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RAPID (EXPLOSIVE) DECOMPRESSION EMERGENCIES IN PRESSURE-SUITED SUBJECTS

by Emanuel M. Roth

Prepared by
THE LOVELACE FOUNDATION
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By Emanuel M. Roth, M.D.

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FOREWORD

This study of explosive decompression emergencies in vacuum test chambers was performed upon request of Dr. E. L. Beckman, Acting Chief, Preventive Medicine Office, Manned Spacecraft Center, Houston, Texas, and submitted through the Directorate of Space Medicine, Headquarters Office of MSF, NASA.

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Rapid (Explosive) Decompression Emergencies in Pressure-Suited Subjects

Exposure of humans to the vacuum of space simulation chambers has become a requirement in the development of full pressure suits and extravehicular assemblies. One of the hazards of such studies is rapid or explosive decompression damage to the lungs brought about by catastrophic failure of any one of several components of the extravehicular assembly. The present analysis will cover the physical factors determining lung damage after explosive decompression of any pressurized structure surrounding a human; the modes of failure and physical processes in the disruption of space suits; the pathological physiology of lung damage by explosive decompression; and the therapeutic considerations in handling such emergencies in vacuum simulation chambers.

I. Physical Considerations in Damage to the Lung During Explosive Decompression

The following discussion is adapted directly from the excellent summary of Luft [123].

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, (suit) and the size of the aperture are known or can be assumed [71, 77, 82, 136]. 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 [77, 136]. 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 [82]. The first of these, which sets the absolute time scale of decompression, will be referred to as the time constant (t_c)

$$t_c = \frac{V\ (m^3)}{A\ (m^2) \cdot C\ (m/sec)} \tag{1}$$

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 1 is a graphic solution of equation (1) relating cabin volume and effective orifice to the time constant in metric units.

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 [82].

$$P_1 = f\left(\frac{P_i}{P_f}\right) \tag{2}$$

The values for P_1 can be read for any desired pressure ratio from the curve in Figure 2. The total duration of decompression (t_d) is the product of the time constant (t_c) and the pressure factor P_1.

$$t_d = t_c \cdot P_1 \tag{3}$$

The relationships expressed in equations (1) and (3), which have been verified in numerous experiments, are convenient for estimating the decompression time on the basis of an aircraft cabin volume and the configuration of windows, doors, or canopy for various cabin pressures at altitudes. Similarly, the volume to orifice ratio and the time constant of any decompression situation can be estimated if the elapsed time of decompression

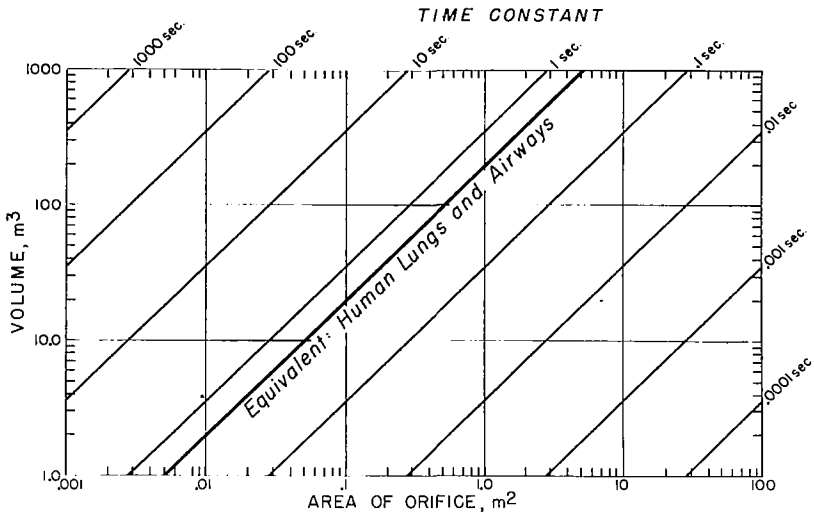

Figure 1. The volume of the pressure cabin relative to the effective area of the decompression orifice determines the time constant of decompression. For the respiratory tract this depends on the lung volume and the flow resistance of the airways at the time of decompression.
(After Luft (123))

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 3 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

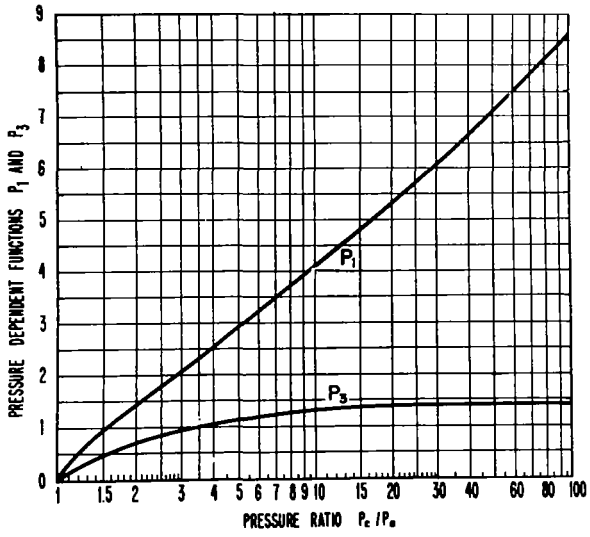

Figure 2. The pressure function P_1 for the total time of decompression and for the "constant rate time" (P_3) as derived from the pressure ratio (P_c/P_a) or (P_i/P_f).
(After Bancroft [12])

(P_3) which may be read from the curve so designated on Figure 2 for the appropriate decompression ratio:

$$t_{cr} = t_c \cdot P_3 \tag{4}$$

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.

$$\Delta P_L = P_L - P_f = P_i - P_f \tag{5}$$

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

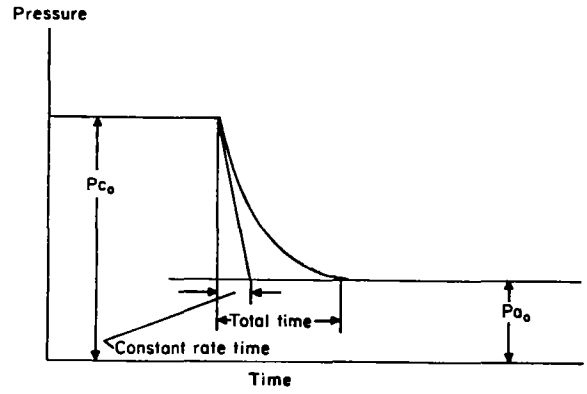

Figure 3. Definition of constant rate time t_{cr}. (After Haber and Clamann [82])

isothermal conditions with water vapor pressure at 47 mm Hg would be [122]

$$\frac{V_f}{V_i} = \frac{P_i - 47}{P_f - 47} = RGE \qquad (6)$$

The lungs are 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 (6) accordingly.

$$\frac{V_{max}}{V_i} = \frac{P_i - 47}{P_L - 47} \qquad (7)$$

and solving for P_L

$$P_L = \frac{V_i}{V_{max}} (P_i - 47) + 47 \qquad (8)$$

The pressure difference, ΔP_L, is found by substituting equation (8) for P_L into equation (5):

$$\Delta P_L = \frac{V_i}{V_{max}} (P_i - 47) + 47 - P_f \qquad (9)$$

It is apparent from equation (9) 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 [58, 86, 98, 123, 155, 167]. 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, tracheal pressures as high as 180 mm Hg are tolerated without discernible damage to the lungs [58, 86, 155]. 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 (9) 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 (9) 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 lung 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 (9) for most instances. As demonstrated by Luft and

co-workers, [123,125,126] 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 4.

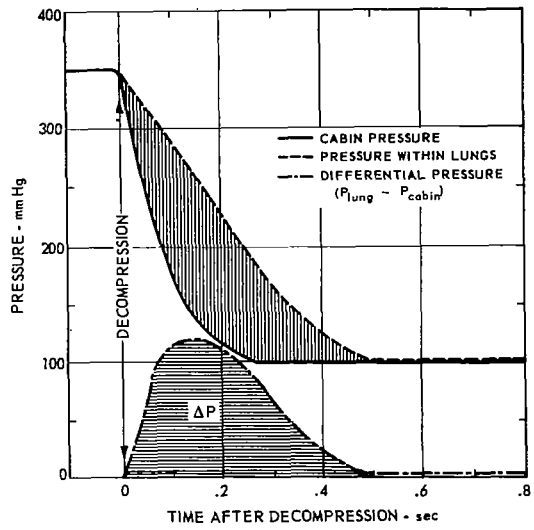

Figure 4. Time characteristics of overpressure in the lungs.
(Modified from Luft [123] by Billings and Roth [22])

The heavy line in Figure 1 represents the time characteristic of the human lung with open glottis on a background of the general volume-to-orifice relationship.

There is a critical V/A ratio of the cabin or suit relative to this ratio of 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 4 remains the same, but the duration of the transient is longer [123,126]. This means that the area under the differential pressure curve which represents the impulse in terms of

$$\frac{\text{force (dyne) x time (sec)}}{\text{area (cm}^2\text{)}} \qquad (10)$$

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 [25,159].

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 (9) for isothermic conditions, with a corresponding drop of pulmonary pressure. In dogs, expansion is not apparent before 10 msec [184]. 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 [60, 167].

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 [43, 168].

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 can 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 [167]. Air embolism may be facilitated at this time at the sites of tissue 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 [123]. There can be no doubt that the rate of decompression is a decisive variable as far as mortality is concerned. In Table 1, eight groups of experiments on small animals have been selected from various studies, comparable in the severe pressure conditions employed. The initial pressure is approximately 1 atm, and the differential of decompression is fairly uniform, being greater than 630 mm Hg (.83 atm) in all cases. The decompression times vary from .630 to .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

Table 1. Mortality in Relation to Decompression Time in Experimental Animals

Reference	Species	Decompression		$P_i - P_f$ mm Hg	P_i/P_f, mm Hg	Decompression time, sec	V/A, m^3/m^2	Mortality, percent
		Initial pressure, P_i, mm Hg	Final pressure, P_f, mm Hg					
Corey ..51	Rat.....	760	21	739	36.2	0.630	33.0	0
Eggleton 64	Rabbit.....	760	122	638	6.2	.200	18.0	0
Eggleton 64	Rat.....	760	122	638	6.2	.200	18.0	0
Kolder 109	Rat.....	735	73	662	10.0	.180	15.0	0
Kolder 109	Rat.....	735	73	662	10.0	.041	3.3	10
Kolder 109	Rat.....	735	73	662	10.0	.015	1.2	50
Stickney 173	Rat.....	738	32	706	23.1	.019	1.14	50
Kolder 109	Rat.....	735	73	662	10.0	.0014	.12	100

All experiments are comparable in the range of decompression from approximately 1 atmosphere to less than 0.2 atmosphere.

(After Luft [123])

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, Kolder used a special decompression device with a V/A of .12 m^3 per m^2 [109]. 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 (9) 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 .47 atm. When rats were exposed to increasing pressure differences from an initial pressure of 735 mm Hg with a V/A of .12, Kolder [109] observed an increasing number of fatalities whenever the final pressure was less than 368 mm Hg (.48 atm). 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 [109]. Complete protection was provided with a pneumothorax of 4 ml in rats that survived decompressions that were otherwise absolutely fatal.

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, Violette gives a value of 100 [184]. 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.

As will be covered below, experience with human exposure to decompression at low cabin V/A ratios is very limited. Well-documented, danger-zone decompressions with open glottis have been limited to those recorded in Table 2. It can be seen that only the first exposure of Sweeney [174] would have had a cabin V/A ratio (1 m^3/m^2) well within the expected lethal range. Luft [123] has calculated for these experiences the overpressures to be expected in the lung for closed airways at midrespiratory volume. Even under these conditions, 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.

Table 2. Rapid Decompression Tolerated by Man

Reference	n	Altitude, ft	Initial pressure in cabin, P_i, mm Hg	Final pressure, P_f, mm Hg	$P_i - P_f$, mm Hg	P_i/P_f	Time, sec	V/A, m^3/m^2	ΔP_L, mm Hg [a]
Sweeney 174	10	27 000 to 45 000	253	112	141	2.23	0.005	1.0	48
Sweeney 174	15	8 000 to 35 000	565	179	386	3.16	.090	13.4	153
Döring ..58	13	9 800 to 49 100	526	90	436	5.83	.230	23.0	220

[a] ΔP_L is the overpressure which would occur in the lungs if the airways were closed at midlung volume; critical pressure is 80 mm Hg (Eq. 9). (See also Table 3.1 in Reference 180 for similar data).

(After Luft [123])

Figure 5 is a summary curve of Violette which represents a rough evaluation of the relative dimensional and pressure-ratio factors defining the zone of possible injury under glottis-open conditions. The curve does have many shortcomings. For instance, it is doubtful that 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. Plots of data from Tables 1 and 2 show this curve to be conservative enough for a first approximation of the safe zone. Lack of direct data regarding V/A ratios in the experiments of Table 1 makes the degree of conservatism difficult to assess. As will be covered in Section III, there is inadequate information on the degree of breathholding and fraction of vital capacity during human exposure where damage to lungs has been recorded. These factors preclude adequate evaluation of the zone above the curve in Figure 5, especially in the pertinent zone of high P_c/P_a ratios. The X-asymptote should also be at lower P_c/P_a ratios (11).

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 [24]. 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 view of these limitations, the zones of Figure 5 must be used with great caution is establishing threshold t_{cr} values in humans at high P_c/P_a ratios. Unfortunately this is the question of immediate interest in evaluating hazards of space suit decompression. All one can really say is that a t_{cr} of less than 0.1 second will more than likely cause lung damage in man at pressure ratios dictated by suit decompression to vacuum. The longer the time beyond 0.1 second, the lower the hazard. Beyond a t_{cr} of several tenths of a second, there is little hazard for the subject if glottis is open and no previous lung pathology is present.

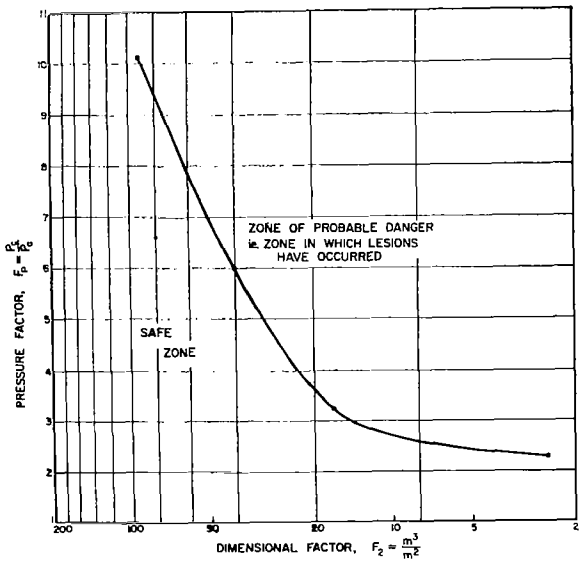

Figure 5. Curve derived from the data of Violette [184] defining the zones of safety and probable danger in explosive decompression. (After Fryer [72])

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 these relatively high pressures with inert gas mixtures [162].

The relative tendency for lung damage on various inert gas-oxygen mixtures has been discussed with respect to space cabins [165].

The flow of gas through the respiratory tract is a critical factor during "explosive" decompression. 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 [25]. 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) anda gas-flow factor involving the specific heat ratio γ. This relationship is shown in the following equation

$$\left(\frac{dP}{dt}\right)_{t=0} \sim \frac{1}{M^{1/2}}\left(\gamma\left[\gamma\left(\frac{2}{\gamma+1}\right)^{\frac{\gamma+1}{\gamma-1}}\right]^{1/2}\right) \qquad (11)$$

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 [165]. 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 [89]. Sensor lag obviously complicates the measurement

to an unknown degree. The lung model of Bowen et al [25] used a value of $\gamma = 1.2$ for air as a polytropic compromise in an unknown situation. It is felt, by this group, however, that the isothermal process probably predominates [69].

In the analysis of the space-cabin situation, calculations were presented for the currently proposed environment of 50 percent inert gas and 50 percent oxygen[165]. Both the isothermal and 50 percent isothermal-50 percent adiabatic specific heat ratios are presented in Table 3. For the isothermal condition, $\gamma = 1$. Table 3 represents the calculations of $\left(\frac{dP}{dt}\right)_{t=0}$ for these gas mixtures and the relative hazard index with nitrogen-oxygen $\square$ 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 $1/M^{\frac{1}{2}}$. 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 secondary factors will 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. Any overlap of the respiratory and cabin flows will reduce the dependence upon molecular weight. Therefore, one can predict that the smaller the hole, the less gas dependent is the decompression hazard. In view of the high probability of flow overlap, the molecular factor should probably be given little weighting in selection of an inert diluent in space suits.

Another variable to be considered in evaluation of the hazard of decompression is the presence of oxygen mask or respiratory equipment which may

Table 3. Relative "Explosive" Decompression and Blast Overpressure Hazards from Atmospheres at 7 psia with 50 Percent Inert Gas and 50 Percent Oxygen

Factor	Gas mixture in cabin						
	$He-O_2$	$Ne-O_2$	$A-O_2$	$Kr-O_2$	$Xe-O_2$	N_2-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 ($N_2-O_2 = 1$)	.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 ($N_2-O_2 = 1$)	.50	.87	1.0	1.2	1.3	1.0	1.0

(After Roth [165])

superimpose an artificial "glottis" over the normal one and increase the effective V/A ratio of the subject [125]. This should not be a consideration in current full pressure suits where large plastic bubble helmets surround the facial area.

Evaluation of damage risk to the lung during space operations in the case of breathholding has been reviewed by Busby using the relationships of the Luft equation (equation 9) [30]. The pressure gradient which may exist across human lungs and passively distended chest wall if an "explosive" decompression to a vacuum occurs while respiratory passages are 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 4. It is most 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 4

Pressure Gradients (ΔP_L) Across Lungs and Passively Distended Chest Wall During "Explosive" Decompression to Vacuum with Respiratory Passages Closed.

Calculations cover different ambient atmospheric pressures (P_i) and lung volumes (V_i) prior to decompression to 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

(After Busby[30] from the unpublished calculations of Luft)

In recent years several mathematical models have been made of the thorax-abdominal system for evaluating the hazards of air blast overpressure and explosive decompression damage to the lungs [25,107]. At the present state of development, these computerized models require more empirical study for confirmation of their validity under the several variables of the current problem. 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 space suit assemblies.

II. Evaluation of Decompression Hazards Following Specific Suit Failures

Section I covered the general physical considerations in assessing the hazards of "explosive" decompression. The next step in the analysis is an evaluation of the V/A ratios associated with failure modes of different types of space suits.

A. Soft Suit

The following modes of failure of soft suits were discussed with Charles Lutz, Matthew Radnofsky, and Edward Michel of the Manned Spacecraft Center, Houston. No published analyses of assembly volumes, areas, destructive testing, or failure mode were available. Any errors in these values should be attributed to the misunderstanding of these verbal reports on the part of the author of this paper. Many of these critical variables are summarized in Table 5 and calculated in Appendix 1.

In the calculation of suit volume, it was assumed that all of the free volume of the suit was rapidly exchangeable. Free volume is defined as the total gas volume of the suit with a human subject inside. By virtue of the complex geometry of the suit-body interposition it is possible that not all of this volume is rapidly exchangeable. Any delay in exchange reduces the effective volume and decreases the V/A ratio. Neglect of this factor minimizes the hazard (vide infra). All orifices have been assumed to have flow coefficients of 1.0. This approximation is on the conservative side and tends to exaggerate the hazard in decreasing the V/A ratio below the actual level.

Helmet Bubble

The new helmet of the soft suit is made of a single unit of polycarbonate plastic which has proven very resistant to fracture on impact testing (no data were readily available). It was felt that catastrophic failure of the plastic helmet could be neglected as a cause of explosive decompression.

