DECOMPRESSION TABLES IN RELATION TO DYSBARIC OSTEONECROSIS

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INTRODUCTION

The possibility that individuals exposed to hyperbaric conditions may develop radiographic evidence of bone lesions after a latent period of several weeks or months is well recognized (McCallum *et al.*, 1966). The exact pathophysiology of dysbarism-induced osteonecrosis is not clear. Hills (this volume) has suggested the possibility that osmotic stresses generated during compression might be a contributing factor. The implications of this and other compression and isobaric phenomena of gas exchange cannot be discounted. But the present paper will be confined to the stresses of decompression and the possibility that dysbaric osteonecrosis is a form of decompression sickness, with delayed manifestations initiated by decompression procedures which were, in some way, inadequate.

The possibility that fatty bone marrow might exchange gas very slowly and thus gradually become dangerously supersaturated after several successive dives will be examined through use of a simple exponential gas-exchange model. Some limitations in this approach and certain implications of the findings will be discussed. So also will be certain theoretical and practical approaches to avoiding supersaturation and consequent gas formation in amounts potentially damaging to bone during decompression.

BACKGROUND

Many theoretical models have been proposed to describe the transport of inert gases within the body, the number of critical tissues that should be considered, and the constraints necessary to prevent decompression sickness. Several of these models have been used to develop decompression tables for animals and man, a few of which have proven reasonably safe for sport, commercial, military, and scientific diving purposes. Several investigators have compared decompression tables and their inherent assumptions, implications, and limitations (Hempleman, 1960; Schreiner and Kelley, 1966; Schreiner, 1967; Hills, 1970; Feld, 1971; Kidd *et al.*, 1971).

Many decompression tables in current use have evolved from concepts developed by Haldane and associates (Boycott *et al.*, 1908). Haldane treated the body as a group of discrete parallel mathematical compartments or half-time tissues that exchange inert gas in solution with the blood, which was assumed to be at equilibrium with the inspired gases. He also assumed that rates of inert-gas exchange in tissue compartments are functions of inert-gas solubility in various bodily tissues and of the blood perfusion rates in those tissues. He further assumed that diffusion of a gas in bodily tissues is very rapid, so that blood-perfusion rates limit tissue gas exchange. Haldane also predicted that when the partial pressure of gas in an individual tissue compartment, divided by the ambient pressure, exceeds a critical ratio (2:1), symptoms of decompression sickness might result. Many of these assumptions have since been challenged.

With the accumulation of experimental data in the intervening years, several modifications of Haldane's concepts have evolved. Tissues with longer half-times than Haldane considered were added and different critical ratios applied to them (Workman, 1965; Bühlmann, 1971; Schreiner and Kelley, 1971). Harvey (1951 *a* and *b*) and Behnke (1951) suggested that safe ratios for various tissues are also a function of depth.

Workman (1965) developed a more flexible approach, postulating for each tissue compartment a maximum allowable inert-gas tissue tension (called *M-value*) that would permit safe incremental changes to a lower ambient pressure. A matrix of M-values was developed and refined as experiments suggested that assumed gas tensions in particular tissues were, in fact, excessive and liable to cause symptoms of decompression sickness. Unfortunately, present-day knowledge of gas-exchange rates in bone and insufficient data on the incidence of osteonecrosis do not clearly identify those exposure profiles and gas tensions productive of that disease. Meaningful modification of Workman's (or any other) mathematical model, and the decompression tables based upon it, are therefore difficult.

However, certain clues are available. McCal-

lum *et al.* (1966) found that bone lesions in caisson workers are related directly to the number of times a man has been decompressed, to the pressure at which he has worked, and to symptoms of dysbarism for which treatment was given. Nellen and Kindwall (1972) reported that men decompressed twice daily during split-shift work have a higher incidence of osteonecrosis than workers decompressed once daily from a single, longer shift. These findings suggest inadequate decompression as the cause of bone lesions.

