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Early Quantitative Studies of Gas Dynamics in Decompression

A. R. BEHNKE

The potential value of quantitative assessment of gas transport has long been appreciated in diving but investigations have been limited in scope and content. Decompression following conventional diving, despite the elaborate mathematical models and sophisticated analysis discussed in previous chapters, has been unsatisfactory and, on occasion, assuredly not safe for any diver. Particularly disturbing is the high incidence of decompression sickness (DCS) occurring under pressure in helium–oxygen diving. Saturation diving has been attended with less difficulty but the prolonged schedules are largely the result of trial and error.

In this section a review of some quantitative studies should serve as an introduction to a systematic program essential in future diving physiology.

INERT GAS TRANSPORT

Many of the handicaps which confronted earlier investigators such as tedious methodology in gas analysis and inability to quantify total body water and fat, which are the chief solvents of inert gases, no longer exist. Helium, for example, can be measured to one part in a million by means of the thermal conductivity cell, and nitrogen can be measured continuously during the course of breathing with the mass spectrometer. With reference to body composition, investigations during the past 25 years have yielded the in vivo content of fat, total body water, blood, potassium and other electrolytes. With knowledge of the solutes of inert gases and total amount of absorption by the body, together with utilization of radioisotopes as part of the newer spectrum of gasometry, essential problems in decompression can be resolved. Models of excellence in recent studies are reports by Groom, Morin and Farhi (1967) and Groom and Farhi (1967). Yet this type of investigation is singularly isolated and discontinuous.

A challenging problem is quantitation of the correct half-time of transport of N_2 and helium following equilibration with body tissues. There is currently a fourfold discrepancy between empirical observations and measurements (admittedly incomplete) of desaturation half-times of the slowest tissue in air (480 min compared with 80 to 150 min) and 240 min compared with 60 min in an helium–oxygen atmosphere (Bühlmann, Frey & Keller 1967). Part of the discrepancy arising from these carefully executed and systematic tests, is that the slowest tissue was subjected to an oversaturation ratio of 1·6 to 1. The decompression procedure was therefore not isobaric, and with evolution of bubbles in early stages, time of decompression may well be prolonged. The decompressions may well represent quasi-therapy.

Nitrogen uptake and elimination in the dog

During the period 1932 to 1935, Shaw et al. designed a closed system of 100 L volume to measure N_2 desaturation (or subsequent uptake following

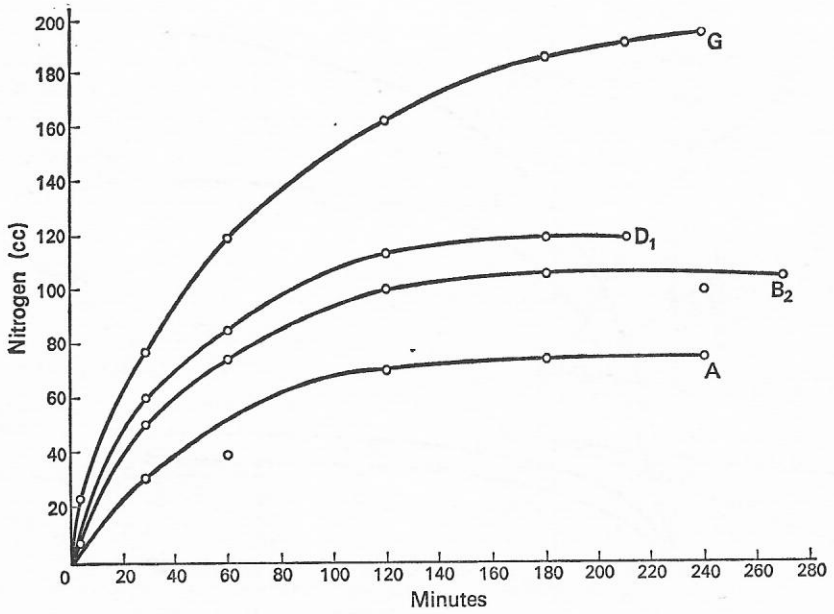


FIG. 21.1. Nitrogen recovery from four anesthetized dogs placed in a closed oxygen (99%) system for periods up to 280 min at 1 ATA. At the end of this period nitrogen elimination is not complete in dog (G) which was old and fat (after Shaw et al. 1935)

initial desaturation) in anesthetized dogs immersed in pure oxygen. Rinsing of the system and the time required for decompression from higher pressures precluded recovery of N_2 during the initial period of 7 to 8 min. The final concentration of N_2 in the system was usually not less than 98·5%. A high degree of analytical accuracy (± 1 part/100,000) was obtained by reduction of large samples following absorption of oxygen, to the 0·25 ml or 0·5 ml calibration of the Van Slyke chamber. The range of variation in control tests was usually within 3 ml. Typical desaturation curves (Fig. 21.1) exhibit individual variation attributed to size, age and fatness. In lean dogs nitrogen recovery approached an endpoint after 3 to 4 hours, in contrast with elimination of nitrogen from a fat dog (G, upper curve on Fig. 21.1). These experiments served to confirm data in goat experiments concerning desaturation time and the importance of fat as a nitrogen reservoir (Boycott & Damant 1908; Boycott, Damant & Haldane 1908). By contrast, in systematic determinations of bends threshold depths for large dogs (48 to 82 lb) exposed in compressed air up to 24 hours followed by rapid 'no-decompression' to 1 ATA, Reeves and Beckman (1966) observed that threshold depth was less following the prolonged exposures than it was up to 7 hours. Either equilibration with nitrogen in compressed air was not attained at the end of 7 hours, or prolonged exposures were conducive to bends independent of the nitrogen content of the dog, at least as measured grossly.

Other curves (Fig. 21.2) revealed noteworthy findings. Thus, during the course of cumulative nitrogen clearance (with the exception of the first 7 min) from tissues of the same dog (D) on different days at 1 and 4 ATA, there was no 'break' or departure from normal (1 ATA) in the nitrogen curves, 4 to 1 ATA. Also, the desaturation curve following partial equilibration is not the reverse of the saturation curve. Thus, following nitrogen clearance at 1 ATA (Fig. 21.2), dog D breathed air for 67 min, and then was cleared of nitrogen (curve B). Following nitrogen clearance after saturation (4-hour exposure) at 4 ATA (curve C), dog D was re-exposed to 4 ATA for 67 min. Nitrogen clearance following abrupt decompression to 1 ATA is represented by curve D.

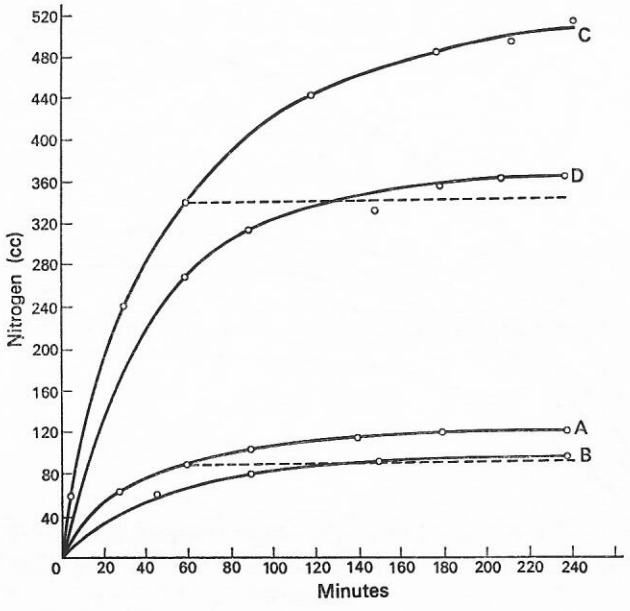


FIG. 21.2. Saturation time compared with desaturation time for anesthetized dog (D). Nitrogen eliminated during the first 7 min (lung rinsing) was not measured. Curve A: N_2 desaturation at 1 ATA; the dog then breathed air for 67 min (1 ATA) followed by N_2 recovery (curve B). Curve C: N_2 desaturation following N_2 equilibration at 4 ATA. The 'N_2-free' dog then breathed air at 4 ATA for 67 min; curve D represents recovery of the N_2 absorbed during the previous 67-min exposure

In an effort to change the shape of the nitrogen recovery curve, say from exponential decay to linear declivity, it was not possible to decompress from higher pressures after long exposures. Either the dog lived and the curve was 'normal' following the sharp drop from higher pressure (i.e. similar to clearance at 1 ATA in the absence of hyperbaric exposure), or the dog succumbed from fulminating gas embolization.

Analysis of the carcass of dog D yielded the following values: weight, 12·23 kg; fat, 1·89 kg; water, 7·23 kg; and dry solids, 3·12 kg. The total body N_2 and component N_2 (Table 21.1) are compared with measured and calculated N_2 during time periods 0 to 7, 8 to 20, and 21 to 180 min. The calculations derive from the equation:

$$\begin{aligned}\text{Total } N_2 \text{ at time } (t) &= \text{Water } N_2(1-e^{-kt}) \\ &\quad + \text{Fat } N_2(1-e^{-kt}) \\ &= 65 \text{ ml} + 102 \text{ ml}\end{aligned}$$

Half-time ($T_{1/2}$) fat from the experimental N_2 curve (see Fig. 21.1) is 30 min ($k=0{\cdot}023$, and 6 time units [TU] = 180 min). Subtraction of calculated fat N_2 from N_2 measured during the period 21 to 180 min (i.e. $85-64=21$ ml) represents clearance of water N_2 for this period, and by difference (total water N_2-21), 44 ml of water N_2 was eliminated from 0 to 21 min.

Measured and calculated N_2 clearances are within the range of analytical error but the N_2 recovery data and computed values from carcass analysis are widely divergent from empirical observations of the time required for N_2 equilibration which is greatly in excess of 180 min (6 TU or 98·4% desaturation).

