Effect of the Hydro-Lab Environment on Pulmonary Function

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INTRODUCTION

It has been known for a number of years that if a person breathes pure oxygen, after about 12 hours he will begin to develop chest pain. Within 24 hours he probably will develop a serious case of pneumonia, and if he continues to breathe pure oxygen it can only culminate in death. This is one form of oxygen toxicity. It is well recognized, therefore, that oxygen can become a deadly poison to the body if the body is exposed for lengthy periods to oxygen equal to 1 atmosphere absolute pressure (ATA), which is equivalent to breathing pure oxygen at ground level. The fact that people can, for short periods, breathe pure oxygen at more than 2 ATA shows that oxygen toxicity depends not only on the partial pressure of oxygen, but also on the length of time the lung is continuously exposed to it. A thorough understanding of the body's time and pressure tolerances to oxygen is essential in saturation diving, in the use of oxygen in chamber treatment of bends or gas gangrene and for decompression schedules.

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Recognition that exposure to elevated partial pressures of oxygen can result in serious physiological damage has caused many workers to become extremely cautious in its use and, indeed, many attempt to avoid any exposure to elevated oxygen partial pressures, except for standard diving, for well-tested decompression profiles or for oxygen treatment tables. They are particularly cautious when the elevated oxygen exposures are to be carried out over long periods of time.

It is not surprising, therefore, to find that from time to time concern has been expressed over the possible adverse effects from extended exposure to the elevated partial pressures of oxygen present in shallow-water saturation diving such as that found in Hydro-Lab. If such habitats employ compressed air, at a 45-ft depth the mean oxygen pressure to which the aquanaut is exposed is approximately 363 mm Hg, or 0.47 ATA, continuing in some cases for 7 days.

To place this problem in its proper perspective, we must first determine to what level the partial pressure of oxygen can be raised without creating symptoms of physiological damage in the aquanaut. Obviously, this value will fall somewhere between 0.2 ATA (160 mm Hg or 20.9% O_2 at ground level), which is that found in our atmosphere and which we consider normal, and 1 ATA (760 mm Hg or 100% O_2 at ground level), at which point toxicity clearly becomes evident within 12 hours.

Although few critical studies to answer this question have been conducted during operational diving, it has frequently been studied in the laboratory. Lambertsen and his associates centered one such study on vital capacity changes which led them to conclude that man can be exposed for an indefinite period of time to oxygen partial pressures of 380 mm Hg. This is equal to that created by breathing 50% O_2 at ground level (0.5 ATA). Even with extended exposure they detected no reduction in vital capacity. This would seem to suggest that an aquanaut should, therefore, be able to live on compressed air for an indefinite period of time at a depth equivalent to 45.9 ft of sea water (FSW) without experiencing any pulmonary deterioration. Many workers are not convinced, however, that this can be applied in operational saturation diving. For example, although the Tektite II habitat was within this depth range, the concentration of oxygen in the habitat environment was reduced to 9%, which, at 40 FSW, would make the oxygen partial pressure equal to that at ground level (normoxic). No pulmonary function studies were carried out on the aquanauts in the Tektite habitat, but there is no reason to presume that Tektite divers suffered any reduced respiratory function.

On the other hand, the use of artificial breathing mixtures in a habitat environment increases by manyfold the cost and the technical difficulties of maintaining a life-support system. Such a system requires techniques for monitoring and maintaining the predetermined level of oxygen, scrubbing CO_2, and if the inert gas is recovered in a closed system, it may require the removal of trace contaminants such as methane, H_2S, CO, etc., produced by the body itself. Any system using an artificial gas mixture requires constant monitoring by a highly trained team of technicians, as well as the operation of many types of sophisticated equipment. This is an expensive penalty to pay both in terms of dollars and time if it is not necessary.

