12. PRESSURE

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TABLE OF CONTENTS

12. PRESSURE

This section covers static pressure, slow and rapid (explosive) decompression, and blast overpressure. The partial pressure environment is covered in Oxygen-CO_2-Energy, (No. 10) and Inert Gas (No. 11).

Static Pressure

The lower limits of static pressure are determined primarily by the availability of an adequate P_{O_2} in the lungs for unimpaired performance (Figure 12-1) and decompression sickness (see below).

The physiological relations between the percentage of oxygen in the atmosphere of an aerospace vehicle and the total pressure of that atmosphere shown in Figure 12-1 are based on continuous exposure for one week or more.

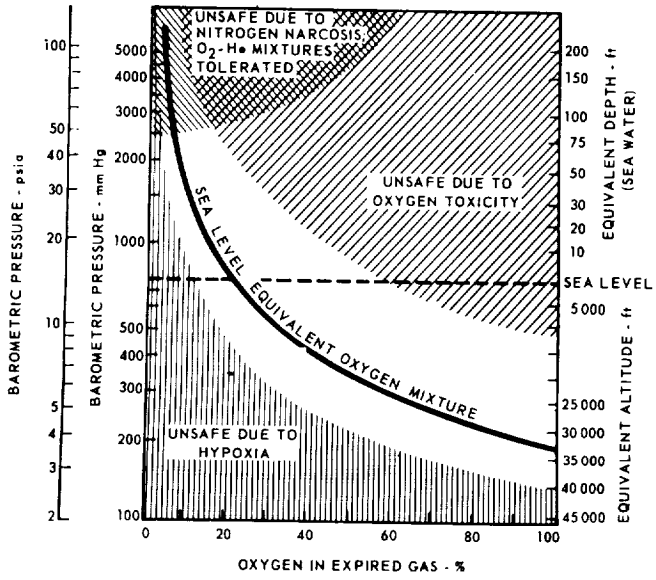


Figure 12-1

The Effect of Barometric Pressure and Altitude on Oxygen Required for Normal Functions

(Adapted from the Space Handbook [115])

Atmospheric air contains 21% oxygen by volume. At sea level, this leads to a blood saturation of 95%. To maintain the same degree of oxygen in the blood at lower pressure, the percentage of oxygen in the atmosphere must increase as shown by the "sea level equivalent" curve. The clear unimpaired performance zone, bounded by the hatched lines, indicates the range of variation that can be tolerated without performance decrement (see Oxygen-CO_2-Energy (No. 10).

Prolonged exposure to low oxygen levels lying to the left of the clear unimpaired performance zone requires acclimatization. Acclimatization is accomplished by continuous exposure to successively lower pressures with

no intermediate return to higher pressures. Acclimatization to 25,000 feet requires 4-6 weeks and performance is still impaired.

The upper limits of pressure are determined by nitrogen narcosis and oxygen toxicity as indicated by the hatched lines in Figure 12-1. The maximum oxygen tolerance (definite pathology) for long periods is currently under investigation. The role of nitrogen and trace contaminants on the symptoms and signs of oxygen toxicity in the 90-100% oxygen range is still open to question, as shown in the hatched area extending into the zone of unimpaired performance (100). (See Inert Gas, No. 11). A P_{O_2} of 258 mm Hg (5 psia-100% oxygen) has been tolerated in operational space cabins for up to 14 days without performance decrement, though abnormal hematological findings were present which may have been related to the elevated partial pressure of oxygen (92, 100). Table 11-8 covers ground-based experiments in atmospheres of different pressures and compositions.

Charts and nomograms are available relating different total pressures and partial pressures of oxygen and inert constituents to the equivalent alveolar oxygen (89).

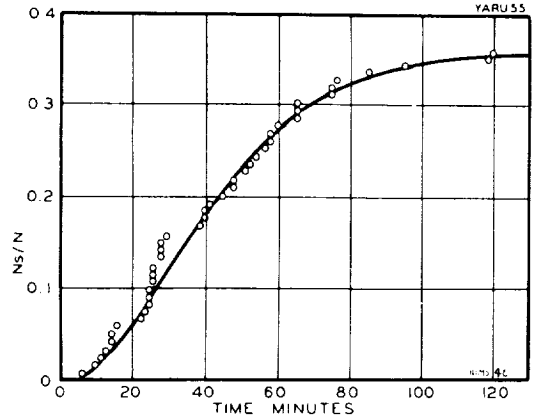
Slow Decompression and Decompression Sickness

During prolonged exposure to atmospheres that contain physiologically inert gases (nitrogen, hydrogen, helium, argon, xenon, and krypton), the body fluids (water and fat) contain amounts of these gases in solution proportional to the partial pressure of the gas in inspired air and to the solubility of the gas in water and fat at body temperature. If the body is subsequently exposed to a much lower barometric pressure, inert gases tend to come out of solution (the phenomenon of effervescence). Oxygen, carbon dioxide, and water vapor also diffuse rapidly into evolved bubbles of gas. Such bubbles, if they form in tissues, may produce pain, especially around the joints. Bubbles within fat cells may cause rupture of the cell walls, allowing fat to enter the circulation. If bubbles form within blood vessels, they are carried to the small terminal vessels of the lungs or the brain where they lodge, cutting off the blood supply of the tissues behind them.

The symptoms caused by evolved gas are known collectively as decompression sickness. This disorder may be mild or it may cause incapacitation. For any one individual, it is unpredictable in its onset and course, though symptoms are rarely seen during the first few minutes of exposure to low barometric pressure. Many factors, among them temperature, muscular work, age, body build, etc., influence susceptibility to decompression sickness and the time course of symptoms (94, 105). A general time course of symptoms experienced in decompression from sea-level air to altitude is shown in Figure 12-2. The fraction of a group having symptoms in a given time interval, is usually at a maximum between twenty and sixty minutes. After two to three hours exposure, very few subjects get new symptoms. The integral of the time curve, a plot of the cumulative fraction of those who have developed any self-judged degree of symptomatology against the time, is an ogive curve having a point of inflection within the same twenty to sixty-minute interval. The ogive curve of Figure 12-2a represents, in most direct

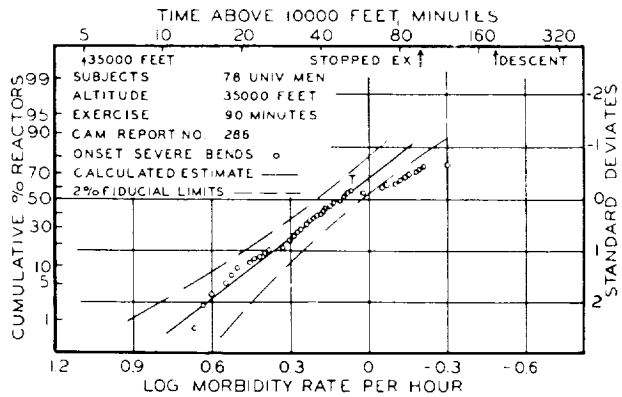
Figure 12-2

Time Course of Symptoms in Decompression Sickness Upon Exposure to Altitude from Prior Equilibration to Air at Sea Level



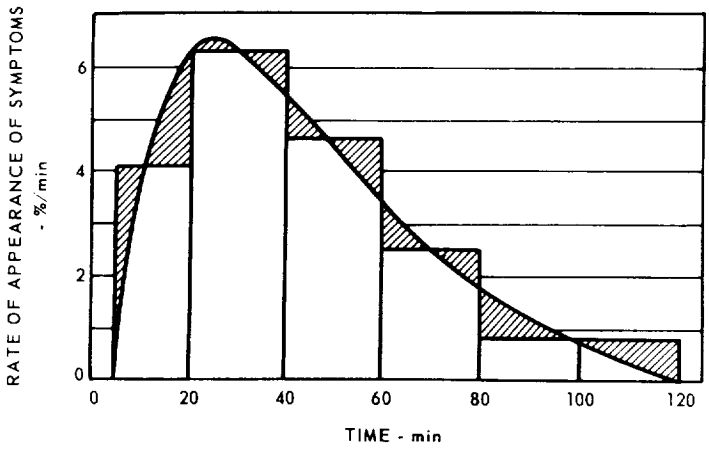
a. The Cumulative Fraction of Group Developing Severe Symptoms of Decompression Sickness as a Function of Duration of Exposure

(After Nims (94))



b. Statistical Treatment of Relationship Between Duration of Exposure and Symptoms of Decompression Sickness

(After Nims (94)



c. Rate of Appearance of New Symptoms

(After Nims[94] from the data of Anthony[2])

and simple way, the quantitative information that can be gained about the group in their reaction to low pressure. The specific shape of the ogive is a function of the final pressure and the secondary factors mentioned above.

Figure 12-2b indicates that if the logarithm of the time of onset of severe bends after exposure to 35,000 ft with exercise is plotted against the cumulative percent reactors expressed in terms of standard deviates, a nearly linear relationship is obtained.

Figure 12-2c shows the percent of exposed subjects per minute experiencing new symptoms (bends of grade 2 or > and chokes) at given times after exposure to 38,000 ft at rest from previous sea level conditions. The curve is thought to reflect the size history of a typical gas bubble in the sensitive tissue (94).

The varied symptoms and pathological physiology of decompression sickness have been reviewed in great detail (1, 44, 102). One can summarize the pathological physiology of the symptom complexes by dividing them into several categories: bends, chokes, skin manifestations, circulatory collapse, and neurological disorders. The relative incidence of the different symptoms varies with the type and partial pressure of the gas of previous equilibration, the level of exercise, and final altitude (102). Relative incidence from work in altitude chambers are available (13, 14).

Bends, the most common symptom, is manifested by pain in the locomotor system. This pain usually begins in the tissue around joints and extends distally along the bone shaft. Pain tends to occur in joints that are being flexed. It is deep and poorly localized with periods of waxing and waning. Relief is obtained by relaxation of the part or application of external pressure to the overlying tissues. Symptoms may spontaneously disappear.

The next most common symptom complex is chokes. Chokes refer to a syndrome of chest pain, cough, and respiratory distress. It usually requires longer altitude exposure than that required for bends. It commences with a burning pain under the breast bone during deep inspiration which is relieved by shallow breathing and gradually becomes more severe and constant. Paroxysms of coughing become more frequent and are followed by cyanosis, anxiety, syncope, and shock.

Skin lesions, causing itching and a red blotchy rash, usually occur only after prolonged altitude exposure and are associated with or presage more serious manifestations of decompression sickness. About 10% of those cases going on to neurocirculatory collapse present previous skin changes. It appears that passage of emboli to the skin is the most probable mechanism.

Neurocirculatory manifestations are the most serious. Cardiovascular symptoms are varied: fainting, low blood pressure, coronary occlusions, heart arrythmias, and shock have all been seen. Rarely, severe and progressive peripheral vascular collapse develops one to five hours post-exposure to altitude. This reaction may or may not have been preceded by fainting. Signs and symptoms of shock with or without neurological findings are seen. Delirium and coma are more common when neurological findings are present. All fatalities following altitude exposure are preceded by this picture of delayed shock. It usually develops in subjects who have experienced severe decompression sickness, especially severe chokes, but may be preceded by few or no symptoms. The types of neurologic symptoms run the gamut of almost every acute neurologic disorder. Convulsions, partial retinal blindness and headaches are the most common.

Several semi-empirical equations have been proposed for rough, first-order, prediction of bends frequency after decompression from atmospheres containing a P_{N_2} other than that of air at sea level (8, 9, 10, 37, 102). These have few other empirical data in their support (10, 34, 37, 55, 68). Unfortunately, decompression from a space cabin involves such conditions. One can assume that a space-cabin atmosphere containing inert diluent should have about 3. 5 psia (180 mmHg) of oxygen and a total pressure of 5 to 7 psia for minimum weight penalty (103). This would allow for 1. 5 to 3. 5 psia of inert

gas. From the point of view of decompression, the lower the equilibrium pressure level of inert gas, the lower the bends hazard upon subsequent decompression to a lower pressure. This would make the cabin with 7 psia 50% inert gas - 50% oxygen more hazardous than one with 5 psia - 70% inert gas - 30% diluent. Prediction of the incidence of bends after decompression from the more hazardous of the mixtures to a space suit pressure of 3.5 psia has been attempted (102). Inadequacy of the empirical data precludes a very precise prediction. The semi-empirical equation of Bateman (8, 9), suggests that after total equilibration to the 7 psia 50% nitrogen - 50% oxygen environment, a well-conditioned astronaut when decompressed to 3.5 psia (35,000 ft) at rest will have less than a 1% chance of experiencing mild, grade I-II bends. If moderate exercise is imposed, the incidence could rise to about 7%. For the general population with only average cardiovascular status and conditioning through exercise, the bends incidence in exercise conditions may be 10-15%. If the space suit pressure could be raised to 5 psia without compromising the mission, the bends incidence should drop by about a factor of 3. Complete equilibration with a 5 psia - 30% nitrogen - 70% oxygen environment and subsequent decompression to 3.5 psia would probably result in no symptoms even with heavy exercise.

In comparison, Figure 12-5 suggests that direct decompression from air at sea level to 3.5 psia presents a more serious hazard. At rest, about 25% of the astronauts would probably experience the bends. Depending on the degree of exercise, from 50 to 100% of the individuals exposed could experience moderate to severe bends. Many would experience chokes and neurocirculatory collapse. Pre-flight or in-flight denitrogenation is certainly an operational requirement in such circumstances.

The decompression hazard prior to the time period of complete equilibration with the space-cabin atmosphere (about 8 to 12 hours) is more difficult to predict. The amount of prior denitrogenation by preoxygenation techniques is a critical factor. Data are available for specific profiles (10, 34, 37). Theoretically, five hours of preoxygenation should reduce the symptom rate to that of the equilibrium condition noted for 7 psia 50% oxygen-50% nitrogen (see below). Shorter periods of denitrogenation possibly dictated by operational restrictions will increase the decompression hazard above this level during the early phases of flight (102).

Presence of inert gases other than nitrogen further complicates predictions of bends, chokes, or neurocirculatory collapse hazards in space cabins. Several theoretical studies of the problem have been made. Both the formation of stable gas micronuclei (10, 37) and rate of bubble growth (102) have been considered as limiting factors in the incidence of symptoms. Both approaches suggest that neon-oxygen mixtures should be safer than helium-oxygen or nitrogen-oxygen as far as bends are concerned. Both indicate that there should be little difference between oxygen-helium and oxygen-nitrogen of the same composition. The few empirical data to the point suggest the helium-oxygen produces slightly more frequent symptoms than oxygen-nitrogen and the symptoms are more difficult to resolve by recompression (10, 34). It should be kept in mind that these experiments were performed under specific M.O.L. profiles in which the body was not

fully denitrogenated and gases were in the unsteady state. Results may well be different for conditions of complete saturation.

It is important that predictions specify whether or not the partial pressure of gas assumed prior to decompression represents equilibrium or non-equilibrium conditions. Age and physical conditioning are also major factors determining incidence. Figure 12-3 represents age dependence of symptoms with no special selection for physical conditioning. Body fat: lean weight ratios are also important (87).

