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CHAPTER 12

INJURIES FROM METEOROID PENETRATION

Knowledge to date has indicated that the probability of penetration of a spacecraft cabin wall or space suit by a meteoroid during missions along relatively meteoroid-free routes in space is quite low [12, 45, 52]. Uncertainties still remain, however, for a great deal more data is required on the frequency, density and frangibility of meteoroids, and on the particle characteristics for penetration of various spacecraft wall and space suit materials. Accordingly, it is apparent that in the face of current optimism, attention must still be given to the possibility of having to treat in space any one or more of a number of injuries which could result from the primary (flash, heat, blast, projectiles) or secondary (e.g., decompression, fire, electrical disruption) effects of meteoroid penetration. This chapter briefly discusses the meteoroid hazard, and the characteristics, diagnosis and treatment of injuries thought possible from meteoroid penetration of the spacecraft cabin wall and space suit.

Meteoroid Hazard

Current information on the meteoroid environment in space can be obtained from the studies of Cosby and Lyle [12] and others [7, 15, 22, 28, 45, 50, 52, 53]. The word "meteoroid" is a general term applied to particles of matter traveling in space. Ninety percent of meteoroids are presumably of cometary origin; the remainder are of asteroidal origin [50]. Seventy percent apparently travel in random directions through space; the remainder travel in streams (45). The majority of meteoroids are thought to be highly porous and frangible with densities as low as 0.5 gm/cm^3 (cometary meteoroids) [28, 37]. A few denser meteoroids have predicted densities of up to 9 gm/cm^3 (asteroidal meteoroids) [22]. The geocentric velocity of meteoroids apparently varies from 11 to 72 Km/sec [28].

Since the Earth passes through streams of meteoroids traveling in heliocentric orbits, the meteoroid flux in the near-Earth environment varies at regular intervals during the calendar year. This flux is highest

in the summer months (May through October) and lowest during the late winter months (January through April) [7]. The various known meteoroid streams and their orbits have been discussed by Burbank and Cour-Palais [7]. Such information could prove quite valuable in mission planning, especially if potentially penetrating particles are present in these streams.

The best data to date on the probability of meteoroid penetration of various thicknesses of aluminum are summarized graphically in Figure 12.1. The best estimate curve shows that for a spherical shell 3 meters in diameter, penetration of an 0.03 cm thick aluminum wall would occur on the average of about every 2.3 years. There is, however, a level of uncertainty of about one order of magnitude surrounding this and other estimates of the penetration hazard [45]. Additional data, especially on meteoroid density and frangibility, are needed to establish more accurate estimates.

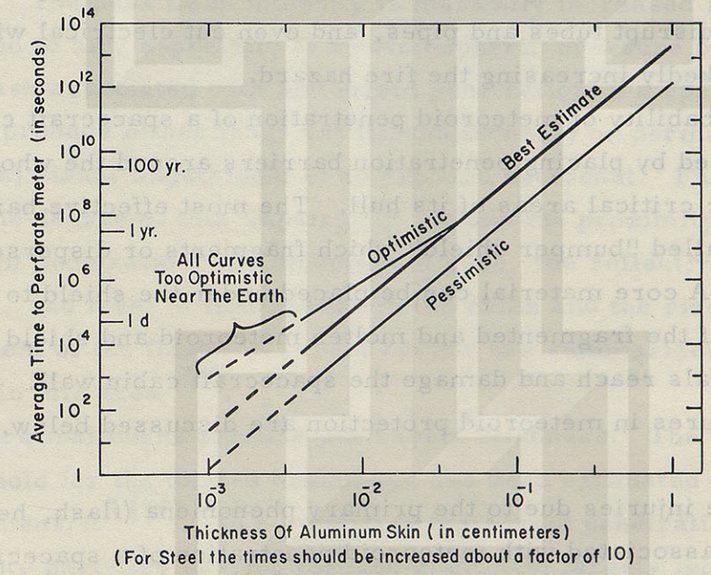

Figure 12.1 Time to meteoroid perforation of a spherical thin metal shell 3 meters in diameter (28 square meters in surface area).

(After Whipple [52]).

Injuries From Meteoroid Penetration of a Spacecraft Cabin Wall

Events and associated injuries from meteoroid penetration of a spacecraft wall have been described by Roth [45]. Pertinent to his considerations were various ground-based studies simulating penetration conditions [23, 33].

Meteoroid penetration of the spacecraft cabin wall might be partial or complete. A partial penetration might cause injuries if particles spall, or chip off the inner surface of the wall. If complete penetration occurs, vaporized molten and hot, fragmented, meteoroid and wall materials will be ejected into the cabin. The vaporized materials will immediately undergo "explosive oxidation", and hence constitute a flash, burn and blast hazard. Molten and hot fragmented materials could travel at high velocities, mechanically injuring an exposed astronaut and damaging equipment. Finally, fragmented meteoroid and wall could penetrate containers, disrupt tubes and pipes, and even cut electrical wires, thereby markedly increasing the fire hazard.

The probability of meteoroid penetration of a spacecraft cabin wall can be reduced by placing penetration barriers around the whole spacecraft or over critical areas of its hull. The most effective barrier appears to be a so-called "bumper shield" which fragments or disperses the meteoroid. A core material can be placed under the shield to accept the momentum of the fragmented and molten meteoroid and shield before these materials reach and damage the spacecraft cabin wall. Further design measures in meteoroid protection are discussed below.

Possible injuries due to the primary phenomena (flash, heat, blast, projectiles) associated with meteoroid penetration of a spacecraft cabin wall are discussed below. It must be kept in mind, however, that the severity of injury and consequent functional impairment of an exposed astronaut will also depend on the magnitude of secondary effects of penetration. Explosive decompression injuries (Chapter 3), acute hypoxia (Chapter 1) and ebullism (Chapter 2) might occur. The magnitude of the decompression hazard will depend on the size of the hole a meteoroid

makes in the cabin wall, the cabin volume, the initial cabin atmospheric pressure and emergency recompression and patching capabilities. There might also be risks of thermal, electrical and chemical burns from fires, disruption of electronic circuitry, and release of chemicals from life support and analytical systems (Chapter 13). Any one or more of a great variety of clinical problems might also result from the release of hazardous particles and droplets into the spacecraft cabin atmosphere (Chapter 8).

Light (Flash) Injury

The "oxidative explosion" of the vaporized meteoroid and spacecraft cabin wall materials will produce a flash which will probably last on the order of 1 millisecond [23]. The intensity of this flash will depend on the magnitude of the penetration, the composition of the wall, and the composition and pressure of the cabin atmosphere. Titanium alloys produce more severe flashes in 100 percent oxygen environments than do aluminum alloys [33]. The peak flash intensity is markedly increased in pure oxygen as compared to air, especially at an atmospheric pressure of 5 psia [23].

The flash associated with meteoroid penetration of a spacecraft cabin wall might produce either transient (flashblindness) or permanent (chorioretinal burn) visual impairment of an exposed astronaut. Factors influencing the degree of this impairment include the proximity of the eye to the site of penetration, the direction of gaze, the reflective properties of surfaces, and level of illuminance in the cabin and the size of pupil at the moment of the flash. A recent review has summarized current knowledge in this area [59].

Chorioretinal burns produce permanent blindness. The chorioretinal burn threshold for the dilated human eye has been estimated to be about 240,000 lumens/ft^2 [34]. In a simulated meteoroid penetration study, hypervelocity particles passing through aluminum targets into a 100 percent oxygen atmosphere at sea level pressure and at 5 psia produced light flashes as high as 273,000 lumens/ft^2 [23]. This value was reduced by 85 percent when the flash occurred in air at sea level pressure and in a 50 percent oxygen-nitrogen atmosphere at a pressure of 7.0 psia. Hence the concentration of oxygen in the spacecraft cabin atmosphere

could markedly influence the risk of blindness from meteoroid penetration of a spacecraft cabin wall. From these considerations, it appears that this risk will be low unless an astronaut is in a high oxygen environment and is in close proximity to the site of penetration. On the other hand, a varying degree of flashblindness is considered highly probable. Further study in this area is warranted, however.

Flashblindness is a transient reduction of visual acuity due to bleaching of visual pigment by a flash of light. The blind area usually occupies the central field of vision, so that useful vision is temporarily lost. The data of Severin and co-workers [21, 48] indicate that the recovery time of flashblindness probably varies between 6 and 48 seconds in the 100,000 to 240,000 lumens/ft^2 flash exposure range. The marked effect an increase in visual field illumination has in reducing visual recovery time has been demonstrated [26, 34, 48].

Various specific measures can be suggested for the protection of an astronaut's eyes from meteoroid flash, especially if a potentially hazardous meteoroid flux is predicted or encountered during a space mission. So-called "fixed filter" goggles use a selected filter of fixed density to absorb or reflect radiant energy before it enters the eye. Since these goggles would have to be worn continuously during high risk situations, there would have to be a compromise between the degree of filter opacity required for protection and the transmission necessary for adequate vision.

The monocular eye patch is simple and reliable, but has some obvious drawbacks [29]. Stereoscopic vision is lost and the visual field is restricted. If the exposed eye is injured, the covered eye would become the sole visual resource of the astronaut [59].

The effectiveness of "peep-hole" or eye slit shields is based on the fact that by reducing the angle of vision, the chance of a flash of light being directly focused on the retina is reduced [59]. The obvious limitation to this technique is restriction of the visual field. A variation of the eye-slit technique is the use of a visor, screen, of bilateral blinders to restrict the field of vision. Again, these measures would be effective

only if a flash occurred outside of the momentary field of view.

The most desirable all-around protective system would be clear eye shields impregnated with a phototropic substance which is darkened by intense light [59]. To be effective in a meteoroid penetration situation, the photochemical reaction time would probably have to be considerably less than 1 millisecond [33]. Various types of photoreactive devices are described in the literature [1, 20, 59].

A drug-induced miosis could serve to prevent chorioretinal burns and reduce the duration of flashblindness. Experiments have indicated that a pilot can perform instrument readings under minimum instrument illumination while his pupils are constricted, that eye discomfort from ciliary spasm was not a significant side-effect of this measure, and that based on the laws of optics, the limitation of light entering the eye should afford chorioretinal burn protection and minimize the duration of flashblindness [35]. However the potential usefullness of a miotic drug in a high risk space situation will require a complete assessment, especially with respect to visual-motor task performance under induced miosis in the simulated spacecraft environment.

There is no definitive therapy for either chorioretinal burn or flashblindness [34, 48].

Burns

Since the flash of "explosive oxidation" extended only a few inches into model spacecraft cabins in simulated meteoroid penetration studies, it is considered likely that the "explosive oxidation" produced by a smaller penetrating meteoroid particle in space will be highly localized in the spacecraft cabin [23]. Even so, there will be a risk of an exposed astronaut suffering a local burn if he is in close proximity to or in direct contact with the "explosive oxidation". Respiratory tract damage from the inhalation of hot gases and noxious fumes is also considered possible. Molten and hot meteoroid and wall materials ejected into the cabin could produce multiple small burns on body areas in direct line with the movement of these materials. Involvement of the eyes, especially corneal burns, could be extremely incapacitating. It should also be remembered

that these missiles could compound the risk of burns by starting fires in the spacecraft cabin.

Burns in general are discussed in Chapter 13. The treatment of corneal injuries is covered in Chapter 8.

Blast Injuries

The "explosive oxidation" of vaporized meteoroid and spacecraft cabin wall materials will not only produce a flash and a local increase in temperature, but also a local increase in pressure which will be rapidly propagated throughout the cabin. The blast hazard based on a comparison of findings in simulated meteoroid penetration studies with estimated pressure-duration relationships required for 50 percent lethality of adult humans has been assessed [45]. However, even though it was concluded that spacecraft penetrations of magnitudes similar to these studies would not cause lethal blast injuries to an astronaut sitting in the center of a 100 cubic foot cabin, many specific factors which are yet to be assessed could greatly modify this hazard. Some of these factors are pointed out below. One should also keep in mind that explosions in the cabin from other causes would produce injuries similar to those from a meteoroid blast.

The physics, biophysics, and pathophysiologic and clinical consequences of blast have been well documented in the literature. Excellent reviews written by Clemedson [11], White and co-workers [5, 13, 39, 56, 57, 58], Chiffelle [10] and others [14, 18, 30, 41, 45, 49] make it unnecessary to discuss this area in detail here. It is important to note, however, that even though a great deal of information has been obtained from the exposures of man and animals to bombs and other sources of blast, much is to be learned of the possible biologic consequences of blast in the closed cabin situation, particularly under conditions of lowered ambient atmospheric pressure and altered atmospheric pressure and gas composition.

The overpressure or "shock" wave of an explosion spreads out radially from its source. Peak overpressure (pressure above ambient atmospheric

pressure) and time-integral of overpressure (impulse) decrease exponentially, so that its injurious power is rapidly lost. The rising phase of a pressure wave hitting a target is steep unless propagation is slowed, as for example, by the interposition of barriers between the source and target. Extremely pertinent to considerations of explosions occurring in a confined volume such as a spacecraft cabin is the fact that pressure reflections occur when a blast wave impinges on a solid object. The magnitude of focused and reinforced reflection can be two to nine times greater than the incident pressure [58]. Hence, depending on such factors as the site of meteoroid penetration, the geometry of the cabin, the pressure reflective and absorptive properties of the cabin walls, and his position in the spacecraft cabin, an astronaut could be exposed to a steep, step-wise, injurious pressure loading from an otherwise non-injurious incident pressure pulse. The underpressure (pressure less than ambient atmospheric pressure) which usually follows the passage of a blast-produced overpressure will probably be reduced in a spacecraft cabin explosion by reflected pressure waves. The role which rapid or "explosive" decompression could play in altering the meteoroid blast hazard has apparently not been determined. Translational wind loading from a meteoroid blast should not be of a significant magnitude to cause injuries by imparting movement to an astronaut or by creating missiles of disrupted cabin structures. On the other hand, such traumatic events could result from associated rapid or "explosive" decompression (Chapter 3).

The amount of bodily injury an individual exposed to a blast overpressure will suffer is determined mainly by the rate of rise, magnitude and duration of the overpressure [5, 39, 55, 57]. The blast hazard in a closed environment such as a spacecraft cabin will probably be modified to a great degree by the step-wise characteristics of the overpressure rise, the atmospheric pressure, and the position and orientation of the individual to the incident pressure wave and reflecting surfaces [55]. Simulated meteoroid penetrations indicate that blast injuries following a meteoroid penetration in space will be due to extremely fast-rising

overpressures of short duration [23]. However, no simulations of penetration of large cabins have been performed.

Tissue damage from the absorption and passage of a blast wave through the body appears most commonly where density gradients of interfaces between tissue components are greatest [10]; however, it is considered possible that some tearing of tissues can result from shear stresses produced by acceleration of adjacent tissues of different densities [11]. Hence most blast pathology is seen within or in close proximity tc air and gas-containing organs, such as the tympanic membranes, lungs, gastrointestinal tract and paranasal sinuses.

Which of many proposed mechanisms is the primary one in producing the tissue-air interface type of injury cannot be stated, however [9]. Passage of a pressure wave across such a sharp density interface appears to have a bursting or shredding effect on tissues, but whether spallation actually occurs is not clear [11]. It might even be possible that the heat associated with bubble compression might be damaging [9]. Disruption of blood vessels might be caused by the forced displacement of blood due to inadequate equalization of pressures in internal body spaces with blast overpressures [58]. Air might even be forced into the circulation when the pressure pulse reaches air spaces into which vessels have ruptured.

Pure air blast injury is characterized by lesions in various internal organs without any signs of external injury [11]. The tympanic membranes are the structures most vulnerable to shock waves [30, 55]. In fact, the threshold for disruption is thought to be about 5 psia for fast-rising overpressures. Tympanic membrane disruption with or without dislocation of the ossicles is common, especially when the rise of overpressure is steep [30]. Structural damage of the organ of Corti can also result from violent inward displacement of the membrane-ossicular system.

The more serious pathophysiologic effects of blast are predominantly attributable to damage to pulmonary tissues. The major determinant of survival in the immediate post-exposure period is the amount of air which enters the vascular system through disrupted pulmonary and

bronchial veins, and capillaries at the time of blast and subsequently with each respiratory cycle [58]. Although it is not definitely known over how long a period this phenomenon can occur, the early steep portions of time-mortality curves of animals exposed to blast overpressures indicate that most air entry takes place within 30 minutes from the time of blast [45, 46]. Reflex vasoconstriction and blood clotting tend to stop air from entering the pulmonary circulation. Aggravating this embolization are coughing, partially obstructed airways, left cardiac decompensation and the ill-advised use of positive pressure breathing in treatment. There is no clear correlation between the degree of pulmonary hemorrhage and the occurrence of air embolism [10]. The air which enters the pulmonary venous system embolizes to the left heart, and thence to the systemic arterial system. By producing local ischemia in such vital tissues as the brain, spinal cord, and heart, these bubbles can cause a great variety of mild to serious, temporary or permanent clinical manifestations.

Air can also pass through disrupted alveolar walls into peribronchial tissues. It can dissect through these tissues into the hilum of the lung and even up into the subcutaneous tissues of the neck and face. In the mediastinum, air can exert pressure on large central vessels, restricting blood flow through them [24]. This serious consequence of mediastinal emphysema is considered unlikely except in extremely severe cases, however [9].

A blast wave can also produce a frank laceration of a lung leading to hemorrhage and the passage of air into the pleural cavity [11]. A torn pleural flap can have a flutter valve effect, allowing air to enter but not leave the pleural cavity. The resulting so-called "tension" pneumothorax can lead to severe cardio-respiratory embarassment and a potentially fatal situation within minutes.