It is clear that if a habitat can utilize compressed air without endangering the health or performance of its occupants, most of the life-support problems can be greatly simplified. A complex gas mixing and monitoring system can be replaced with a simple, low pressure, high-volume air compressor. Since the habitat can be continuously ventilated, oxygen will be automatically supplied and CO_2 removed. The only additional requirement is a simple standby emergency life-support system. Such a facility is, however, limited in the depth at which it can be safely operated. This depth limitation would seem at this time to be about 50-55 FSW (that is, from the entrance hatch to the surface).

Even though Lambertsen's work appears to be above reproach and the Hydro-Lab facility has been in operation for several years without any suggestion of physiological problems arising from the gas mixture, the use of compressed air for saturation diving at a depth of 40-45 ft still has been questioned by some who believe that a 7-day exposure to oxygen partial pressures found at this depth range is contraindicated.

We have therefore undertaken pulmonary studies covering two 7-day saturation missions. Four subjects have thus far been studied, representing a total of 2016 man-hours of bottom time.

METHODS

A preliminary investigation was undertaken to determine if there were vital capacity changes resulting from a 7-day operational exposure to compressed air. Vital capacity measurements were made on each of the three team members less than 1 hour before descent to the habitat, and again within 2 hours after arriving in the habitat. Final measurements were made 2 hours after return to the surface

Subject	Pre-dive	Habitat	2 Hours Post-dive	Post-dive Changes (%)
WS	4.75	4.23	4.74	+ 3.9
LF	5.62	5.05	5.86	+ 0.2
WF	3.70	3.05	3.85	+ 3.9
	In			

Table 1. Vital Capacity Measurements in Liters.

at the end of the 7-day mission. Table 1 shows the result of these determinations.

The second and more definitive study was made two months later and included two of the original three subjects. This second study included the following respiratory measurements:

1. Forced vital capacity (FVC).
2. Maximum voluntary ventilation (free) (MVV).
3. 0.5 sec forced expiratory volume (FEV 0.5).
4. 1.0 sec forced expiratory volume (FEV 1.0).
5. Inspiratory vital capacity (IVC).
6. Mid-expiratory volume (FEV 25-75).

Since neither the habitat entrance nor the dry transfer pots would permit proper transport of a standard-size respirometer of the tank type (Collins), a smaller spirometer (Spirostat*) was used. This unit has the overall dimensions of only 4x4½x 11 in. It thus was easily transported to and from the habitat. This unit is an indirect measuring device. It employs a rotor located in the mouthpiece. Its rotation, which corresponds to airflow, is optically measured and processed electronically. The results are recorded as a volume-time curve on Polaroid film. Analysis of these records is not difficult and was accomplished in the habitat immediately after the measurements were made. The respiratory function measurements outlined above were made before, during and after the mission. Some of these are shown in Figs. 1-4.

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DISCUSSION

It can be seen from Table 1 that even after 7 days exposure to oxygen at a partial pressure approaching 0.5 ATA, two of the three subjects actually showed an increased vital capacity of 3.9% and 4.1%, respectively. The other subject showed no change. These results would tend to indicate that the laboratory findings of Lambertsen and others can be extrapolated to an operational situation and that increasing the partial pressure of oxygen to 0.5 ATA does not cause a deterioration in this pulmonary function even under the conditions of hard work to which the aquanauts were exposed. It was recognized, however, that other more subtle changes might not be reflected by vital capacity measurements alone. It was for this reason that we felt it necessary to examine dynamic volume-flows. We believed that these measurements would permit us to assess the total respiratory system including the respiratory muscles as well as the lungs. We would therefore be able to see the effects of such things as pulmonary edema, bronchial changes, changes in lung elasticity and other related deterioration in the total pulmonary system. These measurements would not, however, measure gas exchange.

