PRELIMINARY BART TUNNEL RESULTS

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The Bay Area Rapid Transit System, known as BART, was the first significant compressed-air tunneling operation in California and the largest civil engineering project ever performed on the West Coast of the United States. The 75-mile network includes duo-rail surface tracks, elevated lines, and subways linking San Francisco to the East Bay counties across San Francisco Bay and beneath the Oakland–San Francisco Bay Bridge. Of 64,576 ft of soft-ground tunneling performed during this project, 22,600 ft were machine-shield-driven by men using compressed air under hazardous soil and obstacle conditions.

Preliminary follow-up studies indicate that dysbaric osteonecrosis can possibly be prevented by instituting sophisticated engineering principles designed to avoid use of compressed air altogether, or to minimize exposure to pressure. Also important in the prevention of this disease in projects such as BART are comprehensive medical supervision, including thorough preemployment examinations; the use of the Washington State decompression tables, as incorporated in the California code (Behnke, 1968); and oxygen decompression in a closed system (Behnke, 1967).

ENGINEERING PRACTICES IN THE BART PROJECT

The BART project was engineered to avoid completely the use of compressed-air workers during construction of the Trans-Bay Tube. Prefabricated 320-ft-long sealed binocularlike tube sections, weighing 10,000 tons each, were floated into position. After gravel ballast was added to overcome buoyancy, each tunnel section was lowered by four cable falls, suspended from a twin-hulled placing barge, into a previously dredged 3.6-mile-long trench extending across the bottom of San Francisco Bay (Fig. 1). Deep-sea divers, using conventional diving suits and hard helmets, worked at approximately 100- to 120-ft depth, where they directed placement and mechanical linkage of the tube sections. No instance of decompression sickness was reported in these divers.

FIG. 1. Cable system used to lower tube sections from barge onto bottom of San Francisco Bay in course of Bay Area Rapid Transit construction. (Photograph courtesy of CIVIL ENGINEERING—ASCE, monthly publication of the American Society of Civil Engineers; September 1967.)

Of 64,576 ft of soft-ground tunneling performed on the BART project, nearly half was hand mined. Of the entire 12 miles of subway tunneling, 9 miles were driven in free air and 3 miles (15,026 ft) under compressed air. However, only 135 ft were driven in compressed air in excess of 17 psig, over a period of 47 days.

Tunneling machines, a recent innovation, were used in the BART project for the other half of the tunnel. A typical tunneling machine has a large cutting wheel (Fig. 2) that scrapes ground off the face and drops it onto a conveyor belt. Three radial doors on the face of the cutting wheel were opened by hydraulic rams. The cutting wheel was turned by planetary gears driven by hydraulic motors, which provided about 2 million ft-lb of torque (Fig. 3). Shove jacks, with the capacity of 115 tons each, were used to hold the cutter against the face during excavation. The forward progress, or shove, sufficient to install a liner ring in the tail of the shield, was 2.5 ft. Although manpower requirements were about equal for both machine-tunneling and hand-tunneling methods, those contractors using tunneling machines averaged advances of 40 ft per day as compared with 25 ft in the hand-mining projects.

About 5100 ft of twin subway tunnels north of the 16th Street Mission Station and an additional 3500 ft of twin tunnels south of the station were driven under compressed air. In both instances the original water level was above the crown of the tunnels.

To reduce the pressure of compressed air, dewatering procedures were introduced by drilling wells, 40 to 140 ft deep, into which submersible pumps were installed. Pumping began prior to tunnel excavation and the water table was lowered approximately 50 ft. It was often impossible to draw water down below the level of the tunnel invert, and air pressure was needed to counterbalance water pressure at the heading. These tunnels were constructed about 50 ft below the surface of the ground and air pressure varied from 9 psig to a maximum of 17 psig.

That portion of the project requiring work in compressed air at pressure great enough to cause dysbaric osteonecrosis involved construction of twin tunnels from the ventilation shaft to the

FIG. 2. Cutting wheel of soft-ground tunneling machine used in BART project.

Fig. 3. Rear view of tunneling machine's cutting wheel, used in BART project.

ventilation caisson through land reclaimed from beyond the shoreline as it existed in 1888. The tunnel is completely founded within new bay mud, which is very unstable material.

It was suspected that an old timber pier might be buried under the street surface, since it had been built as an extension of Market Street from First Street to the present location of the Ferry Building. In addition, the tunnel passes within a few feet of an old rubble seawall and through a forest of piling supporting the foundations of the Ferry Building. The final obstacle confronting this part of the project, and that which required use of progressively higher levels of compressed air, was the unstable backfill and clay blanket surrounding the caisson. Unfortunately, the lower end of Market Street was also the site of major destruction to buildings, which were constructed on unstable ground, during the 1906 San Francisco earthquake.

Timber-pile obstructions were handled by compressed-air workers with extreme caution since each pile was a potential "blow hole" whereby compressed-air pressure could suddenly be lost, with serious potential consequences for the workers. A total of 896 piles had to be disposed of, including one concrete and one steel pile. Locating the piling before each shield advance involved considerable delay. Of the 93 man-hours expended per foot of tunnel construction, 68 were required for excavation, ring erection, and grouting, and 5.2 for pile removal.

When horizontal tunnel excavation had progressed about 200 ft, a muck-lock and man-lock (Fig. 4) were installed adjacent to the working area. Compressed air supplied to the heading was cooled and filtered to prevent excessive fogging; burning and smoking inside the tunnels were prohibited.

The working day in compressed air was divided into four 6-hr shifts for each tunnel. An average heading crew consisted of 23 men with a support crew of 15. Those activities performed

Fig. 4. Photograph of man-lock of 7½-ft diameter. Lock systems were automatically controlled by computer, which the lock tender set for the required decompression time. Highest pressure at which tunnel was operated, 36 psig, required decompression of 3 hr and 28 min. Door through bulkhead into compressed-air tunnel remained open at all times to allow miners to enter lock rapidly in case of emergency.

during a typical soft-ground tunnel-driving cycle included shield advance, ring erection, grouting, and installation of hog rods. Pneumatic impact wrenches were ordinarily used to tighten the bolts between ring segments, pneumatic reciprocating pumps to inject grouting cement, and pneumatic chipping hammers to introduce lead strips into caulking grooves. Unfortunately, much of this work had to be performed overhead, resulting in significant stress to the workers' shoulder joints. Behind the shield was a platform where the soft-ground miners used a pneumatic hoist to lift the ring segments into position. The compressed-air workers were responsible for erecting the steel tunnel liners and for bolting the rings together. Through use of tunneling machines and other hydraulic equipment, however, the heavy overhead lifting usually required of these workers was minimized.

It was during the final phase of tunneling, when the pressure had to be increased, that the majority of severe cases of decompression sickness (Type II) developed. Maximum pressure in the left tunnel was 36 psig for 5 days and, in the right tunnel, 35 psig for 24 hours.

