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The Physiological Basis for Spacecraft Environmental Limits

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The Physiological Basis for Spacecraft Environmental Limits

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This document is intended to provide a description of the physiological effects that determine the environmental limits required in spacecraft. The existing limits for operational environments are described in terms of acceptable physiological changes. Tolerance limits are discussed for exposures to environmental factors during unusual or contingency situations. Where environmental limits may be required but do not presently exist or where additional research is required to refine existing limits, these research needs are specified.

Background information describing physiological systems is presented as required to support the development of physiological limits. References to general works in the physiological area of interest are included for the interested reader. The historical development of physiological limits used in the U.S. manned-space-flight program is also cited as necessary to show the development of the limits currently in use.

This document is intended primarily for use by those responsible for the operational safety and health of space crews. However, the information presented is also of interest to spacecraft design engineers, to those in other fields who may deal with similar environmental factors, and to those interested in the total scope of physiological effects which may be experienced during space flight.

The chapters were written by authors from the NASA Lyndon B. Johnson Space Center, Houston, Texas. The chapters were reviewed by a committee of contributing authors chaired by E. L. Michel, Program Scientist, Life Sciences Experiments Payloads Office. The members of the committee were as follows.

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I. ATMOSPHERE

By D. J. Horrigan

INTRODUCTION

Consideration of the cabin atmosphere for a spacecraft or a space station must be based on biomedical, operational, and engineering requirements. Generally, an atmosphere identical to man's sea-level environment is considered desirable. However, at certain times and/or in selected compartments, an alternative pressure or gas composition may be necessary or desirable. In this chapter, the physiological basis of the limits established for atmospheric pressure as well as the partial pressures of oxygen, carbon dioxide, water vapor, and diluent gas will be reviewed.

BAROMETRIC PRESSURE

Physiological Effects

A wide range of cabin pressures is acceptable physiologically. The limiting effects of static pressure are associated with inadequate partial pressure of oxygen PO_2 in the lungs on the low end of the scale and nitrogen narcosis and/or oxygen toxicity on the upper end of the scale. These conditions would be expected to occur before any effects from the barometric pressure itself. However, animal decompression studies conducted to near-vacuum pressures indicate that death would be imminent unless recompression to a higher pressure - at least 26.66 kN/m^2 (200 torr) - occurred within 60 to 90 seconds (refs. I-1 and I-2). Exposure of the human body to pressures below the vapor pressure of body fluids (6.27 kN/m^2 (47 torr)) results in ebullism, the formation of vapor bubbles in tissues, blood vessels, and body cavities (ref. I-3). Severe hypoxia and lung pathology are present in the ebullism syndrome.

A hyperbaric environment has not been considered for occupants of a spacecraft and, therefore, will not be considered in this chapter. However, in a large space station, it is possible that inclusion of a hyperbaric treatment facility may be required.

The effects of changing pressure can be divided into those which occur during compression and those which occur during decompression. The problems occurring during increasing pressure are associated with body cavities which contain air, such as the paranasal sinuses, ears, and lungs. An "ear block"

or a "sinus block" occurs during compression if the pressure within the cavity concerned cannot be equalized with the outside pressure. Upper respiratory infections tend to make the occurrence of this condition more likely because of swollen mucous membranes. Barodontalgia can occur during compression if gas pockets form below a tooth restoration or in the decayed tooth substance. During the repressurization after extravehicular activity has been completed, the increase in ambient pressure may exceed the rate of pressure increase in the helmet of the space suit. This condition could result in a tendency to squeeze the head area because of the increased pressure below the neck in relation to the pressure in the helmet. The effects of a pressure difference between the surrounding atmospheric pressure and the total gas pressure in the tissues and cavities of the body is termed "dysbarism." This term includes any clinical syndrome resulting from such a pressure difference. The term "decompression sickness" is more specific in that it refers to the syndrome resulting from bubble formation in the tissues. The terms "bends" and "aeroembolism" are also used to describe such a condition. In this chapter, "decompression sickness" is used to denote these effects.

The symptoms of decompression sickness depend on the area of the body involved. Joint pain is a frequent occurrence. Skin involvement may occur with pruritus and a rash with later development of more serious symptoms. The more serious types of decompression sickness involve coughing and chest pain as well as disorders of the cardiovascular system, including vasomotor collapse, and serious disturbances of the central nervous system (ref. I-4).

Decompression occurs during or after ascent in both diving and flying. A more rapid, even "explosive," decompression can occur during a loss of pressurization in an aircraft or a spacecraft. Normally, the ears are not affected during decompression because the anatomical structure of the Eustachian tubes is such that air can escape from the middle ear cavities as it begins to expand. Sinus pain and toothache are more likely to occur than ear pain on ascent when trapped air begins to exert pressure. Pain in the abdominal area caused by gas expansion in the gastrointestinal tract may occur during decompression, especially if a gas-producing food or beverage is ingested before the drop in pressure. Lung damage could occur during a very rapid decompression only if the glottis is closed or if the decompression exceeds the rate capacity of the respiratory tract to exhale air. Lung damage occurs at a pressure difference of approximately 10.67 kN/m^2 (80 torr) between lungs and ambient (ref. I-5).

Decompression sickness is the most serious threat from the loss of cabin pressure or during transition to space-suit pressure. The cause of decompression sickness is thought to be gas bubble formation in body fluids. Inert gas as well as oxygen, carbon dioxide, and water vapor diffuse into evolved bubbles of gas. This process may result in pain, interference with circulation, and impairment of function in the tissues involved. Several factors are correlated with the incidence of decompression sickness. Among them are age, obesity, exercise, and duration of exposure (ref. I-4). The condition is rare in the first 3 to 5 minutes of exposure to reduced pressure, with the maximum number of cases appearing after 20 to 40 minutes.

Physiological Limits

Regarding man's tolerance to extremely low pressures, studies with baboons and dogs suggest that one could survive near-vacuum pressure for a few seconds (ref. I-1). However, the low limit to sustain life for any extended period is 18.62 kN/m^2 (2.7 psia), which is the pressure at an altitude of approximately 12.2 kilometers (40 000 feet), assuming pure oxygen as the breathing atmosphere. This limit is based on the assumption that abdominal gas expansion has not caused an excessive embarrassment of respiration. Spacecraft experience includes a range of 121.59 kN/m^2 (1.2 atmospheres) in the U.S.S.R. Vostok, Voskhod, and Soyuz cabins to 34.4 kN/m^2 (258 torr) in the U.S. space cabin (ref. I-6). Space-suit pressures were 25.33 kN/m^2 (190 torr).

Hypobaric decompression sickness from sea level usually begins only after decompression from 101.32 kN/m^2 (760 torr) to less than 53.33 kN/m^2 (400 torr). During the Apollo-Soyuz missic , the Soyuz spacecraft pressure was maintained at 73.33 kN/m^2 (550 torr) and the Apollo spacecraft at 34.4 kN/m^2 (258 torr) without danger to the crewmen undergoing this decompression repeatedly. The U.S. crewmen had equilibrated to the Apollo spacecraft pressure before the visits to the Soyuz. Previous studies had indicated that repeated compressions and decompressions could be made between these two pressures without symptoms of decompression sickness, as long as the proper denitrogenation had been made before the initial decompression from sea level (ref. I-7). It should be added, however, that some cases of decompression sickness have occurred after a drop in pressure from 101.32 to 50.66 kN/m^2 (760 to 380 torr) (ref. I-8).

The preventive measure for decompression sickness is inert gas washout by breathing pure oxygen for several hours. Although a duration of 3 hours has been used with the astronaut population, a longer time may be required depending on the factors cited previously such as age and body build. A loss of protection has been shown to occur when the washout is interrupted with periods of air breathing. The time required to make up the loss is thought to be greater than the time of the interruption (ref. I-9).

Current research may enable a closer calibration of individuals to ascertain the sufficient time for decompression protection. Another unanswered question which will require in-flight research is the effect of null gravity on the rate of inert gas washout. Respiratory and tissue mass spectrometry are available means to provide quantitative data on washout procedures under one-g and null-gravity conditions.

OXYGEN

Dalton's law states that in a mixture of gases, the total pressure of the mixture is equal to the sum of the partial pressures of the individual gases. The partial pressure of oxygen (PO_2) in the air breathed at sea level is 21.06 kN/m^2 (158 torr). This partial pressure is used as a guideline for establishing oxygen requirements in manmade environments. It should be noted

that in the upper respiratory tract, the addition of water vapor causes the inspired PO_2 to drop to 19.86 kN/m^2 (149 torr). This value is derived as follows: (760 - 47) (1.33 x 10^2) (0.2093), which is the barometric pressure in torr less respiratory water-vapor pressure in torr multiplied by the factor for conversion to newtons per square meter and by the oxygen fraction in air (ref. I-10). Thus, even in a loss of total pressure, the partial pressure of oxygen can be maintained at 21.06 kN/m^2 (158 torr) by increasing the O_2 percentage of the gas mixture. In the Apollo Program, a mixture of nearly 100 percent oxygen was used at a cabin pressure of 34.47 kN/m^2 (5 psia).

The maintenance of the inspired PO_2 at 19.86 kN/m^2 (149 torr) provides an alveolar PO_2 (P_AO_2) of approximately 14 kN/m^2 (105 torr) because of dilution by carbon dioxide (CO_2). This pressure permits arterial blood to become saturated with oxygen to 98 percent of its cap city. Most of the oxygen in the blood is carried by hemoglobin. Each 100 milliliters of blood contains about 15 grams of hemoglobin and each gram combines with 1.36 milliliters of oxygen (ref. I-10). This combination results in about 20 milliliters of oxygen for each 100 milliliters of blood. A relatively small amount of oxygen is carried in solution (0.3 milliliter O_2/100 milliliters of blood).

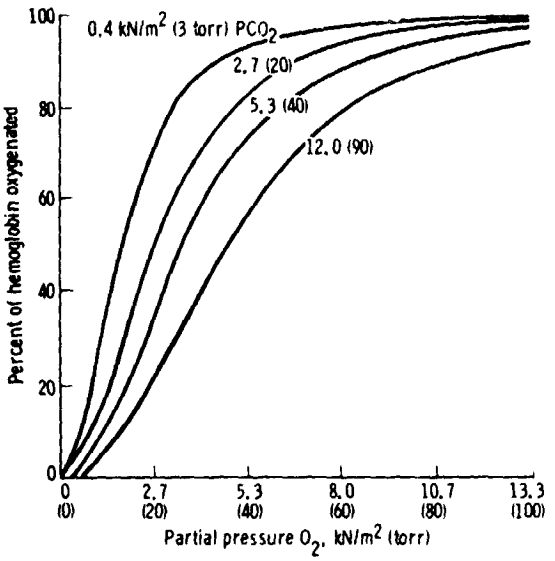
A number of factors which affect the oxygen-carrying capacity of the blood can be understood by referring to the oxygen-hemoglobin dissociation curve as shown in figure I-1. The normal curve is at a pH of 7.4, a temperature of 310 K (37° C), and a CO_2 partial pressure (PCO_2) of 5.33 kN/m^2 (40 torr). The basic sigmoid shape of the curve is maintained as these values change, but the tendency to unload oxygen varies. The curve shifts to the right (i.e., greater dissociation) with an increase in temperature or PCO_2 or with a decrease in pH. The opposite changes would result in the opposite effect (i.e., shift to the left). An increasing metabolic demand, such as from exercise, would produce increases in lactic acid, CO_2, and heat, all of which would tend to unload oxygen to the tissues - a desirable effect. The biochemical mechanism of anaerobic metabolism with the building up of an "oxygen debt" enables maximum exercise levels anaerobically with the oxygen being "paid back" by a later intake.

Physiological Effects

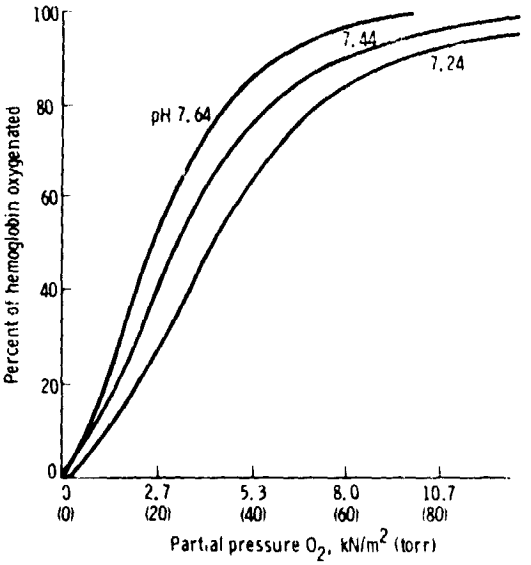
The physiological effects can be those of low PO_2 (hypoxia) or high PO_2 (oxygen toxicity).

Hypoxia.- The earliest symptoms of low PO_2 frequently involve a decrease in night vision and may occur as P_AO_2 is lowered to approximately 10.53 kN/m^2 (79 torr) as it would be at an altitude of 1.5 kilometers (5000 feet). A 3.0-kilometer (10 000 foot) altitude has been used in military aviation as the altitude above which supplemental oxygen is required during daylight flights.

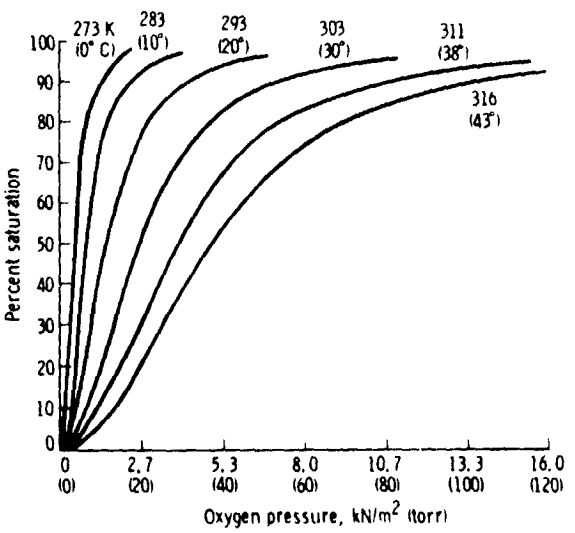
As the partial pressure of oxygen in the inspired air continues to drop acutely, the signs and symptoms of hypoxia include loss of peripheral vision, skin sensations (numbness, tingling, or hot and cold effects), cyanosis, euphoria, and unconsciousness. The euphoria may cause the affected person to



(a) Effect of CO_2 on oxygen dissociation curve of whole blood (ref. I-11).



(b) Effect of acidity on oxygen dissociation curve of blood (ref. I-12).



(c) Effect of temperature on oxygen dissociation curve of blood (ref. I-13).

Figure I-1.- Oxygen-hemoglobin dissociation curves.

be a poor judge of the seriousness of the situation. Table I-1 contains the times of useful consciousness at selected altitudes (ref. I-14).

Chronic exposure to low PO_2 results in hyperventilation such that the arterial PCO_2 could drop to as low as 4.4 kN/m^2 (33 torr). This reduction tends to raise the alveolar PO_2. Another compensatory mechanism is an increase in the red blood cell concentration. Such long-term adjustments to high altitude are not relevant to the transients that can occur in a spacecraft cabin. However, a required diminution of oxygen partial pressure over the duration of a long flight might indeed elicit such compensatory mechanisms.

Oxygen toxicity.- Oxygen in high concentrations can be toxic. Subjects who breathe 100-percent oxygen at sea level for 6 to 24 hours complain of substernal distress and show a diminution of vital capacity of 500 to 800 milliliters (ref. I-10). This loss is probably due to atelectasis, which occurs when the sum of the partial pressures of gas in the alveoli is greater than in venous blood and gas diffuses into the blood to cause collapse of the alveoli. Surface-tension effects tend to prevent reopening of an atelectatic area. Astronauts who breathed pure oxygen at space-suit pressure (26.2 kN/m^2 (3.8 psia)) and Apollo spacecraft cabin pressure (34.5 kN/m^2 (5 psia)) did not experience signs or symptoms of oxygen toxicity.

Physiological Limits

Table I-2 contains the acceptable total and oxygen partial pressures for spacecraft cabin application. The limits established in this table are based on the maintenance of P_AO_2 between 12.67 and 15.33 kN/m^2 (95 and 115 torr). This range was derived from using 14.0 kN/m^2 (105 torr) as an ideal P_AO_2 and allowing $\pm$1.333 kN/m^2 (10 torr) as control limits for the life-support system. To maintain such a P_AO_2 at selected total pressures, the following equation was used to calculate the appropriate partial pressure of oxygen in the breathing atmosphere.

$$P_AO_2 = F_iO_2(P_B - 47) - PCO_2 \times \left[F_iO_2 + \frac{1 - F_iO_2}{0.85}\right]$$

where P_AO_2 = alveolar partial pressure of oxygen

F_iO_2 = oxygen fraction in breathing atmosphere

P_B = barometric pressure of the breathing mixture

0.85 = an assumed respiratory exchange ratio

PCO_2 = partial pressure of CO_2

TABLE I-1.- TIME OF USEFUL CONSCIOUSNESS AFTER ACUTE EXPOSURE TO REDUCED OXYGEN LEVELS

Altitude, km (ft)	Time of useful consciousness, sec	
	Moderate activity	Sitting quietly
6.7 (22 000)	300	600
7.6 (25 000)	120	180
8.5 (28 000)	60	90
9.1 (30 000)	45	75
10.7 (35 000)	30	45
12.2 (40 000)	18	30
19.8 (65 000)	12	12

TABLE I-2.- PHYSIOLOGICALLY ACCEPTABLE TOTAL PRESSURE/OXYGEN PARTIAL PRESSURES[a]

Total pressure, kN/m^2 (psia)	Sea-level equivalent		Acceptable transient limit[b]		Continger limit[c]	
	$\%O_2$	PO_2, kN/m^2 (psia)	$\%O_2$	PO_2, kN/m^2 (psia)	$\%O_2$	PO_2, (p
103.4 (15)	19.3 to 22.0	20.0 to 22.8 (2.90 to 3.30)	17.5	18.099 (2.625)	16.17	16.75
101.4 (14.7)	19.7 to 22.5	20.0 to 22.8 (2.90 to 3.31)	17.9	18.140 (2.631)	16.53	16.75
96.5 (14)	20.8 to 23.7	20.1 to 22.9 (2.91 to 3.32)	18.8	18.147 (2.632)	17.4	16.82
89.6 (13)	22.5 to 25.6	20.2 to 23.0 (2.93 to 3.33)	20.4	18.27 (2.65)	18.8	16.82
82.7 (12)	24.5 to 27.9	20.3 to 23.1 (2.94 to 3.35)	22.2	18.34 (2.66)	20.5	16.96
75.8 (11)	26.9 to 30.6	20.4 to 23.2 (2.96 to 3.37)	24.4	18.48 (2.68)	22.5	17.10
68.9 (10)	29.8 to 34.0	20.5 to 23.4 (2.98 to 3.40)	27.0	18.62 (2.70)	25.0	17.24
[d]62.1 (9)	33.4 to 38.1	20.8 to 23.6 (3.01 to 3.43)	30.3	18.75 (2.72)	28.0	17.37
[d]55.2 (8)	38.0 to 43.4	21.0 to 23.9 (3.04 to 3.47)	34.4	18.96 (2.75)	31.8	17.51

[a]The atmosphere should be an oxygen-nitrogen mixture with an alveolar O_2 partial pressure bet 12.67 and 15.33 kN/m^2 (95 and 115 torr).

[b]1.2-kilometer (4000 foot) equivalent altitude.

[c]1.8-kilometer (6000 foot) equivalent altitude.

[d]The acceptability of total pressures below 68.9 kN/m^2 (10.0 psia) would require extended fli verification for flight durations beyond 6 months.

In table I-2, 1.2 kilometers (4000 feet) was selected as an equivalent altitude for an acceptable transient change in cabin oxygen. A 1.8-kilometer (6000 foot) equivalent is considered as a contingency situation where some remedial action would be necessary, but is not considered an emergency situation.

CARBON DIOXIDE

Man is the major source of the CO_2 in the space cabin. At rest, each crewman would produce about 400 liters of CO_2 per day. During work, production would be much higher. There is no minimum PCO_2 required to sustain normal physiological functions. However, until it is feasible to scrub all of the CO_2 from a cabin atmosphere, guidelines are necessary for short-term and long-term tolerance to increased levels of this gas.

Physiological Effects

The effects of increased carbon dioxide (hypercapnia) in the atmosphere depends on the concentration and duration of exposure. Increases in heart rate, respiratory rate, and minute volume occur as acute reactions to increased PCO_2 (fig. I-2). This powerful stimulatory effect is caused by chemoreceptor and sympathetic nervous system stimulation. Most of the carbon dioxide in the blood is in the form of the bicarbonate ion. As the level of carbon dioxide increases, the acid-alkaline balance is disturbed and acidosis results. Altered P_AO_2 and P_ACO_2 can combine to affect performance. Figure I-3 shows that a lowering of the alveolar PO_2 will tend to lower the concentration of P_ACO_2 at which CO_2 narcosis occurs.

In addition to the physiological effects described, there is some recent evidence that CO_2 may be involved in the initiation of bubble formation during the onset of decompression sickness (ref. I-15). This condition is thought to occur because of the high solubility and rate of diffusion of CO_2 (20 times that of O_2). If correct, this theory adds to the importance of CO_2 control in the space cabin.

Physiological Limits

Based on the data available, the CO_2 limit for space cabins has been set at 1.013 kN/m^2 (7.6 torr). The same limit has been established for inspired gas in the space suit (up to a metabolic rate of 469 watts (1600 Btu/hr)). Additional research is needed to explore the relationship of selected work rates to CO_2 toxicity and to define chronic effects of low CO_2 levels. However, the CO_2 lower limit (at which there is probably no significant physiological, psychological, or adaptive change) is considered to be about 0.5 percent. The 0.5-percent equivalent pressure of 0.5066 kN/m^2 (3.8 torr) is the most prudent for use during long flights. Acceptable transients and duration of exposure are as follows.

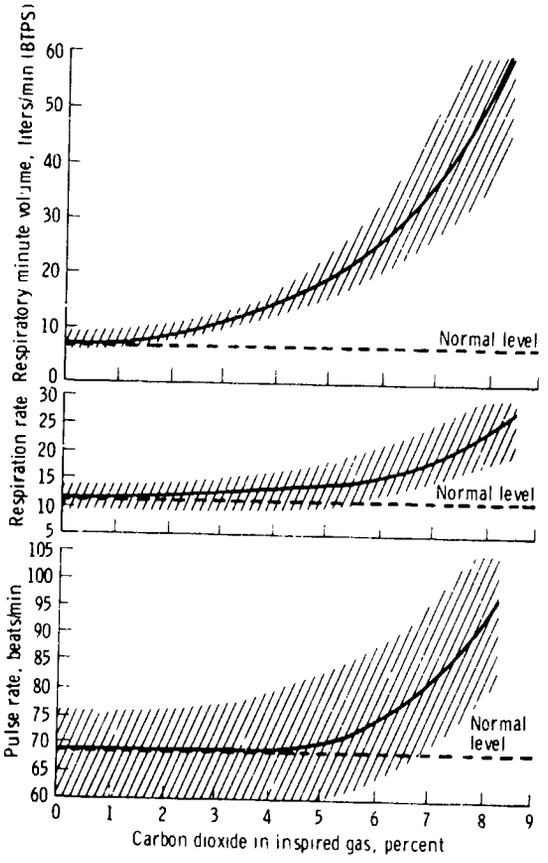


Figure I-2.- Immediate effects of increased CO_2 on pulse rate, respiration rate, and respiratory minute volume (BTPS = body temperature and pressure, saturated with water) for subjects at rest. Hatched areas represent one standard deviation on each side of the mean. To convert percentage CO_2 to partial pressure, multiply percent value by 1.013 for kilonewtons per square meter or by 7.6 for torr.

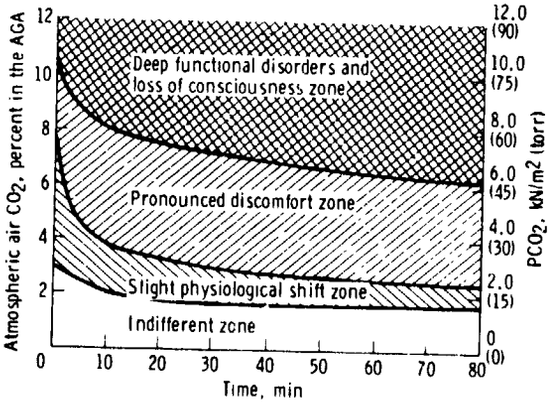


Figure I-3.- Classification of CO_2 toxic action effects in relation to PCO_2 value in artificial gas atmosphere (AGA) (from ref. I-6).

CO_2 limits, kN/m^2 (torr)	Recommended action
0.5066 (3.8)	Nominal, indefinite exposure
1.013 (7.6)	Nominal, for mission of limited duration
1.013 to 2.0 (7.6 to 15)	Restriction on activity, possible mission abort if correction not feasible
2.0 (15)	If correction unsuccessful, immediate action to abort mission

HUMIDITY ENVIRONMENT

Physiological Effects

The partial pressure of water vapor PH_2O affects insensible water loss, respiratory water loss, weight loss, and other comfort and health states related to the eyes, the nose, the throat, and the skin. Excessively high PH_2O tends to prevent adequate evaporation and causes accumulation of water on skin and clothing. Excessively low humidity causes drying of the mucous membranes of the nose, the mouth, and the pharynx as well as chapping of the lips and drying of the eyes (fig. I-4). An increased incidence of upper respiratory infections would be expected with low PH_2O. The suspected reason is that the dryness of the respiratory tract would permit greater bacterial growth.

Physiological Limits

A range of 40 to 70 percent relative humidity was used in past space missions and was generally satisfactory from a comfort and health standpoint. These humidities are equivalent to PH_2O values of 1.0 to 1.733 kN/m^2 (7.5 to 13 torr) at a temperature of 294 K (70° F) and 1.4 to 2.47 kN/m^2 (10.5 to 18.5 torr) at 300 K (80° F). A water-vapor pressure of 1.33 kN/m^2 (10 torr) is considered optimal for habitability. The minimum acceptable value for spacecraft cabins is 0.80 kN/m^2 (6 torr) at 101.4 kN/m^2 (14.7 psia) or 1.067 kN/m^2 (8 torr) at 34.5 kN/m^2 (5 psia), since the drying effect is greater at the low pressure.

DILUENT GAS

To maintain a sea-level atmosphere in a spacecraft cabin, the oxygen must be diluted by an inert gas. This diluent gas must be present because of the flammability problem of substances in a pure oxygen atmosphere and, as

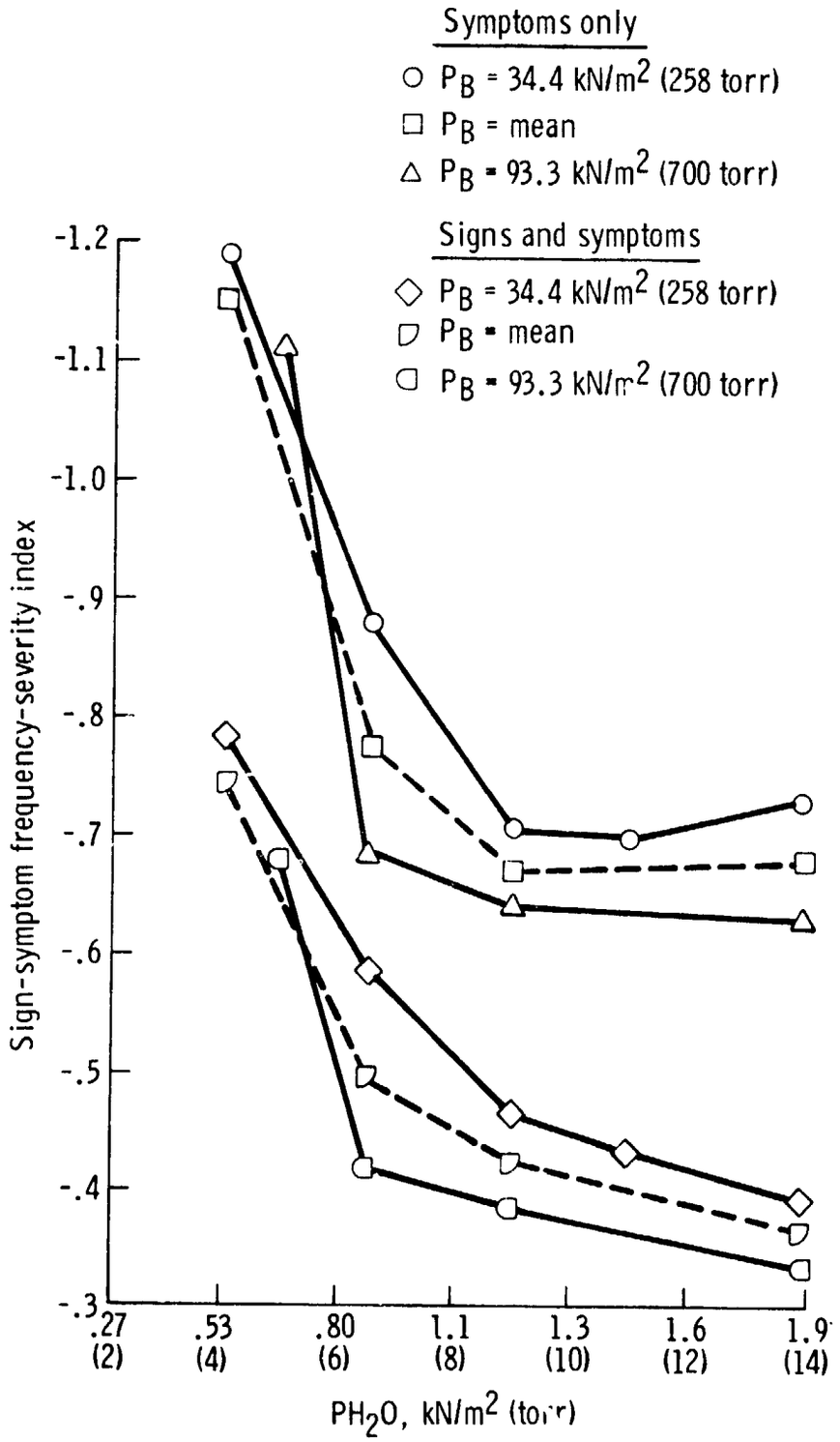


Figure I-4.- Sign and symptom development in relation to PH_2O for 24-hour experiments (from ref. I-16).

described previously, because pure oxygen is toxic at sea-level pressures when breathed for prolonged periods.

Since space crews will be breathing inert gas, their tissue inert gas tensions will be in equilibrium with the partial pressure of the diluent gas in the atmosphere. As described previously, this condition requires a washout procedure before decompression.

Nitrogen is normally used as the diluent gas. There is no evidence at the present time that the use of any other diluent gas would be to advantage.

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II. CONTAMINANTS

By H. L. Kaplan*

DEFINITION AND DESCRIPTION OF CONTAMINANTS IN THE SPACECRAFT ENVIRONMENT

Introduction

The quality of the breathing atmosphere can be a significant factor in determining an individual's physiological and psychological comfort and well-being as well as his performance efficiency. The recent increased awareness of this relationship has led to the establishment of public air quality standards and more stringent control of the contamination of air by industry. Similarly, in the NASA space program, major emphasis has been placed on providing an environment in which the astronaut can perform in comfort and safety with maximum efficiency. The achievement of this objective in manned spacecraft is complicated by the continuous generation of small quantities of contaminants from offgassing of materials and from man, himself, into the closed environment of the spacecraft cabin.

Description of Spacecraft Contaminants

A wide variety of chemical substances has been detected in the spacecraft atmosphere. These contaminants range from gases such as methane, ethane, and ethylene to vapors of high-molecular-weight polynuclear and heterocyclic hydrocarbons, which are solids at room temperature. Approximately 150 contaminants were identified by postflight desorption of the charcoal from the contaminant-removal systems of the Apollo 7 to 17 spacecraft and subsequent gas chromatographic/mass spectrometric (GC-MS) analyses of the desorbate (ref. II-1). These compounds and the spacecraft in which each was detected are presented in table II-1. More recently, organic volatiles in the Skylab 4 cabin atmosphere were collected and concentrated on a synthetic adsorbent, Tenax GC, by drawing the cabin air through tubes of the material for approximately 15-hour sampling periods on 3 days, the 11th, 47th, and 77th, of the mission (refs. II-2 and II-3). Postflight desorption of the Tenax and GC and GC-MS analyses of the desorbates enabled the detection of more than 300 compounds and identification of 107 of these. Approximately 80 of these compounds and their concentrations in the spacecraft cabin on the 3 sampling days are shown in table II-2. However, the concentrations of some compounds, such as the halogenated hydrocarbons, are not exact since compounds with a volatility greater than that of benzene are not quantitatively

*Southwest Foundation for Research and Education, San Antonio, Texas.

TABLE II-1.- APOLLO SPACECRAFT CONTAMINANTS

Contaminant name	Apollo flights in which detected										
	7	8	9	10	11	12	13	14	15	16	17
Butyl alcohol	x	x	x	x	x	x	x	x	x	x	
Capryl alcohol					x	x	x	x	x		
Ethyl alcohol	x	x	x	x	x	x	x	x	x	x	x
Isoamyl alcohol					x	x	x	x	x	x	
Isobutyl alcohol	x	x	x	x	x	x	x	x	x	x	x
Isopropyl alcohol	x	x	x	x	x	x	x	x	x	x	x
Methyl alcohol	x	x	x	x	x	x	x	x	x	x	x
Propyl alcohol	x				x	x			x	x	x
Sec-butyl alcohol	x	x	x	x			x				
Tert-butyl alcohol	x	x	x			x					
Acetaldehyde	x	x	x	x	x	x	x	x	x	x	x
Butyraldehyde	x										
N-butane					x	x	x	x	x	x	x
Cyclohexane	x	x	x	x	x	x				x	x
Cyclopentane	x		x		x	x		x	x	x	
Ethane					x	x	x		x	x	x
Heptane	x	x	x	x		x					
Hexane	x	x	x	x	x	x	x		x	x	
Isobutane					x	x			x	x	
Isopentane	x	x	x	x							x
Methylcyclohexane	x	x	x	x	x	x	x	x	x	x	
Methylcyclopentane	x		x	x		x	x	x	x	x	x
N-octane	x										
Pentane	x	x	x	x	x	x					
Propane					x	x			x	x	x
Trimethyl butane	x			x							
Trimethyl hexane	x		x		x		x			x	
Allene						x	x				
Benzene	x	x	x	x	x	x	x	x	x	x	x
1,3-butadiene						x	x				
1-butene					x	x	x	x	x	x	x
2-butene (cis)					x	x			x	x	x
2-butene (trans)					x	x			x	x	x
Cyclohexane		x				x	x		x	x	
Cyclopentane		x		x							
Ethyl benzene		x	x	x	x	x	x	x	x	x	x
Ethylene	x	x		x	x	x	x	x	x	x	x
2-hexene			x	x	x	x	x	x	x	x	x
Indene	x	x			x	x	x	x	x	x	x
Isoprene				x	x	x	x	x	x	x	x
Mesitylene	x		x		x	x	x	x	x	x	x
Methyl acetylene					x	x	x		x	x	
1-pentene	x		x	x					x		
2-pentene						x	x		x	x	
Dichloroethane					x				x	x	x
Dichloroethylene					x	x					
Dichlorofluoromethane					x	x					
Difluoroethylene							x		x		x
Ethyl chloride					x	x				x	x

TABLE II-1.- Continued

Contaminant name	Apollo flights in which detected										
	7	8	9	10	11	12	13	14	15	16	17
Ethylene dichloride	x										
Ethyl fluoride						x	x	x	x	x	x
Fluoroethane					x		x		x	x	
Fluoropropane						x	x		x	x	
Freon 11	x	x	x	x	x		x		x	x	
Freon 12					x	x	x	x		x	x
Freon 22					x	x	x	x		x	
Freon 113	x	x	x	x	x	x		x	x	x	x
Freon 114					x						
Methyl chloride						x	x		x	x	x
Methyl chloroform	x	x	x	x	x	x					x
Methylene chloride	x	x	x	x	x	x		x	x	x	x
Monochloroacetylene					x						
Pentafluoroethane					x	x	x		x	x	x
Tetrachloroethane		x		x		x		x			
Tetrachloroethylene	x	x	x	x	x	x				x	x
Tetrafluoroethylene					x	x	x		x	x	x
Trichloroethylene	x	x	x	x	x	x		x	x	x	x
Trifluorochloroethylene		x						x	x	x	
Tetrahydrofuran									x	x	
Methyl furan										x	
Freon 21									x	x	
Hexafluoroethane										x	
Trifluoroethylene					x	x	x	x	x	x	x
Trifluoromethane					x	x		x	x	x	
Trifluoropropane						x	x		x	x	
Trifluoropropene					x		x	x	x		
Vinyl chloride						x	x	x	x	x	x
Vinylidene chloride				x	x	x	x		x	x	x
Dimethyldifluorosilane						x	x	x		x	
Trimethylfluorosilane					x	x	x	x	x	x	x
Diethyldisulfide					x						
Dimethyldisulfide				x					x	x	
Dimethylsulfide			x	x	x	x		x	x	x	x
Vinyl fluoride						x	x	x	x	x	
1,1,1-trichloroethane							x	x	x	x	x
Tetrafluorochloroethane							x				
Chlorodifluoroethylene							x	x	x		
Naphthalene							x		x		
Pentyl alcohol							x	x			
Cellosolve acetate							x				
Decahydronaphthalene							x	x			
Propylene	x	x	x	x	x	x	x	x	x	x	x
Styrene	x	x	x	x	x	x	x	x	x		
Toluene	x	x	x	x	x	x	x	x	x	x	x
Trimethyl benzene					x				x		
M-xylene	x	x	x	x	x	x	x	x	x	x	x
O-xylene	x	x	x	x	x	x	x	x	x	x	
P-xylene	x	x				x	x	x	x	x	

TABLE II-1.- Concluded

Contaminant name	Apollo flights in which detected										
	7	8	9	10	11	12	13	14	15	16	17
N-propyl benzene									x	x	
Ethyl acetylene									x		
Trimethyl benzene									x		
2-methyl pentane										x	x
Dimethyl butane										x	x
3-methyl pentane											x
Acetylene					x	x	x		x	x	x
Octyne						x	x	x	x		
Diisopropylamine				x							
Butyl acetate	x	x	x	x	x				x		
Butyl lactate						x		x			
Ethyl acetate	x	x	x	x	x	x			x	x	
Ethyl lactate						x	x	x			
Methyl acetate	x	x	x	x	x	x	x				
Propyl acetate	x						x				
Dimethyl ether					x	x			x	x	x
Dioxane			x	x	x	x	x		x	x	x
Furan		x	x	x	x	x	x	x	x	x	x
Sulfur dioxide				x							x
Acetone	x	x	x	x	x	x	x	x	x	x	x
Cyclohexane	x										
Methyl ethyl ketone	x	x	x	x	x	x	x	x	x	x	x
Methyl isobutyl ketone	x	x	x	x	x	x	x	x	x	x	x
2-pentanone	x	x									
Acetonitrile			x	x	x	x		x	x	x	
Methoxy acetic acid				x				x			
Carbon tetrachloride	x							x			
Chloroacetylene						x					
Chlorobenzene		x	x	x	x	x		x	x		
Chlorofluoroethylene						x		x	x	x	x
Chloroform	x	x			x	x				x	x
Chloropropane	x										
Chlorotetrafluoroethane					x	x			x		x
Chlorotrifluoroethylene					x	x			x	x	x
Dichlorobenzene		x	x	x		x					x
Dichlorodifluoroethylene						x		x	x	x	x
Chlorotrifluoromethane								x			
Fluoroform								x			
Trifluoroacetonitride									x		
Octalfluorobutane								x			
Propadiene								x			
Dichlorodifluoroethane								x		x	x
Dimethylcyclohexane								x		x	
Cyclohexyl alcohol									x		
1-hexene									x	x	x
Octafluoropropane									x		
Ethyl fluoride									x		
Hexafluoropropene									x		
Vinylidene fluoride									x		

TABLE II-2.- VOLATILE COMPONENTS IN THE SKYLAB 4 CABIN ATMOSPHERE

Peak no.	Identification	Mol. wt	Measurement for Julian day -					
			331		002		032	
			% of sample	Concent., p/b	% of sample	Concent., p/b	% of sample	Concent., p/b
1	Difluorodichloromethane	120	0.13	40	0.23	60	0.70	190
2	1,1,2-trichloro-1,2,2-trifluoroethane + 1,1,1-trichloro-2,2,2-trifluoroethane	186	20.8	590	33.3	8700	30.6	8400
3	Hexamethyldisiloxane	162	.28	80	.03	7	.35	100
4	Cyclohexane	84	.10	28	.05	13	.15	41
5	N-heptane	100	.34	97	.13	34	.28	77
	+ monofluorodichloromethane	102						
6	Acetone	58	27.8	7900	27.3	7100	10.0	2800
7	C_8 alkane	114	.04	11	.03	8	.08	22
8	C_8 alkane	114	.06	17	.05	13	.10	28
9	Heptene	98	.02	6	.01	3	.03	8
10	Heptene	98	.31	88	.20	52	.42	116
11	N-octane	114	.10	28	.15	39	.10	28
	+ heptene	98						
12	Ethyl acetate	88	1.60	454	1.50	390	.80	221
13	2-butanone	72	5.30	1505	4.70	1222	2.41	665
14	Octamethyltrisiloxane	236	.37	105	.45	117	.50	138
15	2-propanol	60	2.51	713	4.40	1144	3.10	856
	+ tetrachlorodifluoroethane	202						
16	Hexamethylcyclotrisiloxane	222	.22	63	.10	26	--	--
17	Benzene	78	.41	116	.27	70	.14	38
	+ diacetyl	86						
18	C_9 alkane	128	.08	23	.05	13	.07	19
19	4-methyl-pentanone-2	100	7.01	1990	6.52	1625	3.61	996
	+ octamethylcyclotetrasiloxane	296						
20	Trimethylsilanol (tent.)	90	--	--	.36	94	.55	152
21	Dichloroethane	98	1.6	454	.86	224	.77	213
22	Toluene	92	5.91	1678	4.0	1040	6.22	1717
	+ tetrachloroethylene	164						
23	Decamethyltetrasiloxane	310	.15	43	.20	50	.55	152
24	C_{10} alkane	142	.07	20	.18	47	.15	41
25	C_{10} alkane	142	.02	6	.03	8	.03	8
26	N-decane	142	.67	190	.36	94	.46	127
27	4-methyl-4-pentene-2-one	98	.62	176	.36	94	.45	124

TABLE II-2.- Continued

Peak no.	Identification	Mol. wt	Measurement for Julian day -					
			331		002		032	
			% of sample	Concent., p/b	% of sample	Concent., p/b	% of sample	Concent., p/b
28	C_{11} alkane	156	0.07	20	0.09	23	0.10	28
29	Ethyl benzene	106	.41	116	.20	52	.28	77
	+ dodecamethylpentasiloxane	384						
	+ decamethylcyclopentasiloxane	370						
30	P-xylene	106	.46	131	.59	153	.64	177
31	M-xylene	106	.95	272	.63	164	.60	166
32	Decamethylcyclopentasiloxane	370	.24	68	.12	31	.16	44
33	C_{12} alkane	170	.07	20	.02	52	.04	11
34	Silicone compound	--	.02	43	.01	26	.01	3
35	C_{12} alkane	170	.52	148	.26	68	.23	64
36	O-xylene	106	.52	148	.26	68	.23	64
37	C_3 benzene	120	.01	2	.02	5	.02	5
38	Dodecamethylpentasiloxane	384	.06	17	.05	13	.06	17
39	Silicone compound	--	.19	54	.12	31	.22	61
40	Styrene	104	.11	31	.06	16	.07	19
	+ silicone compound	--						
41	C_3 benzene	120	.15	43	.11	29	.13	36
42	C_{12} alkane	170	.08	23	.04	10	.11	30
43	A hydroxyketone (tent.)	--	2.05	582	1.34	348	1.20	331
44	Limonene	136	.96	273	.61	159	.50	138
45	Dodecamethylcyclohexasiloxane	444	.28	80	.47	122	.57	157
46	2-octanone	128	.09	26	.16	42	.29	80
47	C_3 benzene	120	.04	11	.05	13	.05	14
48	Methyl styrene	118	.09	26	.14	10	.02	5
	+ n-dodecane	170						
49	Tetradecamethylhexasiloxane	458	.12	34	.08	21	.12	33
50	C_3 benzene	120	.07	20	.05	13	.08	22
51	C_4 benzene	134	.12	34	.05	13	.18	50
52	C_4 benzene	134	.07	20	.04	10	.06	17
53	Butoxyethanol	118	--	--	--	--	15.6	4305
54	Silicone compound	--	.04	11	.06	16	.08	22
55	C_4 benzene	134	.04	11	.05	13	.05	14
56	C_4 benzene	134	.01	3	.02	5	.04	11
57	Dichlorobenzene	146	.09	26	.05	13	.09	25
58	C_5 benzene	148	.09	26	.05	13	.09	25

TABLE II-2.- Concluded

Peak no.	Identification	Mol. wt	Measurement for Julian day - 331		002		032	
			% of sample	Concent., p/b	% of sample	Concent., p/b	% of sample	Concent., p/b
59	Tetradecamethylcycloheptasiloxane	518	0.63	179	0.38	99	0.27	75
60	C_5 benzene	148	.07	20	.04	10	.07	19
	+ C_{13} alkane	184						
61	C_5 benzene	148	.23	65	.34	88	.30	83
	+ tetradecamethylcyclo-heptasiloxane	518						
62	Silicone compound	--	22	62	.12	31	.27	75
63	Benzaldehyde	106	.40	114	.24	62	.37	102
64	C_4 benzene	134	.06	17	.05	13	.02	5
	+ C_5 benzene	148						
	+ silicone compound	--						
65	C_5 benzene	148	.15	43	.12	31	.17	47
	+ C_{11} alkene	154						
	+ silicone compound	--						
66	C_4 benzene	134	.09	26	.15	39	.04	11
	+ C_{12} alkene	168						
67	C_2 styrene	132	.16	45	.13	34	.27	75
	+ C_4 benzene	134						
68	Benzonitrile	103	.16	45	.15	39	.15	41
69	C_5 benzene	148	.16	45	.09	23	.17	47
70	C_5 benzene	148	.09	26	.04	10	.09	25
	+ C_{14} alkane	198						
71	C_5 benzene	148	.11	31	.08	21	.10	27
72	Hexadecamethylcyclooctasiloxane	592	.6	170	.05	13	.10	27
73	Acetophenone	120	.21	60	.08	21	.28	77
74	Silicone compound	--	.20	57	.15	39	.16	44
75	Dimethyldihydroindane (tent.)	146	.04	11	.03	8	.05	14
76	C_{15} alkane	212	.08	23	.09	23	.13	35
77	C_5 benzene	148	.04	11	.03	8	.07	19
78	Naphthalene	128	.32	91	.23	60	.41	113
79	Methylnaphthalene	142	.17	48	.11	29	.12	33
80	Methylnaphthalene	142	.01	3	.01	3	.01	3
81	C_{17} alkane	240	.16	45	.13	34	.17	47

retained by Tenax. In table II-3, the data are summarized by categorizing the compounds into four main groups and listing only those contaminants with the greatest concentrations. The contaminants shown in this table comprise more than 95 percent of the total quantities of volatile organics measured on the 3 sampling days. Using the same sampling and analytical techniques, these investigators have sampled air from several urban and industrial environments and compared these samples with the spacecraft atmosphere (ref. II-3). In contrast to urban and industrial air samples in which alkane and substituted aromatics always make up approximately 95 percent of the total organic volatiles, the spacecraft atmosphere contained only a small fraction of these compounds. Also unique to the cabin atmosphere were the large amounts of compounds typical of human metabolism, such as ketones and alcohols, and of halogenated hydrocarbons, and the presence, although in relatively small quantities, of various silicone compounds. Another significant finding in this study was that the concentrations of total volatiles in the cabin atmosphere on the 3 sampling days (i.e., 28.4, 26.0, and 27.6 p/m) were comparable, an indication that an equilibrium has been reached between the generation of contaminants from man and materials and the removal of contaminants by the environmental control life support system (ECLSS) and any cabin leakage.