The second study thus attempted to be more definitive by examining the dynamic function of the lung system. It should be noted that the subjects used in this study were not uniform. Indeed, they covered a rather wide range of age and physiological status. One subject, age 31, was a typical young, healthy, athletic diving scientist in excellent condition and with

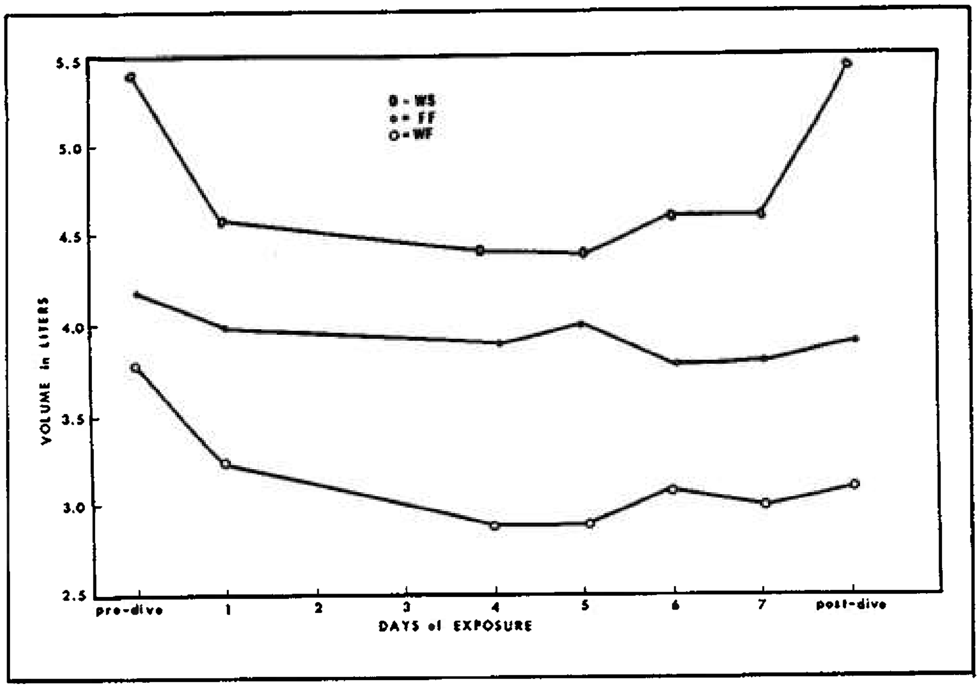

Fig. 1. Forced vital capacity.

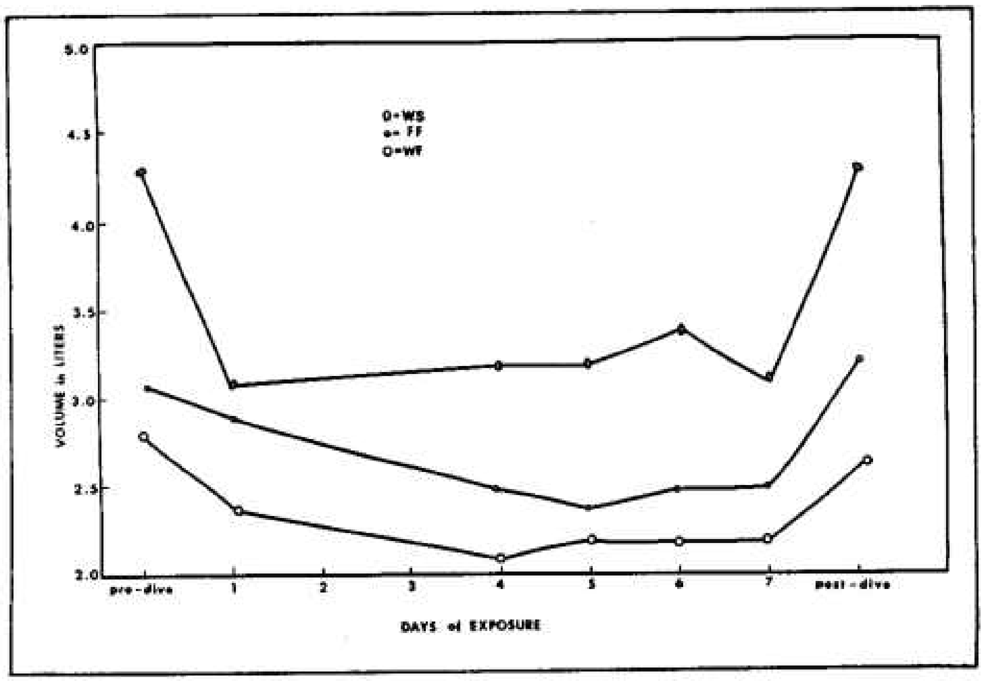

Fig. 2. Forced expiratory volume in 1 sec.

