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CHAPTER 7

ARE DIVERS REALLY DIFFERENT?

During the sixties and seventies there was a remarkable surge of "near folk lore" about differences of the physiological behaviour in divers. These differences were first proposed in informal discussions and later in more formal symposia and journals. In essence the proposal was that divers developed an insensitivity to raised tensions of carbon dioxide and a low ventilatory response to exercise. It was suggested that this was an "adaptation" to the underwater conditions. One enthusiast declared "the underwater man has arrived!". The obvious advantage of reduced ventilatory volumes on exercise is the reduction of respiratory work, particularly with increased gas density and the added resistance of respiratory apparatus. The disadvantage is that the resultant rise of carbon dioxide tensions would increase the risks of carbon dioxide narcosis, of accelerated oxygen poisoning and of the accentuation of nitrogen narcosis.

Nevertheless it became widely considered that all divers developed this feature to different degrees. Those with the most severe degree were titled "carbon dioxide retainers". Some thought that these changes were "for life". Others claimed that they could be reversed by a short period (three months) of abstinence from diving. One worker reported the purported "adaptation" of reduced exercise ventilation and rise of end-tidal PCO_2, with reduction in respiratory discomfort, in a non-diving subject during his second exposure to work at pressure. It was also suggested that, as with oxygen poisoning, the carbon dioxide sensitivity could vary from day to day in a particular diver. Another worker claimed to have shown impaired ventilatory response to exercise in subjects who had been retired from diving for over ten years. He proposed that either their physiological behaviour had changed permanently or that they were

inherently less responsive to exercise and carbon dioxide. It was even suggested that such people might have been unconsciously "self-selected" as divers.

Although these diffuse hypotheses are highly arguable it is, as always, difficult to counter them except by a disciplined and somewhat laborious examination of the evidence. The author has therefore reviewed and commented on a number of frequently cited "accepted" references, mostly those supporting "the difference" of the diver. This review must be, perforce, a limited one and, no doubt, some important references have been omitted, particularly very recent ones. It is for browsing rather than straight reading. I hope the reader will agree that, in these hurried days, a careful and detailed review of well known references can be a useful and sometimes a surprising exercise.

NON-DIVERS AND DIVERS IN UNDERWATER SWIMMING STUDY

Goff & Bartlett (1957, (ii))

This much quoted work showed that trained oxygen breathing underwater swimmers had elevated exercising end-tidal PCO_2 levels in contrast to untrained swimmers, who had normal levels of exercising end-tidal PCO_2. These authors used a circulating channel of a model testing basin. By varying the speed of water circulation the underwater swimmer could be studied in situ, although swimming at different speeds (0.6, 0.7, 0.8, 0.9 and 1.0 knot). Three or four tests were made at each speed on each individual. The exercise was continued for 20 minutes and final measurements taken when a steady state was achieved. The six trained swimmers (age 28-31) were all graduates of the US Navy UDO TWO Training Programme. The untrained swimmers (number not given) were laboratory personnel (age 29-47).

The trained swimmers showed end-tidal PCO_2 levels as follows (mean values): 0.6 knot, 46 mm Hg. 0.7 knot, 48 mm Hg; 0.8 knot, 50.5 mm Hg; 0.9 knot, 51 mm Hg; 1.0 knot, 52 mm Hg. In contrast, the mean end-tidal PCO_2 of the untrained swimmers was 37 to 38 mm Hg at all these speeds.

The authors reported slow deep breaths in the trained swimmers. Four of these also had prolonged post-inspiratory pauses (skip breathing). A figure was shown in this article in which end-tidal PCO_2 (40 to 65 mm Hg) was inversely related to the oxygen ventilatory equivalent (26 to 14 1/per

litre oxygen uptake). However of the 64 points with end-tidal PCO_2 above 48 mm Hg and ventilatory equivalent below 21 litres per litre oxygen uptake, 53 are the plots of three divers with marked post-inspiratory pauses. The authors themselves question "whether the slow, deep breaths with prolonged inspiratory pauses (and therefore a prolonged CO_2 build-up time) in four of the trained swimmers, would apparently accentuate the already great cyclic variations in the alveolar CO_2 as a result of exercise."

This study, although superficially convincing, has some defects. Quite surprisingly, the number of untrained subjects remains unspecified. Despite the authors concern about the effects of skip breathing, the ventilation, respiratory frequency and tidal volumes are not given. The quite remarkable constancy of mean end-tidal PCO_2 (37.1 to 38.5 mm Hg) at all speeds of swimming in the untrained subjects is not discussed.

Again, exactly what is meant by an untrained subject who can swim underwater? Goff et al (1957, (i)) state, in an associated paper on the work efficiency of underwater swimmers, "these laboratory personnel were thoroughly indoctrinated in the use of closed circuit breathing apparatus but not considered to be well trained or conditioned underwater swimmers since training was conducted only to the point of fairly uniform performance." It is possible that some of the laboratory personnel used in this study, had already taken part after training, in about twenty 20-minute dives in the first work-efficiency study (Goff et al, 1957, (i)). Even if they had not done so, one would have thought that some "training" would be apparent after indoctrination and a roughly similar period of underwater swimming in the present series. In the work efficiency study (1957, (i)), Goff and his colleagues carried out a second series of dives (three to four 15-minute dives at five different speeds). They showed a marked rise of the "untrained" divers' efficiency. There was a fall of oxygen uptake of the order of 300 ml in the range of oxygen uptake of 0.9 to 1.7 1/min. (0.7 to 1.0 knot). However throughout both studies, apparently, the increasing experience and efficiency of these subjects did not lead to any increase of end-tidal PCO_2 during underwater swimming. The authors (see above) appear to be inferring that a considerable period of diving is necessary to become "conditioned". One also assumes that the term "conditioned", as they use it, means either the adoption of "controlled" breathing or the development of genuine carbon dioxide insensitivity (or both) causing raised end-tidal PCO_2 when swimming underwater.

Finally it is important to recall that, at the time of this study (1957), many divers were trained to skip-breath both by instruction and by example. Many considered that this procedure economised in the gas demanded and made the increased respiratory work more tolerable. Yet no mention is made of this salient point when discussing the divers' training or respiratory behaviour. Was skip-breathing known to both groups and was there any discussion of the subject between the groups or with the investigators? Was there any tendency to skip-breathing, or reduced respiratory frequency, as the untrained divers became more experienced?

One would like more data and reassurance that we are not dealing with one group of divers trained or allowed to skip-breath and another group which, with or without instruction, breathed more naturally.

VENTILATORY RESPONSE TO EXERCISE IN DIVERS AT ATMOSPHERIC PRESSURE

Lally, Zechman & Tracy (1974)

This much quoted paper is a most interesting study of the ventilatory responses to exercise in divers (n = 8), "controls" who were non-diving healthy students or laboratory personnel (n = 9) and non-diving runners in training (n = 11). Exercise was performed on a treadmill with 10% gradient at 1, 2 and 3 mph. The divers were the same height as the controls but were three years (mean) older and 12.8 Kg (mean) heavier (119% of control).

The authors related the increment in ventilation on exercise to the oxygen uptake. Both were standardised against weight. All figures discussed are group mean values at a particular speed on the treadmill. They showed that the increase in ventilation per Kg of the divers was less than the "control" value at 1, 2 and 3 mph and significantly less (20%) at 2 and 3 mph ($P \leq .01$). The increase in ventilation per Kg of the divers was not significantly different to that of the runners at 1 and 3 mph, but just significantly lower at 2 mph.

The present author felt that this excellent data could be examined in other ways. The total oxygen uptake and total ventilation were calculated from the various data made available (f, VT, ΔVO_2 /Kg etc.). The oxygen uptake is a reliable measure of the work performed during exercise on the inclined (10%) treadmill. This work is largely a function of the subject's weight. The divers showed a consistently higher oxygen uptake (117-

120%) at each speed than the control subjects, which closely matched their greater mean weight (119% of mean control weight). It was therefore necessary to standardise the oxygen uptake in relation to weight in order to be able to compare the degree of ventilatory response in each group to a *certain level of work.*

However the controls and the divers had almost the same mean height (174.8 and 177.2 cm respectively, a 1% difference). It is difficult to see how the extra weight of the divers would greatly change chest cage size or ventilatory capacity. The situation was as if the divers, of almost the same skeletal size as the controls, carried a very well tailored pack, mostly fat, of 12.8 Kg. For these reasons, the present author considers that the exercise ventilation should *not* be standardised in relation to body weight.

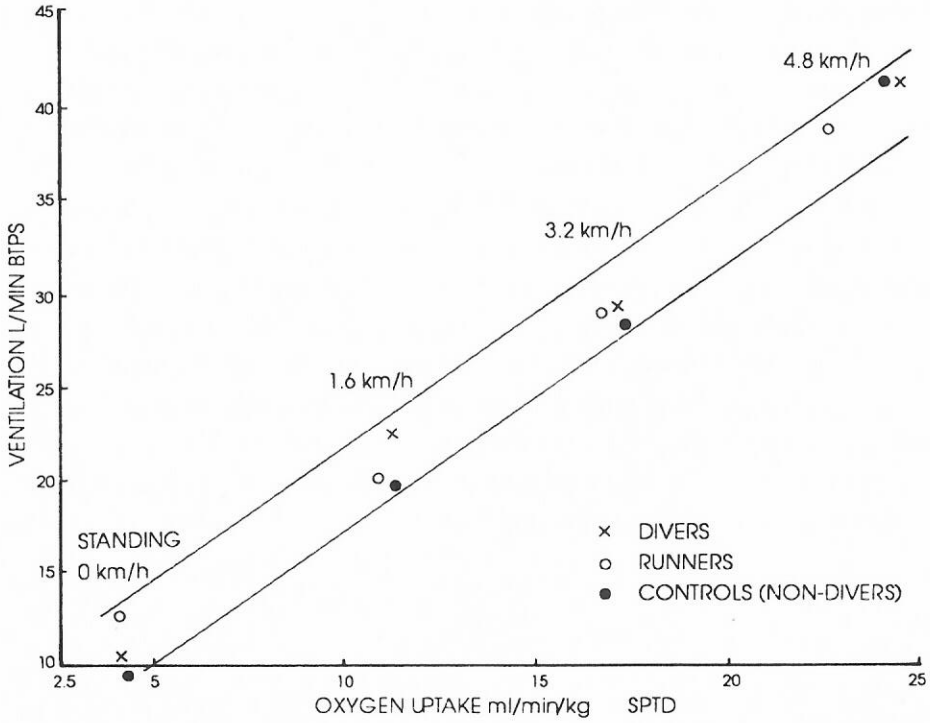

FIG. 23 Ventilatory responses to exercise (treadmill, 10% gradient) in devers, runners and control (see text) ventilation l/lm BTRS V. oxygen/min/Kg. Derived from published data (Lally Et Al, 1974) lines hand-fitted.

In Figure 23 the mean exercise ventilation of the three groups is plotted against the mean standardised (weight) oxygen uptake at 1,2 and 3 mph (10% gradient) on the treadmill. All plots including those of the runners are in a fairly narrow band. The divers' ventilation is almost 3 litres greater than the controls at 1 mph but the ventilation of the two groups become increasingly close at 2 and 3 mph. There is a slight loss of linear relationship at 3 mph. The increased efficiency of the runners (lower oxygen uptake) is also apparent at this speed. It would appear to the present author that the ventilatory response to various degrees of work is almost the same in the three groups.

In this study the expired air PCO_2 was monitored constantly. The PCO_2 at 60% (time) of the expiratory phase was considered a "reasonable approximation" to the alveolar PCO_2 during exercise (Dubois et al, 1952). Using this technique the $PACO_2$ (mm Hg) was between 38 and 39 in all groups when standing before the exercise. At 1, 2 and 3 mph the control subjects mean figures were 42.1, 44.8 and 44.9, the divers 45, 48.4 and 49.6 and the runners 44.7, 47.3 and 47.3 mm Hg. It is of interest that, wearing a "standard SCUBA mouthpiece – valve assembly", the control subjects achieved "alveolar" PCO_2 figures of 44.9 (controls) and 47.3 mm Hg (runners) during high to moderate (VO_2 2.0 to 1.6 1/min.) exertion. Yet some observers would consider these subjects hypercarbic. The divers have a slightly higher figure, presumably because they were doing 20% more work at each stage. Their respiratory rate was lower and the tidal volume about one third greater. Finally, if the end-tidal instead of the 60% expiratory PCO_2 had been measured, the figures would have been even higher.

Kerem, Melamed & Moran (1980)

These investigators studied the respiratory behaviour of large groups of divers (n = 42), ex-divers (n = 58) and "healthy adult males" (n = 49) at rest and during exercise on a bicycle ergometer while breathing oxygen. The subjects were well matched (height, weight, age) except that the ex-divers were ten years older (mean figures). End-tidal PCO_2 and ventilation were measured. There were also breath-holding studies which will not be discussed here. "Each subject was available for one short time only." The subject sat on the ergometer and immediately breathed oxygen ("resting"

state). Measurements were recorded when end-tidal PCO_2, inspiratory flow and pulse rate indicated a steady state (usually after five minutes). The subject then started to pedal. "A steady state gas exchange" was usually achieved after about four minutes of exercise on the bicycle ergometer (650 kpm/min, VO_2 1.6 1/min. approx.). No mention is made of the period of the steady state during which the measurements were made, either at rest or during exercise.

In reporting the results, the authors standardised the respiratory minute volumes for weight, although, unlike Lally's study, the subject's weight did not affect the work done. In any case the mean weight of the three groups were very close (1-1½% difference). Their findings were as follows:

MEAN VENTILATION (1/MIN/KG) BTPS AT REST AND DURING STANDARD EXERCISE

	NON-DIVERS (n = 33)	DIVERS (n = 37)	EX.DIVERS (n = 41)
Rest	0.177	0.141	0.153
Exercise	0.579	0.470	0.490

Both the divers' and ex-divers' mean "resting" and exercise ventilation are significantly different from the non-divers' mean ($P < 0.05$). The authors concluded (abstract) "findings indicated a pronounced hypoventilation and hypercapnia in the divers during exercise."

The reconstructed mean total ventilation figures of each group are more informative and are given in Table 32 with particulars of ventilatory pattern and alveolar PCO_2 (Haldane expiratory sample at rest, end-tidal sample during exercise).