MEDICAL PRACTICES IN THE BART PROJECT

A Transit Compressed Air Medical Center in San Francisco serviced all BART operations conducted under compressed air. The Center performed all medical services required by the "Compressed Air Safety Orders" issued by the Division of Industrial Safety of the State of California. Every prospective workman in the compressed-air projects was required to be qualified by medical examination. After continuous hyperbaric work for one year, he was reexamined to reconfirm his qualification.

Each applicant completed a preemployment health-history form and underwent a comprehensive physical examination, including 11 roentgenograms. He was then subjected to a pressure test in the medical lock before being approved (Behnke, 1969). From June 1967 to November 1969, 1633 initial examinations and 256 annual examinations were performed. Of those examined, 81.1% were qualified for compressed-air work and 18.9% were disqualified, principally because of excessive weight and pulmonary abnormalities. Any applicant whose body weight exceeded 20% of normal was excluded. Pulmonary-function tests were routinely performed; if vital capacity was less than 80% of normal, timed VC measurement of maximal expiratory force was performed (Behnke, 1970) (see Table I).

To date, over 17,000 skeletal roentgenograms have been made on more than 2000 workers. Thirty-three osteonecrosis lesions of varying maturity were discovered in 12 preemployment examinations; 8 additional applicants had sus-

Table I. BART PROJECT: RESULTS OF PHYSICAL EXAMINATIONS OF APPLICANTS FOR WORK IN COMPRESSED AIR

	Number	%
Preemployment examinations		
Applicants qualified	1324	81.1
Applicants disqualified	309	18.9
Totals	1633	100.0
Reasons for disqualifications		
Overweight	96	31.1
Pulmonary dysfunction	66	21.4
Hernia	28	9.0
Ear block	28	9.0
Ear infection	21	6.8
Metabolic disorder	13	4.2
Osteonecrosis	12	3.9
Sickle-cell hemoglobinopathy	8	2.6
Other conditions	37	12.0
Totals	309	100.0

picious lesions and were rejected. Of the 12 subjects, 11 had potentially disabling juxta-articular lesions affecting one or both humeral heads; only 2 had involvement of the femoral heads.

It is significant that all these applicants, with one exception, were allegedly asymptomatic of joint involvement. It would therefore not have been possible to establish a diagnosis of chronic dysbaric osteonecrosis in these men without routine roentgenograms. Two of these rejected workers had previously been exposed to maximum pressures of 16 and 18 psig, respectively, without decompression sickness, but both had a history of alcoholism. All other rejected examinees had previously worked in compressed air for at least one year, and had been exposed to pressures in excess of 30 psig. Only 3 of these applicants admitted having experienced an attack of dysbarism; 7 had previously been exposed to maximum pressures in excess of 45 psig. Eight other applicants (2.6%) were disqualified because of sickle-cell hemoglobinopathy.

DECOMPRESSION PRACTICES IN THE BART PROJECT

The Medical Center had three compression chambers for testing workmen and for treating decompression sickness (DCS). Each had two compartments. The inner one, used for treatment, was equipped with oxygen; the small outer one allowed an attendant to enter without altering the inner compartment's pressure (Fig. 5). Because using compressed oxygen for treatment created a substantial fire hazard, each chamber was equipped with a sprinkler system capable of spraying the interior.

A pressure chamber was carried in an ambulance (Fig. 6) so that a severely injured workman

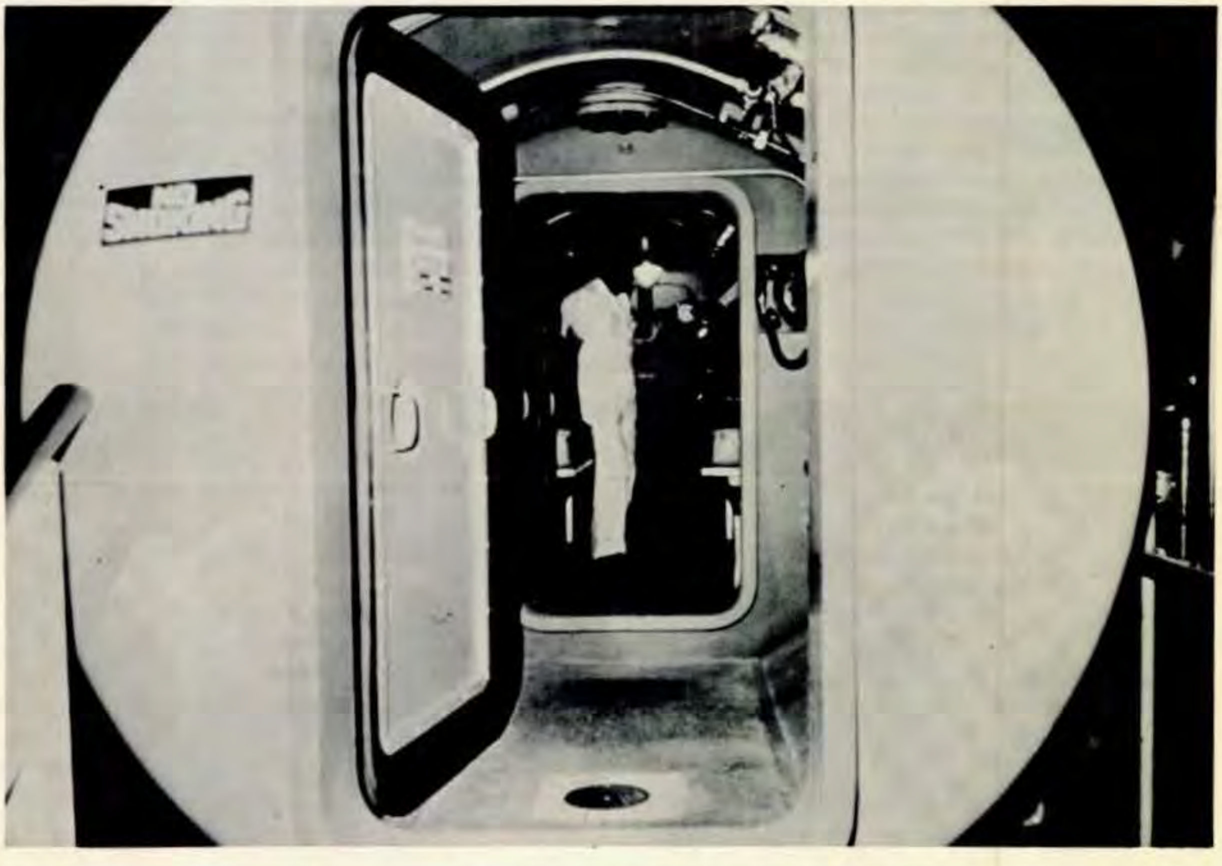

FIG. 5. Front and rear compartments of medical compression chamber at BART Transit Medical Center. Technician is standing inside inner treatment compartment.