Neck Seal

The neck seal of the soft suit appears to be of "fail-safe" design. The seal is even difficult to open in the hands of suit technicians under laboratory conditions. All individuals interviewed felt that the seal would be most secure and need not be considered a site of catastrophic failure. However, were the seal to fail and the helmet blow off, an annular orifice of 295 cm^2 would be available for air flow. (See Table 5 and Appendix I). The effective neck area was calculated by assuming the circular seal at a 17° angle subtended a elliptical area on the cylindrical neck of the astronaut. A mean neck

Table 5

Effective Volumes and Orifices during Explosive Decompression of Soft and Hard Space Suits

Critical Volumes	Apollo Soft Suit	Litton Hard Suit
Total free volume of suit, PLSS, and hoses	28 liters	75 liters
Free volume of helmet	~2.5 liters	4.4 liters
Free volume in PLSS and hoses (2 hoses, 3/4" ID, and 2 1/2 feet and 6 feet long)	3.8 liters	3.8 liters
Free volume of suit below neck ring	22 liters	67 liters
Neck Seal		
Diameter of seal	9" ID	11.8" ID
X-area	411 cm^2	706 cm^2
Angle of elevation of seal	17°	40°
X-area of neck subtended by seal	116 cm^2	145 cm^2
Orifice at neck seal	295 cm^2	561 cm^2
Wrist Seal		
Diameter Seal	4" ID	3.87" ID
X-area of seal	81.4 cm^2	76 cm^2
X-area of wrist at seal	21.5 cm^2	21.5 cm^2
Orifice at wrist seal	60 cm^2	54 cm^2
Thigh Seal		(RX 4 and 5)
Diameter	-	7 7/8"
X-area seal	-	314 cm^2
X-area of lower thigh	-	137 cm^2
Orifice of thigh seal	-	177 cm^2

Table 5 (continued)

Effective Volumes and Orifices during Explosive Decompression of Soft and Hard Space Suits

	Apollo Soft Suit	Litton Hard Suit
Ankle Seal		(RX 3 and 4 only)
Major axes of ellipse	-	5 9/16" and 7 5/32"
X-area of seal	-	207 cm^2
Ankle area (6 1/2" from ground)	-	39 cm^2
Orifice at ankle seal	-	168 cm^2
Waist Seal		
Diameter	-	16" ID
X-area of body seal	-	1300 cm^2
Area of abdomen (1" above umbilicus)	-	490 cm^2
Orifice at waist seal	-	810 cm^2
Fingers		
Diameter of glove finger	1" ID	1" ID
X-area of glove finger	5.1 cm^2	5.1 cm^2
X-section of finger (1/16" clearance)	3.9 cm^2	3.9 cm^2
Orifice at finger	1.2 cm^2	1.2 cm^2
Gas Umbilical Hose from Space Chamber		
Diameter	1 1/4"	1 1/4"
X-area	7.9 cm^2	7.9 cm^2
Gas Umbilicals from PLSS		
Diameter	3/4"	3/4"
X-area per hose	2.8 cm^2	2.8 cm^2

circumference of 37.43 cm^2 was used (See Table 16-4b in Ref. 162) to determine the minor axis of 11.9 cm^2 and major axis of 12.4 cm^2. These calculations were corroborated by taking an elliptical imprint of the neck at the 17^o angle with a stiff wire. Six male subjects, of astronaut size, were measured at the Lovelace Foundation. The difference between seal area and elliptical neck area gives the annular orifice of flow. What is uncertain, of course, is the relative flow restriction presented by the shoulders and suit just below the seal. It was felt, however, that the neck seal would be an adequate approximation of the orifice.

Wrist Seal

The wrist seal appears to be less safe than the neck seal. All agreed that this would probably be the site most likely to be involved in an explosive decompression, either through faulty construction or faulty donning procedure. The annular orifice upon catastrophic seal failure is noted in Table 5 and Appendix 1 as 60 cm^2.

Fingers

Other possible sites of failure are the fingers of the glove. The glove fabric has a single layer of Beta fiber over the bladder and appears more prone to sudden disruption than that of the main body of suit or bellows. A glove finger diameter of 1 inch was chosen and a 1/16 inch clearance assumed between fingers and inflated glove [27]. For the worse case of complete transection of the glove finger, an orifice of 1.2 cm^2 was calculated.

Suit Fabric

It was felt by all interviewed that presence of a multilayered mylar-aluminum outer garmet would tend to reduce the possibility of catastrophic failure of the suit upon disruption by a sharp object. Tears of this type apparently propagate slowly and would not lead to an explosive decompression.

Joint Bellows

Disruption of the joint bellows has occurred in the past. However, the tears tended to propagate slowly. Current development is being directed toward a pressure restraint layer within the bellows which will contain

any disruption of the external bellows and, at worst, cause a slow propagation of any orifice. It was felt, therefore, that the bellows were not to be considered a site of explosive decompression.

Entrance Zipper

The entrance zipper of the suit is of double sealing design and would probably fail slowly even under external trauma. It was doubted that all of the teeth would suddenly give way at once. The zipper was, therefore, not considered as a probable site of explosive decompression.

Umbilicals

One must consider disruption of gas umbilical lines as a cause of rapid decompression. The umbilicals from chamber to suit are 1 1/4" ID.

One connector assembly has already failed at the suit fixture leading to loss of consciousness and hypoxia, but no symptoms or signs of lung damage were noted after the pressure drop from 3.7 psia to minimum reading of 0.1 psia in about 0.5 sec (PLSS 005 Test #3, 14 Dec. 1966). Unfortunately, the actual pressure traces were not available for study [93]. The chamber umbilical held open the baskets of the check valve. Presence of a pressure-sensitive check valve in the umbilical distal to the suit valve assembly could probably have prevented the decompression. The significance of the decompression time for this accident is discussed below.

Testing of a PLSS on the back of a subject would present one of the two 3/4" ID hoses or their connectors as possible sites of explosive decompression.

B. Hard Suit

The Litton hard suit series has advanced through the model RX-5. Each model has presented changes in design which vary the site of potential catastrophic failure. An attempt will be made to cover many of these critical sites, assuming that at some time in the testing process, one or more may be present in any one suit. The data on the hard suit were obtained from Mr. Pierre Brousseau, Manager of Protective Systems, Space Sciences

Laboratory, Litton Industries, and Mr. Joseph Kosmo, Hard Suit Project Engineer, NASA, Manned Spacecraft Center, Houston. The free volume of the hard suit (75 liters) is about 3 times that of the soft suit. The hard suit is made of aluminum metal with compensated bellows joints. A honeycomb layup can be added for micrometeoroid protection[120]. It was felt that the aluminum body of the suit would not be a site of explosive decompression under space chamber conditions.

Helmet

The helmet of this suit is a polycarbonate hemisphere attached by a neck seal to the rest of the suit. The free volume is greater than that of the soft suit helmet. Impact resistance is similar to that of soft suit helmet.

Neck Seal

The neck seal of the hard suit is larger (12" ID) than that of the soft suit and is canted up at a 40° angle. It appears to be of "fail-safe" design with little chance of failure. The annular orifice area, as calculated by the same method as for the soft suit, is 564 cm^2, almost twice that of the soft suit. There is more variation than with the soft suit in subtended neck area. This was measured directly at a 40° angle for six subjects at the Lovelace Foundation. Variation is due to greater diversity in shape of the lower occipital-upper neck area from subject to subject for the 40° ellipse than for the 17° ellipse. The mean area is similar to the calculated area.

Wrist Seal

The wrist seal and glove structure is similar to that of the soft suit. Though also of "fail-safe" design, this seal was felt to be more vulnerable than the neck seal. The annular decompression orifice on seal failure is about 54 cm^2.

Thigh Seal

In models RX 4 and 5, there is a seal at the thigh for easy donning of the suit. It is of design similar to the wrist seal. Calculation of the annular orifice was not as accurate as that of other areas, in that the thigh section

subtended by the seal was not as well defined. The circumference of the "lower thigh" in Table 16-4e of Ref.162 was used to calculate the assumed circular area of the thigh. An annular orifice of 177 cm^2 is recorded in Table 5.

Ankle Seal

In RX 3 and 4 only, there are elliptical seals at the ankle for easy boot removal. The seal is 6 1/2" above the floor. Data for this point on the lower leg is not recorded in standard anthropometric tables. Measurement of the circumference of the leg at this site was made at the Lovelace Foundation on six subjects of general astronaut height and weight. The cross section area at this site is recorded in Table 5 as 39 cm^2 giving an annular orifice at the seal of 168 cm^2.

Waist Seal

The waist seal with a 16" internal diameter is the largest in the hard suit and is the major port of entry. It crosses the abdomen about one inch above the umbilicus. At present this seal is not of "fail safe" design. It was felt that in the future, the design would be improved[111]. The band-and-flange nature of the design, however, makes the current seal quite safe. The annular orifice at the waist is 810 cm^2.

Fingers

The same factors hold for fingers in the hard suit as in the soft suit.

Joint Bellows

While the joint bellows appear to be much more vulnerable than the static structure of the rest of the suit, they are protected internally by a laminated fabric of high tear strength which propagates a rip very slowly. Therefore, as with the soft suit, the bellows are not expected to be a site of explosive decompression.

Umbilical hoses

The PLSS and chamber umbilical hoses are the same size for both the hard and soft suit. Placement of the entrance ports into the suit have varied from model to model of hard and soft suit. In the present analysis,

site of the entrance port on the body of the suit does not significantly affect the calculations.

C. Time of Decompression and Hazard Analysis

No data are available on the pressure profiles for suits catastrophically decompressed to vacuum with dummies inside. Accurate pressure profiles of the one accidental decompression noted above were not available [85]. One curve showing pressure dropping from 3.7 psia to 0.1 psia was recorded as a straight line function over the 0.5 seconds. In view of the exponential nature of the decompression, the shape of the curve is highly suspect. The two other curves available were plotted as exponential but gave the time to reach 0.1 psia as 10 seconds. The time to collapse of subject (12-15 sec) was too short to support the 10 seconds of decompression. It has been suggested that the times on these curves are probably in error and should not be used in this analysis [93]. Figure 6 and pages 29 and 31 cover one of these curves corrected for the proposed 0.5 seconds to pressure nadir.

As covered in Section I, evaluation of the hazard of explosive decompression requires calculation of the V/A ratios of the suit-orifice systems and corresponding times of decompression. Free suit volume as noted in Table 5 refers to the free gas volume in the suit with the subject inside. In a first approximation, the suit is treated as a rapidly exchanging chamber; and orifices, as having a coefficient of 1.0. As discussed above, these approximations will tend to cancel one another, i.e., if only 0.8 of the free suit volume is rapidly exchanged and the orifice coefficients are really 0.8, the V/A ratio will remain unchanged.

Unfortunately, the response time of the PLSS or ECS under conditions of explosive decompression has not yet been evaluated. Rapid addition of gas from the PLSS would tend to increase the effective volume of the suit. By neglecting this factor the worst possible condition is assumed. For the soft suit, initial pressure of 3.7 psia will be assumed. For the hard suit pressures, pressures up to 7 psia can be anticipated.

Calculation of the residual free volume of the suit remaining intact about the chest when the waist and thigh seals of the hard suit are disrupted is somewhat indirect. Empirically determined fractional suit volumes were unavailable. Since the suit segments are approximately cylindrical in cross section, it was felt that calculation of the segmental

areas from frontal projection of the suit would give a fairly accurate indication of fractional volumes. Figure 15 of Reference 100 was used as a model. An Ott planimeter was used to determine frontal areas. The fractional areas were recorded as follows for the four suit sections.

Table 6

Fractional Frontal Areas of Litton Hard Suit	
Area above waist seal	0.47
Pelvic area between waist and thigh seals	0.25
Both legs below thigh seal	0.28
Each leg below thigh seal	0.14

The fractional volumes of the body were also determined from the cylindrical model of man used for calculating radiative surface areas (Fig. 6-22 of Ref. 150 or Ref. 162). From this model it was calculated that the volume of the body above the waist is about 0.54 that of the whole, and the volume of each leg below the thigh seal is about 0.12 that of the whole body. These volumes match closely enough to the corresponding fractional suit volumes noted in Table 6 to permit the assumption that the free gas volumes in the suit are partitioned in the same manner as is total volume within the suit. One can therefore calculate the free volume of the suit above the waist seal as 0.47 x 75 or 35.2 liters and the free volume above the thigh seal of one leg as (1-0.14) x 75 = 64 liters.

The residual suit volumes after disruption of given seals and the orifices at the site of disruption are therefore recorded in Table 7. From these data, one can calculate the V/A ratio of the system converted to meters ($cm^3/cm^2 \div 100$ = ratio in meters).

Equation 1 or Figure 1 can then be used to determine the time constant of decompression t_c. The value of speed of sound at room temperature can be determined by the equation:

$$c = 49.02\sqrt{T} \qquad (11)$$

Table 7

Soft Suits	Residual Suit Volume (cc)	Orifice Area (cm^2)	V/A Ratio (meters)	t_c (Fig. 1) (sec)	P3 (Fig. 2)	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

where c is in ft/sec and T is in degrees R. At 540°R, the speed of sound is 1142 ft/sec or 348 meters/second. The V/A ratios of column 3 of Table 7 divided by 348 give the t_c values in the fourth column.

The appropriate P_3 of Figure 2 is then chosen. Since the P_c/P_a ratio in decompression to a vacuum is ∞, the plateau value of 1.45 of Figure 2 is used in equation (4) to give the constant rate time (t_{cr}) as defined in Figure 3 and recorded in column 6 of Table 7.

Evaluation of the V/A ratios of Table 7 is the first step in the hazard analysis. In Section I of this study, the discussion of Tables 1 and 2 and Figure 5 suggested that V/A ratios above 5 meters would probably be safe for a human exposed with open glottis to pressure ratios below 2 or 3. At these low pressure ratios, but high pressure differentials, acute disruption of only the neck and wrist seals of the soft suit and the waist, neck, thigh, ankle, and wrist seals of the hard suit would probably lead to lung damage. Unfortunately, there are inadequate empirical data for differentials of 3.7 to 7 psia at P_c/P_a ratios of ∞ to evaluate the hazard [11, 61]. The conservative curve of Figure 5 suggests that at higher P_c/P_a ratios, the threshold V/A ratio increases. It is possible that above a V/A of 100 or so, the explosive decompression is safe at any P_c/P_a ratio. The P_c/P_a or P_i/P_f ratios of 36 in the first experiment of Table 1 lead to no death of rats at a V/A ratio of 36. This finding indicates that the curve of Figure 5 may rise far more steeply at high V/A ratios than is conservatively indicated in the figure. As mentioned above, the amplitude of the overpressure is a function of the pressure differential. There is no doubt that decompression from 3.7 psia to vacuum will have a lower amplitude of overpressure than those the studies discussed in Tables 1 and 2 and Figure 5, but the duration of impulse is definitely longer. More data are certainly needed to establish the relative effects of overpressure amplitude, duration, and wave form in the current problem.

Recent studies of explosive decompression of dogs to 2.0 mm Hg did not focus primarily on the damage caused by the rapid rate of drop, but on

the effects of exposure to this low pressure [61]. However, it was noted that dogs which "decompressed in 0.2 seconds" showed more petechial hemorrhages and emphysematous changes than those "decompressed in 1 second," all other variables being constant. What is not clear, however, is the relationship of this 0.2 seconds to the t_{cr}. Bancroft is forwarding the original tracings to the author for analysis [11]. For these dogs at least there is a lung damage threshold on explosive decompression to a near vacuum between the t_{cr} equivalents of the 0.2 and 1.0 second decompressions. More studies within this time of decompression range are certainly needed.

Acute disruptions of the fingers and the PLSS umbilicals in the hard suit appear defintely safe with respect to lung damage. Of questionable safety are disruptions of the PLSS umbilical hose in the hard suit and the 1 1/4" ID chamber umbilical hoses in both suits when the orifices appear at the suit ports.

Unfortunately, the accident involving loss of the 1 1/4" ID umbilical in the chamber accident at MSC (vide supra) does not help one evaluate these questionable disruptions. In Table 7, it is shown that for the soft suit, loss of the 1 1/4" ID chamber umbilical at the suit port should result in a V/A ratio of 32.3, a t_c of 0.93 and a t_{cr} of 0.14. The MSC report indicated that time to reach 0.1 psia was 0.5 seconds. The difference between the predicted t_{cr} of 0.14 seconds and the 0.5 seconds may be accounted for by several factors.

The first is the difference in measurement of time. Figure 6 is the time plot of suit pressure, recorded with times that are corrected to a pressure nadir of 0.5 sec [93]. However, let us focus on the shape of the curve. A straight line plot of t_{cr} noted by (x-x-x) suggests a t_{cr} value of 0.25 if the time to reach 0.1 psia is really the 0.5 seconds suggested above [93]. Bancroft reports that in the pressure tracings of his chamber decompressions from 3.5 to .038 psia in 0.2 seconds, deviation from a straight line appears to be visible on the tracing at about 1.0 psia and is very definite at about 0.4 psia. This suggests that the t_{cr} for the decompression at MSC could indeed have been almost 1/2 that of the actual time to reach 0.1 psia.

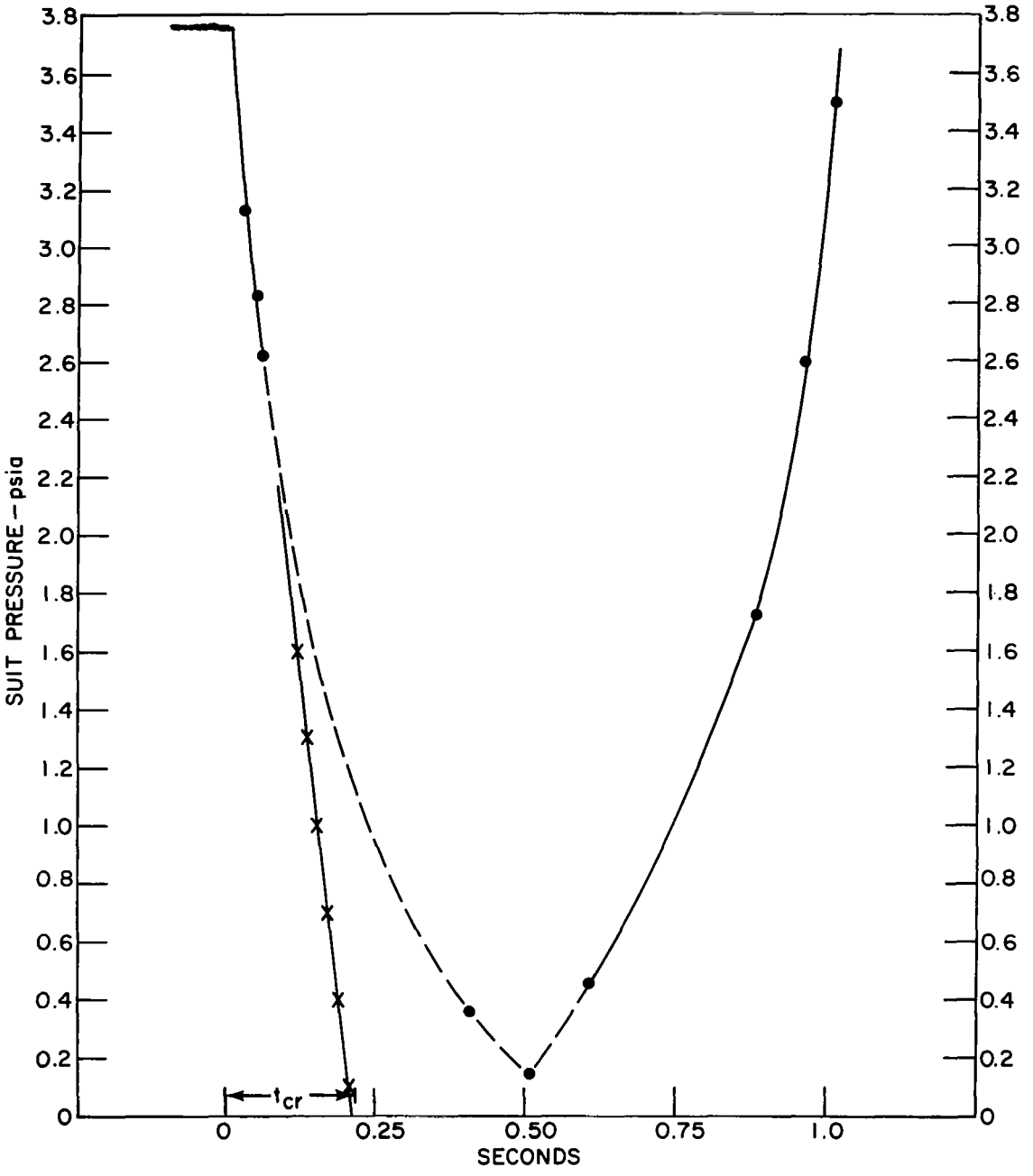

Figure 6

(After Henderson [85])

Another factor is the role of the 64" section of 1 1/4" hose in decreasing the rate of mass flow from the suit. Calculation of frictional factors under such choked-flow conditions is most difficult even for a non-flailing, smooth hose. A vacuum at the distal end of the hose suggests that the problem can be solved as an example of choked flow (exit velocity of Mach 1) of an isentropic nozzle discharging through an adiabatic duct to vacuum [160, 169].