If an individual survives the initial effects of a severe blast, he might be faced with severe respiratory embarrassment due to pulmonary hemorrhage and edema. A bacterial pneumonia is also more likely to occur in a severely damaged lung [9]. An irreversible consequence of blast injury of the lung is chronic respiratory embarrassment from severe

alveolar disruption and subsequent patchy fibrosis of pulmonary tissue (11, 55).

Gastrointestinal hemorrhage and perforation can be produced by shock waves striking the body (11, 58). Ensuing contamination of the peritoneal cavity can lead to fatal peritonitis. Yet another tissue-air effect of blast overpressures is bleeding from the paranasal sinuses.

Contusion of the heart, often associated with disruption of papillary muscles and chordae tendinae, has been observed in individuals exposed to severe blast overpressures (58). Liver and splenic hematomas and lacerations can also result from blast, presumably due to tissue shearing effects.

It is readily apparent that many causes of "shock" and death are possible following blast exposure. Other than the organ injuries completely incompatible with life, such as cardiac rupture, massive air embolization to the brain and heart appears to cause the majority of blast fatalities in the early post-exposure period. Asphyxia from pulmonary hemorrhage and edema, and cardiac decompensation due to myocardial ischemia can occur from minutes to days following blast. Pneumonia and peritonitis could lead to "shock" and death over a period of many hours to several days.

The clinical manifestations of blast trauma are extremely variable. An individual can be killed outright without external signs of injury, although blood-tinged froth or frank blood often appears in the nose and mouth. Survivors frequently suffer from air hunger, with rapid shallow respirations, and are usually quiet, apathetic or even lethargic for a period of time (10).

A marked reflex bradycardia, probably from the stimulation of stretch receptors in the carotic sinus and lungs by the pressure pulse, can cause an immediate profound hypotension which might be severe enough to produce faintness or loss of consciousness (10, 11). In moderate blast injuries, systemic arterial pressure may recover slowly over a period of several days (11).

A period of apnea, lasting several seconds to more than a minute in

duration usually follows exposure to a shock wave. If the lungs are severely injured by blast, breathing may be slow, shallow and weary, often with extreme expiratory dyspnea [11]. Panting respiration in other cases is usually associated with complaints of tightness across the chest and varying degrees of chest or abdominal pain. Coughing will occur, but not usually early [10]. Hemoptysis may appear, often well within an hour, and tends to be repeated [10]. Epistaxis may occur from nasal sinus hemorrhage. Frothy blood coming from the mouth and nose is usually a bad prognostic sign [11]. Physical exertion tends to aggravate pulmonary bleeding, hence giving strong support for early and complete immobilization of an individual injured by blast [10]. Severe lung damage, or cardiac damage, and secondary pulmonary edema can lead to cyanosis and "shock" from impaired ventilatory function.

Any number of clinical manifestations due to focal or diffuse damage of the central nervous system by air emboli can occur. These include unconsciousness, convulsions, general and local paralyses and disturbances in equilibrium. Myocardial ischemia produced by bubble emboli or secondary to contusion can produce anginal pain, fatal cardiac arrhythmias and myocardial insufficiency leading to pulmonary edema, extreme dyspnea, cyanosis, "shock" and death. Less massive air embolization may simply cause temporary chest or abdominal pain [58].

A large pneumothorax, particularly of the "tension" variety, can produce cardiorespiratory embarrassment characterized by severe chest pain, dyspnea, hemoptysis, cyanosis and "shock". Mediastinal emphysema can lead to cardiac insufficiency with much the same clinical picture.

Persistent diffuse abdominal pain with accompanying signs of peritonitis such as fever, vomiting, intestinal ileus, guarding, rebound tenderness and rigidity are usually indicative of a perforated hollow viscus. Confusing this clinical picture is the possibility that except for fever, these signs and symptoms might be present for several hours in a mild form without gastrointestinal perforation having occurred [58].

Damage to the tympanic membrane, and middle and inner ear structures by blast is indicated by ear ache, tinnitus, some degree of hearing

loss, and occasionally vertigo. Unruptured tympanic membranes may demonstrate hemorrhagic blebs and be the cause of serosanguineous oozing from the external ear [10]. When only membrane rupture occurs, there is usually a temporary low tone loss on the order of 10 to 30 decibels and a high tone loss of 40 to 80 decibels until healing occurs [30]. Ossicular disruption produces a permanent conductive deafness for all frequencies. Full or partial recovery from acoustic trauma to the middle ear may be slow, often several months in duration [30].

Possible late sequelae of blast include cardiac decompensation from extensive myocardial damage, recurrence of pulmonary hemorrhage, pneumonia, empyema and even lung abcess. They can become manifest and lead to "shock" and death up to several days after a blast exposure.

Recovery from blast injuries can be fast or slow, complete or incomplete. At best, especially if there have been neurologic manifestations, several days may be required for recovery, which may be surprisingly sudden and complete. Cardiac damage in blast tends to be permanent.

A careful medical assessment of an injured astronaut will be required as soon as possible after meteoroid penetration of a spacecraft cabin wall to determine not only the presence and severity of blast and other injuries mentioned in this chapter, but also to assess the immediate treatment needs of the astronaut. As a rule, the initial history and thorough physical will indicate the over-all injury severity. Close continuous observation will be necessary if there are any signs or symptoms of pulmonary, cardiovascular, nervous system or gastrointestinal involvement. Monitoring of the systemic arterial pressure and heart rate will bring to light any changes in cardiovascular status. If available on board a spacecraft, electroencephalography and electrocardiography might be used for establishing the presence of neurologic and cardiac involvement, respectively. X-rays could also be of some value in determining the degree of lung damage and the occurrence of visceral perforation.

Various measures which attenuate a blast overpressure or offer some

degree of body protection from the consequences of a blast wave can prevent or reduce the possibility of serious injury to an astronaut exposed to a meteoroid blast. The design of the spacecraft cabin structure so as to provide maximum attenuation of blast overpressures might be a highly effective measure. Although rigid thoracoabdominal shielding has been effective in reducing blast injury in animals, this measure might be too impractical for use in space [32, 62]. It should be pointed out, however, that lightweight thoracoabdominal shielding is also suggested for the protection of an astronaut from mechanical injuries while moving about in the spacecraft cabin (Chapter 14). Moreover, from discussion in Chapter 3, such shielding could also offer an astronaut a considerable reduction of risk of internally inflicted injuries from "explosive" decompression.

From the number and severity of different blast injuries and their sequelae, it is readily apparent that no outline of the definitive treatment of an astronaut who has been subjected to blast can be presented. Treatment must be based on sound clinical judgment, supported by a thorough understanding of possible blast effects.

Complete rest is considered to be absolutely essential for most blast injury cases [10, 27, 51, 58]. The work load on damaged lungs and heart must be minimized in order to reduce the risk of incurring further air embolization, pulmonary hemorrhage and edema, and cardiac decompensation. Sedation must be used with caution to prevent masking of various progressive signs which would indicate serious injury.

Positioning an astronaut in the head-down left lateral position in order to promote pulmonary drainage and minimize the migration of air emboli into his coronary and nervous system blood vessels, will obviously be of no therapeutic benefit in the weightless environment, but should be carried out under subgravity conditions. The early use of positive pressure breathing is contraindicated, for this measure may reopen alveolar-venous fistulae and introduce new showers of air emboli into the pulmonary veins [27]. Whole body pressurization to several atmospheres is a proven highly effective measure in the treatment of all air

embolic phenomena. For critical blast injuries, this measure would be required almost immediately after a blast, when most of the air embolization appears to occur [54, 58]. The installation of a recompression facility on board the spacecraft for the treatment of air embolic phenomena is considered under the treatment of decompression sickness in Chapter 4. Since a space suit pressure of possibly up to 5 to 7 psia over the cabin atmospheric pressure might be attainable, a potential mechanism is available for increasing the total body pressure as well as for maximizing the partial pressure of oxygen administered to an astronaut when a recompression facility is not on board the spacecraft.

The administration of 100 percent oxygen might be indicated for the treatment of local (e.g., cerebral ischemia, myocardial ischemia) and systemic (e.g., pulmonary insufficiency, "shock") hypoxia. Since pulmonary hypoxia and reflexes caused by lung damage markedly constrict the pulmonary vessels, administering pure oxygen, which is a proven pulmonary vasodilator, might conceivably aggravate the tendency for lung hemorrhage and edema, and air embolization [2, 11, 19, 42, 54]. Whether increased oxygen tensions can actually overcome the protective vasoconstriction in a lung with blast injury is still open to question, however. Favoring the use of oxygen in the immediate post-blast period is the fact that any bubbles of pure oxygen in the cardiovascular system will be much more rapidly absorbed than bubbles containing an inert gas such as nitrogen [46]. Hence the immediate administration of 100 percent oxygen - which probably will be part of the emergency decompression protocol following meteoroid penetration - should lower the risk of serious complications from air embolization. Because of the high mortality and serious sequelae of air embolization, oxygen should be used immediately after blast, in the hope that adequate reflex vasoconstriction and clotting will seal disrupted pulmonary vessels. Definitive studies on the effect of oxygen on vasoconstriction in a traumatized lung appear in order.

The question arises as to whether pure oxygen should be given beyond 30 minutes, which is considered the upper limit of the embolization period [54]. Cyanosis or other signs of hypoxia would be a definite indication for continuing

or resuming oxygen administration. Because of the pulmonary vasodilatory effect of oxygen, the period of protective vasoconstriction might be prematurely shortened by this therapy. This failure of vasoconstriction has been shown in animal experiments to aggravate bleeding from vessels, disrupted by blast, especially if clotting is inadequate [8]. Again further experimentation is indicated.

The atelectatic tendency of pure oxygen must be kept in mind, especially in instances of treating severe lung injury cases with this gas [44]. As well, it should be noted that the prolonged administration of oxygen at a partial pressure above 400 mm Hg, as could be accomplished in a space suit within a pressurized spacecraft cabin could not only lead to primary pulmonary effects of oxygen toxicity such as toxic bronchitis and pneumonia with hyperemia and edema, but also markedly exaggerate the pathologic effects of blast on the lung [36, 45].

Intravenous digitalization might be indicated for manifest cardiac decompensation [58]. A vasopressor, such as metaraminol, should be used primarily for the treatment of non-hemorrhagic "shock" [16]. Whether or not myocardial damage is suspected or evident, such a drug should probably be administered at the time of digitalization. Intravenous fluids should be used with extreme caution, especially during the first few hours after a blast exposure. A fluid overload could overstrain a damaged heart and aggravate pulmonary hemorrhage and edema. Therefore blood replacement agents, such as blood, plasma and dextran, used for the treatment of hypovolemic "shock" in space should be administered only if absolutely essential for the treatment of evident hemorrhagic "shock" from blast injuries.

The routine use of a vasopressor, even without evidence of non-hemorrhagic "shock", just to stop pulmonary bleeding is questioned. It is known that the bleeding following blast occurs from both the thin-walled subendothelial bronchial and pulmonary veins and capillaries [54]. Arteriolar and venous constriction should reduce this bleeding. Since vasopressors, such as levarterenol and metaraminol, constrict bronchial vessels to the same degree as peripheral vessels, and pulmonary vessels to a lesser degree, such a drug might be useful for decreasing

bronchial and possibly pulmonary hemorrhage [2, 3]. However this indication for a vasopressor might be outweighed by the possibility that the damaged pulmonary vessels, which have been constricted by hypoxia and by local reflex action, might be opened by the increased pulmonary blood pressure and flow caused by the drug [2, 54]. The beneficial effects of the vasopressor drugs primarily for the treatment of pulmonary complications of blast remain to be determined.

An analgesic, sedative or narcotic drug must be given with caution in the immediate post-blast period in order to prevent the possible masking of signs and symptoms which indicate serious injury. An antitussive, such as dehydrocodeinone bitartrate, might be indicated during the first few days after exposure to prevent hemorrhage due to excess coughing. A suitable broad-spectrum antibiotic will be required for peritonitis or pneumonia. It has been suggested that a suitable antibiotic-cortisone combination be administered as a prophylaxis for infection and to minimize pulmonary fibrous tissue formation for at least two weeks if severe lung damage has occurred [9].

Therapeutic pneumothorax as a "last-ditch" measure to control pulmonary hemorrhage, thoracentesis to remove air, fluid and blood from the pleural space, pericardiocentesis to remove pericardial fluid and blood, nasogastric intubation or gastrointestinal decompression for perforated viscus or temporary ileus, and laparotomy for perforated viscus are a few of the highly specialized procedures which may be necessary following a severe blast exposure [25, 31, 58, 60]. These procedures would definitely require a specially trained astronaut and in many cases, a physician-astronaut. In the instance of a perforated viscus, facilities for performing abdominal surgery would also be necessary. Until such sophisticated treatment is possible in space, only more conservative supportive therapy will be available and a high morbidity and mortality from serious blast injuries in space must be accepted.

Penetrating Injuries

Molten and fragmented meteoroid and wall materials ejected into the spacecraft cabin at high velocities could inflict a variety of single or

multiple penetrating injuries. Some associated burning of tissues is also likely. The majority of these missiles will probably be stopped by superficial tissues, with only small superficial lacerations being incurred. However, laceration of major vessels, nerves and other vital deep structures should still be considered possible. The treatment of penetrating injuries in space is discussed in Chapter 14.

One must also keep in mind the potentially great hazard of particles impinging on the eyes at the time of penetration and if these particles remain suspended in the weightless environment, for some time thereafter. Eye problems due to such foreign bodies are discussed in Chapter 8.

Meteoroid Penetration of the Space Suit

Little is known about the injuries which might result from meteoroid penetration of the space suit [4]. Fortunately it appears that through the use of a meteoroid bumper and absorbing materials in the outer layers of the suit, an astronaut will be reasonably well protected from such an event during extravehicular operations in space [6, 38]. What is presently thought to be an extremely low probability of penetration is apparent. If an approximate suit area of 25 square feet (2.5 square meters) and approximate thickness equivalent of 0.040 inch (0.12 cm) aluminum are applied to the "time to meteoroid perforation" data of Whipple (Figure 12.1), it is apparent that there is an extremely low probability of penetration, even though there is a level of uncertainty of about one order of magnitude surrounding this estimate [4]. It is noted that the degree of protection offered by the "shatterproof" space suit helmet and visor materials is many times greater than that offered by other parts of the suit [4].

Molten and vaporized meteoroid and suit materials, and fragmented meteoroid might come into close contact with or impinge on the skin of an astronaut if a space suit is penetrated. An "explosive oxidation" of vaporized materials will probably also occur at the site of penetration. Experiments which are attempting to simulate meteoroid penetration of the space suit in space are demonstrating that the suit materials do not

tear from or spall at the site of penetration and that these materials are not ignited by the penetration even at maximum suit pressurization with 100 percent oxygen [6, 38]. From these studies, it appears that the most likely injury an astronaut will sustain during a penetration of the space suit by a minimum penetrating meteoroid will be a circular-shaped burn at the site of penetration. This burn area might vary from several millimeters to a few centimeters in diameter, depending on the magnitude of the penetration. Undoubtedly the degree of tissue damage by burn will be greatest in the central part of this area. The management of burns in space is discussed in Chapter 13.

Single or multiple meteoroid fragments of larger penetrations might also penetrate the skin and subcutaneous tissues to a variable depth. Due to their high temperatures, these fragments might also produce some thermal damage of the tissues. The majority of these missiles will probably be stopped by superficial tissues with only small, superficial lacerations resulting. However, laceration of major vessels, nerves and other vital deep structures should still be considered possible. The management of such penetrating injuries is discussed in Chapter 14. Finally, if the size of the hole produced by a penetration is so large that the pressure in the space suit cannot be maintained long enough for an astronaut to carry out emergency measures such as returning to the spacecraft, he might be subjected to the decompression effects (Chapters 1, 2, and 3).

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CHAPTER 13

BURNS

In this chapter, the characteristics and principles of management in space of thermal, electrical, and chemical burns will be briefly discussed. It must be kept in mind that a relatively minor burn of a functionally important body area such as a finger or eye could seriously reduce the operational capabilities of an astronaut for a prolonged period of time. Moreover, intensive supportive therapy and definitive surgery normally employed in the treatment of major burns will probably not be possible in space in the foreseeable future. The importance of ensuring maximum burn protection as well as the best therapy possible for burns during space operations cannot be overemphasized.

It should also be mentioned that if programmed decompression-recompression of a spacecraft cabin is ever to be employed for extinguishing a fire or removal of toxic products of combustion or chemical contaminants from a cabin atmosphere, a protected astronaut could suffer acute hypoxia (Chapter 1), and possibly ebullism (Chapter 2) for a short period of time. Although a much less effective immediate emergency measure, purging of the atmosphere with an inert gas such as nitrogen could also lead to acute hypoxia.

Thermal Burns

Design of spacecraft cabins to provide optimum fireproofing and protection from meteoroid penetration, and the installation of adequate fire detection and extinguishing systems should reduce the risk of thermal burns in space to a minimum. The influence of various possible space atmospheres on this risk has been intensively assessed by Roth [45, 46].

It is readily apparent that the type and degree of a thermal burn suffered by an astronaut in space will be determined by one or more of many factors. The relation of time and intensity of applied thermal

energy to burn severity has been discussed in detail by many authors (6, 14, 19, 34, 36, 40, 41) and will, therefore, not be considered here.

From a causative standpoint, if a meteoroid should penetrate a spacecraft cabin wall (Chapter 12), a localized, possibly deep burn anywhere on the body could be produced by contact with the flash or by being struck with molten and hot fragments of both meteoroid and wall. Such a burn could also follow meteoroid penetration of a space suit. A serious linear thermal burn, most likely of the hands, could result from contact with a wire rendered hot by short circuit. A fire in a spacecraft cabin could burn large areas of an astronaut's body. Assuming that fire resistant clothing is worn, only exposed skin areas, such as his face, neck, arms and hands might be burned. Although space suit materials will have a high melting point, heat transfer from fire in contact with the suit might be sufficient to cause extensive thermal skin injury. Respiratory tract damage from the inhalation of hot air and gases of combustion is considered a major potential consequence of unprotected exposure to a fire in a "closed" space environment.