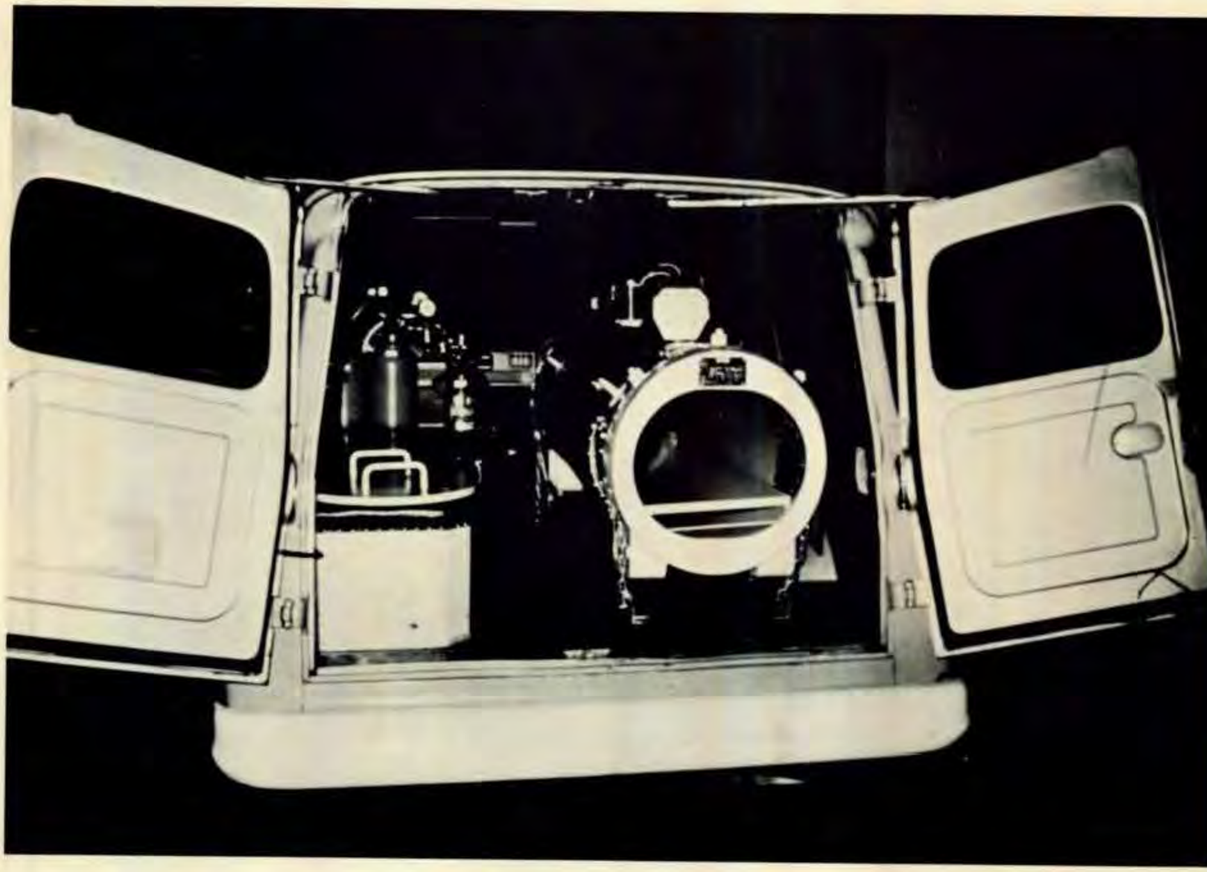

FIG. 6. Portable recompression chamber in rear of ambulance used in BART project, which allowed severely injured workmen to be removed from compressed-air tunnels without undergoing a decompression cycle.

might be extricated from the tunnels without undergoing decompression. Still under compression in the portable chamber, the workman was transported to the Center's medical chamber where his injuries could be treated during decompression.

From November 1967 to May 1969, 80,360 man-decompressions were performed at pressures between 9 and 36 psig. There were 135 instances of DCS involving 85 men among approximately 400 compressed-air workers — 128 of Type I (minor) and 7 of Type II (serious). In constructing the tunnels from the ventilation shaft to the ventilation caisson 473,000 man-hours were worked, followed by 22,359 decompressions. There were 116 episodes of DCS (110 Type I and 6 Type II); 46 men suffered repeat attacks.

In two instances a relatively low pressure was associated with postdecompression symptoms following 6-hr work shifts — 13 psig in one case and 11.5 psig in the other. At relatively high pressures a rather high incidence of DCS was experienced, despite curtailed hours of work. As mentioned, work at higher pressures lasted only one week; hence the time was too short to evaluate the role of acclimatization. However, there appeared to be selective susceptibility in that, of the 85 workers involved, 52 had one attack and 33 suffered repeated ones. One man was treated 6 times for DCS, and 10 of the 33 susceptible individuals had a second attack within 48 hours of the earlier one. No criteria were delineated from these examinations to separate the susceptible workers from those who were apparently resistant, or at least did not report for treatment (approximately 75%). The interval between decompression and the onset of symptoms was prolonged — 5 or 6 hours — which may possibly reflect the individual's reluctance to undergo recompression therapy or, possibly, to driving the 10- to 50-mile distance to the Medical Center.

Only 133 of the 832 miners originally employed for compressed-air work remained on the job and were available for their first annual examination. Results of a radiological bone survey were negative in all these men. Subsequent roentgenographic evaluations have likewise been negative. But since tunnelers are often an itinerant, migratory labor group, follow-up clinical and roentgenographic examinations are virtually impossible.

Four years have passed since the last compressed-air exposure of the BART project. No worker with clinical or roentgenographic evidence of dysbaric osteonecrosis has been reported, and no workmen's compensation claims have been filed in California as a result of the project. However, this is admittedly a preliminary report based on insufficient follow-up examinations.

DISCUSSION

The Decompression Sickness Panel of the Medical Research Council (U.K.) has found that bone lesions are related directly to the number of times that a man has been decompressed, the pressure at which he has worked, and the number of episodes of decompression sickness for which he has required recompression. The basic problem, however, transcends the prevention of DCS and focuses on decompression schedules that do not allow for the separation of inert gas from solution. Thus, nascent bubbles in the circulation, whether "silent" or clinically active, are clearly identified with such secondary phenomena as platelet aggregation, cell clumping, lipid release from lipoproteins, hemoconcentration, and fat embolism (Jones, 1971).

In the BART compressed-air operations, decompression (dc) was in accord with the recently formulated Washington State tables, which provide for single daily work shifts, rather than split shifts, and extended stage dc. The experience with these tables—which have also been adopted by other states, including New York and California — is that no crippling bone disability has been reported to date (November 1972) following trials in the Lake City Tunnel, Seattle (1964–1967), and in the BART project (1967–1969). The incidence of DCS in those two projects was, however, about the same as that in earlier compressed-air operations, which were followed by much shorter dc times.

Before specific regimens in support of these concepts are presented, it is appropriate to examine earlier dc experience resulting in the serious complication of avascular necrosis of bone.