Dr. K. J. Touryan of the Sandia Corporation, using a computer program for isothermal, time-dependent conditions, plotted predictive suit pressure curves for four conditions: smooth orifice flow (coefficient = 1), with and without a hose; and sharp-orifice flow, with and without a hose (see Appendix II). Assuming a volume of 1 cu ft (28 liters) in the suit instead of the 25.5 used in Table 7, the MSC decompression follows very closely that predicted for a smooth-orifice with hose. From Figure A-II-1, it can be seen that the constant rate time (t_{cr}) of about 0.22 seconds represents a throttling of flow of about 50 percent from the t_{cr} of .15 calculated for a smooth orifice without a hose. The prediction in Table 7 of a t_{cr} of 0.14 sec obtained by the Haber-Clamann approximation for smooth orifice flow without the hose is confirmed by this more formal evaluation of the problem.

Unfortunately, the uncertainties regarding the decompression curve and nozzle factors in the duct system preclude a more accurate calculation which would have allowed one to establish a rough estimate for the fraction of the free-suit volume which is exchangeable within the t_{cr} period. The relatively close correspondence between the predicted and semi-empirical values suggests that most of the free volume of the suit probably does exchange within this period.

As a result of this analysis, the following conclusions may be stated.

1. More experimental data on animals are required to establish threshold V/A ratios, pressure differentials, and pressure ratios for lung damage in explosive decompression of space suits in the range of 3.7 psia - 7 psia to final vacuum conditions. Efforts should be directed to analysis of the effect of total impulse and wave form on damage to the lung.

2. Conservative extrapolation of data obtained at higher pressure differentials and lower pressure ratios to the case in point suggests 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. This is true even for open-glottis conditions. The hazard is intensified if the glottis is closed and breath is held. Pathological lung conditions leading to increased sensitivity to lung damage under these conditions will be covered in Section III.

3. Disruption of a glove finger in both suits and the PLSS umbilical in the hard suit would probably not lead to lung damage if the glottis were open, but would lead to difficulty if the breath were held during the decompression.

4. 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. The case is much less clear than in conclusions 2 and 3. Disruption of the umbilical hoses at a distance from the entrance port would lower the probability of damage. The accident at MSC during Test #3 of PLSS 055 is a case in point.

5. 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 laminated fabric of the soft suit and metal shell of the hard suit are also reportedly safe from acute disruption. No

direct data from destructive testing of pressurized space suits were available for analysis. All seal areas should be designed for slow propagation of disruptive processes.

6. 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, especially those noted in conclusion 2, 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.
7. Use of gases other than 3.7 psia O_2 in the suits could affect the above conclusions in accordance with the discussion of Table 3. The higher the percentage of helium in the mixture, the less hazard is presented. Under the most sensitive conditions, a helium-oxygen mixture at 7 psia may be about 0.5 times as dangerous as an oxygen-nitrogen or the equally hazardous pure oxygen mixture at the same pressure. However, this factor will not likely be attained in practical conditions.
8. Since the maximum possible amplitude of transmural pressure is determined by the pressure difference, the higher the initial pressure in the suit, the more hazardous the exposure. A suit at 7 psia would present a maximum possible transmural pressure differential of twice that of a suit at 3.7 psia. The fraction of the total effective differential pressure and time course of overpressure would, as indicated, depend on the V/A and pressure ratios.

Further development of a computerized mathematical model of chest dynamics currently being used in study of blast biology at the Lovelace Foundation may well shed some light on the biomechanics of explosive decompression in the range of V/A ratios, pressure ratios, and pressure differentials considered in the present analysis [25].

III. Pathological Physiology of Explosive Decompression to a Vacuum

In section I of this study it was suggested that during the first 2 phases of pressure change across the chest wall, impulsive loading of the structures could result in shearing of tissues similar to that found in blast injury [19, 41, 43, 45, 47, 159, 168, 192]. 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. Finally, exposure to the vacuum for several minutes can lead to further lung damage and to the ebullism syndrome. Therefore, direct trauma to the lung parenchyma and vasculature, simple and tension pneumothorax, pneumomediastinum, aeroembolism, ebullism, and hypoxia must be considered in the pathologic process.

Contusion and Disruption of Lung Parenchyma

Tearing of the pulmonary parenchyma appears to be most pronounced in the peribronchial areas but can occur elsewhere, especially along the pleural surface [19, 89, 110, 183]. It has been suggested that peribronchial predilection is a result of disproportionate expansion of the alveolar vs. the ductal components of the lung [101]. During normal lung expansion in deep inspiration, the bronchial tree lengthens uniformly along its entire course. The smaller bronchioles and alveoli expand in all directions to fill the peribronchial spaces which are at a relatively negative pressure. With rapid overdistention, the peribronchial alveoli are torn away from the adjacent interstitial tissue; simultaneously the alveoli are ruptured and small veins are torn. The factor responsible for moving the air along the broncho-vascular sheaths is the

lengthening and shortening of the bronchi in normal respiration. The torn vessels are held open by the elastic recoil of all the surrounding pulmonary tissue. Alveolar air is allowed to escape into the peribronchial areas and along the pressure gradient into the disrupted pulmonary veins, as well as along the peribronchial interstitial routes to the mediastinum. After the distention is relieved, similar movements of air probably continue during subsequent respirations, especially if positive and negative intrathoracic pressures are enhanced by partially obstructed airways, painful "grunting" breathing, improper use of full body respirators, or completely ill-advised use of positive pressure resuscitation.

Wedge-shaped, subpleural hemorrhages are also seen on the pleural surface in the visceral and parietal pleura. The lung between the ribs, being expanded more than that opposite the rib, appears to sustain greater damage [110]. In the lung, interstitial, intraalveolar, and perivascular edema and hemorrhage lead to progressive hypoxia [178]. Atelectasis is also found [89]. The response of pulmonary tissue to trauma varies from species to species as does the time course and cause of death. A biphasic time course of death has recently been under study in air-blasted animals [70]. Varied sensitivity of pulmonary veins to tearing, varied lymphatic drainage of interstitial spaces, and other differences may account for the variation in bimodal patterns from species to species [156, 178, 191].

As in the case of blast, expansion of the alveoli and tension on the blood vessels lead to bradycardia and hypotension lasting for variable periods of time [42, 167]. Expansion of gas in the gastrointestinal tract may augment this response by vagal stimulation.

One factor which must be considered in humans 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 human death to be discussed below [119]. 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. These are covered in the next section.

Pneumomediastinum and Pneumothorax

The disruption of the alveoli leads to air dissecting along the peribronchial spaces. Passage of air along the lung roots can lead to pneumomediastinum and to subcutaneous dissection of air in the upper thorax and neck region [39, 91, 98, 131, 167, 178]. The dissecting bubble of air may make its way downwards along the aorta and esophagus into the retroperitoneum, and may rupture into the peritoneal cavity. Symptoms produced by air in the abdominal cavity may simulate acute abdominal conditions for which operation may be mistakenly performed. Air may make its way forward over the heart, whence it may give rise to a loud crunching sound, "Hamman's sign", with each heart beat. It may travel laterally into the vessel sheaths of the other lung, or backward along sheaths of the same lung into areas in which there is no leakage. Air may disrupt the mediastinal wall producing pneumothorax. Collapse of the lung tends to stop the leak, except in cases in which there is violent cough.

In the absence of pneumomediastinum, distal passage of gas along the peribronchial tracts or disruption of alveoli on the pleural surface leads to pneumothorax. Presence of a valving mechanism at the tear site can lead to tension pneumothorax with rapid respiratory and circulatory embarrassment. The tension is built up not during inspiration, but during forced expiration or cough or when the glottis is closed and the intrapulmonary pressure rises above atmospheric. Air continues to leak as long as the factor initiating the original break is operative. In some instances, the leak appears to be favored merely by respiratory movements, especially if they are of a dyspneic character. Even in the absence of frank pneumothorax, respiration is hampered by the splinting action of air in the connective tissues of the lung, preventing the escape of air in expiration and giving rise to dyspnea.

Chest pain, simulating angina pectoris, may possibly be caused by air pressing upon the pulmonary and mediastinal vessels. Circulation is compromised by the collapse of the pulmonary vessels under external pressure, causing venous stasis, and giving rise to cyanosis [153, 178]. Heart action may be inter-

fered with by air in the interstitial tissues of the lung and mediastinum in three ways: (1) the heart is pressed by the distended lungs which prevent heart filling; (2) decreased venous return or stasis due to air bubble pressure on systemic and pulmonary veins; and (3) by direct pressure upon the heart by air bubbles in the precardium and in the posterior mediastinum.

Unilateral tension pneumothorax results in progressive collapse of the lung and displacement of the mediastinum to the opposite side, producing serious respiratory embarrassment. In the presence of mediastinal gas, tension pneumothorax can convert to a bilateral type by disruption of gas into the previously unaffected side, leading to bilateral collapse of the lung.

Clinical conditions predisposing to spontaneous pneumothorax appear most sensitive to the pneumothorax-pneumomediastinum complex after explosive decompression (56, 73, 74, 131). Presence of congenital cysts or post-infectious and asthmatic emphysematous blebs or bullae on the pleural surface are probably the most common conditions of this type (103). Pleural adhesions cause subpleural vesicles at the site of insertion into the visceral pleura (154). Scar tissue vesicles can form distal to valve-like, post-inflammation scars at the bronchiolar level (68, 103).

Some of these blebs or bullae may communicate with the bronchial tree and show no expansion on x-ray when the subject exposed to low pressure in an altitude chanber; whereas others may be fully closed off and expand under these conditions (56, 177). This makes elimination of susceptible candidates far from foolproof. In the absence of frank bullae, air trapping can occur when mucous plugs, edema, or inflammation of bronchioles leading to an air space can act to reduce the V/A ratio of that portion of the lung and predispose it to rupture on decompression (151). Any bronchiole containing pathological structures leading to ball-valve mechanism can also explain appearance of focal, small, emphysematous blebs in an otherwise normal lung (3, 68).

Aeroemboli

As in air blast injury to the lung or in too rapid ascent in diving, gas can be forced into the pulmonary veins during explosive decompression and lead to embolization of the arterial tree (17, 19, 20, 40, 58, 81, 86, 91, 98, 118, 133, 155, 156, 167). The critical transpneumonic overpressure appears to be in the range of 50-80 mm Hg for both interstitial emphysema and aeroemboli in dogs (167). Greater quantities of air appear in the circulation when interstitial emphysema is present than with pneumothorax alone, although gas emboli may appear with neither present (133). As would be expected, the range of symptoms and signs vary from single, focal neurological defects (19, 91) to massive neurocirculatory collapse. The symptoms may appear suddenly after the chest pain or be preceded by prodromal symptoms of dizziness, headache, and angor animi. Focal sensory and motor deficits, paralysis, convulsions, unconsciousness and shock may quickly follow (62, 115). Severe and recurrent convulsions are often seen. Death results from coronary and/or cerebral occlusion with arrythmias, respiratory failure, circulatory collapse and irreversible shock. Figure 7 is a summary of the pathological sequences and cycles occurring in the nervous and other tissue distal to the air embolus.

External signs which distinguish the aeroemboli from other acute neurological or cardiorespiratory accidents are few. Premortem segmentation of blood in the retinal arterioles by gas emboli may be seen, if not gas bubbles themselves traversing the retinal vessels. Liebermeister's sign, consisting of sharply defined areas of pallor on the tongue, has been noted. Marbling of the skin is common. These symptoms and signs associated with sudden chest pain and hemoptysis after rapid decompression should focus attention on the possibility of aeroemboli.

In explosive decompression to a vacuum, the gas bubbles formed in the water vapor cavities of the heart will embolize the lungs as well as the periphery (vide infra). Both pulmonary and arterial emboli must therefore be considered. The effect of inert gases on these embolic phenomena will be covered under ebullism.

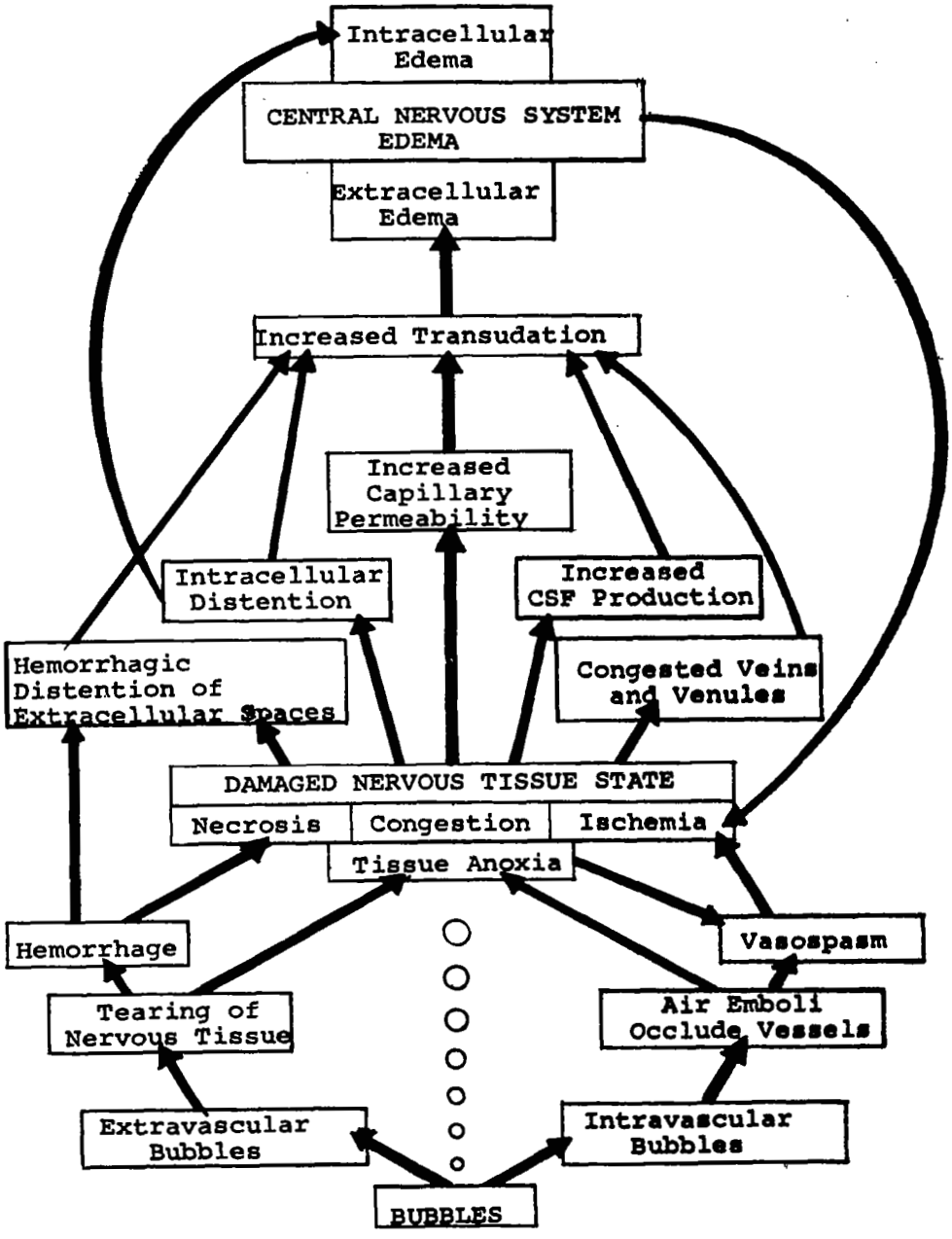

Figure 7. Suggested relationships between intravascular and extravascular bubbles, nervous tissue damage, and nervous system edema.
(After Erde [65])

Ebullism

Exposure to vacuum for several minutes after the explosive decompression can lead to further damage of the lung and to the ebullism syndrome. The pathological physiology of ebullism has received recent review [30, 165]. Only those aspects of the syndrome pertinent to the treatment of explosive decompression injury will be covered.

Early studies of explosive decompression of animals to very low pressures focused on the pathology to the lungs [13, 76, 89, 90]. 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) [13, 49, 61, 95, 105, 106]. Decompressions 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 occured within 0.2 seconds than when a decompression time of 1 second was used [61].

Denitrogenation appeared to reduce the incidence and severity of lung damage, possibly by reducing the inert gas entering the vapor bubble in the right heart [165]. 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.

Exposure of squirrel monkeys results in similar findings [166]. 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.

The chimpanzee can survive without apparent central nervous system damage (as measured by complex task performance), the effects of decompression to a near vacuum for up to 3.5 minutes and return within approximately 4 hours to baseline levels of functioning [105, 106]. 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 post-mortem. Death was attributed to failure in the conducting mechanism of the heart.

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[157]. 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 [95]. 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[13, 33, 48, 49, 61, 106]. These are probably a result of infarct by inert gas bubbles. The problem is aggravated by the concomitant generalized hypoxia. Cooling of the pulmonary blood to as low as $27^{\circ}C$ by rapid evaporation in the alveoli while circulation is still intact, may delay the cardiac and cerebral response to ischemic hypoxia [106, 157]. The short cooling time precludes a more effective temperature drop.

Alteration of the gaseous environment may affect the ebullism syndrome. Analyses of the changing gas compositions of subcutaneous vapor pockets by different investigators have given equivocal results [165]. 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 (165). 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 (α_{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.

Review of Human Accidents Involving Explosive Decompression

A review of the accidental trauma to humans during explosive decompression to altitude appears in order.

Two cases were reported in Germany during World War II at the Erprobungstelle Rechlin [19, 57]. Subjects were decompressed in a period of less than 0.25 seconds from 9800 ft to about 32,000 and 39,400 ft. In the first case, fifteen seconds after explosive decompression, loss of consciousness set in. The subject suddenly dropped the oxygen mask, which he had held to his face as planned, and collapsed. During the descent, cyanosis and clonic contraction of the left facial muscles were noted. Later the cyanosis disappeared. When awakening 3 to 5 minutes after the incident, he suffered from a severe speech disorder and ataxic defects of locomotor function. Half an hour later, both defects had disappeared. Subjective sensations during the decompression were described as a painful blow in the chest. Then the air flowed from the thorax out of the mouth. It was impossible to inhale. Unconsciousness followed until ground pressure was reached. When leaving the chamber, a feeling of unsteadiness persisted "like being drunk." Also, a speech disorder was noted. Thereafter, pains in the chest "like after a K.O." and pains in the larynx "like a swallowed sore throat" persisted for a longer period.

The other subject held his breath at peak inspiration noting that he wanted to let the air escape from the lungs through the mouth without strong resistance of the lips and without closing the glottis. At the instant of explosive decompression, he suffered an intense pain in the thorax. At once, he donned the fighter pilot oxygen mask and breathed oxygen, ordered the descent through the intercommunication system and managed to open

a quick discharge valve. During the descent, which lasted 25 seconds, he noted that his right arm had become numb and motionless. The pain in the thorax decreased a little. He noted a feeling of soreness in the trachea and in the large bronchi. Respiratory movements were painful. About two minutes after reaching ground level, the numbness of the right arm waned within about 5 seconds. He felt very weak and lay several minutes with his head resting on his left arm, bent over the table. Gradually, he felt the onset of nausea, then felt like retching. A cold sweat burst out over the whole body. When he asked for an emesis bowl, speaking was very difficult because of the pains. The ill feeling slowly faded. He remained sitting calmly for some minutes and then left the chamber. No persistent symptoms of nervous defects were noted with the exception of a brief "swimming of the right eye." The sore feeling in the thorax was still persistent 3 hours after the explosive decompression, although it had decreased. Coughing was still very painful. Consciousness was maintained during the whole procedure.

The following day, pains were felt when coughing, bending, and taking deep breaths. On the second day after the incident, deep breathing was still painful.

In 1945, Clark reported two cases of collapse with pneumomediastinum which occurred during rapid decompression from 8,000 ft to 31,000 ft in 0.5 seconds[39]. The original report of the symptoms was not available in time for this study.

In 1954, Luft reported a fatal case at the USAF SAM [124]. Formal statement of the accident and postmortem report are available[119] (see Appendix III).

Three more cases were reported by Holmstrom [91]. The subjects, all healthy men, were decompressed from 8,000 feet to 22,000 feet in approximately 2.0 seconds. Such an exposure appears well within the safe limits for non-breathhold conditions as noted in Sections I and II of the present study and no untoward responses were expected. The first subject was a 42 year old pilot who inadvertently held his breath at the instant of

decompression. He immediately experienced an upper abdominal pain of moderate severity and then lost consciousness. His respirations were noted to be irregular and in the nature of short gasps. Consciousness was regained on reaching ground level about one-half minute after the decompression. Blood pressure at that time was 104/62 with a strong regular pulse. He had an ashen pallor and cold, clammy skin. Physical examination was otherwise essentially normal. The patient improved rapidly. Within a few minutes his color returned to normal and the blood pressure stabilized at 130/76. His abdomen was soft without areas of tenderness. Neurologic examination was normal. Chest x-ray and electrocardiogram showed no abnormalities.

