	103	35	123

NI = No information.

Table 3 *Mean values for indices of lung function before and on three occasions after the dive*

	Before	*1 week*	*1 month*	*1 year*
FEV_1 (l)	4·35	4·59*	4·49	4·27
FVC (l)	5·90	6·32*	6·12*	5·75
FEV_1 × 100/FVC (FEV%)	74·2	73·0	73·7	75·0
Peak expiratory flow rate (l/s)†	11·9	12·6*	12·3*	12·0
Forced expiratory flow ($FEF_{50\%FVC}$ l/s)†	5·42	4·95*	5·23	5·30
($FEF_{75\%FVC}$ l/s)†	1·76	1·69	1·73	1·92
Total lung capacity (l)	7·64	7·70	7·45	7·76
Residual volume (l)	1·48	1·33	1·32	1·54
Transfer factor (*Tl*', mmol/min/kPa)‡	13·9	12·6*	13·1	12·9
*K*co (mmol/min/kPal)‡	2·00	1·78*	1·88	1·85

*Significant change from before the dive.
†At constant lung volume above RV (for details see text).
‡Results for subject 1 omitted (for reasons see text).

rates, lung volumes, transfer factor, compliance, and maximal respiratory pressures; the latter measurements were not made on two subjects. On occasion II subject 2 reported soreness beneath the sternum and a need to take deep breaths at the time of return from saturation. Most of the subjects reported non-respiratory symptoms including weakness in the legs, fatigue, and insomnia.

Between occasions I and II significant increases were observed for FVC, PEFR, and FEV_1. Inspiratory vital capacity and two stage vital capacity obtained in conjunction with measurement of total lung capacity also increased significantly. Significant decreases were observed for transfer fact and *K*co (transfer factor ÷ alveolar volume) despite an increase occurring in one subject; this man had evidence of a raised blood ethanol concentration at the time of the second set of measurements (occasion II). No consistent changes were observed for total lung capacity, residual volume, compliance, recoil pressure, or maximal respiratory pressure. For those indices where significant changes occurred their magnitude was not significantly correlated with the

