Pulmonary mechanics and atelectasis during immersion in oxygen-breathing subjects

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Baer R, Dahlbäck GO, Balldin UI. Pulmonary mechanics and atelectasis during immersion in oxygen-breathing subjects. Undersea Biomed Res 1987; 14(3):229–240.—It has been suggested that vital capacity (VC) reduction seen during head-out immersion and oxygen breathing is due to atelectasis formation. In this study VC was reduced in 8 healthy subjects by 8.7% as an effect of immersion per se and by a further 14.3% as an effect of oxygen breathing during 30 min of immersion. Every 2nd min during the exposure, functional residual capacity (FRC), dynamic compliance (C-dyn), and static esophageal pressure at end-expiration (Poe-frc) were measured by body plethysmographic technique. Results were compared with an air-breathing, immersed control situation to evaluate any possible atelectasis formation. The only significant changes during immersion were observed after 30 min of air breathing, where FRC decreased linearly by 8.5% and C-dyn by 13.2%. The main conclusion is that atelectases acting solely as volume restrictors cannot explain the whole VC reduction without other additive or synergistic mechanisms. We suggest that the linear decrements in FRC and C-dyn as an effect of immersion time might be explained by either inspiratory muscle fatigue or increasing thoracic fluid compartment during the first 30 min of head-out immersion.

atelectasis  immersion
body plethysmography  oxygen
dynamic compliance  pulmonary mechanics
functional residual capacity  vital capacity

It has been shown that lung vital capacity (VC) in human subjects in erect body position diminishes during head-out immersion and oxygen breathing (1, 2). Head-out immersion creates negative pressure breathing by about 3 kPa (3), causing a lung volume reduction by the hydrostatic pressure difference over the rib cage and diaphragm. A considerable pooling of blood in the thorax also occurs (4). These two factors will induce airway closure and pulmonary gas trapping (5, 6). Breathing 100% oxygen when small airways are narrowed and eventually obstructed may lead to absorption atelectasis according to several investigators (1, 7–9). Positive-pressure breathing by 1.5–2.5 kPa in O2-breathing, erect subjects during head-out immersion, as well as in free swimming divers in prone position using a chest mounted, closed circuit, oxygen-breathing apparatus, has been shown to almost eliminate the reduc-
tion in VC (2, 10). Furthermore, the stress on the basal lung parts induced by immersion is similar to that caused by high acceleration force in a fighter aircraft when wearing an anti-G suit (11). If the pilot breathes 100% oxygen, atelectasis will develop as confirmed by x-ray (12).

The purpose of this study was to examine whether atelectasis can be responsible for VC reduction during head-out immersion breathing 100% oxygen, as previously suggested (1, 2, 10), and if so, to estimate the magnitude and time course of its formation. For this reason we followed, by body plethysmographic technique, the functional residual capacity (FRC), dynamic compliance (C-dyn), and static esophageal pressure at FRC (P_{es-frc}) every 2nd min during 30-min exposures.

MATERIAL AND METHODS

Eight healthy male scuba divers took part in the study. Mean (range) age, height, and weight were 25 (18–31) yr, 1.79 (1.70–1.87) m, and 72 (60–79) kg. Three of the subjects were moderate smokers (about 10 cigarettes/d).

The body plethysmograph was a pressure-volume type with pressure correction (13), with a total volume of 234 liter (Fig 1). In the wet experiments it was filled with water up to the subject's chin. Temperature of the water was kept at 35 ± 0.2°C.