Characteristics

Burns of the skin can be divided into three categories - first, second, and third degree burns. First and second degree burns are referred to collectively as partial-thickness burns. Third degree burns are also termed full-thickness burns.

A first degree burn is similar to the familiar "sunburn". It involves only the superficial or outer layer of the epidermis. Erythema, pain, and occasionally slight edema of the involved skin subside within one or two days. During this time, the area is dry, warm, and tender. Destruction of the superficial epidermis to a sufficient depth can lead to scaling, often with an associated severe itching sensation, during the healing phase. Infection of this damaged skin does not characteristically occur.

A second degree burn is slightly deeper than a first degree burn. It involves all the layers of the epidermis. Small islands of germinal cells which remain in the deeper corium eventually reform an intact

epidermis. Blisters, severe pain, and marked subcutaneous edema are characteristic. Removal of blistered tissue leaves a pink, moist, extremely tender surface. Healing occurs uneventfully in 14 to 21 days unless infection supervenes [4]. A deeper second degree burn can be readily converted into a full thickness burn by infection.

In a third degree burn, the entire dermis and corium down to the subcutaneous fat are destroyed. Deeper tissues such as large blood vessels, nerves, muscle and tendon may also be involved. The destroyed skin is insensitive, white or charred in appearance, and dry. Subcutaneous edema is usually more marked than in the second degree burn. In the neck, this edema can produce airway obstruction. In an extremity, it can seriously jeapordize blood flow to the whole or distal part of the extremity. The dead tissue, or eschar usually begins to separate from the living tissue within two to three weeks, and eventually leaves an open wound. If this wound is too large to be covered by normal skin and if it is not covered with a skin graft, a thick layer of granulation tissue can form. Over a period of many months, this tissue becomes a scar and can produce severe contractures of the burn area. It should be remembered that burned dead tissue provides an excellent medium for bacterial growth, so that third degree burns tend to become infected.

Inhalation of hot air, and irritant smoke and gases produced by a fire can lead to some degree of injury to respiratory tract tissues [15, 20, 31, 42, 43, 44, 47]. Even though involvement below the larynx is usually prevented by reflex glottic closure, lower respiratory tract damage is considered to be more likely to occur in a spacecraft because of the rapid spread, high concentrations, and persistence of the products of combustion in a confined environment [31]. An edema of rapid onset in the larynx and epiglottis might seriously obstruct air flow [4]. Involvement of the lungs is usually extensive and bilateral [42]. With extensive alveolar damage, survival is highly improbable [39]. The damaged tracheobronchial mucosa and alveoli can immediately become edematous and can produce sufficient transudate to cause death from suffocation [31]. A severe irritative bronchospasm may also

be a factor in producing asphyxia [15]. Infection of a damaged respiratory tract usually becomes evident within three days [42]. An exudative tracheobronchiolitis usually leads to massive or patchy atelectasis, bronchopneumonia, and all too frequently, death [42, 47]. Respiratory tract damage should be suspected if there is a facial burn, singeing of the nasal hair, redness of the pharyngeal mucosa, hoarseness or rales in the chest [39, 43]. Serious damage is indicated by severe substernal pain, coughing of abundant fluid, and cyanosis.

Burn deaths result from a number of causes other than respiratory tract damage. In extensive second and third degree burns, a rapid excessive loss of protein-rich intravascular fluid into the involved skin to form blisters and into the involved subcutaneous tissues to form edema, may result in profound "shock" unless this fluid is adequately replaced. As discussed in Chapter 10, it is not known to what degree the tendency for "shock" to occur from loss of fluid from the circulation will be aggravated by the decrease of fluid volume which appears to result from exposure to weightlessness. For an unknown reason, paralytic ileus often occurs after an extensive burn and can, especially if oral fluids are administered, lead to vomiting [4]. Fatal aspiration, a not uncommon event in a moribund burn patient, would be even more likely to occur in the weightless space environment (Chapter 8). The vomitus could also be a serious particle and droplet hazard to other astronauts. Beyond the first few days after a burn, death can occur from an overwhelming septicemia, in most cases by hemolytic Staphylococcus aureus or Pseudomonas aeruginosa [2]. These highly pathogenic organisms usually enter the blood stream from a suppurating burn wound. Also during this period, death occasionally occurs due to hemorrhage from an acute upper gastrointestinal, or so-called Curling's ulcer [4].

Weight loss, accompanied by marked negative nitrogen balance, characteristically occurs in an extensively burned individual. It is proportional to the extent of the burn, and is restored at a rate which depends upon the adequacy of the nutritional regimen, the time of removal of necrotic tissue and closure of the burn wound, and whether

or not wound infection is present. It has recently been noted that a significant amount of this weight loss can be attributed to an excessive metabolic demand placed on the body secondary to heat loss from fluid vaporization from the burn surface [38]. Various measures to restore the water barrier to burn areas have apparently successfully reduced this strain on the body [28, 37, 38].

Mild to severe anemia can appear in the period immediately following, or several days after a burn. Deep burns can immediately destroy or increase the fragility of exposed red blood cells. Anemias which appear later are attributed mainly to a combination of suppressed red cell production due to inadequate nutrition, toxins from necrotic tissue and bacteria, and red cell loss from the wound site.

Diagnosis

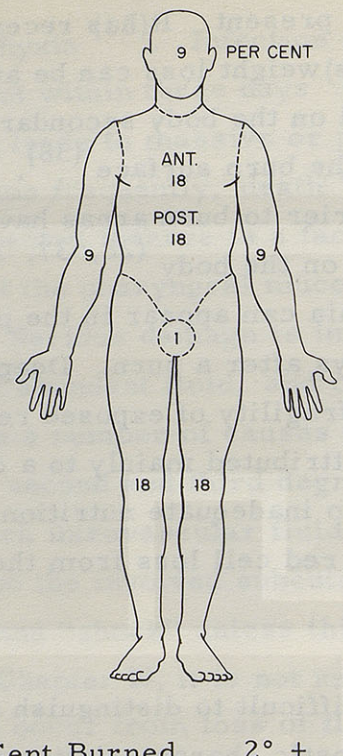
Initially, it can be difficult to distinguish a second from a third degree burn. The diagnostic characteristics of the three degrees of burns have been pointed out above.

A reasonably accurate estimate of the total burned surface area is important, especially to determine whether fluid therapy is needed, and if so, the amount of fluids which should be administered. A rapid popular method is the "Rule of Nines" shown diagramatically in Figure 13.1 [4, 5]. It is not as accurate, however, as the Lund and Browder chart, pictured in Figure 13.2 [4]. Best use can be made of such a method after the burn wound has been cleansed and all loose, devitalized tissue removed. For estimating fluid requirements, the total burn area estimated by either of these methods is the sum of the second and third degree areas only.

If ever possible in space, various laboratory procedures can be of benefit in the assessment and therapy of a seriously burned astronaut. These procedures and the rationale of their use are discussed below.

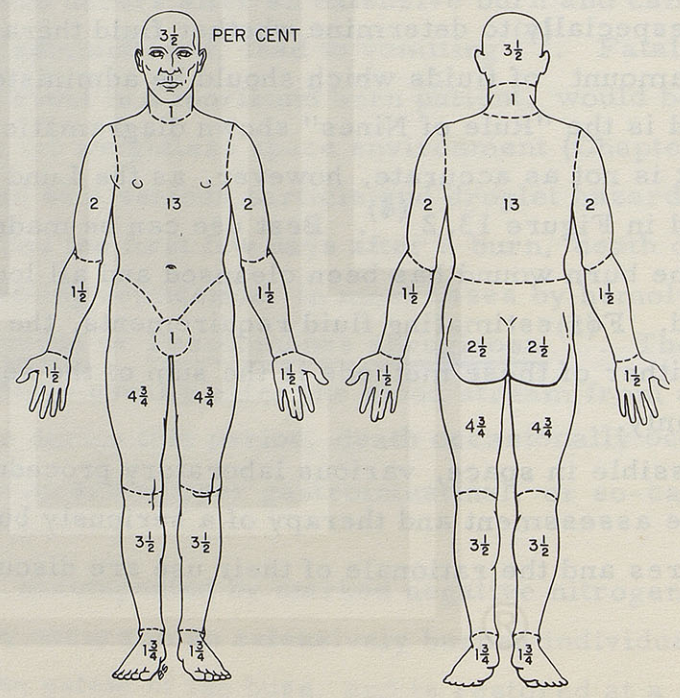
Treatment

Minor burns - Partial-thickness burns covering less than 10 percent of the body surface area, or full-thickness burns of less than



Total Per Cent Burned ___ 2° + ___ 3° = ___

Figure 13.1 The "Rule of Nines" (Adult)



Total Per Cent Burned ___ 2° + ___ 3° = ___

Figure 13.2 Lund and Browder Chart (Adult) (After American College of Surgeons [3]).

2 percent are considered minor burns [4]. Milder first degree burns may involve a much larger area. Fluid replacement for minor burns is usually not required.

First degree burns require no special care. The surface should be protected from irritating garments. A topical anaesthetic, such as 1 percent cinchocaine ointment, may provide some relief of pain and tenderness.

It is recommended that all second and third degree burns, except possibly of certain areas of the face, be treated in space by the so-called "closed" method. This measure will provide less risk of wound infection and further trauma of burned tissue, provide greater comfort, and prevent the release of infected wound material into the spacecraft cabin environment. First, any loose dead tissue, except blisters on the palm of a hand or sole of a foot, should be debrided and if required, the involved surface gently washed with isotonic saline solution. Then the burned surface should be covered with an evenly placed layer of either fine-mesh gauze, petrolatum gauze, carbowax gauze, or a commercial nylon preparation. Finally, a layered, occlusive burn dressing should be applied.

Although locally applied anti-bacterial agents have in general not been successful in preventing and controlling burn sepsis in the past, success has recently been reported with 0.5 percent silver nitrate solution and with 10 percent mafenide acetate cream [5, 24, 28, 33, 37]. For reasons which are readily apparent in the writings cited and because it could present a serious droplet hazard in space, silver nitrate solution does not appear suitable for use in space. On the other hand, if mafenide acetate cream is found suitable for use in space, it might be impregnated in the initial layer of a burn dressing. This water-rich cream has controlled burn wound sepsis, involving even Pseudomonas aeruginosa. It has also minimized weight loss from vaporizational heat loss, from toxic products of bacteria and tissue breakdown, and from inadequate dietary intake associated with repeated general anesthesia for extensive wound debridement. Further clinical use of mafenide

acetate is required before it can be recommended for burn treatment in space.

A burn should be redressed at least every 5 to 7 days. Infected wounds often require more frequent dressing changes. Adherent dressings should be moistened with saline, particularly if over second degree burns, in order to prevent damage to delicate newly formed epithelium. Although some necrotic and liquified burn tissue will be removed along with the dressing, any remaining in the wound should be debrided surgically, especially if the wound becomes infected. Enzymatic debridement of necrotic burn tissue has not proven to be of practical value [4, 8, 26]. It is important to note that extreme care must be taken to prevent the release of infected material into the spacecraft cabin atmosphere. This might be accomplished by means of various suction techniques. A suitable mask should be worn by anyone potentially exposed, including the injured astronaut. An analgesic drug should be administered with care during the immediate post-burn period. It can be used thereafter as required, especially at times of dressing change and surgical debridement.

On the whole, systemic antibiotic therapy has been relatively ineffective in controlling burn wound infections. This generally fails to provide bactericidal levels of drug in infected avascular burn tissue, and promotes the emergence of antibiotic-resistant organisms and sensitizes patients to anti-bacterial drugs [26, 33]. Tetanus antitoxin or toxoid will probably not be required in space if astronauts have been immunized against tetanus prior to space missions.

Major burns - All partial-thickness burns affecting more than 25 percent of the body surface area, or full-thickness burns of more than 15 percent may be considered as major burns [5]. These burns fall into the "potentially lethal" category. They usually require intensive therapy. This therapy centers mainly on providing adequate fluid replacement for the protein-rich fluid lost in burn blisters and edema, minimizing vaporizational fluid losses from the burn surface, protecting the burn from and treating secondary infection, undertaking as much debridement of burn tissue as possible whenever feasible,

closing full-thickness wounds as soon as possible, and maintaining the nutritional status of the patient. It can be expected that if intensive therapy, in particular definitive surgery such as extensive wound debridement and skin grafting cannot be undertaken in space, the usually severe morbidity and high mortality associated with major burns will be markedly increased. This will, of course, be the case for many years to come. Hence a regimen similar to a "shelter plan" may only be possible. A completely austere approach would include medication for pain, proper positioning of affected parts, oral fluids, wound coverage which might be of the simplest sort (e.g. plastic bags for hands), and the use of an oral airway in instances of facial edema and respiratory tract damage [7]. Even if intravenous fluids are carried on board a spacecraft, their use might not be feasible for severe extensive burn lesions.

Burn depth and extent should be assessed as soon as possible according to the criteria discussed above. Second and third degree burn areas are added for determining fluid requirements. Intravenous fluids are usually administered as soon as possible to individuals who have more than 25 percent burns [5]. They may also be given for burns of lesser extent but of greater depth, or for burns with marked edema. As noted in Chapter 10, it is considered possible that due to a decrease of circulating blood volume in the weightless space environment, the tendency for "shock" to occur from intravascular fluid loss in burns might be greater in space than on Earth. Accordingly, appropriate changes might have to be made in any Earth-based formula to be used for estimating fluid replacement for major burns in space.

One of the most popular formulae used to estimate the amount of fluid required by a burned individual is the so-called "Brooke Formula". It states that each of the following fluids should be given intravenously in the first 24 hours after a major burn is sustained [4].

COLLOID (0.5% dextran, plasma or blood)
0.5 ml/Kg/% of body surface burned.
ELECTROLYTE SOLUTION (Ringer's lactate or isotonic saline)
1.5 ml/Kg/% of body surface burned.

WATER REQUIREMENTS (5% glucose in water)
2000 ml.

One-half of the fluids required in the first 24 hours should be administered over the first 8 hours, and one-half over the remaining 16 hours. In the second 24 hours, about one-half of the first 24 hour requirement is recommended. Burns of more than 50 percent of the body surface are calculated as for 50 percent burns, or an excess quantity of fluid can be administered. It is noted that these fluids should be administered with great care if the respiratory tract has been damaged, in order to prevent the initiation or aggravation of pulmonary edema.

Dextran has been considered as effective as plasma in fluid replacement for burns [4, 9, 26]. Opinion to the contrary may have been due to the fact that larger molecular weight dextran preparations used in the past may cause some interference in clotting time and may promote agglutination of red blood cells [26]. The use of blood, even if carried on board spacecraft, in early burn therapy is quite controversial. It is currently thought that blood should be reserved for individuals who have a proven decrease in circulating red cell volume [33]. Ringer's lactate solution is preferable to isotonic saline because a burned individual tends to develop metabolic acidosis in the early post-burn period [4]. In fact, Moyer and coworkers [38] have advocated the use of only Ringer's lactate solution, with its pH adjusted to 8.2, for the treatment of burn "shock". Their reported success with this agent indicated to them that burn "shock" is not primarily oligemic in origin, but is mainly due to an extravascular sodium deficiency resulting from the thermally injured, vascularly isolated tissues taking up large quantities of salt and water. They suggested that Ringer's lactate solution be given fast enough during the 24 to 36 post-burn hours to keep the burned individual safely alive, making urine and remaining oriented - but not so fast or in such quantity to raise central venous pressure significantly and sustainedly above normal. As to whether Ringer's lactate solution will become the sole recommended infusate for burn "shock" will depend upon the results of further clinical use of

this agent.

The great quantities of intravenous fluids are administered to burned individuals through one or more large bore (18 gauge or larger) needles or polyethylene cannulas inserted into a large vein through the skin or by cut-down. At the time of an initial venipuncture, blood can be taken for hematocrit. A narcotic agent, such as morphine, may be administered intravenously, but only if absolutely required for apprehension and pain [4, 5].

The best single indication of the adequacy of fluid therapy is probably an hourly urine output between 20 and 50 ml. This should be determined continuously by means of an indwelling urinary catheter, especially if large areas of the body surface are burned. The catheter can usually be removed within 2 to 3 days. While it is in place and at least for a week after its removal, a suitable urinary antibacterial agent, such as sulfadimethoxine, should be administered.

Venous pressure is an excellent parameter that can be used to avoid over-transfusion. It is generally agreed that to obtain a valid measure of venous pressure, it is necessary to introduce a catheter into the vena cava or right atrium. There appears to be no reason, however, why measurements of venous pressure made in a large peripheral vein should not be valid in the weightless environment.

In a situation where the exact state of total body hydration is in doubt, the serum sodium concentration, if ever possible to determine in space, would be an especially useful guide to fluid therapy. A decrease in serum sodium indicates an excess of body water, and an increase, an excessive water loss. The latter, which is often seen in burn patients who have an enormous insensible water loss, indicates the need for water without salt, rather than more dextran or plasma [11].

The value of the hematocrit in assessing the adequacy of fluid therapy in burns is presently being debated, since the pathophysiology of the changing red cell mass in a burn is complex and hemoconcentration is a result of more than the loss of plasma [11, 38]. Therefore the efficacy of the hematocrit must be thoroughly assessed before recommending its use in space.