This case is characterized by upper abdominal pain, sudden alteration in a previously normal respiratory pattern, and syncope. The role of pain in precipitating vasomotor collapse and syncope was noted. On the other hand, pulmonary and cardiovascular reflexes initiated by an increased intrapulmonary pressure must also be considered (vide supra). The dissection of a small amount of air along the posterior mediastinum to the posterior peritoneum must also be considered.

In the second case, only emboli were probably involved. A twenty-three-year-old altitude chamber technician is believed to have held his breath at the time of decompression. Almost immediately he noted generalized chest pain and collapsed about twenty seconds later. There were no voluntary respiratory movements. Artificial respiration was begun at once. His skin was cyanotic, cold and clammy. Blood pressure was 126/80 and the pulse was regular at 90 per minute. Voluntary respiration began about two minutes after the rapid decompression but he remained unconscious for about five minutes. On recovering consciousness, he noted weakness of the right arm, numbness of the face, headache and blurred vision. He was nauseated and vomited. The paresis and numbness disappeared rapidly but the clinical picture of shock, an ashen pallor with cold wet skin, persisted for a half hour. His blurred vision cleared about

five hours post decompression, the nausea and vomiting lasted six hours and the headache subsided in about eight hours. An x-ray of the chest was normal. The patient has been well and active since.

In the third case, a thirty-three-year-old pilot was near the peak of inspiration when decompression started. Initially, he noted the expulsion of air from his nose and mouth. This was followed by a severe left parasternal pain. Within a few seconds he felt weak and giddy and shortly thereafter became unresponsive. His respirations were irregular, shallow and associated with a hacking cough. During the descent to ground level he exhibited several uncoordinated twitching movements of the upper extremities. The pulse was 45 per minute about two minutes after the decompression. He was in shock and had an ashen pallor and cold, clammy skin. The patient was unconscious for about ten minutes. In the meantime the blood pressure and pulse stabilized at 130/76 and 80 per minute, respectively. The patient had a complete quadriplegia, as well as the loss of tactile sensation for the initial twenty minutes following the decompression. Coincidental with the return of motor function he showed a marked improvement in color and general appearance.

On admission to the hospital, about one-half hour after the decompression, he complained of severe chest pain which was associated with inspiration. Positive physical findings were confined to the chest where a to-and-fro, crepitant, friction rub associated with the cardiac rhythms was heard in the fifth left intercostal space. Neurologic examination was normal. Chest x-rays taken about one hour after the incident showed a pneumomediastinum, a small pneumothorax of the left apex and air in the soft tissues of the neck. The lungs were clear. An electrocardiogram was normal. Subcutaneous emphysema of the neck and upper thorax was noted several hours later.

The patient improved rapidly. He had a headache which subsided about six hours after the rapid decompression. The friction rub disappeared on the second day and the chest pain was much improved. An x-ray taken two days after the incident showed that the pneumomediastinum

had cleared but that the pneumothorax was still present. By the sixth day the pneumothorax had been absorbed and the lung was completely expanded. The patient was discharged from the hospital and has been well and active since.

In these cases, breathholding or expansion of the chest at deep inspiration appear to be the aggravating conditions involved. In the recent explosive decompression accident at MSC, the subject decompressed his pressure suit in a vacuum chamber by disrupting a connection on his chamber umbilical hose [85]. The pressure curve is noted in Figure 6. As discussed above, the effective V/A ratio was probably below that for damage to the lung. The subject lost consciousness in 12 to 15 seconds after decompression. Clonic and tonic movements of the feet were noted. After only 20 seconds in a vacuum, the suit was recompressed to 3.7 psia. Within a period of 27 seconds the chamber was at 6 psia. He regained consciousness at this time. There was no recollection of chest or abdominal pain in the accident report. The subject was found to be pale but not cyanotic. Erythema of both tympanic membranes were noted. Neurological and chest examinations were unremarkable. Electrocardiogram was normal and unchanged from his base line record. Vital capacity, both total and timed, were normal and also unchanged, as were PA and lateral views of the chest. An electroencephalogram was accomplished approximately one week after the incident and was reported as a normal tracing. No base line EEG was available. Several days following the test the subject noted diminished taste sensation. This lasted approximately four days and then remitted spontaneously. He apparently suffered no ill effects from this incident.

The most recent human episode of explosive decompression occurred in a C-141 Flight [14]. The aircraft with 96 passengers on board was flying at 34,600 ft (185 mm Hg) with a cabin altitude of 6500 ft (598 mm Hg). A cargo door of 70 ft^2 (6.5 m^2) suddenly blew off releasing air from a cabin of 7350 cu ft (208 m^3). Under these reported conditions, the

P_i/P_f = 3.26, P_i-P_f = 414.9, P_l (fig. 2) = 2.25, V/A ratio = 32, t_c = .102 sec, and t_d = .23 sec. This calculated time is close to the approximation of 0.3 sec reported by the USAF. Only one of the 96 subjects, many of whom were probably "half-asleep", experienced what may have been aeroembolic phenomena. It is reported in the preliminary summary of the investigator's data only that this passenger's "symptoms and progress are undoubtedly the results of cerebral airembolic phenomenon. The subject was air evacuated to Hickham Field and placed in a recompression chamber for some 24-48 hours. During this period he experienced two or three convulsive seizures. Following decompression to sea level, he experienced one additional seizure, after which he appeared to recover very rapidly and had no further convulsive episodes. He has a retrograde amnesia and is to undergo a complete neurological study." Follow-up data on this patient were not available in time for inclusion in the present study.

In view of the V/A ratio of 32 and P_i/P_f of 3.26, one can speculate that this individual probably held his breath during decompression or had some underlying pathology weakening his lung structure (See discussions of Figure 5 and Table 2).

The rapidity of symptoms in the above case of probable aeroembolism precludes the presence of slow decompression sickness as a cause of his difficulty. A latency period of three to five minutes at altitude is usually required for enough gas to evolve from body fluids to cause symptoms (147, 165).

In addition to this one manifest case of air embolism, at least four other passengers and one Loadmaster were observed to be unconscious. However, on the basis of available information, it is uncertain whether these resulted from lung injury or altitude hypoxia. While only five passengers were observed to be unconscious, it is probable that the majority of passengers were severely hypoxic and/or incapacitated, although not manifestly unconscious, for at least short periods of time. Had decompression occurred during the daytime hours and/or during the cruise

phase of flight, a higher percentage of collapse would have occurred. In this case, the lower metabolic requirements of the seated subjects, all relatively inactive or half asleep, greatly favored the passengers. One of the two loadmasters standing in the forward section of the cabin was blown into surrounding aircraft structure, causing unconsciousness and an observed convulsive seizure. He was aided by a second Loadmaster who gave him oxygen from a portable supply bottle. The Loadmaster recovered completely and was subsequently able to assist in the treatment of other passengers. No report of chest pain was noted.

In addition, there were a large number of ear complaints and at least one ruptured ear drum. It must be assumed that the majority, if not all, of the ear problems resulted from the rapid descent rather than the force of decompression as such. It is possible for a force of this magnitude to cause rupture of a diseased or marginal ear drum. In most cases, the positive pressure in the middle ear will blow the eustachian tube open, thus relieving the middle ear of excessive pressure before rupturing the ear drum.

These case histories represent all of the formally reported episodes of lung damage and related symptoms following explosive decompression. They illustrate the range of problems and their sequelae more formally outlined in the first part of this section. Such varied patterns must be considered in the discussion of therapeutic management of explosive decompression injury.

IV. Therapy in Explosive Decompression Emergencies

A. Prophylactic and Preventive Measures

In view of the hazards discussed above, it appears imperative that the suit design, especially the seals,be examined for reliability under such conditions as internal overpressure, physical trauma, metal fatigue, etc. Simulation of recurrent use of the same suits during testing of PLSS systems and other components, in vacuo, should be used.

Selection of test subjects should also be kept in mind. While it is true that spontaneous pneumothorax is statistically rare in the general pilot population [73, 74], the unique situation of suit testing in vacuum chambers should dictate that special care be taken in the selection of subjects. As covered in Section III, some subjects with occluded bronchi and distal emphysema will show cineroentgenographic evidence of expansion of the blebs or bullae when taken to altitude. This maneuver should be used in the original selection examination of potential candidates. Transient blockage can occur. The death described in Appendix III may well have been caused by a viral bronchitis. Cinerontgenographic examination in an altitude chamber may pick up such transient blockage, but it would be doubtful that such a maneuver is warranted prior to each individual suited exposure in the vacuum chamber.

Another test in the original selection of subjects for vacuum chamber work could be mask breathing of oxygen at 15 to 20 cm of H_2O positive pressure at altitudes of 45,000 ft. Such maneuvers are usually employed in the training of USAF chamber technicians, but should be employed if this is not the case in NASA test operations. Lung disruption during a carefully monitored selection experiment of this type would be far safer than if the subject were accidently decompressed in a vacuum chamber during a suit test. Fluoroscopy during positive pressure breathing or during expiration against a closed glottis may show up some blebs [143]. Stereo x-rays of the lung apices during exhalation in the lordotic position aid diagnosis (132, pers. comm).

Explosive decompression of potential vacuum chamber subjects under the usual USAF training schedules of 8000 ft (564 mm Hg or 10.91 psia) to 23,000 ft (307 mm Hg or 5.9 psia) in several tenths of a second is unlikely to cause lung damage in a normal individual, but may do so in an individual with previous lung disease, even when breath is not held. Once again, such testing under controlled conditions would present a much less dangerous situation than if previously diseased lungs were disrupted during suit testing. It is doubtful, however, that such controlled decompression would be warranted each day prior to vacuum chamber work to select out those individuals with transient bronchial blockage. The ensuing operational problems would probably far outweigh the safety value of such a program.

In no selective test should breathholding be used during the decompression.

Presence of pertinent lung pathology on routine x-ray, on cineroentgenography at altitude or during any of the provocative tests should eliminate the subject from vacuum chamber exposure. Surgical therapy of some of these lung lesions has led to recovery of flying status in the USAF [59, 73, 132]. Such could be considered in the case of space suit subjects in vacuum chamber work but each case should be handled individually.

In no case should subjects with symptoms of respiratory tree infection, or any generalized viral infection for that matter, be allowed to take part in vacuum chamber work in the presence of symptoms. One should consider use of the provocative tests outlined above in clearing the individual for test operations following recovery from his disease.

B. Treatment of Subjects Showing Symptoms after Explosive Decompression

In view of the complex syndromes outlined above, one must consider individually and in combination, therapy of all of the pathological conditions.

1. Lung Contusion

The symptom of chest pain after explosive decompression should lead one to suspect alveolar disruption, pulmonary hemorrhage and edema, pneumomediastinum, pneumothorax (simple and tension), hypoxia, and aeroemboli. Treatment of simple lung contusion has been a problem in the handling of air blast victims [192, 193]. The subject has recently been reviewed from the point of view of space operations [30].

If the lungs are severely injured, breathing may be slow, shallow, and weary, often with extreme expiratory dyspnea [41]. Panting respiration in other cases is usually associated with complaints of tightness across the chest and varying degrees of chest or abdominal pain. Coughing will occur, but is usually not an early manifestation [38].

Hemoptysis may appear, often well within an hour, and tends to be repeated [38]. Frothy blood coming from the mouth and nose is usually

a bad prognostic sign [41]. Pulmonary hemorrhage will lead to airway obstruction varying in degree with the extent of the injury. This, in turn, increases the rate of respiration. There seems to be little or no evidence for a central nervous system role in this process unless there is evidence of associated head injury or cerebrovascular air embolization. It has been suggested that there is a natural vasoconstrictive mechanism in the lung which, after blast injury, becomes manifest over approximately 15 to 20 minutes, limiting hemorrhage from smaller vascular disruptions, and promoting clots in the torn ends of the blood vessels [35]. There seems to be little doubt that in some individuals, virtual suffocation takes place when continued pulmonary bleeding and subsequent development of pulmonary edema obliterates the available air-exchange surfaces, decreasing oxygen saturation and ultimately resulting in a state of generalized hypoxia. The pulmonary edema may be sudden in onset and appear at any time in the first few days of closed compression injury to the chest [131]. Sodium ethacrynate, given intravenously in a dose of 100 mg, is effective in acute pulmonary edema from other causes and may be considered in the present situation.

One must consider the fact that serious hemorrhage is a constant problem and attempt to maintain the pulmonary vascular bed in as constricted a condition as possible. Physical exertion tends to aggravate pulmonary bleeding. This fact gives strong support for early and complete immobilization of an individual injured by explosive decompression [38, 83, 188, 192]. The workload on damaged lungs and heart must be minimized in order to reduce the risk of incurring further air embolization, pulmonary hemorrhage and edema, and cardiac decompensation. Sedation must be used with caution to prevent masking of various progressive signs which would indicate serious injury.

Progressive cyanosis and air hunger are signs that the decline in arterial oxygen saturation resulting from the interstitial and intraalveolar edema must be corrected [34, 44, 41, 178]. Is 100 percent oxygen indicated? Pulmonary insufficiency and shock certainly require oxygen therapy. Administration of 100 percent oxygen might also be indicated for the treatment

of local hypoxic states such as cerebral ischemia and myocardial ischemia. But oxygen may be a two-edged sword.

First of all, the atelectatic tendency of pure oxygen must be kept in mind [59]. Secondly, the problem of vasomotor control in the lung should be considered. Since pulmonary hypoxia and reflexes caused by lung damage markedly constrict the pulmonary vessels, administering pure oxygen, which is a proven pulmonary vasodilator, may conceivably aggravate the tendency for lung hemorrhage and edema, and air embolization [7, 30, 41, 67, 161, 148]. Whether or not increased oxygen tensions can actually overcome the protective vasoconstriction in a lung after decompression injury is still open to question. Failure of vasoconstriction has been shown in animal experiments to aggravate bleeding from vessels, disrupted by blast, especially if clotting is inadequate [35]. Favoring the use of oxygen in the immediate post-decompression period is the fact that any bubbles of pure oxygen in the cardiovascular system will be much more rapidly absorbed than bubbles containing an inert gas such as nitrogen (vide infra) [165]. Because of the high mortality and serious sequelae of air embolization, oxygen should be used immediately after decompression, in the hope that adequate reflex vasoconstriction and clotting will seal disrupted pulmonary vessels. Definitive studies on the effect of oxygen on vasoconstriction in a traumatized lung appear in order [30].

In the absence of progressive or new embolization, should 100 percent oxygen be given beyond 30 minutes, which is considered the upper limit of the embolization period? [190, 191]. Only in mice treated with heparin to decrease clotting is there a distinct tendency for bleeding to restart 15 or 20 minutes after air blast [35]. Because of the pulmonary vasodilatory effect of oxygen, the period of protective vasoconstriction may be prematurely shortened by this therapy. In view of our inadequate data, one can suggest that only cyanosis or other signs of hypoxia should be an indication for continuing or resuming oxygen administration.

Along the same vein, the choice of drugs in the treatment of shock hinges on pulmonary vasomotor factors. This problem will be covered below in the discussion of aeroembolism. Because most bronchodilators tend to be pulmonary vasodilators, addition of helium to the oxygen should be considered in treating bronchospastic or asthmatic response to trauma [6, 132].

An analgesic, sedative or narcotic drug must be given with caution in the immediate post-decompression period in order to prevent the possible masking of signs and symptoms which indicate serious injury. Antitussives, such as dehydrocodeinone bitartrate, may be used during the first few days after exposure to prevent hemorrhage due to excess coughing. A suitable broad-spectrum antibiotic may be required for peritonitis or pneumonia [149]. It has been suggested that a suitable antibiotic-cortisone combination be administered as a prophylaxis for infection and to minimize pulmonary fibrous tissue formation for at least two weeks if severe lung damage has occured [30].

2. Pneumomediastinum, Pneumothorax, and Hemopneumothorax

The acute emergency presented by these entities has been covered in Section III. A large pneumothorax, particularly of the "tension" variety, can produce cardiorespiratory embarrassment characterized by severe chest pain, dyspnea, hemoptysis, cyanosis and "shock". In addition, mediastinal emphysema, indicated by distension of neck veins and livid suffusion of face, can lead to cardiac insufficiency. When a pneumomediastinum has converted to a pneumothorax, treatment of the latter may relieve the former (vide infra). Acute respiratory embarrassment accompanying subcutaneous emphysema about the neck and shoulders cannot usually be relieved by surgical incision just through the skin of the neck or thorax. In severe cases with cardiac embarrassment, an incision in front of the infrathyroid portion of the trachea through the pretracheal fascia will be required to decompress the mediastinum. Continued suction in the incision is often necessary to prevent reaccumulation of gas [9].

The treatment of acute pneumothorax has received much study [9, 10, 21, 50, 54, 94, 99, 102, 104, 158, 175, 186, 194]. Simple pneumothorax should be suspected if the episode of chest pain is not followed by rapid dyspnea and cyanosis. Tympany on percussion, absent breath sounds, and typical x-ray appearance of a collapsed lung with occasional pleural pattern of hemothorax can be found.

If the pneumothorax pocket is small (<30%), not increasing, and not distressing the patient, he may be treated conservatively. Air is allowed to absorb from the pleural space. This will occur at the rate of 1.25 percent per day depending upon the normality of the pleura, the patient's activities, and the nature of the gas. Oxygen would be most rapidly absorbed and so treatment of a patient decompressed in a suit containing 100 percent oxygen may be handled more conservatively than if an inert gas were

present. The patient's activities are usually restricted at least for a few days at the onset. This course should be reserved for cases where only a mantle of air is present about the lung or collapse is less than 30 percent.

If the amount of air is rather large, the duration of disability may be reduced somewhat by aspiration of air from the pleural cavity, instead of waiting for spontaneous absorption. In the past it has been advised to wait twenty-four to forty-eight hours before aspirating air as an elective procedure if the patient is in no real distress, thus giving the pleura time to seal the leak [102]. However, early aspiration or the creation of a high negative pressure in the pleural cavity is now thought to be more effective in in the long run [132] (pers. comm.). The exudative coating often seen over traumatically collapsed lungs interferes with efforts at delayed expansion. Complete aspiration of all air from the pleural cavity may bring the expanding lung into contact with the tip of the aspirating needle, producing an accidental pneumothorax on top of the original spontaneous collapse and care must be taken.

The best site for air aspiration with the patient propped up slightly in dorsal recumbent position is the second anterior interspace in the midclavicular line, if the lung is free of the chest wall in the area. If the pneumothorax is large, aspiration in several stages may be advisable, for removal of most of the air at one sitting may give severe discomfort and cough if the lung has been collapsed and atelectatic for a time and does not expand readily [53]. Insertion of an intercostal catheter at this site, as described below for tension pneumothorax, may be reserved for those cases where collapse is 50 percent or more or if the leak tends to recur. Chest x-ray at least every 12 hours should be used to follow the leak in the first few days. Quantitation of percent collapse (by volume) is difficult, but possible.

In 2-8 percent of benign spontaneous pneumothorax at sea level, hemopneumothorax is present [94]. Transfusion and thoracentesis may be required, the latter to remove a fluid medium suitable for bacterial growth. If aspiration and catheter drainage are not required for removal of air (<30% collapse), no drainage is usually necessary for the blood [50].

Blood spilled into the pleura is also an irritant, and in consequence there is outpouring of pleural fluid. The nature of this effusion varies, depending, probably, upon amount of bacterial contamination and degree of lung trauma. If this effusion is serous, it merely dilutes the blood that is present and this will ultimately be absorbed or can be aspirated. On the other hand, if the effusion has changed in character, it will become exudative in type and richer in fibrin. This deposition of fibrin over the lung will, by subsequent organization, form a coat which can restrain expansion of the lung and entrap it. Aspiration of the more fluid contents of such a hemothorax will not change the ultimate picture. The proteolytic enzymes instilled at this time, when the fibrin has begun its organization, are generally incapable of reversing the process and permitting the lung to expand even though they are entirely capable of lysing unorganized fibrinous masses. They have been discarded as a prophylactic measure in the treatment of uncomplicated hemothorax (50).

The establishment of the diagnosis of continued intrapleural hemorrhage is not based solely upon fluid accumulations within the hemithorax, because a small amount of blood may be followed in 12 to 24 hours by the diluting pleural effusion. Thus, a mere increase in amount of pleural fluid does not necessarily mean further bleeding. The re-accumulating fluid must be whole blood. Measurement of hemoglobin and hematocrit on pleural blood may assist in this determination. On the other hand, if it is suspected that a large vessel may be the cause for the initial blood seen in the pleura, it is probably best to open the chest and assure oneself that this bleeding vessel is well secured and the damaged lung repaired. Also, if the hemothorax clots, rendering aspiration impossible, or becomes infected, thoracotomy is indicated. Mortality rates of 14 percent have been reported when hemorrhage accompanies spontaneous pneumothorax (186).