Fig. 1. Schematic representation of the experimental set up. Cylinder at top decreases the temperature drift of the air-flow signal. Container at back illustrates the compensating chamber, eliminating sudden irrelevant pressure changes in the surroundings, such as door closure, etc.
which can be considered as thermoneutral (14). Volume and pressure changes in the body box were recorded by a differential pressure transducer (DP 103–10, Validyne, Northridge, CA) with pressure range ±90 Pa, inaccuracy 0.4 Pa. The transducer measured the air flow through a net screen (area 56 cm²) at the top of the box. The screen was electrically heated to avoid condensation of water. The flow signal was sampled at a rate of 100 Hz and subsequently integrated to volume by a microcomputer (ABC 800, Luxor, Sweden). Maximal flow that could be recorded was 3.75 liter/s with a resolution of 1.8 ml/s. The subjects breathed dry air or dry oxygen from outside of the box through a demand valve and an airway shutter unit which could close within 0.2 s by the computer during 5 s every time FRC was measured. The dynamic resistance of the open shutter, the demand valve, and connecting hoses was 0.09 kPa (0.9 cmH₂O) at a flow of 1 liter/s at expiration and 0.3 kPa (3 cmH₂O) at a flow of 0.7 liter/s at inspiration. The external dead space amounted to 150 ml.

Mouth pressure was recorded by a differential pressure transducer (Model 270, Sanborn Division, Waltham, MA) with range ± 0.4 kPa, inaccuracy 4 Pa. Esophageal pressure was measured against mouth pressure using the balloon technique of Milic-Emili et al. (15). The length of the balloon (Nolato AB, Torekov, Sweden) was 10 cm and it was filled with 0.5 ml of air when placed in position 38–42 cm from the nares. Phase shift between plethysmographic volume displacement and mouth pressure was corrected for each subject and each experimental condition separately and minimized (13). Temperature drift of the airflow signal was automatically corrected by a multiple regression analysis giving volume changes as a function of pressure and time. All signals were processed by the computer and fed into a 4-channel, strip-chart recorder (Gould, mod. 4400.06, Ballainvilliers, France) for continuous on-line recording.

To estimate the reproducibility of the FRC measurements, the relative SD of 15 consecutive measurements in dry environment was calculated. Breathing air, SD in relation to FRC was 2.25%, breathing oxygen, 2.77%. A corresponding procedure was carried out for C-dyn, giving a SD breath to breath of 15.00%. This higher value was mainly due to cardiac artifacts.

**Experimental procedure**

The subjects were told to avoid food known to produce excessive intestinal gas for 24 h before each experiment, because this could be a source of error in the measurements of FRC. To avoid aerophagia, chewing gum was forbidden. No physical exertion was allowed on the day of the experiment.

After insertion of the esophageal balloon, the subject, wearing swimming trunks, assumed an erect sitting position in the plethysmograph. Calibration, and in the wet experiments filling of the plethysmograph with water, took about 15 min before the measurements could begin.

Vital capacity was measured before and after each experiment, always with air as breathing gas. All VC maneuvers were performed with inspiration first and encouragement by the investigator. FRC was measured during 5 s of breathing maneuvers against closed shutter (two to three inspiratory and expiratory maneuvers applying a pressure of ± 1 to 2 kPa) every 2nd min throughout all experiments. Measurements of C-dyn and Pao2-frc proceeded during 15 s immediately before each shutter closure, thus recording two to five breaths (depending on breathing frequency) every 2nd min.
Tidal volume and breathing frequency were monitored during the 30 min each experiment lasted.

The following experimental conditions were studied: a) Air breathing, dry environment (air-dry). b) Oxygen-breathing, dry environment (O₂-dry). c) Air-breathing, head-out immersion (air-wet). d) Oxygen-breathing, head-out immersion (O₂-wet). In trial experiments the greatest changes occurred in the air-wet condition; therefore, after water had been let out of the plethysmograph (which took about 10 min), we followed immediately with condition e) air-breathing, dry environment, after immersion (air, postwet).

The subjects were instructed to avoid deep breaths throughout all experiments because this could influence any possible atelectasis formation. Each subject performed only 1 experimental condition each day, at the same time of the day on the different occasions. Experimental order was randomized.

Statistics and calculations

Paired Student’s t tests were used and the subjects served as their own controls. Results giving P < 0.05 are considered as statistically significant. Individual linear regression equations were calculated for FRC and C-dyn and P_{\text{dL}}-frc as these seemed to describe the time courses in the best way. Only the mean regression lines and equations for significant changes are presented, however. The individual slopes were also treated by variance analysis with orthogonal comparisons. Results are expressed as the means of individual changes in percent.