Sources of Spacecraft Contaminants

The presence of several hundred contaminants in the spacecraft atmosphere results from several sources, none of which can be completely eliminated. Offgassing of cabin materials, components, and equipment, and metabolic waste products of crewmembers are major sources of spacecraft atmospheric contaminants. Minor sources include thermal decomposition of electrical equipment, plastics, hydraulic fluids, oils, and fire extinguishants; atmospheric gas supply contaminants; leakage from environmental or flight control systems; volatile food components; volatile components of personal hygiene articles; and reaction products of contaminant-removal agents.

Offgassing from cabin construction materials such as plastics, insulation, adhesives, and paints and from components and equipment gives rise to a wide variety of chemical compounds of relatively high vapor pressure. The offgassing products of 70 materials used in the Space Shuttle Orbiter OV-101 are shown in table II-4. A projected cabin concentration of each contaminant, also shown in this table, was determined from offgassing data, the total quantity of material in the cabin, and the cabin volume.

The principal sources of contaminants from the metabolic waste products of man are his expired air, perspiration, urine, feces, and flatus. A comprehensive listing of possible trace contaminants derived from man and their sources was compiled by Roth et al. (ref. II-4).

Minor sources contribute smaller quantities of contaminants to the cabin atmosphere than the major sources but do not, necessarily, generate less toxic compounds. In fact, the conversion of contaminants by reaction with contaminant-removal agents has resulted in some highly toxic products. Two such reaction products, monochloroacetylene and dichloroacetylene, were identified in a manned life support test (MESA 1) (ref. II-5). These highly

TABLE II-3.- VOLATILE ORGANIC COMPONENTS IN THE SKYLAB 4 CABIN ATMOSPHERE

Component	Concentration, p/b, for mission day -		
	11	47	77
Alkanes, alkyl-substituted aromatics (10% to 15%)			
C_7 to C_{17} alkanes, alkenes	844	838	990
Toluene	1678	1040	1717
O-, M-, P-xylenes	551	385	407
Naphthalenes	142	92	149
Benzene derivatives	1414	809	1031
Ketones, alcohols (40% to 45%)			
Acetone	7900	7100	2800
Methyl ethyl ketone	1505	1222	665
Methyl isobutyl ketone	2166	1719	1120
Propanol	713	1144	856
Fluoro-chlorohydrocarbons (30% to 35%)			
Freon 112/113	5900	8700	8400
Dichloroethane	454	224	213
Silicone compounds (3%)			
Siloxanes	657	452	583

TABLE II-4.- OFFGASSING PRODUCTS OF ORBITER OV-101 CABIN MATERIALS

Component trace gas	Total trace-gas amount, mg/m^3
Acetaldehyde	6.5952
Acetone	177.4420
Aliphatic sat. and unsat. hydrocarbons	.0037
Ammonia	14.4844
Benzene	2.6261
Butane	.0004
Butene	.1784
Butyl alcohol	.0200
Butyraldehyde	3.3034
Carbon monoxide	84.2279
Cyclohexanone	1141.1374
C_{10} to C_{12} aliphatic sat. and unsat. hydrocarbons	.0006
C_{10} to C_{12} sat. and unsat. hydrocarbons	.6222
C_{10} to C_{14} aliphatic sat. and unsat. hydrocarbons	.4266
C_{10} unsat. hydrocarbon	.0029
C_{11} unsat. hydrocarbon	.0020
C_{12} unsat. hydrocarbon	.0040
C_5 alcohol	.1827
C_5 aldehyde	.0015
C_6 aliphatic sat. hydrocarbon	.0008
C_6 sat. hydrocarbon	.0001
C_6 unsat. alcohol	.0024
C_7 aliphatic sat. and unsat. hydrocarbons	.0008
C_7 aliphatic unsat. hydrocarbon	.0019
C_7 unsat. hydrocarbon	.2918
C_8 aliphatic sat. hydrocarbon	.0007
C_8 aliphatic unsat. hydrocarbon	.0001
C_8 sat. hydrocarbon	.0001
C_8 unsat. hydrocarbon	.1997
C_9 to C_{12} aliphatic sat. and unsat. hydrocarbons	.0135
Dichloroethylene	.0000
Dichloromethane	.0173
Ethyl acetate	.0006
Ethyl alcohol	.5649
Freon 113	4.4570
Furan	.0001
Isopropyl alcohol	9.1947
Mesityl oxide	367.0741
Methyl ethyl benzene	20.3722
Methyl alcohol	5.9469
Methyl ethyl benzene	.0002

TABLE II-4.- Concluded

Component trace gas	Total trace-gas amount, mg/m^3
Methyl ethyl ketone	253.2882
Methyl isobutyl ketone	.0233
N-butyl alcohol	.0224
N-propyl acetate	.1218
N-propyl alcohol	.0098
Pentane	.0023
Pentane (equivalent)	1.7801
Phorone	407.4446
Propane	.0025
Propionaldehyde	1.6002
Propyl amine	.0558
Propylene	.6300
Pyrrole	.3260
Sec-butyl alcohol	.0090
Siloxane dimer	.3758
Siloxane tetramer	.0082
Siloxane trimer	.2131
Styrene	.0000
T. F. Freon	.7586
Tetrachloroethylene	.0001
Toluene	35.4041
Trichloroethane	.0109
Trimethyl benzene	.0356
Xylene	245.7656
1,1,1-trichloroethane	.0021
1,2-diethoxy ethane	.0004
1,4-dioxane	1.5898
2-ethoxy ethyl acetate	.5063

toxic compounds, which caused the experiment to be aborted, were formed by reaction of two common contaminants, trichloroethylene and vinylidene chloride, with the lithium hydroxide scrubber in the contaminant-removal system. The presence of monochloroacetylene and dichloroacetylene has also been reported in the atmosphere of submarines (refs. II-5 to II-7). In one instance, it was determined that the partial decomposition of methyl chloroform, which had been used as a solvent for an adhesive, in a malfunctioning Hopcalite burner produced trichloroethylene and vinylidene chloride (ref. II-5). The last two compounds reacted with lithium carbonate of the contaminant-removal system to produce the chloroacetylenes.

PHYSIOLOGICAL EFFECTS OF SPACECRAFT CONTAMINANTS

Toxicological Factors

All compounds capable of chemical reaction in the body have an adverse effect on the body at some quantity or concentration (ref. II-4). The primary factors that determine whether a compound produces a toxic effect, and the extent of this effect, are the intensity and duration of exposure. Adverse effects may occur when the magnitude of these factors is such that the body is no longer capable of maintaining a healthy normal environment by homeostatic alterations of physiological and biochemical mechanisms. As a general rule, a single exposure to a chemical, if not carcinogenic or lethal, does not produce persistent deleterious biochemical, physiological, or structural effects. In contrast, substances taken into the body repeatedly or continuously may not produce immediate changes but may slowly exert a deleterious persistent effect. This effect occurs in one of two ways. Either the substance may collect in the body in sufficient concentration to cause change or repeated small injuries may summate and exhaust the normal biochemical, physiological, or tissue-restorative abilities of the body.

The quantitative relationship between the toxic effect and the intensity and duration of exposure is commonly referred to as the dose-response relationship. In general, there are four possible relationships, which can be expressed as follows (ref. II-4).

1. $E \propto C$ - This relationship denotes that the effect E is entirely due to the concentration C of the compound. This relationship probably does not exist in real situations since time, however short, is always involved.

2. $E \propto CT$ - This relationship denotes that the effect is the product of the concentration and the exposure time T within certain limits.

3. $E \propto (dC)/(dT)$ - In this relationship, the effect is dependent on the rate at which the compound enters or leaves the cell.

4. $E \propto (C - A)T$ - This relationship corrects the concentration of the compound for its biotransformation to metabolites A, which may be more or less active than the compound.

The toxicity of a chemical compound may also be influenced by other factors including the time during which the compound remains within the body, individual variations in response to the compound, and environmental conditions. Although the intensity and duration of exposure can modify this factor, the time of residence of a compound within the body is determined primarily by the physical and chemical properties of the compound and depends on its rate and route of absorption into the body and of elimination from the body, its distribution and storage within the body, and its biotransformation. Individual variations in response to a compound may result from sensitization, tolerance, and idiosyncratic reactions to the compound as well as from differences in the general physical condition and state of health of individuals. Environmental conditions such as weightlessness, stress, the presence of other compounds, and radiation also may modify the toxicity of a compound.

Toxicity of Spacecraft Contaminants

Inherent in the wide variety of chemical structures of spacecraft contaminants is the potential of these compounds to produce toxicological effects of comparable diversity. These effects may be classified in several ways. One of the simplest, as suggested by Roth et al. (ref. II-4), is the classification of toxic agents as asphyxiants, irritants, or toxicants as follows.

1. Asphyxiant: An asphyxiant is any agent which interferes with the oxygen supply or its utilization. These may be further differentiated as simple asphyxiants and biochemical asphyxiants.

a. Simple asphyxiant: Simple asphyxiants reduce the concentration of oxygen in the lungs by physical displacement of the oxygen available in the atmosphere. Examples are methane and ethylene. Since this type of agent produces an effect only at relatively high concentrations, it is not likely to be important as a trace contaminant.

b. Biochemical asphyxiant: Biochemical asphyxiants reduce the availability of oxygen to the cells by interfering with the transport or utilization of oxygen. Examples of this type of asphyxiant are carbon monoxide, which reduces oxygen transport by preferential binding of hemoglobin, and hydrogen cyanide, which prevents cell uptake and utilization of oxygen by binding oxidative enzymes in the cell. Since these agents are effective at low concentrations, they are very important as trace contaminants in the spacecraft atmosphere.

2. Irritant: An irritant is any agent which produces an undesirable response of a tissue but not one which results in significant tissue damage. Tissues most frequently involved are the skin, mucous membranes, the cornea, and the respiratory and gastrointestinal tracts. Examples of these agents are most aldehydes and acids. Several other groups of compounds, such as alcohols and esters, may be irritants at low concentrations and toxicants at higher doses.

3. Toxicant: A toxicant is any agent which produces either temporary or permanent interference with normal function. Toxicants which act on the

central nervous system (CNS) are the most rapid acting and constitute the greatest threat to space crews. Toxicants which act on other organs, such as bone marrow, kidney, and liver, are less rapid acting and may permit completion of a mission but can produce severe and permanent effects.

A more specific classification of 175 potential spacecraft contaminants was made by Hine and Weir (ref. II-8), who classified the compounds according to their effects on 13 body systems. The authors selected these compounds as the most frequently present or most likely to cause problems from a review of the literature on contaminants in closed spaces inhabited by man. The potential contaminants and body systems affected are shown in table II-5. Many of the contaminants have the capability, depending on the dose, to affect more than one system. Table II-6, which is a summary of these data, shows that the most common effects of these compounds are mucous membrane and respiratory tract irritation and CNS depression. Contaminants with potential to act on these body systems will predominate in any manned spacecraft since these toxic effects can be produced by several chemical groups prevalent in the spacecraft, such as alcohols, aromatics, ketones, and esters.

Of all of the potential spacecraft contaminants, carbon monoxide (CO) is of particular toxicological concern. In fact, it is regarded by some as a potential limiting toxicant because of its continuous production by man and materials and its effect on health and performance even at low concentrations (ref. II-4). Carbon monoxide is generated continuously in the spacecraft atmosphere by the oxidative degradation of cabin organic materials and as a metabolic waste product of man. In man, this gas is produced principally from the normal catabolism of hemoglobin with a minor fraction contributed by the breakdown of nonhemoglobin heme (refs. II-9 and II-10). In healthy male subjects at rest, the average rate of endogenous CO production is approximately 0.5 ml/hr (refs. II-10 and II-11). Using this value, the buildup in the Orbiter cabin of carbon monoxide from man alone has been calculated for different numbers of cabin occupants and for mission durations of as many as 30 days (fig. II-1). The dilution effect of cabin leakage of 3.2 kilograms (7 pounds) of air per day is shown in figure II-2. This leakage delays the buildup of CO cabin concentration, when 7 men are present, to 17 mg/m^3 from approximately 10 days, with no leakage, to 12 days.

The toxic effects of carbon monoxide are exerted principally by the great affinity of CO for hemoglobin (approximately 200 times greater than the affinity of hemoglobin for oxygen). This formation of carboxyhemoglobin reduces the availability of hemoglobin for oxygen transport and results in oxygen starvation of body tissues. Cardiovascular and CNS functions are particularly vulnerable to reduced oxygen supply.

Measurement of carboxyhemoglobin in the blood is a valid measure of the body burden of carbon monoxide and can be correlated with toxic effects. The relationship among blood carboxyhemoglobin levels, symptomatology, exposure duration, and CO concentration is shown in figure II-3 (refs. II-12 and II-13). The toxic effects that result when blood carboxyhemoglobin levels exceed 10 percent are well documented and universally accepted. However, whether carboxyhemoglobin levels below 10 percent do produce impairment of mental function, as indicated in this figure, is a matter of controversy.

TABLE II-5.- CLASSIFICATION OF POSSIBLE CONTAMINANTS OF THE SPACE CAPSULE ACCORDING TO THEIR TOXIC EFFECTS ON DIFFERENT BODY SYSTEMS

Component	System or effect[a]												
	1	2	3	4	5	6	7	8	9	10	11	12	13
Acetaldehyde				x								x	
Acetic acid									x			x	
Acetone				x					x				
Acetonitrile					x	x						x	
Acetylene				x					x				x
Acrolein									x	x		x	
Allantoin													
Aluminum												x	
Ammonia					x				x			x	
Isoamyl alcohol			x	x					x			x	
N-amyl acetate				x					x			x	
Antimony			x					x		x		x	
Arsine		x						x	x	x		x	
Benzene			x	x			x		x			x	
Benzyl alcohol				x									
Beryllium						x			x	x		x	
N-butane				x									x
Butene-1				x									x
Cis-butene-2				x									x
Trans-butene-2				x									x
N-butyl acetate				x					x			x	
N-butyl alcohol				x				x	x	x		x	
Isobutyl alcohol				x					x			x	
Sec-butyl alcohol				x					x			x	
Tert-butyl alcohol				x					x				
N-butyraldehyde									x			x	
Butyric acid									x				
Cadmium										x		x	
Calcium									x			x	
Carbon dioxide				x									x
Carbon disulfide					x	x					x		
Carbon monoxide		x											
Carbon tetrachloride			x	x				x		x			
Carbonyl sulfide					x	x							
Chlorine									x			x	
Chloroacetone									x			x	
Chromium									x	x		x	

[a]1, autonomic nervous system; 2, blood; 3, cardiovascular system; 4, CNS depressant; 5, CNS stimulant; 6, enzyme inhibitor; 7, hemopoietic tissue; 8, hepatotoxic agent; 9, mucous membrane; 10, nephrotoxic agent; 11, peripheral nervous system; 12, respiratory system; and 13, simple asphyxiant.

TABLE II-5.- Continued

Component	System or effect[a]												
	1	2	3	4	5	6	7	8	9	10	11	12	13
Cigarette smoke (?)													
Copper									x			x	
Creatinine				x									
P-cresol					x				x	x		x	
Cumene				x				x	x			x	
Cyclohexane				x				x		x			
Cyclopentanone				x									
Decaborane				x									
Dimethyl amine												x	
Dimethyl butane				x									
1,3-dimethyl-5-ethyl benzene													
Dimethyl hydrazine, unsymmetrical					x								
Dime hyl sulfide						x							
1,4-dioxane				x				x	x	x		x	
Dioxene				x					x				
Ethanol				x					x				
Ethyl acetate				x					x			x	
Ethyl amine			x						x				
Ethyl benzene				x					x			x	
Ethylene				x									
Ethylene diamine								x	x	x		x	
Ethylene dichloride				x				x		x		x	
Ethylene glycol										x		x	
Ethyl ether				x					x				
Ethyl formate				x					x			x	
Ethyl mercaptan				x									
Ethyl sulfide						x			x				
P-ethyl toluene				x					x			x	
Formaldehyde									x			x	
Formanide									x				
Formic acid									x				
Freon 11				x					x				x
Freon 12				x					x				x
Freon 22				x					x				x
Freon 23				x					x				x
Freon 113				x					x				x
Freon 114, symmetrical													
Freon 114, unsymmetrical													
Freon 125													

[a]1, autonomic nervous system; 2, blood; 3, cardiovascular system; 4, CNS depressant; 5, CNS stimulant; 6, enzyme inhibitor; 7, hemopoietic tissue; 8, hepatotoxic agent; 9, mucous membrane; 10, nephrotoxic agent; 11, peripheral nervous system; 12, respiratory system; and 13, simple asphyxiant.

TABLE II-5.- Continued

Component	System or effect[a]												
	1	2	3	4	5	6	7	8	9	10	11	12	13
Furan				x					x				
Gasoline vapors				x					x				
Gold								x		x			
N-heptane				x					x				
Hexamethylcyclotrisiloxane									x			x	
N-hexane									x			x	x
Hexene-1				x					x				
Hippuric acid									x				
Histamine	x		x										
Hydrazine					x			x		x			
Hydrogen													x
Hydrogen chloride									x			x	
Hydrogen cyanide					x	x							
Hydrogen fluoride						x		x	x	x		x	
Hydrogen sulfide						x			x			x	
Indican									x				
Indole		x							x				
Iron												x	
Isoprene				x					x				
Lactic acid									x				
Lead					x								
Magnesium				x					x				
Mangarese				x				x				x	
Mercury			x		x			x		x			
Methane				x									x
Methanol				x		x					x		
Methyl acetate				x								x	
Methyl acetylene				x								x	
Methyl amine												x	
Methyl bromide				x				x				x	
Methyl chloride									x			x	
Methylene chloride				x					x				
Methyl chloroform			x	x				x	x				
Methyl ethyl ketone				x					x			x	
Methyl formate				x					x			x	
Methyl furan				x					x			x	
Methyl cyanide						x							
Methyl isobutyl ketone				x								x	
Methyl mercaptan						x						x	

[a]1, autonomic nervous system; 2, blood; 3, cardiovascular system; 4, CNS depressant; 5, CNS stimulant; 6, enzyme inhibitor; 7, hemopoietic tissue; 8, hepatotoxic agent; 9, mucous membrane; 10, nephrotoxic agent; 11, peripheral nervous system; 12, respiratory system; and 13, simple asphyxiant.

TABLE II-5.- Continued

Component	System or effect[a]												
	1	2	3	4	5	6	7	8	9	10	11	12	13
Methyl methacrylate				x					x				
3-methyl pentane				x					x				
Methyl sulfide												x	
Molybdenum									x			x	
Monoethanolamine								x	x	x		x	
Nickel									x			x	
Nitric oxide				x					x			x	
Nitrogen													x
Nitrogen dioxide									x			x	
Nitrogen oxychloride									x			x	
Nitrous oxide				x									x
Oxalic acid									x	x			
Oxygen						x						x	
Ozone						x			x			x	
Pentaborane-9					x								
N-pentane				x									
Isopentane				x									
Phenol		x	x					x		x			
Phosgene												x	
Phosphene								x		x		x	
Potassium									x			x	
Propane				x									x
Propionaldehyde				x					x				
Propionic acid									x			x	
N-propyl alcohol				x					x				
Isopropyl alcohol				x					x				
Propylene				x									x
Pseudocumene				x					x				
Pyruvic acid									x			x	
Radon		x					x						
Resorcinol									x			x	
Selenium								x		x			
Silver												x	
Skatole												x	
Skatoxylsulfuric acid												x	
Sodium									x			x	
Stibine		x				x						x	
Sulfur dioxide									x			x	
Sulfuric acid									x			x	
Sulfuryl chloride									x			x	
Tellurium						x		x					

[a]1, autonomic nervous system; 2, blood; 3, cardiovascular system; 4, CNS depressant; 5, CNS stimulant; 6, enzyme inhibitor; 7, hemopoietic tissue; 8, hepatotoxic agent; 9, mucous membrane; 10, nephrotoxic agent; 11, peripheral nervous system; 12, respiratory system; and 13, simple asphyxiant.

TABLE II-5.- Concluded

Component	System or effect[a]												
	1	2	3	4	5	6	7	8	9	10	11	12	13
Tetrahydrofuran				x				x	x	x		x	
Thionylchloride									x			x	
Titanium		x										x	
Toluene				x				x	x	x			
Triaryl phosphate											x		
Trichloroethylene								x	x	x		x	
Trimethyl amine									x			x	
1,3,5-trimethyl benzene				x				x	x				
Urea													
Uric acid													
N-valeraldehyde									x			x	
Isovaleraldehyde									x			x	
Vinyl chloride				x				x					
Vinylidene chloride				x					x				
Water and water vapor													
M-xylene								x	x			x	
O-xylene								x	x			x	
P-xylene								x	x			x	
Zinc									x				

[a]1, autonomic nervous system; 2, blood; 3, cardiovascular system; 4, CNS depressant; 5, CNS stimulant; 6, enzyme inhibitor; 7, hemopoietic tissue; 8, hepatotoxic agent; 9, mucous membrane; 10, nephrotoxic agent; 11, peripheral nervous system; 12, respiratory system; and 13, simple asphyxiant.

TABLE II-6.- SUMMARY OF THE TOXIC EFFECTS OF 175 POTENTIAL SPACECRAFT CONTAMINANTS ACCORDING TO BODY SYSTEM AFFECTED

[Ref. II-8]

Body system affected; contaminant examples	No. of contaminants producing effect
Autonomic nervous system; histamine	1
Blood; carbon monoxide, indole, phenol	6
Cardiovascular system; isoamyl alcohol, ethyl amine, Freons	9
CNS depressants; acetaldehyde, acetone, benzyl alcohol	74
CNS stimulants; acetonitrile, carbon disulfide, hydrogen cyanide	12
Enzyme inhibitors; carbon disulfide, ethyl sulfide, hydrogen sulfide	16
Hemopoietic tissues; benzene	1
Hepatotoxic agents; ethylene dichloride, methyl chloroform, toluene	30
Mucous membranes; acrolein, ammonia, ethyl acetate	97
Nephrotoxic agents; cyclohexane, ethylene glycol, trichloroethylene	26
Peripheral nervous system; carbon disulfide, methanol, triaryl phosphate	3
Respiratory irritants; ammonia, alcohols, formaldehyde	83
Simple asphyxiants; acetylene, methane, propylene	18

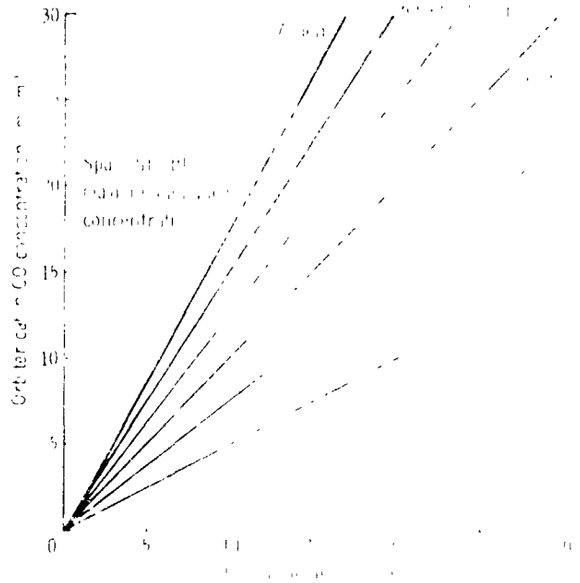

Figure II-1.- Orbiter cabin carbon [illegible] sources as a function of [illegible].

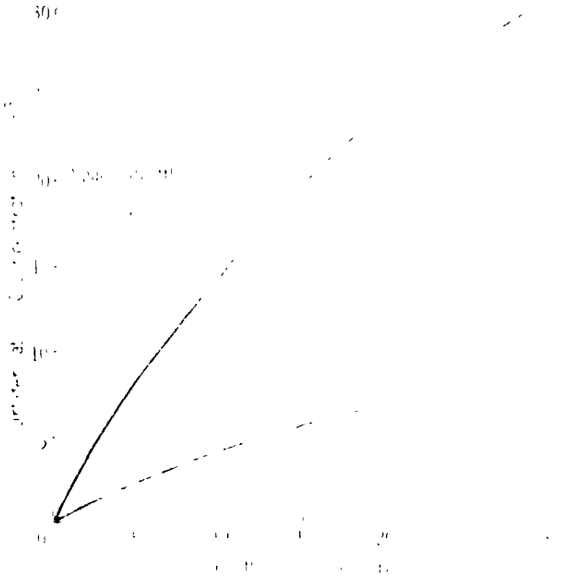

Figure II-2.- Orbiter [illegible] sources as a function of [illegible] (7 lbm/day).

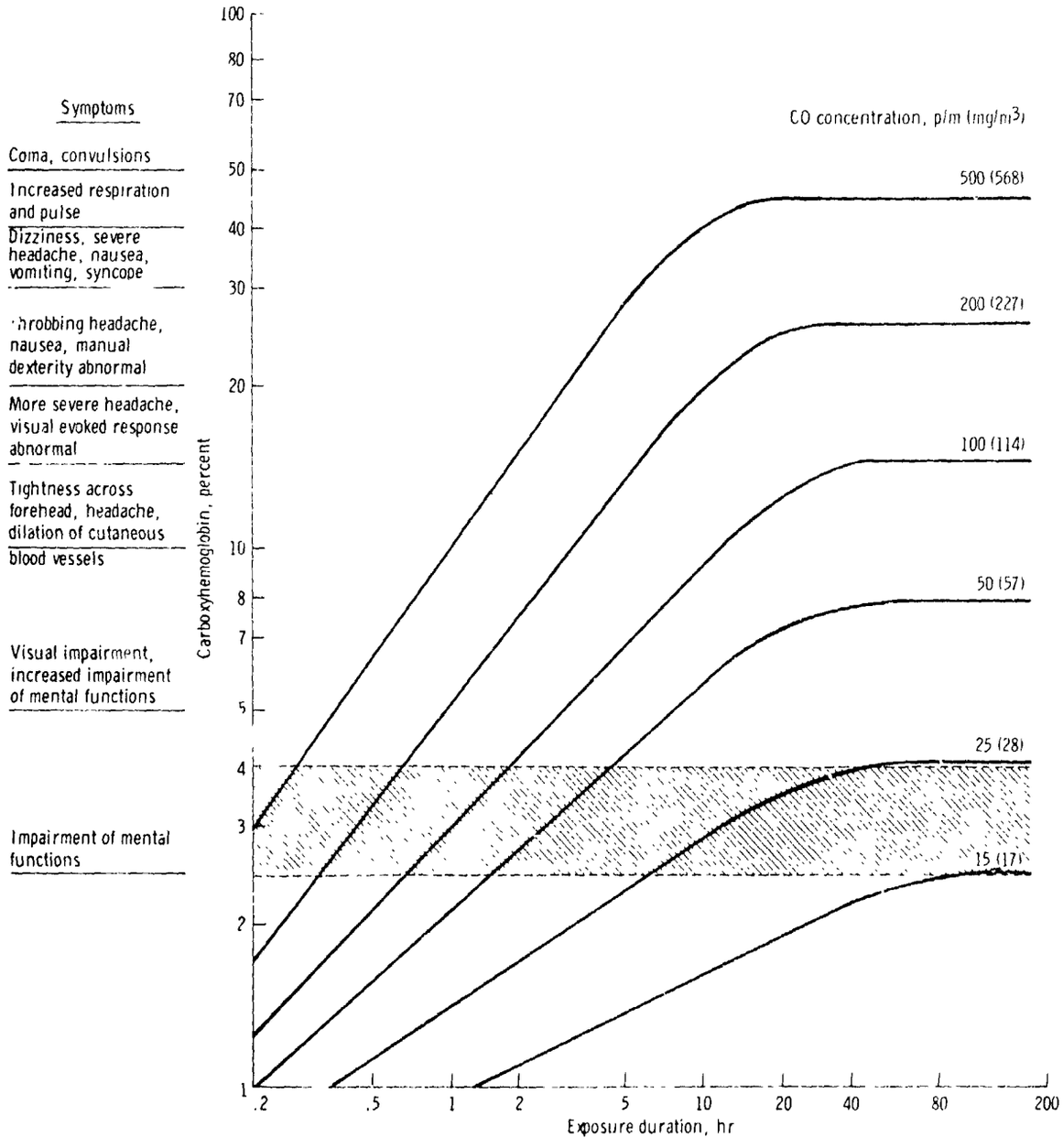

Figure II-3.- Carboxyhemoglobin percentage as a function of exposure duration, including symptoms and relative concentrations of carbon monoxide. The shaded area represents 90-day CO concentration limits imposed for the Space Shuttle Orbiter (15 p/m (17 mg/m^3)) and for U.S. Navy submarines (25 p/m (28 mg/m^3)).

Several investigators have suggested that central nervous system function is impaired at carboxyhemoglobin levels as low as 2 to 5 percent (refs. II-14 to II-16), but contradictory findings have been reported by equally competent scientists (refs. II-17 to II-21). In his review of the effects of carbon monoxide on humans, Stewart (ref. II-13) presented and evaluated the evidence for and against central nervous system impairment by low carboxyhemoglobin levels. In his opinion, the evidence tended to support the view that the ability to perform complex tasks requiring both judgment and motor coordination is not affected adversely by carboxyhemoglobin saturations below 10 percent. Furthermore, reported performance decrements in subjects with carboxyhemoglobin saturations of less than 5 percent, in Stewart's opinion, must be considered suspect until verified by an independent investigator.

Although sudden exposures to high concentrations of carbon monoxide may impair human performance, there is evidence that the slow buildup of this gas in the closed spacecraft environment, although not desirable, may cause an adaptive increase in hemoglobin and red blood cells and permit normal functioning despite elevated carboxyhemoglobin levels (ref. II-22). The occurrence of this environmental adaptation has been reported in studies of prolonged exposure of experimental animals to carbon monoxide. Back and Dominguez (ref. II-23) and Back (ref. II-24) found no detectable effects on learned performance in monkeys exposed to carbon monoxide concentrations of 50, 200, and 400 p/m for periods of as many as 105 days. These investigators attributed the failure of the gas to impair performance in these animals, which had blood carboxyhemoglobin levels of as high as 33 percent, to an increase in red blood cells and total hemoglobin that allowed sufficient oxygen delivery to tissues to prevent cellular hypoxia. This adaptive mechanism was shown to occur in other animal species by Vernot et al. (ref. II-25). Furthermore, cigarette smokers, as well as nonsmokers in metropolitan areas with the greatest air pollution, are reported to also show this compensatory increase in red blood cell mass (ref. II-13).

APPROACHES TO SPACECRAFT ATMOSPHERIC CONTAMINATION

Despite the presence, in the spacecraft atmosphere, of several hundred contaminants with the potential to produce a wide variety of toxic effects, many manned space missions have been completed without any evidence of toxicity to crewmembers. Obviously, the concentrations of these contaminants were maintained below their threshold-effect levels during these missions. Although contaminant levels were controlled primarily by the ECLSS, ancillary programs were essential to provide guidelines for the design of effective systems as well as to restrict the use of materials with high potential toxicity. These programs include materials and spacecraft offgassing tests, materials screening, toxicological evaluations of materials, and postflight analyses of spacecraft adsorbents. The NASA approaches to the spacecraft atmospheric contamination problem are summarized as follows.

1. Identification of contaminants and sources

   a. [illegible]

   b. Spacecraft ground tests

   c. Postflight adsorbent analyses

2. Determination of toxicity of potential contaminants - Establishment of contaminant maximum allowable concentration (MAC) values

3. Contaminant control procedures

   a. Materials manufacture and treatment

   b. Materials selection program

   c. Spacecraft contaminant-removal system

4. Verification procedures

   a. Spacecraft ground tests

   b. Postflight analysis of spacecraft atmosphere

   c. Development of onboard monitoring instrument tion

Some of these approaches have been mentioned previously, and others are discussed in detail in the following sections.

Identification of Contaminants and Sources

Recognition of the potential hazards of materials offgassing from experiences with closed loop systems in submarines prompted the first concern for materials screening during the early days of Project Mercury. In the Gemini Program, increased emphasis was placed on materials screening by conducting analytical and toxicological studies of offgassing products. Since the tragic Apollo AS-204 fire, identification of potentially hazardous materials by offgassing tests and materials selection has assumed even greater importance.

The selection of materials for use in manned spacecraft is controlled by NHB 8060.1A (ref. II-26). This document, which is the responsibility of the Office of Manned Space Flight, NASA Headquarters, establishes the criteria for materials selection on the basis of flammability, odor and offgassing parameters, and the requirements for testing these parameters. Present offgassing criteria for acceptance of cabin materials limit offgassing to 100 micrograms of total organics and 25 micrograms of carbon monoxide per gram of material. However, a planned revision of NHB 8060.1A will replace the measurement of total organics, which does not enable a toxicological assessment of the material, with the requirement for the complete identification of all

offgassing products of every material. In accordance with the latter requirement, complete offgassing analyses are being performed on all cabin materials for Orbiter vehicles. Recently, a computerized program was instituted to supplement materials selection control for the Orbiter. This program will enable the identification of all offgassing products of every material in the cabin, will compile the total quantity of each product, and will calculate the resultant Orbiter cabin concentration of each contaminant. The data from a computer printout of the offgassing products of 70 Orbiter OV-101 cabin materials tested at the NASA White Sands Test Facility are shown in table II-4.

An additional source of contaminant identification data is the total spacecraft offgassing ground test required by NHB 8060.1A as a verification procedure for spacecraft toxicological safety during manned flight. The provisions of this test require the measurement of the total quantity of individual products offgassed by each spacecraft. Additionally, postflight analyses of the charcoal adsorbent of the contaminant-removal systems of many manned spacecraft and of the Tenax adsorbent on the Skylab 4 mission have contributed significantly to the identification of potential spacecraft contaminants and their sources.

Establishment of Spacecraft Contaminant Standards

Even with materials selection controls, the generation of considerable quantities of contaminants in the spacecraft atmosphere from cabin materials, as well as from man, is inevitable. Therefore, it is essential that probable contaminants are identified, that their potential toxicity is ascertained, and that an effective contaminant-removal system is designed to limit their quantities in the atmosphere to levels which do not produce toxic effects.

The determination of appropriate limit values for spacecraft contaminants is not a simple task, primarily because no parallel exists for the space mission in experiences of industrial hygiene or occupational medicine. For example, the threshold limit value (TLV) set established by the American Conference of Governmental Industrial Hygienists represents, for the most part, time-weighted average concentrations to which all workers can be exposed 8 hours daily, 5 days weekly, without adverse effects (ref. II-27). These values are not directly applicable to the continuous long-term exposure conditions of space flight. Extrapolation of these industrial air-limit values to the spacecraft atmosphere is further complicated by other factors inherent in the space environment, such as the multicontaminant atmosphere, weightlessness, radiation, stress, prolonged confinement, and other factors that tend to alter man's normal physiology and, thereby, alter his response to any contaminant. Finally, no industrial air-limit values have been established for many potential spacecraft contaminants because of either their infrequent occurrence in the industrial environment or a lack of available toxicological data.

The first major step in establishing appropriate standards for spacecraft atmospheric contaminants was taken in 1968, when a Panel on Air Standards for Manned Space Flight was assembled, at NASA request, by the National

Academy of Sciences (NAS) to recommend limit values for human exposure to spacecraft contaminants (ref. II-28). Approximately 200 potential contaminants identified from analyses of spacecraft, simulated spacecraft studies, and offgassing experiments were reviewed. The recommendations of this panel included (1) acceptance of the 90-day U.S. Navy submarine missions air quality standards for 23 contaminants (table II-7) for 90-day space-flight missions; (2) adoption, as provisional guidelines for space flight, limits for 11 contaminants for 90-day and 1000-day missions (table II-8); and (3) adoption of 60-minute provisional emergency limits for 5 contaminants in event of a single exposure during flight (table II-9). Although some limit values have been reviewed and revised for usage in the Space Shuttle Program, these limits are generally inapplicable to planned Space Shuttle missions because of their shorter duration.

In 1971, a second panel was established, at the request of NASA, by the NAS Committee on Toxicology to review and extend the limits recommended by the 1968 NAS panel (ref. II-29). Provisional limits were established for 52 potential spacecraft contaminants for a variety of exposure durations (table II-10). The panel emphasized that each limit value was provisional and subject to change, as more information became available, and represented a maximum allowable concentration of a single contaminant, without regard to its occurrence in mixtures of contaminants. For toxicological assessment of contaminant mixtures, the panel recommended the use of a group-limit concept which, essentially, limited the total concentration of a group of structurally related contaminants to the individual limit value of the most toxic member of the group.

More recently, the development of the Space Shuttle Program necessitated the establishment of contaminant limits for 7-day and 30-day missions as guidelines for environmental control system design for the Orbiter and the European Space Agency Spacelab. Appropriate contaminant MAC values were derived by the NASA Lyndon B. Johnson Space Center (JSC) Toxicology Section by extrapolation and modification, when indicated by more recent toxicity data, of limit values recommended by the NAS panels. In the Orbiter contract end item specifications, contaminants are grouped into structurally related classes and the group-limit concept, which limits the total concentration of a group to the MAC value of its most toxic member, is applicable (table II-11; ref. II-30). In Spacelab specifications, in contrast, an MAC value was established for each individual contaminant to meet the engineering requirements for design of its contaminant-removal system.

The establishment of spacecraft contaminant standards for various flight durations and as both group and individual contaminant limits has led to inadequacies and, perhaps, some confusion in the proper usage of these values. Furthermore, although the group-limit concept was adopted to compensate for the additive effects of structurally related compounds, this same provision has not been made for the many contaminants that are not structurally related but do have similar effects. Because of these deficiencies, a program was recently initiated by the NASA JSC Toxicology Section to review and evaluate possible approaches for establishing spacecraft contaminant standards. Upon completion of this effort, standardized MAC values that are applicable to all

TABLE II-7.- U.S. NAVY SUBMARINE CONTAMINANT CONCENTRATION LIMITS

Contaminant	90-day limit, p/m
Acetone	30
Acetylene	2500
Ammonia	25
Benzene	1
Carbon monoxide	25
Chlorine	.1
Dichlorodifluoromethane	1000
1,1,2,2-tetrafluoro-1,2-dichloroethane	1000
Ethyl alcohol	100
Hydrogen	3000
Hydrogen chloride	1
Hydrogen fluoride	.1
Methane	5000
Methyl alcohol	10
Methyl chloroform	500
Monoethanolamine	.5
Nitrogen dioxide	.5
Ozone	.02
Phosgene	.05
Sulfur dioxide	1
Toluene	--
1,1,1-trichloroethane	200
Xylene	--

TABLE II-8.- PROVISIONAL LIMITS FOR SPACE CABIN CONTAMINANTS FOR 90 AND 1000 DAYS[a]

Air contaminant	Air limit, p/m, for -	
	90 days	1000 days
N-butanol	10	10
2-butanone	20	20
Carbon monoxide	15	15
Chloroform	5	1
Dichloromethane	25	5
Dioxane	10	2
Ethyl acetate	40	40
Formaldehyde	.1	.1
2-methyl butanone	20	20
Trichloroethylene	10	2
1,1,2-trichloro-1,2,2-trifluoroethane and related congeners	1000	200

[a]Data from National Academy of Sciences, Space Science Board, 1968.

TABLE II-9.- PROVISIONAL EMERGENCY LIMITS
FOR SPACE CABIN CONTAMINANTS[a]

Air contaminant	Air limit for 60 min	
	p/m	mg/m^3
2-butanone	100	294
Carbonyl fluoride	25	68
Ethylene glycol	100	254
2-methyl butanone	100	352
1,1,2-trichloro-1,2,2,2-trifluoroethane and related congeners	30 000	2320

[a]Data from National Academy of Sciences, Space Science Board, 1968.

TABLE II-10.- ATMOSPHERIC-CONTAMINANT LIMITS FOR MANNED SPACECRAFT

Compound; mol. wt	Air limit, p/m (mg/m^3), for -					
	10 min, (a)	60 min		90 days	6 mo	
Alcohols						
Methyl alcohol; 32.04	--	200	(260)	40 (52)	40	(52)
Ethyl alcohol[b]; 46.07	2000 (3800)	2000	(3800)	50 (95)	50	(95)
N-butyl alcohol; 74.12	--	200	(600)	40 (120)	40	(120)
Isobutyl alcohol; 74.12	--	200	(600)	40 (120)	40	(120)
Sec-butyl alcohol; 74.12	--	200	(600)	40 (120)	40	(120)
Tert-butyl alcohol; 74.12	--	200	(600)	40 (120)	40	(120)
N-propyl alcohol; 60.11	--	200	(500)	40 (100)	40	(100)
Isopropyl alcohol; 60.11	400 (1000)	200	(500)	40 (100)	40	(100)
Esters						
Methyl acetate; 74.0	--	200	(600)	40 (120)	40	(120)
Ethyl acetate; 88.10	--	300	(1080)	50 (180)	50	(180)
Butyl acetate; 116.16	--	200	(940)	40 (188)	40	(188)
Propyl acetate; 102.1	--	200	(840)	40 (168)	40	(168)
Ketones						
Acetone[b]; 58.08	--	1000	(2400)	300 (720)	300	(720)
Methyl ethyl ketone; 72.1	--	100	(290)	20 (58)	20	(58)
Methyl isobutyl ketone; 100.08	--	100	(410)	20 (82)	20	(82)
Methyl isopropyl ketone; 86.77	--	[c]100	(350)	[c]20 (70)	[c]20	(70)
Aldehydes						
Acetaldehyde[d]; 44.05	--	50	(90)	10 (18)	10	(18)
Acrolein; 56.06	--	.2	(.5)	.1 (.2)	.1	(.2)
Formaldehyde; 30.03	--	1.0	(1.0)	.1 (.1)	.1	(.1)

[a]Special area.
[b]Not to be included in group limits.
[c]Estimated levels: more inhalation data with animal models would be desirable.
[d]Based on eye irritation.

TABLE II-10.- Continued

Compound; mol. wt	Air limit, p/m (mg/m^3), for -					
	10 min, (a)	60 min		90 days	6 mo	
Alicyclics						
Cyclohexane; 82.14	--	300	(1 020)	60 (204)	60	(204)
Cyclopentane; 70.13	--	300	(870)	60 (174)	60	(174)
Methyl cyclohexane; 98.14	--	500	(2 000)	[c]15 (60)	[c]15	(60)
Methyl cyclopentane; 84.1	--	300	(1 029)	[c]15 (51)	[c]15	(51)
Halogenated aliphatics						
Chloroform; 119.39	--	100	(490)	5 (24.5)	5	(24.5)
1,2-dichloroethane; 98.97	--	200	(800)	10 (40)	10	(40)
Dichloromethane; 84.94	--	100	(340)	25 (87.5)	25	(87.5)
Methyl chloroform; 133.4	--	300	(1 620)	50 (270)	50	(270)
Tetrachloroethylene; 165.85	--	100	(680)	5 (34)	5	(34)
Trichlorofluoromethane (Freon 11); 140.5	--	5000	(28 500)	100 (570)	100	(570)
Dichlorodifluoromethane (Freon 12); 124.0	--	5000	(25 000)	100 (510)	100	(510)
Trichlorotrifluoroethane (Freon 113), 192.5	--	500	(3 950)	50 (395)	50	(395)
Aromatics						
Benzene; 78.11	--	100	(320)	1.0 (3)	1.0	(3)
Ethyl benzene[e]; 106.16	--	200	(860)	20 (86)	20	(86)
Styrene; 104.1	--	50	(215)	[c]10 (43)	[c]10	(43)
Toluene; 92.1	--	200	(750)	20 (76)	20	(75)
1,3,5-trimethyl benzene; 120.2	--	25	(123)	[c]3 (15)	[c]3	(15)
Xylene (o,m,p); 106.12	--	100	(430)	20 (86)	20	(86)
Halogenated aromatic						
Dichlorobenzene, mixed o- and p-	--	50	(300)	5 (30)	5	(30)

[a]Special area.
[c]Estimated levels; more inhalation data with animal models would be desirable.
[e]Long-term limits based principally on odor.