Uptake of radioactive krypton

Regional studies—uptake in the hand. In 1941, Professor Hildebrand who pioneered the application of helium in diving, initiated a cooperative effort by the Donner Laboratory of Medical

TABLE 21.1

Measured N_2 elimination and calculated N_2 content of fluids and fat derived from carcass analysis of dog (D) following multiple tests (Figs 21.1 and 21.2)

Carcass analysis (kg)		*N_2 content (ml)*	*$T_{1/2}$* (min)*	*6 TU (min)*	*k*
Weight	12·23	—			
Fat	1·89	102	30	180	0·023
Water	7·23	65	12	72	0·058
Solids	3·11	—	—	—	—

Nitrogen elimination

Period (min)	*Measured (ml)*	*Calculated (ml)*		
		Water N_2	*Fat N_2*	*Total*
0–7		22	15	37
8–20	39	22	23	45
21–180	85	21	64	85

* Half-time ($T_{1/2}$) is exponential 50% saturation or desaturation (1 time unit, TU); 6 TU = 98·4% uptake or desaturation; k is the rate constant = $0{\cdot}693/T_{1/2}$ where 0·693 is ln 2.

Physics and the US Navy Experimental Diving Unit to detect and follow the course of intravascular bubbles by use of radioisotopes. Impressive at that time was the air transport of radioactive krypton ($T_{1/2}$ = 36 hours) from California to Washington, D.C. and the subsequent implementation by Dr. Hardin Jones of an inhalation-scanning technique. Although the initial objective of in vivo bubble detection was not realized, it was possible nevertheless to monitor radioactive uptake and elimination of krypton not only in the body as a whole but in regional areas as the forearm and hand as well (Jones 1951). Notable in one series of tests was the accelerated uptake of krypton following release of previously occluded blood supply to the arm for a period of 10 min (Fig. 21.3). The area between curves A and B presumably reflects the relative size of the hyperaemic capillary bed. This type of response may be operative in the remarkable acclimatization associated with repeated exposures in compressed air.

This digression to a discussion of krypton kinetics is apropos of a better definition of the manner of inert gas clearance following *partial saturation* of the body. Use of a radioisotope as a tracer

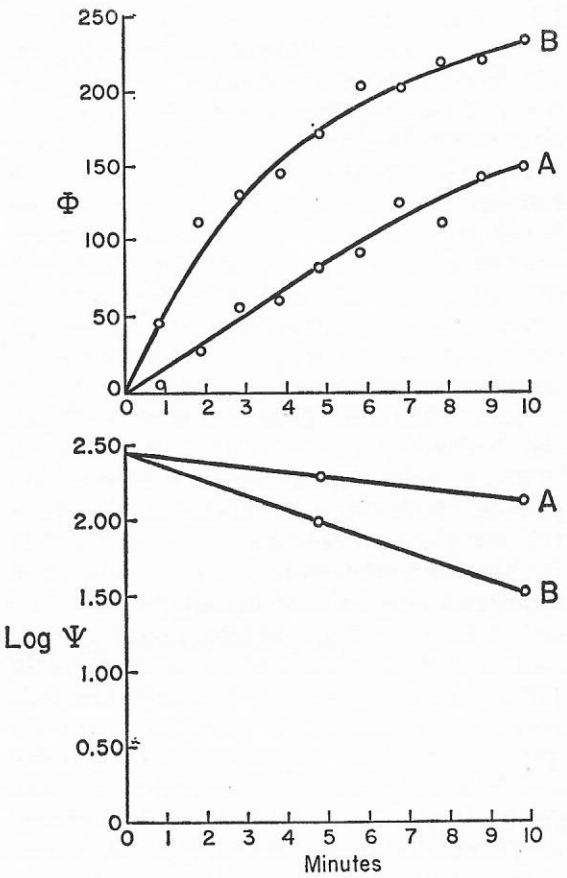


FIG. 21.3. Absorption of inhaled radioactive krypton tracer by the tissues of the shielded hand and forearm. Curve A (upper diagram) depicts absorption of gas by the resting individual. Curve B depicts the absorption of krypton following occlusion of blood supply to the arm for a period of 10 min. Inhalation of krypton began at the end of the occlusive period. Lower diagram shows a semi-logarithmic plot of the upper curves. (Based on the technique of Jones, Hamilton & Lawrence at the Experimental Diving Unit, Washington, D.C., 1942)

affords a refined technique to demonstrate transport of small amounts of gas to or from tissues.

Desaturation time following inhalation of radioactive krypton. A paramount consideration in decompression is the length of time required for complete desaturation following fractional periods of uptake of inert gas. In the studies of Tobias et al. (1949) it was found that an uptake of krypton during a 20-min period of inhalation required more than 100 min for elimination, whereas

inhalation of krypton over a much longer period (117 min) required only slightly longer time (about 120 min) for krypton clearance. However, the shape of the desaturation curves is different for relatively short compared with long exposures.

Tobias et al. (1949) were able to distinguish between 'three distinctly different reservoirs' containing inert gas. The filling of these three reservoirs appeared to be somewhat independent, each characterized by a time-rate constant within a definite range. It is noteworthy that the three half-time values for the respective components are nearly identical both for short and long periods of krypton inhalation. Thus, following a 20-min period of krypton inhalation, the half-times are 6, 39 and 310 min respectively for the three components. Following an inhalation period of 117 min, half-time desaturation values are 6, 41 and 310 min for the respective components. These findings are in accordance with results of N_2 elimination from the dog, and helium recovery from man, namely that the tension of inert gas in various tissues of the body (excluding the bone marrow and perhaps the white matter of the spinal cord) tends toward perfusion–diffusion equalization at a pressure commensurate with the previous level of partial saturation. In a general way, the partial saturation level regulates volume of gas in tissues (i.e. gas content) but not time rate of tissue clearance.

'Bends' retardation of krypton elimination at simulated altitude. In the dog experiments it was not possible to demonstrate any change in the shape of the N_2 elimination curves following abrupt reduction of pressure from several hyperbaric levels presumably sufficient to induce gas phase separation ('silent' bubbles) in the blood stream. Reference will be made in a subsequent paragraph to less effective N_2 elimination during oxygen inhalation at simulated altitudes above 20 000 ft (0·46 ATA), compared with N_2 elimination at ground level (1 ATA) and this was cited as possible retardation of blood flow by 'silent' bubbles. Unequivocal evidence of an altered condition of the blood, and decompression sickness associated with retardation of krypton elimination (Fig. 21.4) is provided by the experiments of Tobias et al. (1949). It is observed that there was a slowing of the rate of krypton desaturation at

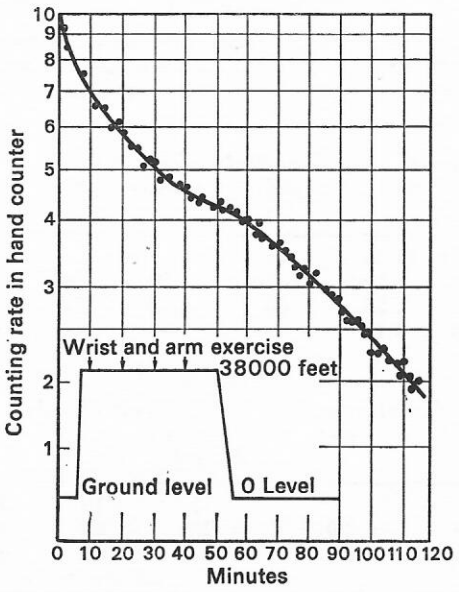


FIG. 21.4. Demonstration of retardation of krypton elimination in the hand during a 1-hour stay at a simulated altitude of 38 000 ft (0·204 ATA) associated with incapacitating bends. On recompression to ground level (1 ATA) pre-recovery rate was restored (after Tobias et al. 1949)

simulated altitude during the time when the subject had incapacitating pain in his knee, shoulder, elbow and wrist. After recompression to ground level the rate of krypton clearance returned to the pre-incapacitation level. Three out of five subjects who remained free from bends at 35 000 ft (0·24 ATA) did not show an altitude-induced change in their krypton curves. One subject, susceptible to bends on previous exposures to altitude, showed a definite slowing of krypton elimination during an altitude test in which he experienced no pain. This retardation could be attributed to the presence of 'silent' bubbles.

Partial saturation relative to diver decompressions and N_2 transport

In 1934, Kagiyama showed that divers could ascend progressively from deeper depths without decompression, provided that exposure time was shortened. Thus a dive could be made to 82 ft (3·5 ATA) for 30 min followed by rapid ascent to the surface, and likewise to a depth of 164 ft (6·0 ATA) for a stay of 15 min without subsequent

decompression stops. In submarine escape tests Shilling and Hawkins (1936) compiled a remarkable series of rapid ascents in the pressurized wet chamber. A large number of simulated escapes (2140) were made with a Momsen lung using air or oxygen from depths of 100 to 200 ft (4 to 7 ATA) with a graded exposure time at each depth until bends supervened. It was found safe under these 'adaptive' conditions to remain 37 min at 100 ft, 18 min at 150 ft and 14 min at 185 ft (6·6 ATA), in no-stop ascents. Incredibly, men of the Royal Navy have made no-stop air-breathing submarine escapes in the open sea from 600 ft (19·9 ATA) with a compression time of 20 sec and decompression of 1 min.

The safe exposure time at depth followed by minimal (no-stop) decompression may be computed from the N_2 elimination curve for the body as a whole (Behnke 1937). The body appears to tolerate a constant volume of excess inert gas for a one-cycle, no-stop decompression, such that the depth times volume relationship is remarkably constant.

N_2 transport at different pressure levels following relatively short exposures. If P_{N_2} tends to equalize by perfusion–diffusion throughout the 'body core' during the course of partial saturation in compressed air, then subsequently, recovery of N_2 during oxygen inhalation should be independent of depth. Preliminary tests supported in part this concept. For example, the quantity of N_2 recovered following a 75-min exposure in compressed air at 100 ft (4 ATA), tended to be the same at the 20, 50, and 100 ft level (Table 21.2). On the other hand, N_2 recovery at 1 ATA following a 2-min decompression from 100 ft (4 ATA) was in two tests strikingly less than it was at higher pressure levels. Early conclusions in regard to partial saturation and N_2 equilibration, possibly naïve in oversimplification, nevertheless are correct in orientation. 'With the exception of tissues with a high fat content (fat deposits, bone marrow and spinal cord) the division of the body into tissues which saturate or desaturate at different rates is largely arbitrary, and the body can be regarded essentially as a unit' (Behnke 1937). An application of this concept in formulation of surface-depth and return decompression is illustrated in Fig. 21.5.