The amount of physical exertion is also important. The effect of exercise rate on incidence of bends after sea-level equilibration in air is seen in Figure 12-4. There is a steady increase in incidence from rest to about 10 deep knee bends every 15 minutes. Incidence varies significantly with the type of exercise.

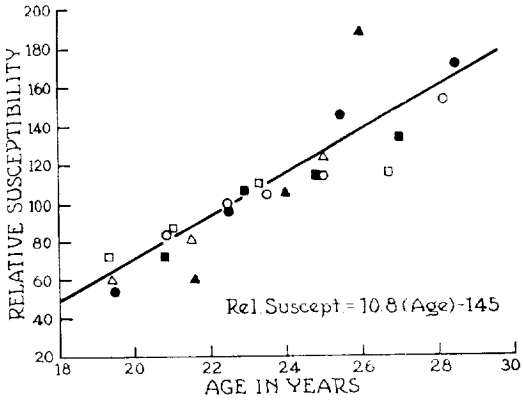


Figure 12-3

Relationship Between Age and Relative Susceptibility to Bends Upon Exposure from Sea Level to Altitude

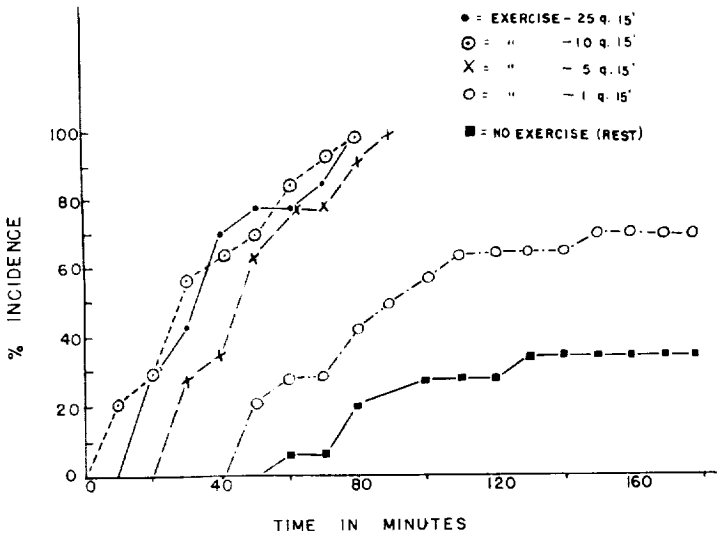
(After Gray [50])

Figure 12-4

Effect of Exercise on Incidence of Bends

Comparison of cumulative incidence of decompression sickness in a group of 14 subjects exposed from sea level equilibration in random fashion to variable degrees of exercise during simulated flights at 35,000 feet (179 mm Hg or 3.5 psia). Exercise was deep knee bends.

(After Ferris and Engle [44])



Protection is afforded by denitrogenation. Total denitrogenation by exposure to 100% oxygen atmospheres for periods of 16 hours or more can be expected to reduce the incidence of bends to zero. Shorter time periods of denitrogenation result in progressively greater incidence of bends. The percent symptoms retained tend to be equal to the percent of residual body nitrogen after previous equilibration with air at sea level. The half time of the second tissue compartment for nitrogen (68-73 minutes) seems to correlate best with the half time of symptom reduction by preoxygenation upon exposure with exercise to 35,000 feet (179 mm Hg or 3.5 psia) (65). For young subjects in good physical condition the half time of nitrogen depletion and incidence of symptoms can be as low as 20 minutes. The dependence of the denitrogenation rates and retained symptoms on age and physical condition in several studies are seen in Figure 12-5. The broken lines represent loss of protection produced by one hour of air breathing after the denitrogenation. Various preoxygenation schedules have been tested in simulation of decompression from space cabins (10, 34, 37, 55, 68).

Denitrogenation schedules for protection against bends caused by exposure to space suit pressures in early phases of flight can be made from Table 12-6 which represents conservative protection factors taken from Figure 12-5. The table is designed to cover groups which eliminate nitrogen slowly. It applies to a suit pressurized at 35,000 feet (179 mm Hg or 3.5 psia) with moderate exercise at altitude. The average nitrogen elimination curves of groups greater than 24 years of age are used in the tables for the category "probable protection." Curve #10 of Figure 12-5 appears in the table as the

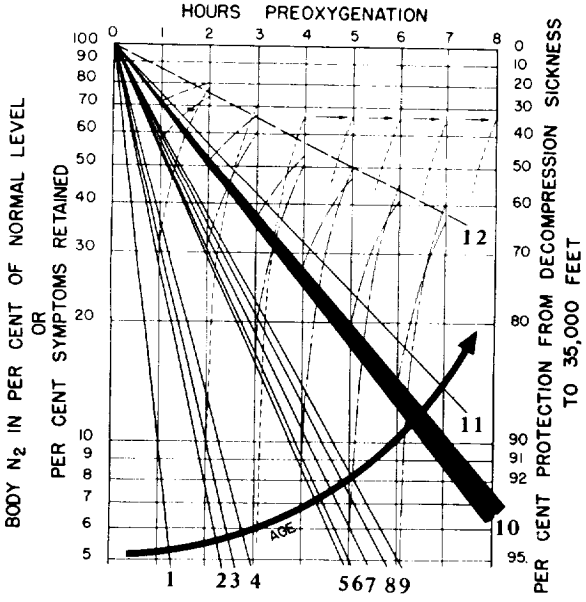


Figure 12-5

Compilation of All Data Bearing on Rate of Protection by Preoxygenation and Rate of Nitrogen Loss from Critical Tissues

Curves 6, 7, and 9 represent data of three different investigators on same age group.

Legend

1. 18 yr old group (fastest curve) - 35,000 ft.
2. 18 yr old group (average curve) - 35,000 ft.
3. <24 yr old group (fastest curve) - 35,000 ft.
4. 17 yr old group (average curve) - 38,000 ft.
5. 27 yr old group (average curve) - 38,000 ft.
6. <24 yr old group (average curve) - 35,000 ft.
7. <24 yr old group (average curve) - 35,000 ft.
8. Mixed group average protection rate - 35,000 ft.
9. <24 yr old group (average curve) - 35,000 ft.
10. <24 yr old group (slowest curve) - 35,000 ft.
11. 35 yr old group (average curve) - 38,000 ft.
12. Single subject (slowest curve) - 35,000 ft.

(After Jones[65])

Table 12-6

Protection[a] of Groups[b] Compared to Ascent Without Preoxygenation

(After Jones[65])

Preoxygenation, hr	Minimum protection, percent	Probable protection, percent
0.5	16	26
1.0	29	45
1.5	41	59
2.0	50	70
2.5	58	77
3.0	61	83
3.5	70	87
4.0	75	91
4.5	79	
5.0	82	
5.5	85	
6.0	86	
6.5	89	
7.0	91	

[a] Zero protection equals incidence of decompression sickness of group without preflight oxygen when ascending to altitude at 4000 feet per minute.

[b] For group prediction and not for individual prediction.

"minimum protection" category. Unless age or nitrogen elimination characteristics of a group are known, prediction should be made with "minimum protection" category. "Protection" is given in percent improvement over the expected condition of preflight oxygenation for that group and ascent to altitude no faster than 4,000 ft/min. For example, if at 35,000 feet a group experiences 70% symptoms, and 50% forced descents with no preoxygenation, after one hour of preoxygenation one would expect from Table 12-6, a "minimal protection" group of:

$$70 \times 0.29 = 20.3\%;\ 70 - 20.3 = 49.7\% \text{ symptoms}$$
$$50 \times 0.29 = 14.5\%;\ 50 - 14.5 = 35.5\% \text{ descents.}$$

Preflight contingencies requiring return to air breathing entail a loss of protection. Table 12-7 represents the protection retained after breathing oxygen for periods of 1 to 7 hours followed by air exposures of 1/2 to 1 hour.

The rate of depletion of inert gas stores in the body after breathing 100% oxygen have been determined for nitrogen and helium. The rate of inert gas elimination follows the exponential equation:

$$dQ_g/dt = k_1 A_1 e^{-k_1 t} = k_2 A_2 e^{-k_2 t} + \ldots + k_n A_n e^{-k_n t} \qquad (1)$$

Table 12-7

Protection[a] Retained When Preoxygenation is Interrupted with Air Breathing

(After Jones (65))

	O_2	Air		O_2	Air		O_2	Air		O_2	Air		O_2	Air		O_2	Air		O_2	Air	
Time, hr.	1	½	1	2	½	1	3	½	1	4	½	1	5	½	1	6	½	1	7	½	1
Minimum protection, percent	29	26	20	50	40	33	64	54	46	75	62	53	82	68	60	86	74	62	91	74	62
Probable protection, percent	45	33	25	70	52	39	83	62	46	91	67	50	95	70	52	97	72	54	97	73	54

[a] Zero protection equals incidence of decompression sickness of group without preflight oxygen when ascending to an altitude of 35 000 feet at 4000 feet per minute.

where: Q_g = the amount of gas lost (cc)

t = time (minutes)

k = exponential time constant of each storage compartment

A = original volume of gas in each exponential storage compartment (cc).

The exponential equation for nitrogen elimination is (65):

$$\frac{dQ_{N_2}}{dt} = 51.2e^{-.462t} + 16.8e^{-.087t} + 10.3e^{-.025t} + 3.3e^{-.0047t} \qquad (2)$$

This is plotted as Figure 12-8a.

The rate of helium elimination is much less certain. Three independent studies have given 3 different equations (11, 42, 66). Uncertainty regarding the early period of elimination is a major cause of difficulty. A desaturation equation defining the fractional rate of helium elimination after a 12 hour saturation period is (42):

$$R_t = 0.25e^{-0.5t} + 0.045e^{-0.135t} + 0.0022e^{-0.025t} + 0.0006e^{-0.0073t} \qquad (3)$$

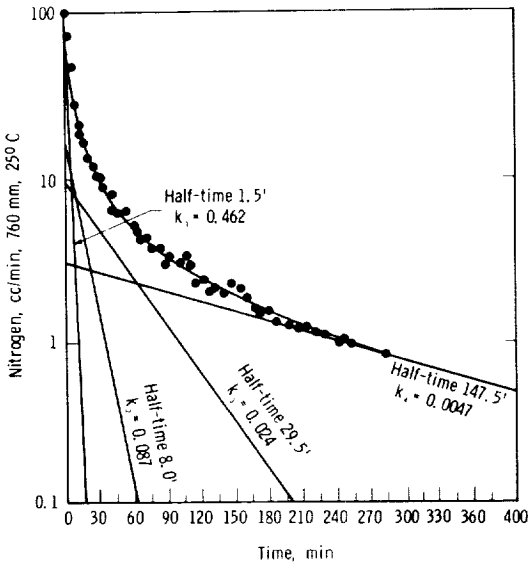
where: R_t is the fraction remaining at any time, t.

Figure 12-8b is the graphic representation of this equation with constants indicated for each exponential.

Further development of recent attempts at theoretical analysis of gas kinetics in diving may allow more definitive predictions of inert gas hazards in the space operations (67, 72, 102, 108, 112). These electrical and pneumatic computer techniques appear particularly promising. Another potential tool in substantiating any theoretical analysis is the ultrasound

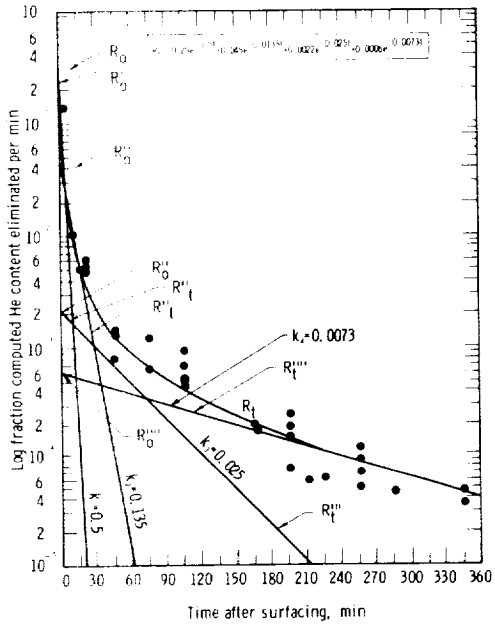
Figure 12-8

Rate of Inert Gas Elimination from Body



a. Rate of Nitrogen Elimination After Switching from a Respiratory Mixture of Air at Sea Level to 100% O_2

(After Jones[65])



b. Rate of Helium Elimination Following 12-Hour Exposure to 1.72 or 2.05 Atmospheres (absolute) Helium In Oxygen

(After Duffner and Snider[42])

technique for bubble detection (53, 88). If the technique can be refined and calibrated it could possibly be used to determine in a more quantitative way the relationship between body movement, tissue supersaturation, the generation of bubbles, and symptoms (102).

A review of the treatment of decompression emergencies in space operations is now available (26). Recompression to the maximum allowable pressure of suit and cabin in 100% oxygen would probably offer the best immediate solution (86). In space chambers the standard U.S. Air Force approach is recommended (87). Supportive treatment of severe emergencies has been reviewed (26).

Protection of astronauts in space by elastic fabric or foam sponge suits is currently under study (54, 96A, 119).This approach may counter the bends problems, and, at the same time, reduce the inconvenience and reduction of mobility presented by current pressure suits.

Several equations have been proposed for rough, first-order, prediction of bends frequency after decompression from atmospheres containing a P_{N_2} other than that of air at sea level (10, 102). These have little empirical data in their support. Unfortunately the physical condition of the subjects and the equilibrium conditions of inert gas saturation have varied in different studies

(9, 10, 34, 102). The data of Tables 11-2 and 11-3 and Figure 11-4 of Inert Gas (No. 11) may be used in the calculation of these equations.

The slow leakage of gas from space cabins is covered in Inert Gas (No. 11).

Rapid (Explosive) Decompression

Rapid decompression of spacecraft or suits can result from accidental trauma or meteorite penetration. Post-meteoritic disruption of space cabin walls and accompanying fire and blast hazards has been reviewed (101). Decompression of space suits by meteorites is a constant hazard during EVA operations (101). Damage to the body by low-velocity impacts is covered under Impact in Acceleration (No. 7). Little is known of hypervelocity impact effects in humans or animals (101). Protection against meteoroid penetration of space suits is accomplished by a combined thermal and anti-meteoroid coverall (85).