TABLE II-10.- Concluded

Compound; mol. wt	Air limit, p/m (mg/m^3), for -							
	10 min, (a)		60 min		90 days		6 mo	
Heterocyclics								
1,4-dioxane[f]; 88.0	--		100	(360)	5	(18)	5	
Furan; 68.07	--		2	(5)	.04	(.1)	.04	
[illegible]; 117.15	1.0	(4.8	.0	(4.6)	.1	(.5	.1	
[illegible]; 131.1	1.0	(5)	1.0	(5)	.1	(.5)	.1	
Inorganics								
[illegible]; 17.03	[illegible]	0)	50	70)	25	17.5)	25	
[illegible]; 44.01	[illegible]	72 000	0	50	5 000	3 000	18 000	10 000
Carbon monoxide[h]; 28.01	--		125	(144	25	(29	25	
Hydrogen chloride, gas; 36.46	--		5.0	(7.5)	1.0	(1.5)	1.0	
Hydrogen fluoride, gas; 20.01	--		5.0	(4)	.1	(.08)	.1	(.1
Nitrogen dioxide; 46.01	--		2.0	(4)	.5	(1.0)	.2	
Phosgene; 98.92	-		.5	(2.0	.05	(.2)	.05	
Sulfur dioxide; 64.1	-		5.0	(13)	1.0	(3)	1.	
Miscellaneous								
Acetonitrile; 41.05	--		40	(68)	4.0	(6.8)	4.0	(6.8)
Methyl mercaptan; 48.11	1.0	(2)	1.0	(2)	.1	(.2)	.1	(.2)

[a]Special area.

[f]Levels are subject to drastic revision downward (<1 p/m) if future research proves that dioxane is carcinogenic in animal models at low (<100 p m/ inhalation concentrations.

[g]Long-term limits based partly on odor.

[h]The 60-minute limit is based on a requirement that the carboxyhemoglobin level not exceed 10 percent, assuming heavy work [illegible] (30 liters min respiration) and conformity to Coburn's equation. If the assumption of heavy work activity in the weightless state proves inaccurate, a value of 300 p/m (330 mg/m^3) is recommended. The milligrams per cubic meter limits are also specified for the 70-percent oxygen, 30-percent nitrogen atmosphere at 34.5 kN/m^2 (5 psia).

TABLE II-11.- MAXIMUM ALLOWABLE CONCENTRATIONS FOR ORBITER TRACE CONTAMINANTS

Compounds	Mol. wt	MAC, mg/m^3 (a)
Families		
Alcohols (as methanol)	32	10
Aldehydes (as acrolein)	56	.1
Aromatic hydrocarbons (as benzene)	78	3.0
Esters (as methyl butyrate)	102	30
Ethers (as furan)	68	3.0
Halocarbons		
Chlorocarbons (as chloroacetone)	93	.2
Chlorofluorocarbons (as chlorofluoromethane)	68	24
Fluorocarbons (as trifluoromethane)	70	12
Hydrocarbons (as n-pentane)	72	3.0
Inorganic acids (as hydrogen fluoride)	20	.08
Ketones (as diisobutyl ketone)	142	29
Mercaptans (as methyl mercaptan)	48	2.0
Oxides of nitrogen (as nitrogen dioxide)	46	.9
Organic acids (as acetic acid)	60	5
Organic nitrogens (as monomethyl hydrazine)	46	.03
Organic sulfides (as diethyl sulfide)	90	.37
Specific		
Ammonia	17	17
Carbon monoxide	28	15
Hydrogen cyanide	27	1.0

[a]The maximum allowable concentration (MAC) values represent the maximum total for a family of compounds and are based on the most toxic member of the family, except in the case of hydrocarbons (n-pentane chosen for convenience of instrumentation calibration). Total is defined as the summation of compounds in a family. If a specific compound is identified, an MAC value will be determined for the "known" compound. The measured concentration of the known compound is subtracted from that of the family's "unknown" constituents; the resultant is then compared to the family MAC value. Until all members of the family are identified, the MAC value for the family of compounds will remain unaltered.

manned spacecraft, with appropriate guidelines for their use, will be established and recommended for incorporation into NHB 8060.1A as an NASA spacecraft criterion. For some contaminants, tentative MAC values will have to suffice until sufficient data from planned in-house or contractual toxicity evaluations become available to enable establishment of more precise standards.

Spacecraft Contaminant-Removal System

The primary purpose in establishing contaminant limit values for the spacecraft atmosphere is to provide guidelines for the design of the contaminant-removal system of the spacecraft ECLSS. The following general requirements for an effective contaminant control system were derived by the first NAS panel (ref. II-28).

1. Contaminants must not produce significant adverse changes in the physiological, biochemical, or mental stability of the crew.

2. The spacecraft environment must not contribute to a performance decrement of the crew that will endanger mission objectives.

3. The spacecraft environment must not interfere with physical or biological experiments nor with medical monitoring.

Contaminant-removal systems for the control of gaseous contaminants in closed systems are generally designed on the principles of chemical removal or of physical removal or a combination of both. A comprehensive review of various methods for conditioning and regenerating air for spacecraft was recently made by Grishayenkov (ref. II-31). He points out that the greatest danger to the space crew, without means of cleansing the air of harmful impurities, is carbon monoxide and hydrocarbons. Although adsorption on activated charcoal is generally effective in removing hydrocarbons, the usual adsorptional methods are ineffective in removal of carbon monoxide. For effective control of CO, several catalytic oxidizing systems for conversion of carbon monoxide to carbon dioxide are available (ref. II-31).

The selection of a specific contaminant-removal system for ensuring the physiologic well-being of space crews in a sealed cabin is determined primarily by the habitation duration (ref. II-31). In Apollo spacecraft, the contaminant-removal system consisted of canisters of lithium hydroxide to remove carbon dioxide and of charcoal to remove volatile organics. Also, condensation of some gases in the heat exchanger unit contributed to the removal of substantial quantities of certain contaminants. A similar system is planned for the Space Shuttle spacecraft, although the potential buildup of carbon monoxide in the Orbiter has necessitated consideration of a catalytic oxidizer in addition. If a catalytic oxidizer is required, the potential conversion by this system of halocarbons to highly toxic halogen acid gases and of nitrogen-containing compounds to more toxic oxides of nitrogen will have to be considered. For some contaminants, limit values, which have been established on the basis of the intrinsic toxicity of the compounds, may have to be reevaluated on the basis of the toxicity of their potential derived products.

Verification Procedures

Offgassing tests of spacecraft materials and of the total spacecraft and manned chamber tests have provided invaluable information about potential cabin contaminants during space flight. In addition, postflight analyses of the charcoal from the contaminant-removal systems of several spacecraft and of the Tenax adsorbent carried on the Skylab 4 mission have contributed to knowledge of the identity and quantities of contaminants in manned spacecraft. Although these offgassing tests and postflight analyses adequately identified potential contaminants in earlier programs, the potential for toxic, and even hazardous, contamination of the atmosphere will be increased considerably in Space Shuttle payload vehicles by the use of chemicals, instruments, and materials for experimental and construction purposes. Therefore, for these missions, the capability for an early warning of an unsafe atmosphere and for the immediate initiation of corrective action is essential. This capability can be provided only by the real-time identification and quantitation of contaminants in the spacecraft atmosphere. In recognition of the necessity for an appropriate warning device, JSC initiated a program to develop an in-flight trace-gas monitoring instrument for use on Space Shuttle payload missions. This instrument will have the configuration of a gas chromatograph/sector-type mass spectrometer with a scan range of 24 to 240 atomic mass units and will be designed to operate on missions of as long as 30 days. Operational specifications include (1) programing capability for 4 daily analyses of the spacecraft atmosphere with the capability for an analysis every 2 hours; (2) the capability for identification and quantitation of more than 300 compounds, including all contaminants identified in previous spacecraft; and (3) instrument sensitivity that will enable detection of contaminants at concentrations below their established MAC values. It is anticipated that miniaturization techniques developed for the Viking mass spectrometer will enable production of an instrument, with the requisite capabilities, of minimum volume and weight (approximately 0.07 cubic meter (2.5 cubic feet) and 38.5 kilograms (85 pounds)), which will fit into a standard 48.3-centimeter (19 inch) equipment rack. This instrument, in addition to providing a means for the early detection of hazardous atmospheric contamination, will significantly increase knowledge of the generation of contaminants in the spacecraft and assist in optimizing the design of spacecraft contaminant-removal systems.

REQUIREMENTS FOR TOXICOLOGICAL RESEARCH

Toxicity Evaluations of Spacecraft Contaminants Under Continuous-Exposure Conditions

In their recommendations of contaminant limit values for manned space flight, the NAS panels emphasized that these values must be considered provisional and subject to revision with the availability of new data (refs. II-28 and II-29). One of the principal reasons given by the panels for this philosophy was the lack of adequate or complete toxicological information on which to derive limits for many potential spacecraft contaminants. Industrial threshold limit values have not been established for many of the compounds

identified in spacecraft; in addition, information on long-term continuous exposure to many contaminants is limited. For these reasons, the NAS panel, in 1972, recommended that chronic inhalation studies be conducted on a number of potential spacecraft contaminants, such as 1,3,5-trimethyl benzene, styrene, methyl cyclohexane, methyl cyclopentane, and methyl isopropyl ketone, to enable the establishment of valid spacecraft MAC values. This list of compounds should be expanded to include a considerable number of other compounds, which were subsequently identified in materials offgassing tests or in spacecraft, for which tentative MAC values had to be assigned, on the basis of limited toxicity data, by the NASA JSC Toxicology Section.

Toxicity Studies of Contaminant Mixtures

The second NAS panel also recommended that the validity of using the group-limit concept be evaluated by research involving chronic inhalation experiments with a multicontaminant atmosphere (ref. II-29). In accordance with this recommendation, contractual efforts have been supported and efforts for in-house experimentation have been initiated by the NASA JSC Toxicology Section to investigate the effects of continuous exposure to mixtures of contaminants. In one contractual study, the combined effects of dichloromethane and 1,1,1-trichloroethane, two prevalent spacecraft contaminants, were investigated (ref. II-32). The results of this 90-day continuous animal exposure study indicated that the combined hepatotoxic effect of the two compounds, at their individual threshold-effect levels, was not additive and therefore was no greater than the effect of each compound alone. Additional studies of the toxicity of mixtures of contaminants that are structurally related or have similar effects are in progress.

In the NASA program to investigate the effects of continuous exposure to a multicontaminant atmosphere, particular emphasis is being placed on the potential interactions of contaminants that have CNS effects. A large number of prevalent spacecraft contaminants, both related and unrelated structurally, represent a major potential hazard by their capability to alter CNS function and impair the performance of complex tasks. In the classification of 175 potential spacecraft contaminants by Hine and Weir (ref. II-8) according to the body systems affected, CNS depressants were the third most prevalent group. For many of these compounds, extrapolation of industrial TLV's to establish spacecraft MAC levels is invalid because industrial standards are often based on the gross toxic effects of the compounds. More subtle effects, such as performance impairment, are seldom considered in establishing industrial TLV's. These effects may be produced at concentrations below those measurably altering physiological and biochemical processes. Therefore, data relevant to the effects on performance of many contaminants are rarely available.

Modification of Contaminant Toxicity by Environmental Factors

The potential modification of contaminant toxicity by a number of environmental factors is another area requiring extensive research to enable the establishment of valid spacecraft MAC values. Industrial TLV's are based on

available toxicity data, most of which have been obtained on subjects in a "normal" physiologic state. The effects of stress, prolonged confinement, weightlessness, radiation, and other factors that might tend to alter man's normal physiology and thus change his response to any given compound cannot accurately be predicted at this time. Therefore, it has not been possible to consider the environmental factors of the space environment in defining spacecraft contaminant standards. Examples of potential interaction between contaminants and environmental factors include the effects of increased levels of radiation on the radiomimetic action of benzene, the effects of increased fatigue from stress factors (e.g., noise, vibration, and weightlessness) on CNS-active agents, and the altered toxicity of gases by condensation on particulates and aerosols in the space cabin.

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III. THERMAL ENVIRONMENT

By J. M. Waligora

DEFINITION AND DESCRIPTION

Man as a homeothermic animal maintains a body temperature 10 to 15 K (10° to 15° C) above his typical ambient temperature. This internal temperature is closely guarded by strong physiological responses that take preference over most other regulatory processes of the body. In addition to physiological control, man is motivated to regulate his temperature behaviorally without active physiological control by a very sensitive sensory response to temperature and change in temperature.

The body temperature of a man is a function of the dynamic balance between internal heat production and heat exchange with his environment. The four avenues of heat exchange between a man and his environment are convection, radiation, conduction, and evaporation. As a result of the multiple pathways of heat loss from the body, a large number of environmental factors affect the thermal balance. A change in any one of these factors can affect the heat balance and thereby the thermal environment.

Convection is the transfer of thermal energy from one surface to another through an intervening fluid in motion. To compute convective heat exchange between a nude man and an air environment, the following measurements must be made.

1. Skin temperature
2. Characteristic dimension of the body (shape, size)
3. Air temperature
4. Pressure
5. Velocity of air across the body
6. Gravity

Radiation is the transfer of thermal energy from one surface to another without dependence on an intervening medium. Radiation depends only on the temperatures and natures of the two surfaces. Thermal energy can be transmitted by radiation throughout the electromagnetic spectrum.

To compute heat exchange between a nude man and his radiant environment, the following measurements must be made.

1. Skin temperature

2. Effective surface areas

3. Reflectivity of the skin

4. Mean radiant temperature

5. Emissivity of the environment

6. Temperature of radiant sources

Conduction is the flow of heat from one surface to another through an intervening medium without the physical transfer of material. Conduction can occur in solids, in gases, and in liquids. To compute conductive heat exchange between man and a conducting surface, the following measurements must be made.

1. Skin temperature

2. Contact area

3. Thickness of the conductor

4. Temperature of the conducting medium

5. Specific thermal conductivity of the medium

Evaporative heat exchange occurs when a change of state of an intervening medium transfers thermal energy from one surface to another. In the body, the skin and the respiratory tract are two major sites for evaporative heat exchange. To compute evaporative heat exchange, the following measurements must be made.

1. Percent wetted area

2. Air temperature

3. Air velocity over body surface

4. Relative humidity

5. Skin temperature

6. Characteristic dimension

Hardy (ref. III-1) has combined the heat loss pathways in a general heat loss equation to arrive at heat loss from the body in kilocalories per hour. (1 kcal/hr = 1.163 watts.) This equation and lists of required measurements

indicate the complexity of the thermal environment and the large number of environmental variables that must be considered. The radiation term is

$$H = 1.37 \times 10^{-11}\left(T_s^{\,4} - T_0^{\,4}\right)t \cdot A \cdot f \cdot e - \left(1 - R_s\right)\left(\frac{1}{m} + \frac{R_c}{m'}\right)H_s \cdot A$$

the conduction term is

$$+ \left(\frac{1}{\frac{d_1}{k_1A_1} + \frac{d_2}{k_2A_2} + \frac{d_3}{k_3A_3} + \cdots}\right)\left(T_s' - T_1\right)$$

the convection term is

$$+ \frac{k}{D}\left[1 + a\left(\frac{DV\rho}{\mu}\right)^{\frac{1}{2}} + b\left(\frac{DV\rho}{\mu}\right)\right]\left(T_s - T_a\right)$$

and the evaporation term is

$$+ W\left(\frac{V}{D}\right)^{\frac{1}{2}}\left(E_s - RH \times E_a\right)$$

where H = total heat exchange

T_s = average skin temperature, kelvin

T_0 = average radiant environmental temperature, kelvin

t = 3600 (seconds in 1 hour)

A = DuBois surface area, square meters

f = ratio of effective radiating surface to the DuBois surface area (0.78 for nude man lying in anatomical position)

e = emissivity of the environment

R_s = reflecting power of skin for solar radiation

m = fraction of the body area exposed to sunlight

m' = fraction of the body area exposed to reflected radiation

R_c = reflecting power of environment

H_s = intensity of solar radiation

k_1, k_2, k_3 = component conductivity of objects in contact with the skin

A_1, A_2, A_3 = component area of objects in contact with the skin

d_1, d_2, d_3 = component thickness of objects in contact with the skin

T_s' = temperature of skin in contact with conducting medium

T_1 = temperature of conducting medium

k = thermal conductivity

D = characteristic dimension of the object; for example, the diameter of a sphere or a cylinder

a, b = constants depending on the particular units used

V = velocity of the gas

ρ = density

μ = viscosity, a factor concerned in the mobility of the gas molecule

T_a = temperature of the air

W = fraction of body area that is completely wet

RH = relative humidity

E_s = water-vapor pressure on the skin

E_a = water-vapor pressure in the air

To solve the equation, the following variables must be measured on the man: T_s, T_s', A, f, m, m', R_s, D, (A_1, A_2 - A_n), and W. The following variables defining the environment must be measured: T_0, e, H_s, R_c, T_a, air pressure p, V, (k_1, k_2 - k_n), (d_1, d_2 - d_n), RH, and T_1. The level of heat production that must be balanced by heat loss varies from 42.8 W/m^2 (36.8 kcal/hr/m^2), the value for basal metabolism for a 30-year-old man (ref. III-2), to 909 W/m^2 (782 kcal/hr/m^2), a value calculated from the oxygen consumption of cross-country skiers (ref. III-3).

The energy released by metabolism appears as heat in the body, except for the energy expended as real work or frictional work outside the body.

The thermoregulatory system of the body acts as a proportional controller. The physiological responses of the body to a thermal imbalance are proportional to the imbalance and tend to reduce the imbalance. Therefore, some change in body temperature can and will occur and can be tolerated up to a critical level. Heat capacity is a very significant term in considering short-term exposures where a balance between heat loss and heat production is not maintained. When heat production exceeds heat loss, and body temperature begins to rise, the physiological responses are vasodilation and sweating. Vasodilation increases blood flow to the skin, which increases skin temperatures and, therefore, increases heat loss to the environment through radiation and convection. Sweating greatly increases the evaporative heat loss from the skin. When heat loss exceeds heat production, and body temperature begins to fall, the major physiological responses are vasoconstriction and shivering. Vasoconstriction decreases blood flow to the skin and thereby decreases the skin temperature and reduces heat loss by radiation and convection from the skin surface. Shivering increases metabolic heat production by involuntary contraction of skeletal muscles and thereby tends to reduce or reverse a trend toward an increasing negative heat balance.

In addition to the physiological response to an imbalance between heat production and heat losses, there is often a sensory behavioral response to body heating or cooling. The behavioral response can provide a degree of control which is as sensitive as and, in some cases, more effective than physiological control mechanisms. A condition of imbalance in heat production and heat loss leads to a sensation of thermal discomfort. Avoidance of this discomfort is accomplished by behavioral responses. These behavioral responses may include change in posture to reduce or increase heat loss area, change in clothing to increase or decrease insulation, and seeking or creating a more suitable environment.

The thermal environment, then, includes all the parameters that bear on the balance of heat production and heat loss. Man attempts to maintain himself in an environment in which his thermal capacity will damp short transients in energy production. If a significant imbalance between man's heat production and heat loss begins to develop, the sensory behavioral response and physiological responses will act to counter the forces tending to move him away from his homeostatic condition.

PHYSIOLOGICAL EFFECT OF THERMAL IMBALANCE

If a thermal environment is such that neither behavioral nor physiological responses are effective in maintaining a thermal balance, significant deviations from the controlled body temperature and physiological effects of this imbalance will occur. If sweating is required for thermoregulation it will be associated with discomfort and a possible decrement in performance. Prolonged sweating may lead to salt and fluid imbalance, which can result in heat cramps or dehydration. Vasodilation will increase as the body tempera-

ture rises until a large fraction of the blood volume is pooled in the skin. If cardiac return becomes inadequate, circulatory collapse may occur and result in heat syncope or fainting. If body temperature continues to rise, tissue disfunction will occur as a direct result of heating. This condition will result in a loss of thermoregulation (heatstroke) and continued uncontrolled rise in body temperature.

If shivering is required for thermoregulation in a cold environment, it will be associated with discomfort and a possible decrement in performance. Vasoconstriction in a cold environment will lead to a reduction in skin temperatures, particularly in the extremities, and to a reduction of heat content of the arms and legs. Skin temperatures below 288 K (15° C) will be perceived as painfully cold, and a loss of dexterity will occur. Temperatures below 273 K (0° C) will result in frostbite and permanent damage. A continued decrease of body temperature will result in unconsciousness and ultimately loss of cardiac rhythm and cardiac failure. Heavy clothing tends to reduce the extent of shivering and vasoconstriction in the face of a negative heat balance. The result is a creeping hypothermia with reduced physiological response.

PHYSIOLOGICAL LIMITS

The element of behavior control in temperature regulation is dependent on a strong and compelling sense of temperature discomfort when the environment is too hot or too cold. Discomfort in the heat or the cold has a deleterious effect on performance. Thermal discomfort is, then, a critical limiting factor for long-duration exposures if optimum performance is to be maintained. The problem remains of defining and specifying the conditions that will provide thermal comfort.

The performance of men at many different tasks has been observed at a variety of temperatures. There is considerable variation in the results of these tests. In general, when motivation is high, when the subject is skilled in his task, and when duration of exposure is short, performance can be maintained to a point just short of collapse. At the opposite extreme, if the task is such that motivation is difficult to maintain, if exposure is prolonged, and if tasks are less familiar, differences in performance can be measured even in the upper border of the thermal comfort zone.

Pepler (ref. III-4), in a review of the literature on performance in heat, gives the following summary: "Heat has many different effects on human skills, depending on the nature of the task performed and on the degree of heat stress. There is evidence that moderate levels of heat have specific effect on the accuracy of skilled movements, interfere with the detection of small infrequent visual signals, and impair the performance of a number of intellectual tasks. Severe heat may have specific effects also, but a more general impairment of performance predominates which, in some respects, is reminiscent of the effect of cerebral anoxia."

A comfort criterion has been devised for use in space-flight design which uses a mathematical model of thermoregulation and environmental heat exchange to correlate changes in environmental factors with changes in body temperature and changes in thermoregulatory response.[1] Empirically determined limits of comfort based on sensations of warmth and coolness, identified under known environmental conditions and known levels of metabolism, have been input to the computer model and theoretical limiting levels of body temperature have been obtained (ref. III-5). By using the theoretical body temperature limits, comfort limits can be defined for any set of environmental conditions. The comfort box depicted in figure III-1 was constructed in this manner and served as a design specification for Skylab. Fanger (ref. III-6) has shown that at least 5 percent of a large group will find any one temperature uncomfortable. This observation suggests that an inclusive and practical approach to achieving comfort is to allow thermostatic temperature control around an optimum point determined by analysis and to provide some means for individuals to modify their heat balance, either by clothing selection or by individual air motion control. This approach has been employed in the Space Shuttle and Spacelab temperature specifications.

In an environment controlled for optimum performance, temperature should be selectable with an accuracy of ± 1.2 K ($\pm 1.2^{\circ}$ C) within a range of 291 to 300 K (18° to 27° C). When mean radiant temperature may vary substantially from gas temperature, the sensor controlling the thermostat should be sensitive to both air and wall temperatures. Under conditions in which a comfortable heat balance is maintained, variations in humidity do not have strong effect on comfort. However, when this heat balance can only be maintained at the upper limit of a comfort band or outside a comfort band, the humidity becomes very significant. To preserve a strong and effective thermoregulation response to overheating, particularly during short transients that may be encountered during exercise, an upper value of humidity should be stated. Since it is the difference in absolute humidity, or water-vapor pressure (pH_2O), between the skin and the ambient gas that limits evaporation, the units of the limiting value should be units of absolute humidity rather than of relative humidity. An upper limit of 1.87 kN/m^2 (14 torr) pH_2O is a component of the Space Shuttle Orbiter specification.

Under contingency conditions in which comfort temperatures cannot be maintained, tolerance is limited by the physiological response of the organism to the heat or cold stress. Leithead and Lind (ref. III-7) provide a detailed description and discussion of these avenues of physiological failure during heat stress. When the heat stress is acute with a duration of several hours, the avenue of physiological limitation is likely to be circulatory collapse. Vasodilation, in response to heat stress, effectively removes blood volume from the circulatory pool. When vasodilation is extreme and arterioles are open wide, capillary pressure is increased and filtration occurs with resultant edema of the skin tissue. At the same time, venous compliance

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[1]Waligora, J. M.: Thermal Comfort and Tolerance Design Criteria. NASA JSC Rep. BRO DB-57-67B, 1970.

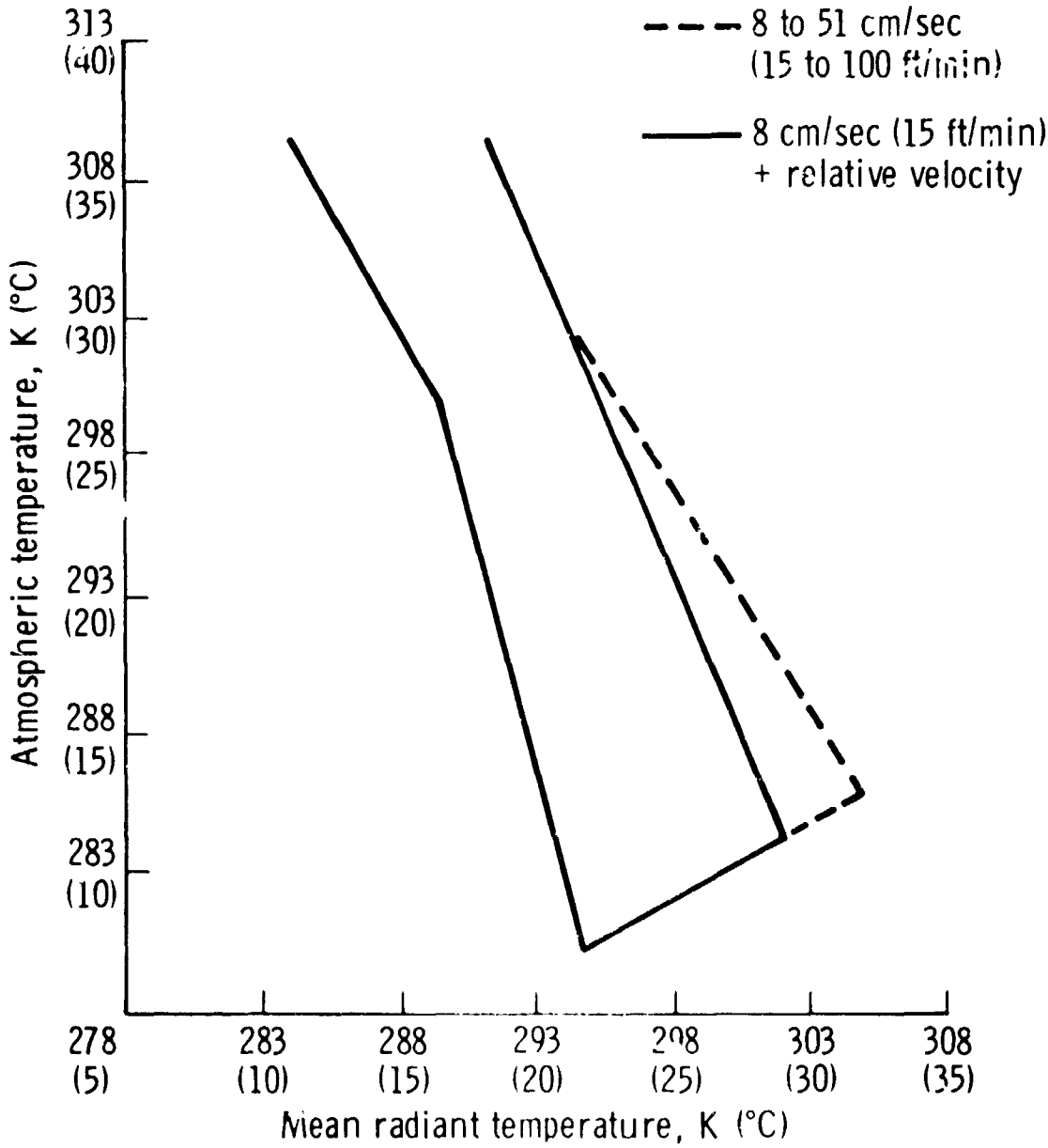

Figure III-1.- Comfort box for metabolic rates of 88 to 176 watts (300 to 600 Btu/hr), assuming thermal resistance (insulation) can be varied from 0.05 to 0.16 K•m^2/W (0.35 to 1.0 clo).

increases and a large quantity of blood remains in the venules and veins of the skin. If cardiac return becomes inadequate, circulatory collapse will occur. Although the point of circulatory collapse is in most cases a function of available blood volume, empirical studies have shown that the point of circulatory collapse can also be correlated with measurement of heat storage in the body, or the change in mean body temperature (refs. III-8 to III-11). This observation is consistent with the fact that thermoregulatory responses, including vasodilation, are closely correlated with a combination of body temperatures that approximates average body temperature (ref. III-12).

Blockley et al., in the U.S. Air Force Design Guide, set a minimum tolerance level for heat storage of approximately 4.2×10^5 joules (100 kilocalories) (ref. III-8). This is the point at which the more susceptible individuals in a group closely approached their physical tolerance limits. Blockley et al. also identified a minimum performance limit of three-fourths the tolerance limit. Beyond this point, adequate physical performance can no longer be relied upon. A number of investigators have reported similar heat storage values at the approach to tolerance, both at rest and during exercise (refs. III-9 to III-11). In 1970, Roth and Blockley (ref. III-13) performed a study for NASA to determine whether heat storage tolerance was extended during heavy exercise. Subjects were exposed to conditions that allowed no heat loss while they were working at high metabolic rates. The exposures were stopped at a point of imminent collapse. Heat storage was shown to be less definitive as a predictor of heat tolerance during exercise than at rest. The range of tolerable heat storage was shown to be much greater during exercise than at rest. However, the minimum tolerance point, the point at which the least tolerant subject approached collapse, was not much greater during work than at rest. In spacecraft life support systems design, 3.16×10^5 joules (75 kilocalories) is used as the heat storage limit where physical performance is essential for survival of the crewmen. In cases where performance of a crewman is not a requisite for his survival (as in the personnel rescue device), 4.22×10^5 joules (100 kilocalories) is used as the heat storage tolerance limit. The heat storage limit is a physiological limit. To use it as an environmental design guide requires a means to derive limiting environmental factors. Blockley used a set of nomograms to determine heat storage from a particular set of environmental factors. To achieve the same end, NASA used a computer model of thermoregulation and tolerance criteria which include limit ranges on some environmental factors. When heat storage is primarily due to a limited heat removal, the predictions of heat storage by the model rely primarily on the heat balance equations and are very reliable. An example would be a specific pressure-suit situation where the total heat removal capability was less than the heat production. When heat storage is primarily due to the effectiveness of the thermoregulatory system, individual variation makes the prediction of heat storage less precise. An example would be a man working very hard in a very hot environment, but one with high air velocities and low humidity. In this case, the heat storage would depend on the gain of the control system (the rise in body temperature necessary to produce sufficient sweat to allow a heat balance). In cases of this nature or in cases involving extreme ranges of some of the environmental factors, it is essential that predictions of heat storage and tolerance be verified by empirical exposures of men to conditions at or near those of the real case.

The loss of the thermal shield on the Skylab 1 flight required that the Skylab 2 crewmen enter and work in the orbital workshop to correct the problem while the wall temperatures were in excess of 327 K (54° C). To provide a guideline for the flight control team, time and tolerance limits were derived. They were based on existing empirically based limits modified and verified by heat storage predictions and empirical data from the literature.

Where temperatures are extreme, heat storage may not be the limiting factor for physiological tolerance. In such cases, a very high heat flux may result in painful skin temperatures. Painful skin temperatures can also occur from local contact. Pain from local skin heating occurs when the skin temperature reaches 318 K (45° C). This is also near the temperature at which protein denaturation begins and a burn will occur. Pain from local skin cooling occurs at temperatures near 288 K (15° C). This end point is not nearly as sharp as that due to skin heating. Permanent damage will not occur from acute exposures to low temperatures above 273 K (0° C).

If the heating is localized, blood flow to the heated area will dissipate approximately 3.5 kW/m^2 (49 $kcal/m^2/min$) (ref. III-14). If the heat flux to the skin does not exceed this level, a burn will not occur. The surface temperature limit for spacecraft and life support equipment is set at 318 K (45° C (113° F)). Exceptions to this limit may be allowed where heat flux analysis or empirical data indicate that contact will not cause a burn.

When heat production in the body does not keep pace with heat loss, a heat decrement or negative heat storage will occur. The thermal tolerance design criterion allows a negative heat storage of 3.165×10^5 joules (75 kilocalories). This is a conservative limit based on maintaining performance. Heat loss beyond this point may result in heavy shivering and loss of manipulative dexterity.

INTERACTING ENVIRONMENTAL FACTORS

Some environmental factors that are not normally associated with the heat balance equation may have an interacting effect with the thermal environment. Factors such as nutrition and fluid intake are likely to affect heat tolerance as are any environmental factors that might affect the cardiovascular system. Any environmental parameter that requires shielding or special clothing for the man, as in the case of a pressure suit, will have a very substantial indirect effect on the thermal balance of a man through the impact of this clothing on the heat exchange with the environment.

EFFECT OF INDIVIDUAL VARIATION AND EXPOSURE DURATION ON LIMITS

Since the population flying in space in the decades of the eighties and nineties will be much more diverse in demographic terms than the astronauts who have flown to date, the question arises: to what extent do intergroup

differences affect the requirements for the thermal environment? By investigating comfort under well controlled conditions, Fanger (ref. III-6) has shown that no significant differences in the optimum temperature for comfort can be demonstrated as a function of age, sex, nationality, or body type. Under the conditions of the tests, levels of metabolism and type of clothing were controlled. In practical situations, however, customary or social differences between population segments, such as clothing differences between men and women or differences in activity levels between younger and older persons, may create practical differences in comfort temperatures. The question of intergroup difference in heat tolerance has not been investigated to the same extent. Most of the tolerance studies have been done on young men. Some of the studies that have examined different grouping factors suggest that the level of physical fitness, as expressed by maximum oxygen uptake, is correlated with a high thermal tolerance and is probably the factor that most differentiates the heat tolerance of the population (ref. III-15).

RESEARCH NEEDED TO FURTHER DEFINE LIMITS FOR SPACECRAFT

In the space environment, zero g has some effects on heat transfer between man and his environment that deserve investigation. In a null-gravity environment, there is no free convection. To provide equivalent levels of forced convection requires considerable amounts of vehicle power. Empirical measurement should be made of convective heat-transfer coefficients under conditions of low-velocity, forced convection. Under zero-g conditions, sweat does not drip from the body but tends to sheet on the skin. This difference in the behavior of sweat may influence comfort and tolerance and should be investigated. Differences in heat tolerance during contingency conditions need to be determined as a function of the diverse population of people that will be exposed to the space environment.

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IV. PHYSICAL FORCES GENERATING ACCELERATION, VIBRATION, AND IMPACT

By J. M. Waligora

Physical forces act on the body to cause acceleration and displacement of the whole or some part of the body. The extent of and the circumstances of the response of the body to force may result in a range of physiological responses from a level at which no effect can be perceived or measured to a level resulting in massive tissue destruction. This section represents an attempt to differentiate and define the types of forces that may act on the body, to identify the physiologic effects, to present limits for these forces, to present the rationale for these limits, and to identify areas in which additional research is required. For the purposes of this chapter, the forces will be divided into those resulting in sustained linear acceleration, in radial acceleration, in impact, and in vibration.

SUSTAINED LINEAR ACCELERATION

Definition and Description

Sustained linear acceleration can be defined as the response of an object or a body to a net directional force applied over a duration of 0.2 second or more. This response is such that the change of the square of the velocity of the object in the direction of the net force is proportional to the duration of the force.

Linear acceleration occurs whenever there is a change in velocity of the spacecraft (e.g., during spacecraft launches and entries). The nature of the response of the body to linear acceleration depends on a number of characteristics of the force and resultant acceleration.

The magnitude of the force acting on the body and the mass of the body determine the magnitude of the acceleration. The duration of the acceleration is a significant factor in the response of the body, particularly at the shorter duration exposures approaching impact (duration <0.2 second). Also significant are the direction of the force vector, the resultant direction of acceleration with respect to the orientation of the body, and the specific body position. The restraint and support systems for the human body in an accelerating vehicle determine the manner in which forces are transmitted from the vehicle to the body and thereby strongly influence the physiological response to the acceleration.

The terminology used in considerations of physical forces and acceleration is confusing but must be understood to comprehend the material covered in this chapter. The symbol g_o ($g_o \approx g$) represents a unit of acceleration

equal to 980.665 cm/sec^2 and equal to the acceleration resulting from the unopposed force of gravity at sea level. The symbol G is used to denote a nondimensional, relative measure of either acceleration expressed in multiples of g_o or a force field expressed in multiples of the force of gravity. The unit G is used to represent acceleration in describing the physiological stresses in response to acceleration and is sometimes referred to as a unit of physiological acceleration.

When an object is dropped near the surface of the Earth, it accelerates toward the center of the Earth at approximately one g. When the object reaches the surface of the Earth, the force of gravity is opposed by the resistive force of the Earth and the object exists in a one-G environment; i.e., a one-G force field or a one-G gravitational f eld. Similarly, when a man is an occupant of a spacecraft accelerating at 4g, a 4G force field, or acceleration field, exists between the structure of the vehicle supporting the man and the man himself, although there is no relative acceleration between the support structure and the man. When discussing the effect of acceleration on this man, the relative unit of acceleration would be used to say that he is undergoing an acceleration stress of 4G or that he is undergoing a 4G acceleration. Acceleration is a vector quantity, and, in the literature on the effect of acceleration on mammalian subjects, the direction or vector of G. or the G load, is denoted by the North Atlantic Treaty Organization Advisory Group for Aeronautical Research and Development (AGARD) convention shown in figure IV-1.

The symbols G_X, G_Y, and G_Z are used as units to express inertial resultants to whole-body linear acceleration in the three orthogonal axes in multiples of the magnitude of the acceleration of gravity g. In this usage, G is the inertial force of the body resisting acceleration and the G vector is in the direction opposite that of the vehicle acceleration. The symbols R_X, R_Y, and R_Z denote angular acceleration about the three orthogonal axes as discussed in the section entitled "Angular Acceleration."

Physiological Effects

The physiological response to sustained linear acceleration is a response to the forces and pressures acting on the body to cause the acceleration. On the surface of Earth, the force of gravity acts on every element of body mass. This force is directional toward the center of the Earth. When a body is stationary on the surface of the Earth, the force of gravity is countered by an equal force vector in the opposite direction. This force is effective at the points of contact of the body with the Earth but must be transmitted to each element of body mass if the acceleration of that element is to be prevented. The structure of the body has evolved under one-G conditions and is adapted to transfer the forces supporting the body (resisting acceleration to the center of the Earth) for those body positions that are normally assumed on Earth. When a space vehicle is accelerated at a given rate, the forces generating the acceleration must be transmitted from the rocket engines to all mass elements of the vehicle and to all mass elements of the passengers. The physiological effect of acceleration occurs when the structure of the body is not capable of transmitting applied forces to each element of the body with

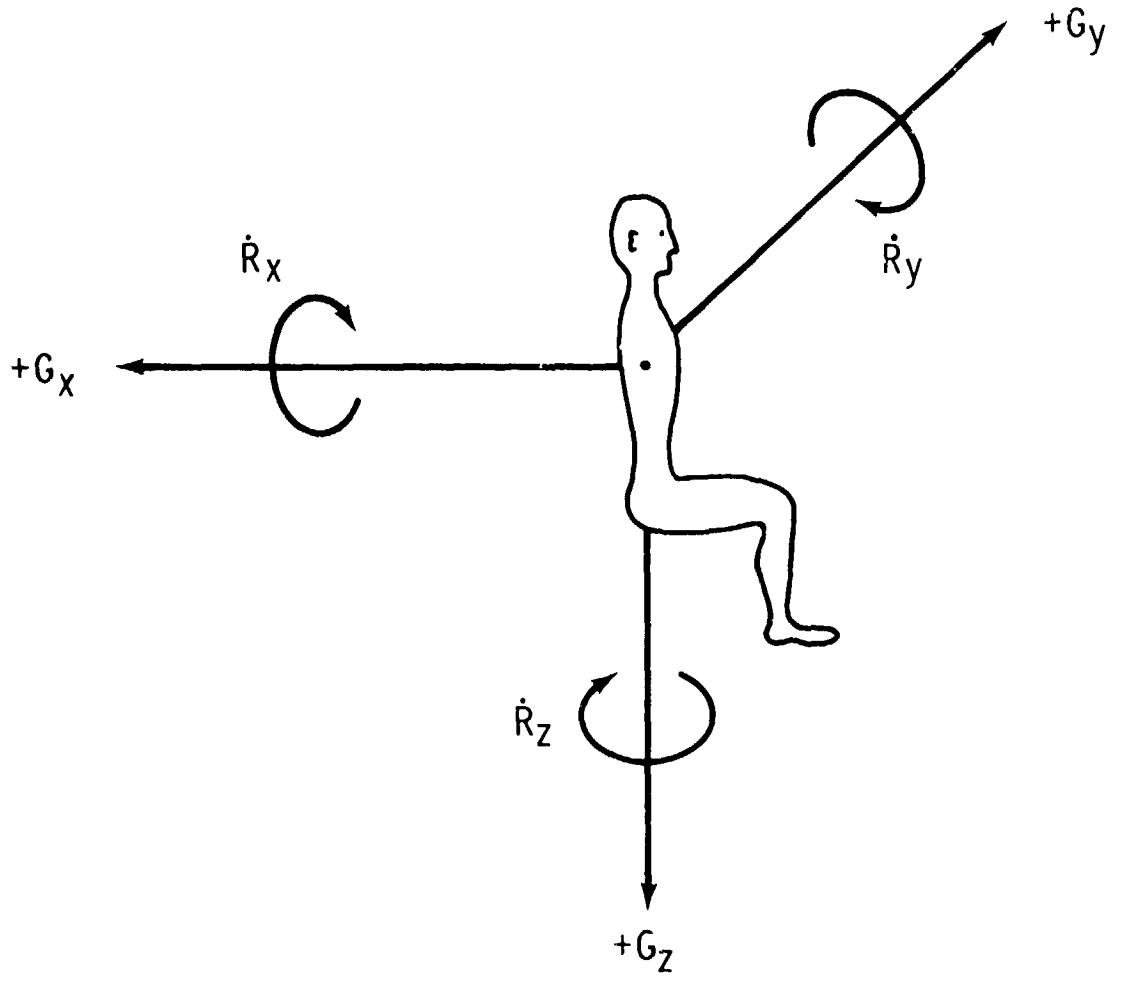


Figure IV-1.- AGARD physiological acceleration system (ref. IV-1). See text for definition of symbols.

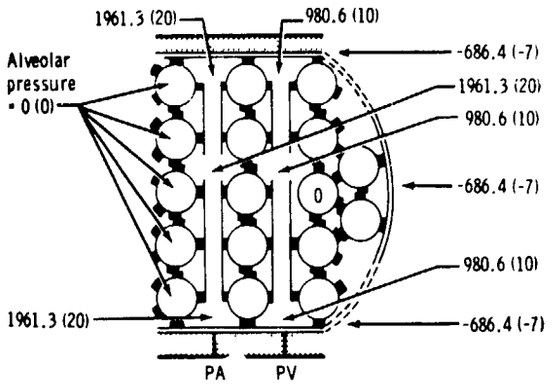
the result that relative movement of body elements occurs and relative pressures within the body are altered. These effects will be considered in the following paragraphs for different axes of acceleration as defined in figure IV-1 and for the physiological systems most limiting in each axis.

The cardiovascular effects of acceleration are generally the most significant. The force causing the acceleration of the body must be transmitted to the blood. This transmittal of force is accomplished by the following sequence of events. Blood is displaced in the direction opposite the accelerative force. This displacement is resisted by the elastic tissues of the body so that a pressure gradient is created in the vector opposite that of the force causing the acceleration. Engorgement and pooling of blood occurs in the areas of the body that experience higher than normal blood pressure. Diminished blood flow rates and poor oxygenation of tissues results in those areas of the body that experience lower than normal blood pressure. The greatest changes in cardiovascular pressures occur when the acceleration vector is parallel to the course of the major blood vessels of the body (G_z). These effects are most severe when these pressures displace blood from the head ($+G_z$). Progressively reduced blood pressure in the head will result in loss of visual acuity, grayout, blackout, and finally unconsciousness as the oxygenation of the eyes and the brain is progressively reduced.