Firstly, as the authors comment, the resting ventilation and "alveolar" PCO_2 figures indicate pronounced hyperventilation which was particularly severe in the non-divers and ex-divers ($PACO_2$ 29 and 32 mm Hg). This was probably due to the unfamiliar environment and experience in the brief "one and only visit" to the laboratory. Such mean resting $PACO_2$ and ventilation findings in groups of this size are, indeed, remarkable. (See Table 32).

With regard to the divers' exercising ventilatory volumes, these are

described as "pronounced hypoventilation". In fact the mean ventilatory volumes of all three groups are within the normal range of ventilation (mean 38, 90% confidence limits 48 and 32 1/min. BTPS) given by Pappenheimer at a VO_2 of 1.6 1/min. working on a bicycle ergometer. Pappenheimer based his figures on material supplied by Dill, Christiansen, Harvard Fatigue Laboratory Data and C. Taylor (The Handbook of Respiratory Data 1944). Nevertheless the difference between the divers' and non-divers' mean exercising ventilation is noteworthy particularly in such large groups. The authors, in considering various factor contributing to or causing this difference, mention the divers' "interrupted' respiration (skip breathing) and their "continued through-a-mouthpiece breathing pattern". They certainly have slower respiration and larger tidal volumes than the non-divers (see Table 32). They also considered the effect of anxiety, manifested by the preliminary hyperventilation, on the levels of exercising ventilation. They found this difficult to assess. Judging by the degree of resting hyperventilation, the non-divers were the most anxious group.

TABLE 32

MEAN TOTAL VENTILATION, VENTILATORY PATTERN AND ALVEOLAR PCO_2 AT REST AND ON EXERCISE

(VO_2 1.6 1/min.approx.)

STATE	RESPIRATORY PARAMETERS	NON-DIVERS	DIVERS	EX.DIVERS
Resting	VE 1/min BTPS	13.17	10.35	11.69
	Respiratory frequency/min.	17	12	11
	Tidal vol. 1 BTPS	0.74	1.01	0.94
	$PACO_2$ (Haldane expy.) mm Hg	29.3	35.6	31.8
Exercise	VE 1/min.BTPS	43.1	34.49	37.44
	Respiratory frequency/min.	20	13	16
	Tidal vol. 1.BTPS	2.15	2.64	2.34
	$PETCO_2$ mm Hg	40.7	48.5	48.0

Data from Kerem et al, 1980

Again, although, in "methods", the authors claimed to have waited for the achievement of a stable pulse rate, ventilatory pattern and end-tidal PCO_2 before commencing and terminating exercise, they state categorically in their discussion that there was no gas exchange steady state during rest. Further they appear to be uncertain about the exercise steady state "although our experience with the system suggested the attainment of a steady state gas exchange after four minutes, even in uninitiated subjects."

Nevertheless the usual resting and exercising periods of 15 minutes would have made a near-steady state more likely and perhaps, would have avoided or reduced the marked hyperventilation in the non-diving normal subjects immediately before the commencement of exercise. It is not unlikely that the most important and critical factor in these studies was the long experience of the divers of breathing circuits in contrast to their "controls".

EXERCISE AT INCREASED PRESSURE (DRY), VENTILATORY & END-TIDAL PCO_2 STUDIES

Jarrett (1960)

This investigator studied four subjects exercising on a bicycle ergometer at 1, 2, 3 and 4 ATA (dry) while breathing air. Each subject worked at three levels (VO_2 0.8, 1.4, and 2.2 1/min. approx.) for 6 minutes with 6 minute rest periods. Three of the subjects were divers and one was a non-diver (laboratory technician) without previous pressure experience.

The three divers all showed raised exercising end-tidal PCO_2 levels particularly at the highest levels of exercise and pressure (64, 65 and 70 mm Hg). End-tidal PCO_2 levels were abnormally raised (above 48 mm Hg) at quite low levels of exercise and pressure. One diver had a raised end-tidal PCO_2 during the three levels of exercise (53, 55 and 58 mm Hg) *at atmospheric pressure.* No diver suffered any respiratory discomfort or fatigue at any time. Only one diver showed a tendency to "controlled" breathing in so far as his respiratory rate was fairly slow (8 to 12) in all states of activity and at all pressures.

The non-diver showed a raised end-tidal PCO_2 (52 mm Hg) during medium exercise (VO_2 1.4 1/min.) at 4 ATA. His ventilation was not

markedly different from that of the three divers at the two lower levels of work. During the highest level of exercise (VO_2 2.2 1/min.) at 3 and 4 ATA he had raised end-tidal PCO_2 levels of 51 and 54 mm Hg, respectively, his ventilation being about 20% greater than the divers' mean. He suffered considerable dyspnoea and exhaustion.

It thus appears that carbon dioxide retention, if judged by end-tidal PCO_2, can occur during exercise at pressure in a non-diving normal subject and that this is not a unique characteristic of divers. When the non-diver repeated the whole experiment (only his second hyperbaric exposure), "he ventilated less, his alveolar PCO_2 rose to higher levels and he was far more comfortable than he had been during his first run." Unfortunately no figures are given. Jarrett claimed priority in describing "the extreme rapidity with which this subject modified his respiratory pattern."

This small but detailed study certainly showed raised end-tidal PCO_2 during exercise at pressure in both divers and a non-diver. However there is no new information as to whether these raised end-tidal PCO_2 levels are truly representative of arterial and body PCO_2 levels in these conditions. There were at least fifteen occasions when the end-tidal PCO_2 during exercise was between 55 and 70 mm Hg. Yet not a single subject appears to have complained of any symptoms of hypercarbia.

Broussolle, Bensimon, Michaud and Vegezzi (1972)

Three experienced naval countermining air divers and three non-diving control subjects were studied at rest and during two levels of exercise (55 & 100 W) at 1, 4, and 7 ATA in a dry pressure chamber. The results are summarised in Table 33, mean group figures being given throughout.

The control subjects showed normal respiratory rates, tidal volumes and ventilation at all pressures and with all degrees of activity. End-tidal PCO_2 levels (continuous sampling, mass spectrometer) were also "normal" although there were readings of 49 mm Hg during the higher levels of exercise at 4 and 7 ATA.

In contrast the divers had remarkably low resting respiratory rates at all pressures (4 to 5 per min.).

TABLE 33

Respiratory data at rest and exercise at 1,4 & 7 ATA

Level of Work	Respiratory Parameters (10th-12th min)	Non-Divers			Divers		
		1 ATA	4ATA	7 ATA	1 AT	4 ATA	7 ATA
0	f	9.4	8.4	11.9	5.0	5.0	4.1
	VTL	1.25	1.1	1.25	1.75	2.05	2.05
	VEL/min	11.7	9.2	14.8	8.7	10.2	8.5
	PETCO2 mm Hg	38	41	42	41	43	44
55W (VO2 1.75 1/min approx)	f	11.7	13.3	11.8	6.2	6.1	4.4
	VTL.	1.5	1.2	1.55	2.3	2.15	3.0
	VEL/min	17.5	16.0	18.3	14.2	13.2	13.3
	PETCO2 mm Hg	41	42	43	45	51	55
110W (VO2 2.4 1/min approx)	f	13.7	13.4	16.8	8.1	6.8	6.3
	VTL.	3.0	2.75	2.2	3.8	3.3	3.5
	VEL/min	41.2	36.95	36.9	30.8	22.3	22.1
	PETCO2 mm Hg	43	49	49	51	61	63

These figures are from published data of Broussolle, Bensimon, Michaud and Vegezzi (1972). Some of the data is derived from diagrams and is approximate.

These rates were only slightly raised on exercise at 1, 4 and 7 ATA (6 to 4 per min at 55 W and 8 to 6 per min at 110 W). The tidal volumes of the divers were therefore considerably greater than those of the controls throughout despite the reduction of the divers' exercising ventilation which was particularly marked at increased pressures. Thus at 110 W the divers' exercise ventilation at 4 & 7 ATA was only 60% of that of the controls. This reduction of exercising ventilation was associated with raised end-tidal PCO_2 levels (51 and 55 mm Hg at 4 and 7 ATA with 55 W exertion and 61 and 63 mm Hg at 4 and 7 ATA with 110 W exertion). The excretion of carbon dioxide was only impaired at the highest level of exertion (VO_2 2.4 1/min. approx.) being 250 ml/min (1 ATA) and 400 ml/min. (4 & 7 ATA) less than that of the controls under the same conditions.

The authors state *"Nous retrouvons la respiration bien caractéristique chez les plongeurs; fréquence basse (environ la moitié de celle des non-plongeurs) qui diminue encore avec la pression; volume courant élevé; pause post-inspiratoire."* Surprisingly they do not identify this as "skip" or "controlled" respiration nor consider whether this marked bradypnoea could be the possible cause of the reduced exercise ventilation and its consequences, at pressure. They demonstrate convincingly that the divers' exercise ventilation was well within the range of their ventilatory capacity. Thus only 40% of the maximal ventilatory capacity was used at 110 W exercise at 7 ATA.

They therefore attributed the reduced ventilation in the divers to their diminished sensitivity to CO_2. Further they emphasised that *"les effets néfastes"* of hypercarbia (CO_2 intoxication and narcosis, increased decompression risks and enhanced nitrogen narcosis and oxygen poisoning) make it necessary to select for *"la plongée profonde avec travail musculaire des sujets qui ne sont pas susceptibles de faire de rétention de CO_2 en milieu hyperbare et qui ont une ventilation maxima élevée."* On these criteria these three senior and highly experienced countermining divers and, presumably, most, if not all, of their colleagues, were unsuitable for diving involving any exertion between 100 and 200 fsw. Again although these divers achieved end-tidal PCO_2 levels between 50 and 63 mm Hg while working at 4 and 7 ATA on air there is no mention of the slightest symptom of hypercarbia or accentuated nitrogen narcosis.

What can explain this difference between these laboratory findings and the reality of diving? It has been suggested that a number of such divers adopt this remarkable slow breathing whenever they are connected to a respiratory circuit in a laboratory. There have been no field studies, as far as I am aware, of the respiratory frequency of such bradypnoeic divers while they are actually on a *real* working dive for significant periods (say 30 to 60 minutes). A strain gauge round the chest and a small recorder would soon be forgotten and would at least determine whether these men have normal respiration during such dives. They should also be studied during their normal activities, including exertion, in everyday life. If these divers' really have a "different" physiology then their respiratory behaviour while breathing air at atmospheric pressure should also be of great interest.

Bradley, Anthonisen, Vorosmarti and Linaweaver (1971)

In this study four experienced and fit divers breathed oxy-helium mixture (30% 1 AT of O_2 throughout) and exercised for 15 minutes on a bicycle ergometer at two levels (VO_2 1.3 and 2.2 1/min. approx.) at sea level, 150, 300, 450 & 600 fsw (dry). 'Alveolar' PCO_2 was calculated (Asmussen and Nielsen, 1956).

The most interesting feature of this study was that two divers had normal respiratory rates, exercise ventilation and 'alveolar' PCO_2 throughout. The other two divers were 'skip breathers' with marked bradypnoea. The ventilatory volumes of the two skip breathers were considerably less at rest and the difference became even greater with increasing exercise and pressure when the minute ventilation was almost half that of the divers with normal respiratory rates. One skip breather (W.L.) had raised 'alveolar' PCO_2 at rest and at all levels of exercise and pressure (up to 61 mm Hg). The other skip breather, surprisingly, only had mildly raised 'alveolar' PCO_2 on two occasions.

TABLE 34

(from data by Bradley et al (1971)

STATE	AMBIENT PRESSURE fsw	SEA LEVEL	150	300	450	600
REST	f per min	4.7	2.3	3.3	3.0	2.7
	VT. L/BTPS	1.6	2.9	2.6	2.0	2.6
EXERCISE	f per min	7.2	3.8	8.2	6.2	7.2
(450 Kg. M/min.)	VT. L/BTPS	3.2	5.4	2.9	3.6	2.9
VO_2 1.2-1.4 1/m						
EXERCISE	f per min	13.2	11.8	11.0	10.6	12.3
(900 Kg. M/min.)	VT. L/BTPS	3.3	3.1	3.4	3.2	3.3
VO_2 2.2-2.3 1/m						

Extreme "controlled breathing" in diver W.L. during rest and exercise.

The respiratory frequency and tidal volumes of diver W.L. are given in Table 34. It will be noted that the resting respiratory rate at pressure is only 2 to 3. Even with moderate exercise (VO_2 1.3 1/min.) the respiratory frequency is between 6 and 8. One reading with moderate exercise at 150 fsw was 3.8 breaths per minute with a tidal volume of 5.4 1.

If the reader tries, with a stop watch, to adopt such rates of breathing, particularly during exercise, he or she will appreciate the degree of abnormality of such respiratory behaviour. In some cases the respiratory cycles were as long as 26 seconds (resting) and 16 seconds (exercising). To achieve such respiratory rates it is necessary to consciously suppress the desire to commence expiration. In other words we are dealing with repetitive breath-holding rather than a natural slow breathing rhythm.

ARTERIAL BLOOD PCO_2 STUDIES DURING EXERCISE AT INCREASED PRESSURE

There are no studies known to the author where end-tidal and arterial blood PCO_2 levels were measured simultaneously during exercise at increased pressures. The only arterial blood studies under such conditions available were those by Salzano and his colleagues (1966 and 1970). In his first study he measured the arterial blood PCO_2 of young, healthy unconditioned subjects carrying out sub-maximal exercise (VO_2 2-2.2 1/min.) on a bicycle ergometer at 1 and 2 ATA, breathing air and oxygen. A Severinghaus CO_2 electrode (gas calibrated) was used. The findings relevant to this review are tabled below (Table 35)

TABLE 35

(Date from Salzano et al, 1966)
Arterial Blood PCO2 During Exercise at 1 & 2 ATA Breathing Air & Oxygen (mean ± SD)

Study	No. of subjects	Gas breathed, ambient pressure. Circuit used	Pre-exercise PCO2 mm Hg	Exercise (8th min) PCO2 mm Hg
1	5	Air, 1 ATA open circuit	33.0 ± 4.9	29.1 ± 9.1
1	5	Oxygen, 1 ATA closed circuit	32.4 ± 5.0	31.4 ± 4.9
2	12	Oxygen, 1 ATA closed circuit	31.6 ± 8.4	39.2 ± 11.5
2	12	Oxygen, 2 ATA closed circuit	32.4 ± 8.3	37.1 ± 7.9
3	8	Oxygen, 2 ATA open circuit	32.9 ± 2.4	43.5 ± 5.4

The mean group resting arterial blood PCO_2 values on air and oxygen at 1 ATA and on oxygen at 2 ATA are extraordinarily low, varying between 31.6 and 33 mm Hg. In the first study (Table 39) the mean exercising level of PCO_2 when breathing oxygen (closed circuit) at 1 ATA was 31.4 + 4.9 mm Hg in contrast to the second study where the mean exercising level breathing oxygen (closed circuit) at 1 ATA was 39.2 + 11.5 mm Hg. The impression gained is that these blood gas estimates (PCO_2) may not be very accurate with a tendency, in some instances, to be too low. This impression is supported by the remarkable finding in this study that in 80 out of 225 instances, during late exercise or early recovery, the PCO_2 of the mixed expired air appeared to exceed that of a simultaneous arterial blood sample (periods of sampling are not specified). As already stated, the end-tidal PCO_2 was unfortunately not recorded in these studies as this may have cast some light on this strange occurrence.