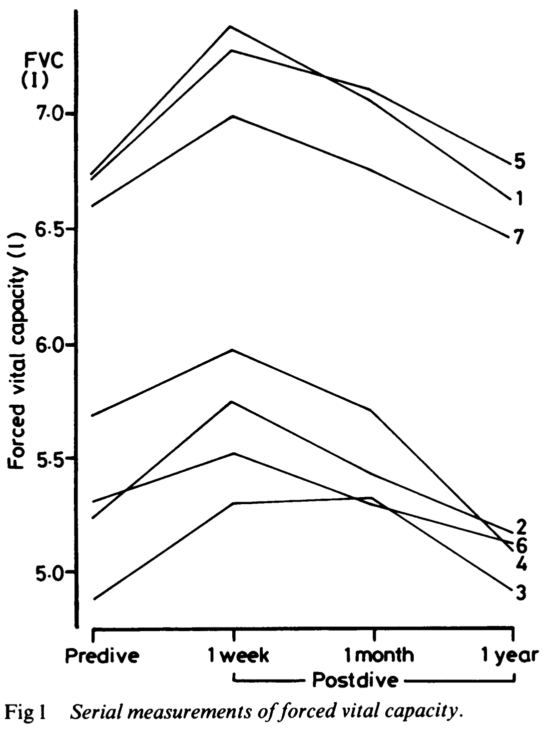

Fig 1 *Serial measurements of forced vital capacity.*

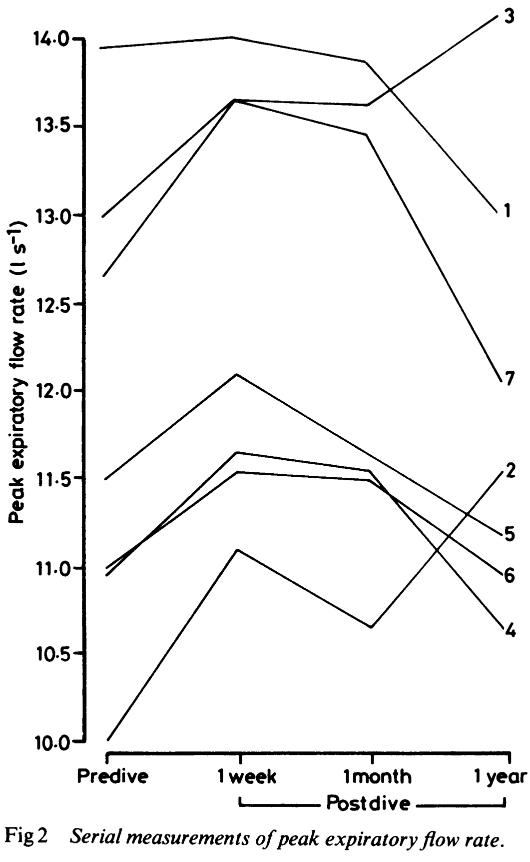

Fig 2 *Serial measurements of peak expiratory flow rate.*

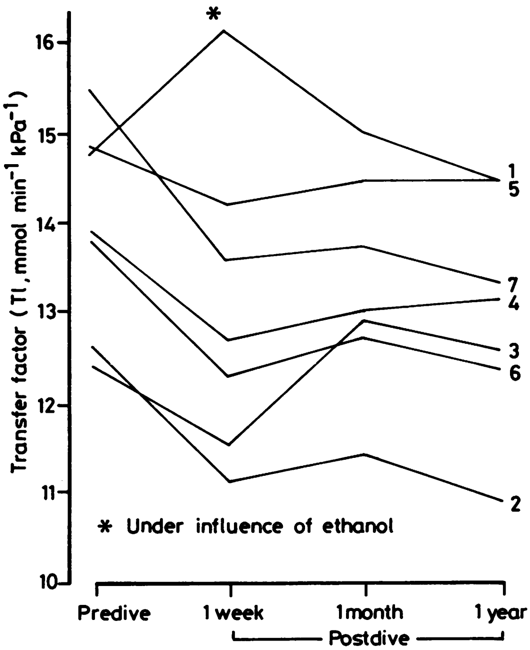

Fig 3 *Serial measurements of lung transfer factor.*

duration of the dive; however, the subject who made the shortest dive (No 6) also had the smallest increase in FVC (0·02 l). On occasion III the FVC and PEF were significantly reduced compared with occasion II but still increased compared with occasion I. By occasion IV both indices had returned to their initial values (figs 1 and 2). Transfer factor in those subjects in whom it was reduced on occasion II was slightly but significantly increased on occasion III but on occasion IV in three subjects showed no evidence of recovery from the low level observed on occasion II (fig 3).

Information on the physiological response to exercise is summarised in table 4. On occasion I the ventilatory and cardiac frequency responses to submaximal exercise were essentially normal as was the maximal oxygen uptake and related indices. Compared with occasion I none of the indices of submaximal exercise differed on occasions II to IV and this was also the case for the maximal indices on occasions III and IV. Estimated $\dot{n}O_2$ max did not differ significantly between any of the four occasions.

Inspection of the accumulated results for each subject individually showed what appeared to be distinct patterns in three of them. Subject 1 on occasion II showed an increase in transfer factor and ventilation and cardiac frequency during submaximal exercise. The results were at their initial levels on occasions III and IV. Subjects 2 and 5 on occasion II showed a reduction in lung compliance, increase in recoil pressure, and increase in ventilation and cardiac frequency during submaximal exercise. These deviations were not apparent on occasions III and IV (fig 4).

Discussion

The present results were obtained in an established laboratory where attention could be given to calibration of equipment and procedures for measurement. But it entailed air travel, an overnight stay, and some inconvenience for the subjects who did not always attend in a basal state or see the need for full cooperation in tests entailing maximal effort. The measurement of compliance was a voluntary extra and only completed by four subjects. The consistency of the results for vital capacity as between FVC, IVC, and two stage VC provided evidence for reliability and this extended to the related flow volume curves and to the derived flow indices which had a high within-session reproducibility. The measurements of lung volumes, transfer factor, compliance, and the response to submaximal exercise were also technically satisfactory though in the case of transfer factor and exercise the results for subject I on occasion II were apparently invalidated by ethanol. Compliance measurement has a poor between-session reproducibility (coefficient of variation approximately 20%) and residual volume is less reproducible than other subdivisions of total lung capacity.[6] The degree of effort put into the measurements of respiratory pressures may not have been maximal in all instances and not all the measurements of maximal oxygen uptake met the definitive criterion (see methods). These considerations influenced the interpretation of some of the present results.