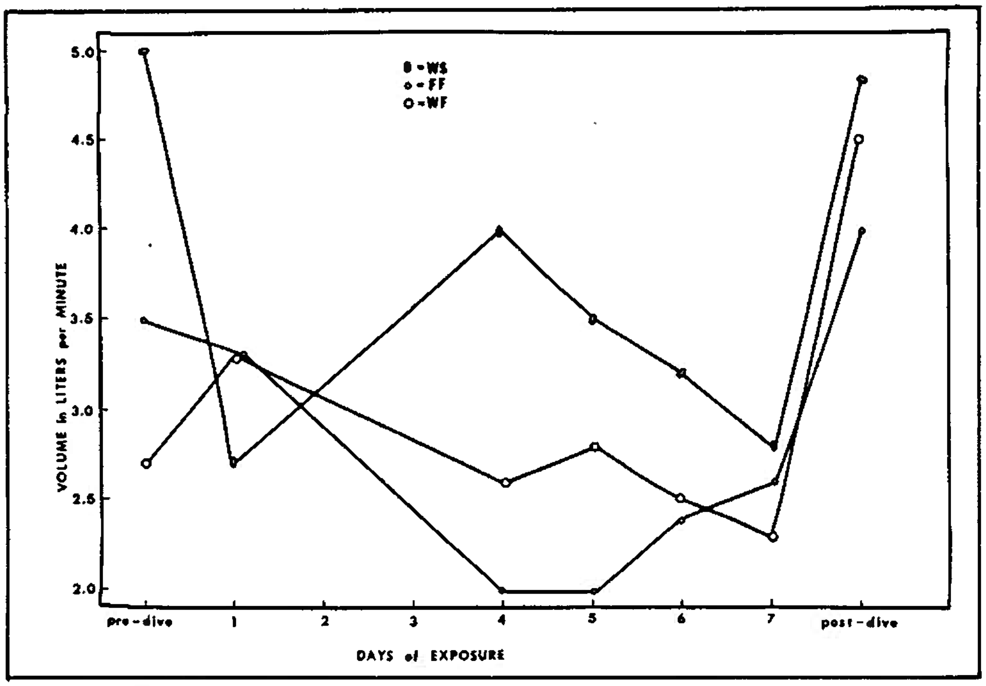

Fig. 3. Forced mid-expiratory flow.