In the second report (Salzano et al, 1970) arterial blood studies were carried out on three subjects (2 divers, aged 27; 1 ex-diver, aged 42) during exercise at three levels (VO_2 1.0, 1.4 and 2.0 1/min.), at one atmosphere (air) and at 31.3 ATA (99.1% He, 0.9% O_2). Each period of exercise was for eight minutes preceded by ten minutes resting seated on the bicycle ergometer. The inspired oxygen at full depth was 220 mm Hg. The oxyhelium mixture at this pressure was 4.4 times denser than air at 1 ATA. At atmospheric pressure no subject's arterial blood PCO_2 exceeded 40 mm Hg during the three levels of exercise. At 1000 fsw the arterial blood PCO_2 during the two lower levels of exercise (VO_2 1.0 & 1.4 1/min.) remained between 40 and 46 mm Hg in all three subjects. However at the sub-maximal level of exercise (VO_2 1.9 - 2.1 1/min.) the two divers raised their arterial blood PCO_2 to 49 and 52 mm Hg respectively. After eight minutes exercise the subjects were taken off the respiratory assembly and breathed ambient gas directly. They continued to exercise for three more minutes. The arterial blood PCO_2 fell to 43 and 47 mm Hg respectively. Although Otis-McKerrow low resistance respiratory valves were used, the external respiratory resistance appeared to be of some significance at this gas density and level of exertion (VO_2 2l/min.). The ex-diver showed normal arterial blood PCO_2 (40 mm Hg or below) even during sub-maximal exercise at full pressure.

Again, it is unfortunate that no end-tidal samples were taken as the previous doubts concerning the accuracy of the arterial blood PCO_2

estimations remain, particularly when one considers the reported arterial blood PCO_2 levels (mm Hg) during rest and exercise in one diver at 1 ATA:

Resting (at least 10 min.) 25: Exercise (VO_2 1.0 1/min.) 31:
Resting (10 min.) 27; Exercise (VO_2 1.4 1/min.) 32;
Resting (10 min.) 26; Exercise (VO_2 1.8 1/min.) 30.

Obviously further hyperbaric studies of arterial blood and end-tidal PCO_2 levels while on circuit at rest and on exercise are desirable.

CARBON DIOXIDE SENSITIVITY IN DIVERS

Schaefer (1955)

Schaefer compared carbon dioxide sensitivity of submarine escape instructors with that of laboratory personnel. These instructors skin-dived, holding their breath, to 90 fsw several times a day for duty periods of up to a year. The ventilatory response was determined while breathing various concentrations of carbon dioxide in air for periods of fifteen minutes. Detailed respiratory data is not given but the results are presented in a figure where mean group values of calculated alveolar ventilation (constant volume of dead space assumed) are plotted against mean group alveolar (end-tidal) PCO_2. The alveolar ventilation is stated in multiples of the values breathing air. The divers' sensitivity to carbon dioxide appears to be less than that of the laboratory personnel. Using the ventilation, breathing air, of the same two groups given in a "hypoxic" study published elsewhere (Schaefer, 1965), we get an approximate idea of the value of S (Δ VE 1/min/Δ $PACO_2$ mm Hg). It is 2.1 for the divers and 2.6 for the controls. The numbers in each group are not given but the author states that the ventilatory response was significantly different, especially in the 5 to 7% CO_2 range (P = .001). It is possible that these results have been published in a more comprehensive form elsewhere.

Schaefer (1965), carried out a further study of seven escape instructors in which he measured the ventilatory response to 5% carbon dioxide in air "during a period of intensive water work" and "after a three month lay-off period". Again the only data presented is a figure showing the mean total ventilation at rest breathing air and that breathing 5% carbon dioxide in air. The minute ventilatory volumes (1/min. BTPS) were as follows:

	Air (1)	5% CO_2 in air (2)	2/1 (Difference n.s.)
Intensive water work	7.3	20.5	2.81
Lay off	8.3	26.0	3.13

The difference between the ventilatory volumes breathing both air and 5% CO_2 in air were significant (P = 0.05).

Finally, it is difficult to see why it was thought that these attendants might adapt to increased carbon dioxide levels. The author showed in a series of tests described in the same paper (1965), that the levels of alveolar PCO_2 (Haldane expiratory samples) were about 25 mm Hg after deep inspiration immediately before the dive, between 40 and 45 mm Hg after the descent to 90 fsw and 40 to 50 mm Hg after an ascent at 1.9 ft/sec. (45.5, SD $\pm$ 3.5). If the ascent is faster (3.5 ft.sec) the alveolar PCO_2 is much lower (31.5 $\pm$ 1.3) due to shortening of the dive and the more rapid expansion and venting of lung gas. Thus, these particular divers only suffer extremely brief periods of very modest hypercarbia.

Froeb (1960)

In this study sixteen professional SCUBA divers were compared with a group of sixteen normal non-diving subjects. Matching of vital statistics was good except that, as is not infrequently the case, the divers' mean weight was 9 Kg (20 lbs) greater than the controls. Froeb found that the absolute values of minute ventilatory responses to 1.6, 3.4 and 5% CO_2 in air, both at rest and during treadmill exercise (VO_2 1.2 - 1.4 1/min.) in the two groups showed no significant difference. However resting minute ventilation on air was different (Divers 7.79; Non-divers 7.16 1/min.) in the two groups, and when minute volume, while breathing carbon dioxide mixtures at rest, was expressed as percentage increase of ventilation on air, there was "significant differences (P = .05)" which the author found "difficult to interpret".

These differences were not found during exercise breathing air and carbon dioxide mixtures (in air). This was because the difference in ventilation during exercise on air in the two groups, was less than in the resting study. Thus, apart from a slight difference of resting ventilation on air, all the other findings at rest and during exercise, while breathing 1.6 to 5% carbon dioxide mixtures, show no significant difference between

divers and non-divers.

Sherman et al (1980) considered that "Froeb's failure to establish a significant difference in the ventilatory response of divers and non-divers could be due to comparison of absolute values of ventilation at relatively low $PICO_2$ where, according to our data, differences are indeed slight." Sherman does not make any such comment about other steady state carbon dioxide studies such as Schaefer (5% CO_2 in one study) or Broussolle (2 to 4% CO_2) which supported the occurrence of reduced CO_2 sensitivity in divers. He also failed to mention Froeb's most impressive demonstration of similar ventilation in non-divers and divers under the combined stimulus of inhaling 5% CO_2 and exercising (2 mph) on a treadmill.

Finally Froeb reported that he found no evidence of slower or deeper breathing than the controls in the divers when breathing air or air and carbon dioxide either at rest or during exercise. This lack of "controlled" breathing may well have contributed to his negative findings. It would have been of great interest to know about these divers' training and whether they had been told not to "skip" breath.

Song, Kang, Kang & Hong (1963)

Song, Kang, Kang & Hong (1963) studied the female breath-holding skin divers (ama) of South Korea. These divers hyperventilate, dive to approximately five metres, collect sea food and then surface after thirty seconds. Each cycle is about one minute and they continue, amazingly, for one hour. Three shifts a day are common. The total time these divers are apnoeic and exercising underwater per day is of the order of ninety minutes. Minute ventilation and alveolar PCO_2 (Haldane expiratory sample) were determined while the subjects were supine and breathing air, 3% CO_2 in pure oxygen and 5% CO_2 in pure oxygen, each for a period of fifteen minutes. Groups of twenty were studied, the controls being housewifes who were well matched apart from being, on average, five years older. The results were as follows, means of groups throughout:

Minute Ventilation 1/min, BTPS and $PACO_2$ mm Hg (Parenthesis)

	Air	3% CO_2	5.5% CO_2
AMA	7.03 (36.0)	12 (40.0)	20 (45.6)
CONTROLS	6.17 (34.6)	12.2 (40.0)	21.6 (44.5)

As will be seen, although ventilation on air is slightly different, the ventilation and alveolar PCO_2 when breathing the carbon dioxide mixtures were remarkably similar. The authors, by expressing the ventilatory response to carbon dioxide in terms of the ventilation on air, purported to show that the ama had a significantly lower CO_2 response ($P<0.05$). A similar exercise with the calculated alveolar ventilation (constant dead space of 100 ml assumed) gave the same significant ($P<0.05$) difference of CO_2 response.

However these are not valid procedures as the air studies are, of course, normoxic and the CO_2 studies are hyperoxic. The data presented here shows only how similar the ventilatory reaction to inspired carbon dioxide in oxygen was in the divers and non-divers. Yet this article is not infrequently cited as an important reference showing that divers are less sensitive to the ventilatory stimulus of carbon dioxide.

These workers also studied the reaction to breathing a hypoxic mixture (8.5% O_2, 91.5% N_2) in the same subjects. The ventilatory response, respiratory rate, tidal volume and alveolar PO_2 and PCO_2 were again remarkably similar in the two groups.

Broussolle, Bensimon, & Onjon (1969)

In this study the ventilatory reaction to 2% and 4% CO_2 in air was observed in eight experienced Naval divers and eight laboratory personnel. Matching was good. The subject breathed air followed by 2 or 4% CO_2 in air for periods of twenty-five minutes. Sampling was carried out during the last five minutes of each state. Each subject carried out four exposures of each carbon dioxide mixture. Only one exposure was carried out on a particular subject in one day. Alveolar end-tidal samples were also collected.

Ventilatory sensitivity to CO_2 (S), stated as Δ 1/min./Δ mm Hg CO_2 was as follows (mean group values):

	Air to 2% CO_2	Air to 4% CO_2
Divers	0.78	1.03
Controls	1.17	1.85

It is unwise to compare 'S' values in different studies with different techniques but these are all very low. Nevertheless the divers show a "significantly" lower sensitivity (P not given). The authors reported very much slower breathing in the divers resulting in the tidal volumes being considerably greater than the controls on 2 and 4% CO_2 (Controls 0.92 & 1.26; Divers 1.47 & 2.18 1, respectively. As the authors comment, there is evidence of some degree of hyperventilation, particularly in the control subjects mean initial end-tidal gas tensions on air, i.e. controls' $PETCO_2$ 34, $PETO_2$ 109 mm Hg; divers' $PETCO_2$ 36, $PETO_2$ 106 mm Hg.

Finally 'B' (non-stimulating level of PCO_2 by extrapolation to zero ventilation) is 31 mm Hg in the controls and 29 mm Hg in the divers. In the absence of any disease these figures can only be caused by hyperventilation.

Florio and Morrison (1979)

In this study the ventilatory response to increased inspired carbon dioxide of ten Royal Navy clearance divers was compared with that of ten non-divers. These control subjects were fit, active laboratory personnel and physiotherapy students. The groups were well matched (age, height, spirometric data) but their weights were not given. The method of Cunningham et al (1957) was used. First the supine subject breathed air for fifteen to twenty minutes until ventilation and end-tidal PCO_2 were stable. The highest level of carbon dioxide (not specified) was delivered to the subject without prior warning. Expiratory volumes and end-tidal PCO_2 and PO_2 were continuously monitored. When these parameters were stable they were recorded for the next five minutes. The process was repeated with at least another four mixtures, each containing less carbon dioxide than the previous mixture. The CO_2 concentrations used were not specified. These exposures were continuous. Stabilisation and measurements generally required about fifteen minutes. End-tidal PO_2 was maintained at approximately 200 Torr regardless of the expired volume or end-tidal PCO_2.

The results were presented in the usual $VE/PETCO_2$ plots. The integrated plots of the individuals of each group show two distinctly separate lines, the divers' ventilation being considerably lower than the non-divers' at all levels of end-tidal PCO_2. The values of 'S'

(1/min/torrCO_2) and 'B' (intercept value, torrCO_2) in the two groups were as follows:

	Divers	Non-Divers	T test (P)
S	2.16 + 0.5	3.25 + 0.99	< 0.05
B	35.3 + 3.4	32.0 + 3.7	< 0.05
VE 1/min. at PCO_2 of 50 torr	32	58	

Thus, as measured in this manner, the mean ventilatory response to carbon dioxide of the divers is "some 33% less than that of the non-divers of similar age and build", 'S' being significantly lower ($P<0.05$) in the divers than the non-divers.

However there are reservations. On first principles the present author is not entirely happy about measuring the sensitivity to the lower levels of inspired carbon dioxide after the subject has been in a continuous hypercarbic state for about an hour. Florio's non-diver $VE/PETCO_2$ plot shows high ventilatory values which are quite abnormally high in the lower range of PCO_2 i.e. VE at 40 torr = 25.5 1/min. When the non-divers plot returned to the original end-tidal PCO_2 on air before the exposure (37.4 torr) the ventilation was very much greater than it had been on air i.e. 17 1/min. in contrast to 9.8 1/min. The inspired PO_2 tension of 200 torr would certainly not account for this marked difference. Thus during these experiments the normal subjects have departed very markedly from their pre-exposure $VE/PETCO_2$ relationship. Again the intercept (VE = O) gives a non-stimulating PCO_2 (B) of 32 torr (Cunningham's normal intercept is of the order of 38 torr).

The divers' plot passes fairly close to the original air VE/PCO_2 point. At 40.3 torr the air breathing VE of the divers was 8.4 1/min. and towards the end of the exposure it was 10 1/min. at this tension.

Thus, although under these experimental conditions the two groups behaved quite differently, it cannot be said that the divers' responsivity is different to the normal as the control plot is, for some reason, undoubtedly abnormal. The levels of ventilation of the control group, particularly in the lower ranges of PCO_2 and the very low value of 'B' (32 torr) suggest that anxiety contributed to the increase in ventilation as well as the induced hypercarbia. We return, as always, to the wisdom of comparative studies of the highly labile function of ventilation in two

groups, when one group has marked and varied experience of breathing from a respiratory circuit and the other group has little or none.