Antimeteoroid Coveralls

In the Gemini G4C extravehicular space suit, the extravehicular coverlayer consisted of an outer protective layer of high-temperature-resistant (HT-1) nylon, a layer of nylon felt for micrometeoroid protection, seven layers of aluminized Mylar and unwoven Dacron superinsulation, and two additional layers of high-temperature nylon for micrometeoroid shock absorption. The meteoroid protective coverlayer design used on the Gemini IV mission was proof tested with simulated meteoroids. The Gemini G4C suit configuration was qualified to provide a 0.999 probability of no penetration, P_o, of the bladder. In a system pressurized to 3.7 psig, samples of 4 by 4" swatches of the meteoroid coverlayer on the bladder were impacted with simulated meteoroids of cork and epoxy, glass and porosilicate in the 5 to 27 km/sec range. Since these projectiles approximate the meteoroidal energy that is absorbed by the coverlayer, a corresponding P_o for a 10-minute exposure was determined. The exposure was for a near-Earth orbit and 25 ft^2 of surface area on the space suit. A pyrex glass sphere 274 microns in diameter at a velocity of 6 km/sec approximates the energy necessary to obtain a P_o of 0.999 for a 10-minute exposure

Samples of lexan and merlon polycarbonate visor material were pressurized to 3.7 psig and impacted with glass spheres accelerated to hypervelocity with the AVCO RAD light gas gun. The projectile impact energy was progressively increased, until the sample was perforated or a leak occurred. An examination of the targets revealed that the 0.098-inch-thick merlon and lexan withstood the impact of a 0.0156-inch glass sphere at a velocity of 6 km/sec without spall or leakage. This projectile energy, when extrapolated to meteoroidal velocity and density, corresponded to a P_o of 0.99993 for 135-minute exposure. The need for reduced coverlayer bulk to improve unpressurized suit mobility and pilot comfort was noted.

The G4C space suit assembly used in the Gemini VIII mission was similar to the one used in the Gemini IV mission. However, the configuration of the micrometeoroid protective layers of the extravehicular coverlayer was modified to utilize two layers of neoprene-coated nylon in lieu of the nylon felt and 6-ounce,HT-1 nylon,micrometeoroid layers. Also, the extravehicular pilot used integrated pressure thermal gloves in lieu of the pressure gloves and overgloves used for Gemini IV. The gloves were designed to protect the hands from micrometeoroids and to prevent conductive heat transfer through the glove palms caused from touching surfaces with temperatures ranging from 250° to -150° F. Structurally and functionally, the gloves were similar to the standard intravehicular pressure gloves with a pressure bladder, a restraint layer, and a wrist connector. A 1/8-inch-thick, flexible, insulating, silastic material was provided on the palm side of the glove for conduction insulation. Micrometeoroid protection was through additional layers of fabric used in the layup of the glove. The micrometeoroid testing of the new coverlayer material demonstrated a P_o of 0.999 for worst-case conditions. The extravehicular space suit components were not used for EVA because of early termination of the mission, However, the reduced coverlayer bulk resulting from the change in micrometeoroid protective materials improved the unpressurized suit mobility for the intravehicular operations.

The addition of the Astronaut Maneuvering Unit (AMU) to the flight plan for Gemini IX-A required extensive modifications to the coverlayer of the G4C space suit. The lower forward-firing and downward-firing AMU thrusters impinged upon the legs of the suit. Temperatures as high as 1300° F were possible at the AMU thruster impingement areas on the suit surface. Since the HT-1 high-temperature nylon, which is normally used for the coverlayer, is not recommended for continuous use at temperatures above 500°F, new suit materials were required. A stainless steel fabric was incorporated into the legs of the suit coverlayer to protect it from the heat generated by AMU thruster impingement. Analysis and testing also indicated that the temperatures inside the thermal insulation layers of the coverlayer would exceed the melting temperature of the aluminized Mylar. Aluminized H-film was developed and found to be adequate for the temperatures expected and, when separated by layers of fiberglas cloth, worked well as a high-temperature thermal insulation. Eleven layers each of aluminized H-film and fiberglas cloth were incorporated into the legs to provide thermal protection during AMU operations. A standard extravehicular coverlayer layup was utilized for the upper torso and the steel outer cover with aluminized H-film and fiberglas cloth was used as thermal insulation for the legs. No meteoroid penetrations of this system were recorded.

The coverall of Gemini X and XI suits were similar to the Gemini VIII. The Gemini XII space suit used by the pilot was a slightly modified version of the one used for the Gemini IX-A mission. The stainless steel fabric on the legs was replaced with high-temperature nylon, and four layers of the aluminized H-film and fiberglas cloth superinsulation were deleted from the suit legs. The coverlayer thermal layup was quilted to the first layer of micrometeoroid protective material. A rectangular pattern was quilted over the torso area, which strengthened the thermal layer and reduced the possibility of tears or rips in the aluminized H-film and aluminized Mylar layers. The suit operated well; no meteoroid punctures were recorded.

The physiological response to rapid decompression must be considered from several points of view: The time of useful consciousness, damage to the lungs by explosive decompression, and the ebullism syndrome.

Time of Useful Consciousness After Rapid Decompression

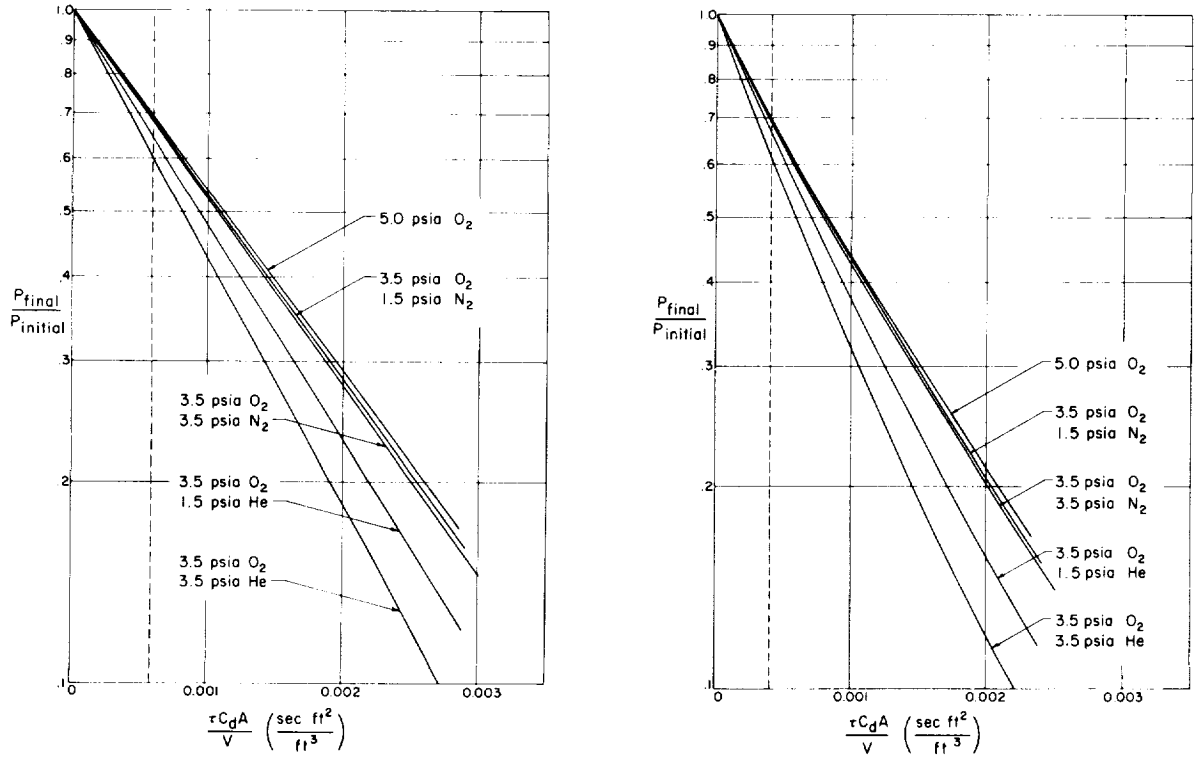
The time of decompression (τ), after puncture of disruption of a cabin wall needed to attain a given ratio of final to initial pressure (P_f/P_i), is a function of the orifice coefficient (C_d) and the ratio of orifice area (A) to cabin volume (V) (17, 103). Figures 12-9a and b represent this relationship for several different gas mixtures suitable as space cabin atmospheres (102, 103). The equation assumes sonic orifice flow for isothermal and isentropic decompression. A sample calculation for isothermal flow using Figure 12-9a: For a hole 1/2 inch in diameter, an orifice coefficient (C_d) of 1, and a psia oxygen can be determined from Figure 12-9a by using the ratio 3.5 to 5.0 or 0.7 to give:

$$\frac{\tau C_d A}{V} = 0.000575 \left(\frac{\text{sec ft}^2}{\text{ft}^3} \right)$$

and

$$\tau = \frac{0.00575 \times 770}{\frac{\pi \times (0.25)^2}{144}} = 325 \text{ sec}$$

Figure 12-9
The Sensitivity of Rapid Decompression to Composition of Atmosphere
(After Roth[103], adapted from Boeing[17])



a. Isothermal Decompression

b. Isentropic Decompression

The time to reach minimum tolerable partial pressure of P_{O2} can be calculated by the factors of Figure 12-1. This reduces the available time considerably. (Table 12-10 represents the time in minutes required to meet minimum tolerable total pressures as determined by minimal P_{O2} levels for 1/2 inch and 3/4 inch holes under isothermal and isentropic conditions with five proposed atmospheres.) It can be seen that in all cases, the oxygen-nitrogen mixture at 7 psia takes the longest times and 100 percent oxygen takes the shortest time to reach the critical condition. The larger the hole, the less the absolute difference between mixtures. Pure oxygen gives more than twice the time of useful work than do the other gas mixtures at 5 psia. The lower the partial pressure of inert gas, the less time required to reach both endpoints and the greater the difference between the two criteria. From the point of view of the human subject, Table 12-10 presents the more valid endpoint than just pressure. At equivalent composition and pressure, nitrogen has a slight advantage over helium.

Table 12-10

Decompression Time to Minimum Tolerable Total Pressure as Determined by Minimum Acceptable P_{O_2}; (Cabin Volume = 770 Ft^3; Orifice Coefficient = 1)

(After Boeing[17])

Leak mode	3.5 psia O_2 3.5 psia N_2 7.0 psia	3.5 psia O_2 3.5 psia He 7.0 psia	3.5 psia O_2 1.5 psia N_2 5.0 psia	3.5 psia O_2 1.5 psia He 5.0 psia	5.0 psia O_2
	Decompression time, min				
Isothermal – 1/2-inch hole	6.17	4.72	2.25	1.93	5.42
Isentropic – 1/2-inch hole	4.54	3.22	1.62	1.35	3.95
Isothermal – 3/4-inch hole	2.75	2.1	1.0	.86	2.41
Isentropic – 3/4-inch hole	2.03	1.42	.72	.59	1.75

There may be operational significance between the maximally divergent times of 6 minutes and 2 minutes for the 1/2-inch hole with isothermal flow. If the mission requires at least 6 minutes for donning an emergency suit in a high-risk phase, this difference may well be critical in the selection. The probability of a penetration producing such a hole size is obviously a major mission-specific factor to be considered.

There are several other minor considerations in the area of fast-flow systems. These are the maximum airlock dumping and repressurization times during extravehicular operations and the maximum rate of cabin pressure dumping during fire emergencies. The dumping of airlock and cabin would, of course, follow the more isentropic type of flow. The faster the flow through the maximum orifice available, the more advantageous the gas mixture. One would therefore have to weight the advantage of having a more rapid dumping capability for a suited crew against a less-rapid emergency dumping after accidental puncture with an unsuited crew.

The repressurization of an airlock from a vacuum to the pressure of the main compartment is most rapidly accomplished by opening a valve between the two chambers. In most cases, the pressure and temperature of the main compartment is maintained constant by the gas feed system and the compression will be close to isothermal because of the great flow turbulence in the

airlock. The flow across the valve starts off as a supercritical pressure ratio and then becomes subcritical when

$$P_c/P_{lk} = \left(\frac{2}{\gamma+1}\right)^{\frac{\gamma}{\gamma-1}} \tag{4}$$

where P_c = cabin pressure
P_{lk} = lock pressure

The approximate time required to recompress a lock isothermally from vacuum, τ_t, can be determined for air of $\gamma = 1.4$ by the equation

$$\tau_t = \frac{V}{130\, C_d A}\sqrt{\frac{m}{T_{lk}}} \tag{5}$$

where m = average molecular weight of the gas
T_{lk} = is the absolute temperature of the lock
γ = ratio of specific heats.

This aspect of a space mission will be critical only when a crewman must be retrieved most rapidly through a lock to a cabin. Since the relatively small volume of the lock suggests that the minimum time for recompression will not in any practical way limit the survival potential of the crewman, the effect of atmospheric composition should have little practical effect on the survival. The difference in time, measured by seconds, which will be given the entering crewman by an optimum gas mixture does not appear to warrant a thorough analysis of the problem in the present context. Such an analysis is available (30). That the gas-specific factor will probably not be critical is indicated by their calculation from equation (5) that a lock of 40 ft^3 can be isothermally pressurized by air to 99 percent of the main compartment pressure through a valve of only 0.58 $in.^2$ in 30 seconds. Doubling the area of the valve can reduce this time to about 15 seconds. Since the time required is proportional to the square root of the molecular weight, substitution of air (molecular weight = 29) by the proposed mixture of lowest molecular weight, helium-oxygen mixture at 7 psia (molecular weight = 18), will reduce minimum compression time by only a few seconds. For larger lock systems, the number of seconds to be saved will increase as will the physiological significance of the savings. However, the valve size can be increased to meet this demand in a large lock.

One must also consider the airlock pumping weight penalties. The airlock may be pumped into a separate storage tank or into the main compartment. The effect of atmosphere composition on this penalty is currently under study (109). Data are also available on a new elastic recovery principle in the design of airlocks (23).

One must be aware of the time of useful consciousness following decompressions lasting several seconds. Following rapid decompression to an ambient P_{O_2} equivalent to altitudes of about 25,000 feet or above, consciousness is rapidly lost (84, 97).

Figure 12-11 represents the mean and minimum times of useful consciousness available when air or 100% oxygen are being breathed at sea level pressures before and during the decompression.

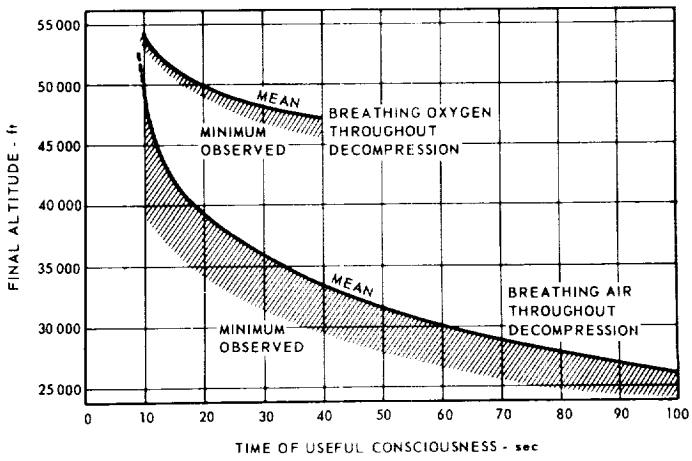


Figure 12-11

Minimum and Mean "Times of Useful Consciousness" Following Rapid Decompression of Humans Who Are Breathing Either Air or Oxygen Throughout Decompression

(After Blockley and Hanifan[16], after data of Luft[83] and others

The "time of useful consciousness" becomes shorter with increasing altitude until a minimum time is reached. This minimum is reached at about 46,000 feet (106 mm Hg or 2.04 psia) when air is breathed throughout the decompression, or, about 52,000 feet (79 mm Hg or 1.53 psia) when oxygen is breathed throughout the decompression. There is a "critical time of exposure" during which an individual must breathe an adequate partial pressure of oxygen if continuous consciousness is to be preserved. This time also reaches a minimum with increasing altitude (3, 24, 83). Oxygen must be given within 7 secs in order to preserve continuous consciousness in subjects decompressed from 8,000 feet (564 mm Hg or 10.91 psia) to 40,000 feet (141 mm Hg or 2.72 psia) in 2.5 sec. The "critical time of exposure" should not exceed 5 to 6 sec in rapid decompressions (2 sec) to altitudes above 52,000 feet (79 mm Hg or 1.53 psia). Specific symptoms resulting from oxygen lack or hypoxia may be found in Oxygen-CO_2-Energy (No. 10).

