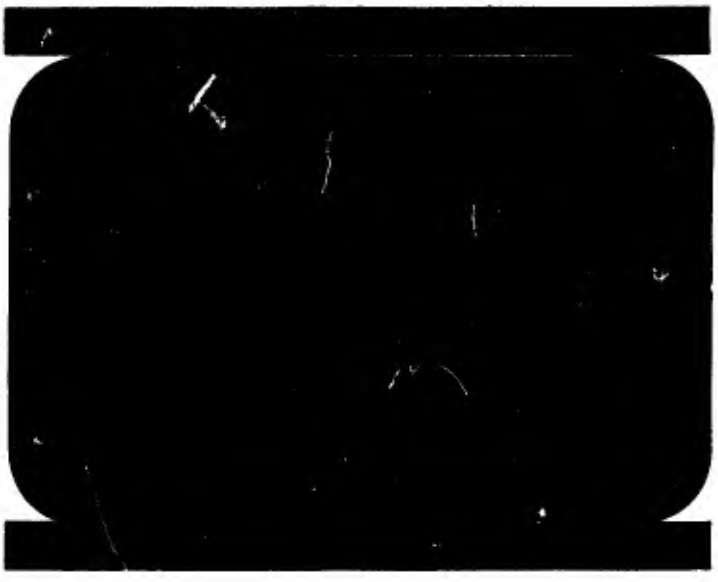

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ATTENUATION OF BIOLOGICAL STRESS FROM COMBINED VIBRATION AND ACCELERATION BY USE OF PRESSURE BREATHING

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By

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ATTENUATION OF BIOLOGICAL STRESS FROM COMBINED VIBRATION AND ACCELERATION BY USE OF PRESSURE BREATHING

ABSTRACT

The complex launch and reentry environment of future manned spacecraft and the associated crew stresses resulting from exposure to the dynamic loads are analyzed. The analysis provides a strong theoretical basis for predicting significant increases in crew stresses from vibrations in future space operations. The biomechanics of g, vibration and hypobaric stress summation are identified. Techniques designed to attenuate the independent and combined stresses are presented along with the theoretical grounds for postulating the protection concepts. Recommended techniques include use of positive pressure breathing and increased space cabin environmental pressure. The theory predicts that positive pressure breathing will reduce tissue stresses by maintaining a near normal one g mid-position of the diaphragm during exposure to the complex stress, will increase the restoring force for a given upward displacement of the vibrating abdominal mass and will increase the resonant frequency of the chest-abdomen system. A significant protective effect expected from an increased total cabin pressure also results from increased restoring forces. Animal experiments were conducted to demonstrate the theorized cardiorespiratory tissue protection provided by pressure breathing during exposure to vibration stress. The results substantiate the theory. Additional complex stress experiments required to fully validate the effectivity and practicality of the proposed attenuation techniques are described.

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INTRODUCTION

A comparison of vibration characteristics of current and future manned spacecraft flags out definite trends which merit careful consideration as regards crew comfort and performance. First, sound pressures in the frequency range of 15 cps and below are progressively greater for Saturn and Nova boosters than for Atlas and Titan. Second, the natural or resonant bending modes of the larger boosters and spacecraft occur at lower frequencies than those of smaller spacecraft. For example, sound pressures measured at equal distances from Atlas and Saturn boosters were 130 db and 140 db, respectively; peak sound pressure for the Atlas occurred at nearly twice as high a frequency as that of Saturn; lateral bending modes of Atlas range from approximately 3-30 cps, whereas a survey indicates Saturn's first three lateral and two torsional bending modes all occur within the range of 3-12 cps.[1-4] These vibrational characteristics appear singularly important when one considers that spacecraft structures transmit acoustic energies most efficiently when the sound energy has a frequency at or near the vehicle's natural resonant frequency. Therefore, it may be expected that compared to the Atlas, future large spacecraft will be exposed to much greater sound pressures transmitted very effectively in the frequency range below 12 cps, precisely the range in which human tolerance is lowest.

The thorax-abdomen system in man contains the group of organs and tissues which display extreme susceptibility to vibration injury and the frequency range for minimal tolerance of this system is approximately 3-10 cps.[5-13] It is very difficult to protect man from vibrations in this frequency range by use of mechanical damping techniques.[8, 10, 11] In addition, since the major vibration exposures occur during launch and reentry, the interaction and possible summation of launch or reentry acceleration stress and vibration stress must be carefully considered. This possibility of synergistic effects is made apparent by the observation that chest pain and respiratory difficulty which determine man's tolerance end point for low frequency vibrations also determine his tolerance end point for launch-type accelerations. There is also likelihood that these stresses will show a relative increase due to transient degradation of human tolerance for dynamic stresses after prolonged exposure to weightlessness. These trends coupled with the nature of man's response to them indicate that space crew stresses from launch and reentry dynamics are likely to increase. Accordingly, new attenuation techniques should be under study now for future application, if required.

Theoretical considerations of probable mechanisms of interaction of linear and sinusoidal accelerations simultaneously applied to man under spaceflight conditions led to a concept for attenuation of the complex stress. The goal of this paper is to describe independent and combined effects of these space vehicle launch dynamics upon man's cardio-respiratory system and how they lead to the theoretical basis for the proposed attenuation technique. The discussion, although limited to launch phase dynamics, could be applied in like manner to the dynamic responses encountered during reentry. Results of animal experiments to validate the attenuation concept are presented.