Sherman, Eilander, Shefer & Kerem (1980)

These workers employed the re-breathing method of Read (1967) to study the ventilatory and "occlusion pressure" response to hypercapnoea. The occlusion pressures will not be discussed here except to say that they appear to correlate well with the ventilatory volumes. Twenty normal subjects and twenty-two SCUBA divers were studied. The diving group consisted of sports (7), semi-professional (5) and professional (10) divers, all of whom had more than four years experience. The non-diving control group consisted of university students and medical and laboratory personnel. They were, apparently, "non-athletes". No mention is made of their aquatic habits or experience as subjects breathing from respiratory circuits. The mean weight of the divers was more than 13 lbs greater than the non-divers.

The subject breathed 50/50 oxygen-nitrogen mixture for about five minutes. At the end of a normal expiration he was switched into re-breathing from a bag containing a 7-8% CO_2, 50% O_2 and 42-43% nitrogen. The bag contained about 6 litres (vital capacity + 1-1½ litres) of this mixture. End-tidal PCO_2, breath-by-breath tidal volume and minute volume were recorded. The appearance of the mixed venous blood PCO_2 plateau after 20-30 seconds showed free mixing throughout the lung bag system. The end-tidal PCO_2 then rises linearly and measurements were made every 20-30 seconds for 4-5 minutes, achieving PCO_2 levels of 70-80 mm Hg. It is fairly assumed that the end-tidal PCO_2 is very close to, or the same as, the central nervous system and respiratory "centres" tissue PCO_2 level. It is considered to be a great advantage that, in this re-breathing procedure, the resultant ventilation no longer affects the alveolar and body PCO_2 levels (open loop). The following results were obtained (means throughout):

	NON-DIVERS	DIVERS	T TEST (P)
VE/PCO_2 (S) 1/min/mm Hg	2.9 ± 0.40 (SD)	1.94 ± 0.20	< 0.05
PCO_2 (VE = 0) (B) mm Hg	39.6	39.4	ns
VE (PCO_2 = 60 mm Hg) 1/min	54.2 ± 5.5	36.3 ± 3.2	< 0.01

In the non-divers the value of 'S' ranged from 0.9 to 7.4 1/min/mm Hg and in the divers from 0.6 to 3.9 1/min/mm Hg. Distribution in both groups was positively skewed.

It was concluded that the carbon dioxide sensitivity of the divers was significantly below that of the non-divers. They also considered that the divers did not represent a distinct population different from the normal one but "rather a group of normal healthy subjects with either an inherent or acquired relatively low CO_2 response." Finally they found that individual carbon dioxide sensitivity did not correlate with either diving experience or current diving activity.

The present author does not propose to discuss the detailed pros and cons of Read's method except to remark that it is a strange procedure to measure the ventilatory response to rising tensions of carbon dioxide while it is being totally frustrated in its purpose and function. The marked inter-individual and intra-individual variation of carbon dioxide sensitivity, as measured by this technique (Rebuck & Slutsky, 1981), suggests that, as in other methods, there are complex and unpredictable events in the nervous system, particularly in such a wholly unnatural physiological situation. Despite claims to the contrary, the ventilatory response during rebreathing is affected by the respiratory rhythm adopted (Rebuck et al, 1974). Rebuck and Slutsky (1981) partly attribute the great variation of carbon dioxide sensitivity in normal subjects to marked individual variation of tidal volume and respiratory frequency. Yet the authors, although continuously monitoring these parameters, make no mention of them, Kerem et al (1980) commented elsewhere on the abnormally slow breathing (skip or controlled) of many divers when connected to a breathing circuit.

The authors claim that these results are similar to those of Florio et al (1979), who used a "high CO_2 first" steady state method. Although the carbon dioxide sensitivity, as measured by slope "S", is of the same order in the two studies, there are conspicuous differences. The non-stimulating PCO_2 (B) in Florio's report is non-divers 32 and divers 35.3 mm Hg. In the present study the figures are 39.6 and 39.4 mm Hg respectively. This difference, is of course, partly due to rebreathing. Further in Florio's study the ventilatory volumes at a PCO_2 of 60 mm Hg (extrapolated) are 77 1/min. in the non-divers and 43 1/min. in the divers. In the present study the respective figures are 54 and 36 1/min. This contrast is a reflection of

the findings being a very considerable function of the method used viz high CO_2 first, steady state v. high CO_2 last, rebreathing, (see Datan et al, 1940).

There is no mention of even slight symptoms of carbon dioxide intoxication during these rebreathing experiments although the end-tidal PCO_2 levels reached were well above 70 mm Hg. One wonders whether the sensations of unreality etc., of early CO_2 narcosis were masked by the music in the subject's earphones.

The authors, who had formidable statistical support, make the important statement that only Froeb (1960) and Florio et al (1979) had sufficiently large groups to warrant statistical conclusions concerning the carbon dioxide sensitivity of divers. The reader will remember that Froeb could find no important difference between the carbon dioxide sensitivity of divers and non-divers and that Florio's conclusions are somewhat jeopardised by the obviously abnormal VE/PET CO_2 plot of his control (non-divers) group.

The present author would again submit that, in this study, the difference in experience of both quiet and stressful breathing from a respiratory circuit (see Rigg et al, 1977) and the almost certain but unspecified difference of respiratory rhythm of the two groups still leaves the issue in some doubt.

In this context it is instructive to consider another group of normal subjects, the Enga people of New Guinea, who also appeared to show diminished sensitivity to carbon dioxide. They were studied by Beral & Read (1971) using Read's rebreathing technique. A portable apparatus allowed the procedure to be carried out in their own environment at 4000 ft. (1300 m) in the Western Highlands. They were non-smokers and most of them were medical assistants at the Baptist Mission Hospital (5 female, 7 male subjects). The control subjects were Caucasians who were studied at sea level in Sydney (6 female, 18 male subjects). Two more Caucasians were investigated in the field (4000 ft.) after a few days "acclimatisation". To eliminate the effect of "psychogenic hyperventilation" the subjects with a ventilation of 15 or more 1/min. in the first half minute were rejected. Numbers rejected in each group were not given.

The Enga subjects showed low levels of CO_2 sensitivity (S) as compared with the Caucasians:

Enga subjects	1.37 ± 0.30 (SD) VE 1/min/mm Hg CO_2
Caucasian subjects	2.51 ± 1.19 (SD) VE 1/min/mm Hg CO_2 ($P < .01$)

Although it is claimed that altitude acclimatisation has a negligible effect on the slope (S) of the hyperoxic CO_2 response line, the ideal experiment would have tested both groups at the same altitude after full adaptation. The Caucasians (24/26) were studied at sea level, the Enga at 4000 ft. The subjects were switched from air breathing to rebreathing. The effect of rebreathing 50% hyperoxic mixture on the Enga subjects, who were naturally adapted to 4000 ft. (18% O_2, PAO_2 75-80 mm Hg) may have decreased the ventilatory response to rising PCO_2 as the body PO_2 also rose. A separate study of the effect of breathing 50% oxygen on the ventilation of each group at their respective altitudes would have been informative and hopefully, reassuring. It is interesting that the two Caucasians subjects studied at 4000 ft. had 'S' values of 2.3 and 1.7.

The cultural and psychological aspects and the inter-reaction between the Caucasian investigators and their Genga subjects would bear some study. Again, holding the nose of the Genga subjects during the rebreathing was a dominating procedure which could tend to "discipline" respiration and reduce ventilation. It is difficult to see why an impersonal nose clip was not used in both studies.

CONCLUSION

The author feels that the evidence presented in these studies does not conclusively support the unlikely thesis that divers become, temporarily or permanently, less sensitive to the ventilatory stimulus of exercise and carbon dioxide. Although there are significant differences between divers and non-divers in a number of studies, this difference may well be due to other factors. The difficulty of finding truly matching control groups of non-divers is considerable, particularly with regard to the divers' far greater experience of respiratory circuits. The less experienced control subjects are certainly more aware of the containment of their respiration and are prone to increased ventilation, as can be seen in several of the above studies. Another problem is the idiosyncratic respiratory behaviour ("controlled" breathing) of many divers, with marked effects on the ventilation, while under observation on a circuit. It is difficult to believe

that divers really breath in this way (see Broussolle et al, 1972, Bradley et al, 1971 above) during their routine diving activities or in everyday life. They would be in an almost permanent state of hypercarbia.

Finally we must return to the consideration of Lanphier's specific proposal that the oxygen-nitrogen mixture diver is far more vulnerable to oxygen poisoning owing to increased gas density, reduced exercise ventilation and increased CO_2 body levels which are tolerated because of reduction of CO_2 sensitivity. He has certainly shown increased end-tidal PCO_2 levels in exercising divers when breathing oxygen-nitrogen mixtures (including air) at 100 fsw and when breathing oxygen or air at 26 and 4 fsw.

Nevertheless there is increasing acceptance that end-tidal PCO_2 measurements do not accurately represent the mean alveolar or arterial blood PCO_2. Lanphier, himself, now takes this view. He stated, with Camporesi, in "Respiration & Exercise", Physiology and Medicine of Diving (1982) "it was widely assumed that end-tidal sampling provided an accurate index of mean alveolar gas composition. This, in turn, was assumed to provide satisfactory indication of arterial blood-gas values. End-tidal PCO_2 values remain useful as non-invasive estimates but they are interpreted with reservation, especially during exertion and at pressure." He also stated in 1987 (personal communication) that he appeared to have had "an unusual concentration" of "CO_2 retainers" in his 1954-1958 investigations. He continued "I would no longer argue that oxygen limits for general use should be conservative enough to be safe for such individuals. I think we must discover and take account of them in other ways." Another important consideration is that, as far as the author knows, there is no reported study of simultaneous arterial blood and end-tidal sampling and PCO_2 measurements in exercising divers at pressure.

Again there have been no convincing signs or symptoms of oxygen poisoning or hypercarbia in any of Lanphier's very large series of oxygen-nitrogen mixture dives to 99 fsw (15 minutes only) described above (Lanphier, 1955, (ii) and 1958). The only instance of undoubted oxygen poisoning, under controlled conditions (Lanphier, 1955, (i)) was the celebrated occasion when an exercising subject convulsed (wet chamber) after breathing (open circuit) 47% ONM at 100 fsw (PIO_2 1.8 ATA, DO_2 29 fsw) for a period of nineteen minutes. This relatively short exposure, which caused such alarm and an immediate dramatic remedy, was, however, within the reported normal range of oxygen tolerance when

breathing pure oxygen at this depth (Donald, 1945).

Finally there is the Royal Navy's and NATO's experience with oxygen-nitrogen mixtures using an oxygen partial pressure limit of 2 ATA in a semi-closed circuit (constant mass flow) without a single case of oxygen poisoning over 50 years.

For all these reasons it is essential to put the matter to the test under controlled conditions and actually demonstrate conclusively, or otherwise, that, apart from end-tidal PCO_2 changes, oxygen-nitrogen mixtures divers suffer from accelerated oxygen toxicity. As already suggested in Chapter 4, a moderate increase of the depth or of the partial pressure of oxygen in the mixture to give equivalent oxygen depths of, say, 1.6 to 2.5 ATA, under safe controlled, experimental conditions, with a significant number of subjects and with pure oxygen "control" dives, would soon show whether there really is an increased danger of oxygen poisoning. As yet neither of Lanphier's hypotheses (possible adverse effect of hyperbaric nitrogen and/or adverse effect of possible carbon dioxide retention on oxygen toxicity) has been proven or convincingly supported. The total lack of any direct demonstration of these purported hazards of oxygen-nitrogen mixture diving, over so long a period, makes it not unlikely that the elaborate and expensive restriction of oxygen-nitrogen mixture diving during the last 35 years in certain countries, has been to avoid dangers which may well not exist. Finally, it is to be emphasized that the putative hazard of *accelerated* oxygen poisoning in oxygen-nitrogen mixture divers is being discussed here and not the established dangers of oxygen poisoning itself.

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APPENDIX 1

THE ADMIRALTY EXPERIMENTAL DIVING UNIT

After serving as Flotilla Medical Officer in the Second (Captain Warburton-Lee VC., RN.) and Fifth (Captain Lord Louis Mountbatten, D.S.O., RN.) Destroyer Flotillas I was posted to HMS *Forth* in Holy Loch. The ship serviced submarines patrolling in the North Atlantic, the Arctic and the North Sea. It was a fascinating experience to meet and work with these extraordinary men. However, although Holy Loch was a beautiful and serene place there was, apart from routine duties, little to do except read, meditate and, occasionally, walk about.

Thus, in March 1942, after volunteering for 'special service', whatever that meant, I found myself proceeding to HMS *Dolphin* (Fort Blockhouse, Gosport). I soon discovered that I was to be associated with underwater activities which were quite apart from those of the submarines based there. The reasons for these developments were highly confidential. The Italian Navy, which has always had a penchant for unusual and gallant underwater operations, had, in December 1941, struck a critical blow against the Royal Navy by severely damaging the two battleships HMS *Queen Elizabeth* and HMS *Valiant* in Alexandria harbour. Both ships were rendered non-operational for a considerable time. The harbour was shallow and lists were rapidly controlled. They successfully deceived the enemy by behaving as if nothing had happened. This was possible as all the human torpedo crews involved had been captured. All drills, shore leave, receptions and band parades continued with due pomp and ceremony. 'Steam' was kept up as if ready to sail at short notice.

This brilliant attack was made by specially designed torpedos which were ridden and controlled by two divers. The powerful warhead, with neutral buoyancy, could be detached from the torpedo and clamped onto the ship's bottom or laid on the seabed. The torpedoes were carried to

within a few miles of the harbour by a parent submarine. This was by no means the first of such underwater operations. A similar planned assault on Alexandria harbour in August 1940 had been unwittingly frustrated by the bombing and sinking of the Italian parent submarine *Iride.* In November, 1940, three 'human torpedos' had launched an attack on HMS *Barham* in Gibraltar harbour. Only one of three craft penetrated the harbour defences. It was ridden solo by Lieutenant Brindelli who had ordered his number two to 'bale out' after respiratory difficulties. Brindelli came within 100 yards of the *Barham* when his torpedo engine failed. He pulled the inert torpedo to within 30 yards of the ship when he suffered breathing apparatus failure and probably, carbon dioxide intoxication. This remarkable man survived and was only captured as he nearly succeeded in boarding a Spanish ship. A similar attack on Gibraltar harbour in September 1941 resulted in the destruction of two large tankers and a cargo vessel. The Italian Navy also had underwater swimming teams who were attaching limpet mines to ships in different harbours with increasing effect.