Lesions are typically found in the proximal humerus and in the proximal and distal ends of the femur (McCallum *et al.*, 1966), which have "yellow" marrow containing large amounts of fat. Because fatty marrow is assumed to be a "slow" tissue (Jones, 1951), decompression would cause a great supersaturation of dissolved nitrogen, which may persist for long periods of time. Further work is needed to reexamine and extend the work of Campbell and Hill (1933 *a* and *b*), who attempted to measure the rates of gas exchange in fatty marrow and other tissues. If fatty marrow is in fact a slow tissue, then one must establish the *slowest* tissues to be considered in calculating decompression tables.

Workman (1965) used a tissue half-time ($T_{1/2}$) of 240 minutes for the slowest tissue in computing the U.S. Navy Standard Air Decompression Tables. Schreiner and Kelley (1971) summarized several pieces of evidence justifying 400 to 500 minutes as the longest half-time compartment for N_2 exchange in the body. Bühlmann (1971) suggested 480 minutes for N_2 exchange. He used 640 minutes in calculating long decompressions after deep dives to compensate for sleeping and other variables that affect human physiology.

Thus if fatty marrow is considered a slow body tissue, and if damage can occur in marrow without the appearance of symptoms elsewhere, it seems necessary to consider gas accumulations in tissues with longer half-saturation times in recalculating standard air decompression profiles. A $T_{1/2}$ of 720 minutes was accordingly chosen by the Institute for Environmental Medicine, University of Pennsylvania — a figure exceeding the longest half-times suggested by earlier workers. Although arbitrarily chosen, the decompression tables were representative of those employed in diving and caisson work in recent years.

Accordingly, several representative tables were subjected to analysis by use of the PADUA* computer program. The program is based on a simple exponential gas-exchange formula (such as those frequently used in a perfusion-limited model of gas exchange).

For gas exchange during periods not involving a change in ambient pressure:

$$\pi_f = \pi_o + (PP_I - \pi_o) \cdot (1 - 2^{-\Delta t/H})$$

For gas exchange during periods involving a linear change in ambient pressure:

$$\pi_f = \pi_o + [(PP_I - mV\Delta t) - \pi_o] \cdot (1 - 2^{-\Delta t/H})$$

Symbols:

π_f = final tissue tension of inert gas
π_o = initial tissue tension of inert gas
PP_I = implied partial pressure
Δt = change in time
H = tissue half-time
m = mole fraction of inspired inert gas
V = velocity of ascent or descent

*Pennsylvania Analysis of Decompression for Undersea and Aerospace

PROCEDURE AND RESULTS

Two sets of M-values, those of Workman and PADUA, were used to calculate tissue tensions at the start of a "dive" and at "surfacing" (see Table I). M-values refer to maximum allowable tissue tensions of inert gas in units of feet of seawater absolute (FSWA). The slightly more conservative PADUA M-values are those employed recently at the Institute in calculating air decompression tables. PADUA M-values represent allowable tissue tensions for a subject's ascent from a final 10-feet-of-seawater (FSW) stop to surface in 10 seconds. Any calculated tissue tension higher than the corresponding M-value indicates that tissue supersaturation would be such that the subject could not surface without significant risk of developing dysbarism.

Table I. COMPARISON OF WORKMAN AND PADUA* M-VALUES ON SURFACING (IN FSWA)

Tissue half-times (min)	5	10	20	40	80	120	160	240	320	480	560	720
Workman	104	88	72	56	54	52	51	50				
PADUA	100	84	68	53	52	51	50	49	49	48	48	48

*PADUA — Pennsylvania Analysis of Decompression for Undersea and Aerospace computer program

The depths and times chosen permitted evaluation of the deeper and longer dives allowed by several commonly used tables (see Table II). (*N.B.*: Regarding Tables II and IV, the "surfacing" nitrogen tensions that exceed the allowable PADUA M-values for several tissues with different half-times — the unsafe supersaturation values — are enclosed in rectangles.) The Blackpool tables (1968) permit a 4-hr working time at 66 FSW, whereas the New York tables (1960) allow two 2-hr exposures to achieve the same 4-hr working time. With the Blackpool tables, 4-hr exposures produced maximum tissue tensions in the 120- and 160-min compartments. There were no M-value violations in tissue tensions upon surfacing in any compartment, even after 7 consecutive days of exposure. The New York table exposures (two 2-hr shifts) produced maximum tissue tensions in the 40- and 80-min tissues. They did not exceed the Workman M-values, although they did slightly exceed the PADUA M-values in the 40-, 80-, and 120-min tissues. The longer theoretical compartments again did not accumulate enough gas to exceed the surfacing M-values even after 7 consecutive days of exposure.