Analysis of nitrogen elimination in man

In man the first measurements of N_2 recovery during the course of oxygen inhalation, were reported in 1913 by Bornstein in studies of cardiac output. It was not until 1931 that Campbell and Hill, employing a modification of the Bornstein method, found that approximately 200 to 300 ml

TABLE 21.2

Nitrogen recovery from a diver exposed to a simulated depth of 100 ft for 75 min compared with nitrogen recovery following an exposure of 30 min at 100 ft. Pure oxygen was inhaled throughout the recovery period

Exposure		*Stop*		*Tests*	*Nitrogen recovery ml*		
(min)	*(ft)*	*(ft)*	*(ATA)*		*3 to 30 min at stop*	*33 to 90 min 0 ft (1 ATA)*	*Total*
75	100	20	1·43	1	1478	834	2312
75	100	50	2·52	2	1533	957	2590
75	100	100	4·0	2	1415	739	2154
30	100	44	2·33	1	1343	548	1891
30	100	50	2·52	1	1312	565	1877
30	100	66	3·0	1	1341	522	1863
30	100	0*	1	1	626	401	1027
30	100	0	1	1	1191	499	1690
30	100	0	1	1	892	856	1748
30	100	0	1	1	1147	511	1658

* 2 min decompression from 100 to 0 ft in the 4 tests.
Data from Willmon and Behnke (1941).

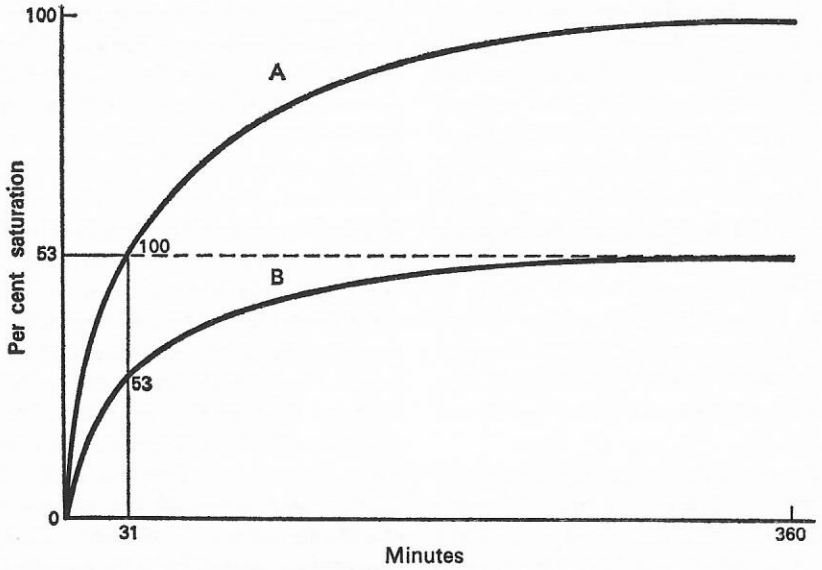


FIG. 21.5. After a dive of 31 min at 100 ft (4 ATA), the body as a whole of this lean diver is 53% saturated as shown on curve A. Equivalent depth is 53 ft and gauge pressure can be reduced to this level. Curve B represents the probable course of N_2 elimination if oxygen were inhaled at the 40 ft level (Behnke 1937)

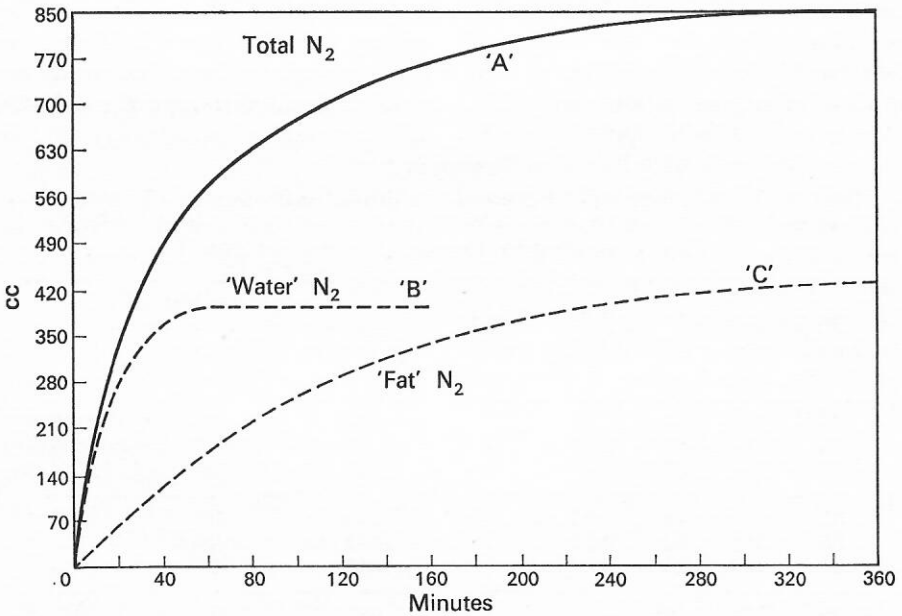


FIG. 21.6. Curve A represents the average values for cumulative nitrogen from three lean men (average weight 64 kg) who breathed oxygen at atmospheric pressure in a helmet system. 'Water' N_2 (B) and 'fat' N_2 (C) are exponential curves with half-times of 7 and 82 min respectively. Nitrogen recovered during the first 5 min (rinsing period) would add a third (faster) half-time component, while progressive fat loading would add a fourth or possibly a fifth half-time component (Behnke 1937)

of nitrogen, about 25 to 33% of the estimated total content in a lean man, were eliminated during the first 9 min of oxygen breathing. Subsequent experiments have confirmed and extended these findings in some degree (Shaw et al. 1935; Behnke et al. 1935; Willmon & Behnke 1941; Stevens et al. 1947; Jones 1950; Boothby, Luft & Benson 1951; Kety 1951; Lundin 1953, 1960).

The amount of N_2 recovered per minute diminishes during the course of oxygen inhalation from an initial calculated 50 ml/min (P_{N_2} = 573 mm Hg) to less than 0·1 ml/min from lean men at the end of 9 hours. At this time diffusion of N_2 through the skin (body, except head surrounded by air) accounts for the greater part of the N_2 recovered; the remainder diffuses into the closed system through the spirometer water seal and other gas-permeable components. The curve depicting cumulative N_2 recovery (Fig. 21.6) was dichotomized, as outlined previously for the dog, into the exponential components comprising the chief body solutes for N_2 in a 64-kg 'composite' diver.

Total N_2 (6 hours)	=	Water N_2 +	Fat N_2
850 ml (6 hours)	=	392	458
Exponential rate constant (k)	=	0·098	0·0085
Half-time ($T_{1/2}$) in min	=	7	82
% desaturation (6 hours)	=	complete	≈ 95

Assessment of body fat and water was not possible at the time; N_2 in the initial period of 6 min was calculated, and the desaturation time of fat (95% in 6 hours) could not be firmly established.

Logarithmic analysis of a similar curve without reference to water and fat solvents, yielded three components:

Total N_2 (at time, t)

$$= 172\, e^{-0.13t} + 353\, e^{-0.028t} + 255\, e^{-0.0079t}$$

The extensive measurements of Jones (1950), over usually not more than several hours, were resolved in one extended experiment into five components referrable to tissues with different rates of blood flow. However, diffusion plays an important role (Hills 1966). Haber (1951) stated that it is hazardous to infer that the exponential components derived from an accurate fit of data are representative of such entities as blood, muscle and fat. He added, 'Furthermore an exponential equation is so flexible that a variable number of constants can be found to fit the data without being representative of the actual physical mechanisms involved.' Haber pointed out the value of recording minute-to-minute quantities of exhaled N_2 as a prerequisite in the calculation of the rate constants. This objective has been realized in part by radioisotopic analysis of Jones (1950), and advanced by Lundin (1960) as a result of development of a highly sensitive N_2 meter.

Lundin's N_2 elimination measurements

In his 1953 paper, Lundin reported cumulative measurements of N_2 from subjects who breathed oxygen in a closed system. He characterized N_2 clearance as a three-stage exponential process:

1. A rapid phase with an elimination half-time of about 1·5 min.
2. A slower phase with a half-time of 12 to 13 min.
3. A slow phase with a half-time of about 100 to 200 min.

From the analysis of Jones (1950) it is reasonable that the first phase corresponds to the N_2 from highly vascular tissue such as liver, brain, heart, intestines, and other organ systems, the middle phase mainly from muscle (and skin), and the slowest from fat. In Lundin's 1960 paper, N_2 elimination was assessed, not in a closed system by cumulative increments, but by an original technique of measurement of end-tidal volume concentrations of N_2 by the sensitive meter previously mentioned. This technique, although not productive of absolute amounts of N_2 eliminated, satisfies Haber's criterion for accurate measurement of rate of N_2 elimination by multiple recordings at short intervals.

In the recording of data, N_2-metered readings in arbitrary units (every 60 sec for the first 20 min, then every 5 min) were plotted on a logarithmic scale against time. N_2 clearance was not measured during the first 6 to 8 min (lung rinsing period during normal breathing) and experiments were terminated at the end of 4 hours (Fig. 21.7). It is this type of technique and newer knowledge of body composition which should provide a firm basis for future studies of gas transport. In the

meantime it seems worthwhile to recast earlier work into a newer mold.