Several other points should be kept in mind when determining the adequacy of fluid therapy in burns. A fluid deficiency can be indicated by severe thirst, tachycardia and systemic arterial hypotension. On the other hand, it is important to point out that blood pressure and pulse can be maintained at normal levels at the expense of a drastic vasoconstrictive reduction of the peripheral vascular bed. Interestingly, death from burn "shock" is often slow, whereas death from hemorrhagic "shock" is often sudden [38]. It should be noted that restlessness, irritability, and disorientation are manifestations of a fluid deficit, the treatment of which is fluid, not a narcotic. Abnormal venous distension and the appearance of rales in apparently undamaged lungs point to a fluid excess.

If the urinary output remains low in spite of an apparently adequate restoration of blood volume and tonicity, some degree of renal failure might have resulted from a period of "shock". Further intravenous fluids should then be given with extreme caution, especially if the lower respiratory tract might be damaged. As pointed out in Chapter 14, the usefulness of an intravenously administered osmotic diuretic, such as mannitol, for preventing renal failure from "shock" has been well established. Whether this agent can also be employed to prevent or reduce burn edema should be investigated, for it is considered possible that edema can contribute markedly to the amount of tissue devitalization resulting from a burn [47].

Since a paralytic ileus, often with accompanying vomiting, frequently occurs in severe burn cases, an astronaut suffering from a major burn should not have any oral intake until it is assured that he is passing gas per rectum, his abdomen is not distended, and good bowel sounds are heard on auscultation [25]. Particular care must be taken when oral fluids are eventually administered, since vomiting in the weightless environment will create a serious droplet hazard (Chapter 8). In fact, if there is a possibility of vomiting in the post-burn period, naso-gastric intubation should be performed.

Although a burn patient might be able to tolerate his metabolic food requirement and large amounts of fluid taken orally, large quantities

of intravenous fluid might still be required after 48 hours because of a marked diuresis due to the reabsorption of edema fluid. Since the body tends to retain sodium at this time, the replacement fluid should be 5 percent glucose in water. Serum sodium determinations would be valuable for assessing the state of sodium balance. Potassium (40 to 80 mEq/day) should be given intravenously if this form of therapy is continued beyond 3 days and there has been an inadequate oral food intake.

A tracheostomy might be indicated if serious respiratory tract damage occurs or if there has been a deep burn of the face and neck. The risk of infection spreading from a septic burn wound and involving the tracheostomy and lower respiratory tract should be kept in mind when assessing the need for this measure, however. Although one would prefer to wait as long as possible before performing a tracheostomy in the space situation, it must be remembered that laryngeal edema can occur quite rapidly and this procedure will be difficult after edema forms in a burned neck.

The prophylactic use of antibiotics in the treatment of major burns is presently being debated [47]. Systemic antibiotics of whatever combination are of value only in the first six to seven days, during which they can prevent septicemia [47]. These drugs should be added to the intravenous fluids in order to maintain continuous high levels in blood and tissues, especially during the first two to three days after a burn. Thereafter the intramuscular route of antibiotic administration is considered adequate. Burn wound or respiratory infection attended by a high fever will usually indicate the need for an increase in dosage, the addition of a different antibiotic or a change to an entirely different antibiotic regimen. Antibiotics might be selected on the basis of culture and sensitivity studies of bacteria taken from the wound, if such studies are ever possible in space. Otherwise, a rather "shotgun" therapeutic approach will have to be used. The possible usefulness of locally applied mafenide acetate cream for the prevention and control of burn wound infection in space has been discussed above. The early removal of dead tissue in preventing the ravages of the septic or autolytic phase

of burns cannot be overemphasized.

The local treatment in space of major burns will be the same as that discussed above for minor second and third degree burns. It must be remembered that eventual closure of third degree burns by skin grafting will have to be undertaken after completion of the mission, so that up to this time the treatment of these burns will be aimed at providing the best supportive therapy and wound care possible in space. However, in the very prolonged mission where extensive skin grafting is not possible, strips of skin taken under local anesthesia by means of an air-driven dermatome might be very advantageous [5].

Respiratory tract damage - A definitive treatment regimen for respiratory tract damage by hot air, and smoke and gases of combustion has not been established [7]. It is possible that intensive therapy with steroids, as in the treatment of chemical inflammation of the lower respiratory tract (Chapter 8), might be of considerable value, not only to combat acute inflammatory edema and bronchospasm but also to reduce tissue necrosis which can eventually lead to infection, bronchiectasis and bronchial stenosis [15]. It is suggested that a suitable steroid, such as dexamethazone, be inhaled in its nebulized form and be administered systemically. Bronchospasm might also be combatted with nebulized isoproterenol or intravenous aminophylline [15].

Otherwise, the treatment of this type of respiratory tract damage is supportive. A tracheostomy appears to be of little value except to relieve early acute localizing obstruction [31]. It might also be required for suction of copious quantities of exudate from a seriously involved lower respiratory tract. Oxygen administered under positive pressure might successfully combat pulmonary edema and hypoxia [7]. Intensive broad spectrum antibiotic therapy is indicated in all cases with respiratory tract damage.

Burns of the hand - The astronaut's hands are thought to be one of the most likely parts of his body to suffer thermal injury in space.

For this reason, and since serious functional impairment can follow even a relatively minor burn of a hand, the treatment of these burns deserves special mention here.

Burn blisters on the palm of the hand have a thick covering which should not be broken or removed at the time of their initial dressing. The occlusive dressing should be applied lightly in such a way as to immobilize the hand and fingers in a position of function. Care must be taken to place the dressing between the fingers. A splint may be or may not be used. Again, debridement should be as thorough as possible at the time of each dressing change. The requirement for early vigorous physiotherapy of a burned hand cannot be overemphasized [32].

Recently, a fabricated mitten type of dressing, saturated with silicone fluid and covered with a plastic bag, has been utilized in the treatment of burned hands [29]. This dressing is changed daily. Finger motion is maintained and removal of eschar and exudate is apparently considerably enhanced. This technique has also reduced the need for skin grafting and has decreased both the extensive care usually required and the morbidity associated with this injury. It is thought that this approach to the treatment of hand burns and possibly cold injuries (Chapter 7) might be especially suited to the space situation, particularly for the reason that some degree of hand function can be maintained.

Electrical Burns

Even though spacecraft electrical systems should be adequately shielded, astronauts might be exposed to an electrical burn hazard if a system is disrupted by a docking or landing accident, or by meteoroid penetration, or if they must make repairs on a system in space. Electrical burns characteristically penetrate deeply into tissues, so that even a small electrical burn of the hand, which is considered by far to be the most likely site of this injury, could produce

marked functional impairment of an astronaut.

Characteristics

For an electrical injury to occur, some part of the body must be interposed between two conductors having different electrical potentials. The current which flows between the conductors tends to follow the most direct path possible. Its type (alternating or direct), intensity, path and duration of flow are the major determinants of the pathophysiologic disturbances it produces. These determinants have been discussed extensively in the literature [12, 13, 17, 21, 22, 23, 35]. Most important to note is the fact that an alternating current is more harmful than a direct current, especially in the 60 cycle range which is particularly disturbing to cardiac and respiratory function [35]. The most frequently observed pathophysiologic disturbances from current flow through the body have been cutaneous burning, deep burning with progressive necrosis, fracture of bones or dislocation of joints by violent uncoordinated muscular contractions, and immediate death from circulatory or respiratory arrest [10].

The degree and site of an electrical burn is determined by the resistance which a tissue offers to current flow [35]. Because dry skin has a high electrical resistance, electrothermal injuries are usually limited to it and immediately subjacent tissues. On the other hand, moist skin has a much lower resistance than dry skin, so that deep burning, especially of muscle, can result from electrical contact.

If the contact between the skin and an external conductor is large, the generation of heat per unit area of surface may be inadequate to produce a burn, yet the current flow may be more than enough to paralyze respiration or produce ventricular fibrillation [35]. Conversely, the heat generated at the site of a small contact, may be sufficient to produce a severe burn, even though the current flow through the body is inadequate to cause a significant degree of physiologic disturbance.

Electrical burns are in most cases deeper than ordinary flame burns [4]. Some degree of coagulation necrosis occurs in the skin and

deeper tissues. A major feature is the extensive vascular thrombosis which usually occurs in surrounding tissues over a period of hours to several days after an electrical burn is inflicted. Consequently, the extent of injury usually does not become apparent for up to several days, and is always much more severe than originally anticipated [4, 16]. Deep structures which are often involved include muscle, blood vessels, nerves, tendons, and even bone. In the hand, all of these structures are in close proximity to one another and to the surface, so that electrical burns of the hand tend to be extremely serious. Because of both the tendency to grasp a wire and the spastic grip produced by electric current flow up the arm, burns on the palm of the hand occur much more frequently than on its dorsum.

Extensive muscular damage can lead to a variety of serious secondary consequences. Loss of potassium from damaged muscle can produce a hyperpotassemia sufficient to give rise to severe failure of cardiac function [17]. Hyponatremia can result from a large scale afflux of sodium ions into severely affected muscle. Release of myoglobin and hemoglobin from hemolysed red cells can lead to anuria and fatal uremia. Damaged muscle is particularly prone to become infected [4].

An electrical burn is usually white or charred, and insensitive. Local edema can be quite marked and, when combined with the tissue necrosis, can give the wound the appearance typical of moist gangrene [4].

One of the most serious complications of electrical burns is hemorrhage, resulting from necrosis often of the walls of major vessels [4, 16]. Profuse arterial or venous bleeding might occur.

Treatment

In general, an electrical burn is managed in a similar fashion to a thermal burn. More intensive fluid therapy is usually required in electrical injuries than in thermal burns [5]. The administration of potassium should, of course, be restricted.

One must be sure that there is no muscle damage beneath a full-thickness burn eschar [5]. When dead muscle is present, it must be

excised. However, an extensive wound debridement may be difficult if not impossible to undertake in space in the foreseeable future.

Since acute renal failure occurs not uncommonly following large electrical burns, an osmotic diuretic, such as mannitol, might be given if the urinary output is low, in spite of adequate fluid administration [4]. Alkalinization of the urine with intravenous and oral bicarbonate might reduce the damaging effects of myoglobin and hemoglobin on the kidneys [17].

If there is no dead muscle, it is recommended that an electrical burn not be debrided until ten to fourteen days, when the living and dead tissues are well demarcated [5]. Since hemorrhage is a common complication of electrical burns, great care must be taken with debridement close to major blood vessels. It is difficult to stop bleeding by local pressure or even hemostatic agents, such as gelfoam or topical thrombin. Arterial bleeding might have to be arrested by ligature.

Finally, if circulation or respiration are arrested by an electrical shock, resuscitative measures discussed in Chapter 1 may be undertaken. It is thought that permanent respiratory arrest is unlikely unless an electrical current is sufficiently great to cause gross burning [23]. Hence artificial respiration might have to be carried out for a prolonged period of time before adequate breathing returns.

Chemical Burns

The burn hazard presented by a corrosive chemical spilled by accident into the spacecraft cabin atmosphere will be greatly magnified in space, for not only will droplets and particles tend to remain suspended in the weightless environment, but also their atmospheric dilution will be limited by the confined environment of the spacecraft cabin. Such a hazard must be seriously considered when analytical systems requiring replacement chemicals are carried on board spacecraft. Notably, a caustic burn hazard will exist while servicing life support systems which contain lithium hydroxide or a superoxide as the

carbon dioxide absorbent.

Characteristics

Corrosive chemicals include mineral acids and alkalis, strong organic acids and alkalis, and inorganic oxidizing agents. Many also possess systemic toxicity, even if absorbed only through the skin. All these agents are protoplasmic poisons through their ability to produce protein hydrolysis, either by a hydrogen or hydroxyl ion effect.

Acute chemical injury of the skin is in many ways similar to that produced by heat [4]. In fact, injurious effects of chemicals are sometimes due in part to the development of heat. A highly variable picture of injury may be present. Of interest is the fact that alkalis tend to penetrate and so burn deeply into tissues, whereas acids burn more superficially. Severe burns are characterized by a central zone of necrosis, surrounded by less damaged, more hyperemic, partial-thickness burn areas. Chemical injuries of the eyes and respiratory tract are discussed in Chapter 8.

Treatment

Immediate irrigation of the involved area of the body with copious amounts of water still remains the best emergency measure for chemical skin burns. Not only does water carry away the chemical, but also the heat of dissolution. This might be accomplished in space with a special body cleansing apparatus. Another suitable measure might be the immediate application of materials soaked with water or a suitable neutralizing or buffering agent to the area of contact. Any irrigation procedure should be carried out for at least 10 minutes or even longer, especially for alkali burns. Other than such an emergency procedure, the treatment of chemical burns will be similar to that described previously for thermal burns.

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CHAPTER 14

INJURIES FROM MECHANICAL FORCES

Astronauts will face a potential risk of injuries from mechanical forces during operations in space. Many causes of this form of trauma can be envisaged. While some injuries may prove to be peculiar to the space environment itself, any type and severity of mechanical injury is possible.

This chapter briefly discusses possible causes, prevention, and principles of diagnosis and treatment of mechanical injuries during missions in space. Notably, it above all others raises the question as to what level of medical care might be given in space. It would seem reasonable that one or more astronauts should be trained in the essential requirements of handling medical problems which might occur in space. However, as is apparent throughout this report, the clinical judgment and skills of a physician-astronaut would be highly desirable for the optimum handling of such problems. The topic will be discussed in greater depth in Chapter 16.

The possibility that the healing of various wounds might be altered to some degree by the weightless environment or by spacecraft atmospheres should be kept in mind. This is an area to which no significant research contributions appear to have been made.

Causes

While moving freely about in the weightless environment of his spacecraft cabin, an astronaut might misjudge the velocity and direction of motion which he imparts to his body [7, 10]. As a result, he might sustain an injury by striking an immovable structure, especially a protruding sharp edge or corner. It is also possible that if unrestrained, he might be injured by being thrown about in the cabin or by being struck by displaced objects during maneuvering, docking, and landing operations. There might be some risk of mechanical injury associated with servicing and repair procedures within the spacecraft. Most injuries in the above situations will probably be of a minor nature, such as abrasions, lacerations, and

contusions. On the other hand, high momentum accidents might lead to more serious consequences, such as concussion and fractures.

As was pointed out in Chapter 12, a great variety of minor and major mechanical injuries could result if a meteoroid should penetrate the wall of the spacecraft cabin. Molten and fragmented meteoroid and wall materials ejected into the cabin at high velocities could inflict single or multiple penetrating injuries and lacerations, probably with some associated burning of tissues. Particles could enter the eyes and produce a variety of problems discussed in Chapter 8. Such injuries might also be produced in partial penetration conditions by metal fragments which spall from the inner surface of the cabin wall. It is conceivable that a translational wind load from a meteoroid blast could be of a sufficient magnitude to create missiles of disrupted cabin structures or force an astronaut against immovable structures in the cabin. If this event should occur, any number of mechanical injuries similar to those discussed below for "explosive" decompression might result.

As discussed in Chapter 3, mechanical injuries might be inflicted externally during an "explosive" decompression of the spacecraft cabin, especially if an astronaut is unrestrained and is either close to the decompression orifice or in a narrow passageway between parts of the cabin. As well, items of equipment or other materials in the cabin might detach or fragment at the moment of decompression and become missiles. This hazard will probably be greatest if the cause of the decompression is a meteoroid penetration. Closed wounds such as contusions and fractures might result if an astronaut is thrown against immovable structures by the blast of escaping air. Violent blows to his body might disrupt hollow viscera and produce contusions or lacerations of solid organs, particularly in the abdominal region. Chest trauma might result in single or multiple rib fractures, hemothorax, pneumothorax, and pulmonary or cardiac contusion. Possible craniocerebral injuries include skull fracture, concussion, cerebral contusion or laceration, subdural or extradural hematomas, chronic subdural hematoma and damage to the labyrinthine system. Open wounds such as abrasions, lacerations and penetrating injuries might result not only if an astronaut is flung against sharp protruding

structures in the spacecraft cabin, but also if he is struck by various missiles. These wounds may involve superficial tissues or vascular, nervous, skeletal or visceral structures, leading to death of an astronaut.

An astronaut might also be injured during an extravehicular operation in space or on a lunar or planetary surface. The velocity and direction of motion which he or his extravehicular maneuvering unit impart to his body might be misjudged, causing him to strike the spacecraft, especially protrusions from its surface, with sufficient velocity to produce injury in spite of the cushioning effect of his inflated space suit. Possible injuries expected in this case are contusions and fractures, especially of the ribs. Closed abdominal and thoracic injuries might also occur. There is also the danger of an astronaut being trapped between docking space vehicles or moving sections of a space station being assembled in space. As a consequence, the involved part of his body might sustain a closed crush injury of any severity, even without suit disruption.

Injury might be sustained during operations on extraterrestrial surfaces. Awkward mobility in the space suit, associated with possible balance and locomotion difficulties while walking in unfamiliar gravity environments and on unfamiliar terrain, will predispose to falls which, despite the cushioning and splinting effects of the space suit, could result in contusions, strains, fractures, and dislocations. For the same reasons, there will also be a risk of penetrating wounds, lacerations and decompression effects (Chapters 1, 2, and 3), especially if sharp or pointed tools are to be used for climbing, digging or chipping, or if an astronaut has to walk or climb over jagged terrain.

Finally, it should be noted that meteoroid penetration of the space suit might occur during extravehicular and extraterrestrial operations. As discussed in Chapter 12, single or multiple meteoroid fragments might penetrate the skin and subcutaneous tissues to any depth, producing mechanical and possible thermal tissue damage. Deep penetration by these missiles might result in serious and potentially fatal organ damage.

Prevention

A number of measures should be taken in order to minimize the risk

of an astronaut being injured by mechanical forces during operations in space and on lunar and planetary surfaces. The need to keep design engineers and astronauts continually aware of this area cannot be overemphasized. It is also important to point out that the astronaut who maintains himself at peak physical condition while in space will keep the risk of certain injuries, such as strains and sprains to a minimum.