LAUNCH VIBRATION EFFECTS IN THE CARDIO-RESPIRATORY SYSTEM

For this discussion, it is assumed that during launch and reentry, the space vehicle structures will be exposed to a broad spectrum of vibrations and will transmit to the crew transient transverse, lateral, and longitudinal vibrations in the 3-10 cps range. Roman determined the major cause of injury to cardio-respiratory organs exposed to these low-frequency vibrations to be the alternate stretching and compression of the tissues induced by oscillations of the pulmonary diaphragm and abdominal tissue mass in and out of the thoracic cavity.(6) This invasion of the thoracic cavity by the abdominal mass was shown to be caused by either transverse or longitudinal vibrations. His work established the concept for protection of these tissues from vibration by employing external restraints to the abdomen and chest to restrict their motion. This mechanism of injury (relative visceral displacement) is the same during transverse ($\pm$ Gx), lateral ($\pm$Gy), and longitudinal ($\pm$ Gz) vibrations, but the magnitude of stress varies with the vibration frequency, amplitude, and direction of application. Studies with human subjects restrained in a Mercury-type contoured couch disclosed tolerance end points to occur at vibration levels of about 3, 2, and 2 g's for $\pm$Gx, $\pm$Gy, and $\pm$Gz, respectively; the critical frequency ranges for $\pm$Gx, $\pm$Gy, and $\pm$Gz vibration stresses were found to be approximately 6-10 cps, 3-6 cps, and 5-7 cps, respectively.(13) Chest pressure, chest pain and respiratory difficulties were major factors limiting tolerance in all cases.

Measurements of the volume of air which oscillated in and out of the chest of supine subjects exposed to longitudinal $\pm$Gz vibrations showed maximum oscillating volume, maximum abdominal displacement and maximum expansion of the thorax all to occur at a frequency of 3-4 cps.(8, 9) When the frequency of

vibration was increased above about 10 cps, nearly all of the above effects disappeared. Seated subjects exposed to constant amplitude ±Gz vibration showed maximum oscillating air volume per oscillation at about 8 cps.(14) Transverse ±Gx vibration of semi-supine subjects demonstrated maximum chest strain and intra-abdominal pressures to occur at about 7 cps.(11, 12)

These data indicate that in the frequency range of maximum vibration stress (3-10 cps), chest pain, respiratory difficulties, chest deformation, and pulmonary gas flow in and out of the chest are also maximum. It is apparent that if this oscillating flow of air were prevented during vibration exposure, the intrapulmonary gas pressure would rise in proportion to reduction of lung volume caused by displacement of the diaphragm into the chest cavity. As the lung pressure increased, the rising pressure exerted against the upper surface of the diaphragm would resist its further invasion of the chest cavity. This very important restoring force created by the increased pulmonary gas pressure would reduce the magnitude of the restoring forces that must otherwise be generated by stretching and compression of thoraco-abdominal organs and connecting tissues to arrest cephalad displacement of the diaphragm and adjacent viscera. This concept for reducing vibration-induced shear and tensile stresses in cardio-respiratory and abdominal tissues by substituting increased pulmonary gas pressure is fundamental to the attenuation technique proposed by results of the theoretical analysis in this study. The effectivity of the technique will be influenced by the total ambient pressure.

EFFECTS OF CABIN PRESSURE ON VIBRATION STRESS IN THE CARDIO-RESPIRATORY SYSTEM

As previously stated, if pulmonary gas flow in and out of the chest were completely prevented during vibration exposure the product of lung volume and pressure would tend to remain constant. In this situation, a decrease in volume caused by elevation of the diaphragm would cause a corresponding increase in lung pressure as expressed by the relationship $P_1V_1 = P_2V_2$. Under these conditions, the magnitude of the restoring force created by a given upward displacement of the diaphragm will be directly proportional to the initial lung pressure. For example, a diaphragmatic elevation which caused a 10% reduction in lung volume would increase the lung pressure by 1/3 PSI if the initial lung pressure were 3 PSI and would increase it by 5/3 PSI if the initial lung pressure were 15 PSI. The additional 4/3 PSI restoring force obtained at the higher initial cabin and lung pressure when applied over the upper surface of the diaphragm (about 40 square inches in man) would be a highly significant force resisting upward motion of vibrated tissues. The example assumes no escape of air from the chest during exposure to the vibration. In the frequency range of concern there will be an oscillating air volume associated with the oscillating lung pressures. The restoring force in the example would therefore be less than the calculated value, but would nevertheless be a very significant factor in damping cephalad motion.

It may therefore be argued that if pressure developed in the lungs is well tolerated and if this increased pressure substitutes for poorly tolerated stresses, it provides a technique for protecting cardio-respiratory tissues from vibration energies. There is a great deal of evidence which indicates that tissues have a relatively great tolerance for compressive forces. Accordingly, the hypothesis is advanced that techniques which selectively increase pulmonary

pressure (within a physiologically acceptable range) during vibration exposure will reduce the absolute cephalad displacement of the diaphragm and will decrease tissue injury. Since, normally, intrapulmonary pressure is essentially equal to the ambient cabin or space suit pressure, the hypothesis can be rewritten to state that an increase in total cabin pressure will decrease cardiorespiratory tissue stresses otherwise resulting from a given exposure to low frequency vibrations. The exception to this general statement arises when increasing the cabin and pulmonary pressure shifts the resonant frequency of the thoraco-abdominal system in the direction of the frequency of the vibrating vehicle and increases the transmitted forces and tissue deformation. The alteration of resonant frequencies of body parts by change in pulmonary gas pressure is discussed in a subsequent section of this paper.