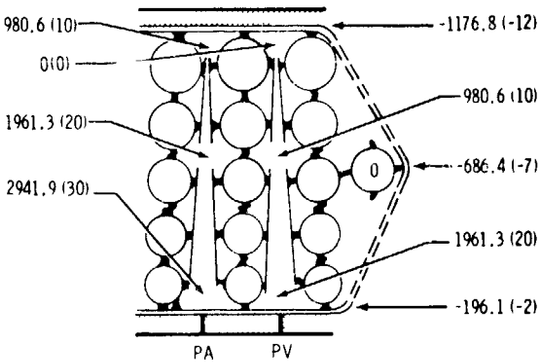
The respiratory effects of acceleration result from a change in relationship of pressure differentials, which, in a one-G environment, facilitate respiration and gas exchange. In a supine subject in a one-G environment (where the force of gravity is effective in the $+G_x$ orientation), there is normally a gradient in intrapleural pressure and in both arterial and venous pressures from dorsal to ventral chest walls (fig. IV-2). These gradients do not interfere with inflation and deflation of the lungs or with perfusion. However, under severe G loading, the pressure differentials at the intrapleural interface resulting from the acceleration become more significant. Modification of intrapleural pressure by exertion of respiratory musculature in the chest wall becomes insufficient to inflate the alveoli near the dorsal chest wall or to empty the alveoli near the ventral chest wall. In addition, the pressure gradients in the blood vessels reduce or eliminate perfusion of the alveoli near the ventral chest wall. Respiration is affected by acceleration in all axes but is affected most significantly by $+G_z$ acceleration. Respiratory effects are more likely to be the limiting factor in $+G_x$ acceleration, however, because there is greater tolerance to cardiovascular effects in that vector.

There are direct effects of forces due to acceleration on all the body organs and systems as well as effects secondary to the hypoxia that may result from the cardiovascular and respiratory alterations and to hormonal response to the general body stress. Figure IV-3 depicts some of the interrelationships that may occur at different functional levels.

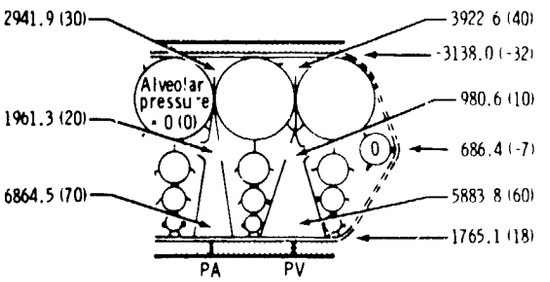
Table IV-1 contains a listing of subjective effects of sustained acceleration in each of the possible vectors. These subjective effects derive from the physiologic effects that have been described previously and bear on the tolerance limits that will be discussed in the following paragraphs.



(a) Null gravity.



(b) Normal gravity.



(c) Acceleration of 5G.

Figure IV-2.- Influence of $+G_x$ accelerative stress on intraperitoneal pressure (ref. IV-2). The dorsoventral dimension of the lung is 20 centimeters; the ventral chest wall is at the top and the dorsal chest wall at the bottom. The single zeros denote atmospheric pressure in the central portion of the thorax in the plane of the heart. The values represent positive and negative intrapleural pressures (with directional arrows) and positive pulmonary pressures (i.e., PA = arterial pressure and PV = venous pressure) expressed in newtons per square meter (centimeters of water).

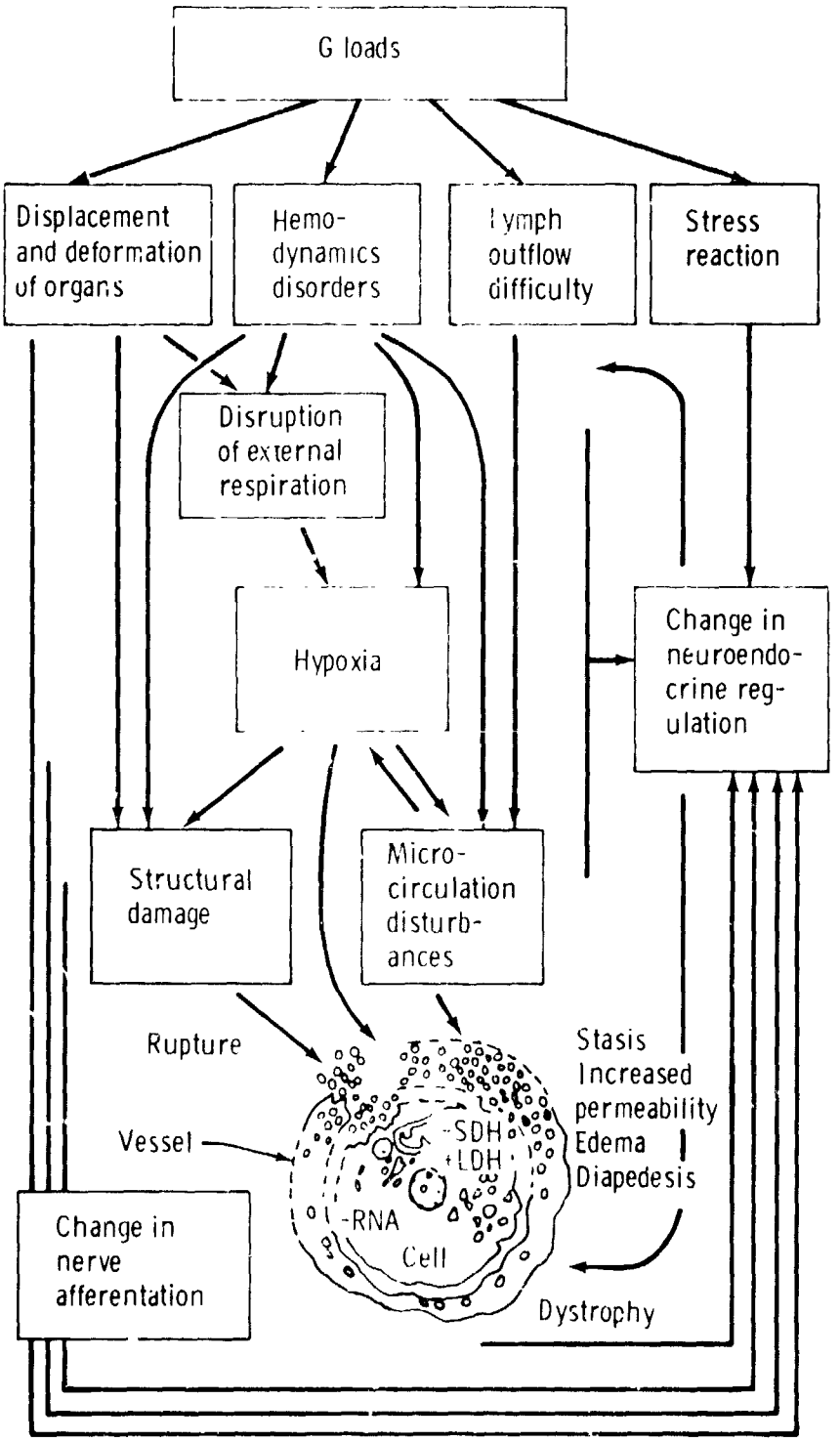


Figure IV-3.- Basic mechanisms of accelerative action on an organism (ref. IV-3). Cell changes consist of increased activity of lactate dehydrogenase (+LDH), reduced activity of succinate dehydrogenase (-SDH), and reduced quantity of ribonucleic acid (-RNA) in cytoplasm.

TABLE IV-1.- SUBJECTIVE EFFECTS OF ACCELERATION

[From ref. IV-4]

Magnitude, G	Effects
	Positive acceleration ($+G_z$)
1	Equivalent to the erect or seated terrestrial posture.
2	Increase in weight, increased pressure on buttocks, drooping of face and soft body tissues.
2.5	Difficult to raise oneself.
3 to 4	Impossible to raise oneself, difficult to raise arms and legs, movement at right angles impossible; progressive dimming of vision after 3 to 4 sec, progressing to tunneling of vision.
4.5 to 6	Diminution of vision, progressive to blackout after approximately 5 sec; hearing and then consciousness lost if exposure continued; mild to severe convulsions in about 50 percent of subjects during or following unconsciousness, frequently with bizarre dreams; occasionally paresthesias, confused states and, rarely, gustatory sensations; no incontinence; pain not common, but tension and congestion of lower limbs with cramps and tingling; inspiration difficult; loss of orientation for time and space as long as 10 sec after acceleration.
	Negative acceleration ($-G_z$)
1	Unpleasant but tolerable facial suffusion and congestion.
2 to 3	Severe facial congestion, throbbing headache; progressive blurring, graying, or occasionally reddening of vision after 5 sec; congestion disappears slowly, may leave petechial hemorrhages, edematous eyelids.
5	Limit of tolerance, 5 sec; rarely reached by most subjects.

TABLE IV-1.- Continued

Magnitude, G	Effects
	Forward acceleration ($+G_x$)
2 to 3	Increased weight and abdominal pressure; progressive slight difficulty in focusing and slight spatial disorientation, each subsiding with experience; $2G_x$ tolerable at least to 24 hr, $4G_x$ for at least 60 min.
3 to 6	Progressive tightness in chest ($6G_x$, 5 min), chest pain, loss of peripheral vision; difficulty in breathing and speaking; blurring of vision; effort required to maintain focus.
6 to 9	Increased chest pain and pressure; breathing difficult, with shallow respiration from position of nearly full inspiration; further reduction in peripheral vision, increased blurring, occasional tunneling, great concentration to maintain focus; occasional lacrimation; body, legs, and arms cannot be lifted at $8G_x$; head cannot be lifted at $9G_x$.
9 to 12	Breathing difficulty severe; increased chest pain; marked fatigue; loss of peripheral vision, diminution of central acuity; lacrimation.
15	Extreme difficulty in breathing and speaking; severe vise-like chest pain; loss of tactile sensation; recurrent complete loss of vision.
	Backward acceleration ($-G_x$)
--	Similar to effects of $+G_x$ acceleration with modifications produced by reversal of force vector. Chest pressure reversed, hence breathing easier; pain and discomfort from outward pressure toward restraint harness manifest at approximately $-8G_x$; with forward head tilt, cerebral hemodynamic effects manifest akin to $-G_z$; distortion of vision at $-6G_x$ to $-8G_x$; feeling of insecurity from pressure against restraint.

C-2

TABLE IV-1.- Concluded

Magnitude, G	Effects
	Lateral acceleration ($\pm G_y$)[a]
3	Discomfort after 10 sec; pressure on restraint system, feeling of supporting entire weight on clavicle; inertial movement of hips and legs, yawing and rotation of head toward shoulder; petechiae and bruising; engorgement of dependent elbow with pain.
5	External hemorrhage; severe postrun headache (14.5-sec duration).

[a]Little information available.

Physiological Limits

The accelerations that occur or are predicted during space flight can be quantified. However, the concentration of force and the relative displacement within the body which are the causative agents of the physiological effects are very difficult to quantify. The physiological limits are therefore most easily stated in terms of acceleration. Different empirically defined limits can be shown to apply under a variety of conditions of body support and restraint (fig. IV-4). The effects of these supports are to distribute the force over the body and to restrict deformation and changes in volume of body segments. The most effective restraint is immersion in water. When the body is immersed in water, it becomes part of the liquid system in which it is immersed and there is good transfer of force without deformation. The air cavities in the body do allow some deformation that becomes limiting at higher G levels. Tolerance varies as a function of acceleration vector as shown in figure IV-5. During practical space operations, it is unlikely that an acceleration will be restricted to one vector. Since reduced tissue oxygenation and reduced respiratory ventilation are both likely to be effects of high levels of acceleration, it is not surprising that pressure breathing and enriched-oxygen breathing mixtures have been shown to increase tolerance to acceleration. Figure IV-6 shows the upper limit of tolerance of a group of highly motivated test pilots suitably restrained without water immersion. It can be compared to the average tolerance values depicted in figure IV-4. The tolerance limits, therefore, must be stated in terms of the acceleration vector and in terms of the specific conditions present during the acceleration. The heavy solid line in figure IV-5 is a plot of required acceleration to achieve Earth orbit against the required duration of that acceleration. Plotted on the same graph are some nominal limits for acceleration. This plot illustrates the physiological trade-off that must be made between short-duration, high-G loads and much longer duration, lower G loads.

Because overall human tolerance to sustained linear acceleration in the transverse ($+G_x$) axis is effectively twice that in the vertical ($+G_z$) axis, all manned space vehicle launches and entries before the Space Shuttle have oriented thrust near the $+G_x$ axis. Maximal peak $+G_x$ forces for the Apollo spacecraft reached approximately 6g on entry with lesser values for launch and orbital maneuvers. Mercury and Gemini spacecraft operated at slightly higher values (fig. IV-7). No acute operational problems, significant physiological deficits, or clinical sequelae related to the cardiovascular and musculoskeletal systems are known to have resulted.

The Space Shuttle vehicle will impose a quite different acceleration environment on the crew. The G loads will be lower but will have a longer duration (fig. IV-8). Visibility requirements during landing necessitate an orientation of the crew couches that results in an acceleration during entry that is primarily in the $+G_z$ vector. An anti-G garment covering the legs and lower torso is being made available for use during Space Shuttle Orbiter entries to reduce the effects of this acceleration.

Acceptable limits of acceleration for normal, healthy adults are in the range of 8G to 10G in the positive x-axis and 3G to 5G in the positive z-axis, depending on individual tolerance, required functions and performance, and

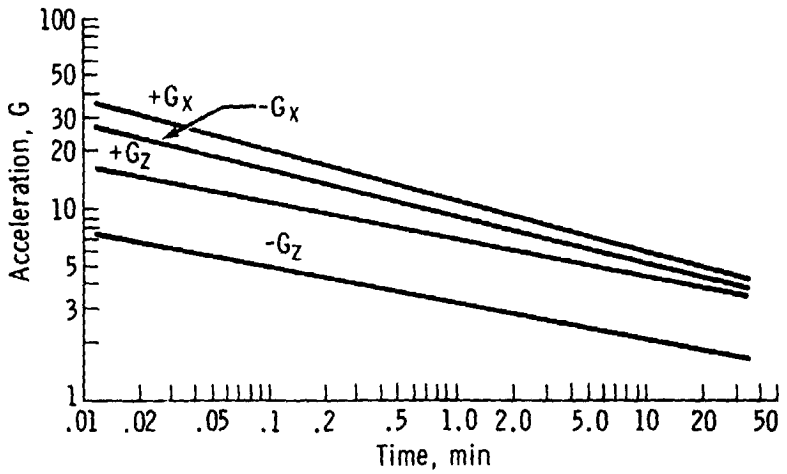


Figure IV-4.- Comparison of average G tolerance in four vectors of sustained linear acceleration (ref. IV-5).

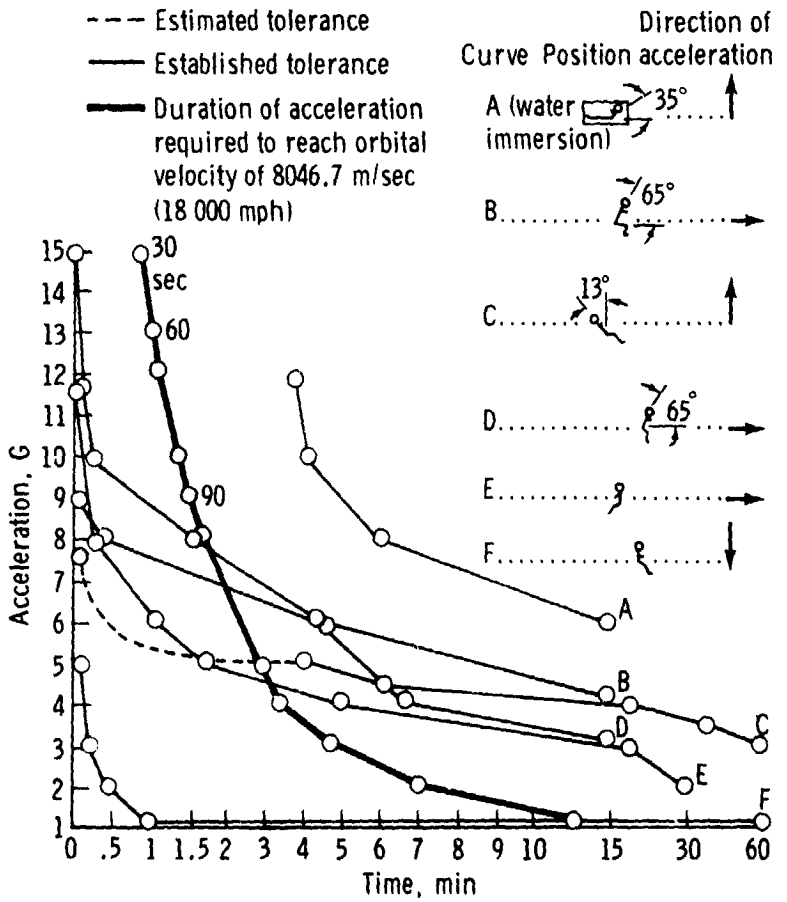


Figure IV-5.- Effect of body position and posture on tolerance to acceleration (ref. IV-6). The time scale (abscissa) is linear but nonproportional.

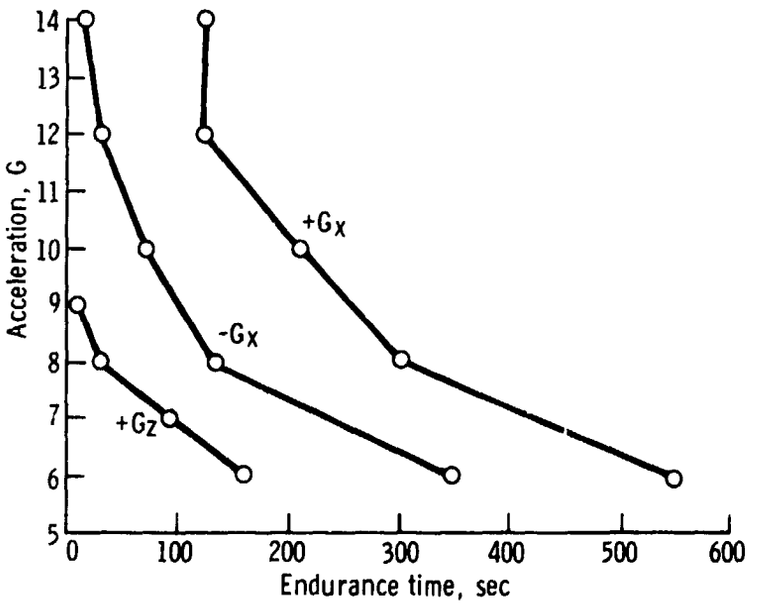


Figure IV-6.- Voluntary endurance of acceleration by highly motivated test pilots (ref. IV-7).

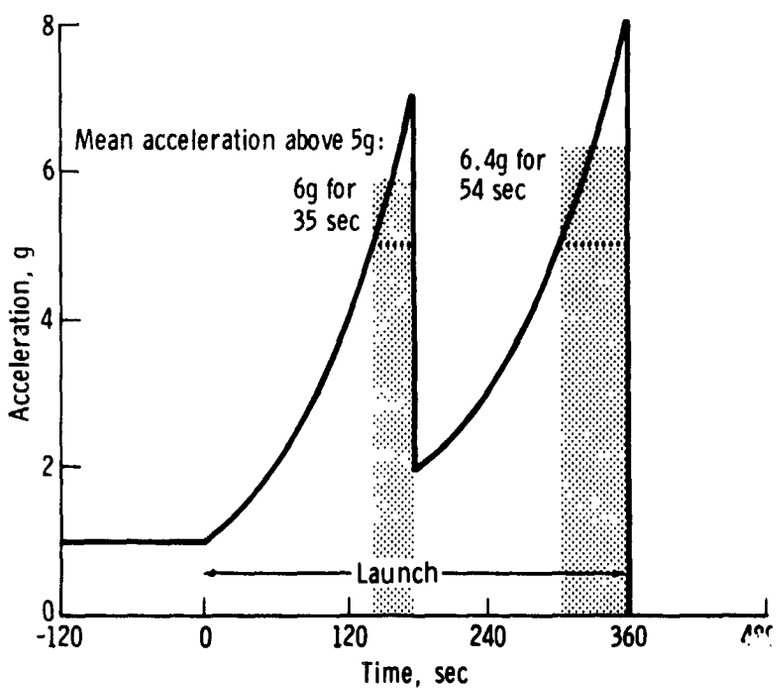
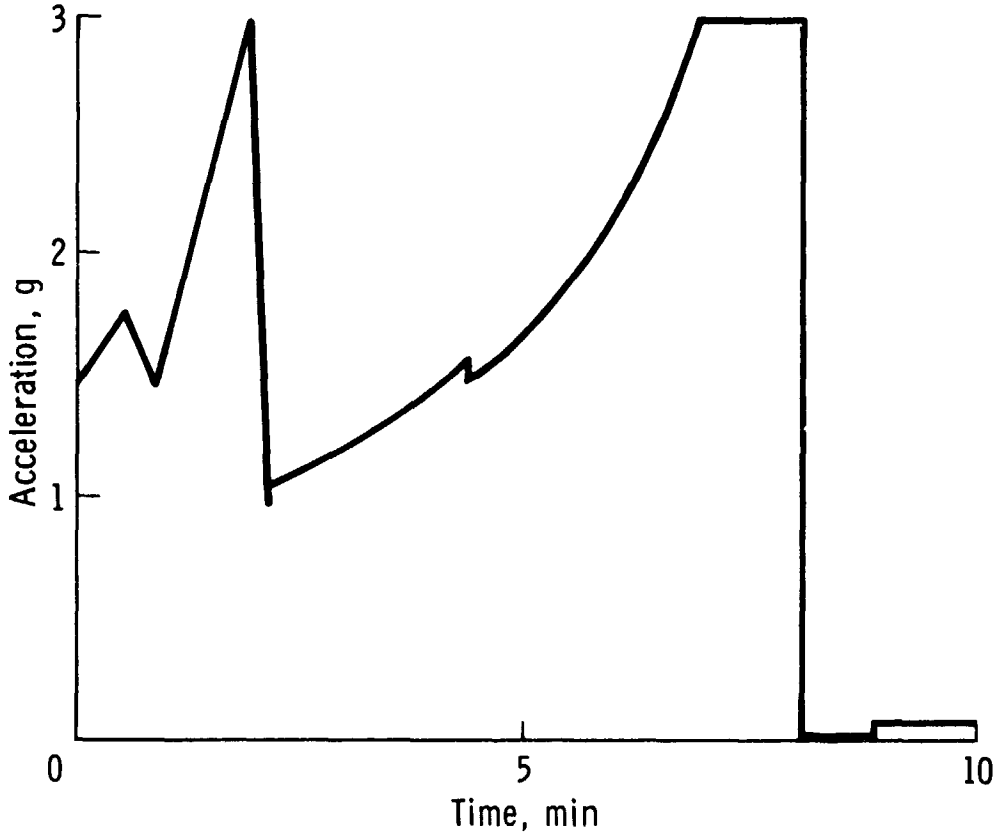
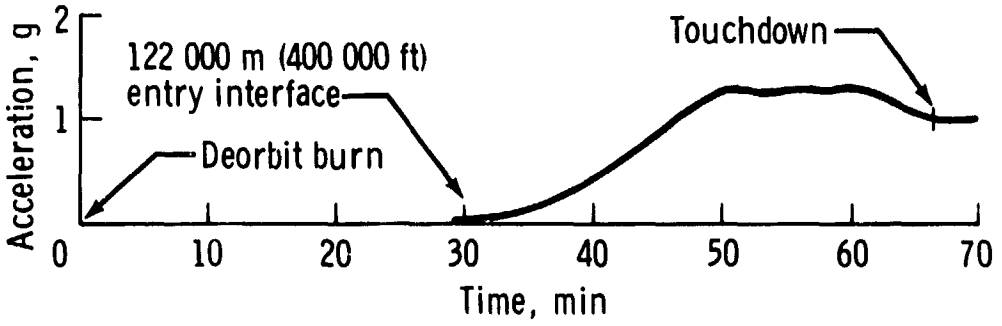


Figure IV-7.- Acceleration profile of launch phase of the manned Mercury-Atlas 6 orbital flight. Stippled areas show periods of acceleration greater than 5g.



(a) Launch.



(b) Entry and landing.

Figure IV-8.- Acceleration profiles of the Space Shuttle vehicle as a function of time.

use of restraining devices, and assuming relatively slow rise gradients and finite durations. The time magnitude integral is very important to overall tolerance.

Interacting Environmental Parameters

Since the physiological effects of an acceleration force field are many, the potential for modification of these effects by a number of environmental factors should be considered. As stated previously, the primary limiting effects of high gravitational forces are a loss of oxygenation due to effects on the cardiovascular and the respiratory systems. Oxygen pressure is, therefore, a very important interacting variable. Temperature can be expected to interact when it results in vasodilatation and decreased cardiac return. Any other environmental factor that might affect the cardiovascular or respiratory system would be expected to influence acceleration tolerance.

Effect of Individual Variation

Figures IV-4 (average tolerance curves) and IV-6 (maximum tolerance for test pilots) provide some indication of the effect of individual variation on tolerance to a force field. However, in Space Shuttle missions and in other future space missions, a much more diverse population may be exposed to force fields other than that due to gravity at sea level.

Research Needed to Further Define Spacecraft Limits

Research is needed to provide the following.

1. Definition of the effects of various durations in a null-gravity environment on subsequent tolerance to force fields in all axes

2. Definition of the range of acceleration forces resulting in physiological effect and of tolerance in the population that may fly in space

3. Optimization of countermeasures that may be used under high-force-field conditions

ANGULAR ACCELERATION

Definition and Description

Angular acceleration can be defined as the response of an object or a body to a combination of force vectors that result in a change in direction of motion of the object. Radial acceleration is angular acceleration that occurs during circular motion with the axis of rotation either within the body or outside the body. Rotary acceleration is radial acceleration for which the axis of rotation is within the body subjected to the acceleration.

The convention for describing orientations of angular acceleration is given in figure IV-1. The symbol $\pm\dot{R}_y$ refers to pitch, tumbling, or rotation around the y-axis; $\pm\dot{R}_x$ refers to rolling, spin, or rotation around the x-axis; and $\pm\dot{R}_z$ refers to yaw, yaw spin, or rotation around the z-axis.

Physiological Effects

The physiological effects of the forces resulting in radial acceleration are in part similar to the effects of the forces resulting in linear acceleration. The forces acting on the body are a function of the radius of rotation and the velocity. As the axis of rotation increases in length relative to the size of the body, the physiological effects of the radial acceleration approach the effects of linear acceleration and the same types of cardiovascular and respiratory effects occur. As the radius of rotation being considered approaches the dimensions of a man, forces act in different directions in different parts of the body, and, since the force increases as the square of the distance from the center of rotation, the cardiovascular and respiratory responses are complicated. The $\pm\dot{R}_x$ rotation around an axis passing through the heart produces pooling of blood in the feet similar to $+G_z$ acceleration and at the same time produces an increased hydrostatic pressure in the head similar to $-G_z$ linear acceleration. Movement of the center of rotation toward the feet increases the $-G_z$ effects. Conversely, movement of the center of rotation toward the head increases the $+G_z$ effects. The same types of considerations apply to $\pm\dot{R}_y$ and $\pm\dot{R}_z$ acceleration.

The other major effect of radial acceleration is on the vestibular system. The discussion of these vestibular effects will be very brief in this section, and the reader is referred to reference IV-8 for more detailed discussion. The vestibular apparatus is made up of three semicircular canals and two otolith organs. These sensory organs provide information that aids in orientation and in eye, head, and body coordination. The otolith organs, the utricle and the saccule, act as detectors for linear acceleration, whereas the semicircular canals operate as detectors of angular acceleration. The vestibular system evolved under normal-gravity, terrestrial conditions, in which acceleration is limited by the normal self-induced motions of a man. When the vestibular system is exposed to much higher accelerations and combinations of forces, the effect can be motion sickness and/or sensory illusions resulting in disorientation. These effects are likely to be limiting factors, at least in terms of performance, during rotational acceleration and in short-radius radial acceleration. If radial acceleration were used to generate an artificial gravity in a future space station, vestibular disturbances (i.e., motion sickness and disorientation) would be a prime determinant of the minimum acceptable radius. The stimulus of the vestibular system in the absence of gravity in the Apollo and Skylab missions has been shown to produce a sequela of motion sickness for 3 to 5 days in some crewmen followed by a period of insensitivity to motion sickness in response to provocative tests (ref. IV-9).

Physiological Limits

Most subjects without prior experience can tolerate rotation rates as large as 0.63 rad/sec (6 rpm) in any axis or combination of axes. Most subjects cannot initially tolerate rotation rates in the region of 1.26 to 3.14 rad/sec (12 to 30 rpm) and rapidly become sick and disoriented above 0.63 rad/sec (6 rpm) unless carefully prepared by a graduated program of exposure (ref. IV-10). On the other hand, rotation rates of 6.28 rad/sec (60 rpm) for as long as 3 or 4 minutes around the y-axis (pitch) and around the z-axis (spin) have been described by subjects as being not only tolerable but pleasant (ref. IV-11). Intolerability becomes manifest again at approximately 8.4 rad/sec (80 rpm) in the pitch mode and at approximately 9.4 to 10.5 rad/sec (90 to 100 rpm) in the spin mode. In the pitch axis, with the center of rotation at the heart level, symptoms of negative acceleration ($-G_z$) are demonstrated at approximately 8.4 rad/sec (80 rpm) and are tolerable for only a few seconds. Some effects of positive acceleration ($+G_z$), namely numbness and pressure in the legs, are also observed but develop slowly, with pain being evident at approximately 9.4 rad/sec (90 rpm). No confusion or loss of consciousness is found, but, in some subjects, disorientation, headache, nausea, or mental depression are noted for several minutes after a few minutes of exposure (ref. IV-11). With rotation in the spin mode, when the head and trunk are inclined forward out of the z-axis, rotation approaches tolerance limits at 6.28 rad/sec (60 rpm) for 4 minutes, although some motivated subjects have endured 9.42 rad/sec (90 rpm) in the same mode (ref. IV-12). Except for unduly susceptible subjects, tolerance tends to improve with frequency of exposure. Long-duration runs in the pitch mode have been endured for as long as approximately 60 minutes at 0.63 rad/sec (6 rpm) in selected subjects (ref. IV-10).

These extreme limits are not likely to be a consideration in design of spacecraft. They may set limits to survivability in contingency situations involving failure of spacecraft orientation control systems.

In considering physiological limits to angular acceleration with long-radius components (such as high-speed aircraft turns), it is appropriate to use the acceleration limits defined for linear accelerations.

Interacting Environmental Parameters

The most significant interacting variable is the presence or absence of gravitational forces, particularly where the vestibular effects of radial acceleration are considered. Those environmental parameters previously mentioned which interact with linear acceleration also interact with radial acceleration.

Effect of Individual Variation

The space motion sickness syndrome that was noted in the Apollo and Skylab flights was not present in all crewmen. The resistance of individuals to this syndrome during flight has not been demonstrated to be related to the susceptibility of the crewmen to motion sickness produced by one-G provocative tests.

Research Needed to Further Define Spacecraft Limits

Research is needed to define the mechanism of action of the motion sickness syndrome under zero-G conditions and to establish preventative and/or ameliorative measures (ref. IV-8). Research is needed to define the effect of various durations of zero-G exposure on subsequent tolerance to angular accelerations in all axes.

IMPACT

Definition and Description

For the purposes of this section, impact will be defined as an acceleration of less than 0.2 second duration. In discussing impact, the same terminology and coordinates will be used as those in the section on linear acceleration. There are, however, some significant differences between impact and linear accelerations. During linear acceleration, all parts of the body ultimately experience the same acceleration. The force causing the acceleration is ultimately effective on all parts of the body, even though there may be some deformation of the body in the process. As a result of the elastic, viscous, and plastic relationship between each of the body elements, each element of the body during impact experiences a different G load. Loads may be stated as response loads of particular elements of the body or as input loads; i.e., the load on a rigid container or support system containing a man. In linear acceleration, the function of the support systems is to evenly transfer the force causing the acceleration to all parts of the body. The support system cannot reduce the acceleration level. In contrast, impact support systems can attenuate forces and greatly reduce the acceleration experienced by the crewman.

Physiological Effects

Information on the physiological effects of impact has been obtained by exposing men to impact under controlled conditions up to a voluntary tolerance level and by analysis of accidental impacts encountered in free falls and other types of accidents. The effects of impact on various physiological systems during voluntary tests are summarized in table IV-2 (ref. IV-13).

In an accidental impact, pathological damage injury to the head is the most frequent and severe manifestation (ref. IV-32). Injuries to the head

TABLE IV-2.- OBSERVED PHYSIOLOGICAL EFFECTS OF IMPACT

[Data from refs. IV-14 to IV-31]

Effects	Impact force parameters	Responses
Bradycardia	5 to 15 $+G_x$	Slowing of heart rate for at least 5 beats.
	15 to 30 $\pm G_x$; 9 to 12 $+G_y$	Slowing of heart rate immediately following impact; slowing is increased at higher accelerations. Elimination of bradycardia by administration of 1.6 mg atropine indicates relationship to vagal reflex.
Shock	>15 $\pm G_x$, 500G/sec	Brief disorientation; drop in systolic/diastolic blood pressure to 12/8 kN/m^2 (90/60 torr) 15 to 30 sec after impact; electrocardiograph nodal rhythm.
	12 $+G_y$	Faint, pallor.
Muscular	>26 $-G_x$, 850G/sec, 0.002 sec	Chest pains, aches in back and neck muscles; stiff neck 1 to 3 days.
Skeletal	>16 $+G_z$, 1160G/sec	Anterior lip vertebral compression fracture; most observed injury first lumbar to seventh thoracic vertebrae.
	>16 $-G_x$, 0.01 to 0.10 sec	Fracture of lumbar vertebrae.
	$\leq$83 $+G_x$, 3800G/sec, 0.04 sec	None.

TABLE IV-2.- Concluded

Effects	Impact force parameters	Responses
Neurological	15 $\pm G_x$	Increased deep tendon reflexes.
	>20 $-G_x$, 400G/sec and 800G/sec	Appear stunned 10 to 15 sec at 20G peak accelerations. Euphoria; hand tremor; decreased coordination; loquacity; increased muscle tone; gross involuntary movements in head, arms, and trunk.
	25 $\pm G_x$	Deep tendon reflexes absent for several seconds, then hyperactive for about 1 min.
	>25 $-G_x$, 1000G/sec	Abnormally slow electroencephalograph wave patterns observed for several minutes after impact.
Hematological	20 $-G_x$, 400G/sec or 800G/sec	Blood thrombocytes reduced 1 hr after impact. A week later, thrombocyte count higher than control value.
Psychological	10 to 25 $+G_x$	Kohn symbol arrangement test shows distinctive changes, increasing with force level.
General stress	>20 $+G_x$	Chemical changes in adrenal blood; alterations in adrenal gland activity; 17-hydroxycorticosteroid excretion levels increase significantly and are related to anxiety and central nervous system stimulation of adrenocortical secretion.

area include whiplash, neck fracture, deformation of the skull, and shear strains through the brain (ref. IV-33). Damage from impact forces to other areas of the body includes bruises, tissue crushing, bone fracture, rupture of membranes or organ capsules, and damage to vertebrae (refs. IV-34 and IV-35).

Physiological Tolerance

Figures IV-9 and IV-10 indicate tolerance to $\pm G_z$ impact and $\pm G_x$ impact, respectively, while the subjects are optimally supported. Figure IV-11 is a chart of impact experience during accidents.

Interacting Environmental Parameters

Zero-G exposure may produce physiological effects that would ultimately influence tolerance to impact. A progressive loss of calcium and other bone minerals in a zero-G environment has been noted (ref. IV-38). If a progressive loss of the structural strength of bones is encountered in long flights, tolerance of the bones to impact will be reduced. A reduction of muscular strength on a long flight may also influence impact tolerance.

Research Needed to Further Define Spacecraft Limits

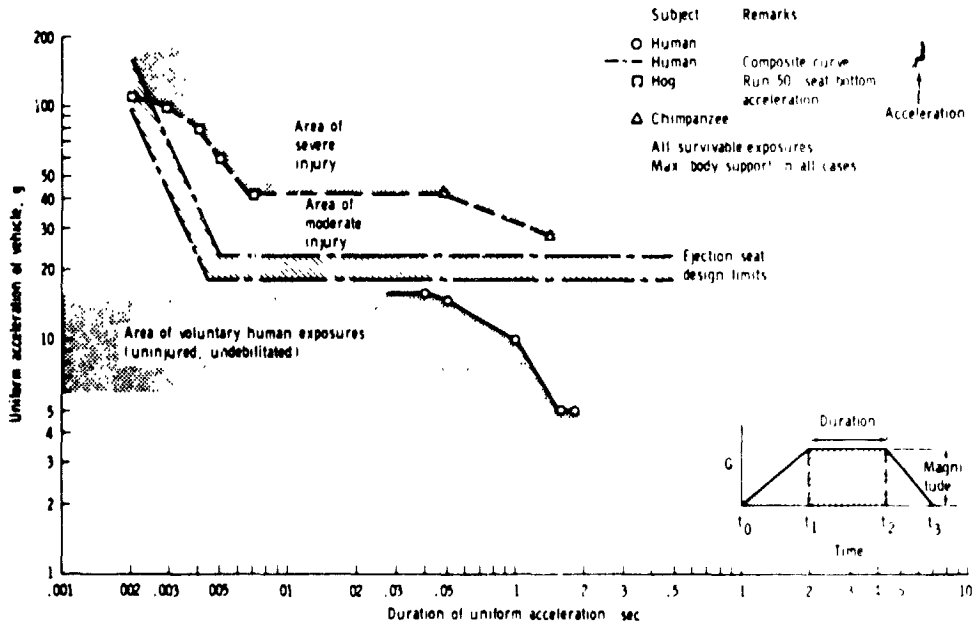
The trends in spacecraft design and design of emergency systems indicate that impact may not be a basic design consideration even for emergency systems. The effect of zero-G exposure on mineral loss from bones would have significance in addition to considerations of impact tolerance.

VIBRATION

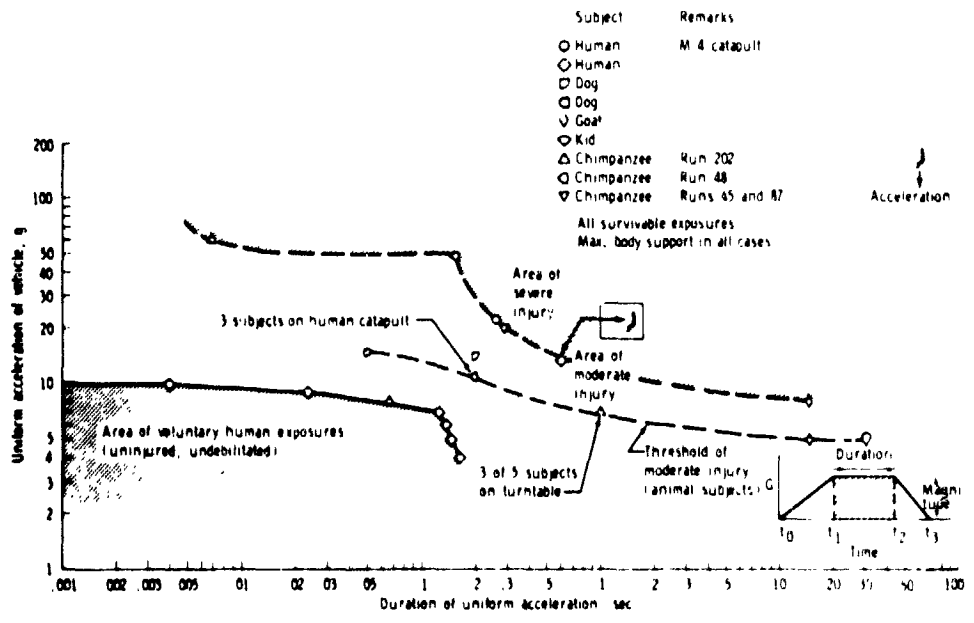
Definition and Description

Vibration is the periodic motion of the particle of an elastic body or medium in alternately opposite directions from the position of equilibrium when that equilibrium has been disturbed. Vibration may occur in fluids or solids. Vibration in fluids is generally discussed as noise. Although the effects of the two types of vibration can be similar, this section will deal only with vibration transmitted to the body through solids. The parameters that describe and differentiate vibrations are frequency, amplitude, velocity (a function of frequency and amplitude), acceleration, and jolt (rate of change of acceleration). The most important range for physi logical effects is 1 to 60 hertz; vibration outside that range is not usually a problem because higher frequencies are damped by body impedance, and frequencies below 1 hertz are not perceived by the body as vibration.

The effects of vibration are accentuated when the vibration occurs at the resonant frequency of the system. The many different couplings of elas-

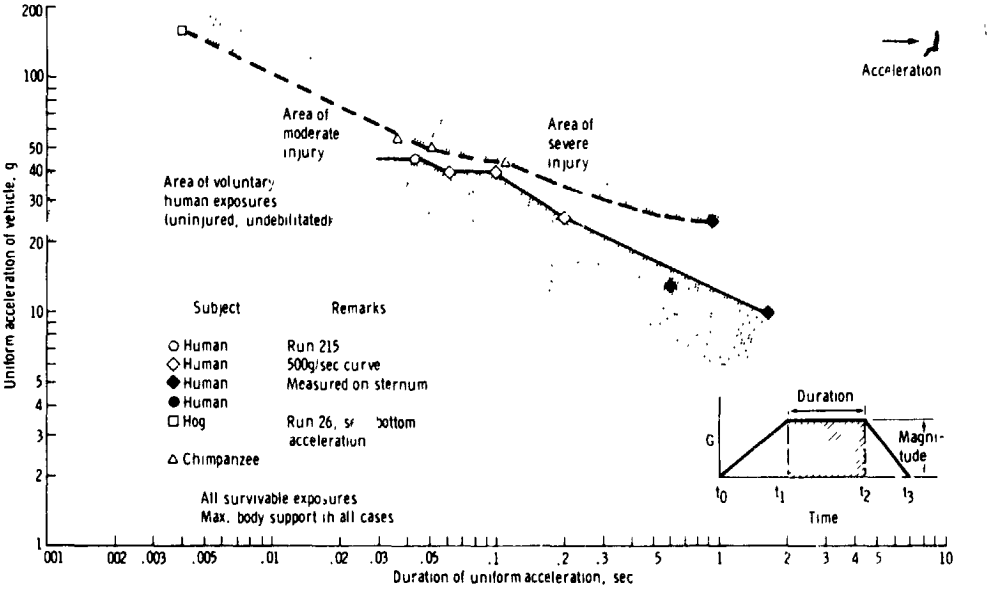


(a) $+G_z$ (tailward) acceleration.

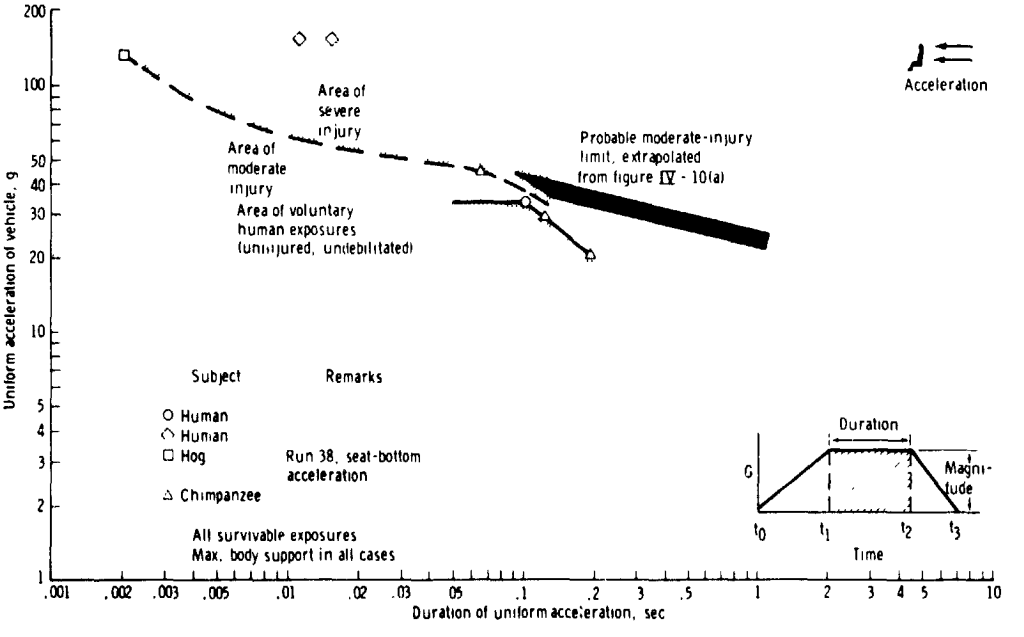


(b) $-G_z$ (headward) acceleration.

Figure IV-9.- Effects of abrupt longitudinal deceleration on various animals and man. Data from reference IV-36, adapted from A. M. Eiband, "Human Tolerance to Rapidly Applied Accelerations: A Summary of the Literature," NASA Memorandum 5-19-59E, 1959. The acceleration vectors shown on the figures are vehicular.



(a) $-G_x$ (back to chest) acceleration.



(b) $+G_x$ (chest to back) acceleration.

Figure IV-10.- Effects of abrupt transverse deceleration on various animals and man. Data from reference IV-36, adapted from A. M. Eiband, "Human Tolerance to Rapidly Applied Accelerations: A Summary of the Literature," NASA Memorandum 5-19-59E, 1959. The acceleration vectors shown on the figures are vehicular.