These events had not gone unnoticed by 'A Former Naval Person' and a brisk signal went to the Chiefs of Staff in January, 1942: "Please report what is being done to emulate the exploits of the Italians in Alexandria harbour . . . Is there any reason why we should be incapable of the same kind of scientific aggressive action that the Italians have shown?"

At long last Their Lordships moved and ordered the development of 'human torpedos' and other unorthodox methods of underwater attack. It was estimated that we were at least seven years behind the Italians in this field, although new types of midget submarines were already being developed. Flag Officer, Submarines, Admiral Max Horton, D.S.O. was placed in charge of all these activities with the support of HMS *Excellent* (Gunnery and Diving School, Portsmouth). Flag Officer, Submarines was represented in this regard at HMS *Dolphin* by Commander G.M. Sladen, D.S.O., RN.

On my arrival at Blockhouse I was put through refresher courses of air diving and submarine escape followed by oxygen diving in Horsea Lake. It was here that the first British 'charioteers' were being trained. They were using a self-contained oxygen breathing apparatus which had a well-designed rubber counterlung and a large radial carbon dioxide absorbent canister (4.5 lbs). Although it had been developed with

considerable urgency it appeared to be more robust and safer than the captured Italian models. Oxygen breathing was essential in these covert underwater attacks on ships and harbours so that no gas needed to be vented and the risk of detection minimised.

Author (centre) with two members of the first charioteer group after a day's oxygen diving in Horsea Lake, Portsmouth, April 1942.

My duties as a humble Surgeon Lieutenant were simple. I attended all oxygen diving carried out by future charioteers in case anyone 'flaked' or suffered other misfortunes. With regard to the safety times and depths when breathing oxygen under water, it was tacitly assumed that these could be directly inferred from the oxygen tolerance determined in the 'dry' hyperbaric chambers when the resting subject breathed oxygen in compressed air. The safety limits at this time (1942, Royal Navy and US Navy) were of the order of 2 hours at 50 feet and 30 minutes at 90 feet of sea water (see Chapter 1). In April and May there were a number of incidents at Horsea Lake (maximal depth just over 25 fsw) when oxygen divers 'did not feel right' or were transiently dissociated. It was felt that minor symptoms were inevitably in groups of men who had not dived before and were under intense training. Again, the mouthpiece, noseclip and soft helmet 'set-up' of the "human torpedo" oxygen diver was far more

claustrophobic than air diving with a large, rigid and windowed helmet in which fresh air sweeps constantly past the face. Then, suddenly, an oxygen diver was lost at these relatively shallow depths. The precise events leading to his death were not fully determined but there was a strong suspicion that oxygen poisoning had occurred. There was a growing realisation that it was desirable, if not essential, to have a constant background of careful investigation to ensure the maximal safety of these men during training and during their dangerous covert operations.

In May, 1942 the Royal Navy took over the experimental wing of Siebe, Gorman and Company at Surbiton. This exceedingly well equipped unit contained a large open 12 foot tank, a smaller open 25 foot tank, a 'wet' pressure chamber (the 'wet pot') and several dry pressure chambers of different sizes. Expert technical help was immediately available. Every member of the company from Sir Robert Davis and his sons to the nurse in the sick bay provided all the assistance possible. It had been decided to test all operational personnel (charioteers, X-craft (miniature submarines) crews, underwater swimmers etc.) in controlled and reasonably safe conditions while breathing oxygen underwater at increased pressures in the wet pressure chamber. I travelled daily from Portsmouth to Surbiton to act as medical officer in these trials and saw experimental diving in hyperbaric chambers for the first time. It was a strange scene as divers were lowered into the 'wet pot', particularly as a considerable number were hauled out unconscious after convulsing. On occasions, the next diver stepped over the last casualty to take his turn. There was an air of extreme urgency as the planning and method of attack on enemy harbours and the proper defence of our own, depended on a full knowledge of how deep these oxygen divers, friendly or hostile, could go with reasonable safety. The grand mal types of convulsion occurring made it absolutely clear that we were encountering acute and severe oxygen poisoning at depths and in times then considered quite safe by all authorities.

After about a fortnight of this unusual and occasionally chilling experience I received a 'chit' from Their Lordships instructing me to proceed forthwith to some obscure posting in the Shetland Islands. Next day I was travelling for the last time from Portsmouth to Surbiton by train and, by sheer chance, Commander Sladen came into the same compartment. Sladen, who had played rugby football for England on a number of occasions, was a man of unlimited energy and quick decisions. He was a

highly successful and much decorated submarine commander having been in many actions, including the torpedoing of the heavy German cruiser, the *Prinz Eugen*. I informed him of my new appointment and must have shown my regret at leaving this brave company. He asked me point-blank if I would like to be responsible full time, for the safety of the subjects and for the investigations and programme at the Unit. I replied that I certainly would. "Give me your chit" he said, "You'll hear no more from Their Lordships." Nor did I.

Thus, there I was, a Surgeon Lieutenant, aged 30, with no research and little hyperbaric experience, with a not inconsiderable research unit on my hands. I was, by some strange and splendid accident, in no way responsible to any senior medical or scientific person or committee and apart from operational demands, I was given a carte blanche in programme and supplies. My immediate responsibility was to Commander Sladen and Commander W.O. Shelford, RN., Submarine Escape Officer and later Superintendent of Diving. In a relatively short time the staff of the Unit expanded and consisted of Commissioned Gunner Mr. E. Crouch, RN., two diving Chief Petty Officers, a gas analyst, a typist secretary and a variable number of hands for diving, routine and maintenance.

A further piece of extreme good fortune was the discovery that J.B.S. Haldane (fils) was working in one of the dry chambers for two days a week completing a contract to investigate the physiological factors relevant to the submarine HMS *Thetis* disaster in 1939, (Alexander et al, 1939; Case & Haldane, 1941; Haldane, 1941). During the next year, while Haldane was completing his programme, he gave most valuable advice particularly during the development of oxygen-nitrogen mixture diving (see Chapter 5) and also on the statistical treatment of the Unit's early studies of oxygen poisoning (see Chapter 2).

In August, 1942, in addition to the many operational personnel passing through the Unit as subjects, we were joined by a team of twelve carefully selected volunteers. These men also acted as experimental subjects, particularly in longer term projects. The Unit programme was rapidly becoming engaged in more and more operational problems. We needed a larger staff who could service and maintain respiratory and other apparatus, who could dress the divers and who could help to attend and observe subjects both in the open water and in chambers. Care was taken to ensure that they were briefed and involved in all Unit activities. One of

my most pleasant memories was of an Able Seaman showing a senior visiting academic how to handle a wayward spirometer during marked pressure changes. The teacher and pupil were completely engaged without a trace of disrespect or false dignity. Another visitor wrote some years later "In spite of the risk and unpleasantness of the job, the experimental department was always a scene of cheerful activity." Some of these divers transferred to operational teams and some operational personnel came back into the 'experimental' group. This leavening both ways was good for morale and increased efficient communication with those in the field.

While ordering notepaper for the Unit I decided that we fully deserved a more definite and prestigious identity. I adopted an Admiralty crest and titled ourselves the Admiralty Experimental Diving Unit. During the next three years we were in constant touch with the Admiralty over a host of problems. They also sent many VIPs, including those of our allies, to see our work. No-one objected to our splendid self-bequeathed title.

In the large study of oxygen poisoning in divers, which is fully described in Chapter 2, it was necessary for the observers to assess the various symptoms and signs reported by the divers. Convulsions are the only unequivocal end-point of oxygen toxicity but other symptoms, even lip twitching in a soft helmet, are largely a matter of report. The morale and reliability of these men were therefore critically important. They were well aware that the success of future operations and the safety and competence of operational personnel completely depended on the accurate and faithful reporting of symptoms in these urgent investigations. When we first transmitted our findings elsewhere concerning the quite unexpected and marked increase of oxygen toxicity under water, there was, initially, almost total disbelief. Most unfortunately, mention must have been made by some unknown persons of possible "feigned symptoms". Such remarks were not only unforgivable but die hard. Indeed, the author only learned of this recently when, to his astonishment, he was asked, in writing, by a US research worker 45 years later "which was the study spoiled by feigned symptoms?" It is therefore necessary to point out, somewhat reluctantly, that the subjects in this oxygen study, including members of the experimental team, received many gallantry awards (Victoria Cross, 4; George Cross, 5; Distinguished Service Order, 10; Distinguished Service Cross, 21; Distinguished Service Medal, 8; George Medal, 5; Conspicuous

Gallantry Medal, 5; British Empire Medal, 16, etc). It is most unusual, if not unique, for an investigator to be able to produce such tangible evidence of the intrepid mould and resolution of his subjects. The extraordinary fantasy of such brave and responsible men feigning convulsive attacks and other signs and symptoms in front of their colleagues and medical officer in these critical trials does not merit polite consideration.

Oxygen-nitrogen Mixtures

In October 1942, it was decided that, as oxygen diving was so dangerous below 30 fsw, it was necessary to develop the use of oxygen-nitrogen mixtures in self-contained counterlung breathing apparatus for use at greater depths. The oxygen breathed could be diluted with nitrogen, although not so much as in air, and this would allow diving to considerably greater depths without the risk of oxygen poisoning.

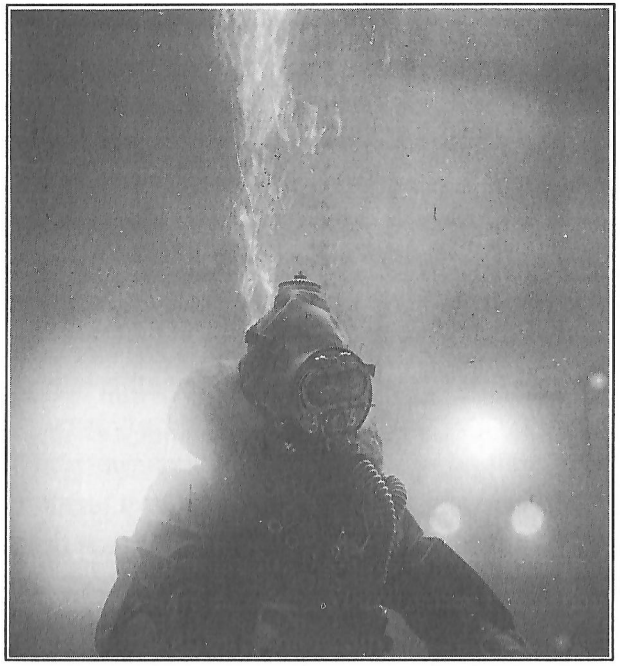
The midget submarines were now working up to operational efficiency. In their harbour-attack procedure, a member of the crew might be required to lock out of the craft, surface and inspect or even cut defensive nets and then return to the submarine and lock back in. This might be necessary at depths greater than 30 fsw and such oxygen-nitrogen mixtures would be very useful in this context as oxygen poisoning could be avoided. The development of oxygen-nitrogen mixture counterlung breathing apparatus is discussed in some detail in Chapter 4. This work was completed and reported in March 1943 (Donald 1943 (i)).

In May 1943, counter-mining officers, on entering Bizerta, Tunisia, found many large German mines with six-day clocks for use in harbours and basins. These had no magnetic or acoustic units and could not be detected and 'swept' by the usual methods. This meant that it would be difficult, if not impossible, to guarantee the safe handling of troop and supply ships in newly captured ports. Unless some means of locating these mines were found, the planned invasion of Europe was in jeopardy. After considerable consultation and much rumination, it was proposed by Officer-in-Charge of Counter Mining that the only way to deal with this problem, was, if possible, to organise large teams of divers to search for these mines systematically. Commander John Stuart Mould, G.C., G.M., RANVR. (called 'Mouldy', of course) came to see me about 'the problem'. Mouldy had achieved considerable distinction in many dangerous counter-mining

operations which, to quote the *London Gazette*, "included the recovery, rendering safe and investigation of the first German magnetic/acoustic unit and moored magnetic mines." Oxygen diving was out of the question as depths up to 80 fsw would be encountered. Thus, a few weeks after completing the first mixture counterlung development, an unexpected and critically important use for this apparatus and technique presented itself.

A number of large teams of divers were required and it was essential that they were highly mobile and able to dive and search anywhere at short notice. Each diver moved on bottom along a jackstay on a grid, searching one side and then, on return, the other. It was necessary to test the efficacy of oxygen-nitrogen mixtures even further and to assess the feasibility of large teams using this not altogether simple procedure requiring scrupulous supervision. During 1943 P Party personnel (P for Port Clearance) were trained at the Admiralty Experimental Diving Unit, particularly in the care and maintenance of mixture breathing apparatus. They also acted as attendants and subjects in many experimental dives 'in the dry' and 'in the wet'. Officers were taught the theory and calculations involved in mixture diving. It was felt strongly that this considerable degree of understanding and involvement was essential for the reliable use of mixtures by counterlung divers in demanding and dangerous operational conditions. It was a great pleasure to work with these brave and intelligent men, whose sense of humour was never far below the surface. In thousands of oxygen-nitrogen mixture dives in Europe after D-day by P Parties, there was no instance of oxygen poisoning, oxygen lack or decompression sickness. Their quite remarkable operational record in Europe is well documented (Grosvenor and Bates, 1956). Their first task was to clear Cherbourg harbour for the US forces. General Eisenhower sent a warm signal of thanks. They worked their way along the coast of Northern Europe, sometimes by-passing unfinished battles and uncaptured ports to return later. The clearing of Antwerp harbour and its approaches was a tremendous undertaking, much of it carried out under enemy fire. Over eight and half million square feet were searched. Rotterdam, Hamburg and Bremen were finally cleared. Bremen was to be the port for the US occupation zone and its clearance was the greatest task of all. All six British P Parties and the Dutch P Party (trained in the United Kingdom) took part in this operation. It is estimated that 9,500,000 square

feet of docks and harbours in Bremen were searched and cleared. These were certainly days of high courage and great achievement.



Original Port Clearance counterlung mixture diver under way.