New York Tables

East River Tunnel Decompression Practices (to 1909). Keays (1909) reported 3692 cases of DCS resulting from 557,000 decompressions in the construction of the East River Tunnels for the Pennsylvania Railroad. There were 20 deaths. At pressures of 32 psig, men worked 8 hours out of 24, taking 30 minutes for lunch at working pressure or one slightly lower. At higher pressures the work shifts consisted of two 3-hr exposures with a 3-hr rest interval at normal pressure. Working pressure never exceeded 42 psig. Keays preferred a 6-hr continuous shift to two 3-hr shifts "as it exposes the man to the risks of only one decompression instead of two."

In 23,000 decompressions from pressures between 40 and 42 psig, there were no serious or fatal cases among the 330 men employed for 36 days. The total dc time for each shift was 48 min, as follows:

1. Pressure was lowered from 40 to 29 psig in 5 min; the men then spent 10 min walking 1000 ft to a second lock.
2. Pressure was lowered from 29 to 12.5 psig in 8 min; the men spent another 10 min walking to a third lock.
3. Pressure was lowered from 12.5 to 0 psig in 15 min.

Considerable time, then, was spent at relatively high pressure levels during dc, and the workers engaged in light exercise (walking). Reporting on 8510 of these decompressions, Keays recorded 1.6% minor cases of DCS (Type I). Noteworthy is his statement that "only seasoned men were employed," conveying Keays' recognition of the remarkable acclimatization or acquired resistance to DCS that has subsequently been confirmed by British medical authorities (Paton and Walder, 1954; McCallum, 1968).

One of the paradoxes of dc practice observed over the years is that decreased incidence of Type I DCS does not correlate with increased dc time beyond minimal requirements. Keays' 3-hr work shift at 40 psig was followed by 48-min dc. This same exposure would require 98-min dc in England (Work in Compressed Air Special Regulations, U.K.); 162-min dc for USN divers (U.S. Navy Standard Air Decompression Tables for exceptional exposures); and 183 min for Seattle tunnelers (Washington State tables). Of possible significance in the occurrence of bends is the probability of extensive bubble evolution during the first-stage drop of 50% in the absolute or gauge pressure in these later tables. In this event the pressure head facilitating gas transport is greatly reduced, and little benefit would accrue from the moderately increased dc time. It is possible that a 48-min dc (Keays, 1909) at higher pressure is as effective as a two- or threefold increase in dc time at lower levels following initially large, abrupt increments of dc.

N. Y. Tables of 1912 and Subsequent Revisions. Progressively, hours of caisson work were decreased and the interval between shifts was lengthened. In contrast to the earlier compressed-air schedules, the 1912 tables (Table II) were seemingly liberal. In 1947 Captain O. E. Van Der Aue, MC, USN (personal communication), conducted dry-chamber tests at 26 psig with 12 divers in good physical condition and at rest. The exposures were the same as those allowed by the 1912 tables (3 hr) but included a rest interval, at normal pressure, of 3 hr (2 hr longer than that stipulated by the 1912 tables). The outcome of these nonwork exposures is shown in Table III.

Table II. 1912 N.Y. STATE DECOMPRESSION TABLES AND SUBSEQUENT REVISIONS

Pressure (psig)	Shift 1 (hr)	Interval at surface	Shift 2 (hr)	Decompression per shift (min)	
1912					
to 22	4.0	0.5	4.0	7 at 17.8 psig	
22 to 30	3.0	1.0	3.0	12 at 26.7 "	
30 to 35	2.0	2.0	2.0	19 at 31.2 "	
35 to 40	1.5	3.0	1.5	24 at 40 "	
40 to 45	1.0	4.0	1.0	— —	
45 to 50	0.75	5.0	0.75	28 at 49 "	
1922*					
to 18	4.0	0.5	4.0	5**	
18 to 26	3.0	1.0	3.0	12**	
26 to 33	2.0	2.0	2.0	21**	
33 to 38	1.5	3.0	3.0	23**	
38 to 43	1.0	4.0	4.0	27**	
43 to 48	0.75	5.0	0.75	29**	
1955–1957* **				1st	2nd
to 22	3.0	2.5	3.0	16**	27**
22 to 30	2.0	3.5	2.0	22**	37**
30 to 35	1.5	4.0	1.5	25**	42**
35 to 40	1.0	4.5	1.0	29**	49**
40 to 45	0.75	4.75	0.75	32**	57**
45 to 50	0.5	5.0	0.5	37**	62**

*Decompression time for maximum pressure in each category

**Stage 1 dc — Reduce gauge pressure by 50% at rate of 5 psi/min

Stage 2 dc — Reduce pressure at uniform rate according to maximum pressure:

Working pressure 0 to <15 psi, rate: 3 psi/min
Working pressure 15 to <20 psi, rate: 2 psi/min
Working pressure 20 to <30 psi, rate: 3 psi/2 min
Working pressure 30 psi and over, rate: 1 psi/min

***Lincoln Tunnel (Third Tube)

Table III. Results of Dry-Chamber Pressure Exposures, with Subjects at Rest*

Diver	Condition between 1st and 2nd shifts	Following 2nd shift
1	No symptoms	Bends, recompression
2	" "	No symptoms
3	" "	Pain, hand
4	" "	Pain, arm
5	" "	Back pain, fatigue
6	" "	No symptoms
7	" "	Stiffness, knee
8	" "	Bends
9	Skin itch	Skin itch
10	Fleeting pain, shoulders	Bends
11	Pain, shoulder (at 175 min)	Bends, recompression
12	Pain, knees (at 120 min)	Bends

*Van Der Aue, personal communication

It is likely that acclimatization may have modified these unexpected results. However, it is difficult to reconcile the striking difference between dc practice relative to divers (stage dc with progressively longer time at successive stops) and to tunnel workers (the split shift and shortened dc time at a uniform rate following the initial 50% drop in gauge pressure).

The 1912 tables proved to be inadequate and were revised in 1922 (Table II). But despite shortened hours of work and somewhat longer intervals in open air, serious morbidity still occurred. Of the 300 instances of DCS reported by Thorne (1941), there were 25 Type II cases involving the CNS, 15 Type II cases with cardiopulmonary symptoms ("chokes"), and 30 Type II cases with vertigo ("staggers").

Bone Necrosis Relative to the 1922 N. Y. Tables. In 1942, Bell *et al.* reported on a radiologic survey of 32 compressed-air workers in New York, none of whom had symptoms or gross signs indicative of bone lesions. The men had worked intermittently for 3 to 33 years in compressed air. The shortest continuous employment was 10 months, the longest, 36 months. Fourteen men gave a history of DCS but 18 stated that they had never had an attack of bends. Yet only 8 of the 32 workers were free of radiologic evidence of bone lesions.

Certain aspects of avascular bone necrosis — specifically, individual variations in response to similar conditions of decompression — were as puzzling then as now. Taylor (1944) reported that the same type of lesions was observed in 38 individuals who had never worked in compressed air: "Individuals with an inadequate circulatory tree who work under increased pressures may develop bone and joint lesions with the ordinary so-called adequate decompression, even in the absence of an acute attack of caisson disease. Agglutination of red cells may play a part."