Treatment of hypoxic emergencies resulting from exposure to vacuum is covered below under ebulism (26, 99).

Lung Damage from Explosive Decompression

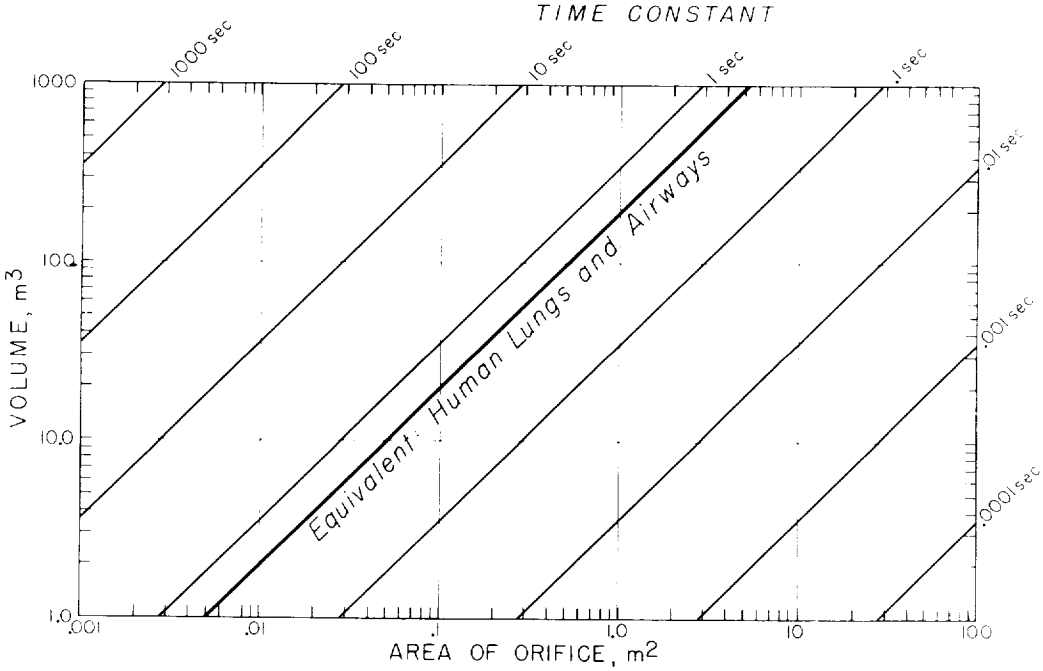
Sudden disruption of a cabin wall or a space suit may decompress an astronaut or test subject rapidly enough to damage his lungs. The problem has been recently summarized by two reviews from which much of the following material is taken (79, 99).

The severity of mechanical effects on the body in rapid decompression is dependent on the change in absolute pressure, the ratio of initial to final pressure, and the rate of decompression. The latter can be defined rather precisely on the basis of physical theory if the pressure conditions, the volume of the cabin or suit and the size of the aperture are known or can be assumed

(46, 48, 51,90). In the presence of humidity, the decompression is neither an adiabatic nor an isothermal process, but is polytropic in character. The rate of flow through the orifice may be of subsonic or sonic velocity, according to the pressure ratio across the orifice. If the critical ratio of approximately 2 to 1 is exceeded, the escape flow will be constant at the speed of sound regardless of how high the pressure head may be. The initial rate of change in pressure is determined by the absolute magnitude of the initial cabin pressure. For all practical purposes, the complex factors that define the decompression transient can be resolved into two principal determinants (51). The firstof these, which sets the absolute time scale of decompression, will be referred to as the time constant (t_c)

$$t_c = \frac{V\,(m^3)}{A\,(m^2) \cdot C\,(m/sec)} \qquad (6)$$

It is defined by the ratio between cabin volume (V) and the effective area of the decompression orifice (A). The velocity of sound (C) is introduced as a characteristic of flow that eliminates the effect of density. It will be seen that t_c must appear in units of time, all other units canceling out. The time constant is independent of pressure. The chart in Figure 12-12 is a graphic solution of equation (6) relating cabin volume and effective orifice to the time constant in metric units.



The volume of the pressure cabin relative to the effective area of the decompression orifice determines the time constant of decompression. For the respiratory tract this depends on the lung volume and the flow resistance of the airways at the time of decompression.

Figure 12-12

Time Constants of Explosive Decompression

(After Luft[79])

The second determinant is the pressure factor (P_1) derived for a polytropic process under subsonic or sonic conditions of flow. P_1 is a function of the initial cabin pressure (P_i) and the final pressure of equilibrium with the environment (P_f), and is independent of the absolute pressure (51).

$$P_1 = f \frac{P_i}{P_f} \tag{7}$$

The values for P_1 can be read for any desired pressure ratio from the curve in 12-13a. The total duration of decompression (t_d) is the product of the time constant (t_c) and the pressure factor P_1.

$$t_d = t_c \cdot P_1 \tag{8}$$

The relationships expressed in equations (6) and (8), which have been verified in numerous experiments, are convenient for estimating the decompression time on the basis of cabin volume and the configuration of windows, doors, or canopy for various cabin pressures at altitude. Similarly, the volume to orifice ratio and the time constant of any decompression situation can be estimated if the elapsed time of decompression and the pressure ratio P_i/P_f are known.

Under vacuum conditions, the duration of decompression becomes extremely long because the final equalization of pressure is very slow. Under these circumstances, the initial part of the transient where the rate of decompression is constant (constant rate time) is more meaningful, as far as biological effects are concerned, than the total duration of decompression. As shown on Figure 12-13b the line of initial rate of change is extended until it intersects the ambient pressure P_{ao}. The point of intersection marks a time which is evidently related to the initial rate of pressure change and the pressure difference. This "constant rate time" (t_{cr}) can be calculated from the time constant (t_c) and another pressure factor (P_3) which may be read from the curve so designated on Figure 12-13a for the appropriate decompressure ratio:

$$t_{cr} = t_c \cdot P_3 \tag{9}$$

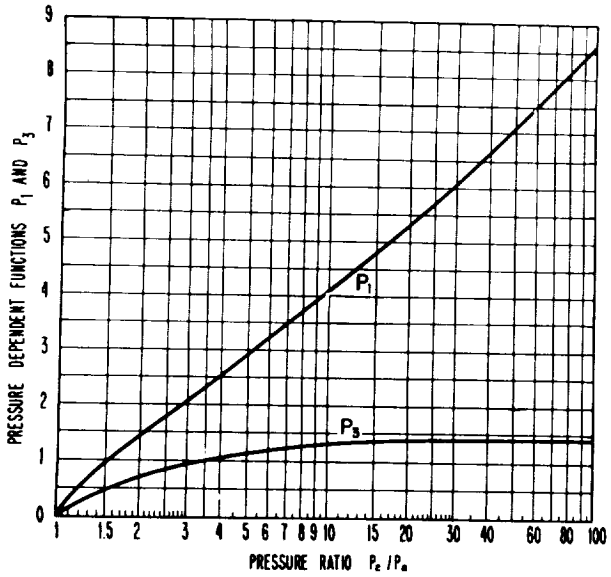
If an individual were decompressed from an initial cabin pressure, P_i, to a final pressure, P_f, at altitude with closed airways in the absence of any change in his lung volume,the pressure in his lungs, P_L, would remain equal to P_i, and the pressure gradient, ΔP_L, sustained by his lungs and chest would be equal to the total pressure difference of decompression.

$$\Delta P_L = P_L - P_f = P_i - P_f \tag{10}$$

On the other hand, if the gas in his lungs could expand without constraint, as in a frictionless piston, its volume would increase from V_i to V_f until P_L became equal to P_f. The relative gas expansion, RGE, assuming isothermal conditions with water vapor pressure at 47 mm Hg would be (78)

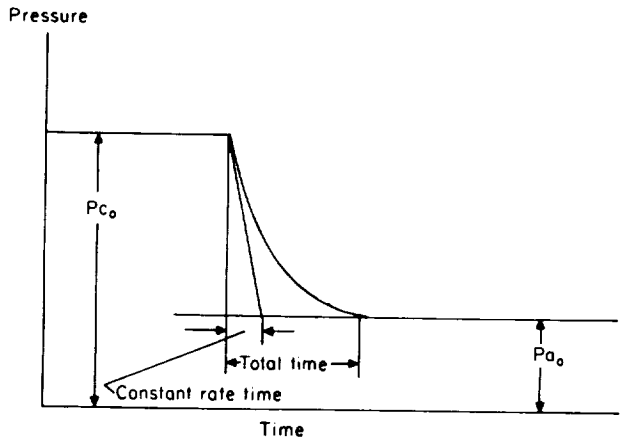
Figure 12-13

Correction Factors for Explosive Decompression to Low Pressures



a. The Pressure Function P_1 for the Total Time of Decompression and for the "Constant Rate Time (P_3) as Dervied from the Pressure Ratio (P_c/P_a) or (P_i/P_f)

(After Bancroft [6])



b. Definition of Constant Rate Time t_R

(After Haber and Clamann [51])

$$\frac{V_f}{V_i} = \frac{P_i - 47}{P_f - 47} = RGE \qquad (11)$$

The lung is neither a rigid container nor a frictionless piston, but an elastic container with limited capacity. The pressure difference across the lungs and chest will tend to expand their contents toward a maximal intact volume, V_{max}, or beyond. The virtual pressure in the lungs, P_L, at the moment in which the maximal intact volume is reached, is estimated by modifying equation (11) accordingly.

$$\frac{V_{max}}{V_i} = \frac{P_i - 47}{P_L - 47} \qquad (12)$$

and solving for P_L,

$$P_L = \frac{V_i}{V_{max}} (P_i - 47) + 47 \qquad (13)$$

The pressure difference, ΔP_L, is found by substituting equation (13) for P_L into equation (10):

$$\Delta P_L = \frac{V_i}{V_{max}} (P_i - 47) + 47 - P_f \qquad (14)$$

It is apparent from equation (14) that when the initial and final pressures of decompression are given, the volume of gas trapped in the lungs relative to the total capacity is the factor determining the critical pressure gradient. According to the animal experiments and human experience, rupture of the lungs is liable to occur when ΔP_L exceeds 80 mm Hg (64, 79, 95, 106). Counterpressure exerted by the chest cage when the lungs are passively distended to their full capacity (relaxation pressure) explains the fact that excised lungs disrupt at a pressure of only 50 mm Hg. Furthermore, when an animal's trunk is bound with inelastic fabric or laid in a plaster cast, trachael pressures as high as 180 mm Hg are tolerated without discernible damage to the lungs (95). These findings point to the fact that high pressure in the lungs is dangerous only if it is permitted to expand pulmonary tissue beyond its tensile limits. In the act of coughing, intrapulmonic pressures of more than 150 mm Hg are tolerated frequently without untoward effects, in the absence of pulmonary pathology. In contrast to the process of passive inflation, the pressure pulse of a cough is the result of active muscular effort, which actually reduces lung volume by compressing its gas content.

By means of equation (14) one can estimate whether the critical pressure for ΔP_L will be exceeded for decompressions of known initial and final pressure with closed airways. If ΔP_L, calculated from equation (14) is less than 30 mm Hg, then the decompression in question would not expand the lungs from V_i to V_{max} and, therefore, would not be dangerous. The initial and final pressures for which the critical overpressures of 80 mm Hg would be reached in the lungs must be calculated for three different lungs volumes: full expiration (Ex), full inspiration (In), and for the normal respiratory position around the midlung volume. The probability is very high that inadvertent decompression would occur during normal respiratory excursions, and it is reasonable to assume a value of 0. 55 for V_i/V_{max} in equation (14) for most instances.

Evaluation of damage risk to the lung during space operations in the case of breathholding has been reviewed using these relationships. The pressure gradient which exists across human lungs and passively distended chest wall during an "explosive" decompression to a vacuum occurring while respiratory passages were closed was calculated for internal pressures of 7 psia and 5 psia which are currently considered for spacecraft and 3. 7 psia for space suits. Three different lung volumes prior to decompression are considered: full inspiration (V_i/V_{max} = 1. 0), the normal end expiratory position (V_i/V_{max} = 0. 55), and full expiration (V_i/V_{max} = 0. 25). These data are presented in Table 12-14. It is interesting to note that all pressure gradients under these conditions are over the previously stated critical level of about 80 mm Hg. Therefore, an "explosive" decompression in a vacuum while respiratory passages are closed is considered a very great hazard from the standpoint of serious lung injury.

Table 12-14

Pressure Gradients Across an Astronaut's Lungs and Passively Distended Chest During "Explosive" Decompression in Space with Closed Glottis

(After Busby[26], from the unpublished calculations of Luft

Pressure gradients ΔP_L calculated for different ambient atmospheric pressures. (P_i) and lung volumes (V_i) prior to decompression to a vacuum (P_f = 0).

$\frac{V_i}{V_{max}}$	ΔP_L at P_i = 7.0 psia (362 mm Hg)	ΔP_L at P_i = 5.0 psia (259 mm Hg)	ΔP_L at P_i = 3.7 psia (191 mm Hg)
1.0	362 mm Hg	259 mm Hg	191 mm Hg
0.55	220 mm Hg	164 mm Hg	121 mm Hg
0.25	126 mm Hg	100 mm Hg	83 mm Hg

In explosive decompression during normal respiration, the case is more complex (79, 81, 82). If the time characteristic of the human lung and airway is greater than the time characteristic of the pressure suit or cabin in which an individual is confined during the decompression, a transient differential pressure will build up between the lungs and ambient atmosphere. This is illustrated diagrammatically in Figure 12-15.