An increase in vital capacity after the deep dive was observed for the forced vital capacity, two stage vital capacity, and inspiratory vital capacity. For FVC it was on average 0·5 l above the mean of the values

Table 4 *Mean values for indices of exercise performance before and on three occasions after the dive*

	Before	*1 week*	*1 month*	*1 year*
Ventilation @ $\dot{n}O_2$ = 67 mmol/min (l/min)	31·8	33·3	30·3	32·6
Cardiac frequency @ $\dot{n}O_2$ = 67 mmol/min (min^{-1})	109	113	105	109
Tidal volume @ ventilation = 30 l/min (l)	1·58	1·73	1·80	1·65
Maximal oxygen uptake (mmol/kg/min)	1·82	1·68*	1·89	1·78

*Estimated from submaximal data.[5]

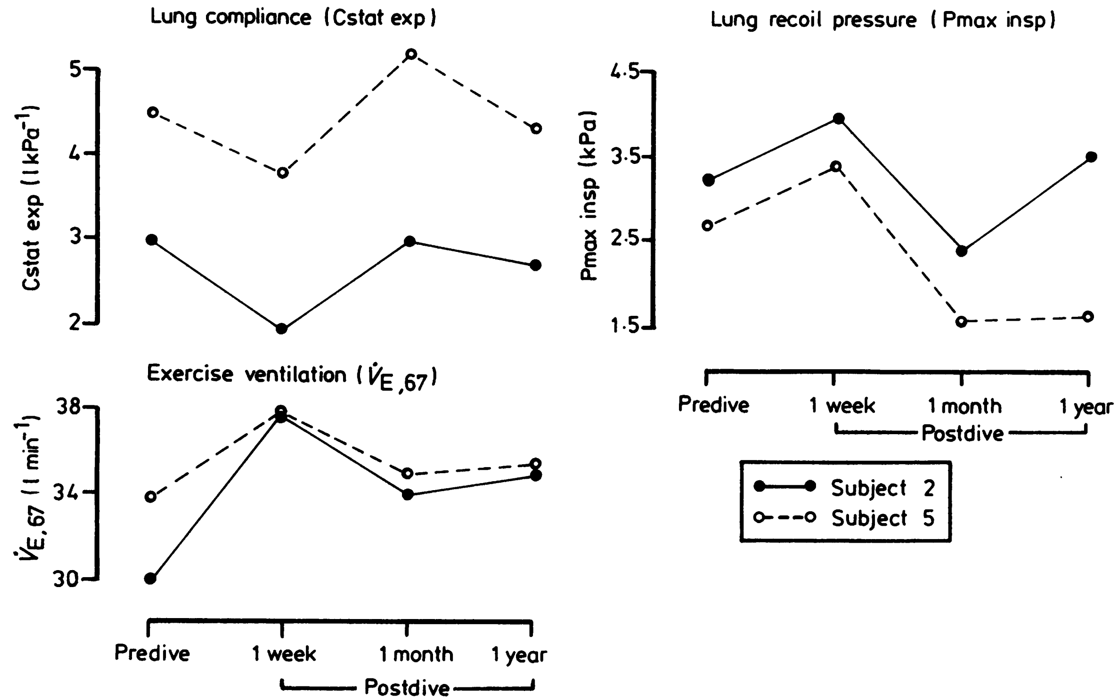

Fig 4 *Lung compliance, recoil pressure, and exercise ventilation in subjects 2 and 5.*

before and one year after the dive and at one month after the dive was almost halfway back to the initial level. On the assumption that recovery was exponential this gives a half time of about 28 days. The forced expiratory volume and peak expiratory flow rate increased respectively by 0·28 l and 0·7 l s^{-1}; the rate of recovery was similar to that for forced vital capacity. The improvement in ventilatory capacity after the dive did not extend to the ventilatory response to either submaximal or maximal exercise so the men did not appear to benefit from the change.