LAUNCH ACCELERATION EFFECTS IN CARDIO-RESPIRATORY SYSTEM

The launch acceleration profile is expected to consist of stages with non-linear rises from zero to approximately 7 or 8 g. For purposes of this discussion, it is assumed that the crew will be restrained in Mercury type contoured couches and oriented in the semi-supine position relative to the launch acceleration vector. During the sustained launch acceleration, as the g load increases, the normal weight of a given volume of tissue or body fluid will be multiplied by the number of g units of acceleration applied. In the case of blood or formed tissues, this will cause a marked increase in the pressure exerted upon underlying body tissues. Therefore, with increasing g, the pressure gradient developed in soft abdominal tissues will cause them to flatten and spread out. Lateral and dorsal motion or deformation of the abdominal mass will be resisted by the couch restraint; motion toward the feet will be resisted by the bony pelvis, perineum and couch seat; headward motion produces pressure against the diaphragm and adjacent thoracic contents.

It is apparent that unless the acceleration produces an equal increase in pressure within the thoracic tissues, the diaphragm will tend to be displaced into the chest. It can be shown that for all practical considerations, the pressure within the lungs remains essentially constant during exposure to acceleration until atelectasis develops. Consider first the forces acting within the lungs in man lying supine in a static one g environment. The principal force maintaining the lungs in their expanded condition is the fluid traction exerted between the visceral and parietal pleura. The lungs per se exert an inward pull against the chest wall and diaphragm and would contract into a small essentially airless mass if the fluid traction force were removed. The pressure of air in the airways and alveoli and blood in the

vascular channels aid in expansion of the lung tissue. However, this air pressure is equal to the ambient air pressure exerted against the external surface of the chest and abdomen and transmitted through abdominal tissues to the lower surface of the diaphragm. Next, consider these forces if the one g acceleration is increased to 10 g's. The weight of the rib cage and abdominal tissues become multiplied by 10 so anterio-posterior flattening and lateral widening will tend to occur due to this exaggerated force acting toward the back. The weight of the lungs and contained blood are also increased tenfold and these tissues tend to shift toward the back. Despite these marked increases in pressure in the lung tissues and blood, the pressure of the gas in the lungs is essentially unchanged by the acceleration due to the extremely low density of air. As long as the air in the lungs is in open communication with air in the cabin or suit environment, any pressure increase in the lungs would merely displace the air to the outside. As air is displaced by the increased tissue pressure, compressing the most dependent lung tissue, the density of the compressed tissue is increased. Inertial forces and pressure would increase as the density of the accelerated tissue increased and the cycle would continue until dependent air spaces were obliterated producing a condition of acceleration atelectasis. Accordingly, except in dependent atelectatic tissue, lung pressure will at any tissue depth tend to equal ambient air pressure during acceleration and an unbalanced force is created by the increased abdominal tissue pressure exerted against the lower surface of the diaphragm. The magnitude of this unbalanced force will increase with both g level and tissue depth and will vary from approximately zero at the ventral margin of the diaphragm to a maximum at the depth where atelectatic lung tissue is met. The usual homogeniety of lung tissue will be altered by the high density

atelectatic tissue in dependent areas and the relatively bloodless air filled alveoli ventrally.[15] The resulting reduced exchange of gases between blood stream and lungs, coupled with impaired circulation, disturb oxygen transport functions. The elevation and dorsal displacement of the diaphragm caused by the unbalanced forces will induce a correlated displacement of the apex of the heart.

The diaphragm and thoraco-abdominal tissues will provide increasing resistance to upward motion as their normal physiological range of motion is exceeded. Tensile stresses developing in stretched connective tissues and associated nerves and vessels will, for a given tissue displacement, be low until the physiological range of motion is exceeded. Tissue stress per unit of added displacement will increase rapidly as elastic limits are approached. These non-linear responses are illustrated by the following measurements of expiratory reserve taken during exposure to increasing transverse acceleration. Expiratory reserve decreased rapidly in a nearly linear fashion up to acceleration levels of about 4 g. Beyond this, the decrease per g became extremely small, such that the total reduction at 4 g was nearly as great as the total reduction measured at 8 g.[16] The measure of the decrease in expiratory reserve may be viewed as a measure of the upward displacement of the diaphragm induced by the transverse g. Symptoms of chest pain, difficult breathing, and abdominal pressures are encountered in the same g range where these volume changes appear to reach a plateau. These findings are consistent with what would be expected from tissues stressed near their elastic limits.

As deformed tissues are stretched, they will exhibit increased stiffness. If their mass remains essentially unchanged, the stiffness to mass ratio will increase and their resonant frequency will be expected to increase approximately as the square root of the change in the ratio.