In tension pneumothorax, rapidly progressive dyspnea and cyanosis are present. If dyspnea occurs in a patient at rest with a unilateral pneumothorax it is likely that dyspnea is due to some factor other than a

simple pneumothorax [53]. Absence of breath sounds, hyperresonance of the chest,cardiac displacement away from the affected side, pallor and a poor pulse are found in tension pneumothorax. As air accumulates under pressure in the pleural cavity,the mediastinum and trachea are displaced. When accompanied by pneumomediastinum, the air may be forced into the opposite pleural cavity leading to fatal bilateral collapse of the lung [152].

In dire emergencies, a trocar and cannula should be immediately introduced between the ribs in the second intercostal space, two inches from the edge of the sternum. This is followed as soon as possible by the insertion of a 16 to 28 F gauge soft rubber catheter (with additional side holes) connected to a water trap. Presence of blood should prompt use of the larger bore catheter to avoid plugging. Catheters smaller than 16 gauge will often plug up even in the absence of frank blood. It has been suggested that for cases not so urgent, the same site can be infiltrated with 10% procaine and a #18 gauge needle used to aspirate the air. The pressure in some of these pockets may be sufficient to blow the plunger of the attached syringe across the room, decompressing the pocket considerably through the open needle. Although additional air can be expressed through the needle by requesting the patient to strain, the possibility of further damage to the lung should discourage such a maneuver. It should also be considered that recurrence of the positive pressure in the chest is most common. In one series of spontaneous pneumothorax, the average re-expansion with catheter was 3 days [175]. In view of this fact, procaine infiltration and the use of the trocar and catheter is, in the long run, the soundest approach upon any sign of tension pneumothorax, with or without bleeding [50]. If the catheter is inserted through the cannula directed slightly upwards through the second interspace for a couple of inches it will come to lie parallel to the surface of the expanding lung and be lifted up by it as it expands and will adequately empty the air pocket and not interfere with expansion of the lung.

The older literature suggests that single, water-seal bottles be used to keep the pressure in the pleural cavity at 1 cm of water positive pressure in order to aid healing. Current surgical philosophy suggests that negative pressure should be maintained right at the onset of therapy [132]. A

suction machine with fine pressure control may be attached to the catheter but care must be exercised to avoid too high a negative pressure or the leak may be reopened or perpetuated. It has been suggested that it is desirable to have a rather large output volume at a very low negative pressure of not over four or five centimeters of water, controlled by a three-bottle system. With such treatment, in the majority of patients, the leak will cease and the lung expand to the chest wall rather rapidly [102]. However, catheter kinking and plugging with exudate often dictates greater negative pressures (Ref. 132) (pers. comm). When the lung is well expanded and seems to stay out for twenty-four to forty-eight hours, as shown by stoppage of bubbles in the water trap as well as by upright chest x-rays, the catheter may be removed from the pleural space.

If there is fluid or blood to be removed from the pleural cavity, this will not be accomplished through an anterior catheter. This must either be aspirated through a posterior site or a posterior basal catheter. When instituting catheter drainage in the presence of hemothorax, it has been suggested that it is sometimes of advantage to use a posterior apical catheter in the posterior third interspace and position the patient so the area of the catheter is dependent before suction is started [99]. This allows for blood to be evacuated first and avoids secondary thoracentesis. However, posterior drainage is more painful and much more uncomfortable for the patient [132].

Most patients with spontaneous or traumatic pneumothorax will seal the leaks and expand the lung under such a program. The catheter should be left in for twenty-four to forty-eight hours after air leak has ceased and the lung is shown to be expanded by chest x-ray examination. There are, however, a few in whom larger leaks do not seal promptly, and continuous air leakage occurs. This is particularly true with rupture of a larger bleb or cyst in the lung. These patients demand prompt surgical intervention and suture of the leak if the patient is to be saved.

Antibiotics may be given where air leak is present, especially when catheter drainage is required. Mild temperature elevations are often seen during the course of drainage [175]. Pneumonitis always lurks as a

problem. Bacterial pneumonias are often a late problem in air-blasted animals [30]. Patchy fibrosis is another late sequela in a lung damaged by air blast [38]. Prophylactic antibiotics may reduce such complications after explosive decompression injury, though this has not been formally established.

3. Aeroembolism

As discussed in Section III, air passing into the pulmonary veins may be ejected into the coronary, cerebral, or other branches of the arterial tree. Focal or diffuse neurological signs, cardiac arrythmias, or failure, and the other signs of arterial embolization should lead one to immediate action in order to minimize the blockage of left ventricular outflow by air bubbles and prevent the coronary and cerebral arteries from receiving the emboli. The usual "mill-wheel" murmur over the heart often heard when air enters the right side from the systemic veins is usually not heard when the gas enters the heart via the pulmonary veins [8, 9]. However, as noted above, ebullism occurring from exposure to vacuum for several minutes may lead to simultaneous gas bubbles in the right side of the heart and may give this murmur in humans.

The patient should be tilted so the head is low. A Trendelenberg position of about 30° has several advantages in treating arterial air embolization. In this position there is less chance for intracardiac bubbles to block the dependent outflow tract to the aorta and less chance for escaping bubbles to enter the cerebral circulation via the dependent orifices of the carotid arteries along the aortic arch. As will be covered below, dissipation of bubbles in the cerebral circulation is also enhanced. There is still a question regarding that rotation of the body which will minimize the ultimate course of bubbles in the left ventricle. If a patient is turned on his right side so that the apex of the left ventricle is uppermost, the air bubbles will tend to fill the apex and thus keep the outflow tract of the ventricle free of large air bubbles (Reference 187, page 144). However, rotation to the left, half-prone position will rotate the coronary ostia to a dependent position and reduce the tendency of escaping bubbles to enter the coronary arteries. See Figure 8.

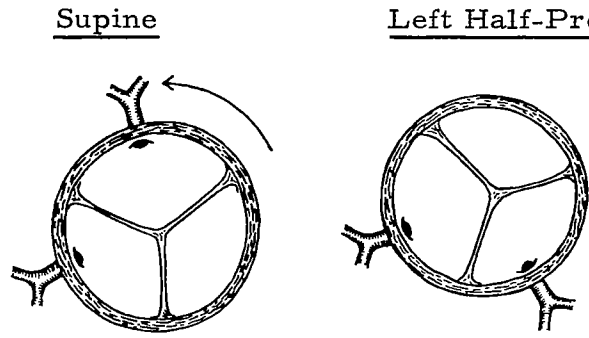

Figure 8. Effect of Body Position on Passage of Gas to the Coronaries.

With the mouths of the coronary arteries dependent, air cannot enter them. (Head to toe view).

(After Bailey [9])

Rotation of the body will probably have little effect on the passage of bubbles through the carotid ostia as long as the head is well depressed [79]. In this position, bubbles will tend to hug the lesser curvature of the aortic arch and pass along the descending aorta to the periphery of the arterial system. During open cardiac surgery, the right side is kept down so that air bubbles trapped in the apices can be aspirated prior to restoration of the circulation. The patient is often kept in this head-and right-side-down position for up to 3 days to prevent sudden release of bubbles to the periphery. One might argue that there should be more concern about sequestering the major bolus of gas than about the path of bubbles in case of inadvertent release. However, examination of a heart model suggests that in the 30° Trendelenberg position, rotation of the body should make very little difference in localizing the gas at the apex of the left ventricle. The left half-prone position would therefore probably be of greater advantage in that the coronary circulation would be more free of embolization.

In addition, bubbles in the right ventricle resulting from ebullism must also be considered. The frothy mass in the right ventricle often seen in animals exposed to vacuum for several minutes can be ejected to the lungs and intensify any contusive damage. In venous gas syndromes, a

mill-wheel murmur is often heard over the precordium and if patient is in the upright position at the time of accident, gas bubbles can actually be felt over the distended jugular veins [8, 9]. Gas in the venous system is usually treated by head down position to keep bubbles from rising into the superior vena cava and cerebral veins and to loculate the gas bolus in the apex. However, the left-side-down position is recommended to keep the gas bolus in the right apex as shown in Figure 9.

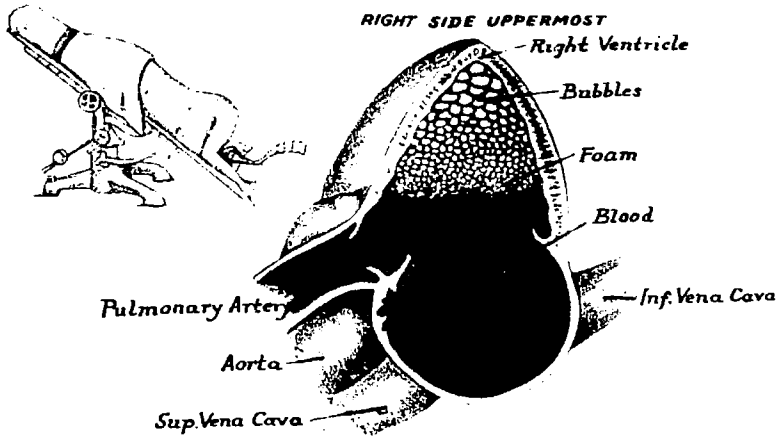

Figure 9. With the Patient on His Left Side and Tilted Head Downwards, Air in the Right Side of the Heart Rises Towards the Apex.
(After Bailey [9])

One might consider that a subject, exposed to explosive decompression in a vacuum and recovered within a minute of exposure so that consciousness is regained shortly thereafter, should be quickly examined for neurological signs and cardiac arrythmias. The demonstration of focal neurological defects in the presence of chest pain and in the absence of a mill-wheel murmur over the right heart, should prompt a left-half prone position for there would be more concern about gas in the left ventricle. If exposure has lasted for more than two minutes, the subject is unconscious, and a mill-wheel murmur is present in the precordium, a head-

down and left-half-front position would probably also be a good plan. This would be effective as long as both apices are kept above their outflow tracts. Further experimental work is required for optimizing the body positioning for simultaneous right and left-sided cardiac gas emboli.

In the face of cardiac standstill of more than 3 minutes duration, it has been suggested that aspiration of the left ventricle through a 3 inch long, #15 needle passed along the left xiphoid margin and directed backward and toward the left axillary line may be of value as a heroic measure. The needle will penetrate the apex of the left ventricle where the air is localized by body positioning and where there is less chance of disrupting a large branch of the coronary artery. If gas appears to be present in the jugular vein and a mill-wheel murmur is heard on the right side, the needle may also be directed from the left paraxiphoid site toward the right axilla in an attempt to aspirate the right ventricle as well. If aspiration of the ventricles does not lead to resumption of heart action, these maneuvers should be followed by the administration of epinephrine ($\frac{1}{2}$ to 1 cc of 1:1000 solution); Na bicarbonate (50 cc containing 3.7 grams); and Ca gluconate (10 cc of a 10% solution) through the needle in the left ventricle [4, 79, 80]. Acidosis of the myocardial tissue responds to the bicarbonate; myocardial tone is improved by the Ca gluconate; and epinephrine improves efficiency of the myocardium. If an EKG shows the heart to be in ventricular fibrillation, 100 to 200 watt seconds pulses of DC current may be applied through large defibrillator electrodes. The pulses may be increased progressively to 400 watt seconds. Beyond this pulse size, little is gained and the heart is actually damaged [79]. (See ebullism for drugs of choice in ventricular arrythmias)

If circulation is not restored by these maneuvers, thoracotomy and cardiac massage should be attempted. Air aspiration should always precede stimulation of the heart by drugs or massage in either the open chest or closed chest condition. This avoids ejection of gas emboli and decreases the excessive stretching of the myocardium which reportedly renders the muscle incapable of contraction (Reference 187, page 131). After pericardiostomy, the left and right ventricles should be aspirated by needle under direct view.

If air emboli are seen in the coronary arteries of the exposed heart (pericardium opened), it has been suggested that the ascending aorta be cross-clamped and firm cardiac compression made to build up a coronary hypertension sufficient to move the air into the venous channels leading to the right side of the heart. External finger massage of the visible air bubbles may be tried [79]. After the heart is filled with blood or saline, defibrillation may be attempted with a DC pulse of 10-20 watt seconds, progressively increased to no more than 50 watt seconds. The above intracardiac drugs may be used again as a last resort.

In view of the times involved in the above sequence, it would appear that thoracotomy and direct cardiac massage will probably be futile but may be worth attempting. A major problem is the necessity for external cardiac compression and artificial respiration to give enough time for cardiopulmonary resuscitation in cases of cardiac arrest not involving gas emboli [80]. Artificial respiration with patient on the left side has been used in the presence of air emboli from the systemic veins [145]. As mentioned above, care must be taken during pulmonary resuscitation in the case of contusion of the lung after airblast or explosive decompression. External cardiac compression prior to attempts at aspiration of the ventricles by paraxiphoid needling appears in itself hazardous, yet the circulation to the brain must be kept going. Cardiac compression in the deep Trendelenberg position may reduce the ejection of bubbles through the mechanism noted above. If operational requirements force a delay beyond 3 minutes from the time of cardiac arrest to attempts at aspiration of the ventricles, external compression in the Trendelenberg position should probably be attempted.

Once emboli have landed in the cerebral arterial tree, a modified Trendelenberg position is useful in dissipation of the bubbles. A recent study of the efficacy of this procedure was prompted by an observation that a severe case of cerebral air emboli in a diver was dramatically relieved by rocking the patient back and forth from a Trendelenberg position to the

horizontal [113]. The mechanism of this phenomenon was studied in cats given air emboli by overpressurization of the lungs [5].

"After an initial observation period of approximately five minutes, during which the stability of the air-blood interface in cortical vessels was observed, the animal was tipped head down with the long axis of the body at 30-60 degree angle. Brain swelling, which had been present following the development of air emboli, became somewhat more pronounced. Marked engorgement of cortical pial veins and venules occurred and blood began to displace the air in the small arteries under observation. Flow through adjacent arteries not containing air emboli underwent no visible change. No air was observed in the veins draining the gyri being fed by air-containing arteries. After blood flow had been established, marked dilation of the small arteries was observed even when the animal was returned to a horizontal position. The following tenative mechanism of the above phenomena is proposed. The point in the branching of the vascular tree at which air lodges is probably determined by the surface tension properties of the air-blood interface within the lumen of the vessel. When arterial pressure is no longer able to move the interface along the vessel walls of diminishing caliber into the capillary bed, stasis results with infarction and the production of neurologic symptoms. Increase in venous pressure through use of the hydrostatic pressure of the venous blood column dilates the venous side of the drainage system and the capillary bed. This allows a more favorable ratio between the circumference of the air-blood interface which tends to be secured to the vessel wall as a result of surface tension differences, and the cross sectional area of the vessel available for arterial pressure which tends to move the interface along the vessel. Once air gets into the venous side of the cerebral circulation it has not been observed to reappear. The volume trapped in these critical vessels is probably quite small."

If this interpretation is correct, it appears that the Trendelenberg position may indeed be of value in restoring circulation to the brain as well as reducing further embolization from the heart. In the presence

of shock, this position may also improve cardiac output, but in some individuals compression of large veins in the chest by diaphragm and abdominal contents may decrease the output. The increased cardiac output may aid the dilation of the cerebral capillary bed in dissipating the emboli through augmentation of the "vis a tergo." In the presence of shock or pulmonary edema from lung damage, the Trendelenberg position should be used with caution [34, 218, 178].

Treatment of shock may be a problem once cardiac function is restored. The original reflex hypotension and bradycardia after air blast is relatively short-lived. Loss of plasma by capillary leakage distal to the many small arterial emboli is a major factor in perpetuating the original hypotensive response [46, 134]. Blood loss from the lungs will intensify the shock state. Contusion of the heart is not as significant a problem as seen in air blast. However, cardiac infarction by air emboli contributes to the shock picture.

It has been suggested that a vasopressor, such as metaraminol should be used primarily for the treatment of non-hemorrhagic "shock" in explosive decompression [30, 63]. Intravenous fluids should be used with extreme caution, especially during the first few hours after a blast exposure. A fluid overload could overstrain a damaged heart and aggravate pulmonary hemorrhage and edema. Therefore blood replacement agents, such as blood, plasma, and dextran, used for the treatment of hypovolemic "shock" should be administered only if absolutely essential for the treatment of plasma loss and hemorrhage [46]. The routine use of a vasoconstrictor in the absence of non-hemorrhagic "shock, just to stop pulmonary bleeding, has been questioned [30]. It is known that the bleeding following blast occurs from both the thin-walled, bronchial and pulmonary veins and capillaries [192]. Arteriolar and venous constriction should reduce this bleeding. Since vasopressors, such as levaterenol and metaraminol, constrict bronchial vessels to the same degree as peripheral vessels, and pulmonary vessels to a lesser degree, such drugs might be useful for decreasing bronchial and possibly pulmonary hemorrhage [7, 67]. However, this indication for a vasopressor might be outweighed by the possibility that

the damaged pulmonary vessels, which have been constricted by hypoxia and by local reflex action, might be opened by the increased pulmonary blood pressure and flow caused by the drugs[6, 7, 30, 190]. The beneficial effects of the vasopressor drugs primarily for the treatment of pulmonary complications still remain to be determined. The therapeutic management of refractory shock has received recent review [97,117, 135]. A central venous pressure catheter (intravenous, subclavian) is of value in evaluating therapy in the face of simultaneous hemorrhage, plasma leakage, and cardiac failure. Analysis of techniques are beyond the scope of the present discussion.

Cardiac decompensation secondary to air embolization should be treated by intravenous digitalization [116, 192]. Treatment of specific arrythmias may also require digitalis or other agents. Again, one should be aware that some of these agents may dilate the pulmonary vessels and increase the bleeding in the lung during the first several hours after the explosive decompression [6, 7, 30]. Evaluation of the anti-arrythmic drugs in the presence of lung hemorrhage remains to be done. In general, the β-adrenegeric drugs such as isoproterenol, used in relieving heart blocks of various types, tend to dilate the pulmonary vasculature. Atropine may be substituted for these agents in treating SA and AV blocks [142]. The vasodilator effects of atropine are much less striking than those of the β-adrenergic agents [7].

Hypothermia has been tried in combating the effects of cerebral edema found after cerebro-vascular embolization following diving accidents (See Figure 7) [16, 65]. This technique has had some success in a few serious cases with CNS symptoms. More definitive data are required before this approach is suggested for the brain syndrome following explosive decompression. Recent studies have suggested that cerebral edema probably plays less of a role after cerebral embolization than has been thought in the past [52]. The role of the abnormal amounts of serotonin found experimentally seems to indicate that the vasoconstrictor factor may overshadow the edema.

The landing of air emboli in the cerebral circulation is associated with a vasoconstrictor response. Preliminary studies on dogs suggest

that this vasoconstriction may be reversed by intracarotid injection of 2 cc of 1% procaine hydrochloride [127]. Procaine hydrochloride has been used in humans to relieve cerebral vasospasm and to dilate the cerebral vasculature prior to arteriography [26, 31, 37]. In the presence of severe pulmonary edema where a deep Trendelenberg position may be contraindicated, intracarotid procaine hydrochloride (200 mg) may be of value in relieving focal neurological signs. The experimental nature of this approach should be realized.

Five percent CO_2 added to a breathing mixture dilates cerebral vessels and, therefore, has been suggested as therapy in occlusive cerebrovascular disease, especially where smaller arteries are involved [140]. However, in the treatment of neurocirculatory collapse associated with decompression sickness, 3% carbon dioxide added to 97% oxygen in the breathing mixture did not seem to be of value [92]. Further studies are required for the specific case of air emboli where the vasoconstrictor reflex may override the local dilatory effect of CO_2.

Infusion under pressure of low molecular weight dextran and heparin therapy have been used experimentally and clinically in the treatment of cerebral air emboli [1, 15, 195]. The mechanism of action is not clear [1]. Preliminary studies in dogs suggest that there may be sludging of blood associated with the gas emboli. Sludging may be involved in the slowing and spasmodic restoration of the circulation in the area of the gas bubble. The dextran may possibly relieve the sludging factor [195]. Peculiarities in the response of the cerebro-arterial system of the dog to air embolization can play a role influencing such observations [55]. These species-specific factors have also interfered with a proper evaluation of the relative merits of LM dextran, heparin, and hyperbaric oxygen in this condition [195]. Before dextran or heparin are to be recommended for use in traumatic air embolization, more definitive studies are required on the mechanism of these agents.