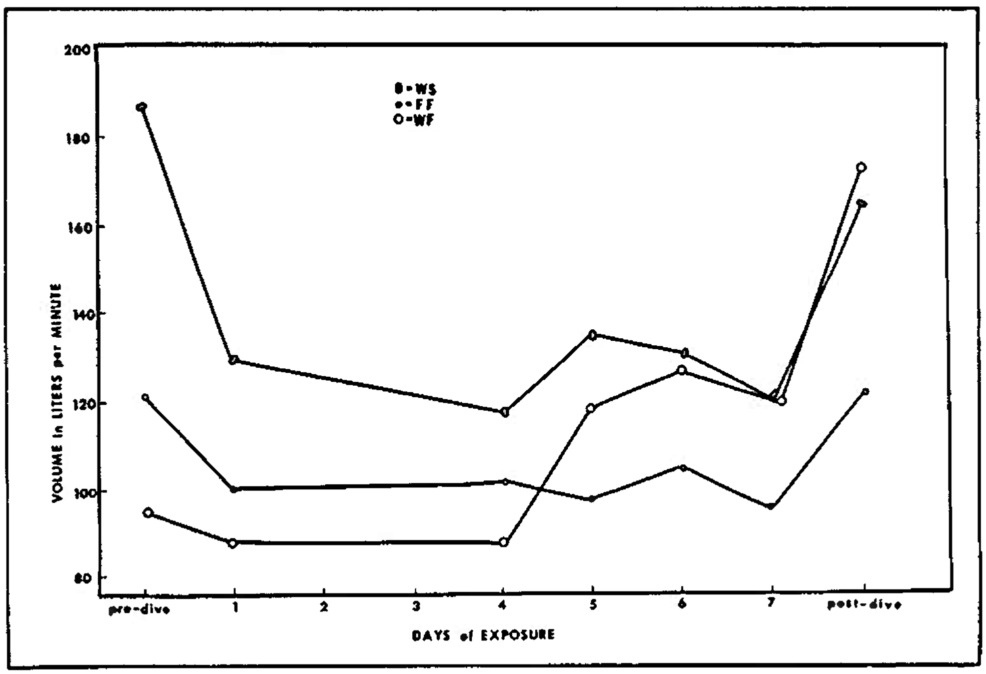

Fig. 4. Maximum voluntary ventilation.

no recent history of illness. The second subject, age 55, also an experienced diver, had a history of mild osteoarthritis (well controlled by aspirin). He was otherwise healthy and in good physical condition for his age. He probably represents the upper age level among active saturation divers. The third subject, age 27, a highly competent scientific diver, had recently recovered from a moderately serious lobar pneumonia that had resulted in some temporary diaphragmatic adhesions. These adhesions had been resolved before the saturation dive. He was pronounced in good physical condition by his physician and had been on a rigorous exercise program prior to the dive.

It may be seen from Figs. 1-4 that there was an immediate and significant decrease in all elements of the respiratory function measurements upon exposure to the increased air density of the habitat. Experiments now are being conducted to determine how much (if any) of this decrease is due to increased air density on the rotation of the spirometer rotor and how much to a bona fide decrease in pulmonary performance. Either or both is possible. However, a study of two subjects who also were a part of the first dive shows that during the first dive they had a vital capacity reduction of 17 and 8.8%, respectively, immediately upon reaching the habitat. Since the spirometer used at that time was a direct measuring device and therefore measured functional changes, we would expect that virtually all of the initial changes shown in Figs. 1-4 were due to functional changes in the subjects as a result of increased air density. However, regardless of the cause of this initial decrease, since it occurred immediately upon descent, it is reasonable to conclude that it does not represent a deterioration of pulmonary function as a result of oxygen toxicity. We therefore feel justified in considering as our baseline the first series of measurements made within a few minutes after reaching the habitat.

We recognize that the data may be attenuated because of the small number of observations. Nevertheless, some tentative conclusions may be drawn by *Groups by Trials Analysis of Variants*. Stated simply, this technique in effect compares each observation with every other observation and asks if there is any significant difference between them. This analysis, which is somewhat tedious indicated that the observations from the fourth to the seventh day of exposure are not statistically significantly different from each other. On the other hand, these observations are significantly different from those made immediately after reaching depth. This would suggest that while there was a subtle but still significant reduction in pulmonary function sometime during the first 3 days of habitat exposure, the respiratory system stabilized at that time and further deterioration did not seem to occur.

In examining our results we should remember that the dynamic measurements carried out during the second saturation dive reflect the mechanical function of the total pulmonary system, not just one component such as the static vital capacity carried out on the first mission. These dynamic functions do not provide any indication of gas diffusion. Most workers believe, however, that any significant amount of oxygen toxicity would be reflected in some of the measurements conducted.