The foregoing discussions indicate several cardio-respiratory system responses to sustained transverse launch and reentry type acceleration which can be expected to interact with and significantly modify this system's response to vibrations when the two stresses are combined. These include: (1) increased inertia or resistance to displacement in the direction of the sustained acceleration vector of otherwise easily mobilized tissues; (2) pre-stressing of thoraco-abdominal tissues due to sustained abnormal cephalad displacement of the midposition of the pulmonary diaphragm; (3) production of a positive density gradient from the aerated ventral to the atelectatic dependent lung tissues; (4) alteration of cardio-respiratory and abdominal tissue stiffness and resonant frequencies; (5) abnormal elevation and rotation of the heart.

COMBINED EFFECTS OF LAUNCH, VIBRATION, AND ACCELERATION IN THE CARDIO-RESPIRATORY SYSTEM

During launch, the dynamic environment will be constantly changing due to changing velocity vectors, reduction in vehicle mass as fuel is expended, buffeting loads, boundary layer phenomena, and other causes. At the time when the linear launch acceleration reaches 5 g, inertial forces equivalent to the 5 g acceleration will restrain the body tightly against the couch. Soft abdominal tissues will be flattened, displaced dorsally and cephalad and will exhibit unusual resistance to motion toward the ventral surface of the body. The diaphragm is displaced near its maximum upward position and the tensile and shear stresses in stretched tissues are expected to be approaching elastic limits. The apex of the heart will tend to be rotated up and displaced toward the dorsum of the thorax. The homogeneous cushion of air-filled lung tissues normally interposed between the heart and the hard structures of the thoracic cage may be replaced dorsally by compressed atelectatic lung tissue. The lung tissue ventral and lateral to the heart would be expected to be of low density and stiffness. It should have resonant frequency modes quite different from the atelectatic tissues and should, by comparison, display loose coupling with the heart.

If a 2-g transverse or longitudinal vibration level were now superimposed on the linear launch g, the mid-point for the oscillatory motion of the vibrated diaphragm would be abnormally elevated. Although the amplitude of the elastic motion induced by vibration of a given frequency and energy level will tend to be decreased by the tissue stiffening and inertial effects of superimposed linear acceleration, the maximum upward position and deformation of tissue would be expected to be greater than if the same vibration were superimposed

on a normal 1-g linear acceleration or if either of the two forces were applied singly.

Since the central area of the diaphragm and the pericardium are fused and the lung bases are held in contact with the upper surface of the diaphragm by fluid traction forces, vibratory motion of the diaphragm causes motion of the tissue sack surrounding the heart and great vessels and causes motion of adjacent lung tissues. Marked posterior displacement of the heart and loss of the normal cushion of lung tissue posterior to the heart could increase the transmission of mechanical impact forces between the heart and vertebral bodies. Mechanical stimulation of the vibrated heart and stretching of major vessels have been suggested as likely causes of chest pain during vibration.(7) The position and dynamic response of the heart during combined vibration and acceleration may be a major determinant of overall crew tolerance. Since maximal vibration effect is anticipated at the resonant frequency, the resonant characteristics of the heart should be considered.

The heart undergoes marked changes in stiffness and mass during its normal function, and should exhibit a relatively wide range of maximum response or resonant frequencies. For example, just prior to systolic contraction of the heart muscle, the relaxed heart and contained blood at low pressure would weigh approximately 700 grams for an average man. Near the end of systolic contraction, the heart is working against the maximum blood pressure and the mass is reduced to something less than 600 grams due to expulsion of blood from the ventricles. For purposes of comparison, if it were assumed that the stiffness of the heart structure were twice as great during systolic contracture as when dilated and relaxed, the resonant frequency equal to the square root of the ratio of the stiffness to the mass could be more than half again as great in

the partially empty, contracted state as in the filled and relaxed condition. In this event, if the resonant frequency were 6 cps for the diastolic condition, it would be about 10 cps near the end of systole. At a forced vibration frequency of 8 cps the heart would oscillate several times during a single cardiac cycle and one could predict a flat maximum effect curve across the 6-10 cps range. A maximum sensitivity might be expected near the lower frequency of this range, since the diastolic phase of the cardiac cycle is longer than the systolic phase and the inertial forces would be greatest in this phase. A marked change in the normal resonant frequency range of the heart might indicate disturbed cardiac dynamics. A narrowing toward the lower end of the normal frequency range might be expected to accompany a condition of incomplete emptying or cardiac dilitation as found in myocardial decompensation or certain valvular defects. Narrowing toward the upper end of the normal frequency range might be expected in the event of inadequate cardiac filling associated with valvular defects or impaired venous return. In the light of these considerations, and assuming the heart is an important determinant of tolerance, one can predict a flat minimum tolerance curve across a relatively broad frequency range to result from the summation of cardio-respiratory effects from simultaneously applied transverse vibration and transverse linear acceleration. This effect is related to the abnormally elevated and dorsally displaced position of the heart. A lower mid-position of the pulmonary diaphragm would tend to stretch the pericardial sack and enclosed heart and great vessels downward and forward relative to the above described position. Likelihood of vibration of the heart against the posterior rib cage and diaphragm would seem to be significantly lessened with the heart restrained in a more normal vertical position.

In accordance with the above discussion, a significant anticipated biological effect of combined vibration and sustained linear acceleration is the increased tissue displacement and deformation that results from summation of the sustained inertial force from the acceleration and the inertial force acting in the same direction during one phase of the vibration. The resulting maximum deformation exceeds, in that one direction, the deformation expected from separate application of either of the stresses, although the amplitude of vibratory motion about the mid-point may be significantly decreased by the inertial effects of the sustained acceleration.