Exposures of 8 hr at 100 FSW were evaluated according to the British tables (1951) and Blackpool tables (1968). On the 1951 tables, maximum tissue tensions were reached on Day 1 in the 160-, 240-, and 320-min tissues, which exceeded M-values in all tissues having a $T_{1/2}$ of 80 min (or longer). Tissues with a $T_{1/2}$ longer than 320 min showed significantly higher gas tensions after Day 7 in comparison with Day 1. On the Blackpool tables the same series of daily 8-hr exposures produced maximum tensions in tissues with a $T_{1/2}$ of 320 min or longer, which slightly exceeded allowable M-values.

On the U.S. Navy tables (1963), 30-min exposures to 180 FSW twice daily for 7 days, with a surface interval of 277 min, produced maximum tissue tensions in the 40- to 120-min tissues. There were no M-value violations.

Under the U.S. Navy Standard Air Decompression Tables (1963) a second dive, following

Table II. COMPARISON OF N_2 TISSUE TENSIONS IN 12 THEORETICAL COMPARTMENTS RESULTING FROM SEVEN-DAY REPETITIVE PRESSURE EXPOSURES FOLLOWED BY DECOMPRESSION ACCORDING TO FIVE DIFFERENT TABLES

Tissue half-times (min)			5	10	20	40	80	120	160	240	320	480	560	720
			Tissue tensions of inert gas (FSWA)											
New York Tables	Day 1	Start	26	26	26	26	26	26	26	26	26	26	26	26
(1960)		Surfacing	30	35	46	[54	54	52]	50	46	43	39	38	36
66 ft, 2 hr	Day 7	Start	26	26	26	26	26	26	26	27	28	30	31	31
twice daily		Surfacing	30	35	46	[54	54	52]	50	46	40	41	40	39
British Tables	Day 1	Start	26	26	26	26	26	26	26	26	26	26	26	26
(1951)		Surfacing	28	29	33	43	[59	68	71	71	68	61	58	53]
100 ft, 8 hr	Day 7	Start	26	26	26	26	26	26	27	30	33	38	40	42
		Surfacing	28	29	33	43	[59	68	71	72	70	66	65	63]
Blackpool Tables	Day 1	Start	26	26	26	26	26	26	26	26	26	26	26	26
(1968)		Surfacing	26	26	26	27	31	37	42	[49	51	52	51	49]
100 ft, 8 hr	Day 7	Start	26	26	26	26	26	27	28	31	34	39	41	44
		Surfacing	26	26	26	27	31	37	42	[49	53	55	56	56]
Blackpool Tables	Day 1	Start	26	26	26	26	26	26	26	26	26	26	26	26
(1968)		Surfacing	27	29	31	38	47	50	50	47	44	40	39	37
66 ft, 4 hr	Day 7	Start	26	26	26	26	26	26	26	27	28	29	30	31
		Surfacing	27	29	31	38	47	50	50	47	45	42	41	40
U.S. Navy	Day 1	Start	26	26	26	26	26	26	26	26	26	26	26	26
Tables (1963)		Surfacing	34	34	39	47	49	47	45	42	40	37	36	34
180 ft, 30 min	Day 7	Start	26	26	26	26	26	26	26	27	28	29	30	30
twice daily		Surfacing	34	34	39	47	49	47	45	43	41	39	38	37

Table III. NITROGEN TISSUE TENSIONS (FSWA) AT START OF AND SURFACING FROM FOUR CONSECUTIVE 30-MIN SIMULATED DIVES TO 180 FSW, ACCORDING TO U.S. NAVY TABLES (1963)*