The heavy line in Figure 12-12 represents the time characteristic of the human lung with open glottis on a background of the volume-to-orifice ratio. There is a critical V/A ratio of the cabin or suit relative to this ratio of

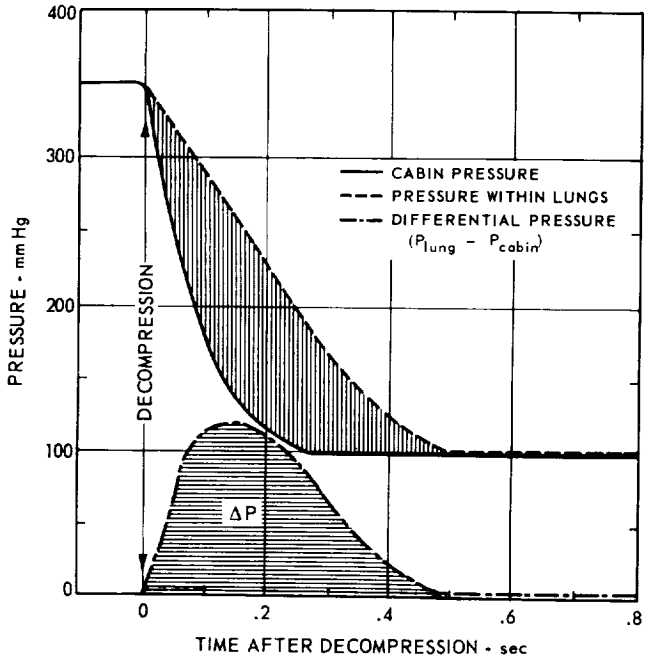


Figure 12-15

Time Characteristics of Overpressure in the Lungs

(Adapted from Luft[80] by Billings and Roth[15])

the human respiratory tract determining the threshold for injury or death. Another factor that influences the transthoracic pressure transient is the pressure ratio (P_i/P_f). It can be shown mathematically and empirically that if decompression takes place over the same pressure difference, but at higher altitude where the pressure ratio is greater, the amplitude of pressure differential of Figure 12-15 remains the same, but the duration of the transient is longer (79, 81). This means that the area under the differential pressure curve which represents the impulse in terms of

$$\frac{\text{force (dyne) x time (sec)}}{\text{area (cm}^2\text{)}} \qquad (15)$$

is a function of the decompression ratio. Unfortunately, there are no data correlating lung damage directly with impulse. The shape and duration of a blast wave is certainly a factor in predicting damage from overpressure (22 , 98).

The conclusions to be drawn from these model analyses can be summarized as follows: 1) The maximal possible amplitude of the transmural pressure in the lung model is equal to the pressure difference of decompression ($P_i - P_f$). 2) The fraction of the total pressure difference effective in the lung is dependent on the V/A ratio in the lung to that of the suit or cabin. 3) The pressure ratio of decompression (P_i/P_f) determines the force x time integral or impulse for any given amplitude of the transthoracic pressure transient and, therefore, the duration of a critical overpressure.

In addition to the perturbing effect of water vapor in the lungs, the most important shortcoming of a rigid model is that it fails to simulate the elastic expansion of lungs and chest in decompression, as would occur according to equation(14)for isothermic conditions, with a corresponding drop of pulmonary pressure. In dogs, expansion is not apparent before 10 msec (117). In man, the time lag is probably even greater, since it is a function of the mechanical impedance of the lungs and chest which increases with body size (41, 106).

According to the cinematographic data, decompression of the lungs takes place in three phases. The first is under essentially isometric conditions with no change in volume, owing to the inertia of the system. The highest transthoracic pressures are probably attained during this phase in which the lungs are comparable to a rigid bottle. In the second phase, the pressure is attenuated due to expansion of the chest and also to the continuing escape of gas through the airways. In the third phase of maximal expansion, the conditions are again isometric until the overpressure is dissipated and the lung volume decreases. Structural damage is conceivable during the first and second phases, when the peak pressure creates powerful dynamic forces opposed by the inertia of the system. In a medium consisting of components with widely different densities, such as the organs in the chest, differences in acceleration under the impulsive pressure loading could result in shearing and spalling lesions similar to those encountered in blast injuries in the vicinity of explosions (29, 107). During the third phase of maximal expansion of the lungs, the mechanism of injury would be comparable to that assumed for decompression with closed airways, namely, rupture of tissues at the limits

of their tensile strength. Penetration of gas bubbles into the bloodstream most likely take place when the lungs are fully expanded and a high gradient is created between the intrapulmonic pressure and that in the pulmonary veins and left atrium (106). Air embolism may be facilitated at this time at the sites of lung damaged in the first two phases of decompression.

Experimental substantiation of this model is difficult. Experimental procedures often do not exclude the influence of hypoxia and decompression sickness or of boiling phenomena on the experimental animals; and more often no effort is made to discriminate between the many factors involved by keeping one or more of these constant. Nevertheless, certain notable relationships emerge that support the following concept. There can be no doubt that the rate of decompression is a decisive variable in the mortality of animals (79). This holds true from initial pressures of 1 atm., at differential pressures of greater than 630 mm Hg (0.83 atm.), and decompression times from 0.630 to 0.0014 second. Since the decompression time is also influenced by the pressure ratio of decompression which differs considerably, the V/A ratio is preferable as a characteristic of the rate of decompression. In all tests where V/A was 15 m^3 per m^2 or more, all animals survived. A significant number of fatalities appears when V/A was 3.3 m^3 per m^2, and the LD_{50} corresponded to a V/A of 1.1 to 1.2 m^3 per m^2. In the only investigation where 100 percent mortality was produced, a special decompression device with a V/A of .12 m^3 per m^2, was used (72). In decompression of such extreme rapidity, there can be very little escape of gas from the lungs before the full pressure gradient becomes effective and the lungs and chest are overdistended with a pressure load practically as great as if the airways had been completely closed. If this were true, one would expect some fatal injuries to occur under the same pressure conditions as found in decompression with closed airways. According to equation (14) solved for decompression from sea level with closed airways at midlung volume, a critical ΔP_L of 80 mm Hg can be predicted when the final pressure is lower than 359 mm Hg or 0.47 atm. When rats were exposed to increasing pressure differences from an initial pressure of 735 mm Hg with a V/A of .12, an increasing number of fatalities were observed whenever the final pressure was less than 368 mm Hg (0.48 atm) (72). Conversely, the fastest decompressions were innocuous when this pressure range was not exceeded. Convincing evidence that the mechanism of fatal injury is overdistention of the lungs and not the pressure pulse per se was obtained by exposing animals with an artificial pneumothorax to extreme decompression and finding less trauma than in the untreated (72).

With slower rates of decompression and open airways only a fraction of the total gradient of decompression will come to bear upon the lungs as more gas has had time to escape before they are fully distended. As pointed out for the rigid model above, the amplitude of the pressure transient in the lungs is dependent on the V/A ratio of the lungs and airways relative to that of the suit or cabin system. From intrathoracic pressure transients recorded in man it has been estimated that the human lungs and airways correspond to a V/A of approximately 180 m^3 per m^2. For dogs, this V/A ratio is 100 (117). This indicates that the dogs may tolerate somewhat lower cabin V/A ratios than humans. However, this difference may well be due to the different experimental techniques used to obtain the values. These figures provide a cue for safety limits in the permissible rate of decompression, since

decompression to unlimited altitudes would not give rise to disruption of the lungs if the V/A of the cabin were no less than the human equivalent.

Experience with human exposure to decompression at low cabin V/A ratios is very limited (12, 28, 39, 40, 62, 76, 79, 99, 113). Well-documented, danger-zone decompressions with open glottis have been limited to those recorded in Table 12-16. It can be seen that only the first exposure

Table 12-16

Rapid Decompression Tolerated by Man

(After Luft[79])

Ref.	No.	Altitude, feet	P_i,* mm Hg	P_f,† mm Hg	$P_i - P_f$, mm Hg	P_i/P_f	Time, sec	V/A‡	ΔP_L§, mm Hg
Sweeney 113	10	27,000-45,000	253	112	141	2.23	.005	1.0	48
Sweeney 6	15	8,000-35,000	565	179	386	3.16	.090	13.4	153
Döring 40	13	9,800-49,100	526	90	436	5.83	.230	23.0	220

[a] ΔP_L is the overpressure which would occure in the lungs if the airways were closed at midlung volume; critical pressure is 80 mm Hg (Eq. 14).

of Sweeney would have had a cabin V/A ratio (1 m^3/m^2) well within the expected lethal range. Even under closed airways at midrespiratory volume, the pressure ratio P_i/P_f would have been small enough in the first case with low V/A ratio to have prevented the critical overpressure of 80 mm Hg from being reached (79).

Figure 12-17 is a summary curve which represents a rough evaluation

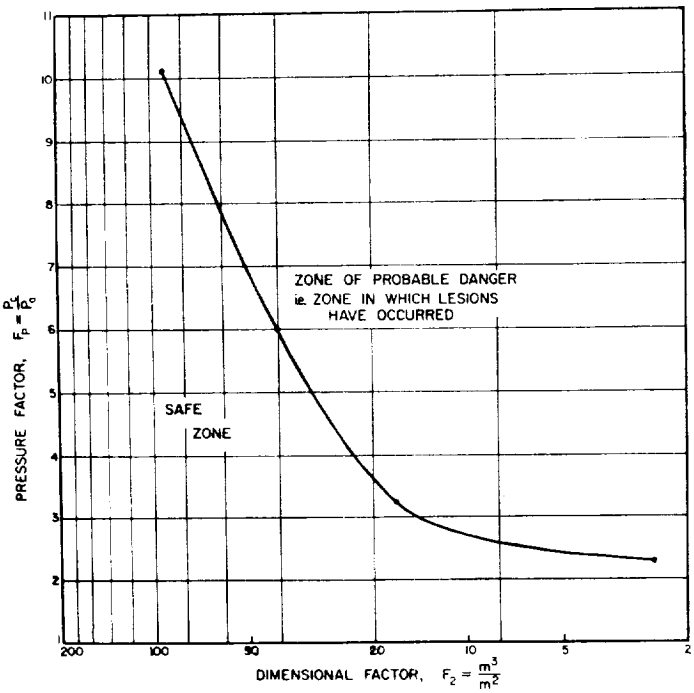


Figure 12-17

Curve Derived from Human and Animal Data Defining the Zones of Safety and Probable Danger in Explosive Decompression with Open Glottis (See text)

(After Fryer[28] from the data of Violette[117])

of the relative dimensional and pressure-ratio factors defining the zone of possible injury under glottis-open conditions. The curve does have some shortcomings. For instance, it is doubtful whether it is permissible to plot animal and human data on a common figure. Again, the degree and actual aetiology of damage in animals in many series of experiments is not fully known. Comparison with animal data (72) and the data of Table 12-16 shows that the curve is conservative enough for a first approximation of the safe zone. Lack of direct data regarding V/A ratios of animal experiments makes the degree of conservatism difficult to assess. In those humans where lung damage was sustained, there is inadequate information about the degree of breathholding and the fraction of vital capacity during exposure. These factors preclude adequate evaluation of the zone above the curve in Figure 12-17, especially in the pertinent zone of high P_c/P_a ratios of vacuum exposure. Another factor controlling the extrapolation of animal data to humans is the relative inertia of the chest wall during phase 2 of the decompression. The time required to move the chest wall should roughly scale directly as the one-third power of the mass of the animal (21). This will determine the rate of application of the tensile forces on the critical lung structures. This factor has not been considered in the above discussion.

In decompression of a space cabin, the composition of atmosphere is a factor in lung damage. For the present, the use of gas other than 100% oxygen is most unlikely in extravehicular suit assemblies. However, there is a possibility that improvement in joint design may permit development of hard suits operating under relatively high pressures with inert gas mixtures. The flow of gas through the respiratory tract is a critical factor during "explosive" decompression (103). A rigid analysis of the flow factor has been made using a mathematical model of the fluid-mechanical response of the thoracoabdominal system to blast overpressure and "explosive" decompression (22). An analysis of the gas-dependent factors in this model leads to the conclusion that the rate of pressure change in the lung with respect to ambient $\left(\frac{dP}{dt}\right)_{t=0}$ is a function of the product of the reciprocal of the square root of the average molecular weight of the gas M and a gas-flow factor involving the specific heat ratio γ. This relationship is shown in the following equation

$$\left(\frac{dP}{dt}\right)_{t=0} \sim \frac{1}{M^{1/2}}\left(\gamma\left[\gamma\left(\frac{2}{\gamma+1}\right)^{\frac{\gamma+1}{\gamma-1}}\right]^{1/2}\right) \qquad (16)$$

The lower the rate of pressure change in the lung with respect to ambient, the more dangerous is the atmosphere. This same relationship would define the hazard from external blast overpressure. For isothermal processes, the value of $\gamma = 1$ can be used. For adiabatic processes the values of γ are obtained from the C_p/C_v ratios (103). The ratios for the inert gases lie in the 1.67 range, except for nitrogen at 1.4. The value for oxygen is 1.4.

It is still not absolutely clear whether adiabatic or isothermal processes predominate in the lung in "explosive" decompression or blast overpressure.

The rapidity of the process suggests adiabatic conditions. It must be remembered, however, that the alveoli of the lung present a large surface for heat exchange and high humidity. This would allow for rapid condensation of water vapor to counteract the adiabatic cooling. The temperature change in the lung during "explosive" decompression has been found to be minimal (58). Sensor lag obviously complicates the measurement to an unknown degree A value of $\gamma = 1.2$ for air has been used as a polytropic compromise in an unknown situation (22) but it is felt that the isothermal process probably predominates (45). In the analysis of the space-cabin situation, calculations were made for the currently proposed environment of 50 percent inert gas and 50 percent oxygen. Both the isothermal and 50 percent isothermal-50 percent adiabatic specific heat ratios are presented in Table 18. For the isothermal condition, $\gamma = 1$. Table 12-18 represents the calculations of

Table 12-18

Relative "Explosive" Decompression and Blast Overpressure Hazards from Atmospheres at 7 Psia with 50 Percent Inert Gas and 50 Percent Oxygen

(After Roth (103))

Factor	Gas mixture in cabin						
	$He\text{-}O_2$	$Ne\text{-}O_2$	$A\text{-}O_2$	$Kr\text{-}O_2$	$Xe\text{-}O_2$	$N_2\text{-}O_2$	O_2
$1/M^{1/2}$	0.34	0.20	0.17	0.15	0.13	0.18	0.18
γ(50 percent adiabatic)	1.25	1.25	1.25	1.25	1.25	1.20	1.20
Isothermal expansion ($\gamma = 1$) $\left(\frac{dP}{dt}\right)_{t=0}$	.34	.20	.17	.15	.13	.18	.18
Relative hazard index ($N_2\text{-}O_2 = 1$)	.53	.90	1.1	1.2	1.4	1.0	1.0
Polytropic expansion (50 percent adiabatic) $\left(\frac{dP}{dt}\right)_{t=0}$	.26	.15	.13	.11	.10	.13	.13
Relative hazard index ($N_2\text{-}O_2 = 1$)	.50	.87	1.0	1.2	1.3	1.0	1.0

$\left(\frac{dP}{dt}\right)_{t=0}$ for these gas mixtures and the relative hazard index with nitrogen-oxygen = 1. The relative hazard index is calculated from the reciprocal of the $\left(\frac{dP}{dt}\right)_{t=0}$ factor. The nitrogen-oxygen and the 100 percent oxygen (7 psi) atmospheres would have the same degree of hazard. It can be seen that the major gas factor is $1/M^{1/2}$. The thermodynamic nature of the expansion has little effect on the relative hazard of the inert gas. Helium-oxygen appears to be about 0.5 as hazardous as nitrogen-oxygen or 100 percent oxygen; neon-oxygen appears to be about 0.9 as hazardous. The relative degree of hazard then increases with increasing molecular weight for the other gases. It should be pointed out that these are the maximum differences expected. Most second-order factors would probably tend to decrease the relative molecular-weight dependence. For example, the rate of gas escaping from the cabin is also dependent upon molecular weight. However, when one reviews the cabin V/A ratios required for lethality in animals, it is evident that the cabin

pressure will have essentially reached ambient well before the flow of gas out of the respiratory tree has ceased. Any overlap of these flows would reduce the dependence upon molecular weight. Therefore, a prediction can be made that the smaller the hole, the less gas-dependent is the hazard. The practical significance of gas-specific factors in lung damage is open to question.