RESULTS

Vital capacity (Fig. 2)

The best measurement of 3 that preceded each experiment was chosen to represent the prevale. Immersion per se reduced VC by 8.7% (SD = 5.2%, P < 0.01), determined as difference between prevale in the air-dry, O₂-dry conditions, and prevale in the air-wet, O₂-wet conditions.

After each experiment the subject performed several VCs until reaching the prevale or until reaching a repeatable postvalue. Our postvalues are represented by the first VC recording after each experiment. Oxygen breathing during 30 min of immersion reduced VC further by 14.3% or 721 ml (SD = 10.1%; P < 0.01), determined as the difference between the prevale and the postvalue in the O₂-wet condition. Most of the subjects normalized within three to seven VC maneuvers with 1 min between each measurement. However, 1 subject still showed a deficit of 4.8% after performing seven VCs, while another subject, who was the only one who could not help taking several deep breaths of over 1.5 liter during the 30-min run, diminished his VC only slightly more than in the air-wet condition. All vital capacities were performed slowly, during approximately 15 s, to avoid coughing. Despite this, 5 of the subjects developed a cough or reported tightness in the chest or unspecified airway irritation.
Fig. 2. Vital capacity before and after the 30-min exposures. ** = P < 0.01; n.s. = not significant. Significance levels within parentheses bear on the effect of immersion.

Minute ventilation

Neither breathing frequencies nor tidal volumes showed any tendency to change over time during any of the environmental conditions, nor was there any noticeable difference when conditions were compared with each other.

Functional residual capacity (Fig 3)

As expected, no changes over time were observed in the air-dry and O₂-dry conditions, and the means did not differ significantly from each other. Therefore the dry means were pooled and compared with intercepts at 0 min of the regression lines in the 2 wet conditions to calculate the immediate effect of immersion on FRC. In our study, FRC diminished by 45.7% (SD = 5.4%, P < 0.001).

Changes over time during wet conditions: In the O₂-wet experiment, FRC diminished by 7.6% (SD = 4.9%) or 140 ml as compared to the larger decrement of 8.5% (SD = 4.5%) or 161 ml in the air-wet condition. If slopes of the mean regression lines
Fig. 3. The mean regression lines showing the changes in FRC (left) and C-dyn (right) (liter/kPa) over time (min) during the 5 experimental conditions. The only statistically significant changes occurred in the air-wet experiment, with the following regression equations: FRC = 1.91 + 5.38 · 10^{-3} · min (P < 0.01); C-dyn = 1.23 − 5.4 · 10^{-3} · min (P < 0.05).

are tested against the zero slope (no change over time) hypothesis, the FRC reduction in the air-wet condition is significant (P < 0.01), whereas FRC reduction in the O_2-wet condition is not. Comparison of the two conditions showed no statistical significance. These findings suggest that if atelectases are formed during 30 min of immersion and oxygen breathing, they do not decrease the functional residual capacity. Furthermore, orthogonal comparisons in variance analysis showed that the 2 wet conditions differ significantly from the corresponding dry conditions (F = 9.18, P < 0.01). This implies a decline in FRC during 30 min of immersion independent of breathing gas (air or oxygen). It should also be pointed out that the changes over time were indeed linear.

Considering the whole group of subjects, FRC had a tendency not to normalize during the immediate 30-min dry followup (air, postwet condition). FRC stayed 4.7% (SD = 7.1%) or 174 ml below the mean air-dry value, calculated as the difference between means of the air-dry and air, postwet conditions. This tendency to sustained reduction in FRC level was not statistically significant.

Dynamic compliance (Fig 3)

The immediate effect of immersion per se was a decrease by 36.2% (SD = 14.3%; P < 0.001) obtained as the difference between pooled means of the air-dry/O_2-dry
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conditions and the pooled intercepts at time 0 min of the mean regression lines in the 2 wet conditions. As with FRC, no significant changes occurred over time during the dry conditions, nor did the means differ from each other.