Tissue half-times (min)	5	10	20	40	80	120	160	240	320	480	560	720
	Tissue tensions of inert gas (FSWA)											
Dive 1, Start	26	26	26	26	26	26	26	26	26	26	26	26
Dive 1, Surface	34	34	35	40	49	50	50	46	44	39	38	36
Dive 2, Start	26	26	26	26	26	26	27	29	30	31	31	31
Dive 2, Surface	34	34	35	40	49	51	50	48	46	43	41	40
Dive 3, Start	26	26	26	26	26	26	27	29	30	32	32	33
Dive 3, Surface	34	34	35	40	49	51	50	48	46	43	43	41
Dive 4, Start	26	26	26	26	26	26	27	29	30	32	33	34
Dive 4, Surface	34	34	35	40	49	51	50	48	46	44	43	42
Dive 5, Start	26	26	26	26	26	26	27	29	30	32	33	34

*All "dives" separated by 12-hr "surface" interval

a surface interval longer than 12 hr, is not subject to the rules of repetitive diving. Table III sets out the tissue tensions at the start and finish of each of 4 consecutive 30-min dives to 180 FSW separated by 12-hr surface intervals.

The same series of dives was made to 190 FSW for 60 min, the longest and deepest dive permitted by the U.S. Navy decompression tables (see Table IV). Maximum tensions developed in the 40-min tissues on the 190-FSW exposures and exceeded the PADUA M-value for 40-min tissues on each dive while approximately equaling the Workman M-value. Maximum tension developed in the 120-min tissue on the 190-FSW dives, but no M-values were exceeded after 4 dives.

Tables II, III, and IV indicate that theoretical gas tensions accumulated in longer half-time tissue compartments may approach or exceed the supersaturation considered permissible with the M-values adopted. In general, however, analysis suggests that these tables — with the exception of the now discarded 1951 British tables, which were included merely for com-

Table IV. NITROGEN TISSUE TENSIONS (FSWA) AT START OF AND SURFACING FROM FOUR CONSECUTIVE 60-MIN SIMULATED DIVES TO 190 FSW, ACCORDING TO U.S. NAVY TABLES (1963)*

Tissue half-times (min)	5	10	20	40	80	120	160	240	320	480	560	720
	Tissue tensions of inert gas (FSWA)											
Dive 1, Start	26	26	26	26	26	26	26	26	26	26	26	26
Dive 1, Surface	34	38	50	[57]	52	47	43	38	36	33	32	31
Dive 2, Start	26	26	26	26	26	26	27	28	28	28	28	28
Dive 2, Surface	34	38	50	[57]	52	47	43	40	37	35	34	33
Dive 3, Start	26	26	26	26	26	26	27	28	28	29	29	29
Dive 3, Surface	34	38	50	[57]	52	47	43	40	38	36	35	34
Dive 4, Start	26	26	26	26	26	26	27	28	29	29	30	30
Dive 4, Surface	34	38	50	[57]	52	47	43	40	38	36	35	34
Dive 5, Start	26	26	26	26	26	26	27	28	29	30	30	30

*All "dives" separated by 12-hr "surface" interval

parison — are reasonably safe, even when used on a daily basis. The qualification is that there must be a minimum surface interval of 12 hr between dives; otherwise they are considered repetitive.

DISCUSSION

The implication of the foregoing calculations may be misleading. Supersaturation in bone marrow may produce damage at gas-tension levels below those causing symptoms of dysbarism elsewhere in the body. Thus M-values for the longer half-time tissues, in particular, may need revision downward.

Hempleman (1960) demonstrated that the rates of N_2 uptake and elimination are unequal. He suggested that "silent" bubbles might form in tissues during decompression, altering the kinetics of gas exchange. The modified Haldane approach is based on the assumption that the rate of gas uptake varies exponentially with time and that several different tissues are involved. Hempleman theorized that the uptake curve is a complex equation represented by a sum of exponentials, and that only one type of tissue is involved. (This model assumes that gas diffusion, rather than tissue perfusion, is the rate-limiting factor for gas exchange in the body.)