Another variable to be considered in evaluation of the hazard of decompression is the presence of oxygen mask or respiratory equipment which may superimpose an artificial "glottis" over the normal one and increase the effective V/A ratio of the subject (82). This should not be a consideration in current full pressure suits where large plastic bubble helmets surround the facial area.

The several mathematical models of the thorax-abdominal system for evaluating the hazards of air blast overpressure and explosive decompression damage to the lungs require more empirical study for confirmation of their validity under the several variables of the current problem (22, 73). When fully validated, these models could be used to give a finer prediction of the hazards under the specific internal pressure profiles presented by disrupting cabins and space suit assemblies.

From the above discussion of critical V/A ratios, it can be calculated that with glottis open, an astronaut in a 50 cu ft cabin of Project Mercury would have to sustain an acute disruption of at least 1 sq foot of cabin wall to receive damage to a normal lung. In the case of suit disruption, the problem is more complex (99). Except for joint and helmet areas, the laminated fabric of the soft suit and metal shell of the hard suit are reportedly safe from acute disruption. Verbal reports from the engineers interviewed suggest that the "fail-safe" nature of neck seal and probably the wrist, thigh, and ankle seals, relegate the chances of acute disruption to a very low category. The waist seal of the hard suit is the most vulnerable site of disruption. The laminated fabric lining the bellows systems reduces their vulnerability to catastrophic disruption.

The time characteristic anticipated from disruption of soft and hard suit seals is shown on Table 12-19. The residual suit volumes were calculated from frontal areas of the suit sections. Orifices were of annular type calculated by subtracting from the cross section area of the suit at the disrupted seals, the cross section of the body segment enclosed. From Table 12-19 and the data presented above, it can be concluded that acute catastrophic disruption of the neck and wrist seals of the soft and hard suits and disruption of the neck, thigh, and ankle seals of the hard suit may well lead to lung damage in a previously normal, suited subject in a vacuum chamber or in space. This is true even for open-glottis conditions. The hazard is intensified if the glottis is closed and breath is held. Disruption of a glove finger in both suits and the portable life support (PLSS) umbilical in the hard suit would probably not lead to lung damage if the glottis were open, but may lead to difficulty if the breath were held during the decompression. Disruption of the chamber umbilical in the hard and soft suits and PLSS umbilical in the soft suit, particularly at the entrance ports to the suit, could possibly lead to lung damage under open-glottis conditions. However, the case is less clear

Table 12-19

Time Characteristics of Explosive Decompression Predicted for Acute Disruption of Space Suits During EVA and in Test Chambers (See text)

(After Roth [99])

Soft Suits	Residual Suit Volume (cc)	Orifice Area (cm^2)	V/A Ratio (meters)	t_c (sec) (Fig. 12-13)	P3	t_{cr} (sec)
Neck Seal (PLSS)	26,000	295	0.88	0.0025	1.45	0.0036
Wrist Seal (PLSS)	28,000	60	4.67	0.0134	1.45	0.019
Chamber umbilical hose	25,500	7.9	32.3	0.093	1.45	0.14
PLSS umbilical hose	28,000	2.8	100.	0.286	1.45	0.41
Fingers (PLSS)	28,000	1.2	233.	0.670	1.45	0.97
Hard Suits						
Waist Seal (PLSS)	35,200	810	0.435	0.00125	1.45	0.0018
Neck Seal (PLSS)	70,600	561	1.25	0.00359	1.45	0.0052
Thigh (PLSS)	64,000	177	3.6	0.0104	1.45	0.015
Ankle (PLSS)	75,000	168	4.46	0.0128	1.45	0.019
Wrist Seal (PLSS)	75,000	54	13.9	0.0399	1.45	0.058
Chamber umbilical hose	71,000	7.9	89.8	0.255	1.45	0.370
PLSS umbilical hose	75,000	2.8	268.	0.77	1.45	1.1
Fingers (PLSS)	75,000	1.2	620.	1.78	1.45	2.6

than the previous one. Disruption of the umbilical hoses at a distance from the entrance port would lower the probability of damage (99, 114). It is clear from this analysis that all seal areas should be designed for slow propagation of disruptive processes. The advisability of preparing therapeutic devices and facilities for handling explosive decompression emergencies would, strictly speaking, depend on the actual reliability of the suit seals under question, and the assumed degree of conservatism used in extrapolating from animal data obtained at pressure regimes different from the case in question. However, these uncertainties suggest that accidents should be anticipated and plans made accordingly.

During the second phase of maximal expansion of the lungs and chest wall, disruption of the tissues would occur as their tensile strength is exceeded. This would also occur during decompression with closed airways. These structural defects lead to pulmonary hemorrhage and edema as well as to pneumoperitoneum and pneumothorax. During the third phase of maximal expansion, penetration of bubbles into the blood stream takes place as a high pressure gradient is formed between the alveoli and the pulmonary veins. Gas emboli enter the blood stream and pass to the arterial circulation. Such embolization may continue to occur upon inspiration for some time after the decompression.

Death is usually caused by hemorrhage from the disrupted lung or by introduction of gas emboli into the venous side of the pulmonary circulation and subsequent infarction of critical sites in the systemic circulation. Exposure to the vacuum for several minutes can lead to further lung damage and to the ebullism syndrome (see below). The pathological physiology and optimum treatment of these syndromes has been recently reviewed (26, 99). All human accidents and the single fatality are reviewed in detail.

One factor which must be considered in the selection of astronauts or test subjects is prior pathology in the lungs. Plugs of mucus in the bronchioles reduce the local V/A ratio and increase the distal transalveolar pressure impulse during the decompression. Such plugs were found in the one reported human death after explosive decompression (112). Those factors which weaken the alveolar walls would increase the hazard of exposure. The same conditions predisposing to spontaneous pneumothorax would be expected to increase the chances of parenchymal damage in decompression. The value of routine and special x-ray examination of subjects and other selection procedures has been analyzed (99).

Treatment of pneumothorax, pneumomediastinum, lung contusion, and aeroemboli resulting from explosive decompression has been recently covered (26, 99). The latest U.S. Navy tables for recompression therapy are recommended (19, 116, 123, 124). The objective of the recompression method is to expose bubbles to the optimum pressure gradient for efficient and rapid resolution while still permitting maximum oxygenation of tissues with circulation impaired with bubbles. Oxygen here has the effect of preserving function in ischemic vital areas and also interrupting the insidious cycle of ischemia, hypoxia, edema, obstruction, and further ischemia. An important collateral benefit is the absence of further inert gas saturation of the patient under recompression with pure oxygen. The volume of any spherical bubble decreases inversely with applied pressure. For chamber therapy, the treatment tables stop recompression at 165 feet gauge pressure because relative decrements of volume with increasing pressure become insignificant past 1/6 of the original bubble volume, while increasing the depth past 6 atmospheres (absolute) enormously increases the difficulties of subsequent decompression back to normal pressure, especially for an injured patient. The geometry of the situation dictates that the radius of the bubble decrease as the cube root of the applied pressure. The diminution of the radius, therefore, begins to become inefficient at shallower depths than 165 feet.

Bubble resolution in decompression sickness depends both on a reduction in size with recompression and on the elimination of inert gas from the bubble and from the surrounding tissue. In severely injured patients treated with recompression to 165 feet, inert gas exchange is grossly impaired in areas distal to obstruction. Bubbles may form during subsequent decompression in areas of tissue injury which have inadequate inert gas elimination rates due to circulatory impairment. The avoidance of further inert gas uptake by compressing only to 60 feet and the acceleration of inert gas elimination by oxygen breathing may overbalance any small decrease in bubble radius from further compression to 6 atmospheres. In patients for whom treatment has

been delayed and in whom vascular obstruction from edema and thrombosis may be of an importance equal to or greater than that from persistent bubbles, the hyperbaric oxygenation given immediately in treatment is believed to be of substantially more benefit than increased bubble compression with compressed air breathing.

The rate of recompression is another factor to be considered. The chambers at the NASA, MSC, Houston, can reach 7 psia within 25 seconds from onset of decompression (26). This time is certainly adequate for handling the emergencies outlined above. Ideally, the repressurization gas should be oxygen. However, the engineering problems and hazards involved in rapid recompression of a huge chamber with oxygen make this approach unfeasible (91, 101). Very rapid recompression can also be hazardous. Compression of animals over 0. 5 atmospheres pressure difference in periods less than several milliseconds can lead to the same type of lung injury as seen in explosive decompression (71, 74). Restricting any emergency repressurization from vacuum through 7 psia to periods longer than 5 seconds should avoid permanent damage to the eardrums in most individuals (4, 56, 110, 111). In case of explosive decompression of space suits during EVA, the space cabin and suits should be raised to their highest design pressures (26). In most cases, this total will not exceed about 10 psia.

Treatment of the accompanying ebullism is covered below.

Ebullism

Exposure to altitude where the total ambient pressure approaches 47 mm Hg, the effective vapor pressure of fluids at body temperature, gives rise to the profuse evaporation associated with formation of vapor bubbles in tissues, blood vessels, and body cavities (102, 118, 122).Selection of vapor site is determined by such local factors as temperature, hydrostatic pressure, tissue elasticity, solute concentration (Clausius-Clapeyron factors) and presence of gas nuclei. As would be expected from these considerations, the large venous channels at the center of the body temperature core are sites of early bubble formation resulting in vapor lock of the heart. Subsequently, vapor pockets forming in the loose subcutaneous tissue are often seen, as are vapor bubbles in the aqueous humor of the eye and in the brain. In looking at the ebullism syndrome, one must also keep in mind damage to the body from hypoxia and lung pathology from explosive decompression (99).

There have been no exposures of the total unprotected human body to pressures in the ebullism range of significant duration. Exposure of only the hand to pressures of 5 to 30 mm Hg results in marked swelling with latencies which range from 0. 5 to 10 minutes (122). The reason for such a range is not clear and may be peculiar to the experimental condition. The wrist and fingers can be flexed and extended through about 50 % of the normal range of motion. There is no pain associated with the swelling but only paresthesias in the area. Sudden recompression results in the swelling resolving at a pressure of 87 to 141 mm Hg in less than 0. 5 minutes. Disruption of gloves in pressurized suits would probably lead to this condition.

The survival time of man exposed to near-vacuum conditions without pressure suit protection must be extrapolated from recent animal data. Early studies of explosive decompression of animals to very low pressures focused on the pathology to the lungs (7, 47, 58, 59). Even in the absence of pneumothorax, atelectasis appeared more severe than after explosive decompression to lower altitudes, probably because of vapothorax. Another key factor is suggested by the finding that only those animals in which respiration had ceased before recompression showed complete atelectasis. It is conceivable that water vapor entering the alveoli displaces the gas content and then recondenses on recompression to cause severe alveolar collapse. Otherwise the lesions were not much different from those of explosive decompression to lower altitudes.

More recent studies of ebullism cover the survival and functional capabilities of animals exposed to altitudes above 100,000 ft (8 mm Hg) (5, 7, 32, 43, 63, 69, 70). Decompression up to 130,000 ft (2 mm Hg) result in violent evolution of water vapor with swelling of the whole body of dogs. Preliminary results indicate that dogs kept as long as 90 seconds at 2 mm Hg did not present a single fatality. The animals were unconscious, gasping, and had bradycardias down to 10 beats per minute from the normal rate of 159 beats per minute, possibly a vagal response due to distortion of the mediastinal structures resulting from sudden expansion of the thorax. Most also had paralysis of hind limbs, yet after 10 to 15 minutes at sea level, they walked about normally. Animals exposed beyond 120 seconds did die frequently. Autopsy of surviving animals exposed less than 120 seconds demonstrate damage to the lung in the form of congestion, petechial hemorrhage, and emphysematous changes, the damage increasing with duration of exposure. Petechial hemorrhages and emphysema were more severe when decompression to altitude occurred within 0.2 seconds than when a decompression time of 1 second was used (43). Denitrogenation appears to reduce the incidence and severity of lung damage, possibly by reducing the inert gas entering the vapor bubble in the right heart (102). For the exposures of more than 120 seconds, gross examinations of the brain and other organs showed increasing amounts of congestion and hemorrhage with time at altitude. Occasionally dogs will die of cardiac arrythmias possibly triggered by aeroemboli to the coronary arteries (see below).

Exposure of squirrel monkeys resulted in similar findings (104). Many of the survivors of 90 seconds exposure showed various defects in locomotion, hearing, vision, and food retrieval, and lost more weight than the control groups. Of interest, however, is the fact that among the survivors there was no loss of proficiency in learning set (69, 70). Chimpanzees can survive without apparent central nervous sytem damage (as measured by complex task performance) the effects of decompression to a near vacuum for up to 2.5 minutes and return within approximately 4 hours to baseline levels of functioning. One chimpanzee with intra-cerebral electrodes was at 2 mm Hg for 3 minutes. His time of useful consciousness was 11 seconds. Cortical silence started at 45 seconds; and subcortical, at 75 seconds. Two months later he still showed mild organic residua with performance and behavioral changes. It is of interest that in one case of death in these chimpanzees, no indication of disruption of the alveoli, alveolar ducts or bronchi was noted

on postmortem. Death was attributed to failure in the conducting mechanism of the heart.

For planning emergency procedures following decompression of protected humans to vacuum or near-vacuum conditions, a maximum survival time of 90 seconds should be used. Times of useful consciousness of about 10 seconds can be anticipated.

From the animal studies it can be inferred that upon prolonged exposure, cardiovascular collapse will be most precipitous and a major cause of death. After exposure to sub-ebullism altitudes, there is a dramatic fall in blood pressure followed by rebound with subsequent anoxic failure. Almost immediately after decompression to an ambient atmospheric pressure at which ebullism can occur, vapor bubbles form at the entrance of the great veins into the heart, then rapidly progress in a retrograde fashion through the venous system to the capillary level. Venous return is blocked by this "vascular vapor lock." This leads to a precipitous fall in cardiac output, a simultaneous reduction of the systemic arterial pressure, and the development of vapor bubbles in the arterial system and in the heart itself, including the coronary arteries. Systemic arterial and venous pressures then approach equilibrium in dogs at 70 mm Hg (96). At ebullism altitudes, one can expect vapor lock of the heart to result in complete cardiac standstill after 10-15 seconds, with increasing lethality for exposures lasting over 90 seconds. Vapor pockets have been seen in the heart of animals as soon as 1 second after decompression to 3 mm Hg (63). Upon recompression, the water vapor returns immediately to liquid form but the gas components remain in the bubble form. When circulation is resumed, these bubbles are ejected as emboli to the lungs and periphery. Cardiac arrythmias often occur as do focal lesions in the nervous system (7, 27, 32, 33, 43, 69). These are probably a result of infarct by inert gas bubbles. The problem is aggravated by the concomitant generalized hypoxia. Cooling of the blood to 9°C by rapid evaporation in the alveoli while circulation is still intact, may delay the cardiac and cerebral response to ischemic hypoxia (4, 69, 96). The short cooling time precludes a more effective temperature drop.