In the treatment of severe convulsions from air emboli, phenobarbital should probably be avoided because its dulling of the sensorium and prolonged action makes general evaluation of the post-traumatic patient most difficult. Intravenous or intramuscular valium, 10 mg repeated about every 4 hours, or Na Amytal, 4 grains repeated every 4 hours can be used. In shock, the intravenous route is preferred.

When facilities are available, compression to 6 atmospheres absolute appears to be the optimum method for treating aeroemboli [2, 18, 23, 141, 172, 185, 196]. Recent studies of air emboli in the cerebral arteries of dogs responding to increased pressure shed light on the physiological processes involved [185]. The results of this study are recorded.

"Typically, the bubbles conformed to the size and shape of the blood vessels. The majority of the air passed through the larger arteries very rapidly, but on reaching the branches of these arteries came to rest in a way that effectively blocked arterial circulation. In some instances, entire branches were filled with air; in others, the air bubbles were lined up in a row with small amounts of blood separating them with thin biconcave menisci. The very small arteriolar vessels were completely filled with air and appeared as a very thin, silvery network on the cortical surface. The largest arteries observed to be blocked by the bubbles were 2 mm in diameter. However, most of the vessels observed which were filled with air and showed evidence of circulatory obstruction were smaller than 2 mm, and were in the range of 30-60 microns. At the blood/air interfaces, the circulation was at a standstill, and the pulsations of the heart could be seen. In other small arteries there was a slow pulsating progression of the bubbles in response to the systolic pressure peaks. The surrounding brain tissue exposed by the cranial windows came under observation and typically showed a pallor which in the untreated cases gave way to a reactive hyperemia. Minor flare hemorrhages and petechial air embolism were also noted. Moderate edema was evident in some cases after an hour or more."

In the six dogs embolized and then recompressed to 165 feet, the following results were observed: "Two showed that all the bubbles observed grossly had vanished by 100 feet (four atmospheres); three showed the same results by 80 feet; and one dog, by 60 feet. In every instance there was evidence of a change in bubble size and partial restoration of the circulation just beyond 33 feet. In none of the experiments were intravascular bubbles seen to persist after pressure equivalent to four atmospheres was reached. Equally important, in no instance was there a reappearance of bubbles during or after decompression, using a Standard Navy Decompression Table for 170 feet (10 min.) at a standard ascent rate of 25 feet per minute. There was no attempt in this series to treat cerebral air embolism with pressures less than six atmospheres absolute, even though there is some indication that this maximum need not be applied.

Recent studies have been performed at the Experimental Diving Unit in Washington, D. C., on the use of recompression with 100% oxygen in treating traumatic air emboli as well as decompression sickness (23). The rationale for this approach is summarized: "The objective of the 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 invidious 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. 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 in an injured patient. The geometry of the situation, however,

is such that the radius of the bubble decreases as the cube root of the applied pressure. The diminution of the radius, therefore, decreases at even 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 efficacy of this approach has been documented in eighteen cases of traumatic air embolism [23]. Details of the therapeutic approach are presented in Appendix IV, a copy of BuMed Instruction 6420.2 [181]. Tables 5A and 6A are of specific interest in the present context. Description of Tables 5 and 6 are included for background and for interpretation of Appendix V.

In explosive decompression of space suits, as in the case of too rapid ascent during buoyant ascent training, inert gas uptake from pressure exposure is minimal in comparison to diving operations. In both, there is little possibility of confusion with decompression sickness, and the problems with recompression to 165 feet as a repetitive dive can be minimized. To reap the benefits of full bubble compression under these circumstances the Commanding Officer, Submarine Medical Center, has

developed treatment tables 5A and 5B which begin with immediate recompression to 165 feet on air. The patient is maintained at this depth for a time and is then decompressed to 60 feet and given hyperbaric oxygenation on a schedule similar to that of Table 5 (Decompression from 60 minutes at 165 feet calls for an ascent at 60 feet per minute to a first stop at 50 feet. Ascents on this treatment have generally been at 25 feet per minute to 60 feet.) Both Table 5 and 5A and Table 6 and 6A provide adequate decompression for the air breathing tender who remains in the chamber throughout the entire treatment, if it is his first dive of the day. If the treatment involves a repetitive dive for the tender or if the schedule of Table 6 is lengthened, then the tender must breath 100 % oxygen during the final 30 minute ascent from 30 feet to the surface. In some situations it is desirable to bring an attendant, such as the Medical Officer, back to the surface before the end of the treatment. If this ascent is made before the end of the stay at 60 feet, the decompression obligation can usually be determined simply by use of the standard air decompression table or the repetitive air decompression tables. If this can not be easily done, it is better to keep the attendant inside the chamber throughout the full treatment schedule rather than risk the problem of treating decompression sickness in a tender while the other patient already occupies the decompression chamber.

Appendix V covers results of recent recompression treatment for traumatic air emboli. Table V-1 covers treatment of divers where decompression sickness may also have been a factor. Twelve buoyant ascent cases have been tabulated in Table V-2. Eleven were taken to 165 feet and kept there for a period ranging from 6 to 30 minutes. Only six had complete relief before leaving 165 feet, and two of these had a recurrence at 30 feet. One patient was taken back to 165 feet and successfully retreated on U.S. Navy Recompression Table 4. The second was recompressed to 60 feet and successfully retreated on the schedule of Table 6. Of the five patients who were taken from 165 feet before they were completely relieved, all also recovered completely. The patient of case 423

was normal shortly after reaching 60 feet but his condition deteriorated after 75 minutes at that depth. He was accordingly kept at 60 feet for three hours breathing alternately oxygen and air. His treatment schedule thereafter was in accordance with Table 6 and he was noted to be completely recovered after treatment. The patient of case 501 was not taken to 165 feet. He recovered completely from a right arm paralysis and paralysis of right conjugate gaze immediately on reaching 60 feet and was therefore treated on Table 5. Treatment was successful.

In this small series the initial success rate with treatment can be stated as only 75%. However, all patients recovered completely before the end of treatment, including treatment of recurrences. In case 060, one of the first in which this method was applied, the decision was made to treat the recurrence on U. S. Navy Recompression Table 4 rather than Table 6 when a convulsion occurred. The patient had been brought up to 30 feet despite his complaints of increasing nervousness, headache, nausea, and numbness. It is not clear whether his convulsion resulted from bubble recurrence or oxygen toxicity. He remained in a stuporous, possibly post-ictal, condition for 155 minutes after return to 165 feet.

It is considered that this method, the optimum approach to treatment of traumatic air embolism occurring under the conditions of ascent training at the submarine escape training facilities, would also hold for air embolism secondary to explosive decompression of space suits. The recompression chamber should be at a location which is accessible within several minutes from the vacuum test facilities. Equipment for preliminary supportive care of the patient should be immediately available at the vacuum test chamber, probably within the lock chambers.

It is clear that the effects of aeroemboli may be alleviated by medical and surgical measures in the absence of a 6 atm. a. recompression chamber. Would a clinical hyperbaric chamber capable of 2-3 atmospheres overpressure available at a nearby medical center be adequate? Cerebral hypoxia and the shock syndrome following traumatic injury to the lungs and aeroemboli may be alleviated at 3 atm absolute in

such chambers [28, 32, 66, 114, 138, 146, 189, 195]. The response of the heart and the coronary circulation to these measures may be also favorable [137, 139]. Effects of anesthesia used in experimental preparations may have confused the evaluation of hyperbaric oxygen in cerebrovascular occlusion [96]. Addition of 2% CO_2 appears to relieve the vasospastic response to hyperbaric oxygenation [75, 176]. The efficacy of additional CO_2 in treatment of such occlusions in humans is as yet not clear. This is true in conjunction with 2-3 atm. a. chambers or during 6 atm. a. therapy in Appendix IV.

In view of the recent experience of the U. S. Navy outlined above and in Appendix V, it would appear that pressures in the range of 4-6 atm. a. would be of greater benefit than hyperbaric oxygen in the 2-3 atm. a. range in relieving the effects of massive air embolization. In milder cases, the typical clinical hyperbaric chamber would probably go far in aiding the patient, but these lower pressures are not ideal. Availability of such a clinical chamber at a local hospital is not always assured. The delay in transporting a patient to such a facility must also be considered. According to Mr. Edward Michel, the recompression chambers recently installed at the NASA MSC do have a 6 atm. a. capability.

In closing this discussion, it should be emphasized 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 [55]. 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. Table 8 represents the results. 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

Table 8. Relative Effects of Oxygen, Carbon Dioxide, Nitrogen, and Helium Emboli

Gas	No. Dogs	Dose (Ml.)	Morbidity	Mortality
O_2	3	0.5	2	0
O_2	2	1	2	0
O_2	2	1.5	2	0
CO_2	2	1	0	0
CO_2	2	1.5	0	0
CO_2	3	2	1	1
N_2	2	0.2	1	0
N_2	3	0.5	2	1
He	3	0.5	2	1
He	3	1	1	1

(After De la Torre [55])

comparative resolution rates and, ultimately, by the permeation coefficients of different gas bubbles [121, 165]. 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 intrarterial 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. As indicated in Part I of this study, the hazard of lung disruption is slightly less with helium than with the other gases under the same pressure variables, but the difference is probably not operationally significant.

4. Ebullism

The rate of recompression following exposure to vacuum should be as rapid as possible. The vacuum chambers at NASA MSC are designed to introduce nitrogen to the chamber to 1 psia, then a mixture of nitrogen and oxygen to a total pressure of 4.5 psia, followed by ambient air to 6 psia [36]. The sequence occurs in 30 seconds. A hold is possible at this point. Ambient air may be introduced to continue

repressurization to 14.7 psia within an additional 60 seconds. This schedule appeared to work satisfactorily during the accidental decompression recorded in Figure 6 where the pressure reached 7 psia within 25 seconds from onset of decompression [85]. 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 [144, 164]. 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 [108, 110]. 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 [11, 88, 17, 171].

From the review of the pathological physiology of ebullism in Part II of this study, it is apparent that in the treatment of this syndrome, one must consider damage to the lungs from exposure to cold [157], 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. The Trendelenberg position may decrease the embolization of the lungs. Aspiration of gas from the right ventricle in case of cardiac arrest may aid in restoration of the circulation and avoid further damage to the lung by gas emboli (vide supra). 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 uncomplication by embolization [163].

Exposure to a hypoxic environment 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 by Busby [30]. 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 recompression 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 [30, 87, 112]. 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 [30]. 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 [84, 197]. The value of steroid-antihistamine combinations for post-hypoxic cerebral edema is yet to be definitively determined [30].

The bizarre electrocardiographic patterns seen in dogs exposed to vacuum range from extrasystoles to idioventricular rhythms and ventricular fibrillation [29, 47, 157]. 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 [79] (see Rx of aeroemboli). 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. 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. (See Letters to the Editor, New England Journal of Medicine, Vol. 278, March 14, 1968).

V. Therapeutic Equipment and Supplies

In view of the several syndromes to be treated, the following is a summary of the equipment and supplies which should be available in the vicinity of the vacuum test chambers. These cover general cardiopulmonary resuscitation [4] and specific therapeutic requirements for explosive decompression. They do not specifically cover other chamber emergencies, but will no doubt go far in doing so. The redundancy of each item will be ultimately determined by the number of simultaneous casualties possible during each operation. The numbers chosen here are minimal for portable emergency carts.

Ventilation Kit

Self-inflating bag-valve-mask unit, with an oxygen reservoir tube at intake, and a valve which also permits spontaneous breathing from the bag.

Mechanical ventilator and chest compressor

Regular oro-pharyngeal tubes (airways) - large adult, medium adult, small adult

Naso-pharyngeal tubes, adult size

S-tubes for mouth-to-mouth ventilation

Tonsil suction tip

Straight suction connectors

A suction machine - portable; fine pressure control; or a suction line

Cricothyrotome with 15mm adaptor (Foregger) or 14 gauge short beveled needle for cricothyroid puncture

Suction catheter (French 14, sterile)

Intubation Kit

Laryngoscope set:

1 battery handle

1 straight adult blade

1 curved adult blade

Bronchoscopy set

Set of orotracheal tubes with straight standard 15mm adaptors (cuffed for adult sizes over 30 French) in approximately the following French sizes: 30, 34, 38, 40.

Cuffed nasotracheal tubes - 28, 30, and 32 French

Stylette (copper wire obturator) for orotracheal tubes

Cuff inflation syringe, adaptor and clamp

Anesthetic,water-soluble,lubricating jelly

Bite block and 1" tape

Right angle adaptor(15mm)for tracheal tubes

Intubation Kit (continued)

McGill forceps for insertion of gastric and nasotracheal tubes

Gastric tubes - French 14 and 16 - 1 each; 2 bulb syringes

Tracheal suction catheters, preferably curved tip - 1 each, French 10, 14 and 16

Y connectors, medium, for suction catheters - 2

Straight connectors - 2

Irrigating saline - 1 liter

Succinyl choline - 20 mg/ml, 10 cc vial (2 vials)

Drug aerosol nebulizer (e. g., Bird breathe-through type) with mouthpiece and 15 mm adaptor (for bronchospasm)

Topical anesthetic spray cannula and atomizer with 4% lidocaine

Drug Kit

Sodium bicarbonate 5%-500 cc-2 bottles (stored with Infusion Kit), or in 50 cc ampule (44. 6 mEq),(10 ampules)

Epinephrine inj. (Adrenalin) 1 mg ampule (10 ampules)

Nor-epinephrine inj. (Levophed) 0. 2%-4 cc ampule (6 ampules)

Succinyl choline-20 mg/ml, 10 cc vial (2 vials) and 1 gm powder (1 vial) for infusion

Metaraminol inj. (Aramine, Pressonex, Pressoral) 1%-10 cc vial (2 vials)

Calcium gluconate 10%-10 cc ampule (2 ampules)

Lidocaine (Xylocaine) (without epinephrine) 1%-20 ml ampule (3 ampules) - for ventricular arrhythmias

Atropine sulfate (0. 5 or 1. 0 mg/ml) 2 cc ampule (4 ampules)

Deslanoside inj. (Cedilanid-D) 0. 4 mg ampule (4 ampules)

Digoxin inj. (Lanoxin) 0. 25 mg/ml-2 cc ampule (4 ampules)

Isoproterenol inj. (Isuprel) 1 mg ampule (6 ampules) -(should not be used for heart block in presence of pulmonary hemorrhage

Quinidine gluconate-80 mg/cc-10 cc vial (2 vials)

Sodium ethacrynate (Lyovac sodium edecrine) - 50 mg, (4 vials) - for intravenous injection in pulmonary edema

Drug Kit (continued)

Procainamide (Pronestyl) 100 mg/cc-10 cc vial (2 vials)

Phenylephrine (Neo-synephrine) 1%-5 cc ampule (1 vial)

Dexamethasone inj. (Decadron, Hexadrol) 4 mg/cc ampules (4 ampules)

Chlorpheniramine maleate (Chlor-trimeton) 100 mg/cc-2 cc vial (2 vials)

Isoproterenol (Isuprel) 1:200-5 cc vial (2 vials) for inhalation therapy (not used in presence of pulmonary hemorrhage)

Aminophylline-250 mg per ampule (4 ampules)

Dihydrocodeinone antitussive tablets and injectable

Diazepam (Valium), injectable (2 cc ampule with 10 mg) for convulsions

Na amytal, injectable (ampule of $7\frac{1}{2}$ gr), for convulsions

Mannitol (20% solution) IV use for post-hypoxic cerebral edema

Antibiotics

Injection/Infusion Kit

Alcohol sponges

Bandaids

Antiobiotic ointment - neosporin

Sterile gauze sponges 4" x 4"

1-inch tape

Needles of various sizes, 25-to 15 gauge

Intracardiac needles - 2 of each: #22 gauge, $3\frac{1}{2}$" and #15 gauge, $3\frac{1}{2}$" for aspiration of gas.

Syringes 4-10 cc; 3-50 cc

Intracaths:

14-gauge - 4

16-gauge - 4

14-gauge 36" radiopaque (central venous) - 2

Injection/Infusion Kit (continued)

Angiocaths (or similar catheter needles):
- 14-gauge - 4
- 16-gauge - 4

I. V. administration sets and poles - 5

Arterial puncture or arterial catheterization tray

Microdrip sets - 2

Blood administration sets - 2 - have blood type on record for all subjects

I. V. extension sets - 2

3-way stopcocks - with 20" extension 2

Blood pumps for bags and bottles

Bedboards or trays

Fluids:
- 5% dextran/saline 500 ml (low molecular weight)
- 5% dextrose/saline 500 ml
- 5% dextrose/water 500 ml
- 5% isotonic saline 500 ml
- 5% sodium bicarbonate/water 500 ml - 2 (see drug kit)

Special Equipment

Cardioscope with cardioscope leads and sterile metal needle electrodes mounted (spare metal needles), plate electrodes and electrode paste

Defibrillator, with external electrodes mounted and sterile internal electrodes available - DC system

Pacemaker, battery-operated, internal

Percutaneous and transvenous electrodes

Portable oxygen with flow meter and delivery tube

Subclavian, percutaneous, central venous pressure catheter

Pleural drainage tray - with four sets covering bilateral pneumothorax for 2 patients, each containing:

Special Equipment (continued)

Trocar

Cannula

Intercostal catheter (MacQuigg type) or 16 to 28F gauge soft catheter with extra side holes

Prep kit - Betadine, isopropyl alcohol, 1% lidocaine, sterile gloves (3 pairs - $7\frac{1}{2}$, 3 pairs - 8), sterile swabs - 2 packages

Thoracotomy tray (for open chest cardiac massage and pretracheal incisions)

Three-bottle suction set for pleural drainage

Emergency operating table with control for up to 30^{o} Trendelenberg

Portable chest x-ray unit

The requirement for a therapeutic chamber stocked with equipment and supplies for continuing the supportive therapy outlined above appears justified. Modification of existing therapeutic chambers will be worth the expense, especially if the chance of critical suit disruption is shown to be large enough. This of course will depend on the review of the integrity of the seals and other parts of the suit system. What quantitative degree of suit reliability would be required to preclude the necessity of a sophisticated therapeutic chamber capable of 6 atmospheres pressure cannot be stated without a more extensive re-evaluation of the suit, equipment, chambers, etc. A thorough review of suit vulnerability by the appropriate groups at the NASA MSC, Houston is suggested. In evaluation of costs it should be realized that the same 6 atm. a. compression chamber can also be used in the treatment of decompression sickness by the current oxygen-pressure method [78, 129, 130].

Availability of the compression treatment does not eliminate the need for the supportive measures. Body position, tracheobronchial clearance, and treatment of pneumomediastinum and tension pneumo-

thorax should still proceed. If the patient appears to be failing in spite of the compression treatment, heroic measures must still be considered as appropriate.

SUMMARY

A review is presented of the biomechanical factors determining lung damage and its sequlae following explosive decompression of space suits in vacuum test chambers. Critical variables such as ratio of free volume of suit to area of orifice, pressure ratio, and pressure differential were outlined. A lack of data was found which precludes precise prediction of the threshold variables for hazardous decompression of space suits. More animal data are needed for decompressions from 3.7 - 7 psia down to vacuum in 0.1 to 1.0 second.

Analysis was made of the vulnerability of space suits to explosive decompression. Calculations were made of orifices presented by catastrophic disruption of typical soft and hard space suits at seal sites. The ratio of the residual free volume of the suit system to the annular orifice in question was used to calculate time characteristics of the man-in-suit system. Conservative extrapolation of data obtained at higher pressure differentials and lower pressure ratios than suit decompression suggests that:

a) 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. This is true even for open-glottis conditions. The hazard is intensified if the glottis is closed and breath is held.

b) Disruption of a glove finger in both suits and the 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.

c) 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. The case is much less clear than in conclusions a) and b). Disruption of the umbilical hoses at a distance from the entrance port would lower the probability of damage. The accident at MSC during Test #3 of PLSS 055 was analyzed as a case in point.

d) 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 laminated fabric of the soft suit and metal shell of the hard suit are also reportedly safe from acute disruption. No direct data from destructive testing of pressurized space suits were available for analysis. 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.

Use of gases other than 3.7 psia O_2 in the suits alters the hazard. The higher the percentage of helium in the mixture, the less hazard is presented. A helium-oxygen mixture at 7 psia would be, at best, 0.5 times as dangerous as an oxygen-nitrogen or the equally hazardous pure oxygen mixture at the same pressure. Since the maximum possible amplitude of transmural pressure is determined by the pressure difference, the higher the initial pressure in the suit, the more hazardous the exposure. A suit at 7 psia would present a maximum possible transmural pressure differential of twice that of a suit at 3.7 psia. The fraction of the total effective differential and time course of overpressure would depend on the V/A and pressure ratios. Further development of a

computerized mathematical model of chest dynamics currently being used in study of blast biology may well shed some light on the biomechanics of explosive decompression in the range of V/A ratios, pressure ratios, and pressure differentials considered in the present analysis.