Based on radiologic evidence of bone lesions, 63 claims for disability were made by tunnel workers in New York in October 1963. Paradoxically, these claims related to the Lincoln Tunnel (Third Tube) operations of 1955–1957 (Table II), which were conducted according to "enlightened" schedules stipulating minimal hours of work and maximal decompression, in accord with principles underlying U.S. Navy practice. This experience merits recounting, since it points up the prohibitive cost of manned, pressurized tunnel operations.

Following World War II, the New York State Department of Health and the Port Authority, in consultation with O. E. Van Der Aue, G. J. Duffner, and A. R. Behnke, Jr., sought to revise the 1922 tables. In addition to curtailing work hours further, about 66% more stage-decompression time was allotted for the second shift of the split-shift regimen. Although Kooperstein and Schuman (1957) reported only 44 cases of DCS out of 138,034 decompressions (3.18 cases/10,000), it is not possible to state with complete assurance that no bone lesions complicated this specific operation. It seems probable that some of the 63 processed claims for disability due to osteonecrosis in the Lincoln Tunnel workers may have occurred as a result of injury received prior to 1955, since preemployment radiological bone surveys were not performed.

Washington State Tables, 1963

Rationale. In 1961 J. Leon Sealey, Medical Consultant to the Municipality of Seattle and Metropolitan Engineers, organized a committee to revise dc tables for compressed-air workers. Their objective was to formulate tables based on the following concepts:

1. A single daily work shift with stage dc limited to a ΔP of 16 psi relative to tissue half-times of 30, 60, and 120 min.
2. A potential 8-hr exposure in compressed air apportioned between work at tunnel pressure and stipulated dc time.

For example, a 6-hr shift at 22 psig requires 103-min dc; total time under pressure, 7 hr and 43 min. A 5-hr shift at 32 psig is accorded 178-min dc; total time under pressure, 7 hr and 58 min. The extended time stipulated for dc in the usual range of tunnel pressures (0 to 35 psig) is more than twice the time allotted for the same work shifts in England, and even exceeds dc time for exceptional air exposures in the U.S. Navy diving tables.

Incidence of DCS Relative to Extended Decompression Time. The innovations outlined above were enforced throughout the Seattle project (1964–1967), the BART project (1967–1969), and recently in Milwaukee (1971–1972). An unexpected finding was that the incidence of DCS did not diminish as a result of the greatly extended dc time (Table IV). The exceptionally high incidence in the BART operation at 29.5 and 35.5 psig, relative to the modest length of the work shifts, may be partially due to a lack of acclimation in the workers. Highly favorable, however, was the response to low-pressure oxygen therapy, as stipulated by U.S. Navy treatment tables 5 and 6, in the 345 cases of DCS (Seattle and BART projects). There were no residual complications in these cases.

Absence of Disabling Bone Lesions. The major benefit now apparent from this protocol is the absence of disabling juxta-articular necrosis. Limited opportunity for radiologic follow-up precludes a conclusive statement regarding ultimate benefits, but restricting the number of work shifts above 26 psig also appears to militate against osseous complications.

Inherent and Extraneous Impediments to Decompression. In the Washington State tables, a 16-psig drop in pressure takes place in 3 min to the first stop. Following a 6-hr work shift at 30 psig, the P_{N_2} resulting from the rapid pressure drop to 15 psig is substantially below that calculated as safe (18 psig) in a 120-min tissue half-time ($T_{1/2}$). The pressure is then reduced from 14 to 4 psig in 35 min, and from 4 to 0 psig in 130 min. The substantial part of dc is therefore spent under conditions of diminishing N_2 transport (*i.e.*, a decreasing size of the "oxygen window"). On the assumption (now experimentally verified) that bubbles form during initial stages of dc, the schedule is, in effect, a treatment regimen. It is beyond the scope of this section to discuss obvious modifications of the Washington State tables, which must be validated by systematic test procedures.

Two undesirable physical effects of a rapid pressure drop in chamber-lock air are fog formation and cooling. When hot and sweating workers become chilled, a peripheral vasoconstriction may occur. These conditions impair blood flow and favor bubble evolution in subcutaneous vessels, which may give rise to pruritus and mottling.

Various physiologic factors impair gas trans-

Table IV. DECOMPRESSION TIME (MIN) ACCORDING TO 1971 MRC (U.K.) TABLES AND TO WASHINGTON STATE TABLES*

Pressure (psig)	Shift, 1 to 2 hr	Shift, 2 to 3 hr	Shift, 3 to 4 hr	Shift, over 4 hr
18 to 20	8 (8)	10 (11)	12 (17)	17½ (63)
24 to 26	17 (27)	27 (52)	37 (92)	51 (122)
28 to 30	39 (41)	51 (98)	58 (127)	70 (153)
34 to 36	52 (98)	72 (151)	82 (178)	98½ (218)
38 to 40	62 (128)	86 (178)	98 (203)	117½ (238)

*The decompression times (min) shown in parentheses are calculated from Washington State tables for the lowest pressure in the range quoted from the MRC (U.K.) tables and for shifts of 2, 3, 4, and 6 hr, respectively.

port — *e.g.*, dehydration in the hot atmosphere surrounding the automated shield at the face of the tunnel, disruption of circadian rhythms because of periodic work-shift rotations, and deficient blood perfusion of tissues as a result of alcoholism, intravascular bubble formation, bubble-related clumping of cells, and decreased blood-clotting time. In the course of long decompressions, the relative immobility of the lower extremities of workers as they play cards (the typical pastime) obviously serves to restrict regional N_2 transport.

British Decompression Experience

In the construction of the road tunnel under the River Tyne (England), 641 men worked in compressed air over a period of 31 months (1962–1965). The maximum pressure was 42 psig and the overall dysbarism rate for work at pressures of 18 psig and higher was 2%. Data extracted from the report of the Decompression Sickness Panel (1971) affirm the high percentage of DCS with increased exposures over 4 hr and with higher pressures (Table V). Inexplicably, the modified U.K. statutory decompression table does not provide increased dc time for work shifts over 4 hr. In an earlier study, Paton and Walder (1954) reported that increase in stipulated dc time did not diminish the incidence of DCS. A possible explanation of this circumstance was commented on earlier.

During construction of the River Tyne road tunnel, radiologic examinations were made of 171 men; avascular osteonecrosis was found in one or more bones of 44 men (26%). Although most of these workers were symptomless three years after the termination of compressed-air exposure, four were partially disabled despite surgery.

The results shown in Table V reveal that work shifts of less than 4 hr produced minimal complications. When the shifts were 8 hr or longer, the incidence of Type I DCS increased fivefold, and there were 16 cases of Type II DCS.

In Table IV the total dc times of the 1971 MRC (U.K.) tables for the usual range of tunnel pressures relative to shift duration are compared with the much greater dc times of the Washington State tables. Note again that absolute pressure is reduced 50% during the rapid pressure decrease to the first stop. With the Doppler ultrasonic detector, it is possible to substantiate the theory that gas bubbles form in the circulation during this initial stage of decompression. The Decompression Sickness Panel concludes that the dc procedures and treatment of bends currently accepted in civil-engineering practice do not prevent aseptic bone necrosis in compressed-air workers.