Hazardous projections into the spacecraft cabin must be eliminated. Necessary projections must be shielded or padded, or have their edges and corners rounded. All detachable items of equipment and other material must be firmly fixed to or enclosed in fixed structures when not in use. Materials with low mass and fragmentation potential must be used whenever possible. Consideration must be given to providing optimum vehicular protection from meteoroid penetration and personal protection from penetrating fragments in various parts of the spacecraft cabin during high risk phases of a mission. Compartmentalization of the spacecraft cabin could be a measure affording protection from mechanical injuries in instances of meteoroid penetration and "explosive" decompression. All hatches on all spacecraft should be designed and standardized to facilitate rescue of injured crew members. Astronauts must be trained in rescue operations.

An astronaut should limit uncontrolled "free-floating" movements about the spacecraft cabin as much as possible. During such movements, the wearing of comfortable light-weight protective equipment such as a helmet, possibly with an attached faceguard, and a rib protector might be indicated. An astronaut should be restrained in the cabin during all spacecraft maneuvering, docking, and landing operations. When the risk of meteoroid penetration and "explosive decompression" is increased, an astronaut should don a space suit and be restrained. Hazards associated with possible servicing and repair procedures on board the spacecraft should be recognized. Procedures should then be outlined and measures taken to ensure that they will be carried out safely.

Several measures should be taken to minimize the risk of mechanical

injuries during extravehicular operations in space. Astronauts should be provided with an adequate propulsion system and be thoroughly proficient at maneuvering in space before attempting major tasks. Close attention must be given to minimizing hazards presented by projections from the surface of the spacecraft, particularly in the hatch area. An astronaut must exercise extreme caution to avoid getting crushed between docking space vehicles or moving sections of a space station being assembled.

During extravehicular operations on lunar and planetary surfaces, an astronaut must exercise extreme caution in walking and climbing over unfamiliar and rough jagged terrain. He might use a mechanical support to assist his balance and locomotion, hence preventing falls. Anchor or safety ropes might be used in certain circumstances. Extreme care must be taken in handling sharp and pointed tools.

Finally, it is assumed that an astronaut will be provided with as durable a space suit as possible. A suitable external protective garment might be indicated in various situations.

Diagnosis

The diagnosis of a mechanical injury in space will be made primarily by history and physical examination. The history must be centered on eliciting symptomatology in detail and the mechanism of injury. The physical examination must be thorough, determining both the extent and severity of tissue damage and assessing the astronaut's total response to the injury. Periodic monitoring of his vital signs is indicated if serious bleeding has occurred or is suspected, or if he is in respiratory distress. If possible on board the spacecraft, basic laboratory and x-ray studies will be useful in confirming and diagnosing injuries.

Treatment

Basic surgical principles will still apply to the treatment of mechanical injury in space. In the foreseeable future, definitive surgical procedures in space will undoubtedly be limited mainly to wound closure, and closed reduction of fractures and dislocations. Thus, in certain cases,

anatomical reconstruction of damaged tissues will have to be done after return to Earth. Due to weight penalties imposed by transporting stored, fresh whole blood into space, only reconstituted plasma or a suitable plasma-expanding agent such as dextran might be available for replacement of blood loss. It is apparent, therefore, that all minor mechanical injuries should be adequately treated in space. On the other hand, limitations placed on the surgical, and possibly the supportive treatment of major mechanical injuries will be such that the duration and degree of functional impairment and the mortality from these injuries will on the whole be much greater in space than on Earth.

Although the processes of repair are fundamentally the same in all body tissues, tissue differences in ability to survive and regain function following various types of damage will make each wound an individual problem in treatment. It is of utmost importance, therefore, to have a sound knowledge of the healing potential of various types of wounds and of the factors that enhance or impede healing. Above all, one must be cognizant of the serious consequences which can result if a wound should become secondarily infected.

Local and regional anesthesia for the repair of wounds in space is considered ideal in the light of the greater weight penalties and problems with atmospheric contamination which would be associated with the administration of a general anesthetic in space. Such might also be used instead of analgesic and sedative drugs if the astronaut must be maintained at an optimum functional capacity.

The immediate care of an injured astronaut must be directed at controlling bleeding, ensuring an adequate airway, and preventing "shock", further tissue damage, and further contamination of an open wound. External bleeding can, in most cases, be arrested by a sterile compressive dressing. As a rule, a well-padded tourniquet should be applied only when a major vessel in an extremity is severed, and should not be removed until definitive treatment of the wound has been instituted. Adequate ventilation of an unconscious astronaut might be attained by inserting a suitable mouthpiece. The respiratory distress associated with a chest injury

such as single or multiple rib fractures may be aided by stabilizing the involved area of the chest wall with strapping or infiltrating the appropriate intercostal nerves with a local anesthetic agent, such as 2 percent lidocaine. If these measures fail or are likely to fail, tracheostomy might be indicated. Sucking wounds of the chest must be securely closed with an airtight dressing. The atelectatic tendency in a 100 percent oxygen atmosphere makes rapid treatment of a sucking wound mandatory.

The general condition of an injured astronaut should be allowed to stabilize before definitive treatment of his injury is commenced. Blood loss should be replaced with colloid or whole blood, if available. If a large crew is on board a spacecraft, use of donor blood from other crew members whole blood is known to be compatible might be possible.

As pointed out in Chapter 10, cardiovascular adaptations to weightlessness will take place during prolonged space missions. A temporary, and possibly persisting decrease of blood volume will occur. Thus an astronaut who suffers from blood loss in space will in essence not receive the benefit of the "transfusion" of pooled blood if he was rendered recumbent due to blood loss prior to leaving Earth. It is thought that cardiovascular mechanisms which compensate for blood loss should not be altered by weightlessness. Since cardiovascular adaptations to weightlessness reduce orthostatic tolerance, "shock" might result from a relatively minor blood loss if measures (Chapter 10) are not taken to adequately protect an astronaut from orthostatic intolerance on return to a gravity environment.

Closed Wounds

Even though the skin remains intact following non-penetrating trauma, any underlying tissue in the body can be damaged. Closed injuries are characterized by the tearing and crushing of soft tissues, fractures and dislocations. From previous discussion, it appears that the majority of closed injuries in space will be contusions, sprains, strains, fractures, and dislocations, of which most will not be life-threatening. On the other hand, violent non-penetrating blows might produce potentially fatal organ disruption and internal hemorrhage.

Contusions are usually produced by direct blunt force and crushing

trauma. Usually only subcutaneous tissues are injured. Small blood vessels are torn and bleed into interstitial spaces. Exudation of serum into the damaged tissues enhances swelling. When muscle is contused, a large vessel might be torn, resulting in profuse hemorrhage and possibly "shock". Extravasated blood may collect between layers of tissues to form a hematoma. Bleeding may also occur into a joint or tendon sheath to irritate and produce inflammation of these structures. An expanding hematoma may produce enough ischemia of skin and pressure on surrounding tissue to cause necrosis and secondary infection. Minor superficial contusions are swollen, tender and usually produce no loss of function. On the other hand, bleeding into a muscle, joint or tendon sheath can produce a severe restriction of movement and pain. A contused nerve in which the axons are not disrupted (neurapraxia) will be associated with temporary pain, paresthesia and paralysis in the nerve distribution, whereas disruption of axons (axonotmesis), usually occuring in association with a closed fracture, results in complete motor and sensory paralyses which recover completely over a period of weeks to months.

The majority of the contusions discussed above are treated by non-surgical measures which are directed first at controlling bleeding, and later at hastening the resolution of the residual hematoma and edema, and restoring the injured part to optimum function. The immediate application of a suitably padded compression bandage over the site of contusion may have some effect in limiting the initial extravasation. If possible, cold compresses might also be effective, especially in areas which are superficial and not easily bandaged. Activity should be curtailed insofar as is practical until an astronaut is over the period of acute local tenderness.

Rarely is it necessary or advisable to evacuate a large superficial hematoma either to hasten return of function or prevent skin necrosis. Evacuation may be accomplished in the first few hours by needle aspiration, or if this is not possible, owing to clotting, by expressing it through a small incision made under local anesthesia. Following these procedures, a pressure dressing must be applied to prevent recurrence of the hematoma. One might also consider aspirating blood from a joint, especially the knee, for the relief of pain. It is important to point out that a definite

risk of introducing infectious organisms into damaged tissues must be assumed when carrying out the above procedures.

To assist the absorption of hematoma and edema fluid, the affected part should be placed at rest and, if possible in space, heat applied periodically. Graduated activity of the part should be commenced when deemed feasible. Recent interest has been focused on the use of orally administered and locally injected protease (plasminogen)-activating enzymes, such as combined streptokinase-streptodornase, to assist absorption of hematoma and edema fluid [2, 6, 12, 13]. Whether agents presently in use significantly ameliorate symptoms and shorten recovery time remains open to question, however.

Tearing of ligaments is defined here as a sprain, and tearing an avulsion of muscle or tendon as a strain. These injuries are caused by either direct or indirect violence, and vary from incomplete to complete disruption of the continuity of these tissues. The resulting pain, swelling and loss of function are dependent upon degree of tissue damage.

The initial treatment of sprains and strains is similar to that for contusions. An analgesic or sedative might be required for pain. If an astronaut must be kept at an optimum functional level, local or regional anesthesia might be used instead of these drugs. Placing these injuries at rest is essential until healing is well progressed. In most cases, bandaging or strapping should provide adequate support. Splinting, as used for the treatment of fractures, might be required for more serious injuries, especially those which would be treated on Earth by operative repair. Graded activity, with care to avoid placing undue strain on healing tissues, should be commenced as dictated by clinical judgment.

Fractures and dislocations are also caused by direct or indirect force. A fracture can be "closed" or "open", depending on whether or not there is a communication between the site of fracture and the outside air through the skin or mucous membrane. It may be incomplete (e.g., fissure, depressed, puncture, and greenstick fractures) or complete (e.g., simple, comminuted, impacted, compression and avulsion fractures)

Fracture fragments may or may not be displaced. In addition to a fracture lesion or dislocation, there may be associated injuries to contiguous nerves, joints, tendons, and viscera. The possible existence of multiple fractures at different levels in the same bone or in other bones, or a combined fracture-dislocation must always be kept in mind. The degree of incapacitation of an astronaut will depend on the site and nature of the fracture or dislocation and the amount of associated soft tissue damage.

Careful handling of an obvious or suspected fracture or dislocation is mandatory to prevent fracture displacement, further soft tissue injury and the possible creation of an open fracture. For quick efficient temporary immobilization of an extremity fracture, a suitable pneumatic splint might be used [7].

A displaced fracture should be reduced into as best alignment as possible in space by traction and manipulation. Ideally, reduction might be most easily and painlessly accomplished during the period of local numbness and paralysis which usually lasts for many minutes after injury. However it might be delayed if an astronaut requires resuscitation, treatment of more serious injuries or debridement of damaged tissues associated with an open fracture. If analgesia is required, an injection of local anesthetic into the fracture site might control pain until reduction is accomplished. More preferable, however, is the use of regional anesthesia, which provides both analgesia and muscular relaxation and eliminates the risk of introducing infection into the fracture site.

The treatment of open wounds with underlying fractures should be similar to that to be discussed for all open wounds. Because of the serious consequences of infection of a fracture site, a suitable broad spectrum antibiotic should be administered prophylactically for a period of time to all open fracture cases.

Complete immobilization of most fractures is essential for their healing. The type of immobilization employed will depend on the site and nature of the fracture and the availability of materials which can be used for such a purpose in space. If specific lightweight materials for

splinting or encasing fractures are not carried into space, a suitable splint might be fashioned from expendable or repair materials on board the spacecraft. Some fractures can be suitably treated by methods other than splinting or encasement. For example, adhesive strapping can be used to immobilize fractures of the clavicle, upper humerus, mandible or ribs. Other fractures may require no specific treatment except rest and perhaps temporary bandaging to minimize associated bleeding, give protection and relieve pain. It is noted that rib fractures frequently require only the relief of pain by infiltration of the appropriate intercostal nerves with a local anesthetic. Finally, it is pointed out that treatment facilities in space in the foreseeable future will be such that open reduction and internal fixation of fractures will not be possible.

Any dislocation should be reduced as soon as possible in order to minimize injury of contiguous structures, especially nerves and blood vessels. Regional anesthesia might be of great benefit by providing temporary relaxation of muscles as well as analgesia around the dislocation. Complete immobilization of the joint for several weeks might be required to allow healing of torn joint structures. Otherwise, movement might be restricted for a short period of time, followed by a graded activity as indicated.

Violent trauma can produce a great variety of closed injuries of a more serious nature than those discussed above. Of particular concern are the consequences of severe blows and crushing forces, especially in the head and thoracoabdominal regions. These forces can disrupt underlying viscera and supporting structures leading to serious internal bleeding. It is important to point out that many serious closed injuries can occur not only in the absence of early or obvious external signs of violence, but also far removed from the site of external impact.

A blow to the head can produce concussion or unconsciousness due to functional or anatomic derangement of the brain stem. It is noted that concussion, brain contusion or laceration, and skull fracture may occur singly or in any combination [5]. Consciousness may return within

seconds following concussion or may take minutes to days following more serious brain injury. Cases which recover slowly pass through a transitional state of semi-consciousness characterized by disorientation and confusion which are attributed mainly to cerebral edema. Common complaints during this period are severe headache, lightheadedness or vertigo, and nausea and vomiting. Permanent residual psychologic and motor defects may result from anatomic damage to the brain. A delayed deterioration of consciousness or a deepening coma may occur following head trauma, due to an enlarging intracranial mass such as an extradural, subdural, or intracerebral hematoma, to a subdural collection of cerebrospinal fluid, or to cerebral edema. Deterioration due to expansion of a chronic subdural hematoma may follow a lucid interval of several days to a few weeks, even after a seemingly minor blow to the head. Fractures through the temporal region may affect vestibular function possibly resulting in severe vertigo, nausea, and vomiting. Open skull fractures, especially those associated with dural laceration, may discharge cerebrospinal fluid and allow the entry of infectious organisms to produce meningitis, osteomyelitis, cerebritis, or brain abcess.

A severe blow or crushing injury of the chest may produce a hemothorax. A fractured rib can penetrate the lung to produce a pneumothorax with or without associated bleeding. The pneumothorax may be of the "tension" variety in which the passage of air back out of the pleural cavity is blocked by the flap of torn lung pleura which acts as a ball valve, so leading to serious pulmonary and cardiovascular insufficiency. Air escaping from the disrupted lung may dissect along bronchovascular roots of the lung into the mediastinum to produce mediastinal emphysema. It may even track into the neck, face, scalp and thoracic wall to produce subcutaneous emphysema. This condition becomes serious when air within the mediastinum produces an acute restriction of venous return to the heart or when secondary mediastinal infection occurs. Severe blows over the cardiac region of the chest may contuse the heart, resulting in a clinical condition not unlike that of coronary artery occlusion with myocardial infarction.

A blunt force to the abdominal region can contuse or lacerate solid viscera, or rupture hollow viscera. All types of abdominal injury, regardless of the organ injured, may produce two primary effects - "shock" and "peritonitis" - either singly or together. A fixed organ is more likely to be injured than one which is more mobile. Organs frequently injured on Earth, in approximate order of frequency, are liver, spleen, small bowel, large bowel, kidneys, stomach, urinary bladder, and diaphragm (left side) [8]. Certain parts of the small intestine are much more prone to injury, such as the third part of the duodenum, the proximal jejunum and the distal ileum. Delayed intra-abdominal hemorrhage may occur from a damaged spleen, liver duodenum, pancreas, or kidney. It is noted that closed abdominal injuries usually present a particularly difficult problem in early diagnosis, for the initial trauma may be remarkably trivial and a severe blow to the abdomen may be followed by rapid recovery and an interval of several hours, and rarely days without implicating signs and symptoms before the actual injury becomes clinically manifest.

As was previously pointed out, definitive surgical procedures in space in the foreseeable future will be limited mainly to wound closure and closed reduction of fractures. Thus the treatment of serious closed injuries will be supportive. Intravenous fluids and electrolytes to maintain water and electrolyte balance, fluids such as plasma, a plasma expanding agent, or whole blood for blood loss, analgesic drugs for the control of pain, and a suitable broad spectrum antibiotic for the prevention and control of infection are considered the most essential forms of therapy to have available in space. Numerous other supportive measures are conceivable. A cerebral dehydrating agent such as mannitol might be used for relieving post-traumatic cerebral edema or preventing post-traumatic renal tubular necrosis. An ataractic drug, such as sodium phenobarbital, might be administered to control agitation associated with brain injury. A tracheostomy might be performed to improve lung ventilation. The removal of air or blood from the pleural cavity might be accomplished by periodic needle aspiration or, in a case with severe bleeding or "tension" pneumothorax, by continuous

suction. Acute restriction of venous return to the heart due to mediastinal emphysema might be relieved by an incision made under local anesthesia at the base of the neck. Cardiovascular drugs such as a rapid-acting cardiac glycoside (e.g., digoxin) or a vasopressor agent (e.g., metaraminol) might be administered to cases which develop myocardial insufficiency due to cardiac contusion. Nasogastric intubation might be necessary to relieve upper gastrointestinal distension or vomiting, decompression of the upper gastrointestinal system, or for feeding. An indwelling urinary bladder catheter might be required for bladder drainage in an unconscious astronaut or to decompress a damaged urinary tract. Finally, even though the view is held that definitive surgery will not be performed in space in the foreseeable future, it is considered possible that physician-astronaut might, under certain circumstances, make a heroic operative attempt to save a fellow astronaut's life using the limited instruments available. Some operations which come to mind are trephining, spenectomy, amputation, and repair of a disrupted hollow or solid abdominal viscus.