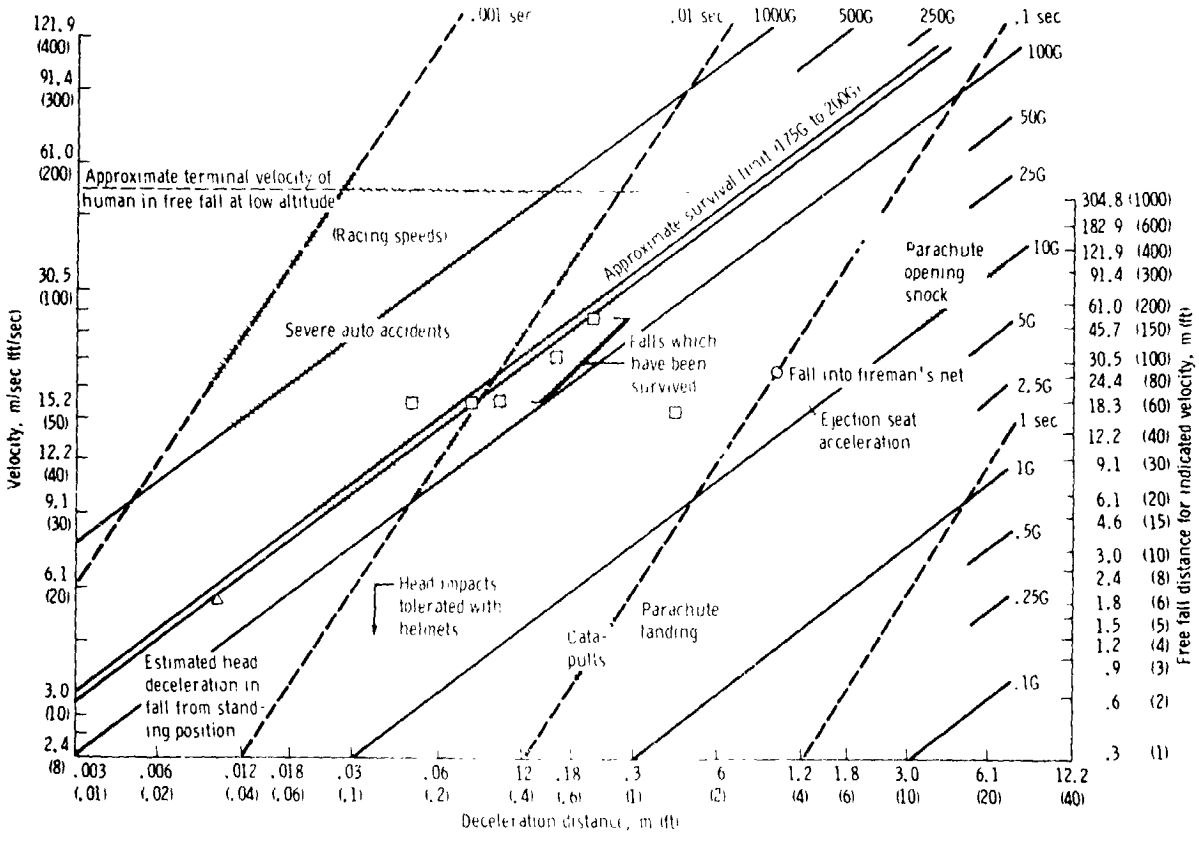


Figure IV-11.- Plot of various types of impact and deceleration experiences on the common axes of deceleration distance and velocity. Stopping time in seconds and impact force in G units are shown as secondary scales. The data points for free falls with survival (open squares) are for falls of 15 to 46 meters (50 to 150 feet). The free-fall-distance scale is calibrated with allowance for air resistance of the human body near sea level. The line labeled "approximate survival limit" must be used with caution, since many biophysical factors influence injuries caused by deceleration. From reference IV-36, using data of reference IV-37.

tic elements in the body give rise to many resonant frequencies. For this reason, the site of major concern and the mechanism of damage to the body vary as a function of frequency.

Physiological Effects

The physiological effects of vibration are summarized in tables IV-3 to IV-5 (ref. IV-39). Terminology for the vibration environment is generally consistent with that described previously in this chapter. However, in discussing the physiological effect of vibration, it is necessary to identify simultaneous values of bias or steady-state acceleration and of the oscillatory acceleration of vibration. An uppercase G is used as the nondimensional unit identified with bias or steady-state acceleration, whereas a lowercase g is used as the nondimensional unit identified with oscillatory acceleration. The vector coordinates remain those described in figure IV-1. In tables IV-3 to IV-5, G and g units are used to describe completely the vibrational environment for a given situation and vibration amplitude is expressed typically as a root-mean-square (rms) value of g.

Physiological Tolerance

The multitude of possible vibration exposures at different frequencies, amplitudes, and accelerations encountered in different body axes and in combinations with simultaneous environmental stresses from other sources makes it difficult to specify precise and rigid limits for vibration. Where vibration environments are suspected to be close to tolerance limits, all the parameters involved in the exposure must be identified and compared with the most similar exposures reported in the literature. However, a unified criterion for human exposure to vibration has been formulated by the International Organization for Standardization (known by the French abbreviation ISO) (ref. IV-80). This standard is well suited to serve as a preliminary standard for future spacecraft design.

The fatigue/decreased-proficiency boundaries from the ISO standard are presented in figures IV-12(a) and IV-12(b). The symbols a_x, a_y, and a_z correspond to the g units of vibration defined previously, in the respective axes. Values for the exposure limit are obtained by doubling the acceleration; values for the reduced comfort boundary are obtained by dividing the acceleration values by 3.15.

Effect of Individual Variation

Individual variations are significant in tolerance to vibration. Therefore, limits for space flight should be based on the least tolerant individuals that may be included as crewmembers or passengers on future spacecraft. Research should be directed to minimizing the sources of such vibration and to improving attenuation of vibration before it can affect the crewmen.

TABLE IV-3.- SUMMARY OF VIBRATION EFFECTS ON VISUAL PERFORMANCE

Vibration conditions	Visual task	Effect	Source
Subject static; display vibrated at 5 Hz, 0.160 cm (0.063 in.) double amplitude (DA)	Reading speed	Speed reduced by about 5.3 percent.	Reference IV-40
$+1G_x \pm ng_z$; 5.8 and 11 Hz	Visual acuity by detection of break in line	Comparison of man vibrating and display static with both man and display vibrating; both conditions result in decrement; for constant displacement, acuity worst at higher frequencies; for constant acceleration, acuity worst at 5 Hz; addition of bite-bar improved vision at 5 Hz.	Reference IV-41
$+1G_z \pm ng_x$ and $\pm ng_y$; 1.5 to 5.5 Hz; 0.15g to 0.35g	Static display, Landolt C's	No decrement in either axis.	Reference IV-42
1 to 30 Hz in combinations of vertical, pitch, and roll motion	Reading printed words, both static and moving display	Visual acuity worst at 4 to 7 Hz; pitch motion caused greatest decrement, roll motion least decrement.	Reference IV-43
$+1G_x \pm ng_x$, $\pm ng_y$, $\pm ng_z$; 6, 11, and 15 Hz; $\pm 0.9g$ to $\pm 2.0g$	Read easy and difficult aircraft instrument dials	No effect with easy dial; errors related to intensity; helmet restraint improves performance in x-axis; improves at 6 Hz but degrades at 11 and 15 Hz in y-axis; no effect in z-axis.	Reference IV-44
$+1G_x \pm 1.1g_x$; 6, 11, and 15 Hz	Read difficult instrument dials; effect of various types of head restraint	Error greatest at 6 Hz; head damped in x-axis results in error reduction; no consistent effect of z-axis restraint.	Reference IV-45
$+1G_z \pm ng_z$; 8, 13, 11, and 23 Hz at 0.13 and 0.25 cm (0.05 and 0.1 in.) DA and 10, 20, 30, 40, and 50 Hz at 0.13 cm (0.05 in.) DA	Read 3-digit numbers, 0.63 cm (0.25 in.) high	Beginning at 8 Hz, errors increase as a function of frequency; no function of amplitude; errors increase to 40 Hz.	Reference IV-46
$+1G_z \pm ng_z$; 1 to 27 Hz; 4 subjective intensity levels	Digit reading	Greatest error in 12- to 23-Hz range; errors increase with intensity.	Reference IV-47

TABLE IV-3.- Concluded

Vibration conditions	Visual task	Effect	Source
$+1G_z \pm ng_z$; 1 to 27 Hz; 4 subjective intensity levels	Digit reading; visual angle 6' to 24' of arc	Greatest error in 12- to 23-Hz range; errors increase with intensity; error only for 9' and 6' of arc digits.	Reference IV-48
$+1G_z \pm ng_z$	Reading aircraft displays	Collimated, head-up display results in fewer errors.	Reference IV-49
$+1G_z \pm 0.25g_z$: 2.4 to 9.5 Hz	Scan letters "c" and count randomly distributed letters "o"	Decrement worst at 3.4 and 4.8 Hz; completion time worst at 3.4 Hz.	Reference IV-50
$+1G_z \pm ng_z$; 1 to 20 Hz; one-half short-time tolerance limit	Detect diverging lines on static rotating disk	Wide individual differences; greatest decrement at 12 to 18 Hz; error also at 5, 7, and 10 Hz; residual effect following vibration.	Reference IV-51
$+1G_z \pm ng_z$; 5 to 37 Hz; $\pm 0.44g_z$ to $\pm 0.51g_z$ and $0.83g_z$ to $1.02g_z$; 6 Hz at $\pm 0.13g_z$ to $\pm 0.36g_z$; 19 Hz at $\pm 0.16g_z$	Read printed numbers, subtending 4.4' of arc; series of studies to compare subject and display motion influence	Increase in error at 5, 14, and 27 Hz for lower intensity; below 10 Hz, influence of vibrating display greater than vibrating subject; above 10 Hz, the reverse.	Reference IV-52
$+1G_x \pm 1.2g_x$ and $\pm 0.9g_x$; 6, 11, and 15 Hz	Dial reading; effects of helmet and liner configuration	Error worst in x-axis for 11 Hz; no effect of liner in x-axis; in y-axis, performance better with liner at 6 Hz but worse with liner at 15 Hz; total errors higher with helmet than without it.	Reference IV-53
$+1G_z \pm ng_y$; 0- to 52-Hz vibration applied directly to head only	Acuity measured by illumination level required to detect break in line; target static and dynamic base	Acuity decrement above $\pm 0.2g_y$; for constant acceleration of $\pm 1.0g_y$, acuity worst from 22 to 34 Hz; for constant amplitude of 0.03 cm, acuity worst at 30 Hz; decrements due to target vibration are smaller than those due to head motion.	Reference IV-54
Random vibration, helicopter spectrum, to $0.41g_z$ rms ($+1G_z$)	Meter reading, Landolt C's	No effects.	Reference IV-55
Random vibration, low-altitude high-speed flight spectrum, various turbulence levels to $0.405g_z$ rms ($+1G_z$)	Target recognition outside of simulated cockpit	No effects.	Reference IV-56
Random vibration, 2- to 7-Hz band, to $0.30g_z$ rms ($+1G_z$)	Read aircraft instruments	No blurring or eyestrain.	Reference IV-57

TABLE IV-4.- SUMMARY OF EFFECTS OF VIBRATION ON BIODYNAMICS, PSYCHOMOTOR PERFORMANCE, SPEECH, HEARING, AND HIGHER MENTAL PROCESSES

Vibration conditions	Measures	Effect	Source
	Biodynamic mechanisms		
$\pm 0.15 g_z$ to $\pm 0.35 g_z$ at 0.9 to 6.5 Hz, low amplitude	Whole-body vertical vibration, hand tremor, body equilibrium, foot pressure	Foot pressure constancy inpaired at 3.5 to 6.5 Hz; error increased with intensity; no residual effects.	Reference IV-58
$\pm n g_x$, $\pm n g_y$ for 0.5 hr	Body sway equilibrium	No effects.	Reference IV-42
$\pm g_z$, 2 to 20 Hz (intensities = one-third short-term tolerance limits)	Control of pitch and roll of a chair	Wide individual differences; decrement between 3 and 12 Hz, worst at 6 Hz.	Reference IV-59
$\pm g_z$ at 0, 2, 5, and 8 Hz	Orientation (orienting body position to face targets at 15°, 30°, and 60° from reference plane)	Only small decrement in accuracy; mean error <0.5°.	(a)
$\pm 0.03 g_z$ to $\pm 0.41 g_z$ at 0, 3, 5, and 8 Hz	Leg muscular power (on bicycle ergometer)	No effects.	(b)
Various peak-to-peak accelerations at 1 Hz with 3 Hz, and at 2 Hz with 6 Hz	Arm-hand steadiness	Positional errors significantly related to rms amplitude and frequency of vibration; 90 percent of error was periodic; 1 Hz with 3 Hz combination produced larger error; small (0.5g to 1g) differences in acceleration had no effect.	Reference IV-60

[a] M. M. Ayoub: Performance and Recovery Under Prolonged Vibration. Unpublished manuscript, School of Engineering. Texas Technological College (Lubbock, Tex.), ca. 1969.

[b] J. Y. Harrison: The Effect of Vibration on the Ability to Generate Human Muscle Power. Unpublished manuscript submitted to Human Factors, 1969.

TABLE IV-4.- Continued

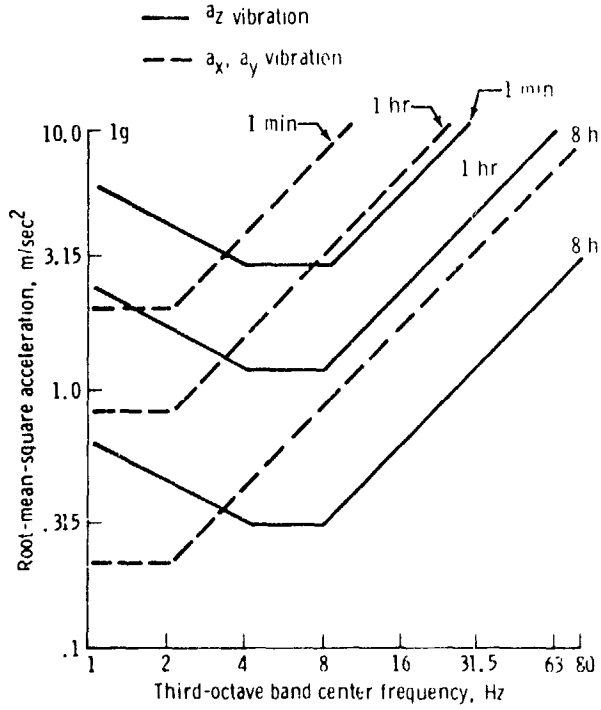
Vibration conditions	Measures	Effect	Source
	Psychomotor performance		
+0.25g_z at 2.4 to 9.5 Hz	Time to pick up markers and place in small circular areas	Completion time worst at 3.4 and 4.8 Hz.	Reference IV-50
+0.5g_z rms at 2 to 30 Hz (13 Hz peak power)	Digital decimal input with push-button, toggle switch, rotary switch, and thumbwheel controls	Accuracy unaffected; insert times increased by 4 percent; push-buttons and toggle switches were most rapidly used, with the former preferred; thumbwheels were most accurate.	Reference IV-61
0g_z, 0.2g_z, 0.4g_z, 0.6g_z, and 0.8g_z rms for 5 min	Same	No effects for 0.2g_z and 0.4g_z rms; significant increase in insert time for 0.6g_z and 0.8g_z rms; *speed:* pushbuttons faster than rotary switches faster than thumbwheels; *error rate:* pushbuttons highest and thumbwheels lowest for high-intensity vibrations.	Reference IV-61
$\pm g_x$ and $\pm g_y$ at 0.33 and 0.80 Hz at amplitude of ±16.0 and ±17.8 cm (±6.3 and ±7.0 in.)	Nut and bolt assembly/disassembly; placement of probe through various size holes	No effects at 0.33 Hz; time required increased by 30 percent at 0.80 Hz with no increase in accuracy.	Reference IV-62
	Speech intelligibility		
$\pm g_z$ at 10, 20, 30, 40, and 50 Hz	Intelligibility	Most effect at 10 and 20 Hz.	Reference IV-63
0.5g_z sinusoidal at 6 Hz; 0.75g_z at 4 and 8 Hz; 1.0g_z at 2 to 20 Hz	Intelligibility and quality	No effect on intelligibility at 65 dB; "quality" poorer than control condition.	Reference IV-64

TABLE IV-4.- Concluded

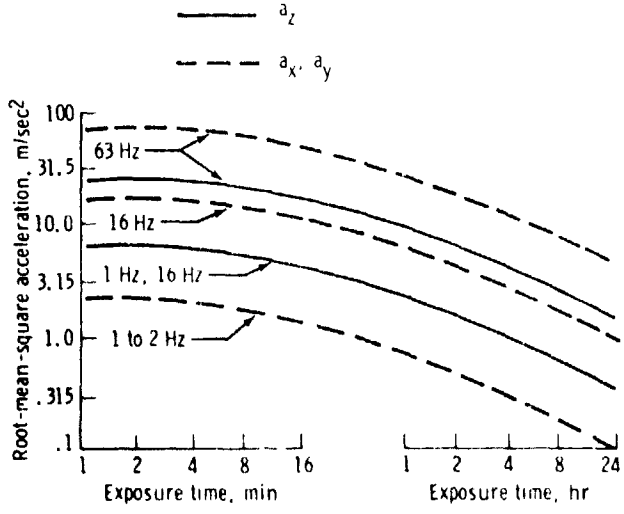
Vibration conditions	Measures	Effect	Source
	Audition		
5 Hz sinusoidal, 5 Hz random amplitude, 4 to 12 Hz random frequency	Frequency (pitch) change (1200 for 1600 Hz) at 86 dB, tones of 0.25-sec duration every second - detection	No effect.	Reference IV-65
$\pm g_z$	1200 Hz at 86 dB presented every 0.25 sec for 1 sec against a 74-dB, 30- to 3000-Hz white noise; pitch change at 86 dB (1600 for 1200 Hz) - detection	No effect.	Reference IV-66
$+1G_z \pm 0.7g_z$ at 15 Hz (amplitude 0.09 cm (0.036 in.)) for 30 min	Temporary threshold shift determined as function of vibration and noise compared to noise alone (acoustical frequencies from 250 to 6000 Hz)	Extremely small vibration effect at low tone frequencies only.	Reference IV-67
	Higher mental processes		
$\pm 0.15g_z$ to $\pm 0.35g_z$ at 2.5 and 3.5 Hz	Mental addition	No effect.	Reference IV-58
$+g_z$ at 5, 7, and 11 Hz	Pattern matching and discrimination	No effect.	Reference IV-68
$0.40g_z$ rms random vibration	Navigational tasks in simulated low-altitude, high-speed flight	No effect.	References IV-56 and IV-69
(1) No vibration; (2) no noise, no vibration; (3) noise only; (4) vibration plus noise; (5) postvibration $+4.0g_z$ at 70 Hz	Continuous counting at a given rate	Decrement, especially during 5 to 7 min of exposure; residual effects noted; 70 percent of decrement attributed to vibration (30 percent to noise); subjects older than 36 yr showed greater decrement.	Reference IV-70

TABLE IV-5.- SUMMARY OF EFFECTS OF VIBRATION ON PHYSIOLOGICAL FUNCTION

Vibration conditions	Physiological effects	Source
	Respiration and heart rate	
$\pm ng_x$ and $\pm ng_y$, 1.5 to 5.5 Hz	Oxygen consumption increased from 7.5 to 11 liters/min without increase in respiration rate, at ±0.25g and ±0.35g.	Reference IV-42
$\pm 1G_z \pm ng_z$ (0.33 to 0.63 cm (0.13 to 0.25 in.) DA), 2 to 15 Hz	No increase in respiration rate; increase in tidal volume at 6, 11, and 15 Hz.	Reference IV-71
$\pm 1G_x \pm ng_z$ (2.54 cm (1 in.) DA), 6.6 Hz	Mild hyperventilation.	Reference IV-72
g_z, rms	Respiratory rate increased as function of vibration intensity to $0.8g_z$ rms, small increase in heart rate.	Reference IV-73
g_z, rms	Heart rate increase at onset of vibration, return to resting levels during exposure.	References IV-73 and IV-74
$+1G_z \pm ng_z$	Same as for rms g_z.	Reference IV-75
$+1G_x \pm ng_{x,y,z}$	Same as for rms g_z.	Reference IV-76
	Blood, kidney, endocrine effects	
$\pm 1G_z \pm ng_z$, 4 to 9 Hz, 1 min	No evidence of effect on renal function; no change in white blood cell count.	Reference IV-77
$0.20g_z$ to $0.40g_z$ rms	No blood, urine, or stool changes.	Reference IV-55
$\pm 1G_z \pm ng_z$, 1 to 20 Hz, 3-min tolerance level	No effect on 17-hydroxycorticosteroids; no consistent change in urine or plasma levels.	Reference IV-78
	Effects in animals	
9.7g, 4 to 40 Hz	14 of 60 animals died; effects included high body temperature, kidney degeneration, increased white blood cells, hemorrhage.	Reference IV-79
g_z, 4 to 8 Hz	Elevated 17-hydroxycorticosteroids; when anesthetized, not as great; death in some dogs.	Reference IV-78
	Skin	
$+g_z$	Skin temperature, no change.	Reference IV-66



(a) The frequency function.



(b) The exposure time function.

Figure IV-12.- Fatigue/decreased-proficiency boundaries for acceleration-vibration environments.

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V. SOUND AND NOISE

By J. L. Homick

INTRODUCTION

Acoustical noise is largely a byproduct of technologically advanced and industrialized societies. Noise produced by the supersonic transport (SST) aircraft is but one glaring example of modern man's ability to create noisy machines.

Partly because few can avoid hearing loud sounds and partly because people are becoming more concerned with what these sounds are or may be doing to them, there has been in recent years an increased awareness of noise in public, private, and government sectors. Legislation has been created to attempt to regulate community and industrial noise. In general, the average individual in modern society is better informed regarding noise and its possible effects. This increased awareness has extended to the design and development of large manned spacecraft systems.

No attempt is made here to summarize all available noise data from the manned space program. It is sufficient to note that the designers of earlier spacecraft (Mercury, Gemini, Apollo, and Skylab) were aware of the detrimental effects of excessive noise exposure and were successful in keeping launch and on-orbit noise to safe, if not tolerable, levels. That is not to say that noise was never a problem. Reports from flight crewmen indicated that noise sometimes interfered with speech communications and disrupted sleep. For these and other reasons, acoustical noise control has received considerable attention in the design of the Space Shuttle spacecraft. How successful these noise control efforts on the Space Shuttle vehicle will be remains to be seen. Inevitably in the design and construction of spacecraft, where cost, weight, and size are critical factors, a compromise must often be reached between what is desirable and what is practical. Nevertheless, in future, more sophisticated vehicles, intended for long-duration habitation by large numbers of men and women, acoustical noise must be given due consideration as a potential detriment to safety, functional efficiency, and physical and mental health.

The purpose of this chapter is to acquaint scientists and engineers associated with the design and/or use of future spacecraft with problems related to sound and noise. The majority of the information presented is on the effects of audible sound and noise on man. Some effects of nonaudible sound (infrasound and ultrasound) are also presented. No attempt has been made to summarize all of the vast literature in this area. Instead, emphasis has been placed on those factors which have potential relevance to the space-flight situation.

Definition and Description

In defining sound, it is appropriate to first consider the basic properties of the sound source, the transmission medium, and the receiver. Any mass which contains the physical properties of inertia and elasticity, and which has been set into vibration by the application of some energy, may be a sound source. Sound is propagated through the surrounding medium (gas, liquid, or solid) in all directions from a vibrating source. The propagation results from the fact that molecules in the medium transmit their motion to one another in a progressive, wavelike manner. That is, condensation and rarefaction phases of molecules in the medium are propagated as compression waves. Sound wave propagation is dependent on the physical characteristics of the transmission medium: inertia, elasticity or compressibility, friction, and density (ref. V-1). For the purposes of this report, the receiver of sound energy is the human body and, specifically, the auditory system, which is exquisitely constructed to transduce mechanical energy (sound) into electrochemical neural energy. The reader is directed elsewhere for a more thorough description of sound generation and propagation, as well as a description of the anatomy and function of the human auditory system (refs. V-2 and V-3).

Perceived or audible sound is of considerable value to man. It can warn him of danger and arouse and activate him. It provides him the immeasurable advantage of communication by speech and language. Sounds can elicit a variety of emotions, both desirable and undesirable.

Unfortunately, excess sound generated by sources irrelevant to the individual may arouse him with excessive frequency, may have no adaptive value, or may simply be offensive. Also, everyday experience indicates that excess sound can interfere with the perception of important, relevant auditory signals. Perhaps most important is the well-documented fact that exposure to excessive sound can cause temporary or permanent loss of hearing function.

Thus, irrelevant or excessive sound is undesirable. By definition, such unwanted sound is noise. The definition of noise often involves a value judgment; sounds that may be considered as noise by some individuals may not be noise to others. Sounds that are valuable in one location may travel to areas where they may interfere with and disrupt useful and desirable activities. Other sounds may be noises only at certain times, in certain places, to certain people. These complexities regarding the definition of noise must be kept in mind, particularly when considering certain psychophysiological and social effects of noise.

Parameters of Sound and Noise

As previously stated, the primary purpose of this report is to summarize relevant information about the effects of audible sound and noise on man. To render a discussion of these areas more meaningful, a brief description of the physical parameters of sound and noise is provided.

The basic parameters used to describe acoustical energy fields include frequency, intensity, and duration. Each of these is discussed separately in the following subsections.

Frequency.- The concept of oscillation at a particular frequency can be represented by the back and forth movement of a clock pendulum. The number of oscillations per unit time defines the frequency, which is specified by the unit hertz (oscillations per second). Audiofrequency refers to the frequencies within the normal range of human hearing, usually specified as 20 to 20 000 hertz. Ultrasound refers to frequencies above 20 000 hertz, and infrasound contains frequencies below 20 hertz.

The period of a sound is the time interval required for one complete oscillation and is the reciprocal of frequency. A sound is periodic if the value of the period (and hence the frequency) remains constant throughout its duration. Pure tones are periodic sounds. Most environmental sounds are nonperiodic, as they contain many frequency components. Such nonperiodic sounds are generally referred to as noise. (This definition of noise is not to be confused with the previous definition of noise as unwanted sound.) Wide-band noise contains a wide or large portion of the frequency spectrum; narrow-band noise contains only a narrow or limited portion of the frequency spectrum.

Since many sound effects of interest to the acoustical environmental engineer and the physiologist are frequency dependent, it is necessary to specify the frequencies which are present in a sound field. These data are usually presented in the form of a frequency spectrum, which is a plot of relative intensity as a function of frequency. Frequencies may be specified in successive octave, half-octave, or third-octave frequency bandwidths or in single cycles. An octave refers to an interval of frequencies in which the lower frequency is one-half the higher frequency (ref. V-1).

Intensity.- Ideally, sound intensity should be measured in terms of the displacement of air particles, their average velocity during oscillation, or the energy contained in their motion. At the magnitudes associated with ordinary sound, however, suitable techniques are not available for performing these measurements. Fortunately, the pressure changes in the sound wave can be determined with special pressure transducers or microphones. The dynamic pressure changes associated with sound differ by several orders of magnitude from static pressures normally encountered. Normal atmospheric pressure is about 10^5 N/m^2 (10^6 dyn/cm^2), whereas pressure fluctuations produced by sounds near the absolute threshold for hearing are on the order of 10^{-5} N/m^2 (10^{-4} dyn/cm^2).

For a simple sinusoidal wave (pure tone), pressure amplitude can be specified as the maximum value of the pressure increase above the ambient pressure or, alternatively, as the root-mean-square (rms) pressure. The relationship between these measures is

$$P_{rms} = \frac{P_{max}}{\sqrt{2}}$$

$$= 0.707P_{max}$$

where P_{max} is the peak pressure and P_{rms} is the rms pressure (ref. V-1).

The relationship in the equation refers to sinusoids. For complex waves (i.e., noise) which contain more than one frequency, there is no unique relationship between peak pressure and rms pressure. Peak pressure varies widely in a complex sound wave; rms pressure is more stable and is, therefore, the preferred measure when describing environmental sounds which usually contain many frequencies. Another reason for using the rms measure is that under specific conditions, the power present in a sound wave can be related to the rms pressure. Power in microwatts per square centimeter is given by

$$J = \frac{P^2}{10R}$$

where P is pressure and R is specific acoustic resistance (a quantity determined by the density and sound propagation velocity of the medium). As with sound pressure, the power associated with sounds one encounters in his everyday activities may vary by several orders of magnitude. Sounds that are of sufficient intensity to destroy the sensory cells of the ear produce as little power as 0.002 watt (ref. V-1).

The ear operates over an enormous range of sound intensities. The most intense sound pressures that can be received without producing damage are approximately one million times greater than the least intense sound pressures that can be detected. To conveniently deal with this range of physical sound intensities, a logarithmic scale is used. Because a logarithmic scale has no zero point, sound intensities must be specified with respect to some standard reference intensity. Sound intensity is commonly scaled as sound pressure level (SPL) in decibels according to the formula

$$SPL = 20 \log_{10} \frac{P_1}{P_0}$$

where P_1 is the pressure of the sound in question and P_0 is the reference pressure. One of the most widely employed reference pressures for sound measurement is $P_0 = 2 \times 10^{-5}$ N/m^2 (0.0002 microbar). This value is approximately equal to the lowest pressure change a young person with normal hearing can detect at a frequency of 1000 hertz. The relationship between decibels, microbars, and newtons per square meter is shown in table V-1 (ref. V-4). Common examples of representative SPL's are as follows.

TABLE V-1.- RELATIONSHIP BETWEEN DECIBELS, NEWTONS PER SQUARE METER, AND MICROBARS

[Adapted from ref. V-4]

dB	N/m^2	μbar
0	0.00002	0.0002
14	.0001	.001
34	.001	.01
54	.01	.1
74	.1	[a]1
94	1	10
114	10	100
134	100	1 000
154	1 000	10 000
174	10 000	100 000

[a]1 microbar = 1 dyn/cm^2.

Location	SPL, dB
A business office	50
Speech at 0.9 meter (3 feet)	65
Subway at 6.1 meters (20 feet)	95
Jet aircraft at 10.7 meters (35 feet)	130
Atlas launch at 45.7 meters (150 feet)	150
On gantry during Saturn V launch	172

For a complex (nonperiodic) sound, the pressure levels within various frequency bands can be measured, and a frequency spectrum can be constructed. The intensity of the total complex wave can also be specified with a single number, usually designated as the overall sound pressure level (OASPL). Equal weight is given to the frequencies between 20 and 20 000 hertz by the OASPL scale. (The OASPL scale is "flat" across this frequency range.) Several other single-value scales of complex sound intensity are in use. These scales apply different weights to the various frequencies. For example, the dBA scale gives more emphasis to the midrange of the audiofrequency spectrum. This scale approximates the ear's loudness response at relatively low sound pressure levels. The dBC scale is nearly flat across the audiofrequency range and is similar to the ear's loudness response at high sound levels (ref. V-1). Sound level meters in common use have filtering networks which allow determinations of A-weighted (dBA), B-weighted (dBB), and C-weighted (dBC) sound intensities as well as the previously described OASPL value. Sound power refers to the total sound energy radiation in all directions from a source per unit time. The power of sound sources is measured in watts and can be expressed by a logarithmic scale of sound power level (PWL) in decibels according to the formula

$$PWL = 10 \log_{10} \frac{W_1}{W_0}$$

where W_1 is the power of the source and W_0 is the arbitrary reference power of 10^{-13} watt (ref. V-5).

As sound waves diverge from a sound source in a spherical manner, the surface area of the wave front increases as the square of the radius. As a result, the energy per unit area decreases inversely as the square of the distance from the source. This is known as the inverse square law of intensity. A 3-decibel decrease in PWL (equivalent to a 6-decibel SPL decrease) occurs with each doubling of the distance from the source.

Duration.- Duration of exposure to a sound is critical when considering possible physiological or behavioral reactions. As will be discussed in later sections, brief exposure to a particular sound may be of no physiological consequence, whereas extended exposure to the same sound could result in irreversible damage. The effects of total duration also vary significantly if the sound is interrupted or if other parameters (e.g., spectral content) of the sound change during the exposure period.

Variations in sound pressure as a function of time may be represented as time-pressure histories. Such histories may be used to represent the temporal pattern of the pressure change directly as in the case of a shock wave or impulse noise. An impulse may be generally defined as an aperiodic pressure phenomenon of less than 1000 milliseconds duration, having a flat rise and decay time and a peak to rms ratio greater than 10 decibels. Common examples include the sound of gunfire and of drop forges. The occurrence of significant impulse noises during space flight is not likely; however, that such noise can occur in flight is exemplified by the thrusters on the astronaut maneuvering unit tested during the Skylab missions. These thrusters produced short noise bursts which peaked at 125 decibels SPL. The more typical sound to be expected during space flight is steady-state sound. Such sounds may be represented by time-pressure histories which specify the temporal change of an envelope within which a complex sound wave is contained. Potential sources of sound and noise during flight are briefly discussed in the following section.

Sources of Sound and Noise

Sound and noise problems may arise at several points during space operations. These problems can be broadly categorized into three general phases.

Launch and boost.- Noise sources during the launch and boost phases include primarily rocket engine noise during lift-off and aerodynamic noise developed during exit through the atmosphere. The duration of exposure for these phases is usually on the order of 3 to 5 minutes.

The external sound field of a space vehicle during launch and boost is created primarily by the booster rocket engines. When the vehicle and engine geometry, exhaust flow factors, and atmospheric conditions are known, it is possible to specify the noise at any point, both in the near and far field. Normally, this specification requires a determination of the total sound power produced by the rocket engine at each frequency, as well as details of how the sound is directed away from the turbulent exhaust flow. These results can then be used with the inverse square law of distance and atmospheric attenuation to describe the sound field at applicable points of interest. Allowance must also be made for refraction and reflection effects.

Because there are no large rotating or reciprocating parts in a rocket engine, the noise of rocket vehicles does not normally contain discrete frequency components. The level of very low frequency noise (1 to 100 hertz) produced by the turbulent mixing of the booster exhaust flow with the surrounding atmosphere generally rises as the booster increases in size and thrust. It is probable that very large superboosters of the future will produce their maximum noise energy in the infrasonic range (below 20 hertz).

As a space vehicle accelerates from its launch pad, lift-off noise can extend far into surrounding community areas but will quickly diminish within the crew compartments of the vehicle itself. With increasing airspeed, however, the crew compartments will be exposed to aerodynamic noise generated by

boundary-layer turbulence. This boundary-layer noise reaches its maximum level as the vehicle passes through the range of maximum dynamic pressure and progressively decreases thereafter (ref. V-6).

Aerodynamic noise increases in level and peaks at lower frequencies as vehicles become larger. For vehicles in the size range of the Apollo spacecraft and greater, noise at maximum dynamic pressure tends to peak below 100 hertz.

On-orbit and interplanetary cruise.- During orbit and interplanetary cruise, vehicle internal sound and noise environments would assume special significance. As discussed in subsequent sections, noise can be a critical factor in safety, comfort, speech communications, performance efficiency, and emotional stability. Specific noise sources will in part be determined by mission requirements. However, some sources of noise such as fans, pumps, motors, and compressors associated with environmental control and waste management systems will always be present, as will be noise from communications systems such as intercoms and teleprinters. It is also probable that noise will be produced by the use of operational or experiment subsystems which contain moving parts or utilize compressed gases. Data that are representative of the noise environments to which crewmen were exposed during previous simulated and actual space flights are summarized in table V-2.

Entry and landing.- During entry, boundary-layer turbulence may again generate an internal sound field containing broadband noise of high intensity. The sound pressure levels reached may be comparable with those produced during the maximum dynamic pressure period at launch, but high intensities may be maintained for longer periods. Other noise sources such as those described previously may contribute to the internal vehicle sound field (ref. V-6).

Auditory Effects of Noise Exposure

Because the auditory system is the primary receiver of sound and is exceptionally sensitive to sound, it is not surprising that noise has its greatest effects on the ear and hearing. One set of auditory effects may become apparent after a noise of sufficient intensity has passed. These include temporary hearing loss, permanent injury to the ear, and permanent hearing loss. Other effects are noticeable while a noise is present. These include masking and interference with speech communication (ref. V-7). Each of these adverse auditory effects, as well as ways to protect against them, is discussed in the following subsections.

Ear damage and hearing loss.- Stimulation with very high intensity (135 to 170 decibels SPL) audiofrequency sound can produce gross destruction of auditory structures, including rupture of the tympanic membrane, separation of the middle ear bones (particularly at the joint between the incus and the stapes), and rupture of the fine membranes or sacs of the cochlea and labyrinth (ref. V-1). The primary site of auditory damage is the receptor organ of the inner ear, the organ of Corti, which contains the sensory cells (hair cells), nerve fibers, and supporting structures. Excessive exposure to noise

TABLE V-2.- REPRESENTATIVE NOISE DATA FROM SIMULATED AND ACTUAL SPACE FLIGHTS

Spacecraft	Mission duration, days	Measurement location	SPL, dBA	Octave band SPL, dB, for frequency, Hz, of -							
				63	125	250	500	1000	2000	4000	8000
[a]Apollo 2TV-1 command module	N/A[b]	Cabin, internal	65	45	48	58	55	61	58	62	55
[a]Apollo LTA-8 lunar module	N/A[b]	Cabin, internal	73	54	52	57	69	63	63	66	66
[c]Skylab Medical Experiment Altitude Test (SMEAT)	56	Wardroom	61	62	62	61	56	54	50	48	49
		Science pilot sleep compartment	54	61	58	56	52	46	43	40	41
		Experiment compartment	58	57	60	58	55	50	46	44	37
[d]Skylab 3	59	Wardroom	49	--	46	48	47	41	35	24	21
		Pilot sleep compartment	43	--	44	41	43	34	26	21	14
		Multiple docking adapter	61.5	--	61	56	55	53	52	47	45
		Airlock module	58	--	62	60	58	52	49	43	40
		Command module	57	--	61	56	56	52	49	43	41
[d]Skylab 4	84	Experiment compartment	55	43	47	46	56	48	36	31	32
		Pilot sleep compartment	41	46	41	38	41	34	25	16	9
		Dome hatch	52	50	53	51	53	47	41	36	28
[c]Spacelab Mission Simulation 3 (SMD-3)	7	Middeck	63	64	65	62	59	59	56	51	42
		Aft Spacelab	66	72	70	64	63	61	54	46	38

[a]Ground test (unmanned).
[b]Not applicable.
[c]Ground test (manned).
[d]Space flight (manned).

can lead to destruction of the hair cells. Other noise-induced injuries to the organ of Corti can range from mild distortion of its structure to collapse or complete degeneration. The auditory neurons may also degenerate. Once these specialized cells are destroyed, they do not regenerate spontaneously and cannot be stimulated to regenerate. Light- and electron-microscope studies have shown that high-frequency sounds produce damage within a limited area toward the basal end of the cochlea, whereas low-frequency sounds produce damage over a greater area toward the apical end of the cochlea.

An increase in hearing threshold level that results from exposure to noise is called a threshold shift. Threshold shifts may result from single or repeated exposures to noise. Also, as will be discussed briefly in a later section, threshold shifts can result from exposure to impulse noises.

Some threshold shifts are temporary and diminish as the ear recovers following the termination of exposure. Frequently, repeated exposures can induce threshold shifts that are chronic, though recoverable after the exposure ceases. When a threshold shift is a mixture of temporary and permanent components, it is a compound threshold shift. When the temporary component shift has disappeared, the remaining threshold shift represents irreversible damage and is permanent (ref. V-7).

Temporary threshold shifts can vary in magnitude from those causing loss of a few decibels in a narrow range of the spectrum to those causing temporary deafness. The time required for the ear to return to near normal can vary from hours to weeks. Noises with energy concentrations in the frequency range between 2000 and 6000 hertz appear to produce greater temporary threshold shifts than do noises elsewhere in the audible range. In general, A-weighted sound levels must exceed 60 to 80 decibels before a typical person will experience temporary threshold shifts even for exposures that last as long as 8 to 24 hours (ref. V-7). The greater the intensity level above 60 to 80 decibels and the longer the time of exposure to noise, the greater the temporary threshold shift. However, exposure durations beyond 8 to 24 hours may not produce further increase in the magnitude of the shift (ref. V-8). This factor has particular relevance with regard to long-duration exposures during space flight. Another property of temporary threshold shifts is that they are usually greatest for test tones one-half to one octave above the frequency region in which the noise that produces the shift has its greatest concentration of energy. Finally, temporary threshold shifts are less severe when an exposure has frequent interruptions than when an exposure is continuous.

People differ in their susceptibility to temporary threshold shifts. These differences, however, are not uniform across the audible range of frequencies. One individual may be especially susceptible to noises of low pitch, another to noises of medium pitch, and another to noises of high pitch. In general, women appear to be less susceptible to temporary threshold shifts from low-frequency noises than are men. This relationship is reversed for high-frequency noises (ref. V-7).

The hypothetical growth of threshold shifts after various single, continuous exposures to noise is presented in figure V-1. Certain facts are obvious from this figure. The more intense the noise, the more rapidly threshold shifts accumulate as the time of exposure to noise is extended. When the noise is only 60 decibels, a typical person has to be exposed for several hours before any significant threshold shift can be detected. However, when the noise is very intense (e.g., 120 decibels), a typical person exposed for only 5 minutes reaches dangerous levels of threshold shift. Combinations of intensity level and duration that produce threshold shifts greater than about 40 decibels are said to be in the region of possible acoustic trauma. In this region, the normal function of the auditory system may break down and permanent threshold shifts, or hearing loss, may occur after even one such exposure (ref. V-7).

Hypothetical recovery from threshold shifts after various single, continuous exposures to noise is illustrated in figure V-2. Although recovery processes are not well understood, it may be seen that complete recovery from temporary threshold shifts can occur within hours to a few days provided that the threshold shift is relatively small (less than 40 decibels) and the duration of exposure is short (less than 8 hours). Recovery from the temporary threshold shifts appears to be very slow when the initial shift exceeds 40 decibels, when the exposure duration exceeds 12 hours, or after long but intermittent exposure to noise. Recovery from very severe exposures may never be complete (ref. V-9). A definition of acceptable exposure limits is presented in the following subsection.

Exposure limits (damage risk criteria).- Considerable effort has been directed toward the establishment of criteria for maximum allowable sound exposure. This effort would be easier if there were a clear distinction between safe and potentially damaging sound exposures across the population. Unfortunately, as has been noted previously, individuals vary widely in their susceptibility to sound-induced hearing loss. This fact poses a problem in that if criteria are set low enough to protect highly susceptible persons, they would be excessively stringent for a majority of the population. Thus, the credibility of the criteria would be weakened. On the other hand, if criteria are set excessively high, susceptible individuals may suffer unnecessary hearing loss.

Short-duration limits: The most detailed and elaborate sound damage risk criteria for short-duration exposures published to date are those developed by the Committee on Hearing, Bioacoustics, and Biomechanics (CHABA) of the U.S. National Academy of Sciences National Research Council (NAS-NRC) (ref. V-9). Damage risk contours proposed by the CHABA group for periodic and nonperiodic sound exposures are presented in figures V-3 and V-4, respectively. The curves of figure V-4 indicate the noise levels at which one exposure per day is likely to produce damage. One-octave band sound levels are given on the left ordinate and one-third-octave narrower band levels are given on the right.

The curves can be applied to particular band levels which may be present in broadband noise. For example, the bottom curve indicates that exposure to a one-octave band of noise centered at 3000 hertz with a level of 85 decibels

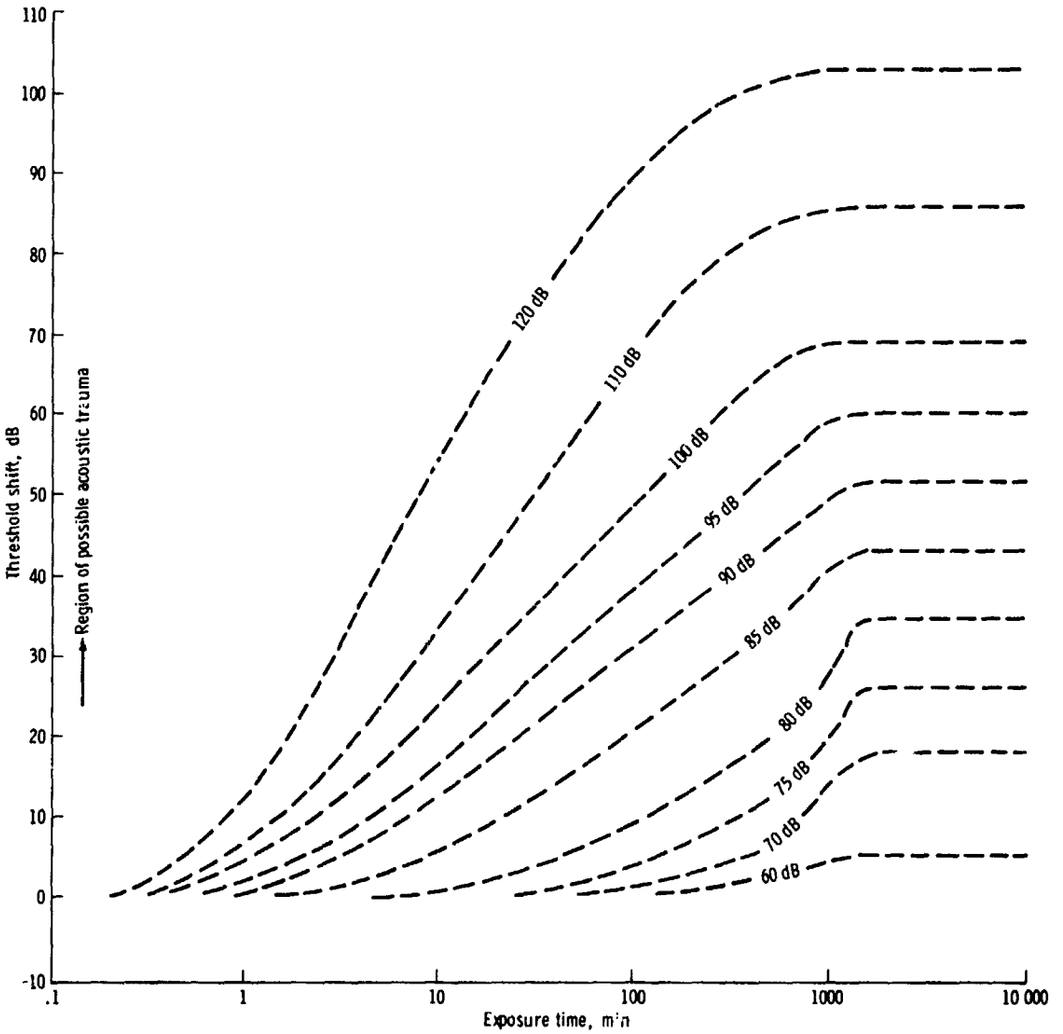

Figure V-1.- Hypothetical growth of threshold shift measured 2 minutes after single, continuous exposure to various levels of noise at a frequency of 4000 hertz. (Adapted from ref. V-7.)