A second anticipated effect of the complex dynamic stress is alteration of natural resonant frequency modes of involved tissues and organs due to changes in stiffness and effective mass. This effect may be an asset or a detriment depending upon resonant modes of adjacent tissues and the frequency of the vibration imparted to the subject from his vehicle environment.

Major detrimental effects expected from the complex stress therefore include excessive cephalad displacement of both the maximum and mid-positions of the vibrating heart, diaphragm, and abdominal mass, increased tissue stresses, and increased transfer of vibration forces to the heart due to loss of the homogeneous cushion of lung tissue that normally surrounds the heart and protects it from mechanical impacts. According to this concept, attenuation of combined g and vibration stress may be expected from any physiologically acceptable technique which prevents excessive upward displacement of the heart, pulmonary diaphragm and apposed abdominal mass otherwise induced by sustained transverse acceleration.

THE ATTENUATION CONCEPT

The foregoing discussions indicate that launch acceleration, launch vibration, and decreased environmental pressure produce a complex stress in the cardiorespiratory system of man through an additive effect which causes excessive elevation of the pulmonary diaphragm and heart with associated alteration of the mechanical coupling between the heart, lungs, and adjacent body structures. Application of positive pressure to the lungs would produce an opposite effect by lowering the position of the diaphragm and precluding pulmonary tissue deformation otherwise encountered.[15, 17] Accordingly, cephalad tissue displacement induced by transverse linear acceleration may be prevented by use of positive pressure breathing allowing the diaphragm and adjacent organs to remain within their normal physiological range of positions during exposure to transverse accelerations. The increased lung pressure and increased resistance to outflow of gas from the lungs resulting from application of positive pressure breathing would significantly increase the restoring forces to inhibit headward tissue excursions during vibration.

As discussed previously, an increased total environmental pressure when reflected in an increased pulmonary pressure would likewise increase the magnitude of restoring forces and decrease the amplitude of cephalad tissue excursions for a given low frequency vibration exposure. Increased cabin pressure should also reduce transverse g stress since the increase in air density accompanying the increase in pressure would tend toward producing a pressure gradient in the accelerated lungs to balance the gradient produced in accelerated abdominal tissue. However, within the constraints on cabin pressure imposed by practical design and gas toxicity considerations--this increase in gas density would not be enough to significantly alter the unbalanced forces acting on the diaphragm. The augmented

resistance to headward displacement of the thoraco-abdominal tissue mass resulting from application of positive pressure breathing and increased ambient pressure when added to the resistance to displacement in all other directions provided by the couch, pelvis, and inertial forces of launch acceleration may be expected to effectively reduce deformation and injury from combined transverse g and vibrations.

In regard to cabin pressure requirements for optimum attenuation, it would appear that the higher the pressure the better the attenuation of g and vibration stress. If so, total cabin pressure should be as high as compatible with gas toxicity, decompression hazards, leak rates and other design constraints.

The optimum amount of positive pressure breathing for attenuation of combined g and vibration forces would be expected to be that required to maintain the diaphragm and heart as nearly within their normal functional range of motion as practical. Intuitively, one would suspect the ideal mid-position of the diaphragm and heart to resist dynamic stress would be the position normally occupied by these structures when man involves himself in dynamic actions of work and stress--the erect standing attitude. It appears impossible to maintain the normal shape of the diaphragm by pressurizing the chest during acceleration exposure because of the presence of the previously discussed pressure gradient acting on the lower surface of the diaphragm and absence of a gradient in the lung pressure acting upon the opposite surface. Consequently, it seems logical to select a positive pressure which balances the average abdominal tissue pressure exerted against the diaphragm at any given acceleration. In this situation, the central area of the diaphragm should remain near its normal functional position during exposure to increasing transverse g while its anterior or ventral areas would be displaced toward the feet and its

posterior areas displaced headward. The amount of positive pressure required to maintain this position of the diaphragm at a given g level will vary with body type, abdominal mass, muscle tonus, amount of atelectasis in lung tissues and position of the subject relative to the g vector. Positive g will lower the diaphragm and reduce the need for positive pressure breathing but will seriously reduce g tolerance. Centrifuge experiments have shown that providing about 2.5 mm Hg positive breathing pressure per g to men exposed to launch type transverse acceleration will maintain their expiratory reserve volumes above the normal 1 g control value for the supine position.(16) This amount of positive pressure breathing should, therefore, maintain the central area of the diaphragm during exposure to transverse g to near its normal position for the standing position in a one g field.

As stated previously, an increase in lung pressure will tend to increase the resonant frequency of the aerated portions of the lungs. Also, since the compressible gas in the chest cavity, coupled with the adjacent noncompressible abdominal tissue mass, resembles a simple spring-mass system, an increase in pulmonary gas pressure should stiffen the spring and raise the resonant frequency of the thoraco-abdominal system as a whole. In like manner, since the restoring force developed in the compressed pulmonary gas during vibration tends to be proportional to initial pulmonary and cabin pressure, the resonant frequency of the thoraco-abdominal system may be expected to vary with cabin altitude. Ability to selectively change the resonant frequency of the chest-abdomen system by changing cabin pressure or by use of positive pressure breathing may be important due to the following observation. A change in vibration frequency of 2 cps away from the maximum effect frequency of the chest-abdomen system of man can reduce the deformation and strain of the chest and

abdomen by more than 50 percent.[11] In the referenced experiment, the frequency of the forcing field (shake table) was altered ±2 cps away from the resonant frequency of the subjects. It is tempting to consider working this effect in reverse when known and relatively fixed vibration characteristics of a space vehicle indicate the crew will be exposed to their maximum effect frequency--say 7 cps. In this case, by control of cabin or suit pressure or use of positive pressure breathing, if the natural frequency of the chest-abdomen system were, for example, increased to 10 cps the biological stress from vibration could be drastically reduced.