Open Wounds

Open wounds denote a break into or an actual loss of the protective skin barrier, with the underlying tissues being damaged to varying degrees and extent. As compared to closed injuries, open wounds are subject to contamination by bacteria introduced into the wound by the wounding agent or by foreign material.

The various types of open wounds include abrasions, lacerations, penetrating injuries, avulsions and crushing injuries. The degree of damage to deeper tissues is often suspected from the nature of the trauma and the type of wound produced. Injured nerves, muscles and tendons in wounds involving the extremities can usually be diagnosed by testing the function of parts distal to the site of injury. On the other hand, the ultimate extent of injury in penetrating injuries of the neck, thorax, and abdomen may not be obvious. No wound in any location should be probed in an attempt to establish the extent or depth of injury, for this maneuver cannot be expected to yield reliable information and can cause further

harm by accentuating hemorrhage and introducing further contamination.

An abrasion is the most superficial type of open wound, with only the skin being destroyed to a variable depth. Slight bleeding and serum exudation occur from the injured surface and form a thin eschar under which the denuded epithelium regenerates. If infection supervenes, healing is delayed.

A laceration is a linear wound in which the skin and underlying tissue damage are localized to the path of the wounding agent, which can be either a sharp or blunt object. It may vary from a neatly incised defect to one with irregular torn and contused edges with much associated tissue loss.

A penetrating wound is created by a missile or sharp object which might pass to any depth into or right through tissues. The greater the velocity of the penetrating agent,the less the likelihood of skin or clothing being carried into the wound by the agent. Due to its mass and velocity, a penetrating agent may dissipate enough kinetic energy in its passage to produce extensive tissue damage around the wound tract. Of note is the fact that in perforating injuries from high velocity missiles, wounds of exit are usually larger than those of entrance. Thus it is conceivable that in the space situation, a penetrating, high velocity, dense meteoroid or spacecraft wall fragment might conceivably damage blood vessels, nerves and other tissues at varying distances from the course of the "missile", with the external appearance of this serious wound being quite misleading. That even more widespread tissue damage might be caused by bone and "missile" fragments is also possible. Reference is made to further discussion of injuries due to meteoroid penetration in Chapter 12.

Avulsions are characterized by the tearing of tissues from their attachments. Skin and subcutaneous tissues can be either partially or completely avulsed from underlying tissues. Any degree of extent of damage to deeper structures can occur. A torn flap may or may not remain viable, depending on the adequacy of its blood supply.

Open crushing wounds can be present with any combination of the various types of open wounds discussed above. Open or closed damage to deeper

tissues, including bones and viscera, may be extensive.

The treatment of all open wounds in space will be governed by the same surgical principles as on Earth. The primary objective will be to convert, as soon as possible, an open contaminated wound into a surgically clean, closed wound. There is a generally accepted optimal time or so-called "golden period" of eight hours during which, from the standpoint of minimizing bacterial invasion of tissues, wound care should be undertaken. However there may be many other factors, such as wound blood supply, and devitalization and contamination of tissues which have to be taken into consideration other than such an arbitrary time limit. If wound care must be delayed until an astronaut's general condition is stabilized or a more serious injury is treated, the wound should be covered with a sterile compressive dressing to minimize further contamination and bleeding. It is noted again that as a rule, a well-padded tourniquet should be applied only when a major vessel in an extremity is severed, and should not be removed until definitive treatment of the wound has been instituted. Whether local or regional anesthesia will be used during wound repair in space will depend upon the nature of the wound.

Wound cleansing, using aseptic precautions (sterile surgical gloves, surgical mask, etc.,) is the first step in definitive open wound treatment. After carefully cleansing the surrounding skin with an antiseptic agent (e.g., hexachlorophene soap) and possibly shaving off hair, it may be necessary to irrigate the wound with a suitable sterile solution (e.g., isotonic saline) and remove loose foreign material. Droplet contamination of the spacecraft cabin atmosphere might be avoided by keeping a sterile absorptive material and strong suction device in close proximity to the wound, or by completely enclosing the wounded area in a container which might be used for washing purposes in space. Draping the wound from unprepared skin surfaces might be accomplished with a sterile adhesive material, or by the circumferential application or tying down of a sterile nonadhesive material.

The next step in definitive wound treatment in space will be to make the wound as surgically clean as possible. This goal is accomplished by debriding or removing devitalized tissues, foreign substances and tissues

which are hopelessly damaged, so leaving a wound which contains only viable tissues with an adequate blood supply. It may be necessary to enlarge a wound in a suitable line to display the full extent of damage. Each type of tissue encountered must be removed only after a careful intelligent evaluation of its viability, for there must be no needless sacrifice of tissue. Blood clots must be removed and careful hemostasis achieved.

The repair of wounds in space will be limited mainly to wound closure. On the other hand, it is considered possible that an experienced physician-astronaut might undertake in space, when indicated, nerve, blood vessel, and tendon repairs with appropriate suture materials.

Every open wound in space should be closed if at all possible, providing that undue tension does not have to be placed on the tissues. Minimal skin tension might be relieved with subcutaneous sutures or by making relaxing incisions. Suturing with a fine, non-reactive material still remains the best all-around technique for skin closure. Experimental and clinical success has recently been reported in closing skin with a sterile surgical adhesive tape [3, 4, 11]. Although such a tape is highly recommended for use in space, it is probable that tape methods of skin closure will never completely replace suture methods, especially in moist, oily or highly mobile surface areas, or for closing jagged or sharply angulated wounds. It appears that where a broad defect exists, taping can oppose skin edges which would otherwise be closed only after undermining of the skin or making relaxing incisions.

After wound closure, a generous dressing must be applied and, if necessary, the part immobilized to promote healing and prevent infection. Broad spectrum antibiotics might be administered prophylactically where there is a high risk of a contaminated wound becoming infected.

Finally it should be mentioned that due to extensive tissue loss, post-traumatic swelling or severe contamination, primary closure of a wound may not either be possible or advisable. The wound must then be packed with fine mesh gauze or other suitable material, a generous dressing applied and the affected part splinted. So-called delayed primary closure of a wound, if clean, should then be attempted in about five

to seven days. If a wound becomes infected or if the defect is too large to close, one will have to allow it to close by granulation, or by so-called "secondary intent". Repeated dressing changes and continued support of the part will be required until healing is complete. Antibiotic therapy should be administered to infected wound cases.

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CHAPTER 15

CARBON DIOXIDE (CO_2) TOXICITY

Astronauts risk exposure to a toxic level of CO_2 during space missions. An accumulation of this waste product of body metabolism could occur in the atmosphere of a spacecraft cabin or space suit due either to a partial to complete failure or a temporary overloading of a CO_2-absorbing system.

Fortunately an astronaut's inspired CO_2 can be prevented from reaching a toxic level in a number of ways. Environmental control systems can be designed to handle all possible peak CO_2 loads. The reliability of these systems can be increased with emergency subsystems which either absorb CO_2 or purge atmospheres of this gas. As well, breathing gas can be supplied directly to an astronaut if he is exposed to a toxic ambient CO_2 level.

Although adequate measures will presumably be taken to prevent the occurrence of CO_2 toxicity in space, it is still considered possible that such measures could become inadequate, especially during prolonged missions and particularly strenuous extravehicular operations. For example, if failure of a CO_2-absorbing system occurs, atmospheric purging or breathing oxygen from an open loop system might have to be limited, and so some elevation of atmospheric CO_2 tolerated, in order to conserve oxygen. Accordingly, it is necessary to consider the medical consequences of such a situation and determine what therapeutic measures might be taken in space to combat CO_2 toxicity and so maintain an astronaut's performance capacity at an optimum level.

This chapter presents various aspects of CO_2 toxicity considered pertinent to the space situation. It will become apparent that differences between the short and long term effects on man, and between the possible circumstances of exposure to CO_2 in space dictate the necessity to discuss this area under the separate headings of "Acute CO_2 Toxicity" and "Chronic CO_2 Toxicity".

Also by way of introduction, it should be pointed out that virtually all

studies in the past have stated inspired CO_2 levels as "percent" rather than as "partial pressure". The partial pressure of CO_2 actually determines pathophysiologic effects of this gas. It also remains constant as the percent composition of this gas changes for different space atmospheres and will probably be sensed by all space atmospheric monitoring systems [35, 134]. Hence the use of this unit is definitely preferable, especially when recommending maximum allowable levels of CO_2 for space atmospheres.

An accurate conversion of percent CO_2 data stated in the literature to partial pressure values will not be attempted in this chapter, for few investigators have made note of ambient atmospheric pressure, temperature, and relative humidity. However, most of the information involving the inhalation of CO_2 has given the inhaled dry air percentages of CO_2. It is thought that atmospheres of past experiments can be reasonably well approximated by assuming a sea level pressure of 14.7 psia (760 mm Hg). Hence a partial pressure of CO_2 of about 7.5 mm Hg would represent one percent CO_2.

A physiological basis of comparison is the partial pressure of tracheal CO_2. This value is stated in terms of body temperature, at ambient atmospheric pressure in air saturated with water vapor (BTPS). The relationship of the partial pressure of tracheal CO_2 and barometric pressure (or altitude) for various sea level equivalent percentages of CO_2 is shown in Figure 15.1.

In this chapter, CO_2 levels will still be stated conventionally as percent, but whenever exposure of an astronaut to CO_2 in space atmospheres comes into consideration, partial pressures of CO_2 equivalent to concentrations in the sea level atmosphere defined above will also be stated.

Acute CO_2 Toxicity

Broadly speaking, acute CO_2 toxicity denotes the effects suffered by an astronaut who is exposed to toxic atmospheric CO_2 levels which are reached within and persist at varying and maintained levels for minutes to as much as 24 hours in duration. An acute toxic condition in this case differs considerably from one resulting from a more prolonged, or chronic

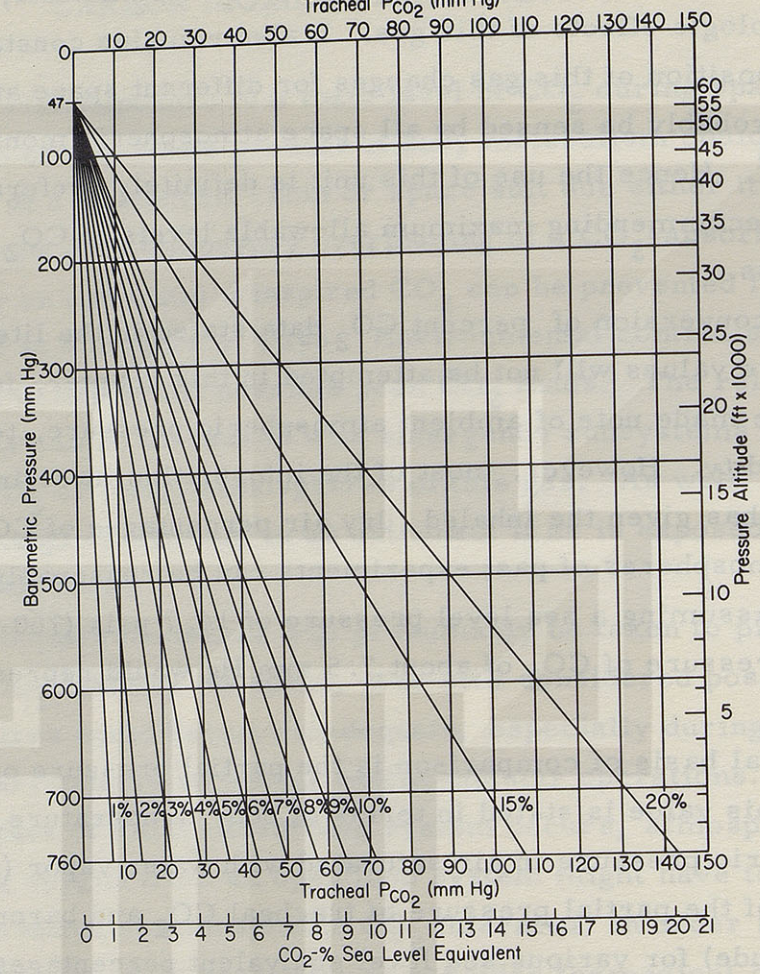

Figure 15.1 Relationship of partial pressure of tracheal CO_2 and barometric pressure (altitude) for various sea level equivalent percentages of CO_2.

(After Luft [124]).

exposure to CO_2. It could arise from a temporary CO_2 build-up in cabin or suit atmospheres due to inadequate functioning of life support systems.

The accumulation of CO_2 will obviously be much faster in a space suit than in a spacecraft cabin atmosphere. Rough calculations based on current suit data indicate that an astronaut who is walking on a lunar or planetary surface can increase his inspired CO_2 to a highly toxic level, as will be defined below, within one to two minutes after a complete cessation of CO_2 absorption by his extravehicular life support system [134, 175].

It is noted, however, that CO_2 storage by the body would have a significant retarding effect on rates of atmospheric CO_2 accumulation only in such a small rebreathing volume as that in a space suit [60, 61, 68, 169]. In fact, recent evidence indicates that the immediate storage of CO_2 involves a body compartment with a volume corresponding to that of the extracellular space [68, 147]. Carbon dioxide storage by the body should therefore be taken into account when attempting to predict such rates accurately. As far as CO_2 accumulation in spacecraft cabins is concerned, it is estimated that three astronauts who are carrying out normal intravehicular operational tasks would not, even in the confined volume of the Apollo Command Module, experience symptoms of CO_2 toxicity until about 6 to 7 hours after CO_2 removal from their atmosphere ceases. From such considerations, then, one can foresee the possibility of toxic levels of CO_2 being reached over a period of minutes in space suit atmospheres and over a period of hours in spacecraft cabin atmospheres. Because of such a marked time difference, the question arises as to whether or not the rate of CO_2 increase can significantly alter an astronaut's response to acute exposures to this gas in space. Fortunately such information can be obtained from reports of past exposures of man to constant and gradually increasing levels of inspired CO_2. For all practical purposes, it can be assumed that exposures to constant levels of CO_2 are equivalent to those that could occur over a brief period of time in a space suit.

Pathophysiology

Since the pathophysiology of man's response to acutely elevated partial pressures of CO_2 in his inspired air is well documented in the literature, it will receive only brief attention here. Particular emphasis will be placed on those aspects which are considered practical from a space operational standpoint and pertinent to ensuing discussions of the clinical manifestations, diagnosis, prevention, and treatment of acute CO_2 toxicity in space. Greater detail in this area is provided by the references to be cited.

Carbon dioxide is a powerful stimulus to breathing. This is an effect

which, within limits, is the major factor protecting man from acutely elevated, otherwise toxic concentrations of this gas in his ambient atmosphere. The increase in pulmonary ventilation produced by CO_2 varies markedly at different times in a normal individual and throughout the normal population [55, 93, 100, 188, 223]. The population response characteristic appears to account to some degree for variations in tolerance to CO_2, for it has been demonstrated that individuals with a relatively large tidal volume and slow respiratory rate show less of a respiratory and sympathetic nervous system response, and less symptoms while breathing low concentrations of CO_2 than individuals with a relatively small tidal volume and fast respiratory rate [178, 188]. Accordingly, knowledge of responses to CO_2 might have some practical value from a monitoring standpoint.

An average effect of various inspired air-CO_2 mixtures upon the steady state alveolar minute ventilation and partial pressure of CO_2 of normal resting man at sea level is shown in Figure 15.2. This response curve, which can be calculated from equations developed by Gray [84, 85], correlates well with other data in the literature [29, 67, 82, 98, 100, 171, 188, 223]. It demonstrates the increasingly inadequate ventilation, notably paralleled by an accelerating rise of alveolar CO_2, as the ambient CO_2 increases. This dulling of man's ventilatory response to progressively increasing levels of CO_2 has been attributed to a combination of the narcotic effect of CO_2 on respiratory center neurons, the stimulation of pressure receptors in the thorax by hyperventilation and the fatiguing of respiratory muscles [41, 55].

Much research has failed to adequately define the mechanisms by which an increase in inspired CO_2 produces an increase in pulmonary ventilation [91, 112, 114]. A few aspects of this area are pertinent to this discussion. For greater detail, the excellent reviews by Hamilton and Brown [91], Heymans and Neil [96], Kellogg [102], Tenney and Lamb [212] and others [40, 51, 52, 85, 88, 99, 113, 120, 155, 211] can be consulted.

It has been observed in humans, and substantiated by animal experimentation, that breathing 100 percent oxygen at pressures near sea level for short periods of time significantly depresses respiratory reactivity to CO_2, presumably through the effects of oxygen on peripheral chemoreceptor sensitivity for CO_2 [19, 91, 113, 114, 121].

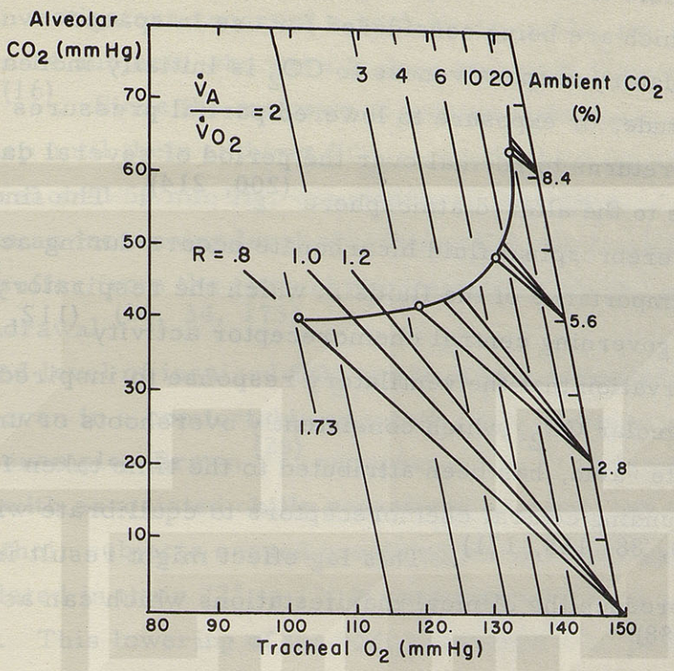

Figure 15.2 Effect of inspiring various CO_2-air mixtures upon the steady state alveolar gas composition of normal man at rest. The ratio $\dot{V}_A/\dot{V}_{O_2}$ represents liters (BTPS) per minute of alveolar ventilation for every 100 ml(STPD) of oxygen consumed per minute. R represents the respiratory exchange ratio (volume of CO_2 output for volume of O_2 intake) and would be equal to the respiratory quotient (RQ) under steady state conditions at sea level.