Changes over time during the wet conditions: In the O₂-wet experiment, C-dyn diminished by 4.1% (SD = 11.1%) as compared to a larger decrement of 13.2% (SD = 11.8%) in the air-wet experiment. The slope of the air-wet mean regression line differs significantly from zero slope (no time change) hypothesis (P < 0.05), which is not the case in the O₂-wet condition. The slopes are not significantly different from each other. Orthogonal comparisons in variance analysis revealed significant difference between the air-dry/O₂-dry and air-wet/O₂-wet conditions (F = 7.33; P < 0.025) whereas no difference was detected comparing both the oxygen-breathing conditions with the corresponding air-breathing conditions.

These results coincide with the changes observed in FRC, suggesting a decrease in C-dyn over time during 30 min of immersion, independent of breathing gas. Also, there was no effect of oxygen-breathing on C-dyn during 30 min of immersion.

During the 30-min air, postwet condition there was a tendency for C-dyn to remain at a lower level as compared to the air-dry condition. This difference was not significant. A normalizing trend occurred during the 30 min after immersion.

Static pleural (esophageal) pressure at FRC (Pc-r-frc)

We observed small and insignificant declines in Pc-r-frc over time in the 2 wet conditions, from +0.24 to +0.19 kPa breathing air, and from +0.22 to +0.13 kPa breathing oxygen. No correlation was found comparing individual decrements in Pc-r-frc with decrements in VC.

DISCUSSION

We used the body plethysmographic method for measuring lung volumes, including trapped air in nonventilated lung regions, because a lung volume reduction measured by this method should reflect a true loss of air volume such as atelectasis. Repeated measurements over a short time period are also possible in contrast to a gas dilution method. Intestinal gas may be a source of error using the body plethysmographic technique. Depending on how much the diaphragm participates in each breathing maneuver against a closed shutter, the measurements may lead to an over- or underestimation of true lung volume (16, 17). Inspiratory maneuvers may result in slight underestimation, whereas expirations may lead to overestimation of the true lung volume by the amount of intestinal gas or even more (16, 17). As our subjects performed both inspiratory and expiratory maneuvers, our high correlation coefficient (0.99–0.9999) between the box flow and mouth pressure signals demonstrated that intestinal gas did not influence our FRC measurements to any appreciable degree. Also, our FRC measurements were too highly reproducible to allow for a significant error of this type. It has been shown that the mean amount of intestinal gas in 60 digestively healthy subjects is only 115 ml (SD = 127 ml) (18), and introduction of up to 600 ml of air into the stomach or colon causes only small and inconsistent errors in the lung volume measurements (19). There is also good agreement in FRC mea-
measurements during dry conditions in healthy subjects between the body plethysmographic and gas dilution methods (5, 19).

During the FRC-measurement periods we used a breathing frequency of 24–36/min with voluntary airway pressures of ±1 to 2 kPa against the closed shutter. We do not know whether these positive or negative pressures, applied during 0.5–1 s at a time by the chest and diaphragm, might have expanded any possible atelectases. Theoretically, this seems unlikely because the pleural pressure rises by the same amount as the intraalveolar pressure, but we cannot exclude that the respiratory maneuvers during the FRC measurements might have influenced a possible alveolar collapse. However, as we still noted a considerable reduction in VC, either atelectases were not formed or the breathing maneuvers did not influence atelectases formation.

Immediate effects of immersion per se

Vital capacity decreased by 8.7%, which is in the same order of magnitude as in most of the earlier investigations, observing a reduction of 6.5–10% (20, 21). FRC decreased by 46% as an immediate effect of immersion, which is in the same order of magnitude as reported by Agostoni et al. (46%) (22) and Dahlbäck (41%) (21), although FRC decrement variability as big as 30–60% is stated as possible (23). Cdyn fell by 36% immediately upon immersion. This is in agreement with the 37% reduction reported by Dahlbäck (21) and the 34% reported by Sterk (24) when calculating the difference between group means. However, Sterk’s subjects wore scuba, which may have caused an even further reduction in FRC, thus decreasing Cdyn more than immersion per se. However, FRC was not measured in that study.