Neither the diffusion nor perfusion theory allows for Hempleman's observation that a "discontinuity in the body physics takes place when large and rapid drops in pressure occur." He therefore suggests that separate theories are necessary for inert-gas uptake and elimination. Bornmann (1970) and Summitt *et al.* (1970) approached the problem of repetitive excursion dives from saturated depths on $He–O_2$ mixtures by calculating uptake according to a 200-min $T_{1/2}$ during excursions and gas elimination according to a 240-min $T_{1/2}$ during the surface interval. Bühlmann (1969) used a 480-min $T_{1/2}$ for N_2 uptake and elimination, but altered the figure to 640 min during sleep.

This inequality of uptake and elimination raises a serious doubt about the validity of calculations based on a conventional exponential equation, as was used to produce the values shown in Tables II, III, and IV. This error could be compounded by repetitive dives.

Kidd and Stubbs (1969) have pointed out that the pneumatic analogue computer offers efficient decompression protocol from random profile dives and repetitive exposures. They have commented, as well, that the computer seems to have an inherent asymmetry similar to that operating in man during gas uptake and elimination.

There is a body of evidence suggesting that "silent" bubbles and gas separation occur in routine asymptomatic decompression (Behnke, 1967). Mackay and Rubissow (1971), employing pulsed ultrasonic energy and the Doppler shift principles, have discussed techniques for studying the occurrence of bubbles and masses of gas within the body. Kidd and Stubbs (1969) noted a small but significant (0.5 dB) ultrasonic signal change during ascent when inert-gas tension, computed by their MkVS pneumatic analogue computer, approximated ambient pressure. A strong signal change (7 dB) was always observed prior to the occurrence of obvious bends symptoms. However, they also noted many instances of 3- to 5-dB signal changes when no symptoms were experienced.

The significance of these silent bubbles is not clear, but repeated showers of bubbles or emboli may be more important in the development of osteonecrosis than a single insult. Stegall and Smith (1972) used repeated exposures to develop lesions in miniature swine. Repetitive dive schedules may therefore need more conservative criteria in their calculations than schedules designed for less frequent use. Further refinement of ultrasonic techniques may make it possible to tailor each decompression protocol to the individual diver by monitoring him constantly.

Behnke (1967) has discussed the principles of isobaric ("oxygen window") decompression, a technique that theoretically avoids bubble formation but "calls for too long a decompression for dives up to one-hour duration, unless it is feasible to breathe pure oxygen for a prolonged period at the +15 psi pressure level." He points out that lengthy half-times, such as those analyzed in Tables II, III, and IV herein, seem much too long to be consistent with the limited physiological data available on N_2 elimination in man. They have been employed, he suggests, only because models used to calculate tables may have questionable physiologic bases. Behnke adds: "It is likely that decompression practice in the past has served to initiate bubble evolution which subsequently has been controlled by prolonged stage decompression at relatively shallow depths."

If these silent bubbles do, in fact, precipitate dysbaric osteonecrosis, tables must be calculated that rely less on ratio principles and, instead, adhere more closely to the O_2-window principle of decompression — even at the cost of longer

stops at the early, deep stages of decompression. No valid data exist on the circulatory changes induced in bone by high-pressure O_2 or on the sensitivity of fatty bone marrow to O_2 toxicity. Such factors might influence the use of high Po_2 to speed inert-gas elimination during decompression.

Hills (1966; 1968; 1970) has suggested that a small amount of gas separation can occur in many tissues without causing symptoms, and that the process is random. His analysis (1970) summarizes several empirical approaches used in the perfusion and diffusion models of inert-gas exchange to allow for the inequality in gas exchange.

Hills also defines an important basic issue in formulating decompression tables. Gas in physical solution is lost from a supersaturated tissue by a conventional exponential format, whereas a very different driving force eliminates gas separated from solution. Conventional supersaturation models keep divers at as shallow a depth as is consistent with their permissible supersaturation; by contrast, Hills's "phase equilibration" model suggests that much deeper staging is needed. The successful use of surface decompression is also explained by the concept of phase equilibration, whereas the concept of acceptable supersaturation makes it hard to explain why there is not more difficulty with bubble formation during the surface interval. Gas formation in the rigid confines of a bone has a more devastating effect than in many other tissues. Unfortunately, the degree to which gas separates from solution in bone marrow during commonly used decompression procedures is unknown.