Alteration of the gaseous environment may affect the ebullism syndrome. Data are available on the nature of the gas bubbles in the vascular system. Analyses of the changing gas compositions of subcutaneous vapor pockets by different investigators have given equivocal results (102). At first there appears to be a rapid conversion of liquid water to the vapor phase which reaches a peak at one minute and continues at a slower rate for several minutes. There is an initial rush of carbon dioxide, nitrogen, and oxygen into the pocket, but carbon dioxide and the nitrogen soon become predominant. If one can extrapolate to the more lethal vaporous bubbles in the great veins and right side of the heart, it would appear that the rate of growth and subsequent stability of bubbles after recompression would probably depend on the permeation coefficient or product of solubility and diffusivity ($\alpha_{blood} D_{blood}$) of the inert gas passing from the blood to the vapor bubble (102). Neon would enter the bubble more slowly than nitrogen, helium, or argon (order of increasing gas permeation . Once emboli have been ejected by the heart and have landed in the arterial system, however, the rate of resolution of the

bubble during therapeutic maneuvers will be inversely proportional to the ($\alpha_{blood}D_{blood}$) factor. Gas emboli containing only oxygen are safest, followed, in increasing order of hazard, by those of argon, helium, nitrogen, and neon. This would also hold for gas emboli entering the circulation from the injured lung.

It should be remembered, however, that presence of 100% oxygen in the space suit does not eliminate the problem of aeroemboli. Comparative effects of O_2, CO_2, N_2, and He emboli have been studied (38). Different volumes of these gases were injected into the internal carotid arteries of dogs prepared surgically so that the gas went only into the cerebral circulation without shunting to the extracranial arteries. Oxygen was tolerated without mortality but all the dogs had clinical or anatomic evidence of cerebral infarction. Carbon dioxide was well tolerated in doses up to 1. 5 ml., but morbidity and mortality occurred with 2 ml. Nitrogen and helium foam produced effects similar to those of air foam, and morbidity and mortality results were comparable to the results obtained with air embolization. The physical basis for this difference is determined by the comparative resolution rates, and ultimately, by the permeation coefficients of different gas bubbles (72, 102). Occlusion of the circulation probably prevents the unsaturation of hemoglobin and reduces the size of the potential oxygen sink in the immediate surround of the intraarterial bubble. One must therefore anticipate that oxygen emboli will be somewhat less dangerous but cannot be neglected. For equal amounts of helium and nitrogen in the cerebral circulation, the hazard is probably equal. Empirical data are needed on gas effects on aero embism and ebullism.

Treatment of ebullism and related syndromes has received little formal attention. From the review of the pathological physiology of ebullism, it is apparent that in the treatment of this syndrome in space operations or in chambers one must consider damage to the lungs from exposure to cold (96), from hypoxia, and from gas embolization arising in the large veins and right side of the heart. One must also consider arterial gas embolization through atrial septal defects or vascular shunts in the lung. Fortunately, therapy of contusive damage to the lung covers damage to the lung from ebullism (99). In chambers on Earth, the Trendelenberg position may decrease the embolization of the lungs (99). Aspiration of gas from the right ventricle in the case of cardiac arrest may aid in restoration of the circulation and avoid further damage to the lung by gas emboli. Treatment of arterial or venous gas emboli after ebullism should be no different than that following lung disruption. Compression therapy suggested for the latter should have no deleterious effects on the former. As in contusive damage, progressive pulmonary edema and atelectasis must be anticipated after prolonged exposure of the lung to vacuum. In view of the atelectatic tendency, prolonged treatment with 100% oxygen should be used only when cyanosis and oxygen unsaturation of the blood are present in cases uncomplicated by obvious embolization (100).

Exposure to hypoxic environments for longer than 3 or 4 minutes may produce several of the post-hypoxic syndromes during the treatment period. These have recently been reviewed in great detail (26). It may be difficult to distinguish post-hypoxic cerebral edema from brain syndromes associated with massive air embolization. Failure of a patient to respond to recom-

pression therapy (persistent coma or delerium) should raise the consideration of post-hypoxic-cerebral edema. Dehydration therapy would then be in order. It is suggested that mannitol be given intravenously in concentrations up to 20% with doses up to 200 Gm per 24 hr period (26, 75). Hypothermia may also be used for the post-hypoxic syndrome to minimize damage to the brain elements and break up the vicious edema-hypoxia cycle (26). It has been recommended that body temperatures between 30°C (86°F) and 32°C (89.6°F) be attained with suppression of shivering by chlorpromazine (52.125). The value of steroid-antihistamine combinations for post-hypoxic cerebral edema is yet to be determined (104).

The bizarre electrocardiographic patterns seen in dogs exposed to vacuum range from extrasystoles to idioventricular rhythms and ventricular fibrillation (25, 31, 96). Cardiac dilatation (from trapped gas), hypoxia, vaporization of intracellular water, exposure to cold blood, and air emboli may all probably play a role. Electrical defibrillation and not just anti-fibrillatory drugs should be used to reverse the ventricular fibrillation and tachycardia (49). Lidocaine is effective if P.V.C.'s or ventricular tachycardia occur after electrical defibrillation. The dose of lidocaine is 1-2 mg/kilogram body weight given intravenously in 1-2 minutes, repeated if necessary once or twice at 20 minute intervals. For idioventricular rhythm with rates greater than 150 per minute,as ventricular tachycardia, the treatment should be lidocaine. Idioventricular rhythm with a rate of less than 100 per minute implies that ventricular escape has occurred and treatment with lidocaine is contraindicated. On Earth, a transvenous pacemaker should be used. This maneuver, by increasing the ventricular rate, will usually suppress the ectopic focus, but if the attempt is not successful, cardioplegic drugs may then be used with a greater measure of assurance. Idioventricular rhythms with rates between 100 and 150 per minute present a difficult problem One can try treatment with lidocaine but if dysfunction of the conduction system appears to be present, an artificial pacemaker should be used (93).

Blast Overpressure

The hazard of blast overpressures from meteoroid penetration or explosions within a spacecraft results from direct blast damage to the ears and lungs and secondary damage to the body from non-penetrating missiles, penetrating missiles, and sudden impact against large structures (101, 121). The time-geometry of the effective blast wave is critical in determining levels of injury expected from any overpressure. Such factors as the incident wave form (rise time, peak pressure, duration , and pulse tail-off), dynamic pressures, reflected waves and their timing, ambient pressures, positioning of subject relative to blast direction, geometry of surroundings, etc., all are critical (97, 121). The term" effective pressure" will be used to cover the overpressure equivalence of all these conditions. For space operations, the ambient pressure is most important in biological scaling (22, 35, 36). For lethality, lung damage, and possibly eardrum damage, the overpressure in psia for 50% effect (P_{50}) at any ambient pressure, (χ) relative to sea level (SL) is:

$$P_{50\ (x)} = P_{50(SL)} \left(\frac{x}{14.7} \right) \qquad (17)$$

The scaling equation in Figure 12-21d includes this pressure factor.

Lethality

Blast overpressures produce their lethal effect by primarily disrupting the alveolar and vascular structures of lungs causing hemorrhage and respiratory embarrassment (101, 120). Gas emboli entering the circulation from disrupted pulmonary veins can pass to coronary or cerebral circulation and cause death by infarction of the tissues. The maximum tolerable overpressure is a function of the pulse duration and geometry of exposure, including such factors as body position relative to the blast wave and to reflecting structures.

The threshold, 50% and 100% lethality levels for short and long duration blast are seen in Table 12-20. For atypical or disturbed wave forms of "long duration," tolerance can be estimated to increase by about a factor of two for pressures rising to a maximum in two "fast" steps and by a factor of 3 to 5 for wave forms rising smoothly to a maximum in 30 or more msec.

Figure 12-21a to c are preliminary estimates of human survival after air blast exposures of different duration and geometry of incidence. Figure 12-21d presents scaling criteria and animal data from which human thresholds were derived. These figures show some of the geometric factors which must be considered in evaluating the blast hazards resulting from explosions of boosters on launch pads or of space cabins in orbit.

Lung

From animal studies it appears that death, if it does occur, will overtake more than 90% of the animals within the first 30 minutes after exposure (120). This can probably be applied to man as well. Threshold overpressures for lung damage and probably secondary emboli are seen in Table 12-20. Figures 12-21a to c give the threshold overpressures for lung damage under different geometries of incidence and duration of exposure.

Damage to the lungs by overpressures resulting from meteoroid penetration of spacecraft has been reviewed (102). Overpressures of less than 1 msec with rise times as short as 15 microseconds may be anticipated. Effects of these overpressures are complicated by the flash-oxidation of molten metal resulting from penetration of the cabin wall by the meteoroids. Inhalation of hot metallic vapors may increase lung damage brought about by the blast overpressure. More quantitative work on this problem is needed.

Damage to the lung from excessively rapid recompression from a vacuum is covered on page 12-30.

Ear

Damage to the eardrum is also a significant factor in blast injury (113, 120, 121). The maximum overpressure is a major blast parameter to be considered, but the rise time and duration of pulse are also significant. The slower the rise time, the greater the peak overpressure needed to disrupt the drum. The longer the duration of overpressure, the greater the percent of disruption at the same peak overpressure (120). Tentative estimates for short-and long-duration effects in man are shown in Table 12-20. The exact scaling of ambient pressure and wave-form effects for man are less certain for eardrum rupture than for lethality.

Figure 12-22 represents an estimate of population response to peak overpressure with drum disruption as an endpoint.

Figure 12-20

Tentative Criteria for Primary-Blast Effects in Man Applicable to "Fast"-Rising Air Blasts of "Short"-Duration (3 msec) and "Long"-Duration (Plateau > 20 msec)*

Critical Organ or Event	Maximal Effective Pressure (psia at sea level)*	
	Short	Long
Eardrum Rupture		
Threshold	6	6
50 Per Cent	18	18-25
Lung Damage		
Threshold	37-49	12-14
Severe	100 and above	–
Lethality		
Threshold	120-140	37-52
50 Per Cent	160-220	51-70
Near 100 Per Cent	250-310	70-98

Effective pressure can be the incident, reflected, or incident plus dynamic, depending on one's geometry of exposure and the location of the explosion. The data on lung damage and lethality correspond to those of Figure 12-21; the data on eardrum rupture (short duration), to Figure 12-22.

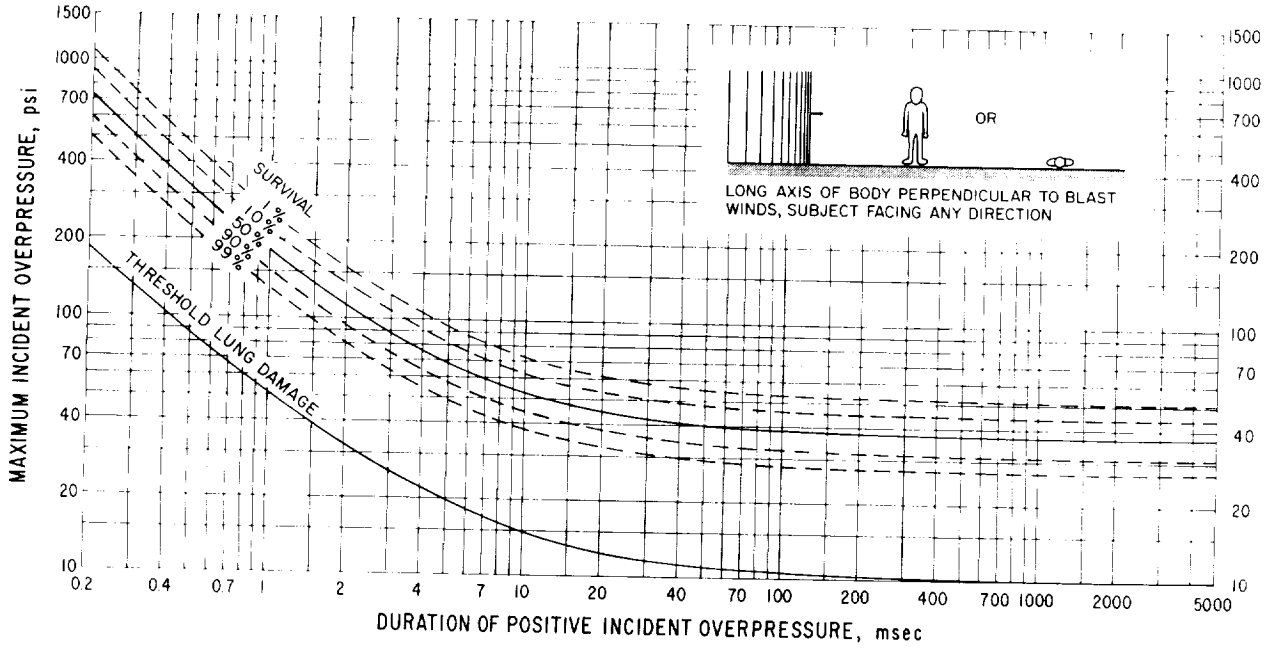
(After Richmond et al[97] and White[121])

Figure 12-21

Estimate of Survival and Lung Damage Thresholds for Humans Exposed to Air Blast

(After Bowen et al[21])

a. Long Axis of Body Perpendicular to Blast Winds, Subject Facing Any Direction



b. Thorax Near a Reflecting Surface Which Is Perpendicular to Blast Winds, Subject Facing Any Direction