Effects of O₂ breathing during 30 min of immersion

Vital capacity decreased in our study by 14.3%, on top of the effect of immersion per se. This may be compared to the 22.4% reduction after 2 h of O₂ breathing while immersed reported by Baldin et al. (1). They put forward the hypothesis that this VC reduction might be due to atelectasis formation, although they did not exclude additional factors such as edema or histotoxic effects of oxygen. They arrived at this conclusion mainly by analogy to the increased gravitational force situation, wearing an anti-G suit, breathing oxygen. Both these otherwise different environments will compress the small airways, affecting the basal parts of the lungs in a similar way (11). Several investigators have demonstrated that increased G-force in combination with anti-G suit inflation and O₂ breathing induces atelectases (11, 12, 25–28). VC has also been reported to be a sensitive index of G-atelectasis formation (11, 12).

Later, pulmonary gas-trapping of 200–300 ml at FRC has been demonstrated during immersion and air breathing (5, 6), which in light of the established concept of absorption atelectasis when breathing oxygen gave support to the alveolar collapse theory even in the immersed environment. Another argument seems to be the countering effect of positive pressure breathing found by Dahlbäck and Baldin (2, 10). In one of their studies, VC decreased by as much as 42%, while this reduction was almost eliminated by application of positive pressure of about 1.5–2.5 kPa to the airways. Positive pressure breathing is also efficient in countering G-atelectases (29).

As the purpose of this investigation was to study the time course of the possible atelectasis formation, we did not want to disturb it by inappropriate breathing mane-
vers in addition to the shutter closures, therefore choosing FRC as the only measure of lung volume. A slight fall in this volume was expected and because of the volume loss a consequent fall in C-dyn. The insignificant reduction in FRC in the O₂-wet experiment indicates that if atelectases of a moderate degree are formed, they do not affect this lung volume. Green and Burgess (12) found atelectasis-induced VC reduction of 40% in 6 subjects exposed to increased G-force, wearing anti-G suits, and breathing oxygen. The mean FRC reduction in their study was 17% but only 2 individuals diminished their FRC significantly. It seems therefore that atelectases may be compensated for. The most likely mechanism for such a compensation would be by expansion of still opened and ventilated alveoli as a result of a decreased pleural pressure created by the collapsed alveoli. To predict the behavior of C-dyn in such a situation, the ventilated and collapsed alveoli may be considered separately. Collapsed units give rise to a volume loss diminishing C-dyn. (This is the case when atelectasis decreases FRC.) The ventilated units are pulled by the decreased pleural pressure expanding to a higher volume, and thus maintaining FRC at the same level. These alveoli may now be more compliant [about half of the difference in compliance between dry and wet environment is caused by the lung change alone (30)], compensating for the C-dyn decrement caused by the collapsed respiratory units. There is a theoretical possibility for C-dyn to remain on the same level with developed atelectasis and unchanged FRC. However, for a compensatory expansion of ventilated alveoli, a decreased pleural pressure is necessary. This decrease may be theoretically calculated to be about 0.6 kPa, with a mean C-dyn in water of 1.2 liter/kPa if atelectases were to explain the whole VC reduction of 720 ml. We observed basically very similar behavior of esophageal pressure during the air-wet and O₂-wet conditions. No statistical significance was reached and therefore we cannot conclude that the decreasing tendency of 0.9 cmH₂O in Pₐr-frc in the O₂-wet experiment is a result of atelectasis formation. The lack of correlation between the individual decrements in Pₐr-frc and VC also goes against the atelectasis theory.

Furthermore, in our subjects pulmonary nitrogen was not washed out by deep breaths of oxygen before starting the O₂-wet run. According to Dahlbäck (6) and Bondi et al. (31) some airways remain closed during tidal ventilation when immersed. Therefore it cannot be excluded that some of our subjects were not washed out of pulmonary nitrogen properly during the whole experiment, thus reducing the possibility of absorption in the closed-off regions as an atelectases-forming mechanism.