Underwater Oxygen Swimmers

In 1930, Commander de Carlieu designed and introduced rubber swim 'fins' attached to the feet. These were widely adopted by sportsmen, some of whom carried air cylinders giving a steady flow of air into their mask. The adoption of a closed circuit with oxygen breathing and without venting was ideal for covert underwater fin-swimmers and was used by all combatants. These divers were, of course, known to the public as frogmen. A 'crawl' leg stroke provided the propulsion, the two swim-fins and legs imitating the driving action of a dolphin's tail.

In view of our recent findings, these oxygen swimmers did not go deeper than 25 feet, except for very brief periods and only for urgent operational reasons. The use of oxygen-nitrogen mixtures was carefully considered but rejected. The higher flow required would have necessitated considerable venting of gas with greater risk of detection. Endurance would have been

reduced and the large cylinders needed would have impaired mobility and stream lining. Water turbulence was most undesirable as it, too could alert harbour and coastal defences.

The Royal Navy frogmen often worked in northern waters and needed protection from the cold. Their beautifully designed and streamlined rubber suits were the most efficient and elegant ever produced. The main contributors were W. Gorham of the Dunlop Rubber Company and Commander Shelford. Colonel H. Hasler, D.S.O, R.M. (of 'cockleshell' fame) and Lt.Cdr. B. Wright, RCNVR. an experienced spear fisherman, both stimulated and helped their development. These underwater swimmers were needed for a host of operational requirements, the sabotage of ships, docks and bridges (limpeteers), boom and ship defence units, beach reconnaissance (the frogman could slip off his mask and carry his flippers), and so on.

In the early days of training these frogmen encountered a new and terrible danger called shallow water blackout (SWBO). These blackouts occurred at depths of 20 fsw or less. Several swimmers were lost. Those who surfaced safely described how they had become dissociated and even unconscious. The investigation and elimination of SWBO is described in detail, with references, in Chapter 5. Briefly it was discovered that these swimmers could have oxygen uptakes and carbon dioxide production far beyond those ever encountered in booted divers. This led to CO_2 absorbent canister overload and the divers being anaesthetised by their own carbon dioxide.

At this time increasing interest was being taken in the oxygen uptake of divers as the safety of both oxygen divers and oxygen-nitrogen mixture divers depended on this being accurately known. In the early development of mixture diving, a somewhat approximate estimation of oxygen uptake had been obtained by balancing the oxygen supply with the oxygen uptake in a steady state at rest and during various degrees of exertion. A new and far more accurate method was developed. This depended on a demand valve* fed through a reducing valve and a long intermediate pressure line to the diver's counterlung. No venting was allowed and the diver's face,

* The demand valve, which releases air or other gases to the diver's breathing apparatus when a slight negative pressure is generated on inspiration, was, astonishingly, invented and used in an independant diver's breathing apparatus by Rouquayroll and Denayruse as early as 1865

mouthpiece and nose clip could usually be observed. The oxygen was supplied from a relatively small cylinder in which exact changes of pressure could be read by the observers at the surface, using a very sensitive and accurate pressure gauge (Donald and Davidson, 1944). This new method could be used in any conditions even in open tidal water and was ideal for determining oxygen uptake (P Party divers, frogmen, etc.), under operational conditions. The very high oxygen uptake of frogmen swimming at speed, already mentioned, was a great surprise to us. Looking back, of course, it should have been obvious that the liberation of the diver's legs from his heavy restraining boots to free swimming would inevitably bring many more powerful muscles into play.

In late 1943 and early 1944 the Unit carried out a large programme of dives to determine oxygen tolerance at relatively shallow depths (25 to 40 fsw). The effect of exercise and temperature on oxygen tolerance underwater was also carefully studied at various depths. The great variability of tolerance in individuals and between individuals, already demonstrated, made large series essential.

A system for diving on oxygen to greater depths in covert operations was evolved at this time. Relatively short stays at 'toxic' depths (50 to 70 fsw) were alternated with brief intermissions near the surface (Donald cit. Admiralty Fleet Order 4565,1944). This potentially valuable technique has not been used operationally since that time.

As D-Day approached we felt ready for all predictable requirements for a major assault from the sea. There were some last-minute requests, such as a light escape apparatus for the crews of amphibious tanks and other heavy vehicles that might be sunk on the way in. The part played by the underwater section of the Royal Navy and Royal Marines in the invasion of Europe is little appreciated. Beach reconnaissance frogmen who determined the slope, the depth of approach and suitability for tanks and heavy vehicles, including sampling, were landed surreptitiously before D-Day from miniature and ordinary submarines and from light surface craft. The formidable array of obstacles to prevent landings, both on the beach and underwater, were located and studied in detail. These obstructions could, of necessity, only be demolished just before landing, usually under heavy enemy fire. The ten gallant teams (6 RN, 4 RM) of Landing Craft Obstruction Clearance units performed one of the most critical and dangerous tasks of the whole war. Miniature

submarines were submerged about three miles off the coast and surfaced at the agreed time to flash hooded lights seaward to guide the assault. The Port Clearance Parties were close behind to search and clear all docks, basins, harbours and canals and to check lock gates and bridges for mines. The harbours, both old and new, were, in turn, protected by the Royal Marine Boom Defence Units who also patrolled under water.

At home the transfer of P Party Headquarters to Brixham gave the Admiralty Experimental Diving Unit better facilities for even more realistic operational trials in the harbour and open sea. In August 1944, Surgeon Lieutenant Commander W. Davidson, RN. joined the Unit to train in hyperbaric work and gave valuable help. As victory in Europe became more certain we turned our attention to a number of new problems. Lighter swim suits were developed and there was a sudden interest in the dangers of the Pacific and other Far Eastern seas. We were able to commence a number of projects that had been set aside because of unremitting operational demands.

One of these projects was to determine the margins of safety in the method of Surface Decompression being used somewhat tentatively by the Royal Navy in air diving during World War II. In this procedure the diver came straight up from the bottom to the surface at about 100 feet per minute. His helmet and weights were removed as quickly as possible (1.5 minutes maximum). He was then recompressed in a deck chamber to his original depth. The maximal total time allowed from 'bottom to bottom' was five minutes. After another five minutes at maximal pressure a standard decompression (air and oxygen) was carried out.

It has since been claimed (Davis, 1951) that this technique had been used during diving on the *Empress of Ireland* (190 fsw) in 1914 and on the *Laurentic* (130 fsw) from 1917 to 1922. In fact the most careful enquiries reveal that immediate recompression and subsequent decompression had only been used after emergency surfacing due to 'blow up' or other severe accidents or illness, as was the case in air diving the world over. The first deliberate, planned use of *immediate* surface decompression in air diving originated in HMS *Tedworth*, the Royal Navy deep diving vessel, during World War II. A table was developed with maximal time allowed on bottom ranging from 50 minutes at 120 fsw to 15 minutes at 250 fsw. The surface decompression procedure and table were not even mentioned in the Royal Navy diving manual of that time, nor had its overall safety ever

been tested and approved. The attendant dangers of almost immediate fatal decompression sickness or of irreversible paralyses were indeed fearsome. Consultation with diving officers of that period revealed that the procedure had only been used on a few important occasions at the "lesser" depths (120 to 200 fsw). Not surprisingly, urgent requests were now being made (1945) that the procedure and table should be formally examined and tested before further use.

In these 1945 Admiralty Experimental Diving Unit trials (Donald & Davidson, 1945), for obvious reasons, the first surface decompression profiles were performed in compressed air using goats as subjects. Human chamber dives were then carried out, followed by full sea dives from HMS *Tedworth*. These investigations showed that it was highly dangerous to use this method with exposures greater than 20 minutes at 190 fsw.

The summary of recommendations made after this investigation were as follows (Donald & Davidson, 1945):

1. The 'Tedworth Method' of Surface Decompression is safe for the following depths and times on the bottom:

Depth	Time on Bottom
Up to 130 fsw	50 minutes
130 - 150 fsw	40 minutes
150 - 170 fsw	30 minutes
170 - 190 fsw	20 minutes

2. These times should never be exceeded. If this occurs inadvertently. e.g. the diver is fouled, then the stops must be carried out on the shot rope.

3. This method should be employed only by *TRAINED* personnel in a properly equipped diving ship, as speed in recompressing the diver is essential.

4. Oxygen breathing is essential during decompression from the 60 fsw stop to surface.

5. This method should not be employed at depths greater than 190 fsw owing to the grave risk of *"CHOKES'* on rapid surfacing."

Air diving is now restricted to 50 msw (165 fsw) but surface decompression is still practised a great deal, especially in 'shallow' oil and gas fields. Recompression to the full depth is not necessary. The main dangers of this procedure are always present if there is any slackening of the rigid discipline of accurately measured and restricted 'times on bottom' and there is the slightest leisureliness and resultant lengthening in 'bottom to bottom' time. Rapid handling of the diver on deck requires ballet-like skill and precision. Any clumsiness or carelessness causing delay at surface is particularly hazardous.

We next turned our attention to the ever-present problem of submarine escape. The number of successful escapes from sunken submarines during World War II, even from moderate depths, had been disappointing. The hazards of main compartment flooding, followed by serial escape through a trunk or escape lock were now realised to be great at shallow depths and appalling at greater depths. The breathing of compressed contaminated air, followed by the breathing of oxygen near equalisation and during escape, was full of dangers which were compounded in a stressed and exhausted group of survivors.

Experience before and during the war has shown that the majority of survivors, particularly from greater depths, were not wearing any breathing apparatus and had escaped from air locks or compartments that had equalised with the outside pressure during or after the incident. A whole new look at submarine escape was patently necessary. We proposed to imitate and exploit these successful but somewhat fortuitous escapes without breathing apparatus from air locks (Donald, Davidson and Shelford AEDU Report XVIII, May 1946, Published J. Hyg. Camb. 1948). The air breathing escape procedure to be adopted was as follows:

Stage 1: Escaper enters the relatively small escape compartment and pressure is raised by flooding to the pressure at which the submarine is lying. An air lock is maintained. Time: 2 minutes approximately.

Stage 2: At full pressure, manipulating and opening hatch and emerging from submarine. Time: variable.

Stage 3: Free ascent from submarine to surface at approximately 2 feet per second. Time: varies with depth, 2-2.5 minutes. The rate of ascent had been determined in open water trials by Commander Shelford.

In view of the considerable risk of irreversible decompression sickness, goats were used in these initial experiments which were conducted in compressed air. Escape profiles were carried out from 150 to 300 fsw. Time at full pressure (Stage 2) was varied from 3 to 7 minutes. Symptomless escapes with no decompression sickness were successfully concluded with up to 5 minutes at maximal pressure (Stage 2) at 150, 200 and 250 fsw. The use of oxygen-nitrogen mixture instead of air (33% oxygen; 67% nitrogen) allowed safe escape profiles from 300 fsw with up to 7 minutes at full pressure. In these experiments the time at full pressure before escape was deliberately excessive. As regards decompression sickness, it was felt that escapes by this method, with a short Stage 2, were feasible from far greater depths. The problem as to whether the escaper could continue to vent and not inhale while ascending from these great depths was yet to be investigated.

Another important question was whether nitrogen intoxication would jeopardise the competence of the escapers. A series of cancellation tests were performed during and after very rapid compression to 300 fsw. It was shown that compression to 300 fsw, in times as short as one minute, did not cause undue psychological disturbance although there was a slight euphoria in several instances. The cancellation tests showed that the subjects were able to concentrate moderately well during compression and after arrival at full pressure. A few subjects showed marked slowing but maintained accuracy and others lost accuracy without slowing. No subject stopped his test or lost control.

All these air breathing submarine escape experiments were performed in 1945 between VE day (8th May) and VJ day (14th August), a good augury, we hoped. The Ruck-Keene Committee on submarine escape (August 1946) supported this new method of escape and recommended further research and development with a view to its adoption, if feasible. A whole series of animal and human experiments in the late forties, fifties and sixties culminated in escapes in the open sea from a submarine lying on the sea bottom at 600 fsw (182 msw) in 1970. To gauge the progress made, in

the 600 fsw escapes the compression time was 33 seconds, the full pressure period 3 seconds and the time of ascent, when most nitrogen is absorbed, (rate 8.5 feet per second) 68 seconds. The great increase in the rate of ascent was largely and somewhat fortuitously due to the development of a survival suit with a hood and a buoyancy stole to prolong survival at surface. Thus the escaping crew were compressed from atmospheric pressure in the submarine to 600 fsw (19 ATA), locked out and ascended to the surface and atmospheric pressure in the astonishing time of 104 seconds. The twenty-five years work to make this feat possible has been described and reviewed by the author elsewhere (Donald, 1970, 1979 and 1991). These two reports made it clear that, although successful escapes were now possible from 600 fsw and even deeper, there were considerable attendant risks which were only justifiable in real escapes from disabled submarines. Calculations had shown an undesirable degree of nitrogen super-saturation of various 'fast' tissues on surfacing after deep escapes (500 to 700 fsw). It was considered highly probable that there were free bubbles of nitrogen in the circulation after such escapes. For these reasons it was recommended that further deep escape trials should be approached with considerable caution and that repeated deep escapes at relatively short intervals by a single individual should be avoided.

The submarine escape study was my last project. I left the Royal Navy in November, 1945. The Admiralty Experimental Diving Unit at Siebe, Gorman, Surbiton was shut down shortly after the war and the remaining staff returned to HMS *Vernon*, Portsmouth, where the diving headquarters and school were now established. It had been an interesting and concentrated three and a half years.