Lundin's analysis of N_2 elimination

The exponential N_2 decay curve (Fig. 21.7) can be resolved into two linear components, one relatively rapid (interpreted as N_2 elimination from muscles, chiefly), the other, a slow component indicative of N_2 clearance from fat (Table 21.3). The arbitrary units referrable to N_2 recovered can

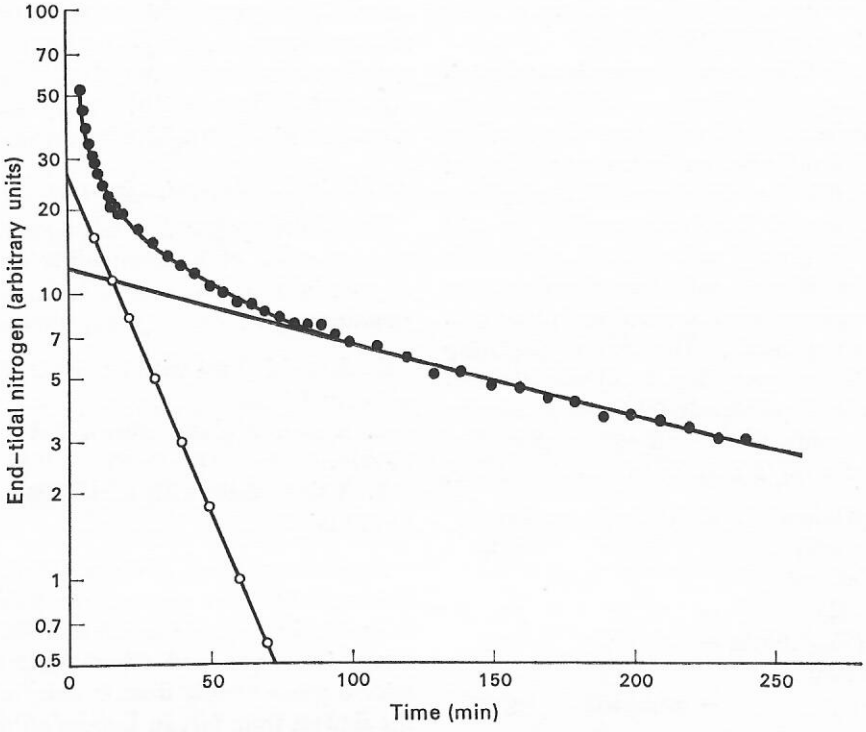


FIG. 21.7. Nitrogen elimination curve, semi-log scale. The end-tidal N_2 (ordinate) is recorded in arbitrary units. (Reproduced from Lundin 1960 by permission of the Editor, *Journal of Physiology, London*)

TABLE 21.3

Semi-logarithmic N_2 clearance from 'muscle' (II) and 'fat' (III) components of the body and the respective k constants during the course of 4-hour end-tidal volume measurements of metered N_2

Subject	*Age* (*years*)	*Weight* (*kg*)	*Height* (*cm*)	*'Muscle'**			*'Fat'**		
				k	$T_{1/2}$ (*min*)	N_2	k	$T_{1/2}$ (*min*)	N_2
1	33	79	182	0·055	12·6	500	0·0059	117	2050
2	28	59	165	0·061	11·4	460	0·0048	144	2670
3	45	80	183	0·051	13·6	588	0·0047	147	2870
4	16	60	163	0·050	13·9	234	0·0051	136	1570
5	16	53	163	0·046	15·1	520	0·0079	88	900
6	22	60	176	0·051	13·6	490	0·0059	117	1290

* N_2 elimination is expressed in arbitrary units relative to the readings of the nitrogen meter. Data from Lundin (1960).

be converted into absolute values derived from indirect estimates of muscle (M) and fat (F) as projected by Lundin.

If body weight $(W) - F \times 0{\cdot}50 = M$ (1)

and if $FN_2 = 6MN_2$, $\text{Fat} = \dfrac{N_2F}{6\,N_2M} \times M$ (2)

Substituting arbitrary units from Table 21.2 in (2), then it follows for Subject 1 that $F = 0{\cdot}684\,M$ and from (1), that Fat $= 0{\cdot}684\ (W - F) \times 0{\cdot}50 =$ 20·1 kg. For Subject 2, Fat = 19·2 kg, and for Subject 3, 23·2 kg.

The correctness of these estimates of body fat derived from logarithmic linear conversions of N_2 clearance data, was confirmed by densitometry (underwater weighing) where densitometric fat was determined as 20·8, 18·7, and 22·8 kg for Subjects 1, 2, and 3 respectively. In order to complete Lundin's novel analysis by addition to categories II and III (Table 21.3), the initial most rapidly desaturating tissues (i.e. in missing category I), it is necessary to outline a practical method for valid assessment of body fat.

QUANTITATIVE APPRAISAL OF BODY FAT RELATIVE TO N_2 TRANSPORT

Reference data

In 1939, following rescue and salvage operations in connection with the USS *Squalus* disaster (Behnke & Willmon 1939), there was renewed interest in a practical method to assess fat in US Navy divers who were then engaged in test dives to 500 ft (15·2 ATA). Since volume displacement of a submarine and buoyancy were matters of daily discussion, serendipity led to the determination of body volume in diving tanks (Fig. 21.8) and the Archimedean parameter of density as a 'third dimension' of the body in addition to weight and stature (Behnke, Feen & Welham 1942). This elementary procedure in discriminate hands has become standard for accurate assessment of body fat (Behnke & Wilmore 1974). If immersion water temperature is 31 to 32°C, then

$$\%\ \text{Body fat} = \frac{1{\cdot}000 - \text{sp. gr. (body)}}{0{\cdot}002}$$

or, in densitometric units:

$$\%\ \text{Body fat} = \frac{495{\cdot}0}{\text{d (body)}} - 450{\cdot}0$$

This is Siri's equation (Siri 1961).

Hundreds of densitometric determinations on young adult males define gross composition of a *reference man* in terms of fat, and with normal hydration, total body water. In the reference man (Table 21.4), fat in adipose tissue is 8 kg, lean body weight is body weight less adipose tissue fat. Fat-free weight is lean body weight less 'essential' fat in lipid-rich tissue as bone marrow and the nervous system. An estimate of 'essential' fat is 2 kg (3% lean body weight), not large, but an entity which merits careful scrutiny in pathology of bone and spinal cord. Total body water is 72%

TABLE 21.4

Gross body composition data of a *reference man* in relation to the inert gas content of body fluid and fat solvents

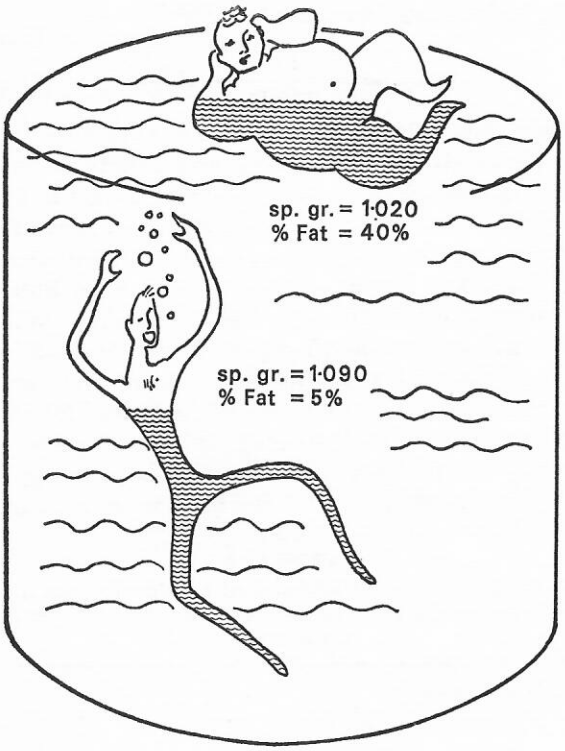
Reference man	
Age, 20 to 24 years; weight, 70 kg; stature, 174 cm	
Fat in adipose tissue (AT)	8 kg
Lean body weight (LBW)	62 kg
'Essential' organ, marrow fat	2 kg
Fat-free weight	60 kg
Total body water (TBW), 72% LBW	44·6 kg
Solubility data (P_{N_2}, $P_{He} = 570$ mm Hg; 37°C)	
N_2 in tissue fluids	9 ml/kg
N_2 in fat	54 ml/kg
He in tissue fluids	5·9 ml/kg
He in fat	12·2 ml/kg

*Variation of parameters**

	-2σ	-1σ	M	$+1\sigma$	$+2\sigma$
LBW (kg)	47·2	54·6	62	68·4	75·8
TBW (kg)	29·3	39·3	44·6	49·2	54·6
Fluid N_2 (ml)	264	354	401	443	491
Fluid He (ml)	172	230	261	288	319

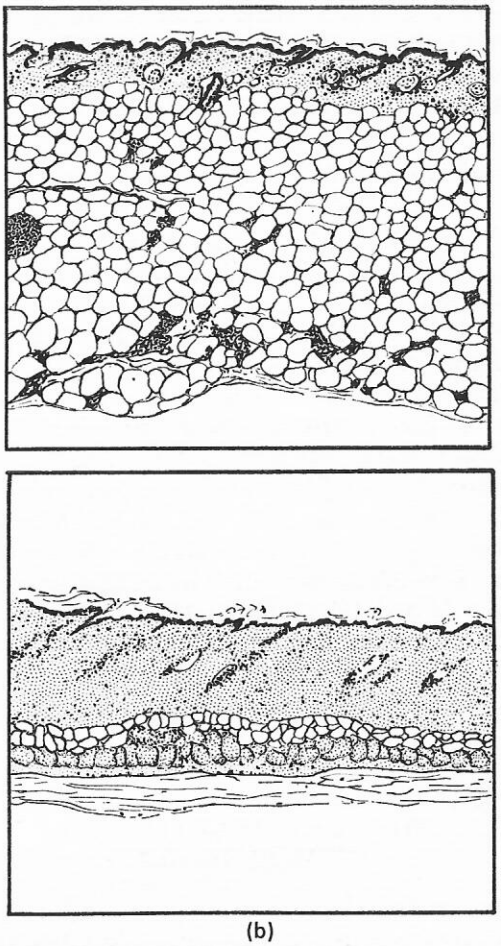
AT fat (kg)	*Very lean*	*Ref. man*	+	++	+++
	4	8	12	16	20
N_2 content (ml)	216	432	648	864	1080
He content (ml)	48	97	145	194	242

* Calculated from representative coefficients of variation of lean and total body weights of 12% (range 10 to 14%).



(a)

FIG. 21.8 (a) The technique of underwater weighing referrable in principle to Archimedes provides an accurate assessment of body fat which is inversely proportional to net body fat sp. gr. (water temp. 31 to 32°C)



(b)

FIG. 21.8 (b) The panniculus adiposus of fat in lean mice is analogous to the variation in fat content of the diver's tela subcutanea (after Hausberger 1957)

(range 68 to 76%) of lean body weight, and in the animal carcass, 73% of fat-free weight.

The following solubility values, derived from Bunsen coefficients (37 to 38°C) are not rigidly established but suffice for this analysis.