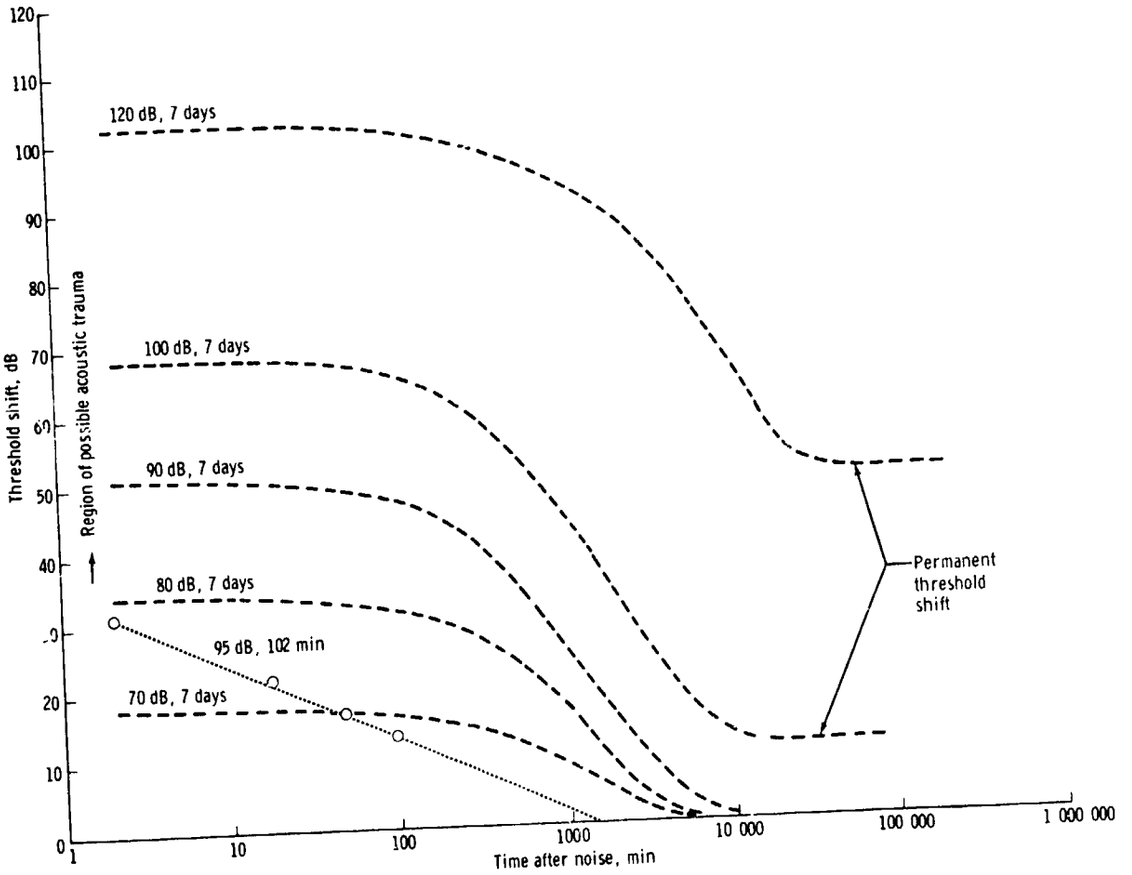

Figure V-2.- Hypothetical recovery from threshold shift after single, continuous exposure to noise of various levels and exposure durations at a frequency of 4000 hertz. (Adapted from ref. V-7.)

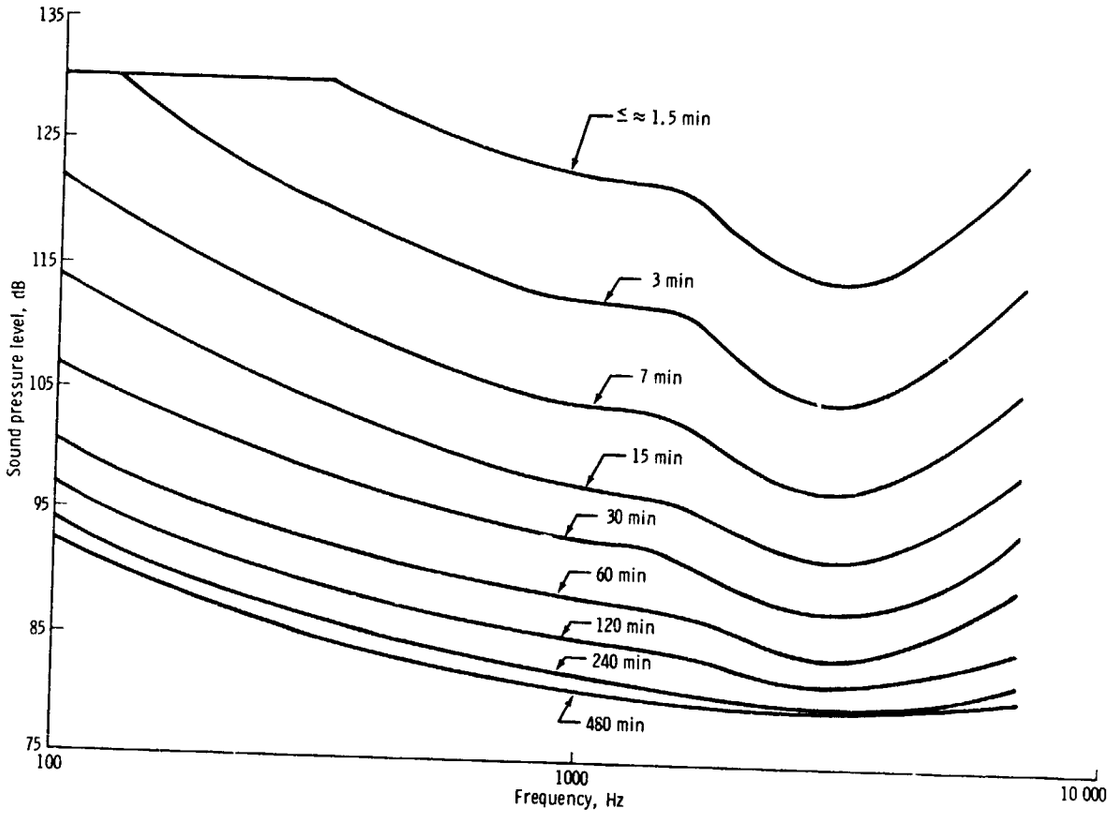

Figure V-3.- Damage risk contours for one exposure per day to pure tones of various durations; reference pressure is 2×10^{-5} N/m^2. (After ref. V-9.)

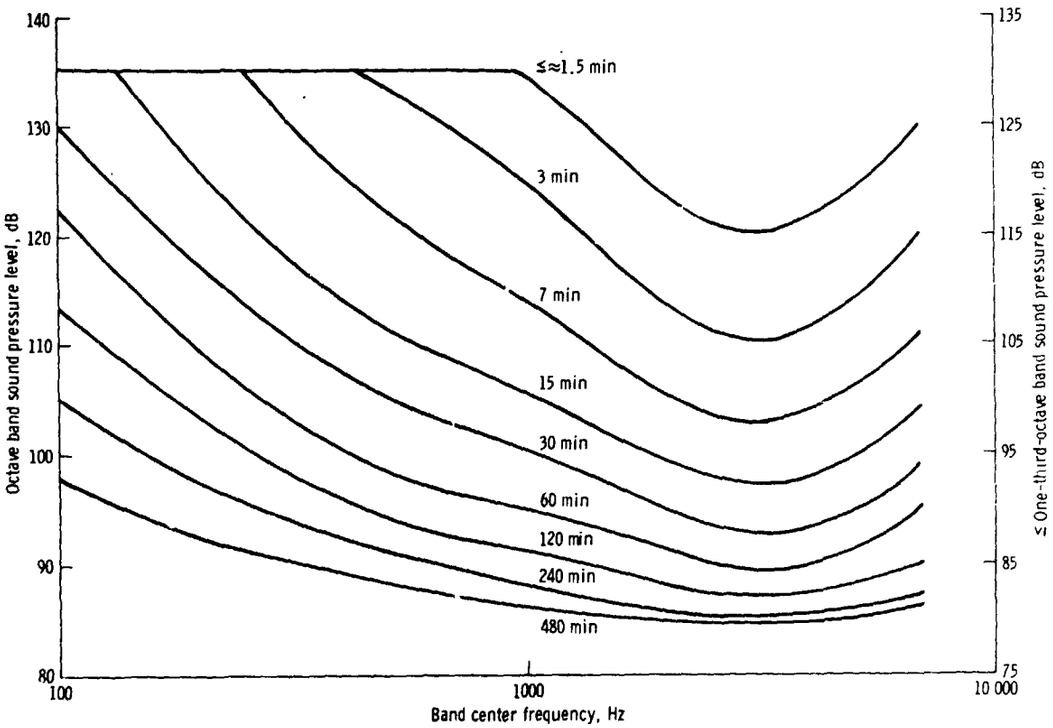

Figure V-4.- Damage risk contours for one exposure per day to one-octave and one-third-octave or narrower bands of noise of various durations; reference pressure for both scales is 2×10^{-5} N/m^2. (After ref. V-9.)

for 8 hours daily constitutes a potential threat to hearing. The curves of figure V-3 for pure tone exposures are interpreted in the same manner as the curves of figure V-4. These damage risk criteria have been incorporated into the design and procedure standard for acoustical noise[1] developed by the NASA Lyndon B. Johnson Space Center (JSC).

Damage risk criteria for impulse noise are not as extensive as the previously defined criteria for continuous ($\leq$8 hours) noise. This deficiency results largely from the fact that the measurement of impulse noise is not as convenient or reliable as the measurement of continuous noise. Nevertheless, tentative limits have been established by the NAS-NRC CHABA (ref. V-10). An idealized impulse sound pressure waveform and exposure limits are presented in figures V-5 and V-6, respectively.

The damage risk criterion (ref. V-10) specifies acceptable exposure levels for two types of impulse waveform duration.

1. A pressure-wave duration, termed A-duration in the criterion, is the time required for the initial or principal pressure wave to reach its positive peak and return momentarily to ambient pressure. In the ideal pressure wave shown in figure V-5(a), the time duration is given by the distance on the time axis C - A.

2. A pressure-envelope duration, termed B-duration in the criterion, is the total time for which the envelope of the pressure fluctuations, both positive and negative, is within 20 decibels of the peak-pressure level. Included in this time is the duration of that part of any reflection pattern that is within 20 decibels of the peak level. In figure V-5(b), the B-duration is the time (D - A) + (F - E).

The damage risk criterion for impulse noise establishes acceptable peak-pressure exposure limits for a sound wave impinging on the ear at a normal incidence, as a function of either A or B pulse duration (fig. V-6). It is considered that below these limits (assuming 100 exposures per day), 95 percent of the exposed normal-hearing population will not sustain a temporary or a permanent threshold shift greater than 10 decibels at or below 1000 hertz, 15 decibels at 2000 hertz, and 20 decibels at or above 3000 hertz. Exposures that exceed any of the specified limits may be considered as potentially dangerous in the long term for the majority of exposed individuals.

Long-duration limits: The maximum allowable once-per-day exposure levels specified in figures V-3 and V-4 are especially applicable to defining exposure limits for rocket launch noise or other on-orbit mission phases where crewmen may occasionally be exposed to intense noise for brief periods. However, these criteria are inadequate with regard to the more typical

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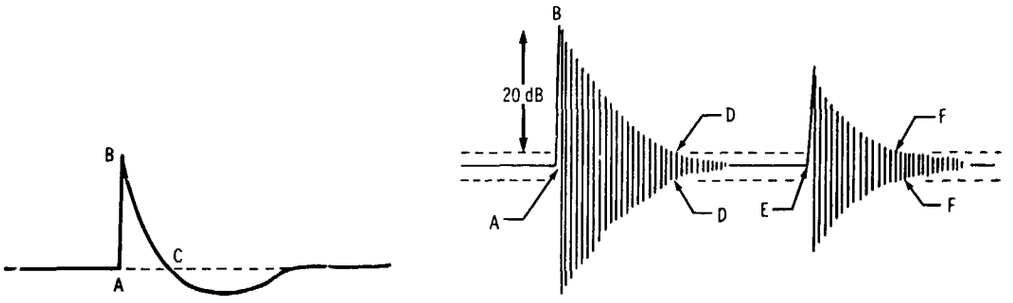
[1]MSC Design and Procedural Standard 145: Acoustic Noise Criteria. NASA Lyndon B. Johnson Space Center, Oct. 16, 1972.



(a) Type A-duration. (b) Type B-duration.

Figure V-5.- Impulse waveforms. See text for explanation. (From ref. V-10.)

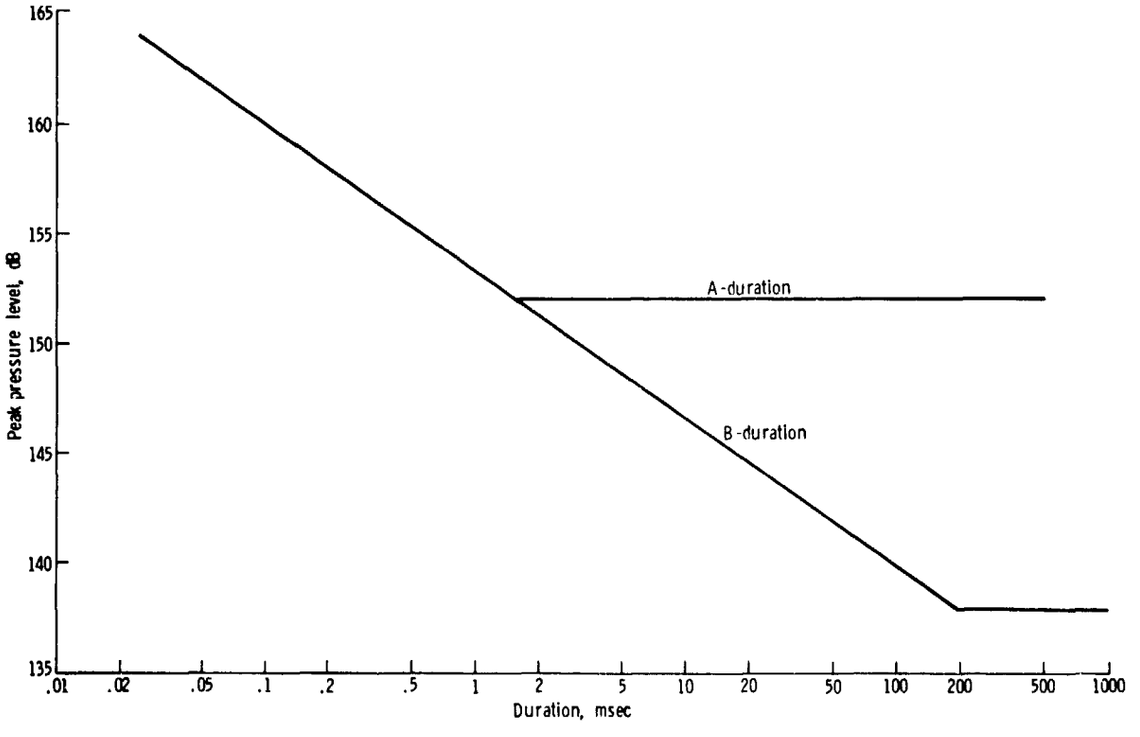


Figure V-6.- Damage risk criterion for impulse noise (gunfire); reference pressure is 2×10^{-5} N/m^2. See text for discussion. (From ref. V-10.)

space-flight situation, in which crewmen will be exposed to noise continuously, 24 hours per day, for periods of 6 months or longer. A need clearly exists for valid standards which define safe limits for continuous, long-duration noise exposures.

Unfortunately, empirical data on which to base such standards do not exist. Therefore, it has become necessary to develop tentative long-duration exposure limits which are based in part on rational assumptions and on careful extrapolations from existing short-duration exposure data or "occupational health" audiometric data. To date, these long-duration exposure standards have been developed primarily for industrial and community noise applications; however, they can also be cautiously applied to the space-flight situation.

One approach that has been used in the past involves the use of noise criterion (NC) curves. These curves were originally developed primarily for defining acceptable workspace background noise where communications interference, loudness, or annoyance by noise are important design considerations. A family of NC curves is presented in figure V-7. It is significant to note that the JSC design and procedural standard for acoustical noise specifies that maximum allowable continuous sound pressure levels produced by normal spacecraft operating equipment or systems shall not exceed the NC-50 contour during work periods and the NC-40 contour during sleep periods. As evidenced by the Skylab orbital workshop (OWS) vehicle, in which sound rarely exceeded the levels specified by the NC-50 curve, these levels are conservative but not unrealistic.

Valid objections can be raised against the use of NC curves for defining allowable spacecraft noise levels. The primary drawback is that NC curves rarely, if ever, accurately represent the typical spacecraft noise spectrum shape. Although knowledge of energy distribution within the audible spectrum is important, it is more convenient and more meaningful in many instances to specify limits in terms of a single dBA value. Indeed, most noise criteria in use today do specify limits in dBA or some version thereof.

One of the most comprehensive and authoritative sources of noise level recommendations currently available is a report published by the U.S. Environmental Protection Agency (EPA) (ref. V-8). No attempt is made here to summarize the document; however, several of the recommendations proposed may be translated to space-flight noise environments. The EPA recommends that the level deemed necessary to protect hearing (with an adequate margin of safety) is a daily equivalent noise level L_{eq} of 70 decibels; L_{eq} is an energy average of A-weighted sound levels over a specified period of time. For reasonably steady sounds of the type anticipated on future spacecraft, L_{eq} is the average sound level meter reading utilizing the A-weighted network. (For purposes of comparison, it should be noted that NC ratings are about 5 decibels less than the measured dBA levels.)

The EPA determination of the 70-decibel L_{eq} value was based on the assumption that people would be exposed on a 5-day/week basis throughout their working lifetime of 40 years, that the noise would be intermittent rather than continuous, and that a period of relative quiet (60 dBA or less for 16

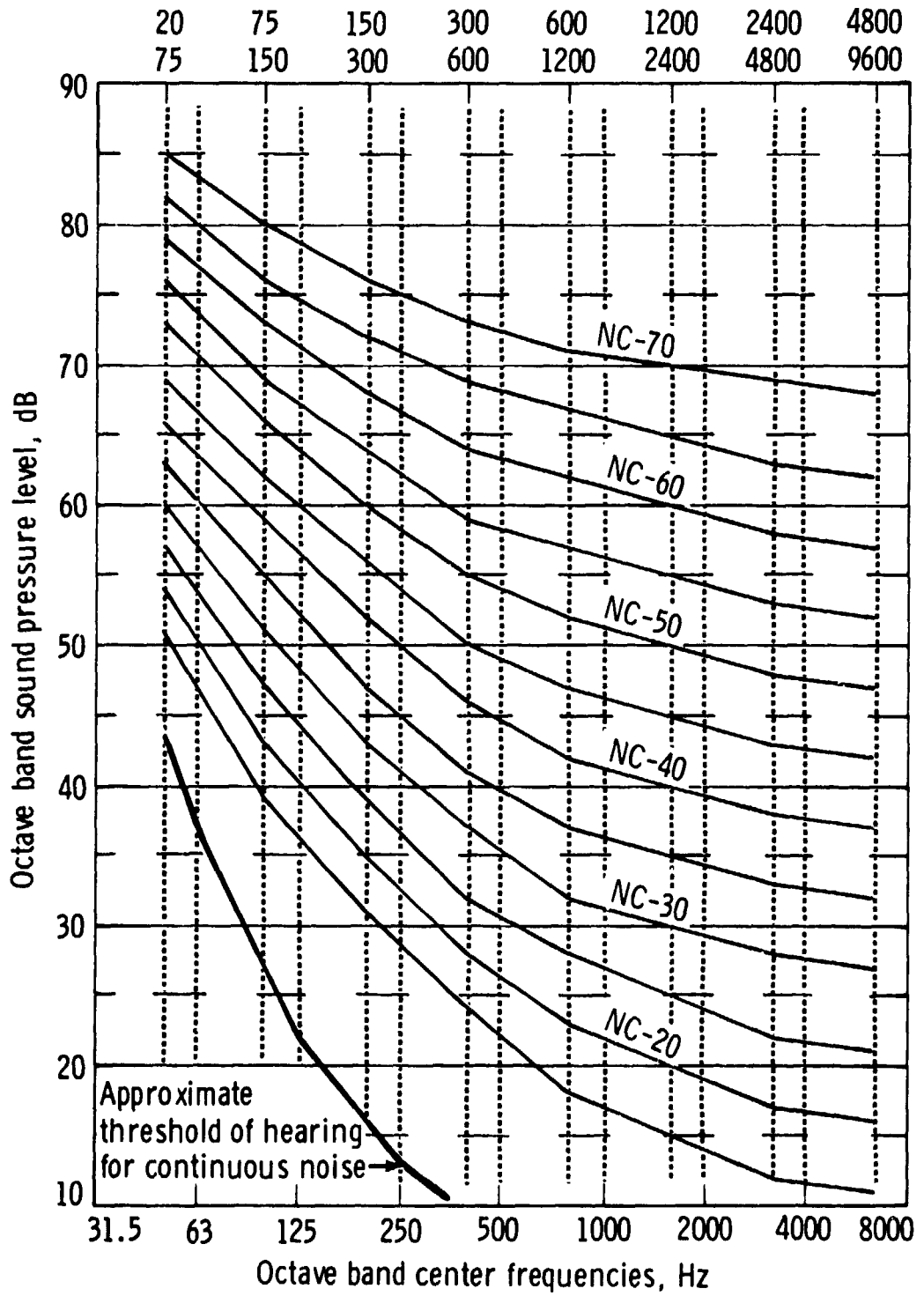


Figure V-7.- Noise criteria curves; reference pressure is 2×10^{-5} N/m^2. Lower abscissa consists of preferred octave bands; upper abscissa, of commercial octave bands.

hours) would occur after each workday to allow complete recovery from temporary threshold shifts. The EPA has also recommended correction factors to the 70-decibel L_{eq} figure to compensate for the effects of continuous noise exposures of 24-hour duration, as well as nonoccupational exposures of as long as 1 year rather than 40 years of occupational noise exposure.

Using the EPA data and recommendations, Bolt, Baranek and Newman (BBN), in a recent study conducted for JSC, determined that to prevent possible hearing damage during a 7- to 30-day Space Shuttle mission, the L_{eq} should not exceed 76 dBA.[2] This level is approximately equal (depending on the spectrum of the noise) to an NC value of 70. This level is considerably higher than the NC-50 value contained in the JSC design and procedural standard for acoustical noise. It must be remembered, however, that the JSC standard was written assuming exposures of indefinite length and considering such factors as communications interference and annoyance. The BBN recommendation of 76 decibels L_{eq} applies only to damage risk and is for a 7- to 30-day exposure. Indeed, BBN recommended that longer exposures require lower limits (e.g., 67 decibels L_{eq} for a 60-day mission; durations greater than 60 days were not addressed by the BBN study). Also, in the same study, BBN recommended that to minimize communications interference, sleep interference, annoyance, and performance decrement, the overall level on a 30-day mission should not exceed an NC value of 50 (approximately 55 dBA L_{eq}).

Prevention of ear damage and hearing loss.- The preferred approach to preventing ear damage and hearing loss is to ensure that people are never placed in a sound field that is potentially damaging. Ideally, the sound source itself should be reduced to acceptable levels if at all possible. Often, however, for a variety of practical reasons, this condition cannot be met. For example, noise levels to which flight crewmen or ground-support personnel may be exposed during the launch phase of a mission may be almost unavoidable.

Under such conditions, steps must be taken to prevent the sound from actually reaching the ear. At least three approaches may be considered. First, the person may be removed to a distance from the noise source such that spherical divergence and attenuation reduce the noise to an acceptable level. This approach has obvious limits as in the case of rocket launch noise. A second approach is to place a sound-attenuating physical barrier (e.g., wall or curtain) between the source and the receiver. The third, and often most practical, approach is to place a mechanical hearing protector over or in the ear canal to attenuate sound energy. The devices may take the form of ear inserts, which are available in several different types, ear muffs, and helmets. Of these three, a properly fitted ear insert generally affords the greatest protection. Even greater protection can be obtained when an insert and ear muffs are used in combination. Regardless of type, ear defenders are more effective in blocking high-frequency sound than low-frequency sound. It must be recognized that the maximum amount of hearing protection that can be obtained with any combination of devices is about

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[2]Pearsons, K. S.: Recommendations for Noise Level in the Space Shuttle. Bolt, Baranek and Newman Job No. 157160, Feb. 28, 1975.

45 to 50 decibels. Beyond this level, sound energy can be transmitted to the inner ear by bone conduction (ref. V-11). Ear protectors of any type must be properly selected and used. Despite the effectiveness of these devices, people often refuse or neglect to wear them for reasons of appearance, discomfort, and bother.

Aside from the devices described previously, it is noteworthy that the auditory system has a built-in mechanism to protect hearing. Sudden or unexpected sounds elicit a reflex contraction of the middle ear muscles which reduces the amount of noise conducted to the inner ear. Attenuation of as much as 10 decibels can be produced by this mechanism. This middle ear reflex is not effective in protecting against high-intensity impulse noise because the noise enters the ear before the reflex has time to occur.

Masking and Speech Communication

The masking phenomenon and the effect of noise on speech communication are discussed in the following paragraphs.

Masking effects.- Noise mixed with a signal (including speech) tends to raise the threshold for hearing that signal above the threshold in quiet. This phenomenon is called masking; the elevated threshold is the masked threshold. The masking effects of a pure tone, or of a noise with strong frequency components, differ somewhat from those of narrow-band and wide-band noise; however, all masking sounds possess several common features. The more intense the masking sound, the greater the effect. Low-frequency sounds (pure tone or noise) produce a greater masking effect than do high-frequency sounds of equal loudness. The masking effect is always greater above the frequency of the masking sound (refs. V-7 and V-11).

Speech communication.- Audible noise can adversely affect speech communications in several ways. First, as described previously, the noise can mask the speech signal. Second, the listener may suffer a temporary threshold shift as a result of the noise exposure and, consequently, higher than usual signal intensities will be required to accurately perceive speech signals. Also, noise may interfere with the efficiency of the total perceptual speech process.

Several different methods are available for predicting the consequences of noisy environments on speech intelligibility. Two of these are the speech interference level (SIL) and the articulation index (AI). Both methods have limitations. Under conditions of extreme noise masking, frequency distortion, and amplitude distortion, the predictive value of these methods breaks down. In such conditions, empirical speech intelligibility tests in the noise environment of concern (or an accurate simulation of that environment) become necessary. Nevertheless, these two predictive methods are widely used and will be briefly discussed. It is important to bear in mind that the articulation index should be used in all carefully designed speech communication systems. The speech interference level can serve as a rule-of-thumb guide in making some engineering decisions regarding face-to-face communications (ref. V-11).

Speech interference level: The speech interference level was designed primarily for use in situations characterized by noise having a relatively continuous spectrum. As such, the SIL is applicable to most spacecraft noise. The SIL is defined as the average sound pressure level in the three octave bands which are most important for speech communication. These bands are 600 to 1200, 1200 to 2400, and 2400 to 4800 hertz. More recently, the average sound pressure level of the octave bands centered at 500, 1000, and 2000 hertz have come into use. This latter calculation is referred to as the preferred speech interference level (PSIL). The SIL (or the PSIL) yields the maximum noise level that will permit correct recognition of 75 percent of phonetically balanced (PB) words and/or about 98 percent of test sentences. (This criterion is equal to an AI of approximately 0.5.) Consequences of selected SIL values on typical face-to-face communication are summarized in table V-3. It is also noted that for speech interference purposes, SIL values are equivalent to NC values. Thus, an NC-50 curve equals an SIL of 50 decibels.

Articulation index: An important advantage of the AI relative to other methods for assessing the effects of noise on communications is that the AI can be used for a wide variety of communication systems. The AI formulation is based on the fact that, to obtain high intelligibility, one must deliver a considerable fraction of the total speech bandwidth to the listener's ear and, also, that the signal-to-noise ratio at the listener's ear must be reasonably high. If the speech peaks are 30 decibels or more above the noise throughout the frequency band from 200 to 6100 hertz, the listener will make essentially no errors (AI = 1.00). If the speech peaks are less than 30 decibels above the noise in any part of the speech band, the listener will make some mistakes (AI < 1.00). If the speech peaks are never above the noise at all (ratio of speech peaks to rms noise less than 0 decibel), the listener will rarely be able to understand anything (AI = 0) (ref. V-11).

The AI can be established by one of two methods, the 20-band method or the weighted-octave-band method. In the 20-band method, the frequency spectrum between 200 and 6100 hertz is divided into 20 bands. The fractional contribution to the AI of each band is 0 to 0.05 depending on the difference (ratio) between the speech peak pressure level and the noise pressure level. The contributions made by the 20 bands when added together give the AI for the communication system in question. The weighted-octave-band method approximates the 20-band method and is computed from octave band measurements of speech and noise. Details on calculating the AI by either of these methods are available elsewhere (ref. V-11). The relationship between intelligibility and the AI for various types of speech materials is shown in figure V-8. Despite the utility of the AI, such factors as noise duty cycle, rate of interruption, frequency and amplitude distortion, reverberation, voice quality, and visual cues from the speaker must be considered.

Behavioral Effects of Noise Exposure

The behavioral effects of noise exposure in the areas of performance, sleep interference, and annoyance are discussed in this subsection.

TABLE V-3.- EFFECTS OF NOISE AS SPECIFIED BY SPEECH INTERFERENCE LEVEL ON PERSON-TO-PERSON COMMUNICATIONS

[From ref. V-11]

Speech interference level, dB	Person-to-person communication
30 to 40	Communication in normal voice satisfactory, 1.8 to 9.1 m (6 to 30 ft). Telephone use satisfactory.
40 to 50	Communication satisfactory in normal voice, 0.9 to 1.8 m (3 to 6 ft); raised voice, 1.8 to 3.6 m (6 to 12 ft). Telephone use satisfactory to slightly difficult.
50 to 60	Communication satisfactory in normal voice, 0.3 to 0.6 m (1 to 2 ft); raised voice, 0.9 to 1.8 m (3 to 6 ft). Telephone use slightly difficult.
60 to 70	Communication with raised voice satisfactory, 0.3 to 0.6 m (1 to 2 ft); slightly difficult, 0.9 to 1.8 m (3 to 6 ft). Telephone use difficult. Earplugs and/or ear muffs can be worn with no adverse effects on communication.
70 to 80	Communication slightly difficult with raised voice, 0.3 to 0.6 m (1 to 2 ft); slightly difficult with shouting, 0.9 to 1.8 m (3 to 6 ft). Telephone use very difficult. Earplugs and/or ear muffs can be worn with no adverse effects on communication.
80 to 85	Communication slightly difficult with shouting, 0.3 to 0.6 m (1 to 2 ft). Telephone use unsatisfactory. Earplugs and/or ear muffs can be worn with no adverse effects on communication.

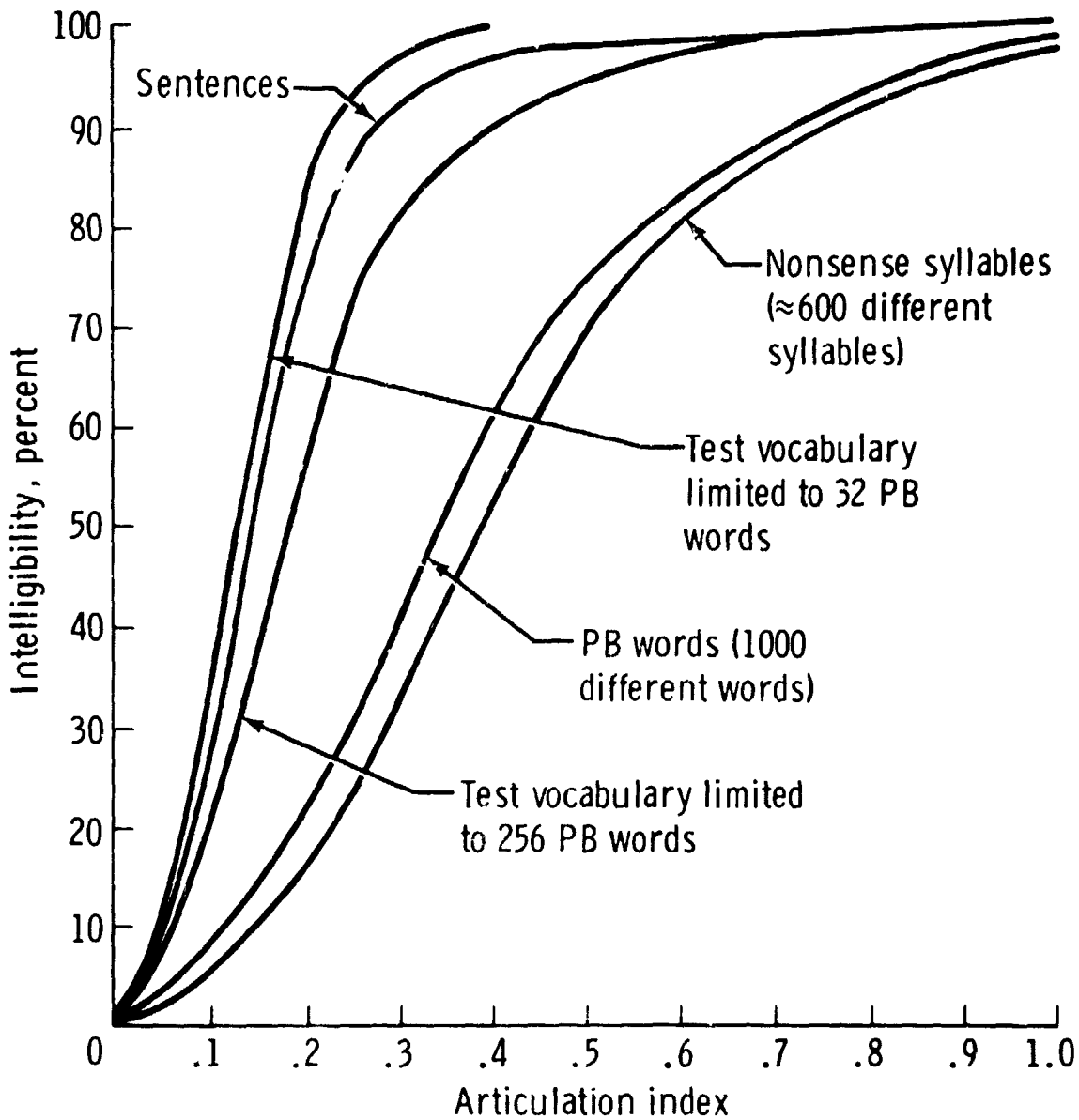

Figure V-8.- Relationship between the articulation index and the intelligibility of various types of speech-test materials. The relationships are approximate; they depend on such factors as skill of talkers and listeners. (From ref. V-11.)

Performance.- The effect of noise on performance has been studied extensively in the laboratory and in actual work situations. Relevant findings from these studies are summarized in the following paragraphs.

When a task requires the use of auditory signals, either speech or nonspeech, noise at any intensity level sufficient to mask or interfere with the perception of these signals will interfere with the performance of the task. When tasks do not involve auditory signals, the effects of noise on their performance have been difficult to evaluate. Nonetheless, certain general conclusions have emerged.

1. Steady noises without special meaning do not seem to interfere with human performance unless the A-weighted noise level exceeds about 90 decibels.

2. Irregular bursts of noise are more disruptive than steady noises. Even when the A-weighted sound levels of irregular bursts are below 90 dBA, they may sometimes interfere with performance of a task.

3. High-frequency components of noise, above about 1000 to 2000 hertz, may produce more interference with performance than low-frequency components of noise.

4. Noise does not seem to influence the overall rate of work, but high levels of noise may increase the variability of the rate of work. There may be noise-induced pauses followed by compensating increases in work rate.

5. Noise is more likely to reduce the accuracy of work than the total quantity of work.

6. Complex tasks are more likely to be adversely influenced by noise than are simple tasks (ref. V-7).

Even when individuals are able to maintain high performance in noise environments, there may be a cost. This cost might include reduced psychological or physiological capacity to react to additional demands and increased fatigue after completion of the task. Many physiological and psychological responses to noise diminish or disappear when the noise is regular or predictable. Strategies can be learned so that the detrimental effects of noise on performance can be avoided. Under certain conditions, noise may even result in better performance because of such factors as auditory isolation provided by masking of other sounds by noise, greater activation and alertness of the worker, or paced performance when the noise is regular or rhythmic. Noises, however, often are not regular and predictable. Adaptation to noise is not always complete, and strategies to eliminate the adverse effects of noise are not always learned.

An ideal acoustical environment is one that does not disturb human performance either because of the properties of the noise itself or because of

irrelevant messages carried by the noise. The task for the spacecraft designer is to eliminate disturbing noises while maximizing the chances that important, relevant messages carried by sound will reach the appropriate receiver.

Sleep interference.- The effects of noise on sleep are not well understood. It is known that the disturbing effects of noise on a sleeping person are related to several factors including the effective perceived noise level, the meaning of the noise to the person, the age of the person, and the stage of sleep the person is in. These effects have been measured in terms of altered electroencephalograph (EEG) activity and observable behavioral responses.

Empirical and anecdotal data also clearly indicate that noise can prevent a person from going to sleep or can awaken one from sleep if it is of sufficient intensity, has important meaning, or is of unusual character. Awakening may, in turn, cause a typical somatic arousal response which tends to alert the individual. The threshold of audibility or hearing of a noise may increase as much as 70 to 80 decibels as one goes from a light to a deep stage of sleep (ref. V-5). Noise above the level of audibility during a particular stage of sleep will usually cause some awakening, but the alertness or stress response may or may not occur depending upon the meaning or information value of the noise.

Some investigators have conjectured that because environmental noise can awaken a sleeping person or cause, without awakening, the changes in EEG activity that usually precede awakening, the noise is physiologically harmful, particularly to a person who is ill or recovering from undue fatigue. Data to substantiate such conjectures are sparse, even though there are data which clearly indicate that sleep deprivation can lead to harmful psychological and physiological effects (ref. V-5).

Individual variations of the effects of noise on sleep are great. Recent reviews of the literature in this area indicate that whereas some people may be disturbed at relatively low levels of noise such as NC-30 (35 dBA), others may sleep through levels equivalent to NC-80 (about 85 dBA). For this reason, the definition of acceptable and reasonable noise standards (limits) is difficult. Recommended levels for bedrooms and sleeping quarters have traditionally ranged from NC-25 to NC-40; however, because of practical design considerations, such levels may be difficult to achieve. (The JSC design and procedural standard for acoustical noise specifies NC-40 as an upper limit for sleep periods.) Nevertheless, that these levels are obtainable is exemplified by the Skylab OWS, in which noise levels in the crew sleeping compartments were at or below NC-40 (45 dBA) levels. In the Skylab environment, the crewmen's sleep appeared to be disturbed only by occasional intermittent noises.

In addition to the Skylab findings, several studies have been performed to evaluate sleep quality and quantity (sometimes with the aid of EEG measurement techniques) during simulated space-flight conditions. In these studies, noise levels ranged from 55 dBA (Skylab Medical Experiment Altitude Test) to

80 dBA (Lunar Module Noise Study). The EEG data obtained from the test subjects indicated little or no change in sleep patterns; however, intermittent noises (e.g., pumps turning on) did cause sleep arousals.

Until more definitive data are available, only general guidelines can be offered with regard to spacecraft noise limits for sleep.

1. The noise should be steady-state and preferably below NC-50 (55 dBA).

2. Intermittent noise sources should be eliminated.

3. Sleep areas should be acoustically isolated from spacecraft noise sources.

4. Provided the level does not exceed approximately 55 dBA, some steady-state broadband masking noise could be employed to alleviate disturbances due to intermittent noise. The level and/or spectral shape of this masking noise should be under control of the crewman.

5. Spacecraft designers should not assume that noise problems can be corrected by requiring crewmen to use hearing protection devices (earplugs or ear muffs).

Annoyance.- Various techniques for predicting potential annoyance caused directly or indirectly by noise exposure have been developed in the past 25 years. A large proportion of this effort has concentrated on annoyance evoked by aircraft noise, including the sonic boom. These techniques have also been used to assess the annoyance associated with a wide range of other environmental sounds.

One annoyance measure is perceived noisiness. This measure relates the noisiness of a particular environmental sound to the noisiness of a band of noise centered around 1000 hertz. The scale of perceived noisiness is PNdB. Details on the calculation of this value are available elsewhere (ref. V-12). Studies have shown that several attributes of an environmental sound, in addition to its loudness, affect this measure. Perceived noisiness increases with the duration of a sound, the "unwantedness" of the sound, the potential of the sound for speech interference, the degree to which the sound disrupts sleep, awareness on the part of a person concerning possible damage to hearing by sound exposure, and some special meaning that the sound might have for the listener (ref. V-1).

An almost bewildering variety of other methods has been devised to measure annoyance and social response to community noise or noise from particular sources such as aircraft. Some of these methods carry acronyms such as CNR, NNI, NEF, TNI, NPL, and CNEL. No attempt is made here to discuss these methods; they are mentioned only to underscore the disagreement, and seeming confusion, that exists in this area.

The annoyance (or, conversely, the acceptability) of sounds in future space-flight vehicles is more likely to be subject to individual variations than perhaps any other effect of noise exposure. As previously indicated, for speech requirements, the level of NC-50 has been widely recommended as an upper limit for such spaces as laboratories, drafting spaces, and engineering rooms. It, therefore, seems reasonable that the NC level required to provide reliable speech communication within a spacecraft environment should also provide a tolerable environment from an annoyance aspect. It should be noted, however, that the use of NC curves requires that the spectra be relatively uniform and contain no discrete frequencies or pure tones. Studies have shown that tones can be as much as 10 decibels more annoying than an octave band of noise of the same level. In other words, to be equally acceptable, a tone would have to be as much as 10 decibels lower in level than the octave band of noise. Correction procedures have been developed to account for this pure tone effect. These corrections are fairly complex and are not in full agreement. For purposes of space vehicle noise level recommendations, BBN has suggested that pure tones be reduced such that no octave band exceeds its adjacent octave bands on average by more than 3 decibels.[2]

Nonauditory (Physiological) Effects

Although considerable attention has been given in the past to the effects of noise on hearing, speech reception, performance, sleep, and mental well-being, only recently has noise been shown to produce potentially significant nonauditory physiological responses. Sudden, unexpected bursts of impulsive or steady-state noise will cause somatic (autonomic nervous system) responses in man and animals including changes in cardiovascular blood pressure and volume, breathing, pulse rate, gastrointestinal motility, endocrine gland excretions, and other neural and body activities. These responses are sometimes designated as arousal, and sometimes as stress responses, and are difficult to distinguish physiologically from responses that occur in emotional states such as fear or anger (ref. V-5). In the past, responses such as these were thought to occur only with relatively intense (e.g., >90 dBA) auditory stimuli. More recent data, however, suggest strongly that these physiological responses can occur at levels below those which produce auditory damage. The precise level at which physiological effects begin and the seriousness of these effects has not been determined, but it may be as low as 75 to 80 dBA (ref. V-3).

It is known that with continued exposure to noise, provided the noise connotes no harmful environmental condition or does not interfere with behavior as the result of auditory masking, man will adapt more or less completely; i.e., will cease to show arousal responses. Sudden noises will elicit an eyeblink response which, unlike the general somatic responses mentioned previously, does not habituate with continued exposure to noise.

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[2]Pearsons, K. S.: Recommendations for Noise Levels in the Space Shuttle. Bolt, Baranek and Newman Job No. 157160, Feb. 28, 1975.

In addition to the previously described types of somatic responses, other nonauditory responses to intense noise have been documented. For example, steady noise above approximately 110 decibels can cause some temporary and permanent (after years of exposure) changes in size of visual field, and noise above 130 decibels can cause nystagmus and vertigo (ref. V-5). However, these effects on visual and vestibular systems are found only with noise of sufficient intensity to cause, if exposures are continued for sufficiently long periods, permanent damage to the auditory system.

As indicated previously, additional research is required before limits can be established for nonauditory physiological effects of noise exposure. However, as with the areas of performance, sleep, and annoyance, if spacecraft noise is limited to those levels (55 dBA) which permit satisfactory communications, then physiological effects should be transient and of no real significance.

Research Required to Refine Limits

Research required to refine limits in the areas of ear damage and hearing loss, communications, behavioral effects, and nonauditory physiological effects is discussed in this subsection.

Ear damage and hearing loss.- As previously indicated, a need exists to better define safe limits for continuous, long-duration noise exposure (i.e., exposures beyond 90 days and for periods of several years assuming manned interplanetary missions). Whether available occupational noise exposure limits will be applicable to the space-flight environment has not been fully determined. In this vein, a number of investigators have tentatively concluded on the basis of limited data that noise-induced hearing threshold shifts plateau after approximately 24 hours (refs. V-7 and V-8). If this supposition proves to be correct, then threshold shifts resulting from 24 or 24 000 hours of exposure to the same noise should be essentially the same. On the contrary, some limited data are available which suggest that temporary threshold shifts may increase in a nearly linear fashion with successively longer exposures to at least 600 hours (ref. V-4). Such conclusions have not been substantiated with valid test data.

