

## Chapter 3

### **EFFECTS OF DECREASED PRESSURE: DECOMPRESSION SICKNESS**

Frederick W. Rudge, MD, MPH  
Benton P. Zwart, MD, MPH (Feb 2002)  
<http://www.brooks.af.mil/web/hyper/>

#### **INTRODUCTION**

Decompression sickness (DCS) is an illness caused by reduced atmospheric pressure on the body that results in formation of bubbles of inert gas (nitrogen) within body tissues. These bubbles result in symptoms of DCS, which can range from mild joint pain to permanent neurologic deficits to death. Each year, 50 - 100 cases of DCS are treated in USAF hyperbaric facilities. The role of the flight surgeon in the diagnosis and initial management of DCS is crucial; inadequately treated, DCS can lead to disability or death.

DCS can occur from decompression during flight, from altitude chamber exposure, from diving, from working in pressurized tunnels or caissons, or from hyperbaric chamber exposure. Most cases of DCS treated by USAF hyperbaric facilities are due to altitude chamber exposures. Exposure to reduced atmospheric pressure during flight can occur in a variety of ways. Some aircraft, such as the T-37, lack a pressurization system, yet are capable of flying at altitudes sufficient to result in DCS. Other aircraft which possess a pressurization system can lose pressure due to failure of the pressurization system, loss of the canopy, or other breach in the integrity of the pressure vessel. Mission requirements may require unpressurized flight at high altitudes, such as high altitude parachute drops, which place both the jumpers and crew at risk. In other aircraft, such as the TR-1, the maximum pressure differential the aircraft can maintain at very high altitudes (greater than 50,000 feet) is not sufficient to protect the crewmember.

#### **HISTORY**

Sir Robert Boyle first described bubble formation in 1670 when he noted a bubble in the eye of a snake which he had exposed to decreased atmospheric pressure in an evacuated bell jar. He did not know what the bubbles were composed of, nor why they were generated. The first description of the symptoms of DCS in humans was published by Triger in 1841. He noted that coal miners working under increased atmospheric pressure suffered from muscular cramping and pains after decompressing to the surface. In 1854, Pol and Watelle also noted similar symptoms in compressed air workers. They also noted that the symptoms were alleviated upon return to the

compressed air environment. None of these investigators could explain the etiology of the workers symptoms.

The first scientific approach to decompression sickness was begun by the French physiologist, Paul Bert. In 1878 he published his book, *Barometric Pressure*. He was the first to correctly deduce that the symptoms of DCS were caused by bubbles of nitrogen which evolved due to a rapid decrease in atmospheric pressure. He also discovered that oxygen is toxic to the central nervous system when breathed at pressures greater 33 feet (2 atmospheres) for extended periods, resulting in convulsions. This CNS toxicity is now termed the "Paul Bert effect."

During this time, the term "bends" was coined as a synonym for DCS. During the construction of the Brooklyn Bridge in the late 1800's, many workers developed DCS from working in pressurized caissons used to build the bridge abutments. (DCS was often referred to as caisson's disease.) Fashionable ladies of the day wore corsets that caused them to assume a bent over posture which was referred to as the "Grecian bend." Caisson workers afflicted with decompression sickness often assumed a similar posture due to back and abdominal pain, and were chided by other workers as doing the Grecian bend. This was later shortened to "the bends," a term which is still frequently used.

At the turn of the century, the Royal Navy was noting a high incidence of DCS in it's military divers. A noted physiologist, J.S. Haldane, was commissioned to work out a schedule of dive exposures to reduce this problem. Through extensive animal experiments, he concluded that the body could tolerate a two-to-one reduction in ambient pressure without symptoms. His findings were published in 1908, and were the basis of the first decompression tables.

In 1917, Yandell and Henderson predicted the possibility of DCS in aviators. In 1919, Herringer formulated the theory that the mechanisms of diving and altitude DCS were identical. DCS was not a problem in early powered flight due to the inability to achieve sufficiently high altitudes. This changed with the advent of the reciprocal, supercharged aircraft engine. During World War II, decompression sickness became a significant problem, stimulating a great deal of interest in DCS research. A screening program was developed in which crewmembers exercised at high altitudes in an altitude chamber to determine susceptibility to DCS. This program tragically resulted in many deaths, and was later determined to be invalid. Progress was made when it was discovered that the breathing of pure oxygen prior to decompression lowered the risk of DCS. The use of pure oxygen to decrease the risk of DCS continues to be used extensively. Research regarding methods to reduce the risk of DCS during flight continues at several sites, including the Armstrong Laboratory at Brooks AFB, Texas.

## **ETIOLOGY OF DCS**

DCS occurs when a person is subjected to a sudden reduction in ambient pressure that causes inert gas bubbles to form and grow within body tissues and vascular spaces. According to Henry's Law, the amount of inert gas which can dissolve in body tissues is directly proportional to the partial pressure of the inert gas in the persons breathing environment prior to decompression.

During any decompression, the partial pressure of inert gas (usually nitrogen) in the environment decreases by Daltons Law, creating supersaturation; a situation in which the body tissues contain a greater amount of nitrogen than predicted by Henry's Law at equilibrium for the new, lower pressure conditions. During the decompression, this increased level of nitrogen equilibrates by diffusing out of the body tissues and traveling in the blood to the lungs, where it is expelled in the expired air. If the ambient pressure decreases faster than the body can off-load the excess nitrogen, a condition of critical supersaturation may develop. When the critical supersaturation threshold is exceeded, the amount of dissolved nitrogen exceeds the ability of the tissues to hold it in solution, and the nitrogen comes out of solution in the form of bubbles. The point at which nitrogen gas forms bubbles, was described by Haldane as the *critical supersaturation point* and was determined by him to be a ratio of two-to-one (2:1). If enough bubbles form, and they grow large enough, the pathophysiologic mechanisms of DCS are initiated.