Table V. INCIDENCE OF DECOMPRESSION SICKNESS RELATIVE TO MAN-DECOMPRESSIONS, TYNE ROAD TUNNEL, ENGLAND*

Pressure (psig)	Number of man-decompressions			
	Shift, under 4 hr	Shift, 4 to 6 hr	Shift, 6 to 8 hr	Shift, 8 hr and over
14 to 19	7625 [0.53]**	1504 —	2332 [5.58]	8698 [5.98]
20 to 29	5262 [4.6]	1073 [27.0]	1617 [33.4]	9367 [20.8]
30 to 36	1547 [8.5]	359 [61.3]	404 [40.4]	2977 [53.4]
37 to 41	495 [18.2]	105 —	38 —	564 [67.4]
Totals	14,929 [3.35]	3041 [23.4]	4391 [19.1]	25,606 [17.1]
Number of Type II (Serious) DCS	1	3	3	16

*Abstracted from Medical Research Council, 1971

**Incidence of Type I (minor) DCS per 1000 man-decompressions shown in brackets

Physiologic Principles Pertaining to Gas Transport during Decompression

Body Composition and Gas Transport. An estimate, supported in part by quantitative data, can be made of blood flow to various bodily tissues of a hypothetical lean (70-kg) man relative to N_2 content (Table VI). About 50% of the N_2 content of this man (10% fat in adipose tissue) is in aqueous tissues with a large blood supply. The other 50% is in fatty tissues with a meager blood supply. The $T_{1/2}$ for N_2 transport in aqueous tissues varies from less than 2 min to not more than about 16 min. In this man's fatty tissues, $T_{1/2}$ for N_2 transport ranges between 85 and 120 min.

Definite end-points for N_2 elimination have not been determined. But Lundin's data (1960) clearly show the association between body fat and prolongation of desaturation time. Calculations indicate that about 54 ml of N_2 will be added to the body's N_2 store for every kg of fat gain. Moreover, the "heavy" men in Gray's analysis (1951) manifested progressive susceptibility to altitude decompression sickness in relation to weight.

Haldane's Ratio Concept and Stage Method. It is apparent from dc data that exposures to 15 psig and even somewhat higher pressures are well tolerated in acclimatized workers without need for more than a few minutes' dc (minimal). Haldane postulated (Boycott *et al.*, 1908) that, since the body could be decompressed rapidly from 2 to 1 atm abs, it would be safe to reduce the absolute pressure at higher levels by 50% as the first stage in dc. For the diving depths and exposure times with which Haldane was concerned, it appeared that the 2:1 supersaturation ratio was satisfactory. But subsequent deeper dives and longer exposures have demonstrated unequivocally that no single ratio applies in decompression and that, probably, no degree of inert-gas supersaturation is maintained in circulating blood for any appreciable time. *What appears to be a ratio indicative of inert-gas supersaturation in blood may, in reality, be an index of the degree of air embolism that the body can tolerate.*

The ΔP Principle. Another concept applied to decompression-schedule calculation is based upon the assumption that tissue pressure can be safely decreased from 2 to 1 atm abs, creating a pressure head of air (*i.e.*, about 12 psi N_2) that can be safely sustained during decompression at all pressure levels above ambient pressure. The ΔP principle has been widely applied, notably in the computation of the Washington State tables. However, the physiologic and physical bases underlying gas transport do not explain decompres-

Table VI. ESTIMATED WEIGHT, FLUID, AND LIPID CONTENT OF 5 TISSUE CATEGORIES FOR 70-KG MAN, PLUS CALCULATED N_2 CONTENT RELATIVE TO BLOOD PERFUSION AND $T_{1/2}$ FOR N_2 TRANSPORT

Parameter	Tissue-organ group				
	Blood, organs, red marrow, GI tract	Muscles, skin, spinal cord, nerves	Bone matrix* (fat, mineral-free)	Bone marrow (fat-rich)	Adipose tissue (lean man)**
Weight (g)	15,000	37,000	4,000	1,500	9,500
Fluid (g)	12,000	28,000	2,500	200	2,000
Lipid (g)	400	400	—	1,200	7,000
N_2 content (ml)***					
Fluid	108	252	23	2	18
Lipid	22	22	—	65	378
Blood perfusion (ml/min)	4,000	1,200	80?	50?	400
N_2 transport ($T_{1/2}$/min)	2 to 16		85? to 120?		

*Weight of mineral in bone: 3000 g

**Lean man: 10% of body weight is lipid in adipose tissue

***N_2 solubility per kg fluid, 9 ml; per kg lipid, 54 ml (P_{N_2}=570 mm Hg; temp., 37°C) sion at a fixed oversaturation pressure of 12 psig any better than they do decompression at a fixed 2:1 ratio.

The Isobaric ("Oxygen Window") Concept. During the course of blood flow through capillaries, oxygen is unloaded in different quantities to various tissues. During the late 1930s Capt. Charles B. Momsen, USN, and his medical officers (U.S. Navy Experimental Diving Unit) postulated that O_2 diffusion from capillary blood into tissues creates a "partial pressure vacancy" to permit isobaric transport of an equivalent amount of inert gas from tissues to capillaries to lungs. This concept holds that, essentially, an "oxygen window" exists in the capillary blood through which inert gas diffuses from tissues. This window is proportional in size to the alveolar Po_2.

At normal pressure during air inhalation, arterial Po_2 (100 mm Hg) falls to about 40 mm Hg in the capillaries. The size of the potential window (60 mm Hg) is reduced to about 53 mm Hg by an increase in capillary CO_2 tension. If arterial Po_2 is elevated to 287 mm Hg through inhalation of a mixture richer in O_2 than air, mean capillary Po_2 rises to about 50 mm Hg. With the addition of 7 mm Hg of CO_2, the O_2 window $[287-(50+7)]$ equals 230 mm Hg, the equivalent of 10 FSW.

How does this concept apply to diving decompression? It is postulated currently that, during the course of decompression following a helium saturation dive, there is permissible oversaturation of inert gas, ΔP, equal to $\pi - P$, where π is inert-gas tension in the slowest desaturation tissue ($T_{1/2}$, 240 min) and where P is total ambient pressure. The total driving force implementing inert-gas transport is $\Delta P +$ alveolar Po_2. Ascent rate for desaturating 1 ft of He from a 240-min tissue is represented by:

$$\frac{d\pi}{dt} = -\frac{0.693}{240 \text{ min}}(20+10) \quad = \quad 11.6 \text{ min per ft.}$$

π is equal to inert-gas tissue tension, $0.693 = \ln 2$, $\Delta P = 20$ FSW, and $Po_2 = 0.3$ atm abs $= 10$ FSW (Workman, 1969). However, if permissible oversaturation — *i.e.*, ΔP of 20 FSW — is incompatible with transport of inert gas *in solution*, an ascent rate of 11.6 min/ft would still hold if the $T_{1/2}$ of the slowest tissue is 80 min rather than 240 min, as indicated by earlier (admittedly incomplete) measurement of He transport. The following comparative data demonstrate isobaric gas transport from tissues to lungs.