	Water	*Oil*
N_2	0·013	0·065
He	0·0085	0·015
He/N_2	64·5%	23·1%

Gas solubility in tissue fluid (water) requires a correction for dissolved solutes. With reference to N_2 (solubility 0·975 vol %, P_N = 570 mm Hg) the correction derived from urine analysis (to be discussed subsequently) is,

$$0{\cdot}975 - \frac{\text{sp. gr. (fluids)} - 1{\cdot}0}{0{\cdot}2} = 0{\cdot}90$$

(sp. gr. [fluids] 1·015)

Updating this type of analysis is the authoritative investigation of Klocke and Rahn (1961).

In Table 21.4, fat has been converted to kg by multiplying volume by 0·90 (d fat 0·90) to obtain a rounded value of 6 as the ratio in Lundin's analysis of fat to fluid nitrogen. The Gausserian distribution of lean body weight and total body weight relates to a coefficient of variation of 12% (range 10 to 14%), and includes about 95% of mean values of lean body weight and total body weight for young adult males whose stature (h) is 174 cm, and for different statures, mean lean body weight = 0·204 h^2 (h in decimeters). The range of values for adipose tissue fat are projected as 4 kg

(very lean), 8 kg (reference diver), and 4(+), 8(++), and 12(+++) of excess fatness.

Effect of increased fatness on N_2 transport

If 12 kg of fat accumulate during the course of hypertrophy of adipose tissue (i.e. without hyperplasia or additional blood supply), weight (reference man) is increased from 70 to 82 kg and total body fat from 11·4 to 24·4%. The following range of estimates pertain to the time course of N_2 desaturation of the *excess* plus reference fat (12+8 kg) compared with reference fat (which includes, in category III, 'essential' fat and water). water and fat in each category are derived from reliable source data. The blood supply in the resting state (not given in Table 21.5) is about 4 L for category I, 1·2 L for category II, and 0·5 L for category III. It is apparent that the disparity between the distribution of blood flow and tissue mass in category III severely impairs the transport of lipophilic gases such as nitrogen.

The k values and half-times $T_{1/2}$ for category II are from Lundin's data; for category III, from earlier measurements of N_2 elimination in lean men (Behnke 1937); and for category I, from an estimate of N_2 clearance from highly vascularized

	N_2 *content*	k	$T_{1/2}$ (*min*)	*Cumulative* N_2 (*ml*)				
min				*6*	*30*	*60*	*90*	*6 TU**
Reference fat	542	0·0082	85	26	118	210	282	533
(1)+12 kg	1190	0·0048	143	35	159	300	421	1171
(2)+12 kg	1190	0·0037	187	26	138	238	339	1171

* 6 TU (reference fat) 510 min, excess fat (1) 864 min, excess fat (2) 1122 min.

The tabular values pertaining to (1) approximate Lundin's half-times for the slowest tissue in his moderately fat subjects, and were calculated from the ratio,

$$\frac{(1190)^{2/3}}{(542)^{2/3}} \times 85 \text{ min}$$

where 542 ml is the N_2 content of category III (reference man) and 85 min is half-time. The values (2) are calculated as an increase in half-time proportional to N_2 content of fat (reference man). The challenging aspect of N_2 measurements of young men of variable fatness (assessed by underwater weighing) is the insight afforded into the relative role of diffusion compared with perfusion. Impressive in earlier but incomplete studies was the unusually large amount of N_2 recovered in the first 2 hours which was well in excess of the quantity calculated for perfusion-limited adipose tissue. Measurements currently should be carried out, preferably over a period of 24 hours, starting at 1 ATA and continuing the N_2 recovery in an altitude chamber at 0·25 ATA. In such tests it is necessary to circumvent diffusion of N_2 through the skin.

In Table 21·5, three tissue categories are projected for the reference man and the amounts of tissue. If the lungs are rinsed free of N_2 within 45 sec, N_2 from category I can be quantified.

Application of 3-component analysis to Lundin's data

The partition of N_2 dissolved in fluid and fat into three categories has been applied to four of Lundin's subjects (Table 21.6). Total body water referrable to tissue content is allocated to I (27%), II (62%) and III (11%) categories. Essential fat (in organs) approximated as 1% of lean body weight is in category I, and bone marrow fat (2% lean body weight) is in category III which also harbours all of the adipose tissue fat. The N_2 clearance half-times are 5 min (I) and 14 min (II) since the range of variation appears to be narrow for these largely aqueous categories. In III, the half-times are those derived from logarithmic analysis.

The principles underlying this analysis are applicable to techniques of N_2 measurement in which appreciable time (5 to 7 min) elapses for clearance of lung nitrogen, and in which estimates of body fat and the rate constants are derived from logarithmic analysis. Densitometric quantification of body fat provides a check on the logarithmic derivation as well as an accurate estimate of total

TABLE 21.5

Nitrogen solvents, content, and transport in three categories of tissues referrable to a young, lean, adult male

Entity	*Reference man*	*Tissue category*			
		I *Organs* *Viscera*	*II* *Muscle* *Skin* *Spinal cord* *Nerves*	*III* *Bone**	*Adipose tissue*
Weight (kg)	70				
Fluids (kg)	44·6	12	27·6	3	2
(%)	72	27	62	11	
Fat:					
Essential (kg)	2·0	0·4	0·4	1·2	—
Adipose tissue (kg)	8·0	—	—	—	8·0
Nitrogen (ml)					
Fluids	401	108	248	27	18
Fat:					
Essential	109	22	22	65	—
Adipose tissue	432	—	—	—	432
Total N_2	942	130	270	542	

Cumulative N_2 *transport (ml)*		*Category*		
		I	*II*	*III*
Time period (min)	*Total*			
6	169	73	70	26
30	454	128	208	118
60	597	130	257	210
90	679	130	267	282
510 (6 TU)	933	130	270	533
$T_{1/2}$ (min)		5	14	85
k		0·14	0·05	0·0082

* N_2 clearance of bone marrow not known; tentatively projected to have a half-time of 85 min.

TABLE 21.6

Partition of body N_2 into aqueous and lipid solvents and assignment of the aliquots to tissue categories I, II and III for four subjects in Table 21.3

Subject	*Lean body weight (kg)*	*Fat** (*kg*)			*Total body water (kg)*	*Category* N_2† (*ml*)						*Category* $T_{1/2}$ (*min*)		
		Adipose tissue	*Essential* *O*	*Essential* *B*		*I* *w*	*I* *f*	*II* *w*	*III* *w*	*III* *f*		*I*	*II*	*III*
1	58·2	20·8	0·58	1·16	41·9	102	31	234	42	1186		5	14	117
2	40·3	18·7	0·40	0·81	29·0	70	22	162	29	1054		5	14	144
3	57·2	22·8	0·57	1·14	41·2	100	31	230	41	1293		5	14	147
4	46·4	6·7	0·46	0·93	33·4	81	25	187	33	412		5	14	88

* Adipose tissue lipid and 'essential' organ (O) and marrow (B) fat.
† Category N_2 allocated to aqueous (w) and lipid (f) solvents.

N_2 content and subsequent allocation to fluid and fat. Determination only of end-tidal N_2 at small intervals greatly simplifies the technique and should provide the missing data pertaining to prolonged N_2 recovery.

CUTANEOUS DIFFUSION OF INERT GASES

Until the last decade practically no attention was focused on a factor which during the course of prolonged decompression from saturation dives may play an appreciable role. Earlier tests in man indicated that about 10% of the body's store of helium diffused through the skin per hour when oxygen was inhaled and the body immersed in helium. The technique employed at the time permitted measurement of inward (skin to lungs) or outward (lungs to skin) transport of helium. The data in Table 21.7 show a three- to four-fold increase in transport of helium at elevated ambient temperature (Fig. 21.9).

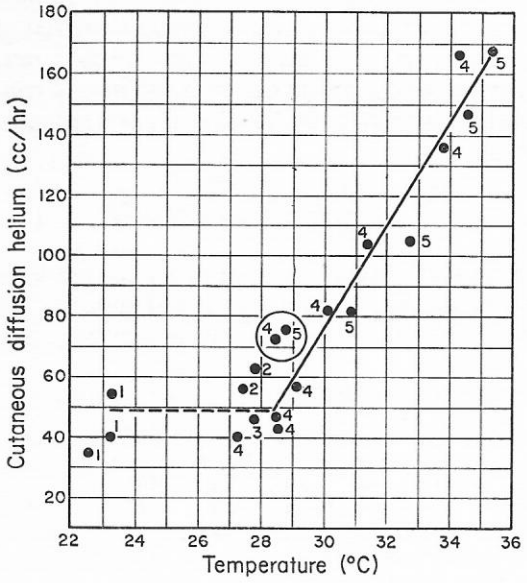


FIG. 21.9. Cutaneous diffusion of helium in relation to temperature, measured as ml (cc/hr) of helium recovered from the lungs per hour when the body (head out) is immersed in helium ($P_{He} = 700$ mm Hg). Numbers 1 to 5 refer to different subjects. The encircled values were obtained after the previously heated ambient helium had been cooled to 29°C (from Behnke & Willmon 1941)

With regard to N_2, cutaneous diffusion through the dog's tissues will replace the entire N_2 store every 24 hours (Groom & Farhi 1967). In a representative test the half-time of the slowest washout compartment of the dog surrounded by air was 332 min in contrast to 117 min with the dog immersed in oxygen. Open skin incisions greatly augment the diffusion of helium into tissues. During the course of N_2 clearance tests conducted at the Harvard School of Public Health (1932 to 1935), oxygen was inhaled by the anesthetized dog through a tracheal cannula. Not more than 4 hours (in later experiments) were required to reduce N_2 recovery from the lungs to 3 ml/hour. However, in earlier tests, about 10 ml of N_2 per hour were recovered in the reservoir spirometer for periods of at least 14 hours (Fig. 2.10). 'What appeared to be spontaneous generation of N_2 in the dog's body was found to be diffusion of N_2 from air into tissues through two incisions, one made for the tracheal cannula, and the other over