The question should be asked as to whether the apparent deterioration in pulmonary function found during the first 1 - 3 days of hyperbaric exposure is sufficient to cause alarm. As indicated above, it cannot yet be said how much of the changes represent changes in function of the spirometer, a change in pulmonary function, or both. This question might be considered from two views. First, although studies have not been completed to show what the normal range of function should be at the pressures at which these tests were made, it seems reasonable to assume that approximately the same variability from normal mean values is permitted in the hyperbaric environment as at 1 ATA. If so, the changes noted after the first day are well within normal limits. On the other hand, even if the immediate changes seen upon reaching the habitat do indeed represent reduced pulmonary function, these changes must be due to the increased density of the breathing mixture and not to oxygen toxicity or some other physiological deterioration. If this is the case, similar changes should be seen in any aquanaut exposed to breathing mixtures of the same molecular density. This would suggest that the same changes would have been found in Tektite aquanauts since the density of their breathing mixture closely resembled that found in Hydro-Lab. We are not aware that any of the aquanauts from either the Tektite or Hydro-Lab habitats felt that the increased breathing gas density affected their performance in any way. Indeed, although the increased density might be easily measurable by sophisticated techniques, it would be doubtful if such small changes reflected here could be detected subjectively by an individual exposed to such slight increases in breathing gas densities.

We do not know at this time if the stabilization in respiratory function seen to occur sometime during the first 3 days represents an adjustment process in which the respiratory system learns to cope with a more dense breathing gas, or whether perhaps at that point the respiratory system is repairing pulmonary deterioration as rapidly as it is produced. If the former is the case, the adaptation should be permanent while at depth. If the latter is the case, there is no indication that the body cannot continue to cope with this problem for an indefinite period. It would seem wise, however, to examine this question in more detail by a longer exposure.

Special attention should be given to the pre-dive and first two post-dive MVV values for subject WF. It may be noted that bronchiospasm developed during these maneuvers, both pre-dive and to a lesser extent on the first two MVV maneuvers after reaching the habitat. The MVV test is a rather taxing experience for a subject. It is interesting to note that this value returned to normal in the habitat environment. WF is allergic to tobacco smoke. For the first two days after arriving at the dive site, he continued to feel the effects of exposure to tobacco smoke that took place during the plane flight to the Bahamas. Although it is not common for divers (or the population at large) to complain about forced exposure to smoke in enclosed spaces, it is probable that a significant number of people would show the same reaction if tested. In any event, the fact that an individual who has a rather reactive respiratory system found an improvement in some of his respiratory function measurements during the long-term exposure to the habitat environment would further indicate that this environment is not stressful on the pulmonary system.

CONCLUSIONS

We must first of all acknowledge that there are several questions concerning the physiological effect of the Hydro-Lab environment on the diver which we cannot yet answer. It seems clear, however, that the operational use of compressed air at pressures equivalent to 45 FSW for at least 7 days is not contraindicated and produces no progressive or alarming changes in the respiratory system of man. Indeed, although it is not cogent to our present discussion, it would seem that, as unintentionally illustrated by our data, the routine inescapable exposure to tobacco smoke constantly faced by the general population produces more deleterious effects on the respiratory system than a 7-day exposure to the Hydro-Lab environment. It is clear, therefore, that the laboratory tests conducted by Lambertsen and others can be applied to the operational situation such as we described. There appears to be no good evidence of oxygen toxicity.

The question now raised is whether or not a 14-day exposure to the Hydro-Lab environment is contraindicated. We see no evidence from our data that there is any reason why an aquanaut should not be exposed to these conditions for a 14-day period, or perhaps even longer. Since the capability exists for daily monitoring of subjects, should any adverse effects begin to be manifested, the operation could easily be aborted before serious damage developed.

We believe that a 14-day test should be carried out, and further, that these data should be extended to the female diving population, since it is to be expected that hyperbaric pulmonary function values for women are significantly different from men and cannot be extrapolated from data derived from men.

Finally, since the pulmonary function studies described above are not difficult to carry out, consideration might be given to testing all aquanauts periodically throughout a mission to assure that no unrecognized pulmonary deterioration takes place during saturation.