(After Fenn [64, 65]).

This effect was not found, however, when a few individuals were exposed to 100 percent oxygen at an atmospheric pressure of 200 mm Hg for 4 days, or when sleeping subjects near sea level breathed 40 to 100 percent oxygen for periods of 40 minutes to a few hours in duration [34, 47]. The reason for these divergent results cannot be given. As far as the potential exposure of an astronaut to increased levels of CO_2 is concerned, studies of man's response to CO_2 in various possible space atmospheres which contain oxygen above that breathed on Earth appear indicated. As well, it is

deemed advisable to assess this response in atmospheres containing inert gases which are being considered for use in space.

The ventilatory responsiveness to CO_2 is initially increased on exposure to altitude, or exposure to lowered partial pressures of oxygen [200, 214]. It then returns to normal over the period of several days required to acclimatize to the altered atmosphere [200, 214]. The finding that a reduction of cerebrospinal fluid bicarbonate occurs during acclimatization points to the importance of the fluids in which the respiratory neurons are bathed in governing central chemoreceptor activity [112, 200].

The observation that the ventilatory response to inspired CO_2 lags changes in alveolar CO_2, which consistently overshoots or undershoots its steady state value, has been attributed to the time taken for CO_2 in tissues surrounding central chemoreceptors to equilibrate with altered blood CO_2 [73, 88, 158, 171]. This lag effect might result in a hypocapnia sufficient to produce the clinical manifestations which can accompany CO_2 withdrawal [188].

The question arises as to whether or not an increasing level of CO_2 in an astronaut's ambient atmosphere could actually confer some protection on him from decreasing otherwise hypoxic levels of oxygen. It has been suggested in theory and established empirically that by stimulating ventilation with a proper amount of CO_2 in the inspired air, the alveolar oxygen tension can be somewhat increased, and so performance and well-being at moderate altitudes maintained [123, 161, 194, 210]. However, since the major factors underlying this phenomenon are the displacement mainly of nitrogen in alveolar air by CO_2, with an associated elevation of the alveolar oxygen tension due to increased ventilation, it is readily apparent that CO_2 can not confer any protection from hypoxia in a pure oxygen space atmosphere [123]. Moreover, it is doubtful if this effect could exist to a significant degree in proposed space atmospheres, which have a much lower inert gas concentration than air [124].

Although it has not been shown by some investigators, there appears to be a significant decrease of man's respiratory responsiveness to CO_2 with increasing depth of natural sleep [10, 16, 34, 126, 170, 173]. As well, there is an associated respiratory depression resulting in an

elevation of alveolar and hence arterial CO_2 [10, 16, 17, 34, 66, 73, 173]. One group of investigators recorded a peak alveolar CO_2 increase averaging 9 mm Hg in 14 subjects who were in deep sleep not assisted with a hypnotic drug [16]. Since the majority of normal individuals studied remained asleep until their ambient CO_2 reached 4 percent or their alveolar CO_2 reached 50 mm Hg, one wonders, therefore, if an astronaut exposed to an increasing level of inspired CO_2 while asleep might on awakening suffer from the clinical manifestations which can accompany CO_2 withdrawal [16, 34, 173].

An elevated level of inspired CO_2 can lead to a decrease in body temperature, even in a comfortable or warm high humidity environment [28, 32, 90]. For example, Brown [28] recorded a 1 to 3° F decrease in body temperature, with associated chilly sensations, during, and for many minutes after their subjects ceased breathing about 5 percent CO_2, which accumulated in their 72° to 77° F environment over a period of several hours. This lowering of the body heat store may be due to a combination of a number of CO_2 effects on the body. Increased heat loss will result from CO_2-induced cutaneous vasodilatation and hyperventilation [33, 75, 186]. In the past few years, attention has been focused on the marked increase in sweating which accompanies acute exposures to toxic levels of CO_2 [31, 109, 178]. This phenomenon, which cannot be attributed per se to an increase in ventilatory work, may be due to one or more of several factors, such as a lowering of the thermostatic setting of the hypothalamus, an increased sensitivity of cutaneous thermoreceptors, an increase in sympathetic nervous system activity, or an augmentation of sweat gland effector activity [32]. It has also been shown that toxic levels of CO_2 markedly suppressed the shivering which followed exposure to a cold environment [33]. In the light of the above considerations one wonders, therefore, if acutely elevated CO_2 concentrations could increase an astronaut's susceptibility to cold, leading to a lowering of body temperature and associated symptoms sufficient to reduce his functional capacity or even to render him significantly more susceptible to hypothermia (Chapter 7).

A practical question that arises is whether the oxygen cost of increased

ventilation in response to elevated levels of inspired CO_2, especially if this gas is maintained at tolerable levels for a prolonged period of time, could possibly impose a significant drain on space suit or spacecraft cabin oxygen stores. In one study, the oxygen consumption of 12 normal subjects exposed to elevated concentrations of CO_2 in air increased linearly by 2.3 ml per minute per mm Hg rise in alveolar CO_2, or by 1.4 ml per liter of air breathed [24]. The latter value corresponds quite well to other data on the oxygen cost of breathing [37, 38, 44]. Applying the former value to the CO_2 response curve in Figure 15.2 and assuming an R of 0.8, the inhalation of 2.8, 5.6, and 8.4 percent CO_2 would on the average increase the oxygen uptake by about 6, 21, and 47 ml per minute, respectively. Such data indicates, therefore, that until the inspired CO_2 reaches highly toxic levels, as will be defined below, increased ventilation should place a relatively insignificant demand on oxygen stores in space systems. By the same token, it is noted that at highly toxic CO_2 levels, there will also be a significant increase in CO_2 output, and hence an accelerated rise of atmospheric CO_2.

Data on the effects on man of breathing various concentrations of CO_2 while performing at various work loads have been confusing. In one study it was shown that 3 percent CO_2 augmented the ventilatory response on commencing work, yet in another, 5 percent CO_2 did not alter this response [97, 109]. Sustained work has been found to depress the threshold of the respiratory center to CO_2, presumably due to lactic acid accumulation and elevated body temperature [5]. Combined work and CO_2 have reportedly had additive and multiplicative effects on ventilation [4, 111]. Craig [45] generalized from his own as well as other data that although the increment of minute ventilation produced by a change from rest to exercise is increased by CO_2, the ratio of exercise minute volume to resting minute volume is decreased by this gas. It was assumed that this effect was due mainly to a depression of the respiratory center by increasing partial pressures of alveolar CO_2.

From a space operational standpoint, however, it is important to point out that an astronaut's work capacity could be significantly limited, possibly at CO_2 levels, which would not have an observable effect on him

at rest. In 1908, Hill and Flack [98] reported that the work output of a mine worker diminished when the level of CO_2 accumulating in his breathing apparatus passed 2 percent. It is a well known fact among submariners that dyspnea and fatigue can severely limit the performance of heavy manual tasks when the ambient atmospheric CO_2 concentration rapidly reaches 3.0 to 3.5 percent. Although such information is of great operational value with respect to manned space missions, the effects of various partial pressures of acutely elevated inspired CO_2 on man's maximum work capacity have apparently not been reported in the open literature. Instead, interest has been focused on more physiologic aspects of exercise-CO_2 interactions, especially on associated changes in lung ventilation.

The cause of this inordinate dyspnea and fatigue, which characteristically occur at much lower levels of ventilation than that produced by severe work or by breathing a high CO_2 level alone, is unknown [224]. It has recently been shown in animal experiments that the ability of the body to increase metabolism above basal levels is significantly inhibited by hypercapnic acidosis [145, 165]. Since the availability of free acids, which are the main fuel utilized by the body during exertion, is limited by acidosis, the combined acidotic effects of work and CO_2 breathing on this metabolic pathway might account for the lowered CO_2 tolerance for a given work level, and vice versa [141].

Since dyspnea is the major symptom which limits work performance in a CO_2-containing atmosphere, the ventilatory responses to low levels of inspired CO_2 at rest and during exercise might indicate what minimum level of acutely inspired CO_2 might possibly affect an astronaut's maximum work capacity. Haldane and Priestley [89] reported that during work in a closed chamber, accumulating CO_2 in the chamber appears to "affect respiration" at an atmospheric concentration of 2 percent, as compared to 3 percent at rest, due to increased metabolic production of CO_2. Krogh and Lindhard [109] observed that the initial increas in ventilation on commencing severe exercise is markedly elevated in a 3 percent CO_2 atmosphere as compared to when the same degree of work is performed in air. Brown [28] noted that active exertion had a slight

effect on the respiration of his subjects when their ambient atmospheric CO_2 accumulated to the 2 percent level. Data has also been compiled showing that sustained exercise can markedly lower the ventilatory threshold for CO_2 [5]. Thus it is conceivable that a relatively low level of atmospheric CO_2, possibly as low as 2 percent, might degrade an astronaut's maximum work capacity significantly. Again, it is apparent that space-oriented research aimed at better defining man's work capacity at various levels of inspired CO_2, especially at maximum recommended levels of CO_2 for space atmospheres, is definitely indicated.

An increase in sympatho-adrenal activity is another major physiologic response which appears to protect man from otherwise toxic concentrations of CO_2 to which he is acutely exposed. It is a well established fact that up to highly narcotic levels of CO_2, an increase output of catecholamines counteracts the depression of cardiac and smooth muscle produced by the CO_2-induced acidosis and thereby prevents eventual secondary hypotension and "shock" [29, 131, 136, 144, 155, 167, 194, 198, 212]. It should be remembered, however, that hypercapnic acidosis produces two antagonistic effects in the mammalian preparation [127, 129, 141, 145, 165]. On the one hand, the sympatho-adrenal system is stimulated by the decrease of arterial pH. On the other hand, the functional effects of the catecholamines are inhibited, again presumably due to the lowering of pH. The net result of this antagonism on the cardiovascular system might be altered considerably by such factors as drugs, hemorrhage, plasma and fluid loss, or concomitant metabolic acidosis or alkalosis.

Many investigators have shown that systolic and diastolic blood pressures and heart rate increase when normal individuals breathe CO_2-rich mixtures [29, 55, 82, 119, 138, 188, 194, 198]. In spite of earlier reports to the contrary, cardiac output also increases [3, 55, 128, 167, 194, 198]. These cardiovascular events have been attributed to two actions of CO_2. First, the increase in ventilation in response to CO_2 could augment venous return, and so cardiac output through the thoracic pump mechanism [3, 75, 198]. This mechanism may well operate alone up to

inspired CO_2 concentrations of about 6 percent, and appears to exert its greatest effect when the subject is in a horizontal position, which minimizes venous pooling in the extremities [3, 82]. Second and most important, Sechzer and coworkers [198] have demonstrated in normal subjects that at alveolar CO_2 levels above 50 mm Hg (corresponding to an inspired CO_2 of about 6 percent in Figure 15.2), the concentration of catecholamines and 17-OH corticosteroids in the blood begins to increase. They believe that epinephrine liberated from the adrenal medulla and norepinephrine secreted in the myocardium, together with the effects of increased respiratory effort account for the cardiovascular events produced by CO_2 above this level. The importance of the sympathetic response in protecting man from high concentrations of CO_2 is demonstrated by the fact that sympathectomized subjects respond to CO_2 with hypotension rather than hypertension [43, 207]. The sympathetic response to CO_2 may also have been primarily responsible for preventing orthostatic intolerance both in subjects who breathed 4 to 7 percent CO_2 for varying periods of time after exercise and in quadriplegics who breathed 5 percent CO_2 during tilting [54, 130]. Although 17-OH corticosteroids do appear to help maintain normal myocardial contractility and may also be important in regulating smooth muscle activity, their role in orthostasis has not been defined [205, 212].

It cannot be stated with certainty if CO_2 accumulation in an astronaut's ambient atmosphere would enhance his susceptibility to or protect him from orthostatic intolerance on return to a gravity environment, especially if he has sustained some degree of cardiovascular deconditioning during his exposure to weightlessness (Chapter 10. It is a well established fact that both CO_2 and exercise are vasodilatory. Accordingly, it is thought possible that this susceptibility might be significantly increased, especially when an astronaut ceases muscular activity, since the latter assists in maintaining an adequate venous return and provides some protection against orthostatic intolerance.

Finally, it should be noted that studies of the effects of intravenous buffering agents on the sympatho-adrenal response to CO_2 appear to have elicited the probable major mechanism by which CO_2 produces toxic

manifestations. The CO_2-induced changes in blood pH in animals have been buffered with intravenous sodium carbonate or an amine buffer, tris (hydroxymethyl)-aminomethane. In a study using sodium carbonate, the blood catecholamines increased, whereas in studies using the amine buffer, the blood catecholamines remained unaltered [101, 117, 118, 143, 144]. Based on earlier work in this area, the difference in these observations was attributed to the fact that blood sodium bicarbonate, which increased during the sodium carbonate infusion, diffused less rapidly than CO_2 into the intracellular space, whereas the amine buffer rapidly distributed itself throughout both the intra- and extracellular spaces, and thus maintained more constant intracellular bicarbonate-carbonic acid relationships [101, 117, 142, 143, 156, 174, 220]. These findings suggest, therefore, that the sympatho-adrenal response to CO_2 results from intracellular pH changes. Since administration of the amine produces a marked suppression of respiratory activity, it is probable that intracellular acidosis is the major mechanism of CO_2 toxicity [14, 22, 27, 122, 142, 154]. This action of tris (hydroxymethyl)-aminomethane will again come into consideration when its potential value in treating CO_2 toxicity in space is assessed.

Natural body buffering activity does not respond rapidly enough to counteract the acidotic effects of an acute exposure to CO_2 [20, 46, 53, 57, 69, 81, 152, 168, 196, 197, 212, 219]. This fact was borne out well in the experiments of Schwartz and coworkers, who noted that if humans breathed high concentrations of CO_2 for one to 2 hours, there was only a slight increment in blood bicarbonate in the face of a doubling of the arterial CO_2 from 40 to 80 mm Hg and a profound drop in arterial pH [20, 196]. As will be pointed out in the discussion of chronic CO_2 toxicity, many hours, days, and even weeks may be required for extrarenal and renal buffer mechanisms to achieve presumably adequate total body buffering of the acidosis produced by a given level of inspired CO_2. Accordingly, it is reasonable to suggest that buffering agents be administered to compensate for this deficiency.

Very little is known about the relative contributions of an increased

partial pressure of CO_2 *per se* and the concomitant CO_2-induced decrease in the pH of body tissues to the primarily neurological signs and symptoms of acute CO_2 toxicity [136, 155, 201, 215, 221, 225]. There appears to be little doubt that CO_2 exerts a narcotic effect, possibly through the combination of CO_2 and amino acids which then participate in reversible biochemical equilibrium reactions, or through a mechanism involved in the production of general anesthesia [60, 202]. On the other hand, it may well be that the manifestations of CO_2 toxicity are mainly due to alterations of the hydrogen ion concentration in the cellular environment. This is supported by the fact that the tolerance to high partial pressures of blood CO_2 of patients suffering from chronic respiratory acidosis correlates well with the ability of the body to maintain blood pH at or near normal by buffering mechanisms [136, 157, 201, 221]. Westlake and coworkers [221] presented a plausible explanation for this hydrogen ion effect, noting that even small changes in intracellular pH produced by CO_2 will depress tissue oxygen consumption, which is a vital factor for the maintenance of consciousness. This again brings up the question of whether practical measures could be taken in an emergency situation in space to enhance the intracellular buffering capacity and so increase an astronaut's tolerance to inspired CO_2.

The diuresis produced by even low toxic levels of CO_2 is a physiologic reaction which might conceivably have adverse effects on an astronaut. Barbour and coworkers [6] found that exposure of normal recumbent subjects to 5 and 7 percent CO_2 produced a threefold increase in urine output over and above the normal diuretic response to recumbency. These investigators also showed that exposure to 5 percent CO_2 for over 3 hours without replacing the fluid loss could lead to marked hemoconcentration. They and others have noted that the diuretic response is slight in the sitting position, may be abolished by the erect posture or by applying high tourniquets on the thighs while supine, and that it may be restored by standing in a tank of water or by mild exercise [218]. Accordingly, it has been suggested but not proven that this response results from stimulation of intravascular stretch receptors in the left

atrium and pulmonary vessels by an increase in central blood volume, by some mechanical action on the atrial wall from exaggerated respiratory movements, or by an increase on the atrial transmural pressure gradient (58, 141, 218). If one or more of these mechanisms does operate to some degree, afferent connections from these receptors would inhibit the production of antidiuretic hormone by the neurohypophysis (95). Since voluntary hyperventilation, with alveolar CO_2 being maintained constant by inhaling a 2 percent mixture, has been shown to produce much less of a diuresis than CO_2 *per se*, it is also likely that CO_2 acts directly on the neurohypophysis (218). Support for this action being hydrogen ion dependent is given by the findings that a CO_2-induced diuresis did not occur either when the blood pH was maintained near normal by administering a buffer on exposure to CO_2, or during the first few days at altitude when an uncompensated respiratory alkalosis would presumably have prevented the attainment of a critical intracellular pH on breathing CO_2 (163, 217, 218). Finally, there still remains the possibility that CO_2 might inhibit the effectiveness of antidiuretic hormone on the renal tubule (209).