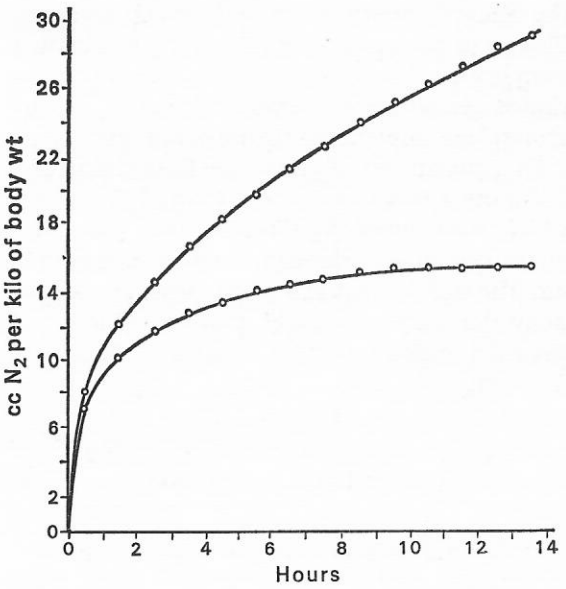


FIG. 21.10. The upper curve represents the elimination of N_2 via the lungs of anesthetized dogs with open incisions over the trachea and femoral artery of one leg. The lower curve represents the elimination of N_2 under the same conditions with the exception that the incisions were closed by suture. The area between the curves reflects the quantity of N_2 diffusing from ambient air into the dog's tissues through the incisions (data from Behnke & Shaw 1934)

TABLE 21.7
Cutaneous diffusion of helium in relation to skin temperature

	*Body in helium**		*Body in air†*	
Subject	*Skin T (°C)*	*Helium‡*	*Skin T (°C)*	*Helium‡*
1	37·7	104	37·2	61
2	37·8	57	36·9	54
3	38·2	107	36·9	109
4	37·2	93	37·1	100
Average		90		81
1	35·0	23		
2	34·6	16		
3	34·2	20		
4	33·8	27		
Average		21·5		

* Body in a gas-tight bag (head out connected to spirometer system).
† Body in a gas-tight bag (subject breathed an helium–oxygen mixture).
‡ ml of He/m²/hour, computed for $P_{He} = 760$ mm Hg. When body was in helium, helium was recovered from the lungs; when the body was in air, helium was recovered from the bag system.

the femoral artery to record blood pressure. Closure of the incisions rendered N_2 elimination complete in the expected 4 to 5 hours and concluded the attempt to measure the N_2 of the atmosphere through the tissues of the dog.'

In a human test, N_2 clearance from tissues of a 70-kg diver was reduced to a level of 47 ml/hour which represented N_2 diffusion into the spirometer system and through the skin immersed in air. The replacement of air by oxygen around the body skin surface brought about a reduction of inward diffusion from 47 to 24 ml/hour (Behnke & Willmon 1941).

a factor of three or more (diffusion and perfusion are complementary in this phenomenon). Diffusivity of helium in oil is 3 times greater than that of N_2, and more than 2·5 times greater in water. These values are well in excess of the general relationship postulated for diffusion of gases in tissue, namely,

$$\frac{\dot{V}_{He}}{\dot{V}_{N}} = \frac{\text{soln.}/\sqrt{\text{mol. wt.}}}{\text{soln.}/\sqrt{\text{mol. wt.}}} = 1{\cdot}73$$

Systematic investigation of cutaneous gas diffusion merits prosecution for several reasons. The perceptive and in-depth demonstration that

Nitrogen recovery from the lungs relative to ambient gas

Subject 1	*Air*			*Oxygen*				*Air*
Hours of test	7·5	8·5	9·5	11	12	13	14	15
N_2 recovery (ml)	53	52	47	46	33	34	24	48

Subject 2	*Oxygen*					*Air*	
Hours of test	9·5	11	12	13	14	15	16
N_2 recovery (ml)	55	49	47	46	41	60	67

These preliminary tests were terminated by military exigency but it appeared that in contrast with diffusion of helium, the quantity of N_2 diffusing percutaneously into the circulation was less by

bubble formation can occur in tissues by 'counter-diffusion' at fixed ambient pressure (Graves et al. 1975) serves to explain the intense itching and skin rash which supervened when a switch was

made to air at hyperbaric pressure and the body surface was immersed in helium. The practice of substitution of air for helium–oxygen mixture during decompression was routine at the Experimental Diving Unit during the 1938–1941 test period. Pruritus, macular skin lesions and delayed onset of 'diver's fatigue' were attributed to bubbles in subcutaneous vessels as a result of abridged decompression, and notably following chilling of the skin. Hypothermic circulatory stasis served to explain the adverse phenomena. Since the changeover from inhalation of helium–oxygen to air was gradual, it is not likely that the counter-diffusion phenomenon compounded the decompression problem. On the other hand, the importance of the phenomenon during decompression diving has been demonstrated under certain conditions.

Investigation of cutaneous diffusion is essential in assessment of the feasibility of head-out immersion in water to eliminate inward diffusion through the skin during decompression in helium atmospheres as well as for body temperature control. Immersion to the neck in water not only serves to circumvent inward percutaneous diffusion of helium and to regulate body temperature, but also to enhance inert gas transport (Balldin & Lundgren 1972). The final consideration is that the challenging problems outlined are amenable to ready solution by current refinements in gas analysis, in temperature control, in measurements of blood flow and tissue diffusion, and by fabrication of 'lung-body' collection systems impervious to gas diffusion and accurately calibrated as to volume.

INERT GAS CONTENT OF URINE

Leonard Hill (1912) analysed urine of divers to ascertain N_2 equilibration time with kidney tissue. Behnke and Yarbrough (1938–39) and subsequently, Van Der Aue, Brinton and Kellar (1945) analysed inert gas content of urine before and following dives in wet and dry chambers. The objective was to provide a simple and practical test presumptive of gas held in supersaturation or in bubbles circulating in blood. In recent years procedures directed to analyses of inert gas content of urine and blood, relative to alveolar concentrations of P_{N_2} have been refined in elegant tests of Klocke and Rahn (1961) and Farhi, Edwards and Homma (1963). The earlier unpublished studies did not lead to an unequivocal prognosis of dysbarism but the techniques with subsequent refinement appear to have wide application in current decompression procedure.

Underlying methodology is a comparison of urinary P_{N_2} (without loss of inert gas) with that of an aliquot of urine equilibrated with room air at 38°C. A condition of equilibrium through the media of arterial blood and kidneys is presumed to exist between gas tension in urine, lungs, venous blood and tissues at constant barometric pressure. There is temporary disequilibria when a diver is compressed or decompressed; re-establishment of gaseous equilibrium between P_{N_2} in urine and alveolar air presumably will be delayed by phase separation of gas in circulating blood.

In initial procedures, urine from divers who breathed a helium–oxygen mixture under pressure, and subsequently air during decompression, was collected before a dive (control sample), immediately after surfacing, and then at hourly intervals until normal gaseous equilibria prevailed. Large samples of urine were analysed by extraction of gases in the Van Slyke chamber and after chemical absorption of oxygen and carbon dioxide the inert residual was assessed for nitrogen, and helium, if present.

Analytical data

In 76 analyses, the N_2 content of bladder urine (P_{N_2}=570 mm Hg) averaged 0·015 vol. % higher than the gas content of urine equilibrated at 38°C by bubbling air slowly through the sample (P_{N_2}= 563 mm Hg). The greatest difference was 0·039 vol. %; in eight analyses urine N_2 was slightly lower (from 0·006 to 0·016 vol. %) than that of the equilibrated sample.

Inert gas content of equilibrated samples of urine was inversely proportional to specific gravity (i.e., 'salt' lowering effect on solubility) and a correction was made previously applied to N_2 solubility in body fluids.

N_2 content of urine (570 mm Hg, 38°C) in vol. % corrected for sp. gr. is:

$$0{\cdot}975-\frac{\text{sp. gr. urine}-1{\cdot}0}{0{\cdot}2}$$

TABLE 21.8

Results of analysis of inert gas (I) and carbon dioxide (free and combined) in the urine of an 'alkalinized' and an 'acidified' diver in dry chamber tests to 100 ft 4·03 ATA)*

Time	*Sample*	*Urine*‡ *(ml)*	*Sp. gr.*	*Sample (I) (vol.%)*	*Equilibrated (vol.%)*	*Excess (vol.%)*§	CO_2 *(vol.%)*
Diver 'alkalinized'†							
0800	control	405	1·022	0·901	0·862	0·039	57·5
0850	surface	30	1·023	1·865	(0·860)¶	1·005	110·0
0950	1 hour	50	1·025	1·251	(0·850)	0·401	104·0
1050	2 hour	80	1·016	0·971	0·893	0·078	105·0
1150	3 hour	180	1·006	0·979	0·960	0·019	94·4
Diver 'acidified'							
0715	control	75	1·019	0·916	0·914	0·002	4·1
0850	surface	130	1·015	1·810	(0·90)	0·910	6·0
0950	1 hour	165	1·016	1·220	(0·895)	0·325	2·8
1050	2 hour	80	1·014	0·996	0·916	0·080	3·5
1150	3 hour	245	1·005	0·986	0·952	0·034	3·8

* 2 min compression, 28 min on bottom, 2 min decompression.
† 'Alkalinized', a total of 60 g $NaHCO_3$ in divided doses preceding day and morning of dive; 'acidified' with ammonium mandelate on day preceding and morning of dive.
‡ Fluid intake: 250 ml water at 0800, 0900, 1000, and 1100.
§ Sample (I)—equilibrated aliquot with air at 1 ATA = Excess inert.
¶ In parentheses: calculated vol. % from sp. gr. (see text).

TABLE 21.9

Inert (I) gas content of urine in relation to forced fluid intake during the period of air compression in a dry chamber at 2 ATA, and during the post-decompression period

Time period (min)	*Fluid intake*	*Urine voided*		*Vol. % inert*
		(ml)	*sp. gr.*	
Pre-compression	—	55	1·022	0·865
Compression				
30	250	275	1·016	1·471
60	250	275	1·005	1·917
90	250	605	1·001	1·965
120	250	600	1·001	1·972
150	250	365	1·001	—
180	—	460	1·001	1·948
	1250	2580		
Post-decompression				
30	250	260	1·003	1·550
70	250	75	1·011	1·052
90	250	175	1·003	1·005
120	250	370	1·004	0·997
150	250	100	1·006	0·968
180	250	275	1·001	0·979
210	—	125	1·001	0·968
	1500	1380		

Thus for every 0·002 'units' of sp. gr., N_2 solubility is lowered by 0·001 vol. %. Pertinent is the finding that no substances, except ethanol, will give false positive values of inert gas content of urine. Van Der Aue, Brinton & Keller (1945) found in analysis of urine of divers with 'hangovers' that ethanol vapour rendered invalid assessment of inert gas. On the other hand, such measures as high degree of acidification or alkalinization of divers failed to influence residual urinary gas content (Table 21.8).