Harvey *et al.* (1944) and Aggazzotti and Ligabue (1942) suggested the importance of preexisting gas nuclei in gas-bubble formation. Walder (1969) has suggested that reducing the number of these nuclei by repeated decompressions might explain the observation that divers are more resistant to dysbarism when they have been diving regularly (*e.g.*, daily). Hills (1970) has offered an alternative explanation — namely, that successive formation and elimination of a stable-gas phase could cause a fall in the "elastic modulus" of a critical tissue and, hence, in the pressure differential between gas and tissue, tending to distort a nerve ending beyond its pain-provoking threshold.

Walder's theory would permit less conservative decompression procedures during the later dives of a series than in earlier ones. Hills's theory, on the other hand, suggests following the same protocol in every dive, since rigid bone structures might be less capable of change in elastic modulus than many other tissues would. Repeated insults might therefore produce permanent damage to bone.

Use of a gas (or a combination of gases) other than N_2 for hyperbaric work would modify the concepts discussed in this paper. Modification would depend on many physical characteristics — *e.g.*, partition coefficient, fat solubility, oil–water solubility ratios, molecular weight, and molecular size of the specific gas. The exact risk of developing dysbaric osteonecrosis in helium, neon, or hydrogen exposures on existing tables following dives deeper than 200 FSW is not known. Until further data are accumulated, the risk cannot be predicted accurately.

The effect of mixing or switching inert gases for the purpose of shortening total decompression time (Bühlmann *et al.*, 1967) as a causative mechanism in the production of osteonecrosis is also open to question. (Cutaneous, vascular, and vestibular manifestations have recently been observed by Idicula *et al.* [1972] in studying isobaric skin–gas exchanges involving different inert gases.)

Yet to be explored is the possibility that bone marrow saturated with a slowly exchanged gas (such as N_2) might become temporarily supersaturated should the subject begin breathing a "fast" gas (such as He). This possibility might have a limiting effect on the development of tables for diving with multiple inert gases, although Keller and Bühlmann (1965) have demonstrated that decompression time might be shortened by breathing mixed gases.

Alteration in blood components relative to diving and silent bubbles has been reported (Philp *et al.*, 1972). Stegall and Smith (1972) reported alterations in blood constituents in mini-pigs that developed dysbaric osteonecrosis. Further studies of blood–bubble interactions may give additional insight into the pathophysiology of osteonecrosis and the stresses that must be limited or avoided altogether during decompression.

SUMMARY

In areas of developing dysbaric osteonecrosis, bone marrow probably exchanges inert gas slowly. Lesions have developed even with tables of apparent safety — as evaluated in terms of incidence of dysbarism or by a simple exponential gas-exchange model (as used in the modified Haldane perfusion-limited model). Inequality in

gas uptake and elimination, as well as the low tolerance of bone for inert-gas supersaturation, may precipitate development of lesions when present-day decompression tables are followed. Showers of silent embolic bubbles during repeated decompressions may also be contributory factors, possibly because of secondary changes in blood constituents. Reflex circulatory and toxic changes in bone from an elevated Po_2 need further evaluation. Inert-gas exchange rates and the consequences of mixing or switching inert gases should also be studied in many tissues.

A reasonable approach to the modification of current decompression practices might reasonably include *1*) more positive consideration of tissues with longer half-times; 2) reduction of supersaturation in those tissues; and *3*) greater emphasis on the inequality of inert-gas uptake and elimination. Of possible importance, as well, would be efforts to decrease the separation of gas from solution into a gas phase (silent bubbles) by using deeper decompression stages, by closer adherence to the O_2 window principle of decompression, and by monitoring with ultrasonic techniques — particularly in repetitive diving.

However, expenditure of the large amounts of time, money, and energy required to arbitrarily modify and retest existing tables seems inefficient until a better understanding of the pathophysiology of dysbaric osteonecrosis helps reduce the choice of modifications. Meanwhile, a central registry to collect data on cases in the United States would allow a better evaluation of existing decompression tables. Statistical relationships could be established that might suggest certain changes that would make existing tables safer.

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