	Gas tensions (mm Hg)	
	Lungs	*Tissue-capillary blood*
Helium	2000	2230
Oxygen	287	50
Carbon dioxide	40	47
Water vapor	47	47
Totals	2374	2374

The O_2 window for He transport is 230 mm Hg (≈ 10 FSW). Isobaric decompression can progress uniformly at a rate of about 11.5 min/ft, a rate concurrent with a 230-mm-Hg fall per min of He in the 80-min tissue.

The isobaric principle of decompression is mandatory in deep saturation diving, but it requires too long a time to be practical in subsaturation diving. Hence an unfortunate compromise between the ideal and the practical has been made with the consequence of an appreciable incidence of decompression sickness (Behnke, 1967; Behnke, 1969).

Oxygen in Decompression and Recompression Practice

Oxygen is not routinely employed in the dc of tunnel workers, chiefly because a regimen has not been developed to control the fire hazard involved. However, effective fire control has been maintained in hyperbaric chambers in the dc of divers, treatment of DCS, and administration of hyperbaric oxygen to patients. During salvage operations of the Submarine *Squalus* in 1939, oxygen was first used in surface decompression. Divers were brought rapidly to the surface, then were recompressed in a deck decompression chamber on O_2 to pressures between 26.7 psig (60 FSW) and 17.8-psig (40 FSW). At the end of the scheduled 17.8 psig period, pressure was decreased to 1 atm abs in 5 min. There were no decompression stages at less than 40 FSW (17.8 psig).

Oxygen decompression of divers and O_2 treatment of DCS have become routine in the U.S. Navy, but there is either stage or uniform decompression at pressures lower than 15 psig. These procedures are questionable for two reasons: *1*) the possibility of bubble growth increases, and *2*) the inert-gas transport capacity of blood diminishes as the pressure gradient falls below 15 psig.

Sealey (1970) found that hyperbaric O_2 treatment in accord with U.S. Navy treatment tables 5 and 6 was singularly effective in the management of 210 cases of DCS. This therapy, modified to eliminate uniform decompression from 15 psig, was also highly effective in promoting the prompt recovery of 135 BART workers stricken with DCS, despite an average 5- to 6-hr delay before they reported for treatment. With few exceptions the patients, who had received 90 and 120 min of O_2 therapy, resumed their next regular work shift (Behnke, 1970).

Oxygen Decompression of Tunnel Workers. Oxygen rather than air inhalation during the dc of tunnel workers would reduce the decompression time of the Washington State tables by at least a factor of 2. This regimen also holds great promise in preventing both DCS and dysbaric bone necrosis, since excess N_2 can be eliminated from tissues under isobaric conditions. In addition, work shifts could be lengthened safely.

Table VII incorporates data for various isobaric decompressions utilizing O_2 inhalation following work shifts of 6 hr at 20, 30, and 36 psig, and of 4 hr at 40 psig.

Calculation Based on a 120-min Half-time Tissue. At the end of a 6-hr shift, $P_{N_2} = 11 + 0.875$ (80% psig) and at the end of a 4-hr shift, $P_{N_2} = 11 + 0.75$ (80% psig). The values 0.875 and 0.75 represent % saturation of 120-min tissue following 3 and 2 half-saturation time units (T.U.), respectively; 11 psia is tissue N_2 partial pressure in equilibrium with alveolar N_2 at 1 atm abs.

Excess N_2 (ΔP_{N_2}) is the difference between the P_{N_2} of the 120-min tissue and the permissible level, which is set at the conservative figure of 19 psia (11 psia + 8 psia). The 8-psia P_{N_2} value for the 120-min tissue is highly conservative, a level requiring no decompression after saturation exposure.

The length of time for O_2 decompression is calculated as follows:

$$\frac{\Delta P}{P_{N_2}\ (\text{120-min tissue})}.$$

For example, after a 6-hr shift at 36 psig, P_{N_2} in the 120-min tissue is 36.2 psia and ΔP_{N_2} is 17.2 psi (36.2 − 19). Oxygen decompression time is computed from 17.2/36.2, or 47.5% of the total. This percentage of N_2 is eliminated in 112 min from the 120-min tissue. In the last column of Table VII are shown rounded values for O_2 dc per hr of work. These values relate conservatively to the estimate that 80% to 85% is the "effective" O_2 concentration in the lungs.

The size of the O_2 window (*i.e.*, the approximate pressure level for O_2 inhalation) is at all times not less than the P_{N_2} in the 120-min tissue (Table VII). Inhalation of O_2 at these pressures has been well tolerated in practice. In preliminary tests of isobaric decompression, the following schedule of moderate exercise, performed under the voluntarily imposed adverse chamber conditions of high humidity and no ventilation, was well tolerated by a 65-yr-old subject:

Pressure (psig)	Duration (hr)	O_2 decompression (min) per hr of work
20	1 to 6	10
30	1 to 6	20
40	1 to 4	30

After the 40-psig exposures, the subject inhaled O_2 continuously at pressures no higher than 30 psig (for short periods) and no lower

Table VII. PROTOTYPE OXYGEN DECOMPRESSION TABLE FOR TUNNEL WORKERS

Tunnel pressure (psig)	Work shift (hr)	P_{N_2}* (psia)	ΔP_{N_2}** (psi)	Calculated decompression time (min)***	
				Total	*Per hr work*
20	6	25.0	6.0	48	10
30	6	33.0	14.0	96	20
36	6	36.2	17.2	112	23
40	4	35.0	16.0	102	30

*In 120-min tissue at end of shift

**Excess N_2 to be eliminated from 120-min tissue + P_{N_2} − 19

***Rounded estimates based on 80% to 85% alveolar O_2

than 15 psig. The closed-circuit O_2 inhalation apparatus was periodically rinsed by means of a "demand" regulator. Continuous O_2 inhalation against some expiratory resistance, interspersed with periodic deep breaths, was well tolerated for 2-hr periods. At the termination of O_2 breathing, the subject was decompressed on air in 5 min to normal pressure.

Few data exist regarding daily inhalation of O_2 for the purpose of decompression. Tests by Jacobs *et al.* (1970) demonstrated that elderly persons with senile impairment could tolerate inhalation of 100% O_2 for 90-min periods twice daily at 2.5 atm abs without ill effects. These exposures were repeated for 150 to 435 days with only an occasional break. What is now needed is the systematic daily exposure of healthy men to an O_2 dc regimen following the work shifts described in this paper.

Periodic Residence in Compressed Air

It has been shown repeatedly that habitation in dry chambers and undersea habitats is feasible for at least two weeks in air atmospheres at depths equivalent to 50 FSW (22.2 psig). Larsen and Mazzone (1967) conducted tests showing that a 4-hr no-decompression excursion can be made to a simulated depth of 100 FSW (44.5 psig) from a saturation pressure of 15.6 psig (35 FSW). The following tabulation indicates the scope of no-decompression excursions that are considered theoretically safe:

Saturation pressure (psig)	Working pressure (psig)	Work time (hr)
15	25	6
15	30	4
15	35	3
15	40	2

The feasibility of two work shifts daily per man remains to be tested with and without O_2 inhalation following saturation exposures at 15 psig.