The N_2 content of urine was determined relative to time periods following compression to 15 psi gauge (2 ATA) and subsequently after rapid decompression to normal pressure (Table 21.9). Approximately 90 min were required both after compression and decompression to attain N_2 equilibrium in voided and equilibrated samples. During the compression period in this particular test, the quantity of urine excreted was strikingly in excess of the forced fluid intake.

In the initial tests (not confirmed by Van Der Aue, Brinton & Kellar 1945) the elevated N_2 content of urine voided during the second hour post dive, appeared to have prognostic import as to bends occurrence. Although higher N_2 values compared with equilibrated samples were frequently associated with subsequent onset of bends (Table 21.10), inhalation of oxygen during the decompression period tended to clear the

TABLE 21.10

Excess inert gas in urine in the second hour sample following long exposures in compressed air. Group A was decompressed in two min; Group B received extended air decompression; and Group C was given relatively short oxygen decompression at the 60 ft (2·82 ATA) level

Divers	*Depth (ft)*	*Duration (hrs)*	*Excess N_2* 2-hour sample (vol. %)*	*Decompression sickness*
Group A				
1	38	12	0·127	yes
2	38	12	0·124	yes
3	38	24	0·143	yes
4	38	9	0·061	yes
5	38	9	0·039	yes
6	30	12	0·047	no
7	30	12·5	0·010	no
8	30	12	−0·006	no
9	30	18	0·047	no
Group B				
1	60	6	0·020	no
2	90	6	0·038	no
3	90	6	0·005	no
4	90	6	0·022	yes (12-hour delay)
5	90	9	0·047	yes
Group C†				
1	60	6	0·000	no
2	60	12	0·012	yes
3	60	12	0·005	yes
4	60	12	0·002	yes
5	60	12	0·015	yes

* N_2 in 2-hour sample less N_2 in sample equilibrated with room air.
† Oxygen decompression at 60 ft level, then 5 min to 1 ATA followed by air inhalation 2 hours.

urine of N_2 and (although air was breathed at normal pressure for a 2-hour period) to mask any prognostic implications.

Rapid diffusion of helium

In one diver unusually resistant to bends, high values of urinary inert gas persisted through the second and third hours post dive. Helium was detected in these samples as well as in venous blood. This is the only instance in numerous helium–oxygen dives featured by air decompression in which helium could be detected in the 2-hour urine sample. Helium was usually present however, in the immediate post-dive (surface) sample.

During decompression when air was inhaled in place of the helium–oxygen mixture, there was reason to believe that helium diffused out of the bladder urine, notably if the bladder were distended. In a test designed to quantify helium loss from retained bladder urine, it was found that over a period of 3 hours, 1·63 vol. % helium dissolved in isotonic saline and introduced into the bladder, was reduced to 0·07 vol. %. Making allowance for dilution of helium by urine secreted into the bladder during the 3-hour period, it was computed that nine-tenths of the helium initially present had diffused through the bladder wall.

Abnormal urinary secretion during deeper helium dives

As much as a liter of urine was secreted during the course of wet tank helium dives (350 to 400 ft, 11·6 to 13·1 ATA) of 20 min bottom time followed by 220 min decompression. In 12 dives to 300 ft (10 ATA) of 20 min duration followed by 86 min decompression, the following urinary volumes were recorded.

Surface	*1 hour*	*2 hours*
610	114	182

The surface sample had been retained during the dive and decompression for 106 min; the average per hour during this period was 345 ml. Prior to the 1- and 2-hour samples, 250 ml of fluid was ingested.

Urinary volumes and inert gas content are recorded for a 500 ft (16·2 ATA) wet chamber dive (Table 21.11). Diver was in helmet and conventional diving dress, tightly laced around the lower extremities with snugly fitting lead belt around the waist. On bottom he breathed 85% helium, 15% oxygen for 12 min; during decompression there was a switch to air at 210 ft (7·4 ATA). The diuresis has a substantial explanation in the hydrostatic pressure gradient acting on the tightly laced lower extremities and body trunk distal to the weighted belt. The effect of this gradient is engorgement of intrathoracic circulation comparable to that in head-out immersion in water extensively investigated in connection with space flight. This redistribution of blood volume is similar to that of continuous negative pressure breathing. As in any procedure which causes engorgement of intrathoracic circulation, diuresis follows. The phenomenon has been interpreted as

TABLE 21.11

Volumes of urine excreted and inert (I) gas content prior to and following a 500 ft (16·2 ATA) wet chamber dive of 12 min bottom time followed by air decompression from 210 ft (7·36 ATA)

Time	*Sample*	*Urine* (ml)*	*Sp. gr.*	*Sample (I) (vol. %)*	*Equilibrated (vol. %)*	*Excess† (vol. %)*	CO_2‡ *(vol. %)*
0820	Control	40	1·019	0·890	0·898	−0·008	4·2
1310	Surface	1040	1·005	1·643	0·967	0·686	23·4
1410	1 hour	100	1·013	1·259	0·927	0·332	10·0
1510	2 hour	45	1·019	0·998	0·897	0·101	4·2
1600	3 hour	30	—	0·908	0·883	0·025	—

* Fluids: 500 ml night prior to dive, 375 ml morning of dive, and 375 ml immediately following dive, then 250 ml each hour thereafter.

† Excess inert gas = Sample (I) less equilibrated aliquot with air (1 ATA).

‡ Free (dissolved) and combined CO_2 liberated from urinary base.

an expression of volume regulation by stimulation of receptors in the intrathoracic circulation (Gauer, Henry & Behn 1970). Hormonal changes as well as suppression of plasma renin activity and aldosterone excretion are observed in water-to-the-neck immersion (Epstein, Duncan & Fishman 1972).

In 1945, at the instigation of Captain Van Der Aue, Dr. Gregory Pincus reported surprisingly low values of ketosteroids in urine from divers engaged in saturation dives at times which were attended by a high incidence of decompression sickness. The need for follow-up of these incomplete studies is apparent.

EMPIRICAL CONCEPTS RELEVANT TO CURRENT DECOMPRESSION PRACTICE

Asymptomatic ('silent') bubbles

The Doppler ultrasonic technique (Mackay & Rubissow 1971; Spencer, Johanson & Campbell 1975) in support of previous observations and surmise, detects bubbles in the circulation which do not give rise to decompression sickness. In altitude chamber tests at the Experimental Diving Unit (1939 to 1940) oxygen inhalation above 20 000 ft (0·46 ATA) afforded less protection against dysbarism than pre-oxygenation at 1 ATA. A debilitating fatigue, often delayed until return to ground level, frequently supervened during several hours' exposure at 20 000 to 25 000 ft (0·46 to 0·37 ATA) in the absence of pre-oxygenation at normal pressure (1 ATA). This altitude 'gray' zone was designated as one productive of 'silent' bubbles (Behnke 1942). The concept may be considered with reference to events depicted in Fig. 21.11 which shows that out of 39 divers in an older age group, only one was able to remain at 38 500 ft (0·2 ATA) for 4 hours in the absence of pre-oxygenation (1 ATA). It is inferred that those divers who developed decompression sickness after varying intervals at altitude, had circulating bubbles prior to onset of symptoms. All of the divers, except one who had 4 hours of pre-oxygenation (1 ATA), were able to remain symptom-free at altitude for at least 4 hours, and several subjects

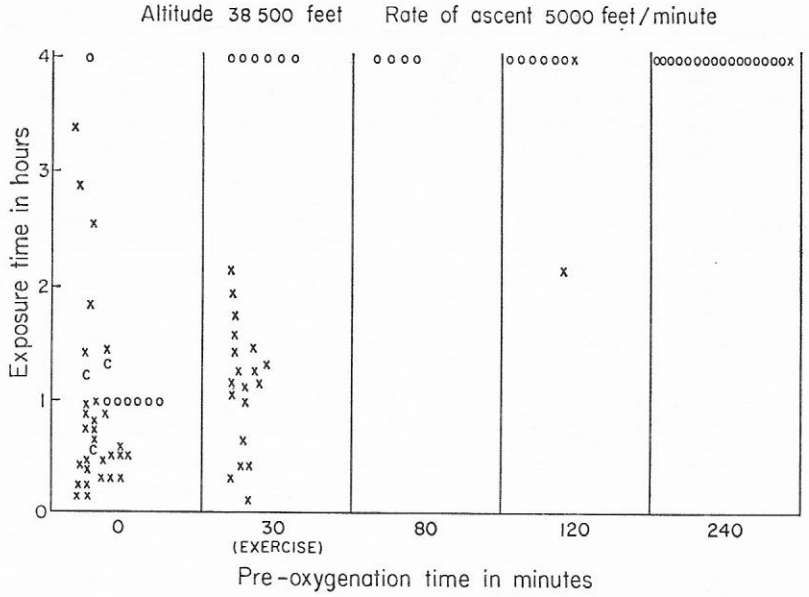


FIG. 21.11. The relationship between pre-oxygenation time (1 ATA) and duration of stay at simulated altitude of 38 500 ft (0·2 ATA) for periods up to 4 hours. Incidence of bends (×) of chokes (c) and of tolerance (O) relate closely to degree of N_2 clearance prior to ascent. One diver with an old injury (leg) was susceptible to bends despite 4 hours of pre-oxygenation

(not divers) were in good condition for at least 1·5 hours at 48 000 to 52 000 ft (0·13 to 0·10 ATA).