Three techniques for attenuating combined vibration and acceleration stresses are suggested by the theoretical considerations presented:

(1) Application of positive pressure breathing in the amount required to maintain the normal one g mid-position of the diaphragm during exposure to sustained launch accelerations, plus vibrations (approximately 2.5 mm Hg positive pressure per transverse g).

(2) Utilization of the highest cabin or suit pressure compatible with design constraints, gas toxicity and decompression hazards.

(3) Application of selected cabin and/or positive breathing pressure to shift the resonant frequency of the chest-abdomen system 2 to 3 cps away from maximum biological effect frequencies transmitted to the crew by vehicle structures.

If subsequent test results validate the attenuation theory and future missions require application of these techniques, they could be exercised in various combinations.

Cabin pressure could be lowered to minimize gas leakage and decompression hazards during all mission phases except launch and reentry.

Positive pressure could be built up as launch acceleration increased to maintain the normal 1 g position of the diaphragm. In this situation, however, the full potential of the pressure breathing for attenuating vibration stress by creating maximal restoring forces for a given diaphragmatic tissue displacement would be encountered only at maximum g. The average crew member can breathe against a positive pressure of 15-20 mm Hg for time periods well in excess of launch and reentry phase durations without unduly compromizing venous return and cardiac output. Consideration can therefore be given to maintaining 15-20 mm Hg positive breathing pressure during the entire launch period while preventing excessive footward displacement of the diaphragm during periods of low g by providing a rigid abdominal restraint. The rigid restraint could be a padded contoured plate designed as a removable continuation of the contoured couch which would encircle the occupant's abdomen and lower chest area. It could be fit loose enough to enable free respiration when not using pressure breathing. Application of pressure breathing would be expected to create expansion of the chest and abdomen to create a closer fit to the restraint. Roman demonstrated rigid restraint provides remarkable protection of these body tissues from low frequency vibrations. The combination of rigid external restraint plus internal restraint and augmented restoring forces provided by increased cabin pressure and positive pressure breathing gives promise of producing a highly significant method for attenuation of combined high level transverse g and vibration stresses. The method can be implemented with minimal weight penalty since it requires only minor modification of existing onboard equipment.

ATTENUATION TEST RESULTS AND REQUIREMENTS

Demonstration of the effectivity of the three proposed attenuation techniques requires experimentation combining transverse g with vibration stress at different levels of ambient pressure and positive pressure. These combined stress experiments have not been performed but results of isolated stress experiments employing positive pressure breathing and increased ambient pressure to reduce g and vibration stress have been encouraging.

Human centrifuge experiments employing the current launch position (12^{o} elevation of the back from the supine) have demonstrated great increases in subjective comfort and time tolerance for the g stress to result from use of positive pressure breathing.(15, 16)

Megal, et al, found lung injury and mortality in rats subjected to 60 cps transverse vibration at various simulated altitudes to increase with increase in altitude above 10,000 feet.(18) The 60 cps vibration used in the above referenced study, since probably well above the resonant frequency for the thoraco-abdominal system of the rat, would not be expected to show maximal potentiation effect as regards mechanical damage to cardio-respiratory tissue. The authors concluded that under the conditions of their experiment, the hypoxic effect of altitude was the predominant factor eliciting increased injury and mortality. They reported that the primary pathological difference between animals vibrated at sea level and those vibrated at altitude is in the lungs where extensive pulmonary damage was noted. The increased pulmonary damage with increased altitude reported by the referenced research appears to be consistent with the theoretical considerations in this paper.

The absence of experimental data confirming the theorized effects of positive pressure breathing upon biological tolerance for vibration stress

stimulated our study of these effects, described in detail in another report,[19] and briefly summarized here as follows.

Mice were exposed to $\pm$Gz vibration while restrained in the apparatus shown in Figure 1. Controls and experimental animals were vibrated simultaneously so that all experimental conditions were identical except positive pressure breathing was provided to the experimental animals. Use of a few inches H_2O positive pressure breathing markedly decreased mice mortality as shown in Figure 2. Histological examination disclosed marked difference in extent of injury to the cardiopulmonary and abdominal tissues of controls as compared to experimental animals indicating a very significant protective effect (Figure 3).

The experiments described above have demonstrated that positive pressure breathing will attenuate biological stress of transverse g when the g is applied separately and of vibration when applied in a background of one g. They have demonstrated that decreased cabin and pulmonary pressure increases vibration stress when applied in a one g field. It would be enlightening to test the dependency of cardiorespiratory system vibration tolerance upon ambient pressure in the resonant frequency range of this system where increase of tolerance with increase of ambient pressure should be optimally demonstrated.

Subsequent experiments are required to determine the optimum body attitude to best tolerate complex g, vibration and hypobaric stress. As the ratio of severity of vibration stress to g stress increases, the optimum elevation of the back may exceed the 12^{o} currently utilized.