Unfortunately, such data may not be easy to obtain. Ideally, these data should be obtained in operational situations or high-fidelity simulations in which individuals are exposed continuously to noise of known levels and spectra for long periods. Such studies are costly and it is improbable that they could be implemented for the evaluation of noise effects as such. Therefore, advantage should be taken of any opportunity to collect these data where men live and work in confined spaces for long periods. One such planned effort is to obtain internal cabin acoustical noise measurements during the early Space Shuttle flights.

Communications.- Acceptable levels for effective speech communication have been reasonably well defined. These levels are applicable regardless of mission duration. It should be cautioned, however, that if very long duration exposures do result in a progressive buildup of temporary threshold

shifts (which would remain chronic as long as the exposure is maintained) or even permanent threshold shifts, then communications will be degraded because of the reduced ability of crewmen to accurately perceive auditory information. In this event, desired auditory signals would have to be increased in intensity to be heard.

Behavioral effects.- As previously stated, large individual variations exist with regard to the effects of noise on performance, sleep, and tranquillity. Because of this variation and because available data have been interpreted differently by different investigators, valid limits cannot be established in this area currently. As a first step to resolving this problem, it is recommended that all available information be carefully reevaluated with a view toward its specific application to the space-flight environment. If satisfactory conclusions cannot be reached, then additional research will be required. To be of value, such research should be performed with human subjects in a setting which matches to the greatest extent possible not only the types of acoustical but also the functional (including psychosociological) environments that may be encountered during space flight.

Nonauditory physiological effects.- Conclusions and recommendations for future research on the physiological effects of noise have been offered by a working group of the NAS-NRC Committee on Hearing, Bioacoustics, and Biomechanics (ref. V-5). These recommendations are applicable to research required to refine limits (physiological noise effects) for spacecraft and are as follows.

1. So-called stress reactions in humans, when continued for sufficiently long periods, can be physiologically harmful. However, it appears that the psychological and physiological responses to noise (excluding changes in hearing) are transitory and that they adapt out with continued exposure to the noise and, therefore, do not constitute harmful physiological stress. This conclusion is deduced from a relatively small amount of research and from incompletely tested concepts. For these reasons, research involving at least weeks or months of psychological and physiological testing with human subjects exposed, when awake, to quiet and to both low-level background and higher level intermittent noise is needed. Included should be further laboratory and field research on the effects of noise on sleep.

2. Physiological stress reactions that sometimes appear in certain noisy environments are likely to be the result of frustration or anger that occurs when the noise interferes with the reception of a wanted auditory signal or when the noise distracts from some other activity. Setting tolerable limits for environmental noise in terms of its subjective acceptability to people and its damaging effects on the inner ear would appear to provide levels of normally present environmental noise that are lower than those which can directly cause harmful nonauditory physiological stress conditions in man. It is recommended that laboratory studies be undertaken to study individual differences in sensitivity of humans to noise and to multiple stress conditions, including the performance of tasks requiring use of auditory cues and tasks not requiring such cues. Some real-life situations may be found in industries or in various societies that permit useful research studies in

this problem area; however, these studies must be undertaken with considerable caution because of the possible presence of conditions having effects that outweigh those the noise may have on the psychological and physiological conditions of the people involved.

3. Nonauditory physiological stress responses in an organism to normally present environmental noise are often the result of interactions between specific behavioral activities and the noise rather than the noise as such. Therefore, research aimed at understanding the nonauditory effects of noise on man should, under most circumstances, not involve lower animals.

NONAUDITORY SOUND

Definition and Description

As indicated earlier in this report, nonauditory sound is sound having energy at frequencies either above or below the normal audible spectrum. Ultrasound typically refers to frequencies above 20 000 hertz; infrasound refers to frequencies below approximately 20 hertz. Both infrasound and ultrasound have been demonstrated to produce physiological and behavioral effects. Sounds in both of these categories are likely to occur to some extent during future space-flight missions and, therefore, must be of concern to the spacecraft designer and the space physiologist. Observed physiological and behavior effects of nonauditory sound, as well as currently defined limits, are summarized in the following paragraphs. It will be noted that some of these effects are not unlike responses produced by exposure to high-intensity audiofrequency sound.

Infrasound

Noise spectra containing very low audiofrequency and infrasonic energy may excite body structures such as the chest, the abdomen, the eyes, and the sinus cavities. Under these conditions, subjects have reported chest wall vibration, mild respiratory alterations, gag sensations, blurred vision, and speech tremor. Auditory pain and tissue damage occur with high-intensity exposures. Ear pain occurs at levels on the order of 160 to 170 decibels at 2 hertz to 140 decibels at 50 hertz. Usually, such damaging effects can be prevented with properly fitted earplugs or ear muffs. At lower intensities, tickling sensations of the tympanic membrane and sensations of pressure buildup in the middle ear have been frequently reported when ear protection devices are not used. Temporary threshold shifts occur which are a direct function of exposure intensity and duration. Responses indicating vestibular system stimulation have been observed following exposure to intense sound (135 to 140 decibels SPL) in the low audiofrequency and infrasonic range. Included have been observations of nystagmus, dizziness, nausea, and loss of balance. Behavioral effects stemming from some of these physiological responses include annoyance, discomfort, fatigue, and slower task performance

rates (refs. V-1, V-4, V-6, and V-13). Limited data suggest that infrasound does not significantly affect speech reception. No data can be found regarding the effects of infrasound on sleep.

Research interests on the effects of infrasound appeared to peak during the 1960's, when researchers were concerned particularly about launch noise from the Apollo booster rockets. Since that period, little meaningful work has been done in this area. One study on the effects of infrasound on man is summarized in table V-4 (ref. V-13). Representative noises to which subjects were exposed in a series of tests are indicated on the left. Typical subjective reports associated with each of the noise exposures are listed on the right. These findings indicated that voluntary tolerance limits for frequencies below 100 hertz occurred around 150 to 154 decibels as determined by symptoms of nausea, giddiness, coughing, etc. The results of this study confirmed that the maximum permissible exposure to infrasound is approximately 150 decibels SPL. It should be stressed that the findings in table V-4 are maximum subjective tolerance levels. As previously indicated, various other effects of infrasound on man may occur at lower levels. In a recent review of the literature on infrasound, Von Gierke and Parker (ref. V-14) proposed a set of limits for long-duration exposure to infrasound (fig. V-9). These proposed limits, which are similar to a 24-hour exposure of 75 dBA, are based on extrapolation from a set of admittedly incomplete experimental data.

Ultrasound

Documented evidence of detrimental effects of airborne ultrasound on man are scarce, largely because ultrasound is especially amenable to noise control measures and atmospheric absorption. The proper use of hearing protection devices can eliminate most undesirable effects. Ultrasonic noise levels produced by several types of equipment and associated subjective responses of exposed individuals are summarized in table V-5 (ref. V-13). In this study, subjective complaints increased with increased noise intensities and exposure durations. Sensations of malaise and fatigue were experienced with levels as low as 90 decibels SPL. In interpreting the significance of the findings summarized in table V-5, it has been well established that many ultrasonic exposures also contain considerable audiofrequency energy. Often, it is this lower frequency energy that results in undesirable sensations and complaints. Eliminating or reducing the audiofrequency energy often eliminates the symptoms reported. Nevertheless, when airborne ultrasound exceeds the levels specified in figure V-10, subjective responses such as those in table V-5 may occur (ref. V-12).

The preceding findings and guidelines apply to airborne ultrasound. Structural or liquidborne ultrasound presents potential problems of a different nature. A large amount of energy can be carried in ultrasound vibrations. Direct contact of any part of the body with structures or liquids carrying ultrasonic energy will result in transmission of that energy to the body, where it is converted to heat. If the exposure is excessive, tissue damage resembling burns may result (ref. V-1). Ultrasound fields of sufficient energy to cause such tissue damage could conceivably be encountered

TABLE V-4.- HUMAN RESPONSES TO LOW-FREQUENCY AND INFRASONIC NOISE EXPOSURE

[Adapted from ref. V-13]

Exposure	Observed behavior
Frequency ranges	
0 to 50 Hz, $\leq$145 dB	Chest wall vibration, gag sensations, respiratory rhythm changes, postexposure fatigue; voluntary tolerance not exceeded.
50 to 100 Hz, $\leq$154 dB	Headache, choking, coughing, visual blurring, and fatigue; voluntary tolerance limit reached.
Discrete frequencies	
100 Hz at 153 dB	Mild nausea, giddiness, subcostal discomfort, cutaneous flushing; tolerance limit symptoms.
60 Hz at 154 dB, 73 Hz at 150 dB	Coughing, severe substernal pressure, choking, respiration, salivation, pain on swallowing, giddiness; tolerance limit symptoms.

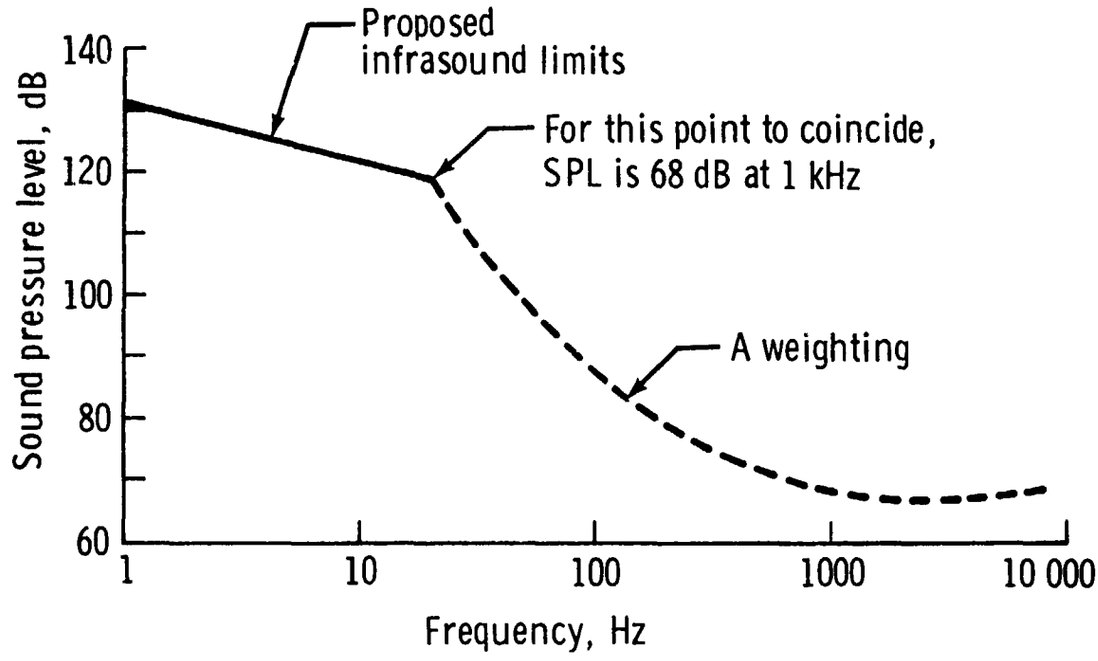

Figure V-9.- Proposed 24-hour infrasound limit, approximately equal to audio-frequency 24-hour limit of 75 dBA. Reference pressure is 2×10^{-5} N/m^2. (From ref. V-14.)

TABLE V-5.- SUBJECTIVE RESPONSES TO VARIOUS AIRBORNE ULTRASONIC EXPOSURES GENERATED BY ULTRASONIC INSTRUMENTATION

[From ref. V-13]

Item no.	Ultrasonic instrument type	Operating characteristics	Subjective response	Sound pressure levels, dB, at midpoint frequencies (third-octave bands), kHz, of -					
				10	12.5	16	20	25	31.5
1	Drill	Input freq. - 23.1 to 26.1 kHz, 400 mA	Pain in ears after few minutes. Headache after 1 hr. Nausea after 2 hr.	91	85	90	81	107.5	84
2	Welder	Input freq. - 15 750 Hz, intermittent exposure. On 15 sec, off 5 sec; 1 transducer	Malaise, backache; individual wore earplugs and symptoms disappeared. Operator had a cold when symptoms were experienced.	76	95	115	93	73	69
3	Cleaner	Input freq. - 20 kHz	Operator did not use the machine; the machine noise was too intense.	125	123.5	119.5	113	133.5	110
4	Cleaner	Input freq. - 40 kHz	Headache, malaise, fatigue; when necessary to use, the operator leaves the room.	77	78	79	96	77	77
5	Cleaner	Input freq. - 20 kHz, 1 transducer; water circulating at 293 K (20° C)	Fatigue and malaise. Shrill sound of 94 dB at 4000-Hz midpoint frequency.	81	78	84	93	76	76

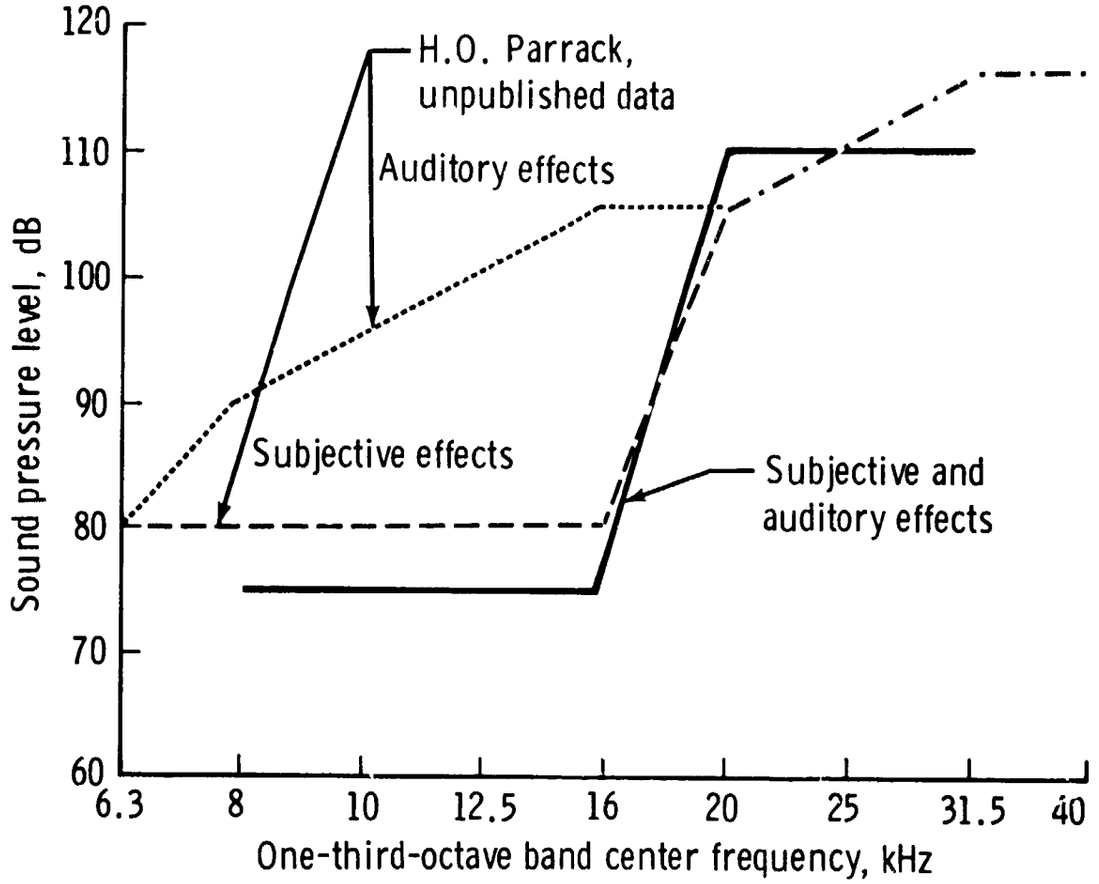

Figure V-10.- Proposed criteria for subjective and auditory effects of ultrasound; reference pressure is 2×10^{-5} N/m^2. (Adapted from ref. V-13.) The curve labeled "Subjective and auditory effects" (solid line) is from W. I. Acton, Institute of Sound and Vibration (ISAV no. 208), University of Southampton, Southampton, England.

in future space-flight environments. Limits for maximum allowable structural or liquidborne ultrasound are not available currently.

Research Required to Refine Limits for Spacecraft

Research required to refine limits for spacecraft is discussed in the areas of infrasound and ultrasound.

Infrasound.- It would appear that upper tolerance limits for short-duration exposures to infrasound have been reasonably well established. Data are sparse, however, with regard to long-duration exposure to lower level (75- to 100-decibel range) infrasound. Attempts should be made to acquire additional usable information in this latter area.

Ultrasound.- In general, on the basis of data available at the time of this writing, it would seem that the overall effects of ultrasound on man are not well understood. The literature must be more thoroughly evaluated. If sufficient data for the establishment of valid, safe standards for spacecraft design are not available, then appropriate research studies must be conducted.

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VI. RADIOFREQUENCY RADIATION

By D. S. Nachtwey

DEFINITION AND DESCRIPTION OF THE RADIOFREQUENCY ENVIRONMENT

Spacecraft personnel may be exposed to microwaves and other radiofrequency (rf) radiations. At sufficiently high power densities, rf radiation can be hazardous or lead to performance decrements. This chapter contains descriptions of sources of rf radiation, some of the biophysical characteristics of rf radiation, some of the potential biological effects, the U.S. rf radiation exposure standards, the philosophy behind the standards, and areas requiring additional research. The major source of information is a review by S. M. Michaelson (ref. VI-1), parts of which have been used verbatim.

Sources of Radiofrequency Radiation

Potential sources of rf radiation in spacecraft environments are (1) radar units: used during approach and docking, approach and landing in transfer spacecraft, or as a payload (e.g., Earth observation radar), (2) communications equipment (e.g., television transmitters), and (3) power beams: during tests of power beam transmission or during maintenance at solar power stations (i.e., side lobes and scatter as well as the direct beam must be considered).

Biophysical Aspects of Radiofrequency Radiation

Because the assessment of the biological impact of rf radiation is highly dependent on the basic biophysical aspects of the interaction of the radiation with biological systems, a somewhat extensive discussion of these biophysical aspects may be fruitful. Like all electromagnetic (EM) radiation, rf radiation consists of a stream of photons, each possessing a discrete energy. The behavior of EM photons can be described by wave equations; therefore, the different types of EM radiation are designated by their energy (in electronvolts or joules), their wavelengths (in meters, centimeters, or nanometers), or the frequency of the waves (in hertz).

The energy of the photon E is directly related to its frequency ν and inversely related to its wavelength λ. Equations (VI-1) and (VI-2) and figure VI-1 depict this relationship and the EM spectrum, which contains radiofrequency radiation.

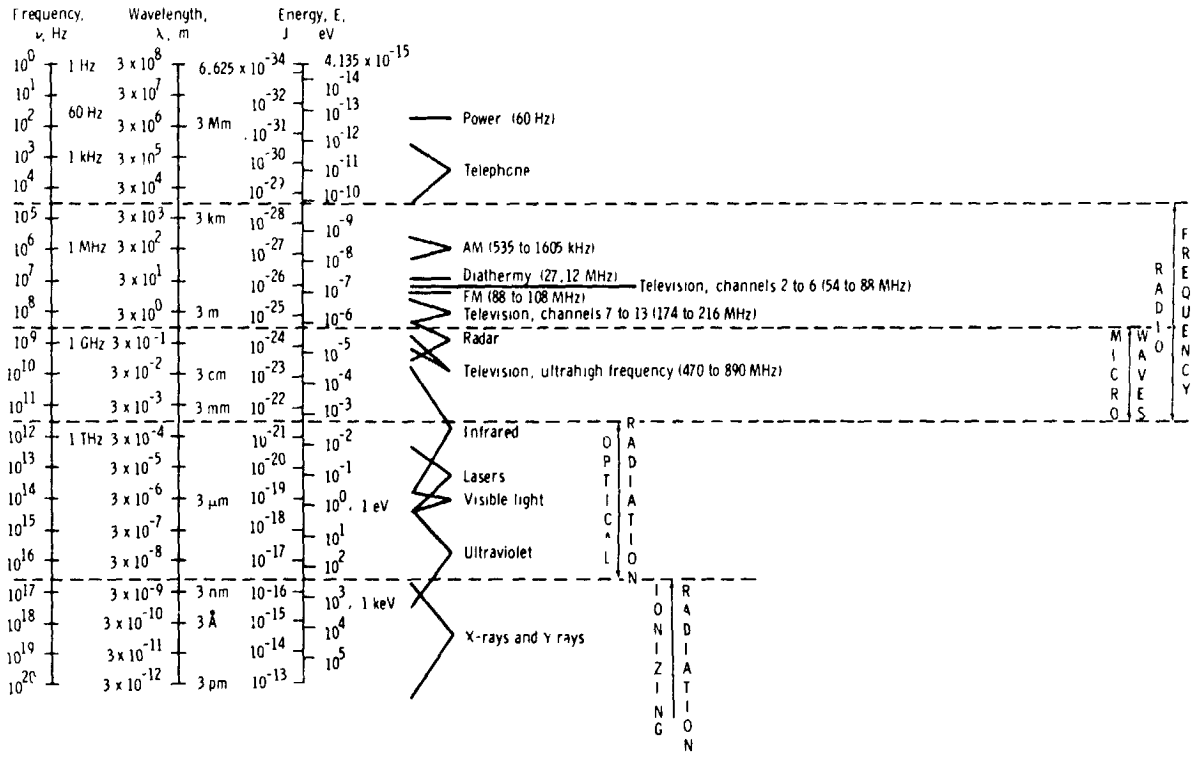


Figure VI-1.- Electromagnetic radiation spectrum. (After ref. VI-1.)

$$E = h\nu \qquad \text{(VI-1)}$$

$$\nu = \frac{c}{\lambda} \qquad \text{(VI-2)}$$

where h is the Planck constant and c is the speed of light in a vacuum.

To obtain biological effects from any EM radiation requires that the photon interact with molecules within the organism and that the energy of the photon be absorbed; without absorption of energy, there is no possibility for a biological effect. Electromagnetic photons are absorbed in a body in an exponential manner, e^{-kd}, with the absorption as a function of depth d being determined by the probability k of a photon interacting with the biological matter. The probability of interaction depends on a number of factors, but if the probability is high, essentially all of the photons will be absorbed in the surface layers. On the other hand, if the probability is very low, most of the photons may pass completely through the body; i.e., the body will be essentially transparent. Thus, not all photons in a beam of EM radiation will necessarily be absorbed nor will they necessarily be uniformly absorbed in a body. However, some photons will be absorbed in any case. When a high-energy photon, such as in X-rays or gamma rays, is absorbed, the absorbed energy of the photon can cause ejection of an orbital electron; i.e., it can cause ionization and thus detrimental alteration of the molecule and possibly of the organism. When a moderately low energy photon, such as in ultraviolet radiation, is absorbed, it cannot cause ionization but can raise an orbital electron in a molecule to an excited state, which can then lead to a detrimental chemical change in the molecule. When an even lower energy photon, such as in infrared or radiofrequency radiation, is absorbed, it cannot excite orbital electrons but can affect the molecule by increasing its vibrational, rotational, or translational energy; i.e., by increasing its temperature. In general, rf photons do not have sufficient energy to ionize or excite orbital electrons no matter how many are absorbed. The necessary minimum energy for ionization of biological molecules is approximately 10 electronvolts; for excitation, it is approximately 1 electronvolt. In contrast, the energy values of rf photons all lie in the range 10^{-3} to 10^{-10} electronvolt. Therefore, in biologic systems, absorbed rf energy does not cause direct chemical change but is rapidly equilibrated among the degrees of freedom of the system with the net effect being only an increase in general or localized temperature of the tissue. Such heating results from both ionic conduction and vibration of the dipole molecules of water and proteins (ref. VI-2). If sufficiently great, heating can, of course, cause damage to molecules (e.g., denaturation of proteins), cells (e.g., membrane breakdown), and organs.

Radiofrequency radiation transmission, scattering, and absorption are usually considered at the macroscopic wave level rather than as photonic interactions. Thus, these EM waves are characterized by an electric field vector $\vec{E}$ (volts per meter), a magnetic field vector $\vec{H}$ (amperes per meter), and a propagation vector $\vec{k}$ (per meter). Far from the wave source, the electric

and magnetic field vectors are mutually perpendicular and both are perpendicular to the direction of propagation along $\vec{k}$. This far-field rf radiation is plane-wave radiation. Close to electromagnetic sources and around electromagnetic scattering objects, the radiation is not plane wave: different configurations and relative magnitudes of the $\vec{E}$, $\vec{H}$, and $\vec{k}$ vectors are possible.

The total amount and the distribution of the absorbed electromagnetic power in biological tissue exposed to rf fields depends on many factors, including reflective loss, the magnitude of the electric field E, the magnitude of the magnetic field H, the relative stored energy in the magnetic and electric fields, the polarization of the fields, the frequency of the radiation, the source and tissue configurations, the tissue composition, and environmental factors. In general, however, the penetration and absorption of rf energy is dependent on the electrical properties of the absorbing medium, specifically, on its dielectric constant and electrical conductivity, both of which change as the frequency of the applied rf field changes. Values of dielectric constant, electrical conductivity, and depth of penetration have been determined for many tissues. (See table VI-1.) The absorption of rf radiation is high and, therefore, the depth of penetration is low in tissues of high water content such as muscle, brain tissue, internal organs, and skin, whereas the absorption is lower and, therefore, the depth of penetration is higher in tissues of low water content such as fat and bone. Reflections between interfaces separating tissues of high and low water content can produce standing waves accompanied by hot spots that can be maximum in either tissue, regardless of dielectric constant or conductivity. The degree of these reflections is also frequency dependent (table VI-1).

In considering the biological effects of rf radiation, the wavelength or frequency of the radiation and its relationship to the physical dimensions of the exposed objects become very important factors with major implications for the extrapolation of results of animal studies to effects on man. The absorption of power radiating from a source into space depends on the relative absorption cross section of the irradiated object. Thus, the size of the object with relation to the wavelength of the incident field of photons is significant. At very low frequencies (long wavelengths), a biological specimen absorbs very little electromagnetic power; that is, it is essentially transparent. Absorbed power increases rapidly with frequency up to a resonance region where the animal body dimensions are approximately 0.4 of the wavelength in free space. At frequencies greater than the resonance frequency, total absorbed power slowly decreases. The orientation of the animal body with its variable dimensions (height, width, thickness) to the planes of the various fields also is significant. Johnson et al. (ref. VI-3) and Durney et al. (ref. VI-4) have calculated the absorbed powers for prolate spheroids approximating the dimensions of man and laboratory animals used in microwave research. In figure VI-2, the specific absorbed power or specific absorbed radiation is shown as a function of frequency and long-axis orientation of a man-sized prolate spheroid to the various field vectors. (Other studies are in progress using more realistic simulated configurations; e.g., ellipsoids or block models.) It may be seen in figure VI-2 that orientation to the E-field vector is an order of magnitude more effective than orientation to the other vectors at frequencies below the resonance point at $\approx$70 megahertz.

TABLE VI-1.- PROPERTIES OF RADIOFREQUENCY RADIATION IN BIOLOGICAL MEDIA

[From ref. VI-2]

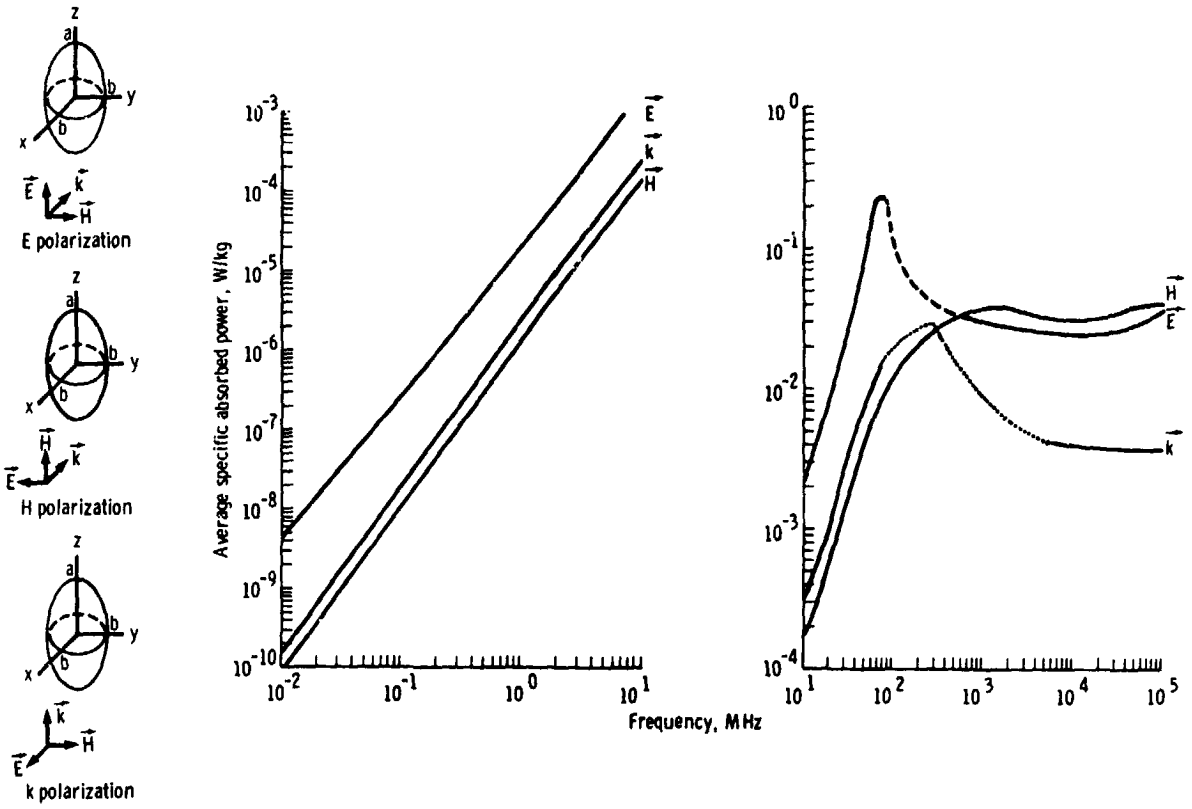
(a) Media with high water content

RF radiation		Muscle, skin, and tissues							
Frequency, MHz	Wavelength in air, cm	Dielectric constant ϵ_M	Conductivity σ_M, S/m	Wavelength[a] λ_M, cm	Depth of penetration,[b] cm	Reflection coefficient			
						Air/muscle interface		Muscle/fat interface	
						r	ϕ	r	ϕ
1	30 000	2000	0.400	436	91.3	0.982	+179	--	--
10	3 000	160	.625	118	21.6	.956	+178	--	--
27.12	1 106	113	.612	68.1	14.3	.925	+177	0.651	-11.13
40.68	738	97.3	.693	51.3	11.2	.913	+176	.652	-10.21
100	300	71.7	.889	27	6.66	.881	+175	.650	-7.96
200	150	56.5	1.28	16.6	4.79	.844	+175	.612	-8.06
300	100	54	1.37	11.9	3.89	.825	+175	.592	-8.14
433	69.3	53	1.43	8.76	3.57	.803	+175	.562	-7.06
750	40	52	1.54	5.34	3.18	.779	+176	.532	-5.69
915	32.8	51	1.60	4.46	3.04	.772	+177	.519	-4.32
1 500	20	49	1.77	2.81	2.42	.761	+177	.506	-3.66
2 450	12.2	47	2.21	1.76	1.70	.754	+177	.500	-3.88
3 000	10	46	2.26	1.45	1.61	.751	+178	.495	-3.20
5 000	6	44	3.92	.89	.788	.749	+177	.502	-4.95
5 800	5.1.	43.3	4.73	.775	.720	.746	+177	.502	-4.29
8 000	3.75	40	7.65	.578	.413	.744	+176	.513	-6.65
10 000	3	39.9	10.3	.464	.343	.743	+176	.518	-5.95

(b) Media with low water content

RF radiation		Fat, bone, and tissues							
Frequency, MHz	Wavelength in air, cm	Dielectric constant ϵ_L	Conductivity σ_L, mS/m	Wavelength[a] λ_L, cm	Depth of penetration,[b] cm	Reflection coefficient			
						Air/fat interface		Fat/muscle interface	
						r	ϕ	r	ϕ
1	30 000	--	--	--	--	--	--	--	--
10	3 000	--	--	--	--	--	--	--	--
27.12	1 106	20	10.9 to 43.2	241	159	0.660	+174	0.651	+169
40.68	738	14.6	12.6 to 52.8	187	118	.617	+173	.652	+170
100	300	7.45	19.1 to 75.9	106	60.4	.511	+168	.650	+172
200	150	5.95	25.8 to 94.2	59.7	39.2	.458	+168	.612	+172
300	100	5.7	31.6 to 107	41	32.1	.438	+169	.592	+172
433	69.3	5.6	37.9 to 118	28.8	26.2	.427	+170	.562	+173
750	40	5.6	49.8 to 138	16.8	23	.415	+173	.532	+174
915	32.8	5.6	55.6 to 147	13.7	17.7	.417	+173	.519	+176
1 500	20	5.6	70.8 to 171	8.41	13.9	.412	+174	.506	+176
2 450	12.2	5.5	96.4 to 213	5.21	11.2	.406	+176	.500	+176
3 000	10	5.5	110 to 234	4.25	9.74	.406	+176	.495	+177
5 000	6	5.5	162 to 309	2.63	6.67	.393	+176	.502	+175
5 800	5.17	5.05	186 to 338	2.29	5.24	.388	+176	.502	+176
8 000	3.75	4.7	255 to 431	1.73	4.61	.371	+176	.513	+173
10 000	3	4.5	324 to 549	1.41	3.39	.363	+175	.518	+174

[a]In medium.
[b]Depth at which power density reduced to e^{-2} (13.5 percent).



(a) Frequency range of 10^{-2} to 10^{1} megahertz (ref. VI-3).

(b) Frequency range of 10^{1} to 10^{5} megahertz (ref. VI-4).

Figure VI-2.- Average specific absorbed power (or radiation) in a prolate spheroidal model of an average man, for the three standard polarizations; a = 0.875 meter, b = 0.138 meter, volume V = 0.07 cubic meter. Incident power density is 1 mW/cm^2.

From a few hundred megahertz to >100 gigahertz, the orientations to the E-field and H-field vectors are about equally effective.

In figure VI-3, similar calculations for a small rat are presented. A comparison between the graphs for man and those for the rat shows that, for a given power density at a given frequency, the amount of absorbed power in man can be orders of magnitude greater than that for the rat, whereas at higher frequencies, absorption can be orders of magnitude greater for the rat than for man. Thus, if some biological effect can be produced in a rat at a power density of 100 mW/cm^2, for example, the power density required to produce the same amount of absorbed power in man may be substantially different.

Although these calculations using a simple model provide only approximations of energy absorption, they serve to illustrate the following points.

1. The power density alone is a poor indicator of absorbed radiation.

2. The frequency is the primary factor in determining the absorbed power from a given power density. Assessment of risks and setting of exposure standards should be further divided into smaller frequency bands.

3. For humans, the frequencies near and beyond 70 megahertz are the most important to consider because the radiations are absorbed more readily.

4. Extrapolation of results from animal studies, performed at some specific frequency and power density, to an expectation for effects on man exposed at the same given frequency and power density may be grossly misleading.

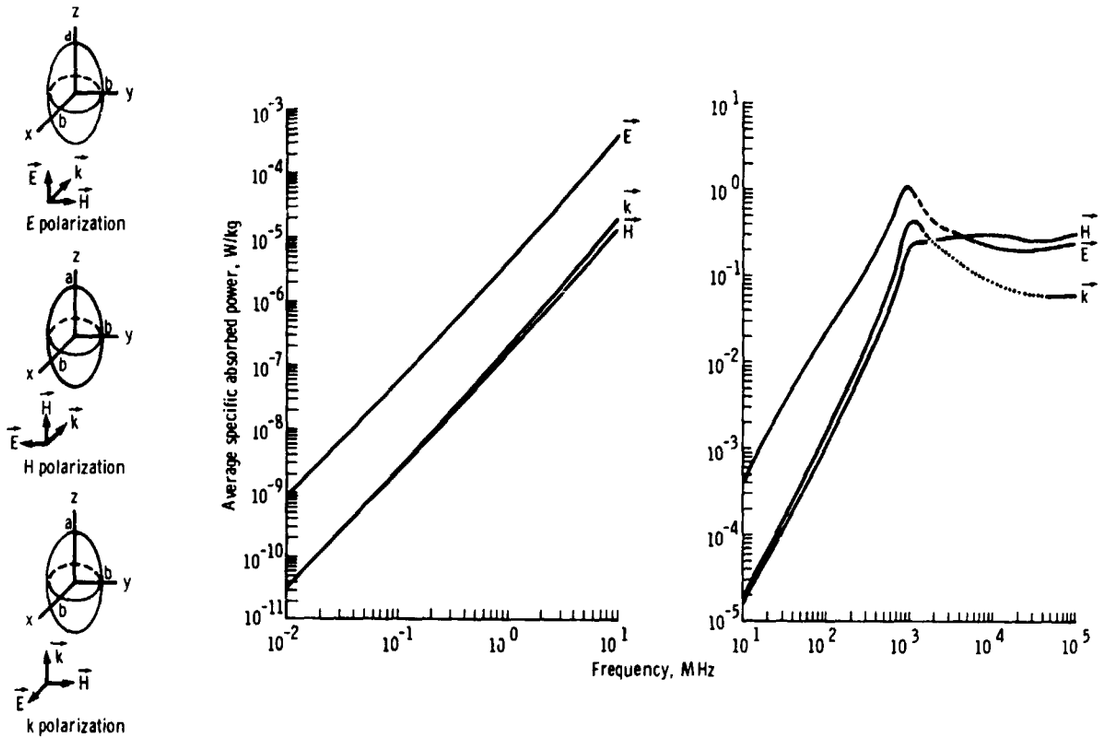
5. In setting exposure standards, the random orientation of persons to the various vectors of the beam should be considered; the most critical orientation should set the limiting factor.

For a more detailed discussion of the interaction of rf radiation with biological materials, references VI-3 to VI-5 may be consulted.

Thermal Effects on Organisms

Body temperature increase during exposure to rf radiation depends on (1) the specific area of the body exposed, (2) the efficiency of heat elimination, (3) the power density or field strength, (4) the duration of exposure, (5) the specific frequency or wavelength, and (6) the thickness of skin and subcutaneous tissue. These variables determine the percentage of radiant energy absorbed by various tissues of the body (refs. VI-6 and VI-7).

In partial body exposure under normal conditions, the body acts as a heat sink, which stabilizes the temperature of the exposed part. The stabilization is due to an equilibrium established between the energy absorbed by the exposed part of the body and the amount of heat carried away from it. This heat transport is due to increased blood flow to other parts of the body, which are maintained at normal temperature by heat-regulating mechanisms of



(a) Frequency range of 10^{-2} to 10^{1} megahertz (ref. VI-3).

(b) Frequency range of 10^{1} to 10^{5} megahertz (ref. VI-4).

Figure VI-3.- Average specific absorbed power (or radiation) in a prolate spheroidal model of a small rat, for the three standard polarizations; a = 0.07 meter, b = 0.0194 meter, V = 1.1×10^{-4} cubic meter. Incident power density is 1 mW/cm^2.

the body such as heat loss due to sweat evaporation, radiation, and convection. (See Ch. III.) If the amount of absorbed energy exceeds the optimal amount of heat energy which can be handled by the mechanisms of temperature regulation, the excess energy will cause continuous temperature rise with time. Fever and, under some circumstances, local tissue destruction can result (refs. VI-6 and VI-7). Not all parts of the body are equally susceptible to temperature rise; different parts vary in the ability to sense thermal stimulation and respond by increasing blood flow. The degree of innervation and vascularization together determine the body's response. Thus, the most susceptible parts of the body to localized heating are those which are not as well protected by these physiological mechanisms. Such areas include the eye lens, the testes, the gall bladder, and parts of the gastrointestinal tract. It has been shown that damage to these tissues can occur without significant rise in oral or rectal temperature (ref. VI-8).

Total body exposure of organisms the size of humans may be considered in large measure to be partial body irradiation. As seen in table VI-1, the depth of penetration may be only a few centimeters for many rf radiation frequencies. Fortuitously, as the frequency of the radiation and its associated photon energy increases, the depth of penetration decreases and allows the highly vascularized surface tissues to effectively dispose of the heat (ref. VI-8).

Nonthermal Effects in Organisms

There have been many reports of so-called nonthermal effects of rf radiation, especially by investigators in the Eastern European countries. The existence of true nonthermal effects of low levels of rf radiation has been challenged by American and Western European investigators. In part, the controversy reflects a difference in definition of nonthermal effects. The Eastern Europeans consider any biological effect of rf radiation that is not accompanied by a generalized heating of the organism to be a "nonthermal" effect. In contrast, the definition used by Western investigators allows for localized, hot-spot-type heating, even though unmeasurable because of technical difficulties.

Given the very low energy of the photons of rf radiation, it is most likely that almost all observed biological effects can be attributed to thermal effects (ref. VI-1). It does not appear to be fruitful to dwell further on any distinction between thermal and nonthermal effects.

PHYSIOLOGICAL EFFECTS OF RADIOFREQUENCY RADIATION

Systematic research on the biological effects of rf radiation was begun immediately after World War II. The results of these investigations are available in reports of the "Tri-Service Program" (ref. VI-9), reviews (refs. VI-1 and VI-10 to VI-14), books (refs. VI-15 to VI-19), and symposium proceedings (refs. VI-20 to VI-22). Glaser (ref. VI-23) has prepared a comprehensive bibliography of the literature on rf and microwave bioeffects. Some reported

effects are listed in table VI-2 (compiled from ref. VI-1). The listed effects are presented primarily as examples of the physiological systems and responses that have been studied. They include results with various experimental animals and with humans exposed experimentally (perception and pain) or occupationally (some central nervous system effects). The listed effects are not characterized in most cases as to animal size or frequency of rf radiation employed. Therefore, the effects should be judged only as examples of potential responses and not as indicating that rf radiation used in space at comparable power densities will have similar effects.

Extensive investigations into microwave bioeffects during the last quarter century show conclusively that, for frequencies between 1200 and 24 500 megahertz, exposure to a power density of 100 mW/cm^2 for 1 hour or more can produce pathophysiologic effects of a thermal nature. Such effects are characterized by temperature rise, which is a function of the thermal regulatory processes and active adaptation of the animal. The end result is either reversible or irreversible change, depending on the conditions of the irradiation and the physiologic state of the animal. At power densities below 100 mW/cm^2, however, evidence of pathological changes is nonexistent or equivocal (ref. VI-1).

The literature on the biologic effects of rf and low-frequency ($\leq$30 megahertz) electromagnetic radiation has been reviewed by several authors[1] (refs. VI-16 and VI-24 to VI-26). Bollinger (ref. VI-27) has reported on an extensive biomedical study of low-frequency rf radiation. Short-term (1 hour) exposures of monkeys to frequencies of 10.5, 19.3, and 26.6 megahertz, under experimental conditions which employed power densities of 100 to 200 mW/cm^2, did not produce discernible biologic effects.

Michaelson (ref. VI-1) has reviewed in greater detail the results briefly listed in table VI-2. He has pointed out the limitations in some of the data that make the data inapplicable for supporting the idea that exposures to low power densities cause pathological effects; for example, he points out the lack of appropriate controls in some cases, other mitigating circumstances (e.g., concurrent exposure to ionizing radiation) that might be responsible for an effect, and the reversibility of most of the phenomena observable at low exposure levels. He indicates that many of the biological responses observed with exposures at power densities less than 100 mW/cm^2 are not to be considered pathological but reflect physiological adaptations and stress reactions.

With regard to effects on the human central nervous and cardiovascular systems produced by exposure to low levels of rf radiation, Michaelson (ref. VI-28) summarizes the reported physiologic symptoms as being primarily subjective and consisting of fatigability, headache, drowsiness, irritability, loss of appetite, memory impairment, and chest pain. He reports that such psychic

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[1]Behling, U. H.: Biological Effects of Radio- and Low-Frequency Electromagnetic Radiation. U.S. Public Health Service, Bureau of Radiological Health, Biological Effects Division, 1969, pp. 1-7. (Unpublished manuscript.)

TABLE VI-2.- SOME BIOLOGICAL RESPONSES TO RADIOFREQUENCY RADIATION

[Compiled from ref. VI-1]

Response	Remarks
Perception of heat	13 to 59 mW/cm^2 for 4 sec at 3000 and 10 000 MHz (observations in humans).
Pain threshold	1800 mW/cm^2 for 60 sec at 3000 MHz (observations in humans).
Cataracts	Lens clouding when temperature of lens increases by 4 K (4° C). Accumulation of subclinical damage at low power densities for short durations may yield cataracts. Evidence still equivocal.
Reproductive detriment	
Testes	Intrascrotal temperature rise of >1 K (>1° C) by rf radiation or any other means reduces viable sperm count; this effect is usually reversible. Exposure to 2880 MHz at 5 mW/cm^2 for an indefinite period is the "threshold" for evidence of testicular damage in the most sensitive dog out of 35 dogs tested. Exposure to 3000 MHz at 8 mW/cm^2 did not affect mating of mice or rats.
Ovaries	No evidence that exposures to 10 mW/cm^2 or even somewhat greater interfere with reproduction in female mice.
Visceral effects	
Gastric ulcers	>100 mW/cm^2 for ≥10 min.
Delay of gastric secretion and emptying	0.05 to 1 mW/cm^2 for 30 min. Reversible.
Hematopoietic effects - Leukocytosis, lymphocytopenia, eosinopenia, red blood cell lifespan alteration, impaired bone marrow function, hemoglobin decreases, platelet decrease, reticulocytosis, etc.	Generally, long exposures to >10 mW/cm^2 are required to yield an effect. Effects are generally reversible.
Cardiovascular effects - Blood flow changes, blood pressure decrease, heart rate increase, etc.	Effects generally attributable to peripheral vasodilation and hemodilution in response to heat stress.
Central nervous system - Agitation, drowsiness, muscular weakness, electroencephalogram changes, avoidance behavior, altered conditioned response, decreased endurance, headache, etc.	Large number of studies; some with occupationally exposed humans; some conflicting results. Eastern Europeans claim effects <10 mW/cm^2; investigators in Western countries have not always observed these effects even at higher exposure levels. This is the area of greatest controversy.

changes as unstable mood, anxiety, insecurity, hypochondriasis, suicidal thoughts, delirium, terror, visual and auditory hallucinations, and sleep impairment have been observed. Among the objective symptoms cited by Michaelson are persistent dermographia, hyperhidrosis, unstable arterial pressure, retinal angiopathy, bradycardia, and disturbance in intraventricular conduction. Michaelson's conclusions are as follows. "Most of the subjective symptoms are reversible, and pathological damage to neural structures is insignificant. Most of the reports are based on subjective rather than objective findings. It should be noted that individuals suffering from a variety of chronic diseases may exhibit the same dysfunctions of the central nervous and cardiovascular systems as those reported to be a result of exposure to microwaves."