In submarine escape training during the course of successive exposures to 100 ft (4 ATA) followed by 30 sec ascent to the surface, the first ascent may be symptom-free, the second accompanied by delayed fatigue, while the third ascent may be followed by frank bends and other involvement. In test dives presumptive evidence of graded separation of gas from solution is afforded by the systematic work dives followed by limited oxygen 'surface decompression' conducted by Van Der Aue et al. (1951). The gradation of reactions varies from no symptoms, to fatigue, itch, fatigue and itch, and mild decompression sickness (Table 21.12). These responses followed the systematic shortening of oxygen recompression time (i.e. following the short surface interval) of divers who engaged in heavy work at depth. A special contribution of this solid investigation is the greater susceptibility to bends brought about by a heavy work load and presumably related to the excess CO_2 which augmented latent bubble size. The results are similar to the thoroughly established

TABLE 21.12

Reactions attending the systematic shortening of oxygen recompression time of divers who engaged in heavy work at a depth of 100 ft (4 ATA) and were then bought rapidly to the surface*

Number of dives	*Time at depth (min)*	*Water stops (min)*	*Recomp. oxygen 2·21 ATA*	*Reactions*				
				None	*Fatigue*	*Itch*	*F & I†*	*Bends*
4	26	0	0	4	—	—	—	—
4	28	0	0	1	3	—	—	—
4	30	0	0	2	2	—	—	—
4	40	0	0	1	1	1	1	—
4	50	0	10	4	—	—	—	—
8	60	0	33	5	3	—	—	—
4	60	0	26	—	2	—	2	—
4	60	0	20	1	2	1	—	—
4	60	0	15	4	—	—	—	—
4	60	0	9	1	—	2	1	—
4	60	0	7	2	1	—	1	—
4	60	0	5	1	—	1	—	2
8	75	0	19	6	1	1	—	—
4	75	0	16	3	—	—	—	1
4	75	0	13	2	—	1	—	1
4	75	0	11	1	—	—	—	3
8	85	0	51	8	—	—	—	—
4	85	0	25	3	—	1	—	—
4	85	0	19	2	1	—	—	1
4	85	0	16	1	—	—	—	3
4	100	0	35	4	—	—	—	—
4	120	0	47	3	—	—	—	1
4	120	3 (at 30 ft)	50	3	1	—	—	—
4	120	3 (at 30 ft)	41	1	—	—	—	3

* Rate of ascent, 25 ft/min; at surface for 3·5 min; time elapsed between bottom depth and chamber recompression was 10 min.

† F & I (fatigue and itch). Note: Bends were usually mild and did not require recompression.

Data from Van Der Aue, Brinton and Kellar (1945) and Van Der Aue et al. (1951).

increase in decompression sickness incident to exercise at altitude. Fit divers with a lower respiratory quotient ($RQ = 1{\cdot}2$) would be less susceptible than unfit divers with a work RQ of 1·4 or higher. A rest period following heavy work should be enforced prior to decompression to permit elimination of excess CO_2 which otherwise complicates the transport of inert gas.

During the latent period prior to onset of decompression sickness in man, bubbles in the circulation can now be identified by the Doppler technique. In the rapidly decompressed dog, these presymptomatic bubbles are observed in rapid circulatory transit. More amenable to confirmation is the observation that during the oxygen recompression period (to +30 psi gauge; 3 ATA) visible bubbles in blood vessels vanish and cardinal signs of decompression sickness are reversed (tachypnea, bradycardia, rise in pulmonic and fall in peripheral arterial pressures), only to reappear when pressure is prematurely reduced to 1 ATA (Behnke et al. 1936).

With reference again to Van Der Aue's tests, surface decompression was attended by a high incidence of fatigue and other mild impediments which usually did not require treatment. The remarkable ability of aqueous tissues to contain, at least initially, high pressures of N_2 without symptoms during the early stages of decompression, is probably responsible for the long-standing error of decompressing divers and caisson workers too rapidly in the early stages of decompression. The venerable postulate that bubbles form as soon as a condition of supersaturation is initiated supported the conclusion that supersaturation ratios were probably indices of permissible embolization. It may well be that the separation of gas from solution and consequent retardation of tissue perfusion may explain the empiricism underlying the prolonged desaturation time of 'the slowest tissue' postulated both for air and helium–oxygen dives. In effect decompression tables for subsaturation dives are treatment tables. Yet there is no proof that, in the absence of symptoms, phase separation of gas (silent bubbles) impairs normal circulation and thus nitrogen transport in tissues.

Decompression experience in earlier chamber tests

Decompressions according to Haldane's stage method following extended exposures in compressed air of 3 to 4 hours' duration (45 psi gauge, 4 ATA) were frequently complicated by bends at the Harvard School of Public Health during the period 1932 to 1935. In 1934, a bend was recorded by an engineer at a level of 3 psi gauge during the course of stage decompression following an exposure of 99 hours at 30 psi gauge (3 ATA). The initial drop in pressure, according to a conservative schedule at the time, was from 30 to 15 psi gauge and some 7 hours were taken to arrive at the 3 psi gauge level. This unfavorable experience stimulated physiologic investigation at Harvard and served to emphasize the need for decompression studies following saturation exposures. Such tests were conducted at the Experimental Diving Unit during the period 1940 to 1945. These tests in compressed air served to establish a depth (dry) of 33 ft (2 ATA) as limiting for a saturation exposure (12 hours) followed by no-decompression stoppages. Exposures of longer duration were only occasionally complicated by tolerated bends which did not require recompression. Of special interest was the role apparently of the small quantity of N_2 (which could not be measured at the time) absorbed after 6 hours relative to occurrence of bends. At depths deeper than 33 ft (2 ATA) the attempt was made to decompress on oxygen at a single level (60 ft; 2·82 ATA) which would assure isobaric transport of N_2 at an optimal level (Table 21.13). Despite this precaution, bends occurred frequently and it was clearly evident that the time allocated for oxygen inhalation (calculated to permit a surfacing ratio of N_2 of about 1·6 to 1) was inadequate.

The isobaric (oxygen window) principle of decompression

In any gas mixture there is a certain percentage of oxygen compatible with well being. During the course of blood perfusion of tissues, oxygen is unloaded in different quantities to the various tissues. The result of this transfer of oxygen is that the blood can transport an equivalent amount of inert gas from tissues to lungs. During the late Thirties, Momsen (1939) and his group at the EDU referred to the 'space' available for transport of inert gas in solution as the 'partial pressure vacancy'.

TABLE 21.13

Saturation air dives in the dry or wet tank

No. tests	*ATA*	*ft*	*Exposure time (hours)*	*Decompression*		*Outcome**
				Depth stop (ft)	*Time (min)*	
4 Resting	2·00	33	12	—	—	NS
4 Work	2·00	33	12	—	—	NS
4 Resting	2·00	33	24	—	—	NS
8 Resting	2·00	33	36			2(X) 6 NS
4 Resting	2·06	35	12	—	—	1(O) 3 NS
4 Work	2·06	35	12	—	—	NS
14 Resting	2·21	40	12	—	—	4(O) 9 NS
14 Work	2·21	40	12	—	—	5(O) 4 NS 5(X)
1 Resting	2·82	60	12	60 oxygen	63	(X)
1 Resting	2·82	60	12	60 oxygen	69	(X)
1 Resting	2·82	60	12	60 oxygen	80	(X)
1 Resting	2·82	60	12	60 oxygen	92	(X)
1 Resting	3·82	90	6	40 oxygen	111	NS
1 Resting	3·82	90	6	40 oxygen	111	(X)
4 Resting	4·00	99	6	33 air	12 hours	1(X) 3(NS)
2 Resting	4·00	99	9	33 air	12 hours	1(X) 1(O)
2 Resting	4·00	99	9	33 air	18 hours	NS
2 Resting	4·00	99	12	33 air	24 hours	1(X) 1(O)

* NS = No symptoms; (X) = Bends; (O) = Mild bends, no recompression.
Data from Behnke (1940) and Van Der Aue (1945).

At normal pressure during inhalation of air, the partial pressure of oxygen in the arterial blood falls to about 40 mm Hg on the average in capillaries. If Pa_{O2} is elevated to 287 mm Hg, there is a subsequent decrease in the capillaries to about 50 mm Hg, only 10 mm higher than the previous level. Noteworthy is the possibility of an increase of Pa_{O2} to about 1500 mm Hg without incurring a rise of more than several hundred mm of oxygen pressure in the capillaries. Since oxygen becomes abruptly toxic above the level of 2 ATA for work or sustained inhalation, there is a physiologic limit to the size of the *oxygen window*.

In non-saturation diving the isobaric principle is compromised between the ideal 'bubble-free' decompression and a relatively abridged schedule based on empiricism and dictated by expediency. This limitation did not preclude helium–oxygen dives in the open sea to depths greater than 500 ft (16·2 ATA) in the earlier era but the exposures were short and generally limited to accomplishment of a single task, previously well rehearsed on the surface. With standard oxygen decompression at essentially two stops, 50 and 40 ft (2·25 and 2·21 ATA), as it was during the salvage operations to raise the USS *Squalus*, then out of hundreds of dives there were only occasional cases of decompression sickness, and minimal, if any, residual disability.

Current calculations pertaining to decompression following helium–oxygen saturation dives postulate a half-time for the slowest desaturating tissue of at least 240 min, which is greatly in excess of measurements, admittedly incomplete, made in a previous era and of deductions referrable to helium solubility in fluid and fat. Thus if

π is the pressure of helium in the 'slowest' tissue ($T_{1/2}$ = 240 min), ΔP permissible supersaturation in ft of seawater, and Po_2 the *oxygen window*, then the rate of clearance of the slowest tissue is

$$\frac{d\pi}{dt} = -\frac{0{\cdot}693}{240}\, T_{1/2} \cdot (20+10) = \frac{11{\cdot}6 \text{ min}}{1 \text{ ft}}$$

If, however, ΔP is non-existent or a mathematical strategem, and if we consider only oxygen exchange (i.e. 10 ft equiv.), half-time for the slowest tissue becomes 80 min. To complete the analysis of isobaric transport, consider,

	Gas tensions LUNGS	*Gas tensions* TISSUES-CAPILLARIES
Helium	2000	2230
Oxygen	287	50
CO_2	40	47
H_2O vapor	47	47
	2374 mm	2374 mm

Isobaric decompression can now progress uniformly at a rate of 1 ft every 11·5 min and concomitantly there is an isobaric tension decrease of helium of 230 mm Hg during the course of each foot of ascent.

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