Ability to create controlled shifts of natural resonant frequency of the cardiorespiratory and chest-abdomen systems by change in position relative to the g vectors, restraints, and change in cabin and positive breathing pressures

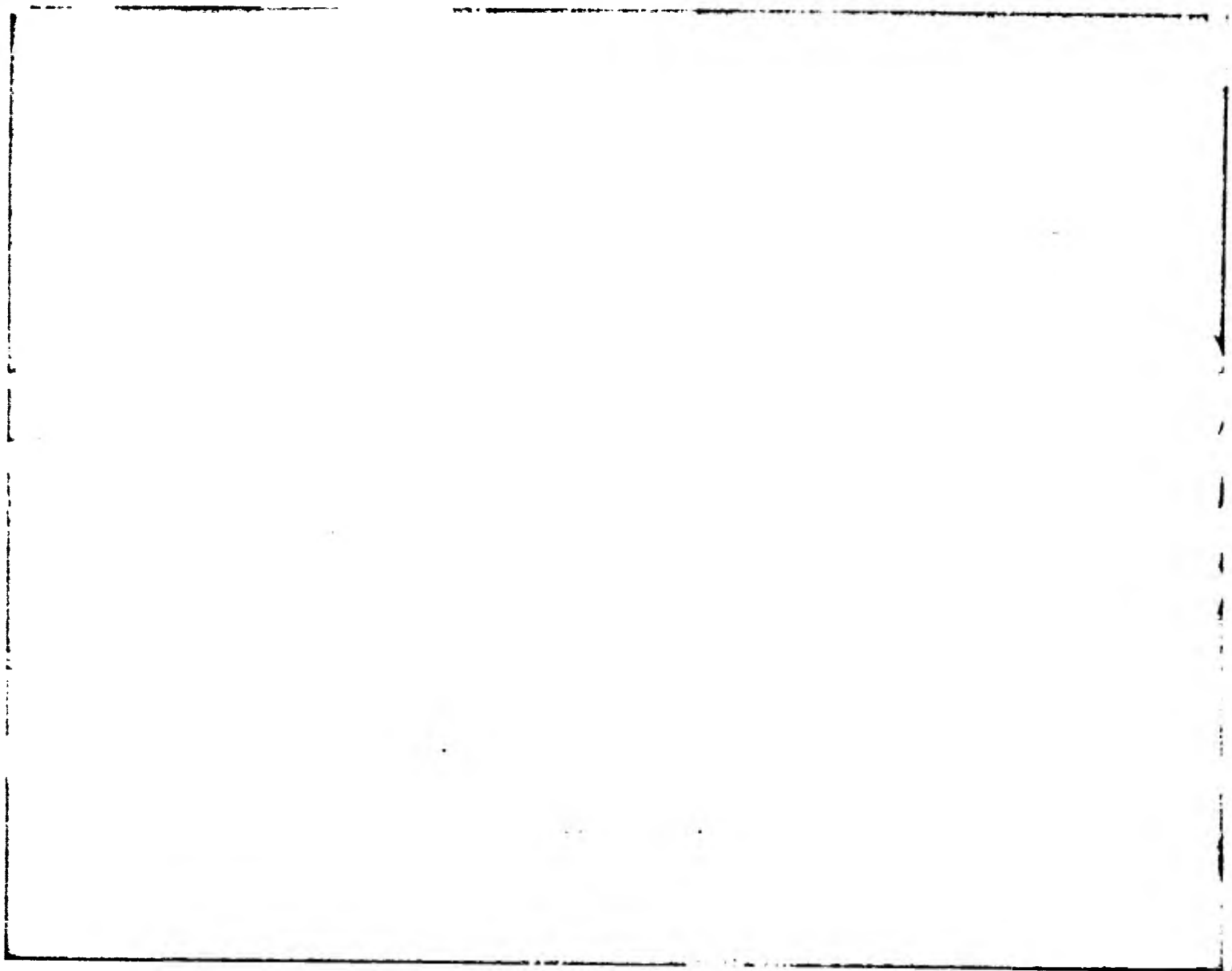
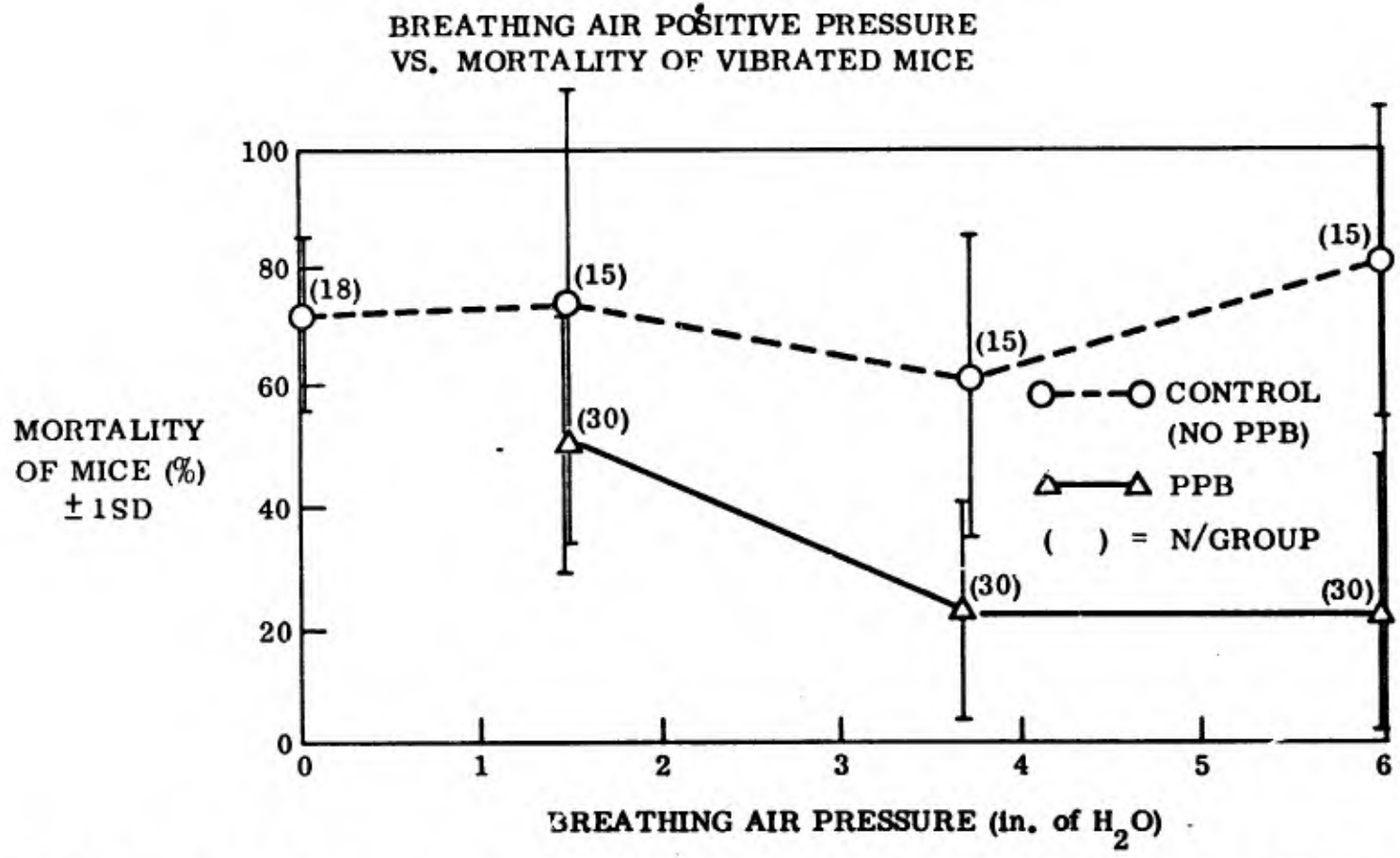