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APPENDIX 2

TABLE 1

OXYGEN POISONING AT 90 FEET IN THE DRY IN 37 SUBJECTS

Name	Early symptoms	Time of Onset of Early Symptoms (min)	Symptoms at Final End Point	Time of End Point (min)	Age (yrs)	ht. (ins)	wt. (lbs)
Derrick	Slight vertigo	43	prolonged dazzle, severe spasmodic vomiting	96	37	72	120
Sims	Slight twitch left upper lip	55	Severe lip twitching	67	23	70	190
Harman	Dilatation of pupils (sign)	23	Euphora & lip twitching	62	23	65	141
Goldsworthy	Tremor left upper arm	38	Nausea and vertigo	62	34	64	118
Whittington	Fine tremor of lips	37	Severe lip twitching	54	18	64	143
Brown	Slight lip twitching	7	Dazzle and lip twitching	51	21	66	140
Warren	Drowsiness & pouting of upper lip	1	Blubbering of lips and fell asleep	50	30	69	146
McInnes	Cheek tremor	4	Dazed & lip twitching	50	36	63	122
Fraser	Slight lip twitching	7	Nausea, vertigo, lip twitching	34	19	66	131
Ward	Slight facial twitch	25	Convulsed	33	23	69	148

Name	Early symptoms	Time of Onset of Early Symptoms (min)	Symptoms at Final End Point	Time of End Point (min)	Age (yrs)	ht. (ins)	wt. (lbs)
Murton	Fine twitch of lips	18	Convulsed	32	20	72	168
Dickie	Fine twitch upper lip	3	Severe lip twitching	32	21	68	150
Shields	No warning	–	Convulsed	30	20	67	142
Wadsley	Slight twitch of lips	11	Convulsed	26	35	66	164
McAtamney	Drowsiness	1	Drowsiness lip twitching	25	25	69	142
Robertson	Slight tremor left upper lip	18	Severe lip twitching	24	25	65	161
Wallis	Sensation of epigastric tension	1	Lip twitching	23	23	67	142
Gray	Fine twitch right upper lip	10	Lip twitch, twitch left arm. Amnesia	20	34	70	138
Martin AB	Slight lip twitching	–	Convulsed	19	24	69	138
Rogers	Slight twitch upper lip	10	Vertigo & Severe lip twitching	18	25	67	169
Martin	Sensation of epigastric tension	6	Vertigo plus plus	18	21	67	145
Smith	No warning	–	Lip twitching	17	23	67	154
McCourt	–	–	Lip twitching Spasmodic respiration	17	23	68	159

Turner	Fine twitching of lips and pallor	10	Lip twitching Spasmodic respiration	17	40	70	157
McLaughlin	Lip twitching	1 1/4	Lip twitching	16 1/4	19	63	111
Mould	Slight lip twitching	10	Severe lip twitching & spasmodic respiration	16	33	71	154
Callieu	Inspiratory 'predominance'	8	Lip twitching and syncope	15	32	67	145
Donald	Nausea and weakness	5	Syncope and confusion	15	31	73	165
Green	Pins and needles in hands	12	Lip twitching	14	19	69	149
Knight	Twitch left lower lip	1	Lip twitching	12	18	64	125
Tewson	Stinging sensation beneath eyes	2	Dazed & lip twitching	9	27	72	146
Mulberry	Fine twitch lips		Lip twitching & vertigo	9	23	69	143
Gray	Twitch upper lip	2	Severe lip twitching	7	21	67	141
Gibson	Slight twitch upper lip	2	Diaphragmatic spasm	7	23	68	133
Kirk	Fine twitch both lips		Severe nausea	6	21	68	133
Williams	Slight tremor of lips	2	Severe lip twitching	6	23	71	166

TABLE 2

DIVES TO 50 FT. IN THE WET. LIMIT OF TIME 30 MINS.

No.	Time	Symptoms	Work done
1	30	No symptoms	Exercised with 2-7 lb weights
2	25	CONVULSED	10 mins. with weights
3	30	No symptoms	Mild exercise
4	14	CONVULSED	None
5	21	Lip twitching	Mild exercise
6	12	Malaise	5 mins. gently with weights
7	30	No symptoms	Mild exercise
8	9	Lip twitching	Mild exercise
9	10	Lip twitching	Mild exercise
10	30	No symptoms	Hard exercise
11	11	Nausea and respiratory distress	Mild exercise
12	13	CONVULSED	None
13	18	CONVULSED	Mild exercise
14	16	Lip twitching	Intermittent exercise
15	28	Lip twitching	Mild exercise
16	30	No symptoms	Mild exercise
17	30	No symptoms	None
18	30	No symptoms	Mild exercise
19	10	CONVULSED	Mild exercise
20	30	No symptoms	Mild exercise
21	20	Lip twitching	Mild exercise
22	30	No symptoms	Mild exercise
23	30	No symptoms	Mild exercise
24	30	No symptoms	Mild exercise
25	27	CONVULSED	Mild exercise
26	30	No symptoms	Work with weights
27	30	No symptoms	Mild exercise
28	30	No symptoms	Mild exercise
29	20	Lip twitching	None
30	23	CONVULSED	Mild exercise
31	27	CONVULSED	Mild exercise
32	30	Lip twitching	Mild exercise
33	30	No symptoms	Mild exercise
34	18	CONVULSED	None
35	19	CONVULSED	None
36	30	No symptoms	Mild exercise
37	30	CONVULSED	Mild exercise
38	25	Lip twitching	None
39	30	No symptoms	Mild exercise
40	29	Lip twitching	None
41	10	Lip twitching	None
42	30	No symptoms	None
43	30	No symptoms	None
44	30	No symptoms	Mild exercise
45	25	Lip twitching	Mild exercise
46	30	No symptoms	Mild exercise
47	30	No symptoms	None
48	30	No symptoms	Mild exercise
49	24	CONVULSED	None
50	30	No symptoms	None
51	20	CONVULSED	None
52	30	Lip twitching	None
53	30	No symptoms	None

No.	Time	Symptoms	Work done
54	30	No symptoms	None
55	30	No symptoms	None
56	29	CONVULSED	None
57	30	No symptoms	None
58	30	No symptoms	None
59	27	CONVULSED	Mild exercise
60	30	No symptoms	None
61	30	No symptoms	None
62	30	No symptoms	None
63	30	No symptoms	None
64	30	No symptoms	None
65	30	No symptoms	None
66	30	No symptoms	None
67	30	No symptoms	None
68	30	No symptoms	None
69	22	CONVULSED	None
70	29	CONVULSED	None
71	16	CONVULSED	None
72	30	Lip twitching	None
73	30	No symptoms	None
74	29	CONVULSED	None
75	30	No symptoms	None
76	30	No symptoms	None
77	30	No symptoms	None
78	30	No symptoms	None
79	20	CONVULSED	None
80	30	No symptoms	None
81	30	No symptoms	None
82	7	CONVULSED	None
83	30	No symptoms	None
84	20	Severe lip twitching	None
85	28	CONVULSED	None
86	20	CONVULSED	None
87	22	Lip twitching	None
88	10	Lip twitching	None
89	18	Lip twitching	None
90	30	No symptoms	None
91	31	Nausea	None
92	30	No symptoms	None
93	24	CONVULSED	None
94	30	No symptoms	None
95	30	No symptoms	None
96	15	CONVULSED	None
97	12	CONVULSED	None
98	17	Lip twitching	None
99	18	Lip twitching	None
100	11	Lip twitching	None

TABLE 4

TIMES OF TOLERANCE OF FIVE SUBJECTS AT VARIOUS DEPTHS IN THE WET

	50FT.		60FT.		70FT.		80FT.		90FT.		100FT.	
					DEPTH OF FEET OF SEAWATER							
	Time in Mins	Day and Symptoms	Time in Mins	Day and Symptoms	Time in Mins	Day and Symptoms	Time in Mins	Day and Symptoms	Time in Mins	Day and Symptoms	Time in Mins.	Day and Symptoms
WHITTINGTON	58	23rd Severe lip twitching	61	5th Bad lip twitching	21	30th Lip twitching	8	37th Lip twitching	11	41st Lip twitching	12	28th Lip twitching
BROWN	44	26th Severe lip twitching	19	5th Convulsed	14	33rd Lip twitching	37	37th Lip twitching	12	40th Lip twitching	15	28th Lip twitching
HARMAN	112	22nd Severe lip twitching	76	6th Severe lip twitching	44	29th Lip twitching	26	35th Lip twitching	25	44th Lip twitching	19	27th Lip twitching
FRASER	120	19th No symptoms	37	7th Lip twitching	74	34th Lip twitching	45	36th Lip twitching	18	41st Lip twitching	19	29th Lip twitching
DICKIE	121	20th No symptoms	12	9th Lip twitching	55	33rd Lip twitching	28	35th Convulsed	9	42nd Lip twitching	24	29th Lip twitching

TABLE 7

TOLERANCE OF DIVERS IN THE WET AT 50 ft RESTING

Minutes	Diver	Symptoms
3	Kirk	Lip twitching
10	Martin	Lip twitching
11	Mulberry	Lip twitching
12	Gibson	CONVULSED
15	Morton	CONVULSED
15	Witham	CONVULSED
16	Morfett	CONVULSED
17	Knight	CONVULSED
17	Hutton	Lip twitching
18	Green	Slight lip twitching
19	Hill	CONVULSED
19	Hill	Lip twitching, Nausea
19	McInnes	Lip twitching
20	Donnelly	CONVULSED
20	Carpenter	CONVULSED
20	McCann	Lip twitching
22	Dodd	Lip twitching
22	Ladham	CONVULSED
24	McLaughlin	CONVULSED
26	Sims	Lip twitching, headache
27	Liddle	CONVULSED
28	Gray	CONVULSED
28	Powell	Slight lip twitching, vertigo

Minutes	Diver	Symptoms
28	Smith	CONVULSED
29	Symington	CONVULSED
29	Woolcott	CONVULSED
35	Ward	Lip twitching, nausea
35	McAtamney	Lip twitching and loss of consciousness
35	Robertson	Lip twitching
44	Brown	Slight lip twitching
59	Whittington	Lip twitching and vertigo
69	Shields	CONVULSED
85	Smith I	Sudden lip twitching
90	Rogers	Lip twitching and nausea
90	Derrick	Coughing
99	Wallis	Lip twitching
112	Harman	Severe lip twitching
120	Fraser	No symptoms
120	Warren	No symptoms
121	Dickie	No symptoms

Total No. of Divers – 40.

TABLE 8

TOLERANCE OF DIVERS IN THE WET AT 60 ft RESTING

Minutes	Diver	Symptoms
2	Kirk	Lip twitching and vertigo
8	Ward	Lip twitching
9	Hutton	Lip twitching
12	Dickie	Lip twitching
13	Sims	Lip twitching
15	Mulberry	Lip twitching
16	McAtamney	Vertigo
19	Brown	CONVULSED
19	Robertson	Lip twitching
23	Martin	Lip twitching
24	Wallis	Lip twitching
25	Warren	CONVULSED
37	Rogers	Severe lip twitching
37	Fraser	Lip twitching
40	Derrick	Lip twitching
73	Whittington	Severe lip twitching
76	Harman	Lip twitching and body tremors
80	Smith II	Severe lip twitching

Total No. of Divers – 18

TABLE 9

TOLERANCE OF DIVERS IN THE WET AT 70 ft RESTING

Minutes	Diver	Symptoms
1½	Kirk	Lip twitching
4	Mulberry	Lip twitching
8	Martin	Vertigo and Nausea
10	Wallis	Lip twitching and vertigo
11	Robertson	Lip twitching
12	Sims	Lip twitching
14	Brown	Slight lip twitching and dyspnoea
15	Liddle	CONVULSED
15	Powell	Lip twitching
17	Clarkson	Lip twitching
17	Symington	CONVULSED
19	Whittington	Lip twitching
20	Baldwin	Lip twitching
23	Hill	Syncope
26	Rogers	Lip twitching
26	Herrett	Lip twitching
32	Miller	CONVULSED
38	Ward	Lip twitching
39	McAtamney	Vertigo, feeling of exhaustion, dazed.
43	Warren	Slight lip twitching
44	Harman	Lip twitching
55	Dickie	Lip twitching
74	Fraser	Slight lip twitching
77	Derrick	Slight lip twitching

Total No. of Divers – 24

TABLE 10

TOLERANCE OF DIVERS IN THE WET AT 80 ft RESTING

Minutes	Divers	Symptoms
2	Martin	Vertigo
3	Kirk	Lip twitching
4	Mulberry	Lip twitching, vertigo
7	Hutton	Lip twitching
8	Smith I	Lip twitching
8	Whittington	Lip twitching
8	Wallis	Lip twitching
9	Robertson	Severe lip twitching
11	Woolcott	Severe lip twitching, nearly out
13	Clarkson	Lip twitching
13	Sims	Lip twitching, headache
13	Smith II	Spasmodic respiration, manual tremors
13	Ward	Lip twitching. headache
14	Baldwin	Lip twitching
15	Herrett	CONVULSED
17	Hill	CONVULSED
18	McAtamney	Vertigo, weariness
20	Derrick	Lip twitching
22	Warren	Lip twitching
25	Harman	Lip twitching
27	Rogers	Lip twitching, nausea
27	Powell	CONVULSED

Minutes	Divers	Symptoms
28	Dickie	Coughing, CONVULSED
33	Ware	CONVULSED
35	Brown	Lip twitching
44	Fraser	Slight lip twitching, dyspnoea
75	Miller	Lip twitching

Total No. of Diver – 27.

TABLE 11

TOLERANCE OF DIVERS IN THE WET AT 90 ft RESTING

Minutes	Diver	Symptoms
2	Kirk	Lip twitching
4	Wallis	Lip twitching
4	Mulberry	Lip twitching, body and leg tremors
7	Ward	Lip twitching
9	McAtamney	Drowsiness, CONVULSED
9	Sims	Severe lip twitching
9	Dickie	Lip twitching
11	Whittington	Lip twitching
11	Rogers	Lip twitching and vertigo
11	Robertson	Lip twitching, CONVULSED
12	Brown	Lip twitching
18	Fraser	Lip twitching
25	Harman	Lip twitching
38	Derrick	Coughing
71	Smith II	Lip twitching

Total No. of Drivers – 15

TABLE 12

TOLERANCE OF DIVERS IN THE WET AT 100 ft RESTING

Minutes	Diver	Symptoms
2	Kirk	Lip twitching
3	Wallis	Lip twitching, headache
3	Mulberry	Lip twitching, Pulsation in arms
3	Ward	Vertigo, drowsiness
4	Robertson	Lip twitching
4	Hutton	Lip twitching, vertigo
5	McAtamney	Vertigo, weariness
7	Sims	Lip twitching
7	Rogers	Lip twitching, nausea, slight vertigo
11	Brown	Severe lip twitching
12	Whittington	Lip twitching
19	Fraser	Lip twitching
24	Dickie	Lip twitching
36	Derrick	Lip twitching

Total No. of Divers – 14.