In summary, the qualitatively and quantitatively very similar and small changes in FRC, C-dyn, and Pₐr-frc, breathing air or oxygen during immersion, suggest that atelectases solely as a volume restrictor cannot explain the relatively large VC reduction during immersion breathing oxygen. However, there is still a possibility that small, by our method undetectable, atelectases might be only the first factor in a chain of events eventually decreasing the VC considerably more. Terms such as "miliary collapse" and "dispersed absorption collapse" are used to describe individually small (radiographically nonapparent) but widely scattered atelectases during anesthesia as well as during oxygen-breathing in the elderly (32). Further, such small atelectases might retract the bronchiolar linings of their airways, thus causing irritative friction and in turn setting off a neural reflex preventing a full inspiration. The cough and airway irritation so often experienced after oxygen breathing during immersion might perhaps support such a speculation.
Effect of time during immersion

Significant declines in FRC and C-dyn according to the t tests were observed only when breathing air. Orthogonal comparisons in variance analysis emphasized this time effect of immersion. Over the 30-min air-breathing, FRC was brought down by 161 ml or 7.6% and C-dyn by 13.2%. Sterk (24) noted a decline of 21% in C-dyn during 25 min of upright immersion. Regarding FRC, however, we found nothing in the literature with which to compare our result.

Trying to explain the continuous FRC decline, it is possible that circumference of the chest wall decreased, or the thoracic fluid compartment increased, or a combination of these two factors occurred. Sterk (24) considered it possible that during prolonged periods of negative-pressure breathing, inspiratory muscles could become fatigued. However, no such evidence has been brought out yet. There is an indication that pulmonary capillary blood flow and pulmonary diffusing capacity may increase during the first 0.5 h of seated immersion (33). Also, total plasma volume has been found to increase by about 300 ml during the first 30 min of immersion when the hypervolemia is maximal before significant diuresis occurs (20). Therefore, the other possible explanation for the slightly falling FRC and C-dyn during the first 30 min of immersion is slightly increased thoracic fluid compartment, either intravascularly or interstitially in the lung.

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Baer R, Dahlbäck GO, Baldin UI. Mécaniques pulmonaires et atélectasie durant l’immersion chez des sujets respirant de l’oxygène. Undersea Biomed Res 1987; 14(3):229–240.—Il a été proposé que la réduction de la capacité vitale (CV), observée durant l’immersion jusqu’au cou et la respiration d’oxygène, est attribuable à la formation d’atélectasie. Dans cette étude, la CV chez 8 sujets en santé fut réduite de 8.7% par un effet de l’immersion en soin, et de 14.3% additionnel par un effet de la respiration d’oxygène pendant 30 min d’immersion. À chaque 2 min pendant l’exposition, la capacité résiduelle fonctionnelle (CRF), la compliance dynamique (C-dyn) et la pression statique oesophagienne en fin d’expiration (Pş,crf) furent mesurées au moyen de la technique de la pléthysmographie corporelle. Les résultats furent comparés avec ceux d’une condition d’immersion témoins sous respiration à l’air afin d’évaluer toute formation possible d’atélectasie. Les seuls changements significatifs durant l’immersion furent observés après 30 min de respiration à l’air, temps auquel la CRF et C-dyn diminuèrent linéairement de 8.5% et 13.2%, respectivement. La conclusion principale est que les atélectasies agissant uniquement en tant que restricteurs de volume ne peuvent expliquer l’entière réduction de la CV sans l’intervention d’autres mécanismes additifs ou synergistes. Il est suggéré que les diminutions linéaires de la CRF et C-dyn par un effet de temps d’immersion peuvent être expliquées soit par une fatigue des muscles inspiratoires ou une augmentation progressive de liquide dans le compartiment thoracique durant les 30 premières minutes de l’immersion jusqu’au cou.

atélectasie
pléthysmographie corporelle
compliance dynamique
capacité résiduelle fonctionnelle

immersion
oxygène
mécaniques pulmonaires
capacité vitale

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