The pathological physiology of explosive decompression was then reviewed. This included analysis of contusion of lung parenchyma; pleural and peribronchial hemorrhage; interstitial, peribronchial, and mediastinal emphysema, and pneumopericardium. Simple and tension pneumothorax were then presented along with a review of cardiorespiratory embarrassment brought about by all of these conditions. Aeroembolism arising from air injected into the pulmonary veins and passing to the coronary, cerebral, and the systemic arterial system was then reviewed. Analysis was made of the critical factors caused by exposure to vacuum conditions in the ebullism syndrome. Finally, a review of all of the reported cases of explosive decompression damage in humans was presented. Pre-existing lung pathology was noted as a major variable.

The next section reviewed the most recent ideas in selection and testing of subjects to reduce the hazard and in the treatment of the individual syndromes and conditions. Questions were raised about the dangers of oxygen therapy and certain drugs which can cause pulmonary vasodilation and prolong hemorrhage and aeroembolism. Emergency incisions of the pretracheal fascia to relieve mediastinal emphysema and thoracentesis for tension pneumo-and hemothorax were discussed. A weighted plan for appropriately handling situations of increasing severity was outlined.

Supportive treatment of aeroembolism was then covered. Such procedures as cardiopulmonary resuscitation, aspiration of bubbles from both ventricles, 30^{o} Trendelenberg position, and the heroic, last-ditch measures of thoracotomy and cardiac massage were presented. The use of recompression chambers up to 6 atm. a. appears to be the best therapeutic measure for gas emboli. The latest schedules developed by the

U. S. Navy for treatment of buoyant ascent injuries appear most reasonable for space suit decompression. The rationale and results of this approach were presented. Finally, the treatment of the ebullism syndrome was pointed out to be similar to that outlined for the other aspects of explosive decompression. Ventricular arrythmias should be anticipated.

A tabular summary is given of the equipment and supplies needed to optimally handle explosive decompression injuries at test chambers. The redundancy of each item depends on the numbers of subjects at simultaneous risk. Chambers of 6 atm. a. capacity should be outfitted for handling the supportive measures suggested for each of the syndromes. They should also cover support of other accidents which may occur.

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Appendix I

Calculation of Annular Orifices and Volumes.

I. Neck Seal

A. Diameters - 9" ID soft suit, 11.8" hard suit.

B. Area of seal = $\pi \times (4.5 \times 2.54)^2 = 411\ cm^2$ soft suit

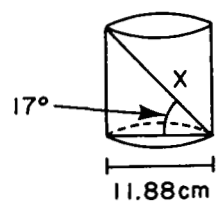
Area of seal = $\pi \times (5.9 \times 2.54)^2 = 706\ cm^2$ hard suit

C. Human neck X-area in soft suit

1) Calculated from Ref. 162, Standards #16.

a) Circumference of astronaut neck (mean) = 37.43".

b) Diameter = $\frac{3.743}{\pi} \approx 11.88$ cm .



c) $X = 11.88 \sec 17^\circ \approx 11.88 \times 1.046 = 12.43$.

d) Area of ellipse = $\pi ab \approx \frac{\pi \times 11.88 \times 12.43}{4} = 116\ cm^2$.

2) Measured on six subjects at Lovelace Foundation by wire imprint of neck (mean values).

a) $2a = 5.4" = 13.72$ cm

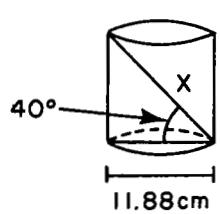
$2b = 4.0" = 10.18$ cm

b) Area of ellipse = $\pi ab = \frac{\pi \times 13.72 \times 10.18}{4} = 110\ cm^2$

D. Human neck area in hard suit

1) Calculated from Ref. 162, Standards #16.

a) Diameter ≈ 11.88 cm



b) $X = 11.88 \sec 40^{\circ} = 11.88 \times 1.31 = 15.50.$

c) Area of ellipse $= \pi ab = \frac{\pi \times 11.88 \times 15.50}{4} = 145 \text{ cm}^2$

E. Annular orifices.

1) Soft suit: $411 - 116 = 295 \text{ cm}^2$.

2) Hard suit: $706 - 145 = 561 \text{ cm}^2$.

II. Wrist Seal

A. Diameters - 4" ID soft suit, 3.87" ID hard suit.

B. Area of seal $= \pi \times (2 \times 2.54)^2 = 81.4 \text{ cm}^2$ soft suit

Area of seal $= \pi \times (1.93 \times 2.54)^2 = 76 \text{ cm}^2$ hard suit

C. Human wrist X-area

From Ref. 162, Standards #16 and measurement at Lovelace.

1) Breadth (2a) = 5.95 from Ref. 162, Standards #16

2) Thickness (2b) = 4.60 from measurement at Lovelace Foundation

3) X- Area $= \pi ab = \frac{\pi \times 5.95 \times 4.60}{4} = 21.5 \text{ cm}^2$

D. Annular orifices

1) Soft suit: $81.4 - 21.5 = 60 \text{ cm}^2$

2) Hard suit: $76.0 - 21.5 = 54 \text{ cm}^2$

III Thigh Seal - hard suit only

A. Diameter of seal = 7 7/8" ID

B. Area of seal $= \pi \times \left(\frac{63}{16} \times 2.54\right)^2 = \pi \times 100 = 31.4 \text{ cm}^2$

C. X - Area of human thigh (lower part)

1) Circumference - from Ref. 162, Standards #16 = 41.6 cm

2) $r = 41.6/2\pi = 6.6$ cm

3) $A = \pi r^2 = \pi \times (6.6)^2 = 137 \text{ cm}^2$

D. Annular orifice $= 314 - 137 = 177 \text{ cm}^2$

IV. Ankle Seal - hard suit only

A. Diameter of elliptical seal: (2a) = 5 9/16" ID, (2b) = 7 5/32" ID

1) a = 89/32 x 2.54 = 7.24 cm

2) b = 229/64 x 2.54 = 9.1 cm

B. Area of seal = $\pi ab = \pi \times 7.24 \times 9.10 = 207\ cm^2$

C. X-area of human ankle 6 1/2" above floor

1) Circumference measured at Lovelace Foundation = 8 5/8" average of 6.

2) Radius = $\frac{69}{8 \times 2\pi}$ = 1.38" or 1.38 x 2.54 = 3.5 cm

3) $A = \pi \times (3.5)^2 = 39\ cm^2$

D. Annular orifice = 207 - 39 = $168\ cm^2$

V. Waist Seal - hard suit

A. Diameter of waist seal = 16" ID

B. Area of waist seal = $3.14 \times (8 \times 2.54)^2 = 1300\ cm^2$

C. X-area of body 1" above umbilicus (assume waist)

1) From Ref. 162, Standards #16, the
Depth of waist = 21.1 cm
Breadth of waist = 29.6 cm

2) Area of waist = $\pi ab = \frac{\pi \times 21.1 \times 29.6}{4} = 490\ cm^2$

D. Annular orifice of waist seal = 1300 - 490 = $810\ cm^2$

VI. Fingers - both suits

A. Diameter of typical glove finger = 1" ID

B. X-area of glove finger = $\pi \times (2.54 \times 0.5)^2 = 5.07\ cm^2$

C. X-section of typical finger

1) Assume 1/16" clearance between glove and finger

2) Radius of finger = $\frac{8}{16} - \frac{1}{16} = \frac{7}{16}$"

3) X-area of finger = $\pi \times (\frac{7}{16} \times 2.54)^2 = 3.87\ cm^2$

D. Annular orifice at finger

1) 5.07 - 3.87 = $1.2\ cm^2$

VII. Chamber umbilical hose

A. Diameter = 1 1/4" ID

B. X area = $\pi \times (\frac{1.25}{2} \times 2.54)^2 = 2.8\ cm^2$

VIII. PLSS umbilical hose

A. Diameter = 3/4" ID

B. Area = $\pi \times (\frac{0.75}{2} \times 2.54)^2 = 7.9\ cm^2$

IX. Free volume of hard suit helmet

A. Diameter of helmet = 12" ID

B. Volume of helmet (hemisphere)

$= \frac{1}{2} \times \frac{1}{6} \times \pi D^3 = \frac{\pi}{12} \times (12 \times 2.54)^3 = 7400$ cc

C. Volume of human head (Ref. 162, Standards #16)

1) Depth of head = 19.96 cm

2) Breadth of head = 15.55 cm

3) Ave. diameter of head = 19.96 + 15.55/2 = 17.8 cm

4) Volume of head - assuming it to be spherical

$= \frac{1}{6} \pi D^3 = \frac{1}{6} \pi (17.8)^3 = \sim 2950\ cm^2$

D. Free volume in helmet of hard suit

= 7400 - 3000 = ~ 4400 cc.

X. Free volume of soft suit helmet

The free volume of the soft suit helmet varies from model to model as the helmets vary in size and shape. The general range of free gas volume is estimated by Mr. Michel of MSC as 2-3 liters.

Appendix II

Effect of Umbilical Hose on Mass Flow During PLSS 055 Test #3, NASA, MSC

Dr. K. J. Touryan, Supervisor, Reentry Studies of the Aerodynamics Laboratory of the Sandia Corporation, analyzed the effect of the 64" long, 1 1/4" ID hose on smooth orifice and sharp orifice flow during the MSC accident of PLSS 055 Test #3. Calculations were performed using the program of this laboratory for isothermal, time-dependent flow conditions to vacuum.

A free volume of 1 cu ft (28 liters) was assumed. This is somewhat smaller than the 25.5 liters of the suit without back pack. The volume is 0.9 that assumed in Table 7. The friction coefficient (s or f) was selected for a moderately smooth tube. The difference between a very rough tube and a very smooth tube is an increase in mass flow rate of 70%, i.e., if the nondimensional mass flow rate is 0.50 for a very rough tube, it is 0.85 for a very smooth tube. His calculations were based on a flow rate of 0.80.

Figure A-II-1 is a plot of the calculations for the four conditions and that of MSC curve taken from Figure 6. The lower abscissa of Figure 6 was used. Deviation of the lower part of the MSC curve from the predicted curve of smooth orifice flow with hose is difficult to interpret in view of the several assumptions made as to the suit volume, the exchangeability of suit gas, the orifice coefficient, the hose friction factor, and accuracy of the few points plotted in Figure 6. The correspondence of the first 0.3 seconds is quite close. The correspondence of the calculated t_{cr} for smooth orifice flow without hose of 0.15 seconds with that of 0.14 seconds in Table 7 using the Haber-Clamann approximation is also of interest.

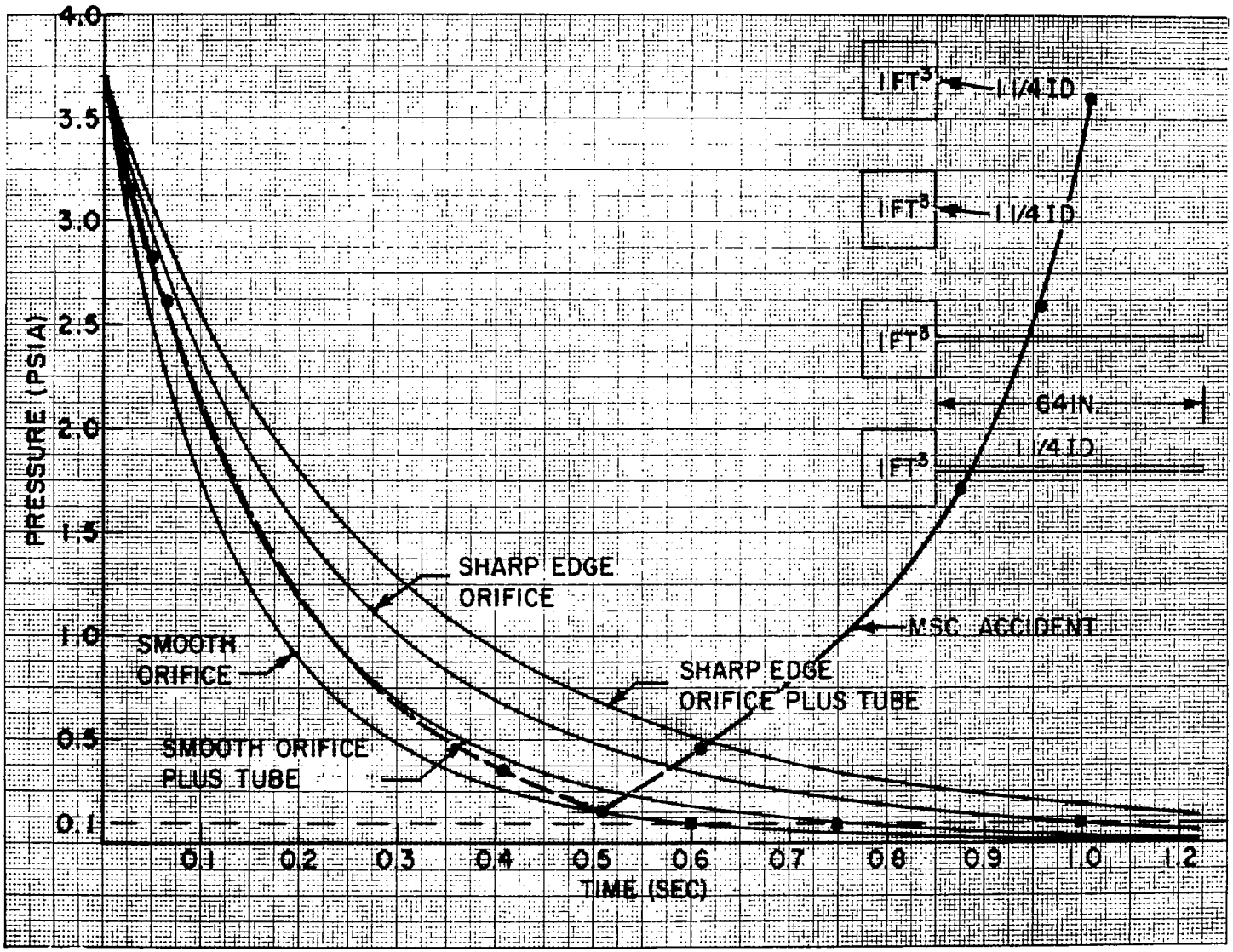


Figure A-II-1

(After Touryan [179])

Appendix III

Summary of Accident Report and Postmortem Examination - Brooke Army Hospital #42428 (A-241-53) 18 August 1953 (Reference 119)

The subject, a 24-year-old white male was decompressed in a routine chamber flight from 8,000 to 30,000 feet in ~1 second. Oxygen equipment was functioning normally. Just prior to the rapid decompression, the patient was seen to be in apparent inspiratory position with his chest fixed, and arms held rigid as if he were apprehensive and holding his breath. Immediately following rapid decompression, it was noted that he began to cough moderately. Very shortly after this he was seen to lose consciousness, and the picture described by the physicians on duty was that the patient remained deeply cyanotic, totally unresponsive and flaccid during the 2-3 minutes down to the ground level.

At this time, the patient was seen to be apneic, very deeply cyanotic, completely flaccid, and covered with a cold, clammy sweat. He was seen to have bilateral, widely, dilated pupils with the eyes fixed. After about two minutes, at ground level, there was urinary incontinence. It is also to be noted that he was given 100 percent oxygen for breathing from the time that treatment was instituted after leaving the chamber. Blood pressure and pulse determinations were attempted immediately but were unobtainable and no heart sounds were heard by stethoscopic examination. Manual artificial respiration was begun immediately. In a few moments a mechanical resuscitator was used to respire the patient. Mechanical resuscitation was continued for several more minutes while a full body respirator was prepared. Within five minutes of reaching ground level the subject was placed in the full body respirator. The patient at no time breathed spontaneously; however, at the moment ground level was reached he was seen to give a few gasps. These were very irregular and only two or three in number. The artificial respiration seemed to be perfectly adequate as air could be heard rushing in and out of the mouth; there was

no difficulty in keeping the airway clear. In spite of this respiration, mechanically induced, the color of the face and neck and the chest failed to improve. Heart sounds were not heard, although detection was attempted by several physicians who were on hand. After an interval of approximately ten to fifteen minutes, when heart sounds were still not audible, 1 cc of 1:1000 adrenalin was given intracardiacly. In spite of continued, deep, mechanical resuscitation the patient's color did not improve but developed a waxy pallor.

Postmortem examination showed cyanosis and frothy blood tinged fluid exuding from nostrils upon abdominal pressure. Distinct hyperson-ance to percussion was noted over the left anterior chest.

Prior to incision, a PA chest x-ray was taken with the patient in the upright position. This film revealed a 50% pneumothorax on the left side. There was no distinct radiologic evidence of pneumothorax on the right side. No shifting of mediastinal structures or emphysema within the neck structures were evident. Intrapleural pressures were taken using the pneumothorax-induction apparatus with the patient in the supine position. These were recorded as 0-1 mm. of water positive pressure on the left and 3 mm. of water negative pressure on the right.

In the process of reflecting the tissues overlying the thoracic cage, numerous minute, discrete gas bubbles, each less than 1 mm. in diameter, were noted escaping from transected blood vessels, especially small veins. Each axillary space was filled with water and the hemithoraces were entered under water by stab incisions. Large, coarse gas bubbles were detected escaping from the left hemithorax. There were not detected escaping from the right hemithorax. Booming resonance was evident with the cutting of individual ribs of the anterior thoracic plate. There was no evidence of rib fractures. Upon removal of the anterior thoracic plate, bilateral pneumothorax was found, more on the left, and no significant pleural fluid or adhesions were encountered. The lungs appeared in-completely collapsed. Collapse was more evident in the left lung. Each lung was distinctly subcrepitant to palpation. The pericardial sac did not appear enlarged. No air was detected within mediastinal tissues. The

entire thoracic cavity was then filled with water. Needle aspiration of each of the chambers of the heart produced finely bubbled blood. Innumerable minute, discrete gas bubbles were noted upon entering the root of the aorta and pulmonary conus. As in the chest, minute gas bubbles escaped from transected vessels throughout the abdomen.

Moderate amounts of frothy and finely-bubbled, blood-tinged fluid were removed from these passages. A moderate amount of mucoid material was additionally recovered from the smaller bronchi of each lung. Distinct bronchial occlusion by mucus plugs could not be grossly established. No significant intrinsic or extrinsic lesions of these passages or of the larynx were found.

Diffuse subpleural emphysema was noted on all pleural surfaces of both lungs. The antero-lateral aspect of the lower portion of the left upper lobe presented a small area of pleural disruption surrounded by an area of pleural and subpleural hemorrhage measuring 5x3 mm. An additional 2x2 mm. area of pleural hemorrhage without distinct disruption were noted in the mid-portion of the anterolateral aspect of the left lower lobe. Several minute fresh subpleural hemorrhages were additionally noted over the hilar aspects of the left lung. These measured 2 to 4 mm. in diameter. Examination of the right lung revealed no distinct pleural tears, however, its hilar region presented several minute subpleural hemorrhages similar to those seen on the hilar aspects of the left lung. With the lungs and trachea intact, the organs were insufflated with air under water. The entire left lung could be expanded by this process. Only the lower lobe of the right lung could be expanded to approximately normal dimensions. Two areas of pleural disruption were noted on the anterior and diaphragmatic aspects of the right lower lobe. The lesion on the anterior aspect appeared to be secondary to postmortem needle puncture. The other lesion was surrounded by a small area of hemorrhage. Gas bubbles escaped from each upon manual pressure. The two sites of pleural disruption in the left lung were found again and air escaped under water from each of these sites upon pressure. Multiple sections through each

lung revealed tiny mucus plugs within small bronchi and finely bubbled blood exuding from transected vessels. Save for the aforementioned pleural hemorrhages and subpleural emphysema, no distinct lesions of either lung were recognized.

The exposed heart showed moderately severe enlargement and engorgement of the right ventricle and right atrium. No distinct epicardial lesions were found. Fetal passages were closed. Three small areas of fresh subendocardial hemorrhage were detected near the tricuspid valvular ring and the adjacent posterior wall of the right atrium. The auricular appendages were not remarkable. No additional distinct endocardial or myocardial lesions were noted upon sectioning. The coronary arteries were thin-walled and patent throughout.

The brain appeared turgid with distinct flatting of gyral convolutions, and narrowing of sulci. A slight cerebromedullary pressure cone was seen. Cerebral edema and meningral congestion were noted.