TABLE 15

ASSESSMENT OF INDIVIDUAL AND GROUP VARIATION
LOGARITHM OF DIVER'S TIME MINUS LOGARITHM OF MEAN VALUE FOR EACH DEPTH

Pressure	50 ft. under-water	60 ft. under-water	70 ft. under-water	80 ft. under-water	90 ft. under-water	100 ft. under-water	90 ft. in air	Variance	Standard Deviation
Diver									
Kirk	-1.2553	-1.0038	-1.1152	-0.6603	-0.7154	-0.4717	-0.6639	0.08159	0.2856
Mulberry	-0.6910	-0.1267	-0.6892	-0.4842	-0.4143	-0.3925	-0.4879	0.03717	0.1928
Wallis	+0.2632	+0.0844	-0.2701	-0.2060	-0.4143	-0.3925	-0.0804	0.06280	0.2506
Robertson	-0.1822	-0.0148	-0.2499	-0.1597	+0.0443	-0.2675	-0.0529	0.01440	0.1200
Ward	-0.1883	-0.4017	+0.2885	-0.0235	-0.1413	-0.3577	+0.0764	0.05909	0.2431
Sims	-0.3029	-0.1909	-0.2121	-0.0235	-0.387	+0.0055	+0.3840	0.05090	0.2256
McAtamney	-0.1822	-.01007	+0.2998	+0.1179	-0.0622	-0.170b	-0.0356	0.02980	0.1726
Brown	-0.0840	-0.0260	-0.1452	+0.4128	+0.0628	+0.1911	+0.2655	0.04078	0.2019
Rogers	+0.2218	+0.2634	+0.1237	+0.3019	+0.0443	+0.0055	-0.1968	0.02939	0.1714
Whittington	+0.0421	+0.5615	-0.0013	-0.2343	+0.0250	+0.2185	+0.2943	0.06495	0.2549
Dickie	+0.5281	-0.2079	+0.4491	+0.3098	+0.0387	+0.5240	+0.0631	0.08397	0.2898
Smith II	+0.1970	+0.5983	+0.3458	-0.0235	+0.8349	+0.0055	-0.2117	0.13719	0.3704
Fraser	+0.5281	+0.2692	+0.5779	+0.5110	+0.2508	+0.4092	+0.0957	0.03247	0.1802
Derrick	+0.2218	+0.2973	+0,5980	+0.1636	+0.5634	+0.6927	+0.5402	0.04308	0.2076
Mean								0.05483	0.2342

TABLE 17

DIVES IN THE WET ON OXYGEN AT 40 FT WITHOUT WORK

Name of Diver	Time in Min.	Symptoms
Donnelly	150	Nil
Brown	67	Lip twitching
Mulberry	92	Lip twitching, nausea
McAtamney	90	Lip twitching
Robertson	120	None
McAtamney	120	None
Rogers	120	None
Wallis	120	None
Allender	28	CONVULSED
Brewster	28	CONVULSED
Gibson	120	None
Kirk	18	Lip twitching
Brown	30	CONVULSED
Smith	12	CONVULSED
Ward	120	None
Kirk	18	Lip twitching
Martin	19	Lip twitching and nausea
Hunt	120	None
Hutton	29	Lip twitching
Gibson	50	Lip twitching, nausea
Hutton	120	None
Rutter	29	Lip twitching

Snelling	120	None
Kirk	27	Lip twitching
Gibson	55	Nausea and lip twitching
Senner	120	None
Rolfe	120	None
Elsby	120	None
Symmington	150	None

TABLE 18

DIVES IN THE WET ON OXYGEN AT 35 FT WITHOUT WORK

Name of Diver	Time in Min.	Symptoms
Brown	180	None
Gibson	180	None
Kirk	30	CONVULSED
Williams	14	Lip twitching
Kirk	32	Lip twitching
Hunt	120	None
Hutton	120	None
Senner	40	Throat twitching
Elsby	120	None
Snelling	120	None
Rutter	31	Lip twitching
Sims	90	None
Smith	30	Lip twitching
Rogers	90	None
McAtamney	90	None
Ward	90	None
Scott	90	None
Martin	90	None
Wallis	90	None
Mulberry	90	None
Hutton	90	None

TABLE 19

DIVES IN THE WET ON OXYGEN TO 30 FT WITHOUT WORK

Name of Diver	Time in Min.	Symptoms
Robertson	120	None
Kirk	48	CONVULSED
Gibson	120	None
Ward	120	None
Hunt	120	None
Wallis	120	None
Rogers	120	None
Hutton	120	None
McAtamney	43	CONVULSED
Snelling	120	None
Senner	120	None
Rutter	24	Vertigo
Rolfe	120	None
Robertson	90	None
Mulberry	90	None
Sims	90	None
Kirk	90	None
Scott	90	None
Martin	90	None
McAtamney	90	None

TABLE 20

DIVES IN THE WET ON OXYGEN TO 25 FT WITHOUT WORK

NAME OF DIVER		
Robertson	Downer	Kenny
Kirk	Shean	Butt
McAtamney	Mays	Leslie
Ward	Cotter	Carr
Wallis	Morley	Locke
Gibson	Stubbs	Snelling
DeAsha	Payne	Rolfe
Merriman	A. Smith	Senner
McIlduff	Maidment	Rutter
	Elsby	

All these divers completed 120 minutes, at 25 feet, with no symptoms, except Butt, who suffered from nausea and vomiting after 44 minutes. This subject had similar symptoms when breathing oxygen at atmospheric pressure.

TABLE 21

OXYGEN POISONING AT 50 ft. IN THE WET WITH WORK

Subject	Times in min.	Symptoms	Total Work done in ft. lbs	Control without Work	
				Time in Min.	Symptoms
Elsby	8	Lip twitching	12,801	120	None
Elsby	27	Lip twitching	28,105	120	None
Elsby	33	Lip twitching	31,500	–	–
Friels	22	Lip twitching	25,200	21	Nausea. Vertigo
Friels	–	–	–	28	Nausea
Grabham	15	Lip twitching Vertigo	14,630	10	Lip twitching
Grabham	–	–	–	12	Lip twitching
Gibson	8	Lip twitching	5,101	19	Body tremors
Gibson	5	Lip twitching	11,068	20	Lip twitching
Gibson	10	Lip twitching	5,740	12	Lip twitching
Hunt	58	Lip twitching	53,033	120	None
Hunt	60	Vertigo	56,017	67	Lip twitching
Hunt	63	Lip twitching	70,350	–	–
Hutton	13	Lip twitching	22,137	20	Body tremors
Hutton	22	Lip twitching	29,741	20	Lip twitching
James	64	Lip twitching	66,640	120	None
Kirk	1	Lip twitching	1,480	73	Lip twitching
Kirk	3	Vertigo	3,712	4	Lip twitching
Kirk	4	Lip twitching	4,372	7	Lip twitching
McAtamney	10	CONVULSED	28,328	42	Lip twitching
McAtamney	10	Vertigo	10,395	20	Vertigo

Subject	Times in min.	Symptoms	Total Work done in ft. lbs	Control without Work	
				Time in Min.	Symptoms
McAtamney	3	Vertigo	5,390	38	Vertigo
Mulberry	6	Lip twitching,nausea	15,134	12	Lip twitching
Robertson	8	Lip twitching	18,826	120	None
Robertson	7	Lip twitching, nausea	10,202	120	None
Robertson	20	Lip twitching	35,011	–	–
Rogers	16	Vertigo	28,216	120	None
Rogers	32	CONVULSED	61,710	101	Lip twitching
Rogers	48	Pain in chest (probably not O_2	50,380	121	None
Rogers	24	Lip twitching, Vertigo	25,130	–	–
Rolfe	43	Lip twitching	45,141	120	None
Rolfe	38	Lip twitching	41,404	120	None
Rolfe	18	Lip twitching	17,010	–	–
Rutter	5	Lip twitching	6,545	10	Lip twitching
Rutter	12	Lip twitching	19,346	–	–
Senner	3	Body tremors	7,988	120	None
Senner	–	–	–	43	CONVULSED
Senner	6	CONVULSED	6,256	8	Lip twitching
Senner	–	–	–	5	Throat tremors
Senner	4	Throat tremors, nausea	3,920	–	–
Sims	22	Lip twitching	26,516	26	Lip twitching
Snelling	37	Lip twitching	49,761	120	None
Snelling	56	CONVULSED	48,895	–	–
Wallis	11	Lip twitching	28,918	99	Lip twitching, nausea
Wallis	12	Lip twitching	22,618	28	Lip twitching, vertigo
Wallis	16	Lip twitching	34,168	65	Lip twitching
Wallis	40	Lip twitching vertigo, nausea	44,590	–	–
Ward	13	Nausea	12,993	35	Nausea
Ward	29	Nausea	42,446	79	Lip twitching
Ward	33	Nausea	–	39	Lip twitching

TABLE 22

OXYGEN POISONING AT 40 ft. IN THE WET WITH WORK

Subject	Time in Min.	Symptoms	Total Work done in ft. lbs.	Control without Work	
				Time in Min.	Symptoms
Elsby	15	Lip twitching	22,137	120	None
Elsby	60	None	81,427	–	–
Elsby	120	None	113,400	–	–
Friels	45	Vertigo, nausea	38,500	–	–
Gibson	10	Lip twitching	9,336	120	None
Gibson	34	Lip twitching, body tremor	50,435	50	Lips, nausea
Gibson	13	Lip twitching	12,110	–	–
Grabham	16	Lip twitching	16,100	–	–
Hunt	60	None	93,940	120	None
Hunt	30	Nausea	44,563	–	–
Hunt	48	Lip twitching	99,099	–	–
Hunt	120	None	187,790	–	–
Hutton	22	Lip twitching	38,981	120	None
Hutton	38	Lip twitching	43,601	–	–
James	120	None	97,020	–	–
Kirk	8	Vertigo	7,837	24	Lip twitching
Kirk	6	Lip twitching	6,765	18	Lip twitching
Kirk	6	Lip twitching	4,200	27	Lip twitching
McAtamney	7	Nausea	11,646	120	None
McAtamney	8	Vertigo	7,604	–	–
Robertson	60	None	89,801	90	None
Robertson	60	None	80,753	120	None
Rogers	60	None	109,670	120	None
Rogers	120	None	141,180	90	None
Rogers	43	Nausea	78,650	–	–
Rogers	39	CONVULSED	40,040	–	–
Rogers	35	Dyspnoea	65,230	–	–
Rolfe	38	Lip twitching	40,714	120	None
Rolfe	23	Dyspnoea	17,768	–	–
Rolfe	41	Vertigo	27,790	–	–
Rutter	6	Vertigo	5,293	29	Lip twitching
Rutter	3	Dyspnoea	1,925	–	–
Senner	5	Throat tremors	8,566	120	None
Senner	17	Vertigo	22,426	5	Lip twitching
Senner	9	Lip twitching, throat tremors	10,220	–	–
Snelling	60	None	79,046	120	None
Snelling	60	None	63,525	–	–
Wallis	60	Lip twitching	92,592	120	None
Wallis	30	Lip twitching	54,092	90	None
Wallis	35	Nausea	38,981	–	–
Wallis	57	Lip twitching	131,670	–	–
Wallis	64	Lip twitching	104,896	–	–
Wallis	28	Nausea	29,750	–	–
Ward	30	Vomiting	31,281	120	None
Ward	35	Lip twitching	38,981	90	None
Ward	46	Nausea	63,070	–	–

TABLE 23

OXYGEN POISONING AT 30 ft. IN THE WET WITH WORK

Subject	Time in Minutes	Symptoms	Total Work done in ft. lbs.	Control without Work	
				Time in Minutes	Symptoms
Friels	51	Nausea	40,110	–	–
Gibson	39	Vertigo	24,736	120	None
Gibson	61	Nausea	74,270	–	-
Kirk	34	Lip twitching Vertigo	–	48	CONVULSED
Kirk	37	Vertigo	24,640	90	None
McAtamney	19	Vertigo	22,715	43	CONVULSED
Rogers	60	Vertigo, nausea	114,950	120	None
Rogers	24	Vertigo	56,107	–	–
Rogers	120	None	152,180	–	–
Rutter	17	Lip twitching	24,447	24	Vertigo
Senner	10	CONVULSED	14,533	120	None
Senner	21	Lip twitching	19,880	–	–
Wallis	120	None	126,770	120	None
Ward	120	None	129,150	120	None

TABLE 24

OXYGEN POISONING AT 25 ft. IN THE WET WITH WORK

Subject	Time in Min.	Symptoms	Total Work done in ft. lbs.	Control without Work	
				Time in Min.	Symptoms
Friels	120	None	169,190	–	–
Gibson	60	Vertigo	61,118	120	None
Gibson	120	None	126,400	–	–
Kirk	42	Lip twitching	37,785	120	None
Kirk	120	None	119,420	–	–
Senner	28	Lip twitching	38,211	120	None
Senner	54	Lip twitching Vertigo	56,017	–	–
Senner	120	None	89,110	–	–
Ward	60	None	60,076	120	None
Wallis	60	None	111,833	120	None
Rogers	60	None	148,449	60	None
Robertson	60	None	130,322	120	None
McAtamney	60	None	111,553	120	None
Hutton	54	Vertigo	94,806	–	–
Rolfe	60	None	81,138	120	None
Snelling	60	None	160,448	120	None
Elsby	60	None	227,150	120	None
Rutter	60	None	91,630	120	None

APPENDIX 3

SYMBOLS

These are based on those recommended by Pappenheimer (Fed. Proc.,1950, 9, 602).

Primary Symbols: are printed in italics and are as follows:

V	volume of gas or blood	F	Fractional concentration of gas
V	gas volume per unit time	f	Frequency of respiration per minute
P	pressure	D	equivalent depth

The symbol P is used in several ways:

(a) Describing atmospheric or ambient pressure
(b) When representing partial pressure exerted by a designated gas in a mixture of gases.
(c) When representing tension of a designated gas dissolved in a liquid.

Suffixes: qualify the primary symbol and define the anatomical site and/or substance to which the measurement refers.

I	Inspired gas	B	Barometric pressure
E	Expired gas	AMB	Diver's ambient pressure
A	Alveolar gas	a	Arterial blood
T	Tidal gas	v	Venous blood
ET	End-tidal gas	$\bar{v}$	Mixed venous blood

Suffixes are printed in Roman type, capitals when in gas phase and lower case when in liquid phase.

DEPTH AND PRESSURE

Atmospheres (or bars) are employed to define pressure, ATA for atmospsheres absolute, ATS for atmospheres above one atmosphere.

Metres or feet of sea water (msw, fsw) are used when giving the depth of the diver.

These conventions are not strictly observed in pressure work.

Useful 'running' approximations are:

10 msw = 33 fsw = 1 ATS = 2 ATA = 2 bar
30 msw = 100 fsw = 3 ATS = 4 ATA = 4 bar

Finally, units of volume or pressure are specified throughout and are always those used by the author whose work is being described or reviewed.