Since CO_2 exerts such a marked diuretic effect on man in the recumbent position, it is probable that this effect would be of similar, if not greater magnitude in the weightless environment. Study of the diuretic response to various concentrations of CO_2, especially with the exposed individual performing at various work loads, is indicated before potential hazards of such a diuresis can be implied. One would think that if a significant diuresis can occur at relatively asymptomatic levels of CO_2, exposure of an astronaut in a space suit to such levels might limit the duration of his extravehicular activity by virtue of a need to void urine. It should also be kept in mind that excess loss of body fluid will decrease tolerance to heat and cold and increase the orthostatic intolerance of an astronaut entering a gravity environment.

The effect of combined CO_2 and heat stresses on man has apparently not been determined. Since they are both vasodilatory and, as previously mentioned, CO_2 markedly increases sweating, it is likely that CO_2 could

enhance an astronaut's susceptibility to heat disorders (Chapter 6). This could be particularly significant if an astronaut commences activity in a gravity environment after exposure to a period of weightlessness which might render him more susceptible to orthostatic intolerance.

As was pointed out in Chapter 4, CO_2 markedly increases an individual's susceptibility to decompression sickness. The risk of development of manifestations of decompression sickness would, of course, be enhanced still further if a CO_2 exposure occurs while an astronaut is performing work under conditions of decompression (Chapter 4).

A review of the literature has failed to find reference to gross or microscopic pathologic changes which can result from an acute exposure to CO_2. The often marked increase in intracranial pressure, frequently accompanied by papilledema, in patients suffering from respiratory acidosis has been attributed to marked CO_2-induced cerebral vasodilatation, with or without cerebral edema secondary to the altered hemostatics [136, 167]. It could well be that many symptoms of acute CO_2 toxicity, especially those that continue for some time beyond the period of exposure, result from either cerebral edema or a vasomotor phenomenon caused by exposure to, then withdrawal from CO_2 environments [186]. Except for these effects, which do not seem to produce permanent pathologic changes, it appears that any other temporary or permanent toxic consequences of CO_2 exposure would be found at the biochemical level.

Clinical Manifestations

The clinical manifestations of acute CO_2 toxicity are also well documented in the literature. Although several excellent reviews [56, 136, 155, 157, 201, 221] have discussed the consequences of acute CO_2 retention in patients suffering from lung disease, such information is quite impractical from the standpoint of predicting an astronaut's response to acutely elevated partial pressures of CO_2 in his inspired air. Caution must be taken in extrapolating to space operational situations the results

of numerous experiments in which man has been exposed from minutes to several hours to constant or gradually increasing levels of CO_2. As will be pointed out in the following brief summary of these experiments, practically all CO_2 exposures have been carried out on resting subjects. Since exercise appears to markedly effect man's tolerance to CO_2, one would not expect the results of resting exposures to be applicable to a situation in which an astronaut is exposed to elevated levels of inspired CO_2 while having to perform work, such as during extravehicular operations in space. It will also become apparent that past experiments have actually yielded very little information on the time of onset and the degree of functional impairment which occurred during and in the immediate period after various acute exposures to CO_2. Although past experiments have yielded enough information for reasonable recommendations of maximum allowable levels of CO_2 for acute exposures to this gas in space, confirmatory data should be obtained in studies which simulate possible modes of exposure during operations in space, especially extravehicular activity while performing various work loads. Finally, it should be kept in mind as experimental data is taken into account, that air resistance imparted by breathing circuits could have aggravated toxic responses to CO_2, and so could have produced the variable results in various studies where subjects breathed the same concentrations of CO_2 (8, 24, 38, 72, 160).

Much has been written on man's clinical response to various constant CO_2 levels to which he is acutely exposed. As pointed out above, such exposures simulate for all practical purposes the rapid increase of CO_2 in space suits.

Schaefer and coworkers (183, 188) recorded the symptoms experienced by 39 normal resting subjects who were alternately exposed for 15 minutes to air and, in order, 1.5, 3.3, 5.4, and 7.5 percent CO_2. No symptoms were reported at the 1.5 percent level; those at other concentrations are listed in Table 15.1. It was noted that these symptoms usually appeared during the last 5 minutes of the exposures. The marked

effect which 7.5 percent CO_2 had on the nervous system is readily apparent. Symptoms at this level would no doubt have led to severe impairment of performance of a psychomotor task. Also of interest was the observation that individual differences in response to CO_2 in this study were related to the pre-exposure respiratory patterns of the subjects. It was noted that those individuals who had the combined respiratory characteristics of a relatively high tidal volume, slow respiratory rate and high partial pressure of alveolar CO_2 before exposure to a given level of CO_2, showed less of a ventilatory response and experienced much milder symptoms than those who had the combination of a relatively low tidal volume, fast respiratory rate and low alveolar partial pressure of CO_2.

	3.3% CO_2	5.4% CO_2	7.5% CO_2
Dyspnea	2	4	24
Headache	0	0	15
Stomach ache	0	0	1
Dizziness	0	0	6
Sweating	1	1	5
Salivation	0	0	1
Numbness of extremities	0	0	5
Cold sensations	1	1	3
Warmth sensations	1	1	4
Increased motor activity	0	0	10
Restlessness	0	0	10
Loss of control over limbs (overactivity)	0	0	4
Loss of balance (spatial disorientation)	0	0	7
Color distortion	0	0	2
Visual distortion	0	0	6
Irritability	0	0	4
Mental disorientation	0	0	2

Table 15.1 Symptoms occurring in 39 resting subjects who inhaled CO_2 for 15 minutes.

(After Schaefer et al [188]).

From numerous other studies, a more detailed CO_2 response spectrum can be described. During the first day of their exposure to 3 percent CO_2, several individuals remained mentally keen in spite of exhibiting general excitement and increased activity [36, 183]. Four percent CO_2 was found to be the upper limit tolerated by sleeping individuals and has been shown to increase the auditory threshold significantly and to

lengthen the latent period of the negative afterimage [16, 78, 79].

Proficiency at card naming and sorting was unaltered during the exposure of 31 subjects to 5 percent CO_2 for 16 minutes, although all of these subjects were moderately dyspneic, most reported fatigue, fogginess and an effort to concentrate, two experienced visual disturbances, and one failed to complete the last minute because of dizziness, marked dyspnea and impending fainting [223]. It is noted that most of these individuals, of whom many were experienced pilots, were of the opinion that 5 percent CO_2 for a 16 minute period was close to a marginal concentration for the safe operation of an automobile or airplane [225]. Other studies carried out at the 5 percent level have found a significant increase in the pain threshold and decrease in the fusion frequency of flicker [189, 204, 209].

Two observers who entered a 5.7 percent CO_2 atmosphere in which several individuals were tolerating a gradual increase of CO_2 immediately became so dyspneic that they were unable to make observations [28]. Seven subjects tolerated 6 percent CO_2 for about 22 minutes, but experienced marked dyspnea, flushing and sweating of the face, and feelings of stupification and impending collapse, especially toward the end of the exposure [29]. Visual intensity discrimination has also been shown to be affected in studies at the 6 percent level [74]. Prolongation of the time required for addition and cancellation tests, and the existence of dissociation, perseveration and an increase in the unusualness of response has been demonstrated in subjects breathing 6 to 7 percent CO_2 [76, 77].

In contrast to the symptoms reported in the above exposures to 6 percent CO_2, the "mental status seemed unaffected" in 7 subjects who breathed 7 percent CO_2 for 40 to 90 minutes, although all suffered from dyspnea and some complained of mild headache and burning of the eyes [20]. Exposure to 7.5 percent CO_2 for 3.5 to 6 minutes has been tolerated, but symptoms had a shorter lag time than in 7 percent CO_2 [29]. The 7.5 percent CO_2 level has also been found to decrease the inhibitory effect of light stimulation on brain waves - a finding which demonstrated the depressive or narcotic action of CO_2 on the central nervous system [189]. An experiment in which 42 subjects who breathed 7.6 percent CO_2 for

2.5 to 10 minutes yielded results similar to the other experiments near this CO_2 level, although one subject did lose consciousness [29, 188].

Individuals who have been exposed to 10 percent CO_2 have immediately experienced one or more of a number of clinical manifestations, such as extreme dyspnea, visual and auditory hallucinations, chilliness, nausea, and vomiting, a strangling sensation, burning of the eyes, cloudiness of vision and profuse sweating. They have usually become stuporous within 10 minutes and lose consciousness within 15 minutes [29, 41, 55, 64, 199, 222]. Although CO_2 concentrations of over 20 percent have been used for the treatment of mental disorders and experimentally for anesthesia, it is considered probable that if an individual who does not have the benefit of therapeutic support is exposed to CO_2 levels above 10 percent, he will rapidly suffer the sequence of respiratory depression, convulsions, "shock", and death [116, 132, 135, 222].

The classic work of Haldane and Smith in 1892 [90] elicited many of the clinical manifestations to be expected from exposure to gradually increasing inspired CO_2. These investigators had normal resting subjects rebreathe air while enclosed in a 70 cubic foot chamber. As noted previously, the rate of CO_2 accumulation in this volume would apply to that generally expected in spacecraft cabin atmospheres. In one experiment, chamber CO_2 increased linearly over an 8 hour period to 6.4 percent, while oxygen decreased to 13 percent. About 4 percent CO_2, the subject became aware of increased breathing and began to complain of headache and nausea. For the last two hours of exposure, when CO_2 had passed about 5.2 percent, breathing was "painfully labored and required so much exertion as to cause great exhaustion". This marked dyspnea eventually caused termination of the experiment. Another subject showed a similar response, having to end his 7 hours in the chamber after linear CO_2 and oxygen changes to 5.8 and about 14 percent, respectively. By allowing changes in either CO_2 or oxygen in the chamber, Haldane and Smith [90] showed that the decrease in inspired oxygen in the above experiments could not have been a factor in producing these various manifestations of rebreathing air until the oxygen concentration fell below

13 percent. Finally, they demonstrated that resting subjects who rebreathed air from a 225 liter bag could tolerate a maximum CO_2 concentration of about 10 percent, attained in about 1.5 hours. They suffered from mental confusion and extreme perspiration in addition to the manifestations described above, as this level was reached.

Studies by Brown in 1930 [28, 29] have also yielded valuable information on man's response to gradually increasing inspired CO_2. Normal resting subjects in groups of four rebreathed 654 cubic feet of chamber air (164 cubic feet per man). In the first experiment described, the chamber CO_2 increased linearly to 4.7 percent, while oxygen decreased to 15.5 percent, over a period of 10 hours. At about 2 percent CO_2, "active exertion had an effect on respiration". All subjects tolerated this exposure, none complaining of actual dyspnea; one suffered from a mild headache for the last 3 hours. General fatigue and listlessness experienced during the exposure did not seem to alter subject alertness. In the second experiment, CO_2 was gradually introduced into the chamber, with linear changes in this gas and oxygen to 4.8 and 17.8 percent, respectively, taking about 5.3 hours. Notably, all subjects reported chilly sensations during the final hours, in spite of an average chamber temperature of 75° F and relative humidity of 74 percent. Oral temperatures recorded in three subjects fell 1.0, 2.2, and 3° F. Again, dyspnea was noted, mild headache occurred in one subject, and general fatigue and listlessness were outstanding. In the third experiment, CO_2 was again introduced into the chamber, and changed along with oxygen in an essentially linear manner until these gases reached 5.2 and 15.6 percent, respectively, by the end of 8 hours. Breathing became decidedly labored for all subjects, with two reporting dyspnea in the last half hour, when the CO_2 level was above about 4.8 percent. Fatigue was more marked than in the above experiments and again all subjects had chilly sensations, associated with a fall in body temperature. Three experiments similar to the last reached 5.6, 5.7, and 5.8 percent CO_2 and 16.1, 15.4, and 14.2 percent oxygen in 8 hours. All subjects made note of experiencing a pronounced increase in the depth of breathing soon after the 4 percent CO_2 level was reached, the

stage of panting virtually setting in at this time. During the last hour in all three of these experiments, when CO_2 was above 5.1 percent, all subjects except one who was found to be relatively resistant to CO_2, complained of dyspnea in addition to exhaustion from severe panting. Most reported headache, which was frequently associated with nausea, for the last two hours, when the CO_2 was above 4.6 percent. Again, mild hypothermia with associated chilly sensations occurred in spite of the subjects being in a presumably comfortable environment. A number of experiments with similar increases of CO_2 as those above, but with oxygen maintained near normal levels, ruled out a significant contribution of decreased oxygen, at least down to the 15.5 percent level, in producing these various toxic manifestations. From his own and other past studies, Brown estimated that oxygen depletion would begin to have an effect about the 14 percent level. In the experiments where CO_2 and oxygen reached respective levels of 5.8 and 14.2 percent, and 5.3 and 20.5 percent, Brown found evidence that excellent psychological reserve was maintained, with subjects failing to show an altered response to a variety of tests of attention, memory, association, deduction, and motor coordination.

Other studies have substantiated the findings of Haldane and Smith, and Brown [1, 42, 89, 92, 98, 194]. In experiments in which inspired CO_2 increased to the 5 to 7 percent range in one to 4 hours, it was also noted that the incidence of clinical manifestations of CO_2 toxicity increased rapidly above 5 percent CO_2 [92]. Particular attention is drawn to the occurrence of nausea and vomiting above this level, for vomiting in the weightless environment will be an extremely hazardous event (Chapter 8) [192]. Mental confusion and dizziness occurred about 7 percent CO_2 [92]. In other rebreathing experiments, the maximum tolerable limit was again about 10 percent CO_2, this level being reached in about 4 hours [98].

As mentioned above, symptoms can be experienced after the cessation of certain exposures to CO_2 and, as the examples given below will show, can result in even greater functional impairment than symptoms experienced

during such exposures. This phenomenon may have been a major factor preventing the successful escape of personnel from the sunken submarine Thetis [1].

A number of CO_2-withdrawal symptoms have been reported following exposures to various maintained levels of CO_2. This reaction and its marked variability was well demonstrated by a study in which 5 subjects breathed 6.7 percent CO_2 for one hour [1]. On ceasing their exposure, one subject immediately vomited repeatedly and complained of nausea and headache, two experienced temporary severe incapacitating headaches, and two complained of only slight headache. In other studies, subjects exposed to 3 percent CO_2 for many hours apparently complained of only a mild headache on returning to air [36, 179]. Headache was also complained of after exposures to 5.2 and 6.4 percent CO_2 for 2 hours [50]. A frequent symptom after cessation of exposures to 7.6 percent CO_2 for an average of 7.4 minutes and 10.4 percent CO_2 for an average of 3.8 minutes was temporary dizziness [55].

Clinical manifestations have also occurred after withdrawal from exposure to gradually increasing ambient CO_2 levels. A classical example of a particularly severe CO_2-withdrawal reaction was cited by Alexander and coworkers [1]. Their subject tolerated an increase in CO_2 to 6.6 percent over a 14.5 hour period, yet, when subsequently exposed to oxygen, immediately vomited a pint of clear fluid in spite of not having ingested anything for over 16 hours. He also began to suffer immediately from a violent diffuse headache, which appears to have incapacitated him for about an hour. In the Haldane and Smith study [90] the headache and nausea experienced by the two subjects, as CO_2 increased to 6.4 and 5.8 percent over 7 and 8 hours, was reported to have temporarily worsened when they left the chamber. In Brown's experiments [28, 29], withdrawal from concentrations of CO_2 reaching from 4.8 to 5.8 percent over 5.3 to 10 hours was accompanied either by the onset or aggravation of headache or nausea which lasted for 1 to 3 hours. Hayter and Duffner [92] found that for an as yet unexplained reason, the headaches resulting from exposure to CO_2, which increased to 5 to 7 percent over one to 3 hours, were much worse, occurred with

greater frequency, and lasted much longer in subjects who breathed air as compared to those who breathed oxygen after exposure.

The cause of the above clinical manifestations of CO_2 withdrawal is unknown. The brief hypotension which coincided with the temporary dizziness immediately after ceasing the brief exposures to 7.6 and 10.4 percent CO_2 might possibly be due to the vasodilatory action of CO_2 persisting beyond its sympathetic action in the immediate post-exposure period [55, 167]. Other effects of altered sympatho-adrenal activity, which could accompany CO_2 withdrawal, might conceivably cause symptoms [141]. Whether the temporary undershoot of alveolar CO_2, observed when 15 minute exposures to 5.4 and 7.5 percent CO_2 were terminated, might produce a hypocapnia of a sufficient magnitude to produce a symptom such as dizziness remains to be determined [188]. Finally, it is conceivable that a cerebral vasomotor phenomenon caused by exposure to, then withdrawal from a CO_2 environment might be a major etiologic factor [186].

A review of the pertinent literature indicates it is unlikely that CO_2 exposures in space will ever be severe enough to cause such serious consequences of CO_2 withdrawal as prolonged profound hypotension and grave cardiac arrhythmias which are prone to occur following marked CO_2 retention in anesthetized patients [25, 26, 31, 39, 83, 137, 155, 166, 167].

Finally, it is pointed out that certain symptoms which are not really specific effects of CO_2 withdrawal might occur in the post-exposure period. Marked, general fatigue, and soreness in the region of the diaphragm have been reported after most of the prolonged acute exposures to over 4 percent CO_2 described above. Such symptoms could no doubt limit an astronaut's physical work capacity for several hours after such an exposure. Conceivably, intense shivering might be experienced after certain exposures to CO_2 which, as mentioned above, can cause an excessive loss of body heat during exposure.

The results of most of the experiments in acute CO_2 toxicity have been summarized graphically in Figure 15.3 for CO_2 exposures of up to