The forced expiratory flows during expiration when 50% and 75% of observed vital capacity had been expired were not significantly changed by the deep dive. These flow rates, however, are affected by lung volume which should be standardised if the rates on different occasions are to be compared.[8] In the present instance the volumes were those after 50% and 75% of forced vital capacity had been expired from total lung capacity on the first attendance. The corresponding volumes on the other occasions might have been obtained by whole body plethysmography but this was not performed. In its absence the correction was made assuming a fixed reference point which could have been either total lung capacity or residual volume. Use of these two reference points were associated respectively with increases in both flow rates and with a reduction in $FEF_{50\%FVC}$ but no change in $FEF_{75\%FVC}$. Therefore, the choice of reference point was important but made difficult by the absence of significant changes in either total lung capacity or residual volume when these volumes were measured independently by the helium dilution methods. There was no significant increase in maximal inspiratory pressure, which, if present, would have been in favour of total lung capacity increasing as a result of diving exposure. There was also no significant decrease in static lung compliance or increase in recoil pressure which, if present, would have been in favour of a reduction in residual volume. The absence of significant changes may have reflected the variability of the measurements and the small numbers of subjects. The increase in vital capacity, however, was probably mainly due to increased muscle strength[9,10]; this might have been expected to increase the total lung capacity but not the residual volume which would then have been the appropriate reference point. On this basis the flow rate at small lung volumes was not affected by the deep dive. The results for $FEF_{75\%}$ based on the observed FVC pointed to a similar conclusion and hence that small airway calibre and recoil pressure remained consistent. Thus the reduction in flow rates at small lung volumes observed in a previous cross sectional study[1] must have had some other cause.

The transfer factor and *K*CO both decreased significantly after the deep dive although a small increase in the former and decrease in the latter might have been expected on account of the increase in vital capacity also affecting the alveolar volume; in the event the increase in single breath alveolar volume was only 0·1 l, too small to affect the result materially. The reduction in transfer factor might have been due to the hyperoxia associated with the dive reducing the haemoglobin concentration. For this to have been the explanation, however, the concentration should have fallen by approximately 20% and there should have been complete recovery afterwards. Neither of these conditions were met (see below). The absence of changes in small airway calibre likewise militates against structural changes in the acinus being responsible.

In two individual subjects combinations of changes were observed which suggested abnormalities not shared by their colleagues. In subject 1 on occasion II a rise in transfer factor (against the trend) and in ventilation and cardiac frequency during submaximal exercise were probably due to heavy drinking during the night before the tests. In subject 2 the concurrence of transient changes in compliance, recoil pressure, and exercise ventilation in association with symptoms was suggestive of oxygen toxicity. A reduction in FVC might also have been expected; its absence was possibly due to predominance of the factors discussed earlier. Similar physiological changes occurred without specific symptoms in subject 5.

The present results were obtained on only seven diver/welders during a single deep dive. Thus the odds were against observing significant effects; indeed the absence of material effects has been commented on in a general account of the expedition which also involved 14 commercial divers investigated at two other centres (Y Giran *et al*, at VIII Symposium, European Undersea Medical Society, Marseille, 1984). The pooled results failed to demonstrate an unequivocal increase in vital capacity. This may have been due to several factors; the work undertaken during the excursions may have been more strenuous for the welders than for the other divers. The diving exposure during the weeks after the dive may have been more intense in the non-welders or the reproducibility of the measurements may have been insufficient. More information is needed on these points. By contrast the transfer factor was significantly reduced as a result of the dive in the whole group as well as in the present subjects. There was an associated transient reduction in venous blood haemoglobin concentration of approximately 6%; this might have been expected to cause an approximate 2% reduction in transfer factor due to less haemoglobin being available in the alveolar capillaries for combination with carbon monoxide in the test gas.[11] This reduction is materially less than the mean reduction in transfer factor of 9·6% observed between occasions I and II and consistent with the view that the two phenomena are not cause and effect.

Thus the present study shows that a single deep dive can increase the vital capacity and indices of forced expiratory flow at large lung volumes, including the peak expiratory flow rate and FEV_1 but not the flow rate at small lung volumes. The changes could have been due to increased muscle strength but in the five subjects in whom this measurement was made no increase was shown. Recovery afterwards appeared to have a half time of about 28 days. Transfer factor was materially reduced by the dive and although significant recovery occurred, this appeared not to be complete. The reduction was larger than could be explained by any concurrent reduction in haemoglobin concentration. One subject developed unequivocal features of oxygen toxicity.

These findings are evidence that a single deep dive can cause material pulmonary changes which may persist. The changes should be monitored by regular surveillance in a physiological laboratory.

Dr T L Fallowfield introduced the subjects. Dr D J Chinn, Miss J J Weller, Miss N G Bridges, and Mrs J Askew made some of the physiological measurements: Dr Y Giran supplied the haemoglobin concentrations: Mr C Elliott and Dr V Woolley contributed to the analysis of results. We are indebted to the Medical Research Council and the University of Newcastle upon Tyne for financial support.

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