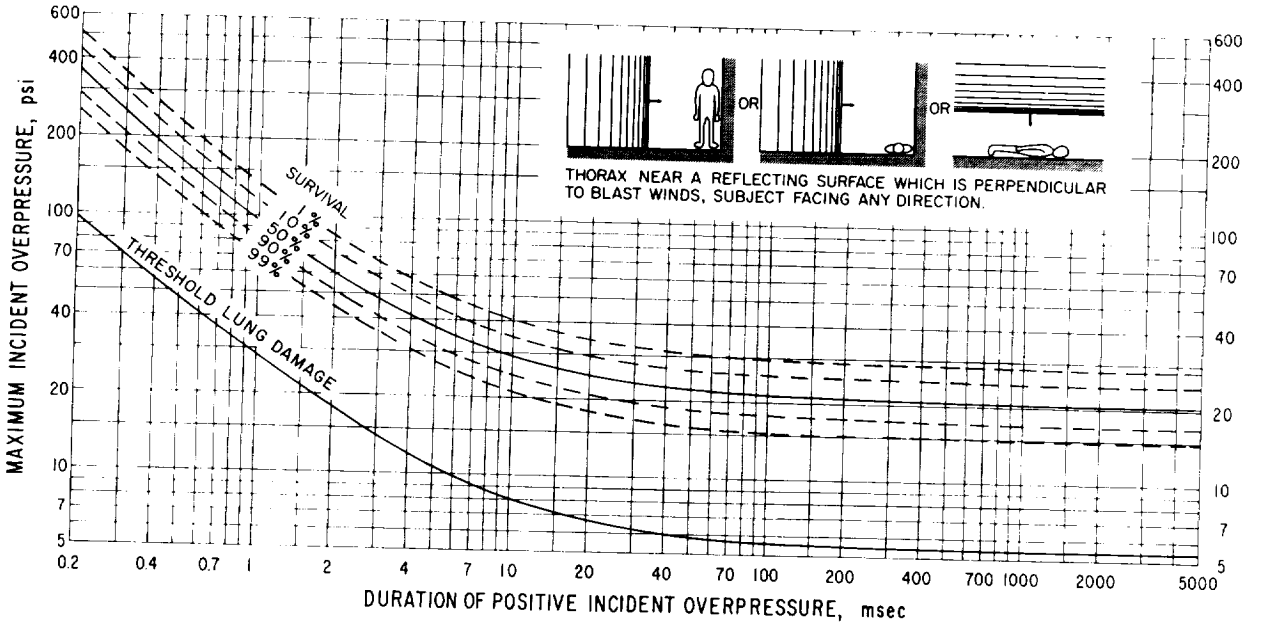
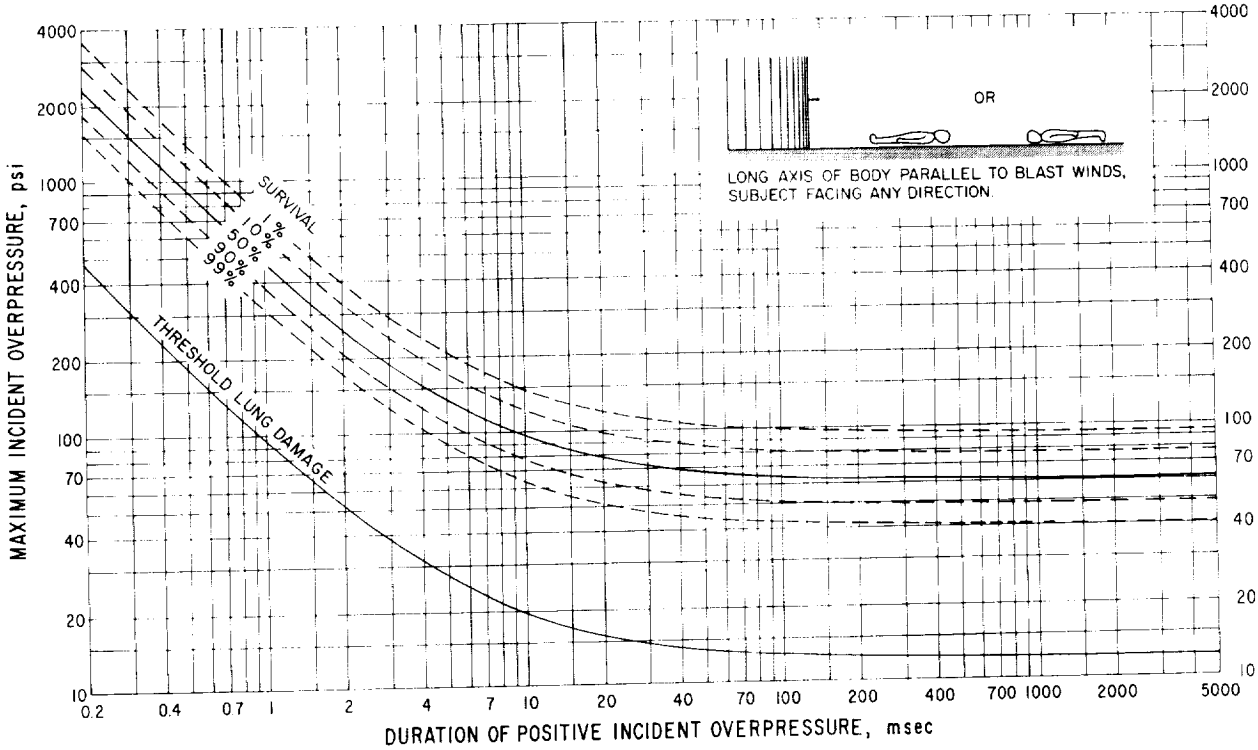
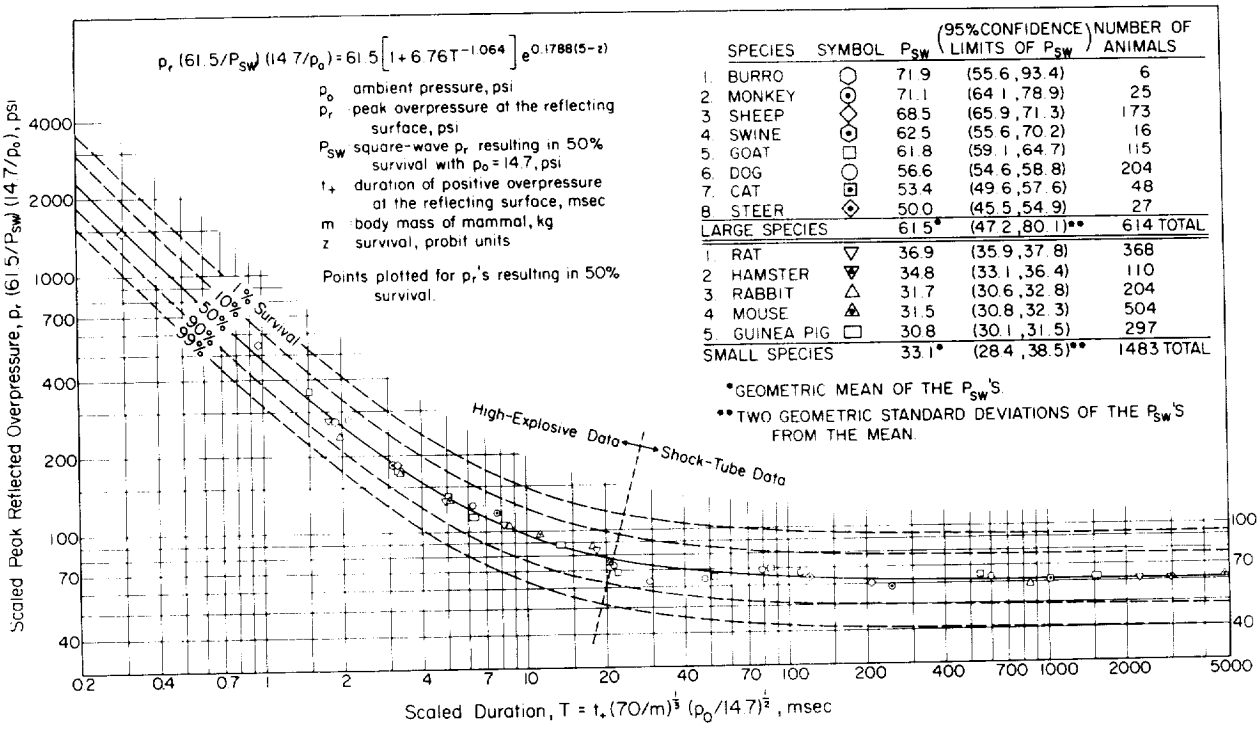


Figure 12-21 (continued)

c. Long Axis of Body Parallel to Blast Winds, Subject Facing Any Direction



d. Scaling Factors by Which Animal Data Have Been Extrapolated to Humans



	SPECIES	SYMBOL	P_{sw}	(95% CONFIDENCE LIMITS OF P_{sw})	NUMBER OF ANIMALS
1.	BURRO	○	71.9	(55.6, 93.4)	6
2.	MONKEY	⊙	71.1	(64.1, 78.9)	25
3	SHEEP	◇	68.5	(65.9, 71.3)	173
4	SWINE	⊙	62.5	(55.6, 70.2)	16
5.	GOAT	□	61.8	(59.1, 64.7)	115
6.	DOG	○	56.6	(54.6, 58.8)	204
7.	CAT	⊡	53.4	(49.6, 57.6)	48
8	STEER	◈	50.0	(45.5, 54.9)	27
LARGE SPECIES			61.5*	(47.2, 80.1)**	614 TOTAL
1.	RAT	▽	36.9	(35.9, 37.8)	368
2	HAMSTER	⧩	34.8	(33.1, 36.4)	110
3	RABBIT	△	31.7	(30.6, 32.8)	204
4	MOUSE	⧋	31.5	(30.8, 32.3)	504
5	GUINEA PIG	□	30.8	(30.1, 31.5)	297
SMALL SPECIES			33.1*	(28.4, 38.5)**	1483 TOTAL

*GEOMETRIC MEAN OF THE P_{sw}'S.

**TWO GEOMETRIC STANDARD DEVIATIONS OF THE P_{sw}'S FROM THE MEAN.

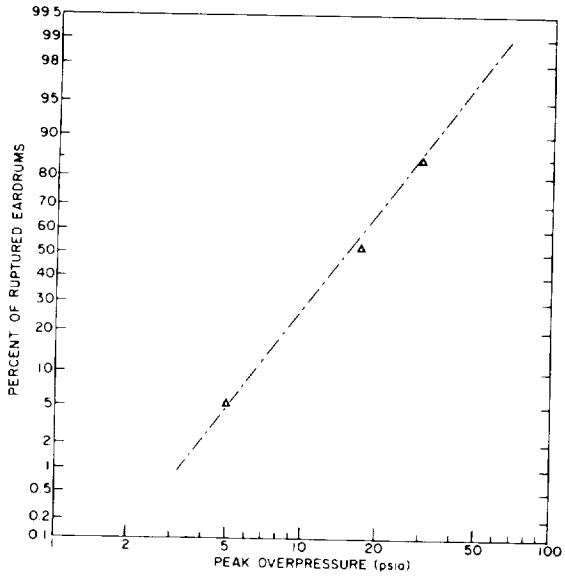


Figure 12-22

Tolerance of Eardrums to Fast-Rising Overpressures Estimated from Combined Human Data (Δ)

(After Hirsch[57])

The most significant effect of blast injury to the ear is damage to the organ of Corti and resulting loss of hearing, both temporary and permanent. It is felt that when the eardrum is disrupted, injury to the inner ear is less and the deafness less grave and less permanent than when the same overpressure does not disrupt the drum (57). In the presence of drum rupture, the hearing loss is of mixed type with both low-frequency loss of middle ear damage and high-frequency loss of inner ear damage. (See Sound and Noise, No. 9). Usually the low-tone loss will be in the order of 10-30 dB and the high-tone loss of 40-80 dB. When dislocation of the ossicles accompany drum rupture, usually in "long-duration" blast, a permanent, severe, conductive loss is sustained. The size of the perforation does not correlate with hearing loss. Up to 78% of cases of temporary deafness can occur without perforation (18). There is an accumulative effect of multiple blast insults to the ear with progressive conversion to permanent hearing loss.

Because of the great variation with age and secondary factors, there is little correlation of dB loss at any frequency with the blast overpressure sustained at the ear level. Figure 12-23 represents a typical audiogram following a fast rise-short duration overpressure of 30 dB which disrupted both eardrums. After healing of the perforation the audiogram returns to normal except for residual high frequency loss. As is indicated, conversational tone reception may not be compromised. The effect of impulse noise is under study (60, 61).

Recompression damage to the ear is covered on page 12-30.

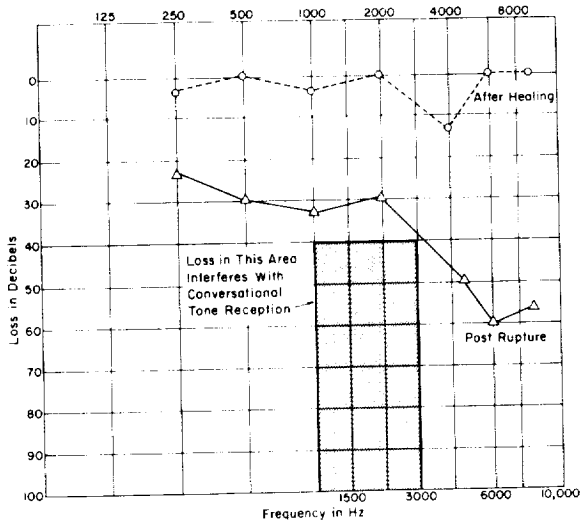


Figure 12-23

Typical Audiogram Following Short Rise Time-Short Duration Overpressure of 30 Psia with Drum Rupture

(After Hirsch[57])

Heart

Lowering of the blood pressure sometimes to the point of shock, slowing of the pulse and an increase in the respiratory rate have been frequently observed in experimental animals exposed to explosions. The shock-like state occurs too rapidly to be associated with loss of blood in the lung tissue, so explanations have been sought through reflex mechanisms. It thus appears that the tissue injury of the lung may induce reflexly through the vagus nerve a shock-like state with a slow heartbeat. The situation is later complicated by loss of blood into the lungs and reduced oxygen supply secondary to the reduced capacity of the lung for air. Heart failure secondary to failure of the circulation of blood to the heart muscle brought about by an emboli has also been noted and sometimes appears to be related to sudden death in experimental animals.

Abdominal Viscera

Hemorrhage into air-containing abdominal viscera, particularly the gastrointestinal tract, has been reported following both air and water blast. As for the lungs, the basic mechanism for injury would appear to be relative displacements among tissues at boundaries where the medium changes abruptly from fluid to gaseous. In air, injury to the lungs occurs more easily than injury to the abdominal viscera. Because of better shock-wave coupling, underwater explosions of mines near sailors swimming in water have been noted to produce more severe injuries to the air-filled abdominal viscera than to the lungs.

Brain

The mechanism of injury to the brain from shock waves in air remains somewhat obscure. Both large and small hemorrhages in the substance of the brain and in the tissues surrounding the brain have been observed in both man and experimental animals. Such injuries usually occur after exposure to extreme and nearly mutilating blast conditions. There is experimental

evidence that under less severe conditions some of these hemorrhages are secondary to interference with the circulation to the brain by bubbles of air in much the same way as described above for the heart (120).

Skeleton

Injuries to the bony skeleton from shock waves reaching man through air or water have not been reported. On the other hand, shock waves reaching man through solid supporting structures have produced severe fractures and dislocations of the skeleton. Transients applied to the base of the spine of a seated man can produce fractures and dislocations of the spine with paraplegia as a consequence. Although injuries to the lower extremities while standing and to the spine while seated are most common, other postures with varying degrees of contact with solid structures can produce other more bizarre injuries (120). (See Impact in Acceleration, No. 7.)

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