It is not clear whether the preceding reported subjective effects can be applied to a large fraction of the population, merely to a small, highly susceptible subpopulation, or to a subpopulation concomitantly affected by other factors. These observations, nonetheless, indicate that rf radiation stress at power densities below 10 mW/cm^2 may lead to performance decrements, perhaps similar to those occurring in hot environments.

PHYSIOLOGICAL LIMITS (STANDARDS)

Adequate protection of spacecraft personnel from potential rf radiation hazards should be achieved by using the rf radiation exposure standards of the American National Standards Institute (ANSI) or the American Conference of Governmental Industrial Hygienists (ACGIH) as criteria for spacecraft design and/or operational procedures. These standards plus those of other agencies and countries are listed in table VI-3.

The value of 10 mW/cm^2 listed as a maximum permissible intensity for continuous exposure reflects the simple physiological consideration that the amount of heat which the human body can transfer to the external environment is, under normal circumstances, approximately 10 mW/cm^2 of body surface. Under very favorable circumstances, this amount may be raised about tenfold; thus, the human body's ability to absorb rf radiation energy without causing a continuous temperature rise is limited to a value somewhere between 100 and 1000 watts. These values may be compared to the metabolic energy produced by a 70-kilogram man: about 100 watts at rest and more than 1000 watts during heavy labor. (See Ch. III.) Calculations indicate that, for most frequencies, only a fraction of the 10-mW/cm^2 intensity to which a human might be exposed would actually be absorbed (fig. VI-2). These considerations, plus a review of the extensive body of experimental data then available from the Tri-Service-sponsored studies (ref. VI-9), led a committee of the ANSI to recommend in 1966 the 10-mW/cm^2 value as the standard (ref. VI-31). They reaffirmed the standard in 1973 (ref. VI-1). The basic standard reads as follows.

> For normal environmental conditions and for incident electromagnetic energy of frequencies from 10 to 100,000 MHz the radiation protection guide is 10 mW/cm^2 as averaged over any possible 0.1 hour period.

TABLE VI-3.- OCCUPATIONAL SAFETY LIMITS FOR NONIONIZING RADIATION

[Compiled from refs. VI-1, VI-29, and VI-30, and Draft Report of Committee No. 12 of the International Electrotechnical Commission (unpublished)]

Average power density, mW/cm^2	Country and/or organization	Frequency range, MHz	Specifications
0.01	U.S.S.R., 1965	>300	Max. level for continuous exposure
	Poland, 1972	300 to 300 000	Limit of safe zone for stationary fields
	Czechoslovakia, 1968	300	Pulsed; 8 hr/day
0.025	Czechoslovakia, 1968	>300	Continuous wave; 8 hr/day
0.1	U.S.S.R., 1965	>300	2 hr/day
	Poland, 1972	300 to 300 000	Limit of safe zone for nonstationary fields
0.2	Poland, 1972	300 to 300 000	3-hr limit for stationary fields[a]
1.0	U.S.S.R., 1965	>300	20 min/day (protective goggles required)
	Poland, 1972	300 to 300 000	20-min limit for stationary fields[a] 8-hr limit for nonstationary fields[a]
	Germany, 1976 (draft standard) VDE 0865	30 to 300 000	Max. level for continuous exposure
	Sweden, 1977	300 to 300 000	Max. level for continuous exposure
5.0	Sweden, 1977	10 to 300	Max. level for continuous exposure
10.0	Poland, 1972	300 to 300 000	11.5-sec limit for stationary fields[a] 4.8-min limit for nonstationary fields[a]
	Germany, 1976 (draft standard) VDE 0866	30 to 30 000	1-hr limit for continuous exposure
	E.C.C.[b] (draft recommendation)	300 to 300 000	Max. level for continuous exposure, averaged each 0.1 hr
	United Kingdom, 1971	30 to 30 000	Max. level for continuous exposure
	United States (ANSI), 1973, adopted by OSHA, DOD[c]	10 to 100 000	Max. level for continuous exposure, averaged each 0.1 hr
	United States (ACGIH), 1978	300 to 300 000	8-hr limit for continuous exposure
25.0	United States (ACGIH), 1978	300 to 300 000	Maximum permissible exposure level; limited to 2.4 min out of each 6-min interval (radiation energy not to exceed 36 kJ m^2 (1 mW/cm^2) in any 0.1-hr period) during 8-hr work period
30	E.C.C. (draft recommendation)	300 to 300 000	2 min in any 0.1-hr period. No power density limit is specified but radiant energy must not exceed 36 kJ/m^2 (1 mW/cm^2) in any 0.1 hr period
	United States (ANSI)	10 to 100 000	
	United States, 1971	30 to 30 000	
50	U.S. Air Force, 1976	0.001 to 10	Continuous exposure
55	U.S. military	100 to 100 000	2 min/hr[d]

[a]Time limits at any power density are calculated by $t = 3.6/p^2$ for stationary fields and $t = 800\ p^2$ for rotating fields (e.g., radar antennas), where t = time in hours per day and p = average power density in watts per square meter. (1 mW/cm^2 is equivalent to 10 W/m^2.)

[b]European Communities Commission.

[c]Occupational Safety and Health Administration, Dept. of Defense.

[d]Time limits for power densities between 10 and 55 mW/cm^2 are calculated as $t = 6000/p^2$, where t = time in minutes per hour and p = average power density in milliwatts per square centimeter.

This standard does not set an upper intensity limit for very short term (<0.1 hour) exposures but sets a maximum energy density of 3.6×10^4 J/m^2 (1 mWh/cm^2) averaged over the 0.1-hour period (a time-weighted average). The ANSI standard permits indefinite exposure to a maximum power density of $\leq$10 mW/cm^2. The guide applies whether the radiation is continuous or intermittent. The Occupational Safety and Health Administration and the Department of Defense have adopted the ANSI recommendations as advisory guidelines (ref. VI-29).

These guides and exposure levels in force today appear to be entirely safe. So far, there is no documented evidence of injury to military or industrial personnel, or to the general public, from the operation and maintenance of radar equipment and other rf and microwave-emitting sources within the 10-mW/cm^2 limit of exposure (ref. VI-1).

Despite the apparent safety of the ANSI standards, microwave standards are undergoing frequent review and more detailed specification. The most recent statement on microwave standards is in "TLVs, Threshold Limit Values for Physical Agents Adopted by ACGIH for 1978" (ref. VI-30). The full text is as follows.

> [The following] Threshold Limit Values refer to microwave energy in the frequency range of 300 MHz to 300 GHz and represent conditions under which it is believed that nearly all workers may be repeatedly exposed without adverse effect.
>
> Under conditions of moderate to severe heat stress the recommended values may need to be reduced.* Therefore, these values should be used as guides in the control of exposure to microwave energy and should not be regarded as a fine line between safe and dangerous levels.
>
> Recommended Values:
>
> The Threshold Limit Value for occupational exposure to microwave energy, where power density or field intensity is known and exposure time is controlled, is as follows:
>
> 1. For exposure to continuous wave (CW) sources, the power density level shall not exceed 10 milliwatts per square centimeter (mW/cm^2) for continuous exposure and the total exposure time shall be limited to an 8-hour workday. This power density is approximately equivalent to a free-space electric field strength of 200 volts-per-meter rms (V/m) and a free-space magnetic field strength of 0.5 ampere-per-meter rms (A/m).

*Mumford, William Walden: Heat Stress Due to RF Radiation. Proc. IEEE, vol. 57, no. 2, Feb. 1969, pp. 171-178.

2. Exposures to CW power density levels greater than 10 mW/cm^2 are permissible up to a maximum of 25 mW/cm^2 based upon an average energy density of 1 milliwatt-hour per square centimeter (mWh/cm^2) averaged over any 0.1 hour period. For example, at 25 mW/cm^2, the permissible exposure duration is approximately 2.4 minutes in any 0.1 hour period.

3. For repetitively pulsed microwave sources, the average field strength or power density is calculated by multiplying the peak-pulse value by the duty cycle. The duty cycle is equal to the pulse duration in seconds times the pulse repetition rate in Hertz. Exposure during an 8-hour workday shall not exceed the following values which are averaged over any 0.1 hour period:

Power Density	10 mW/cm^2
Energy Density	1 mWh/cm^2
Mean Squared Electric Field Strength	40,000 V^2/m^2
Mean Squared Magnetic Field Strength	0.25 A^2/m^2

4. Exposure is not permissible in CW or repetitively pulsed fields with an average power density in excess of 25 mW/cm^2 or approximate equivalent free-space field strengths of 300 V/m or 0.75 A/m.

These standards appear to be the appropriate ones for the physiological limits to consider in spacecraft design and operations.

The ACGIH standards (ref. VI-30) apply only to the frequencies 300 megahertz to 300 gigahertz, but the ACGIH has under study rf radiation of frequencies from 10 to 100 megahertz. The possibility exists that future standards may be more specific as to frequency (ref. VI-8). For example, Rogers and King (ref. VI-32) suggest that under plane-wave (far field) conditions, the body could endure an rf radiation power density greater than 10 mW/cm^2 (E-field strength = 200 V/m) for frequencies in the high-frequency band (3 to 30 megahertz) and suggest that an electric field strength of 1000 V/m can be considered the safe limit for continuous daily exposure to rf radiation in the frequency range below 30 megahertz.

Mention should be made of the lower standards established by the Eastern European countries. (See table VI-3.) These lower standards reflect the industrial hygiene philosophy of the U.S.S.R., which, according to Magnuson et al. (ref. VI-33), basically consists of the following.

1. The maximum exposure is defined as that level at which daily work in that environment will not result in any deviation from the normal state, as well as not result in pathological effects.

2. Standards are based entirely on presence or absence of biological effects without regard to the feasibility of reaching such levels in practice.

3. The values are maximum exposures rather than time-weighted averages.

4. Regardless of the value set, the optimum value and goal is zero. The U.S.S.R. maximum permissible exposure (MPE) values are not rigid ceilings, but, in fact, excursions above these values within reasonable limits are permitted and the MPE's represent desirable values for which to strive rather than absolute values to be used in practice. Thus, the standards used in the United States and in the U.S.S.R. are not as irreconcilable as they might appear (ref. VI-1).

RESEARCH NEEDS

The main areas of uncertainty in the application of the ANSI or ACGIH standards as design and/or operations criteria are as follows.

1. Dosimetric methods and models for studying the rf power densities or E- and H-field strengths at frequencies to be expected in various zones of the spacecraft or inside extravehicular activity (EVA) suits need to be developed to ascertain whether the limits might be exceeded. Consideration should be given to the absorbed power from mixed fields produced by focusing and scattering effects within both the spacecraft and the bodies of spacecraft personnel. Dosimetric units that include a weighting or quality factor based on the frequency of the rf radiation should be established.

2. The amount of heat which the human body can transfer to the environment of the space station in zero g, where natural convection does not occur, needs to be determined. The 10-mW/cm^2 value appropriate for Earth conditions may not be applicable in a zero-g environment.

3. The extent to which exposure to low power densities (<10 mW/cm^2) for extended periods will cause performance decrements by inducing headaches, fatigue, muscular weakness, irritability, etc., should be studied further.

4. The potential interactions of other environmental factors in space with the responses to rf radiation should be examined. For example, rf heating effects on the biological responses to ionizing radiation need further study. Also, additive thermal loading problems of rf radiation at 10 mW/cm^2 coinciding with heat stress induced by partial failures of environmental control and life support systems or with potential heat stress from heavy physical activity such as in EVA may cause a detrimental body temperature rise.

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VII. WEIGHTLESSNESS

By D. J. Grounds*

INTRODUCTION

The weightless environment of space flight has only been available for scientific investigation since the early 1960's. The ability of the human physiological system to adapt to this unique environment and subsequently to readapt to a one-g environment may ultimately limit the duration of manned space flights. This chapter consists of a description of the general physiological effects of weightlessness, with emphasis on the physiological effects that could limit mission durations in the absence of effective countermeasures. The "human factors" of weightlessness in the design of spacecraft facilities and equipment (i.e., anthropometric changes, habitability, man-machine interface) are not included in this document, and the interested reader is directed elsewhere for this information (ref. VII-1).[1] The influence of weightlessness on food systems and hygiene facilities that could affect man's physiological state is not considered in this chapter. Since it has been demonstrated in previous space flights that adequate services can be provided, they do not represent an area of major concern.

DEFINITION AND DESCRIPTION

The gravitational, inertial, and external forces that produce weight, weightlessness, and reduced gravity are defined in the context of this chapter. Care should be taken not to interpret the zero-g state as being outside the influence of the Earth's (or some other celestial body's) gravitational field. The gravitational attraction between any two bodies is given by Newton's familiar equation $F = g_o(m_1 m_2/r^2)$, where F is the force of gravity, g_o is the universal gravitational constant, m_1 and m_2 are the masses of the two bodies, and r is the distance between them. For a body (e.g., spacecraft or man) in low Earth orbit, the distance between the orbiting body and the Earth's surface may differ by only a small fraction of the Earth's radius. This relative change in distance (r) is not great enough to affect seriously the gravitational forces as calculated by Newton's equation

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[1]Spacecraft Design Division: Habitability Data Handbook, Vol. 1: Mobility and Restraint. NASA Lyndon B. Johnson Space Center rep. MSC-03909, July 31, 1971.

(about 5 percent at an altitude of 1111 kilometers (600 nautical miles)). However, because the inertial forces of an orbiting body exactly balance the gravitational forces, the resultant forces impinging on the body have a magnitude of zero. This state is called zero g, null gravity, or weightlessness.

It is only during space flight that zero g can be produced for any extended period. However, other methods are available for producing the zero-g state for brief periods within the Earth's atmosphere. Aircraft flying a Keplerian parabolic trajectory have produced periods of weightlessness of as long as 60 seconds (ref. VII-2). These brief periods of zero g have limited usefulness for investigations of physiological effects of weightlessness but are most useful for developing and evaluating space-flight hardware and procedures. Other methods are also available by which certain effects of the weightless environment on living systems can be simulated more or less realistically, depending on the phenomena under observation. Bed rest and liquid immersion have been used for many years to simulate the effects of space flight on the human cardiovascular system, musculoskeletal system, and fluid, hormonal, and electrolyte control system (refs. VII-3 to VII-7). The degree to which these stresses are analogous to zero g is the subject of controversy even today (ref. VII-8). Given the complexity of the systems under study, the controversy may not be resolved in the foreseeable future. Nevertheless, these methods provide an opportunity to test hypotheses and evaluate countermeasures in areas where physiological effects similar to those observed in zero g are known to occur.

PHYSIOLOGICAL EFFECTS

Zero g is known to affect several physiological systems including cardiovascular, vestibular, fluid and electrolyte control, other hormonal, and musculoskeletal. Many of the changes that occur are not fully realized until return to the one-g environment.

Gravitational forces have been present in the evolutionary development of every species of land animal and plant. The anatomical placement and mass of the skeleton and musculature of land animals have subsequently evolved to maintain posture and provide locomotion against these gravitational forces. This observation is illustrated by the fact that the skeletal systems of terrestrial mammals have relatively greater mass than those of aquatic mammals, which benefit from buoyancy forces.

To enable man and other animals to acquire and maintain posture and bodily orientation in normal gravity, the central nervous system interprets visual, kinesthetic, vestibular, and statokinetic signals to produce the "appropriate" gravity-dependent sensorimotor response. Both the mechanoreceptors within the body and the learned central nervous system interpretations are tuned to the terrestrial weight/force relationship. Further, the cardiovascular systems of man and of other animals have evolved anatomical structures and physiological mechanisms (e.g., carotid sinus reflex) to overcome changes of orientation with respect to gravitational forces.

In essence, the "stress" of zero g is the removal of forces to which the body is adapted and for which the body is genetically designed to counteract. Adaptation to the zero-g environment involves disuse or modified use of these structures and mechanisms. Rapid and complete adaptation to a zero-g environment is desirable for enhanced performance during space flight. However, the degree of adaptation during zero-g exposure may affect the severity of problems encountered upon return and readaptation to the one-g environment. Zero-g effects in some major physiological areas are summarized in the following paragraphs.

Energy Metabolism

Data gathered before and during the three manned missions of the Skylab Program (SL-2, SL-3, and SL-4; 28, 59, and 84 days duration, respectively) do not show a change in metabolic energy requirements during space flight that could be extrapolated for extended mission durations. The overall energy consumption during flight was not statistically different from preflight controls. However, results of Skylab energy balance studies show that energy consumption was below preflight levels during the first in-flight month and was approximately equal to the preflight levels for the second and third in-flight months (ref. VII-9). The decreased energy use reported during the earlier in-flight period is likely due to the anorexia of acute space motion sickness, which lasts for 3 to 5 days. With the use of individual direct measurements of the gas exchange level, U.S.S.R. investigators have found both increases and decreases in resting oxygen consumption during flight (refs. VII-10 and VII-11). Further investigations will be required to determine the effect of space flight on basal metabolic rates. The metabolic requirements to perform exercise during space flight were slightly decreased in six of nine Skylab astronauts (ref. VII-12). Currently available data on overall energy metabolism do not as yet suggest a limit to flight duration.

Cardiovascular

The most significant effects of cardiovascular changes due to space flight are noted during the period immediately after flight. The ability to tolerate the upright posture in one g (orthostatic tolerance) is reduced after space flight. This condition has subjective symptoms of lightheadedness, dizziness, weakness, and transient instability upon standing. Objective cardiovascular measurements show increased heart rate, decreased pulse pressure, and presyncopal indications. The signs and symptoms may be present for as long as 1 to 2 weeks after flight, and recovery durations have not been shown to be dependent on mission duration.

The changes in the human cardiovascular system as a result of space flight are thought to be a function of changes in extracellular fluid volume (including plasma volume), changes in the tone of venous capacitance vessels after fluid redistribution, and possible functional changes in pressure-regulating mechanisms (ref. VII-12). During Skylab missions, the cardiovascular adjustments due to weightlessness did not produce any impairment in the performance of in-flight exercise (ref. VII-13). However, a significant

decrement was observed in the response to submaximal exercise immediately after flight. The degradation was, in large part, evidenced by decreases in oxygen pulse, cardiac output, and stroke volume. These postflight changes have been interpreted as resulting from decreased venous return due to readjustments in body fluid volumes and distributions (ref. VII-13).

Lower body negative pressure (LBNP) was also employed during flight as well as after flight during Skylab missions for assessment of orthostatic tolerance. Differences between in-flight responses and preflight responses to LBNP stress included greater heart-rate and leg-volume increases in all crewmen and, in most, higher diastolic pressures and mean arterial pressures and lower systolic blood pressures and pulse pressures. The loss of orthostatic tolerance had developed by the first in-flight tests performed after 4 to 6 days of flight. The greatest instability and decrement in orthostatic tolerance occurred during the first 3 weeks of flight. After approximately 5 to 7 weeks, cardiovascular responses became more stable and evidence of improved orthostatic tolerance appeared (ref. VII-14). The response to LBNP immediately after flight paralleled those seen in the first weeks of flight despite evidence that blood volume displaced to the lower body was much smaller after flight than during weightlessness. These data indicate intense sympathetic activity and an adequate cardiac and peripheral arteriolar response (ref. VII-14). A special concern for these cardiovascular changes arises when acceleration forces ($+G_z$) are applied during crucial tasks (e.g., spacecraft landing). The effect of the reappearance of hydrostatic forces due to reentry accelerations may accentuate the effects of an inadequate circulating blood volume after even a few days in space. Skylab experience indicates that adequate protection against orthostatic hypotension during reentry and during the first few hours after flight can be provided by counterpressure garments. This countermeasure is indicated during any significant exposure to $+G_z$ accelerations, as will be experienced by the Space Shuttle crewmen. ($+G_z$ accelerations are further discussed in Ch. IV.) Recumbency can be of further benefit during the first few hours after flight in cushioning the cardiovascular effects of return to one g by preventing large shifts of intravascular fluids to lower extremity vessels and extravascular compartments (ref. VII-14).

Decreased heart sizes have been found from postflight chest films in all Apollo crewmen (marked by great individual variability) and from echocardiographic measurements made on Skylab crewmen (refs. VII-15 and VII-16). However, findings have not shown evidence of specific cardial effects. All measurements of cardiac size and performance returned to control values after 30 days of recovery. Cardiovascular effects of space flight and its associated recovery currently do not appear to pose a limit to the duration of space missions, as long as appropriate countermeasures (e.g., in-flight exercise, counterpressure garments) are provided.

Hematologic and Immunologic

Decreased red cell mass (RCM) following space flight has been a consistent finding in all U.S. space programs (ref. VII-17). Before the Skylab Program, the decrease in RCM had been thought to be caused by exposure to a

hyperoxic environment (100-p[illegible] oxygen at sea-level pressure before flight and hypobaric pressures during flight (34.0 kN/m² (258 torr)), since hyperoxia is known to inhibit [illegible] and to cause hemolysis (refs. VII-18 and VII-19). However, small reductions in RCM were observed during the Skylab missions, which were observed under normoxic conditions, the hyperoxic hypothesis for RCM decrease has been ruled out. Further experience with the Skylab flights has suggested that the decreased RCM does not become more pronounced with increasing flight duration. The changes observed for the Skylab missions were -14.3, -12.2, and -6.8 percent for SL-2, SL-3, and SL-4, respectively (ref. VII-17). A preliminary interpretation which has been placed on these data is that red cell mass is returning to preflight levels during the longer flights (ref. VII-17). However, further investigations are required to examine the effects of individual variation, exercise, and other in-flight factors before these conclusions can be drawn with certainty. The etiology of the reduction in red cell mass has been related to hemoconcentration secondary to plasma volume losses. The proposed mechanism for the decrease in circulating red cell mass is a decreased red cell production rather than increased destruction; however, verification of this hypothesis under space-flight conditions remains to be demonstrated. Plasma volume changes alone may not provide a complete explanation of this phenomenon, and other mechanisms are being sought (ref. VII-17).

From Skylab and Apollo-Soyuz Test Project (ASTP) experiments (refs. VII-20 and VII-21), it has been shown that the function of the human immune system is degraded after space flight as demonstrated by a decreased lymphocyte count and diminished response to a mitogenic challenge. Although the etiology of these changes remains speculative, the implications are that man may be more susceptible to disease in space. These findings call for maintaining present standards of microbial monitoring and providing medical services commensurate with duration of stay and exposed populations. Further, appropriate countermeasures should be considered to remedy the observed immunosuppression (e.g., gamma globulin injections).

Fluid, Electrolyte, and Endocrine

Fluid shifts in zero g have been mentioned previously in this chapter as contributing factors in both cardiovascular and hematological changes during space flight. Loss of extracellular fluid has been a consistent finding for flights longer than 2 days. When one enters the weightless state, the lack of hydrostatic forces allows blood and interstitial fluid to migrate from the legs toward the head and thereby to create a relative hypervolemia in the upper body (ref. VII-12). This process, which initiates several adaptive changes in fluid and electrolyte control, is illustrated in figure VII-1.

The total volume of the legs is reduced shortly after entering the zero-g environment as compared to preflight supine measurements. Direct leg-volume measurements on one ASTP crewmember, taken 6 hours after launch, showed that 1 liter of fluid had left the legs. By the second day, an additional 0.5 liter had been shifted headward. Further, data from the SL-4 mission show that leg volume was reduced 1.8 liters by the third in-flight day and had decreased to a deficit of 2.2 liters by the end of the 84-day mission

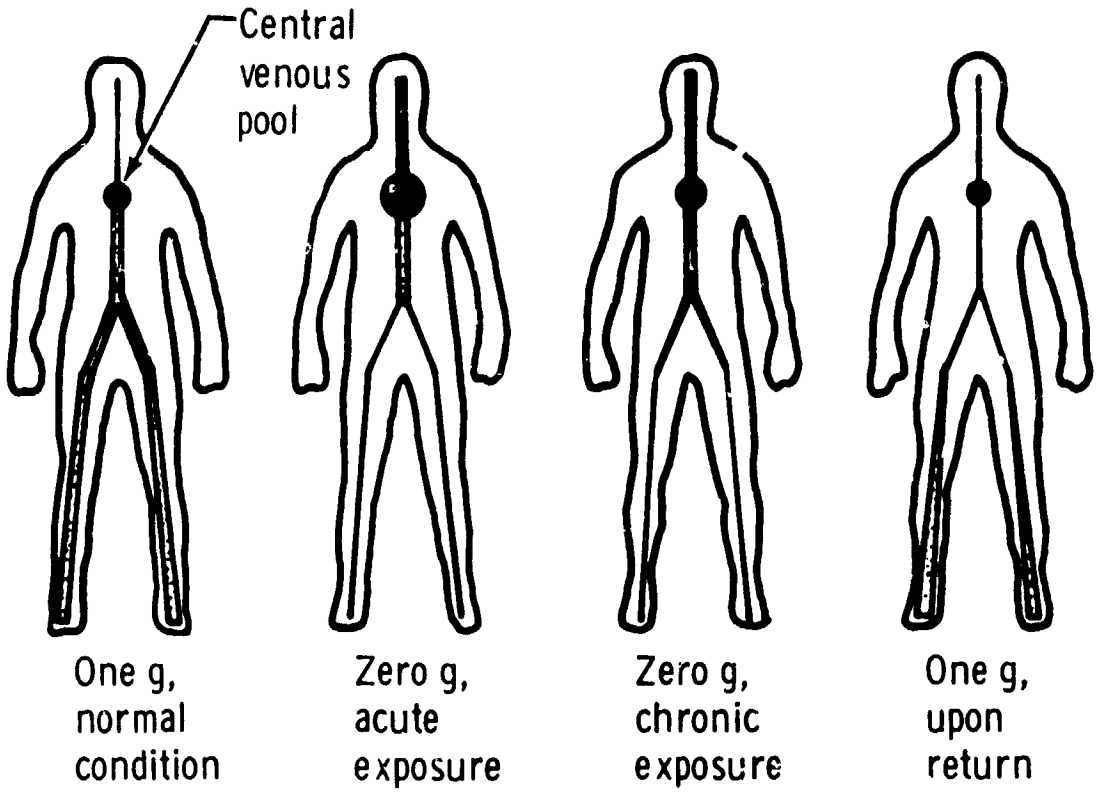

Figure VII-1.- Schematic representation of fluid shifts during zero-g exposure.

(ref. VII-12). These changes are illustrated in figure VII-2. It can be assumed that the fluid component of this volume shift is transferred to the vasculature of the upper body and from there is excreted from the body and/or is stored in vascular and extravascular spaces.

The concept that this fluid is transferred to the upper body and excreted through the kidneys is in accordance with the Henry-Gauer reflex (ref. VII-22). This concept can be briefly stated as follows: pressure receptors operating at the relatively low pressure of the left atrium sense an overfilling of blood volume and initiate renal hormonal reflexes for removing this excess fluid from the body. Although no such diuresis has yet been directly observed in U.S. space flight, a complete water balance analysis of the Skylab missions shows a decrease in total body water (TBW) of 1.5 liters by the third day of flight (ref. VII-23). The decrease in TBW without an observed diuresis was largely due to reduced water intake during the first few days in flight without a commensurate reduction in urinary excretion.

Total body water measurements and the in-flight water balance suggest that after the initial loss of fluid on the first few days of flight, TBW did not continue to decrease but remained approximately constant with a tendency to return toward control (ref. VII-23). At recovery, the Skylab data indicate a deficit of 0.8 to 1.1 liters in TBW (ref. VII-24). Since this quantity is less than leg-volume losses, it suggests that as much as 0.3 liter of fluid may have been stored in the upper body. If this fluid storage does occur, it can account for the reported in-flight symptoms of head fullness, nasal stuffiness, facial plethora, and distended neck and scalp veins (ref. VII-12). It is uncertain whether the excess fluid was accepted by the vascular or extravascular compartments in the upper body. Whether this fluid was available to supplement circulating blood volume during LBNP tests also is not known.

The fluid shifts discussed previously are causally related to a number of adjustments in the hormonal and electrolyte systems. A flow diagram that relates some of the environmental stimuli, observed changes, and hypothesized mechanisms is shown in figure VII-3. During Skylab, urinary sodium, potassium, chloride, nitrogen, and phosphorus are shown to increase during the in-flight phase (ref. VII-26). Some of the increased excretion of electrolytes is associated with a loss of body fluids. Sodium, for example, is a major ionic component of extracellular fluid, and even though urinary sodium excretion remains elevated throughout the flight period, extracellular fluids have been shown to level off or tend to return toward control levels (ref. VII-23). Also, increased urinary potassium losses are far too great to be completely associated with extracellular fluids. One plausible explanation for these observations is that a decrease in sweating occurred during the flight relative to preflight levels. Sweat losses and sweat composition have not been measured in previous flights, and further investigations are warranted to resolve these parodoxical findings. If the hypothesized decrease in sweating occurs, then causative mechanisms such as differences in free convection, metabolic activity, or sweat suppression (hydromieosis) should be examined.

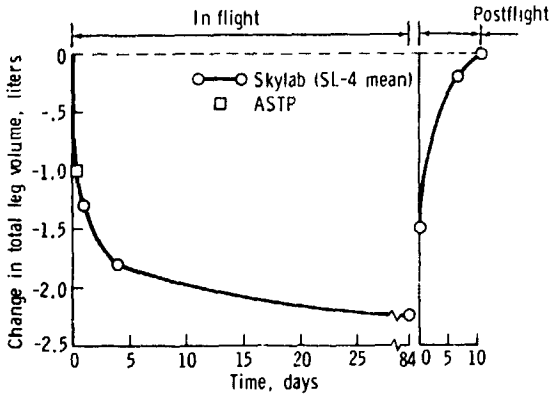

Figure VII-2.- Effect of space flight on total leg volume.

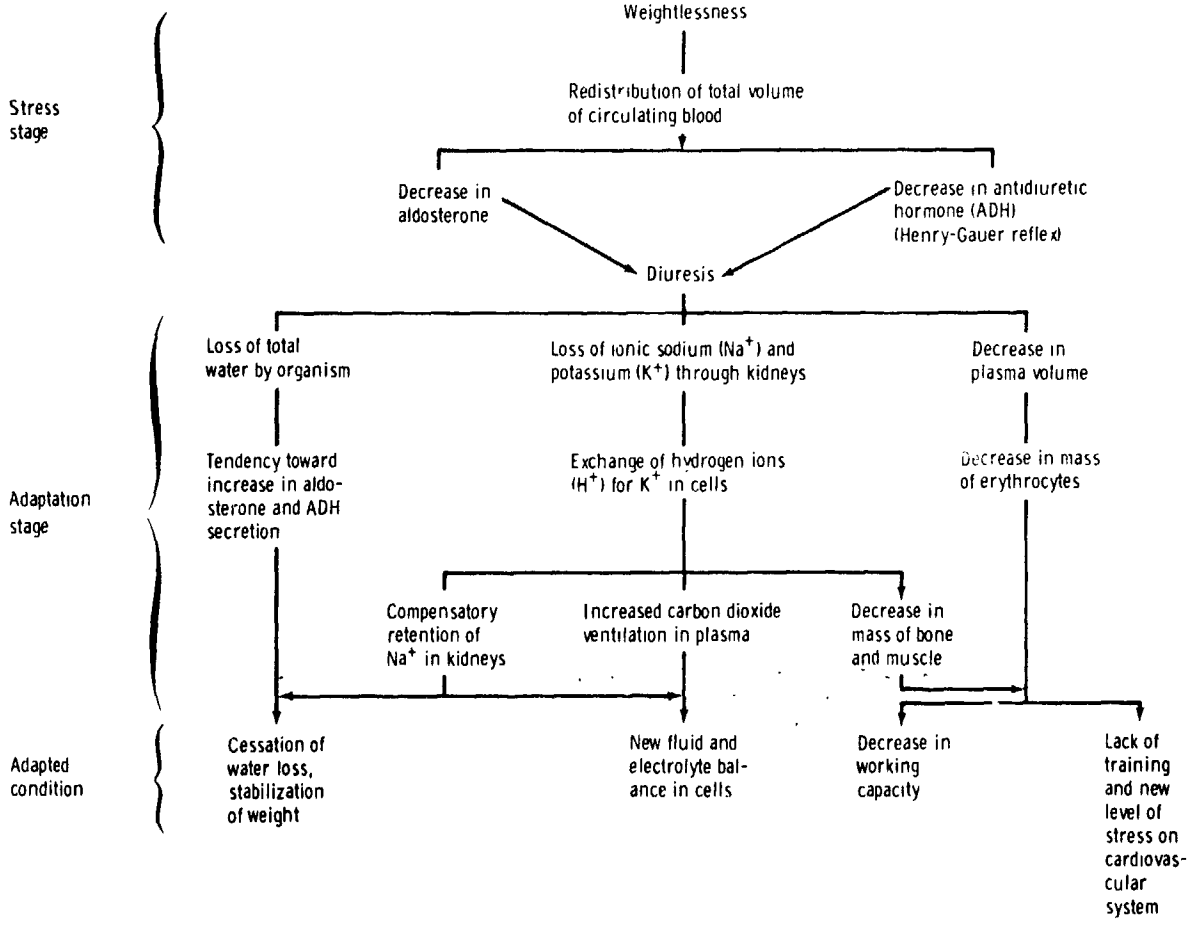

Figure VII-3.- Proposed process of adaptation to weightlessness. (From ref. VII-25.)

Other Skylab findings included a decrease in plasma sodium concentration during the in-flight period (ref. VII-26). Urinary levels of antidiuretic hormone were decreased during flight. This finding is in agreement with the Henry-Gauer reflex response mentioned earlier, and no tendency to adapt was demonstrated during the in-flight period (ref. VII-26). Aldosterone, a hormone produced in the adrenal cortex and normally associated with sodium and water retention, was increased in the urine during flight (ref. VII-26). Further investigations are required to explain fully these observations including the increased aldosterone excretion, but current hypotheses are based on the presence of a "third factor," or natriuretic hormone (ref. VII-27). Urinary cortisol, a hormone with general catabolic properties produced by the adrenal cortex, was increased during flight. Other urinary electrolytes and hormones were increased in flight, including inorganic phosphates, total 17-ketosteroids, creatinine, and urine osmolality. Decreases were found in urinary levels of uric acid, epinephrine, and norepinephrine. A great deal remains to be learned about the significance of these hormonal changes to the long-term health and well-being of people living in a zero-g environment. However, in the face of fluid shifts which in Skylab appear to be approaching a new homeostatic level, it would seem reasonable that related hormones are also reaching a new steady-state level. Other hormones, however, such as corticosteroids may have long-term effects (e.g., catabolism, immunosuppression) that may limit space-flight duration unless suitable countermeasures are developed. The influence of altered renal blood flow and other possible mechanisms of hormonal changes induced by space flight should be further investigated to enable timely development of these required countermeasures.

Musculoskeletal

Demineralization of bone tissue has been observed in returning space crewmen and in subjects of bed-rest studies (refs. VII-28 and VII-29). This process is evidently a consequence of decreased forces in the skeleton since such forces are closely related to bone mineralization (ref. VII-30).

From studies conducted during the Skylab Program, Smith et al. reported decreases in the bone mineral content of the os calcis (heel bone) in four of the nine Skylab astronauts, with greater average decreases found in each mission (ref. VII-31). Bone mineral losses of -4.5 and -7.9 percent were found immediately after flight in two of the SL-4 crewmen. Preflight control levels in these individuals were not regained until 95 days after flight. In their study, Smith et al. suggest a prediction formula for os calcis losses based on data from bed-rest subjects that is a function of the control levels of os calcis mineral, urinary hydroxyproline, and urinary creatinine (ref. VII-31). It has been concluded on the basis of knowledge about the reduced mechanical strength of bone that missions of 6 to 12 months duration could be safely undertaken (ref. VII-32).

The increased levels of urinary and plasma calcium measured during Skylab show the greatest losses within the first month of space flight.[2] Results of balance studies also show that the calcium losses continue with exposures of as long as 84 days in space. The calcium excretion in the feces shows an unabated increase, which eventually exceeds urine losses.[2] Whether these changes are due to an alteration in the feedback control of calcium absorption or due to increased active calcium secretion into the feces is not known.

Possible countermeasures that provide mechanical stress to the skeleton may be necessary for very long duration missions or for repeated zero-g exposures that may have cumulative effects. Other possible countermeasures include dietary and pharmaceutical control based on extended knowledge of the physiological mechanisms of calcium loss.

Decreases in bioelectric activity have been reported by U.S. and U.S.S.R. authors in the musculature of the neck and in back and pelvis muscles during bed rest and weightlessness (refs. VII-33 and VII-34). Also, as discussed previously, a decrease is thought to occur in the volume of leg muscles. Skylab data indicate a negative nitrogen balance, loss of exchangeable potassium, and altered amino acid metabolism.[3] These observations indicate a breakdown in muscle protein as a result of exposure to weightlessness, despite adequate dietary protein intake. Although other interpretations are possible, it is most likely that these changes result from muscle disuse atrophy.

In-flight exercise may constitute a significant countermeasure to some effects of hypogravity and hypodynamia. During the three Skylab missions, the amount of personal exercise increased with each successive crew and was particularly high for the SL-4 crew (ref. VII-35). The effect of increased in-flight exercise in the SL-4 crew is only one factor that may have influenced the recovery response. It is nonetheless tempting to speculate on the remedial effects it may have had on regaining exercise tolerance as well as on reducing orthostatic intolerance and RCM losses, which were less pronounced for SL-4 crewmen than for the other Skylab crews. Hence, until more experimental evidence is gathered specifically on the effects of exercise during prolonged weightlessness, in-flight exercise will be considered a valuable countermeasure for muscle deconditioning. Specific exercises for postural muscles could also be considered for future extended missions.

Vestibular

Exposure to the weightless space-flight environment poses two potential problems for man with regard to vestibular and related sensory system

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[2]Rambaut, P. C.; Leach, C. S.; and Whedon, G. D.: Calcium and Nitrogen Balance in Crewmembers of the 84-Day Skylab IV Orbital Mission. Acta Astronaut., in press.

[3]Leach, C. S.; Rambaut, P. C.; and Di Ferrante, N.: Amino Aciduria in Weightlessness. Acta Astronaut., in press.

function. The most significant of these is space motion sickness, which may occur within hours following transition into weightlessness. Although space motion sickness was not reported by any of the Mercury or Gemini crewmen, approximately 30 percent of the Apollo flight crewmembers experienced symptoms indicative of this syndrome (ref. VII-36). During the Skylab missions, approximately 50 percent of the crewmen experienced space motion sickness (ref. VII-37). Complete recovery from in-flight symptoms has typically occurred within 3 to 5 days. After this initial period of adaptation, crewmen have been virtually immune to the further development of motion sickness symptomatology, as evidenced by experimental data from Skylab (ref. VII-37). Upon return to one g, minimal problems with motion sickness have occurred following zero-g exposures of as long as 84 days.

The mechanisms underlying space motion sickness are not well understood. However, it is generally accepted that the otolithic receptors in the vestibular system have a significant role in the process. It is probable that upon entry into zero g, the otolith organs generate conflicting neural signals which mismatch signals from the semicircular canals, the visual system, the proprioceptors, and the touch-pressure-kinesthetic receptors. The initial inability of the organism to resolve the resultant sensory conflict may be the major causal factor in the space sickness syndrome. It has also been postulated that the cephalad shift of body fluids that is known to occur upon entry into weightlessness may contribute to space motion sickness. Research conducted to date has failed to substantiate the fluid shift theory.

To minimize the potential deleterious impact of space motion sickness on early-mission crew well-being and performance, suitable countermeasures for this syndrome must be identified. Currently, emphasis is being given to the development of more effective anti-motion-sickness drugs and special preflight vestibular adaptation training techniques. Additionally, efforts must be made to develop reliable predictive tests that may be used to select for space flight individuals minimally susceptible to space motion sickness.

The second vestibular system effect that may occur as a result of zero-g exposure is much more speculative in nature. It involves potential irreversible or very slowly reversing functional and/or anatomical alterations that may occur in the gravity receptors as a result of very long duration exposure to zero g. It is known, for example, that in mammals, the otoconia (small calcite crystals) are constantly interacting with their environment as evidenced by calcium ion exchange measurements in rats.[4] The prolonged absence of gravitational forces could conceivably influence calcium metabolism in the otolith organs and cause subtle alterations in the mass or distribution of the otoconia. As a result, sensitivity to linear accelerations could be altered and could cause perceptual illusions during subsequent exposure to acceleration forces. Other possible adverse reactions encountered upon return to one g could include ataxia, postural dysequilibrium, and altered motion sickness susceptibility. Similarly, other neurosensory and neuromuscular adaptive changes may occur that are appropriate for the zero-g environment

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[4]Ross, M. D.: Calcium Metabolism of the Gravity Receptors. Personal communication.

but inappropriate for the one-g environment. Indeed, limited Apollo and Skylab data indicated several postflight response changes including ataxia, dysequilibrium (ref. VII-38), and altered susceptibility to motion sickness (ref. VII-37). However, fairly rapid and complete recovery from these changes did occur even after zero-g exposures of as long as 84 days.

Beyond the initial period of adaptation when motion sickness and/or spatial disorientation may occur, there is no known limit to the duration of vestibular system exposure to zero g. Whether or not subtle anatomical and/or functional sensory system alterations induced by prolonged zero-g exposure will manifest themselves in the form of serious sensorimotor or perceptual disturbances upon return to one g remains to be determined. If the results of required human and animal research that hopefully will be performed during the Space Shuttle/Spacelab era indicate trends toward debilitating changes, then countermeasures may become necessary. These countermeasures may include limiting human exposure to zero g to safe durations or providing some level of artificial gravity during all or designated portions of long-duration exposures (e.g., interplanetary travel or space colonization). It must be recognized, however, that the creation of artificial gravity can itself become a source of stressful stimuli for the vestibular system because of the Coriolis effects that would be experienced by individuals moving about in a rotating space habitat (refs. VII-39 and VII-40). Hence, careful planning would have to be done in the design and use of artificial gravity.

PHYSIOLOGICAL LIMITS

No hard limits have yet been established for continuous or cumulative exposure to the weightlessness of space flight. However, several physiological effects mentioned in the previous discussion could ultimately impose a limit on zero-g exposure. Bone demineralization is the most apparent effect of weightlessness that could limit flight durations. Degeneration of gravity receptors is another potential limiting factor for space-flight duration; however, the nature and time course for the development of this effect remain speculative. Both of these effects are only realized with reappearance of gravitational or other external forces. Other physiological effects of weightlessness, such as cardiovascular deconditioning and immunosuppression, may not be current limiting factors to space-flight duration but will require monitoring and deployment of appropriate countermeasures.

Exposure limits must be derived through further experimentation and/or progressively increased exposures to zero g. When established, these limits will likely be a function of the availability of effective countermeasures. Moreover, with sufficient progress in these countermeasures, weightlessness as such may not be the most critical factor for limiting long-duration space flights.

Experience with space flight to date has shown that man can live and work in space for periods of as long as 84 days. It has also shown that the most deleterious effects of zero-g exposure may not be realized until return to one g and subsequent readaptation. Also, from physiological evidence

amassed through extensive experimentation and data analysis in cardiovascular, hematological, endocrinological, vestibular, and musculoskeletal areas, it can be stated that the allowable exposure period to zero g can be safely extended to 6 months. Physiological measurements and performance parameters should be monitored during these extended missions to detect debilitating changes that might occur. This approach of systematically increasing exposure times can be continued until physiological limits are approached or until operational goals are realized.

RESEARCH NEEDS

A number of research needs have been noted throughout the discussion of each physiological area together with related countermeasures. It should be mentioned that the physiological effects of weightlessness have, heretofore, only been measured in the astronaut and cosmonaut populations. An adequate data base in each physiological area should be compiled on the response to zero g of the much more diverse populations (i.e., age, sex, physical conditioning) that will be using the Space Shuttle in the near future.

Research will be necessary to study the interaction of physiological adaptation during extended-duration space flight and unusual physical stresses and disease states which may occur. These interactions must be recognized for accurate diagnosis and effective treatment.

In general, physiological effects of zero g should be studied where an interaction with environmental stresses may produce different performance or exposure limits from those established under one-g conditions.

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15. Supplementary Notes

16. Abstract

This document is intended to provide a description of the physiological effects that determine the environmental limits required in spacecraft. The existing limits for operational environments are described in terms of acceptable physiological changes. Tolerance limits are discussed for exposures to environmental factors during unusual or contingency situations. Where environmental limits may be required but do not presently exist or where additional research is required to refine existing limits, these research needs are specified. Background information describing physiological systems is presented as required to support the development of physiological limits. References to general works in the physiological area of interest are included for the interested reader. The historical development of physiological limits used in the U.S. manned-space-flight program is also cited as necessary to show the development of the limits currently in use.

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