If the pressure in an undersea habitat can be safely raised to an upper level of 22.2 psig (50 FSW), it should be possible to conduct work at tunnel pressures up to 32 psig for indefinite periods followed by isobaric decompression back to habitat pressure. At higher working pressures O_2 could be inhaled for short periods at habitat pressure.

A detailed schedule applicable to tunnel workers remains to be developed and validated by repeated tests.

SUMMARY

Osteonecrosis is a major hazard when compressed air is used in civil-engineering projects (tunnel or caisson work). Preliminary analysis of the BART project's history suggests that dysbaric osteonecrosis may possibly be prevented by following certain engineering and medical practices.

The risk of decompression sickness is minimal if working pressure can be maintained below 11 psig; maintaining pressure below 17 psig minimizes the risk of dysbaric osteonecrosis. Engineering consideration should therefore be given to avoiding the use of compressed air altogether, or minimizing the pressure necessary in tunnel, caisson, or diving work. Engineering principles should also include *1*) use of prefabricated tunnels deployed with minimum direct supervision by divers breathing compressed air; *2*) dewatering, probing, and elimination of obstacles in the heading; *3*) use of machine-tunneling apparatus and mechanically operated erector arms, which reduce the requirement for pneumatic hand-operated equipment and heavy overhead lifting; and *4*) maintaining a man-lock and emergency lock adjacent to the working area.

To disqualify from work those applicants with antecedent dysbaric osteonecrosis or coexistent illnesses associated with avascular osteonecrosis, comprehensive preemployment examinations are essential — including special laboratory studies and roentgenograms. Bone lesions have been associated with the primary effects of nascent bubbles liberated during rapid decompression and with such secondary complications incident to bubble release as platelet aggregation, cellular clumping, release of lipids with embolic potential, and circulatory stasis. Predisposing factors are repeated decompressions, the pressure under which the individual has worked, and the number of attacks of dysbarism that he has experienced.

During the past 50 years steps have been taken in New York State to reduce the incidence of dysbarism and the complication of bone necrosis by shortening hours of work. In both U.S. and U.K. experience, it has not been possible to limit the hazard of decompression sickness.

Tables have recently been computed by the State of Washington that greatly extend decompression time following prolonged pressure exposure. But use of these tables in the Seattle tunnel project (1964–1967) and in San Francisco's BART project (1967–1969) did not appear to reduce the incidence of dysbarism. Nevertheless, no case of disabling bone necrosis has been

reported as of this writing (1972).

To circumvent future dysbarism-related injury in compressed-air work, two positive procedures are suggested: *1*) O_2 decompression under conditions favoring isobaric N_2 elimination from tissues; and *2*) residence in compressed-air habitats with pressure adjusted to that of the tunnel. The feasibility of these procedures has been demonstrated in diving practice, but they remain to be implemented in compressed-air tunnel operations.

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REFERENCES

Behnke, A. R. (1967). The isobaric (oxygen window) principle of decompression. In The New Thrust Seaward. pp. 213-228. Washington, D.C.: Marine Tech. Soc.

Behnke, A. R. (1968). Medical aspects of work in pressurized tunnel operations. Transit Insurance Administrators Publ.

Behnke, A. R. (1969). New approaches to medical aspects of work in compressed air. J. Occupational Med. 11, 259-272.

Behnke, A. R. (1970). Medical aspects of pressurized tunnel operations. J. Occupational Med. 12, 101-112.

Bell, A. L., Edson, G. N., and Hornick, N. (1942). Characteristic bone and joint changes in compressed-air workers: A survey of symptomless cases. Radiology 38, 698-707.

Boycott, A. E., Damant, G. C. C., and Haldane, J. S. (1908). The prevention of compressed-air illness. J. Hyg., Camb. 8, 342-443.

Decompression Sickness Panel, Medical Research Council. (1971). Decompression sickness and aseptic necrosis of bone. Brit. J. Med. 28, 1-21.

Gray, J. S. (1951). Constitutional factors affecting susceptibility in decompression sickness. In Decompression Sickness. p. 182. Ed. Fulton, J. F. Philadelphia: Saunders.

Jacobs, E. A., Winter, P. M., Alvis, H. J., and Small, S. M. (1970). Hyperoxygenation effect on cognitive functions. In Proceedings of the Fourth International Congress on Hyperbaric Medicine. pp. 448-452. Tokyo: Shoin.

Jones, J. P., Jr. (1971). Alcoholism, hypercortisonism, fat embolism and avascular necrosis. In Idiopathic Ischemic Necrosis of the Femoral Head in Adults. p. 130. Ed. Zinn, W. M. Stuttgart, Germany: Thieme.

Keays, F. L. (1909). Compressed air illness with a report of 3,692 cases. Publ. Cornell Univ. Med. Coll. 2, 1-55.

Kooperstein, S. J., and Schuman, B. J. (1957). Acute decompression illness: A report of 44 cases. Ind. Med. Surg. 26, 492-496.

Larsen, R. T., and Mazzone, W. F. (1967). Excursion diving from saturation exposures at depth. In Proceedings of the Third Symposium on Underwater Physiology. pp. 241-254. Ed. Lambertsen, C. J. Baltimore: Williams & Wilkins.

Lundin, G. (1960). Nitrogen elimination from the tissues during oxygen breathing and its relationship to fat:muscle ratio and the localization of bends. J. Physiol. 152, 167-175.

McCallum, R. I. (1968). Decompression sickness: A review. Brit. J. Ind. Med. 25, 4-21.

Paton, W. D. M., and Walder, D. N. (1954). Compressed air illness: An investigation during the construction of the Tyne Tunnel, 1948-50. p. 18. Special Rep. Series, Medical Research Council (Lond.), No. 281. London: Her Majesty's Stationery Office.

Sealey, J. L. (1969). Safe exit from the hyperbaric environment: Medical experience with pressurized tunnel operations. J. Occupational Med. 11, 273-275.

Sealey, J. L. (1970). Minimal recompression, hyperbaric oxygen treatment of decompression sickness in tunnel workers. In Proceedings of the Fourth International Congress on Hyperbaric Medicine. pp. 100-104. Tokyo: Shoin.

Taylor, H. K. (1944). Aseptic necrosis in adults: Caisson workers and others. Radiology 42, 550-569.

Thorne, I. J. (1941). Caisson disease: A study based on three hundred cases observed at the Queens-Midtown Tunnel project. J. Amer. Med. Assoc. 117, 585-588.

Workman, R. D. (1969). American decompression theory and practice. In The Physiology and Medicine of Diving and Compressed Air Work. Ed. Bennett, P. B., and Elliott, D. H. Baltimore: Williams & Wilkins.