All other organs were unremarkable.

Microscopic examination of the heart revealed fresh epicardial and sub-endo cardial hemorrhage and vascular congestion. Section of the lung taken at or adjacent to an area of pleural hemorrhage showed distinct subpleural interstitial emphysema, disruption of subpleural parenchyma, numerous fresh intra-alveolar, intra-bronchiolar, intra-bronchial and perivascular hemorrhages and focal atelectasis. Elsewhere, sections showed a mild acute and chronic bronchitis and bronchiolitis. Moderate pulmonary edema and congestion were present throughout. A section of right lower lobe showed moderate atelectasis and postmortem changes. An additional section taken from the lower lobe of the right lung showed multiple fresh intra-alveolar and intra-bronchial hemorrhages and a moderate degree of parenchymal congestion.

Liver showed mild centrolobular congestion associated with mild parenchymal degenerative changes within these zones. Other abdominal organs showed congestion. Sections of stomach, small and large intestines showed a moderate degree of congestion and, in addition, a

moderate degree or lymph follicular hyperplasia within the ileum, cecum and ascending colon. Representative mesenteric and para-aortic lymph nodes revealed a moderate, non-specific, chronic lymphadenitis.

Sixteen representative brain sections uniformly showed evidence of moderate cerebral edema, acute disintegrative changes of ganglion cells with occasional satellitosis and neuronophagia, scattered fresh perivascular hemorrhages, meningeal and parenchymal congestion, occasional areas of perivascular cuffing by round cells with and without associated hemosiderin deposits and mild calcification with medial degeneration of lenticulostriate arterioles.

The conclusion of the report was as follows: "The major pathologic changes as outlined above are consistent with asphyxia. It is felt that the underlying cause of death in this case may be attributed to acute cardiorespiratory failure, secondary to bilateral pneumothorax. The finding of myriads of minute, discrete gas bubbles within intact vessels throughout the body strongly indicates an associated gas embolization. It must be stated, however, that tissue degeneration ascribable to embolism was not demonstrable either grossly or microscopically. Further, it is felt that the nature of these bubbles and whether or not true dynamic embolism occurred cannot be stated with any degree of assurance. That these gas bubbles entered the blood stream by virtue of pulmonary parenchymal disruption appears very likely. Accordingly, these bubbles are presumed to be undissolved air.

The histologic cerebral changes, in the main, are felt to be consistent with hypoxia. However, the findings of occasional perivascular round cell cuffing associated with hemosiderin depositions strongly suggest pre-existing cerebral pathology of mild degree. Further, the combined findings of mesenteric lymphadenitis, lymph follicular hyperplasia within the bowel, mild acute and chronic bronchitis and focal collections of polymorphonuclear leukocytes within the liver with round cell infiltrations into portal areas lend additional evidence to a pre-existing systemic illness, possibly of viral etiology. It must be emphasized, however, that these latter changes are of minor degree and are felt to contribute very little to the patient's ultimate demise."

It is still not clear whether or not pneumothorax would have occurred even if the subject had not held his breath, as was alleged in the accident report. The blocked bronchi may have, by themselves, created a hazardous condition for explosive decompression. The pleural shock syndrome may also have played a role in his rapid demise [53].

Appendix IV

DEPARTMENT OF THE NAVY
Bureau of Medicine and Surgery
Washington, D.C. 20390

BUMEDINST 6420.2
BUMED-74
22 August 1967

BUMED INSTRUCTION 6420.2

From: Chief, Bureau of Medicine and Surgery
To: All Ships and Stations

Subj: Oxygen breathing treatment for decompression sickness and air embolism

Ref: (a) U.S. Navy Diving Manual (NAVSHIPS 250-538)
(b) MANMED Article 14-17(3)

Encl: (1) Minimal Recompression, Oxygen Breathing Method for Treatment of Decompression Sickness and Air Embolism (Tables 5 and 6)
(2) Oxygen Breathing Method for Treatment of Air Embolism Incurred During Submarine Escape Training (Tables 5A and 6A)

1. Purpose. This Instruction promulgates the regulations for use of the oxygen breathing methods (tables 5, 5A, 6 and 6A) in the treatment of decompression sickness and air embolism.

2. Background. The U.S. Navy treatment tables of reference (a) have been utilized as the treatment of choice for decompression sickness and air embolism for the past 20 years with a significant degree of success. However, the recent emergence of diving as a popular pastime has increased the incidence and severity of diving accidents and has led to an accelerated effort to establish more effective, less time consuming methods of treatment. A new regimen of treatment of decompression sickness and air embolism utilizing minimal recompression while breathing oxygen (tables 5 and 6) has now been shown to be both safe and effective, especially for those severe injuries in which treatment has been delayed or which have responded poorly in the past to the standard treatment tables (tables 1 - 21) of reference (a). It has also been demonstrated that air embolism incurred during submarine escape training can be most effectively treated with tables 5A and 6A.

3. Discussion

a. 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.

b. The pathophysiology of air embolism incurred during submarine escape training, however, is such that maximum benefit is obtained chiefly through immediate recompression to 165 feet but does not necessitate prolonged ensuing decompression. Rapid decompression to 60 feet after symptomatic improvement at 165 feet and the addition of oxygen breathing at 60 feet minimizes the possibility of decompression sickness onset and is immediately supportive to the cerebral tissues assaulted by the embolism.

c. The symptomatic manifestations of decompression sickness and air embolism can be further aggravated by development of secondary edema and cardiovascular impairment or collapse. It may therefore be of benefit to the patient to utilize other forms of therapy aimed at correction of these complications in conjunction with recompression. Decisions to use such adjuvant therapeutic measures as steroids, dextran or plasma expanders should be made by the responsible medical officer in light of the patient's condition and response to treatment.

4. Action

a. Approval is hereby granted for use of these new therapeutic regimens in the treatment of diving injuries at all naval recompression facilities with oxygen administration capabilities when, in the opinion of the responsible medical officer, such therapy would benefit the patient.

b. Regulations for use of the Minimal Recompression, Oxygen Breathing Method for treatment of Decompression Sickness and Air Embolism (tables 5 and 6) are given in enclosure (1). Use

of these tables must be under the supervision of a medical officer. A qualified medical attendant must always accompany the patient in the chamber during treatment.

c. Regulations for use of tables 5A and 6A for treatment of air embolism incurred during submarine escape training are given in enclosure (2). Use of these tables must be under the supervision of a medical officer. A qualified medical attendant must always accompany the patient in the chamber during treatment.

d. The use of oxygen breathing without recompression is not approved in any situation except in case of an emergency, during transport to a compression chamber, or during an interim period while recompression facilities are being prepared for use.

e. The responsible medical officer shall, in accordance with reference (b), submit a Report of Decompression Sickness and all Diving Accidents (MED-6420-1) on NAVMED 6420/1 after each use of table 5, 5A, 6 or 6A. Details for submissions are given in paragraph 1.9.8 of reference (a).

R. B. BROWN

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Minimal Recompression, Oxygen Breathing Method for Treatment of Decompression Sickness and Air Embolism (Tables 5 & 6)

TABLE 5

Depth (Feet)	Time (Minutes)	Breathing Media	Total Elapsed Time (Minutes)
60	20	O_2	20
60	5	Air	25
60	20	O_2	45
60-30	30	O_2	75
30	5	Air	80
30	20	O_2	100
30	5	Air	105
30-0	30	O_2	135

TABLE 6

Depth (feet)	Time (minutes)	Breathing Media	Total Elapsed Time (minutes)
60	20	O_2	20
60	5	Air	25
60	20	O_2	45
60	5	Air	50
60	20	O_2	70
60	5	Air	75
60-30	30	O_2	105
30	15	Air	120
30	60	O_2	180
30	15	Air	195
30	60	O_2	255
30-0	30	O_2	285

INSTRUCTIONS FOR USE OF TABLES 5 and 6

1. Choice of Table. The short (135 minute) schedule of table 5 is used for treatment of "pain only" bends if all pain is completely relieved within 10 minutes of reaching 60 feet. The long (285 minute) schedule of table 6 is used for all serious symptoms, for recurrence, or if pain is not completely resolved after 10 minutes at 60 feet.

2. Oxygen Breathing. Commence O_2 breathing prior to descent. Descent time is not counted as time at 60 feet. If oxygen intolerance develops it should be treated as described in figure 1.

3. Descent. Normal rate of descent is 25 feet per minute. A more rapid descent is desirable if more serious symptoms are present.

4. Ascent. Ascent is continuous at 1 foot per minute. Do not compensate for slowing of the rate by subsequent acceleration. Do compensate if the rate is exceeded. If necessary halt ascent and hold depth while ventilating the chamber.

5. Relief not Complete. If relief is not complete at 60 feet, proceed with table 6 and observe patient's condition closely for any change, lengthen the schedule if thought necessary, or compress to 165 feet and treat patient on table 2, 2A, 3 or 4 of reference (a), as appropriate.

Enclosure (1)

6. Recurrence. If symptoms recur or if new symptoms appear during course of treatment with Tables 5 or 6, return to 60 feet and re-treat the patient on Table 6.

7. Lengthened Treatment. Table 6 can be lengthened by an additional 25 minutes at 60 feet (20 minutes O_2 - 5 minutes air) or an additional 75 minutes at 30 feet (15 minutes air - 60 minutes O_2) or both.

8. Medical Evaluation. Before making a recommendation the responsible medical officer should carefully consider:

a. The diagnosis and exact condition of the patient.
b. The nature of any defect remaining.
c. The diving schedule which precipitated his injury and the magnitude of the omitted decompression, if any.
d. The time intervals elapsed between the end of the patient's last dive, the onset of injury, and the commencement of treatment.
e. The circulo-pulmonary condition of the patient and the status of his inert gas exchange.
f. The presence of other medical conditions which might complicate treatment or necessitate later transfer to a hospital.
g. Adjuvant medical treatment which might be of benefit.

9. Serious Symptoms. Unconsciousness, convulsions, weakness or inability to use arms or legs, air embolism, any visual disturbances, dizziness, loss of speech or hearing, chokes, bends under pressure.

10. Tender. Tender routinely breathes chamber air. If the treatment schedule is lengthened or if the treatment constitutes a repetitive dive for the tender, he must breathe oxygen for the final 30 minutes of ascent from 30 feet to the surface.

FIGURE 1.

Oxygen Administration: Rules, Routines, Reactions and Precautions

If Oxygen Intolerance Occurs or is Anticipated

a. Halt ascent; remove mask at once; maintain depth constant;
b. protect a convulsing patient from injury due to violent contact with fixtures, deckplates or hull, but do not forcefully oppose convulsive movements;
c. with a padded mouthbit protect the tongue of a convulsing patient;
d. for non-convulsive reactions, have patient hyperventilate-with chamber air-for several breaths;
e. administer sedative drugs upon direction of a medical officer;
f. 15 minutes after the reaction has entirely subsided resume the schedule at the point of its interruption;
g. if the reaction occurred at 60 feet, on the 135 minute schedule; upon arrival at 30 feet switch to 285 minute-schedule (15 minutes air-60 minutes oxygen, 15 minutes air-60 minutes oxygen).

Oxygen Reactions - Symptoms

Twitching (fasciculations or tremors) of facial muscles and lips; nausea; dizziness and vertigo; vomiting; convulsions; anxiety, confusion; restlessness and irritability; malaise; disturbances of vision and narrowing of visual fields; incoordination; tremors of arms and legs; numbness or "tingling" of fingers or toes; fainting; spasmodic breathing.

Oxygen Administration-Preparedness	Oxygen Administration-Routine Practices	Fire Warning
a. Sufficient cylinder supply b. Demand valves operative c. Emergency kit stocked d. Tenders trained to manage reactions e. O_2 humidified if possible f. Depth gauges currently in calibration	a. Insure patient is as comfortable as possible b. Patient at complete rest c. Insure snug face-mask fit d. Follow air-O_2 schedule closely e. Be alert for signs or symptoms of reactions f. Patient to take a few deep breaths every five minutes during treatment	Danger of ignition and propagation of fire increased under pressure, as O_2 is exhaled into the chamber atmosphere the hazard is magnified. Ample ventilation must be provided. Do not use electrical appliances. Keep combustibles clear of the chamber.

Enclosure (1)

Oxygen Breathing Method for Treatment of Air Embolism Incurred During Submarine Escape Training (Tables 5A and 6A)

TABLE 5A

Depth (Ft.)	Time (Min)	Breathing Media	Total Time (Min.)
165	15*	Air	15
165-60	4	Air	19
60	20	O_2	39
60	5	Air	44
60	20	O_2	64
60 - 30	30	O_2	94
30	5	Air	99
30	20	O_2	119
30	5	Air	124
30 - 0	30	O_2	154

*Total time will vary as function of this stop. Medical attendant should take enough time to accomplish a thorough physical examination, since the ensuing treatment is based on patient's physical status.

TABLE 6A

Depth (Ft.)	Time (Min)	Breathing Media	Total Time (Min.)
165	30	Air	30
165 - 60	4	Air	34
60	20	O_2	54
60	5	Air	59
60	20	O_2	79
60	5	Air	84
60	20	O_2	104
60	5	Air	109
60 - 30	30	O_2	139
30	15	Air	154
30	60	O_2	214
30	15	Air	229
30	60	O_2	289
30 - 0	30	O_2	319

Instructions for Use of Tables 5A and 6A

1. Recompression to 165 feet should be accomplished as rapidly as possible (usually less than 1 minute).

2. Total time at 165 feet will vary with the clinical status of the patient. The medical attendant should take the time to make a thorough physical appraisal of the patient. If all major symptoms and signs are gone before 15 minutes total bottom time, proceed to 60 feet at 25 feet per minute on air and begin oxygen as in table 5A.

3. If serious or major symptoms or signs persist beyond 15 minutes, but show signs of moderating within 30 minutes (total bottom time), proceed to 60 feet at 25 feet per minute and begin oxygen as in table 6A.

4. Should serious symptoms and signs persist beyond 30 minutes at 165 feet without moderation, begin treatment on table 4 of reference (a).

Enclosure (2)

5. Recurrence. If symptoms recur or if new symptoms appear during course of treatment with tables 5A or 6A, return to 60 feet and retreat patient according to table 6A.

6. Relief not Complete. If relief is not complete at 60 feet, proceed with table 6A and observe patient's condition closely for any change, lengthen the schedule if thought necessary or compress to 165 feet and treat patient on table 4 of reference (a).

7. Lengthened Treatment. Table 6A can be lengthened by an additional 25 minutes at 60 feet (20 minutes O_2 · 5 minutes air) or an additional 75 minutes at 30 feet (15 minutes air - 60 minutes O_2) or both.

8. Oxygen Toxicity. Should oxygen intolerance develop during the course of treatment, discontinue the oxygen until 15 minutes after the reaction has entirely subsided, then resume the schedule at its point of interruption. If the reaction occurred at 60 feet on table 5A, upon arrival at 30 feet switch to table 6A.

9. Tenders. Inside tenders routinely breathe air; however, if treatment constitutes a repetitive dive for a tender, he must breathe O_2 from 30 feet to the surface.

10. Serious Symptoms. Unconsciousness, convulsions, major paralysis or weakness, cranial nerves signs, cerebellar signs.

11. Followup. Patients with air embolism on leaving the chamber should be routinely admitted for observation and given a thorough medical examination, including X-ray carefully checking for pneumothorax. Patients should be released to duty only if completely asymptomatic and medical clearance is indicated by examination.

Appendix V.

Clinical Experience with O_2-Recompression for Traumatic Air Emboli.

Explanation of Tables and included abbreviations for Tables V-1 and V-2.

All time is given in minutes only or in hours:minutes

Dive — Denotes purpose of dive, number of dives, and identifies cases other than decompression sickness of divers.

A	Altitude exposure	R	Recreational dive
B	Buoyant ascent	W	Working dive
C	Civilian diver	2-5	Number of dives, if more than one
E	Experimental dive		

Mix — Breathing mixture during exposure.

Max Depth or Max Altitude — Greatest depth or altitude attained has been listed in cases with multilevel or repetitive exposures.

Total Bottom Time or Time — Sums of individual bottom times have been listed in cases with repetitive dives. Time at greatest altitude is given for altitude exposures.

Ascent — 1st or 2nd ascent of escape training cycle.

Pain, Neurological, Other

1	Minimal	M	Motor deficit	C	Convulsion
2	Mild	S	Sensory deficit	U	Unconsciousness
3	Moderate	SS	Special sense organ involvement (e.g. eye)	N	Nausea
4	Severe			V	Vomiting

Onset Symptoms — Time from surfacing to appearance of first symptoms, or time from reaching altitude to appearance of first symptoms.

Onset Therapy — Time from onset of first symptom to beginning of adequate therapy.

Onset Relief — Time from beginning of adequate therapy to onset of complete relief.

Table
- 5 Table 5 Short oxygen treatment table
- 6 Table 6 Long oxygen treatment table
- SMC Submarine Medical Center modification for treatment of air embolism.

Result
- 1 Relief complete
- 2 Residual deficit
- 4 Recurrent symptoms

Treatment of Traumatic Air Embolism in Divers

Table V-1

CASE		EXPOSURE					SYNDROME			TREATMENT					REMARKS
EDU	NR	DIVE	AGE	MIX	MAX. DEPTH	TOTAL BOTT. TIME	PAIN	NEURO	OTHER	ONSET SYMPTOMS	ONSET THERAPY	RELIEF	TABLE	RESULT	
—	R24	E	29	AIR	245	21	2	2S	—	P	—	—	6	1	
272	S1	E	30	AIR	66	17	—	4M	—	10	4	5	5	1	
061	U1	CR	21	AIR	20	90	2	4U	C–S	15	10:27	8	—	1	TREATMENT TO 33 FEET ONLY. PNEUMOTHORAX, MEDIASTINAL EMPHYSEMA
280	34	W	31	HeO_2	150	10	—	3SM	—	2	60	—	6	1	SYMPTOMS RELIEVED WITH OXYGEN BREATHING ON THE SURFACE
401	35	CW2	50	AIR	90	35	4	4UM	—	1	45	—	SMC	2	RESIDUAL MINIMAL WEAKNESS AND NUMBNESS OF RIGHT SHOULDER.
471	36	W	34	AIR	150	6	—	4MS	—	P	38	27	6	1	THIRD AIR EMBOLISM IN THIS DIVER

Treatment of Traumatic Air Embolism in Submarine Escape Trainees

Table V-2

CASE		EXPOSURE				SYNDROME			TREATMENT							REMARKS
EDU	NR	DIVE	AGE	DEPTH	ASCENT	PAIN	NEURO	OTHER	ONSET SYMP.	ONSET THERAPY	TABLE	TIME AT 165 FT.	RELIEF	DEPTH OF RELIEF	RESULT	
054	N1	B	18	50	1ST	—	U4M	—	0	1	SMC	27	8	165	1	MEDIASTINAL EMPHYSEMA
060	P2	B	20	50	1ST	—	U-C	—	0	3	SMC	30	30	165	6	RECURRENCE AT 30 FEET. RETREATED ON TABLE 4. (Ref.182)
248	N2	B	27	50	1ST	3	4SS-M-S	—	0	3	SMC	30	56	30	1	
249	P5	B	21	50	2ND	—	3M-S	—	0	1	SMC	30	20	165	1	
269	P10	B	27	100	1ST	—	C-4M	—	P	3	SMC	30	67	60	1	PNEUMOTHORAX, PNEUMOPERICARDIUM
423	27	B	20	60	1ST	—	U	—	0	1	SMC	30			4	DETERIORATION IMPROVED WITH OXYGEN BREATHING AT 60 FEET. COMPLETE RECOVERY. PNEUMOTHORAX. (Ref 182)
425	28	B	18	50	1ST	—	U	—	0	1	SMC	22	22	165	1	
426	29	B	21	50	2ND	—	U-3M	—	P	1	SMC	6	2	165	1	VERTIGO IN WATER
473	30	B	19	50	2ND	3	U-4M	—	0	1	SMC	17	35	60	1	PNEUMOPERICARDIUM. CHEST PAIN AFTER FIRST ASCENT NOT REPORTED
490	31	B	20	50	1ST	2	3S-M	—	10	14	SMC	9	2:00	30	1	
501	32	B	19	50	1ST	—	4M	—	0	3	5	—	1	60	1	
479	33	B	20	50	2ND	—	U4M-SS	—	0	1	SMC	30	15	165	4	NEUROLOGIC DETERIORATION AT 30 FEET. RETURN TO 60 FEET AND RE-TREATED. COMPLETE RECOVERY. PNEUMOTHORAX, MEDIASTINAL EMPHYSEMA, AND RUPTURED EARDRUMS. (Ref.182)