Figure 1

Restraint Apparatus for Vibration of Mice While Applying Positive Pressure Breathing



ACCELERATION = 7.07 g rms FREQUENCY = 20 cps EXPOSURE DURATION = 10 MIN.

Figure 2

Figure 3

Excised Lungs from Simultaneously Vibrated Mice. Left (No PPB) and Right (PPB).

should be studied. Ideally, subsequent to animal studies the anticipated future space vehicle complex dynamic and pressure environment during launch and reentry should be simulated in the laboratory to measure crew stresses and performance responses. Against these baseline values, changes could be measured in stress and performance levels that resulted from change of position, adding external body restraint and changing ambient pressure and/or positive breathing pressure, with the subjects instrumented to enable measurement of alteration of resonant frequencies of the chest-abdomen system.

SUMMARY

The complex launch and reentry environment of future manned spacecraft and the associated crew stresses resulting from exposure to the dynamic loads are analyzed. The analysis provides a strong theoretical basis for predicting significant increases in crew stresses from vibrations in future space operations. The biomechanics of g, vibration, and hypobaric stress summation are identified. One such mechanism is summation in one direction of inertial forces of vibration with those from transverse acceleration resulting in excessive elevation of the heart and diaphragm and altered mechanical coupling between the heart and adjacent body structures. This creates severe tissue stresses within the cardiorespiratory system. Techniques designed to attenuate the independent and combined stresses are presented along with the theoretical grounds for postulating the protection concepts. Recommended techniques include use of positive pressure breathing and increased space cabin environmental pressure. The theory predicts that positive pressure breathing will reduce tissue stresses by maintaining a near normal one-g mid-position of the diaphragm during exposure to the complex stress, will increase the restoring force for a given upward displacement of the vibrating abdominal mass, and will increase the resonant frequency of the chest-abdomen system.

A significant protective effect expected from an increased total cabin pressure also results from increased restoring forces which inhibit excessive invasion of the chest by the vibrating thoracoabdominal mass. Selected levels of cabin and positive breathing pressure are expected to reduce tissue stress by providing a controlled shift of the resonant frequency of the chest-abdomen system away from maximum biological stress frequencies transmitted by vehicle structures. A rigid chest abdomen restraint designed as a removable extension

to the existing Mercury-type contoured couch is suggested for use with combined internal tissue restraint provided by pressure breathing. Animal experiments were conducted to demonstrate the theorized cardiorespiratory tissue protection provided by pressure breathing during exposure to vibration stress. The results substantiate the theory.

Results are presented of other experiments which have likewise demonstrated the protective effect of pressure breathing and increased cabin pressure when provided to subjects exposed to linear g and to vibration. Additional complex stress experiments required to fully validate the effectivity and practicality of the proposed attenuation techniques are described.

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