The simple in-vivo existence of bubbles may not be sufficient to result in DCS symptoms. Moderate numbers of "Silent" bubbles have been shown by Doppler monitoring to occur during many normal, asymptomatic decompressions. Indeed, silent bubbles may be very prevalent in most flying and diving operations, including altitude chamber exposures. These bubbles usually disappear without producing symptoms and with no consequence to the body.

Nitrogen is the most prevalent inert gas in the air we breathe (78%), and therefore is the primary gas involved in the development of DCS. Bubbles do not form solely by the action of oxygen or carbon dioxide because these metabolically active gases have unique blood transport mechanisms, are rapidly consumed or excreted, and their concentrations are controlled by the respiratory and circulatory systems. Nitrogen is inert: neither consumed, excreted, bound to other molecules, nor actively controlled. The solubility of nitrogen in water is relatively low, but in some tissues such as fat, is relatively high. Nitrogen diffuses slowly from fat tissues to the blood.

Once formed, bubble size varies according to Boyle's Law. Any decrease in pressure causes bubbles to grow, while any increase in pressure, such as treatment in a hyperbaric chamber, will cause bubbles to shrink. Flying after an altitude chamber exposure is not recommended, because silent bubbles and bubble nuclei formed during the altitude chamber exposure, may expand and become symptomatic during aircraft flight at decreased atmospheric pressure.

## **BUBBLE EFFECTS ON THE BODY**

Regardless of their precise site of origin, bubbles have two major categories of effect in the body: mechanical effects and blood-bubble interface effects.

Bubbles can be detected in experimental altitude chamber research subjects by Doppler techniques once they reach a size of 40 to 50 microns, especially if there are many of them. A grading scale of bubble severity using a precordial Doppler over the right ventricular outflow tract has been developed by Spencer. Grade 0 indicates no detectable bubbles. Grade 1 indicates an

occasional bubble, with most cardiac cycles free. Grade 2 indicates that many, but less than half, of the cycles contain bubbles. Grade 3 indicates that most cardiac cycles contain bubbles. Grade 4 indicates severe bubbling which obscures the cardiac signal. With grade 0 bubbles, there is little chance of DCS, while with grades 1 and 2 there is moderate risk. The risk of DCS is greatest when grade 3 or 4 bubbling is present. It must be emphasized that the presence of bubbles does not mean the subject has DCS. DCS occasionally occurs in a subject without Doppler evidence of bubbles, and subjects with grade 4 bubbles are frequently asymptomatic.

### **Mechanical Effects**

A gaseous bubble growing in the tissues will displace and deform adjacent structures. It is conjectured that when bubbles reach a certain size, they impinge on nerve endings, resulting in pain. If the deformation pressure of the bubble becomes too great, tearing of tissues takes place, and destruction of formed elements occurs. The resulting tissue trauma may account for post-exposure symptoms, even after the bubbles have dissipated.

Bubbles can mechanically obstruct vessels, leading to distal ischemia. Ischemia in the CNS may result in neurologic symptoms of DCS, and pulmonary symptoms (chokes) in the lungs. A large volume of gas bubbles in the right side of the heart may interfere with cardiac contraction by forming an air lock. This could cause a massive infarction and death, or may lead to right ventricular failure and circulatory collapse.

Unless the bubbles have produced direct tissue damage by overexpansion, the symptoms caused by these mechanical effects usually disappear once the bubble has been reduced in size below a certain critical diameter. This explains why patients with DCS begin to feel better upon compression to treatment depth in the hyperbaric chamber. Recompression produces a reduction in bubble size by Boyles Law, which relieves mechanical pressure on surrounding tissues and nerve endings, and also may relieve capillary occlusion and ischemic pain.

### **Blood-bubble interface effects**

Intravascular bubbles appear to the body's host defenses as foreign bodies, causing enzyme release, complement activation, and release of vasoactive substances. Platelets and red blood cells tend to clump on the surface of bubbles. As venous bubbles produce platelet aggregation, there is a release of vasoactive substances such as serotonin and epinephrine, leading to vasoconstriction. Kinins are also released, causing leakage of fluids from the intravascular to the extravascular spaces. Aggregation in the post-capillary vessels and small veins blocks capillary flow, causing tissue ischemia. The release of platelet factors accelerates clotting and creates further circulatory embarrassment. Blood viscosity increases, along with increasing capillary flow resistance and capillary pressure. These effects lead to large shifts of fluid from the intravascular to the extravascular spaces. The volume loss decreases venous return and further reduces blood flow. The resultant hemoconcentration increases the hematocrit and blood viscosity, and raises the fibrinogen concentration. These relationships generate a vicious cycle which can be reversed only when DCS is adequately treated. The longer definitive treatment is delayed, the greater the pathological effects of blood-bubble interface. These effects may persist for some time after the bubbles are adequately treated and removed, and may explain the residual plateau of symptoms sometimes seen in DCS despite adequate therapy.

## CLINICAL MANIFESTATIONS OF DECOMPRESSION SICKNESS

DCS symptoms have been classically grouped into two types, Type I DCS and Type II DCS. Recently, there has been a trend to drop these groupings in favor of using a more clinically descriptive classification based on specific symptoms. For simplicity of presentation, the classic groupings will be used here.

### **Type I DCS**

Type I DCS includes joint pain (musculoskeletal, or "pain only" symptoms) and symptoms involving the skin (cutaneous symptoms) and lymphatics.

(1) ***Cutaneous Manifestations*** . Skin symptoms can result from extravascular or intravascular bubble formation in the skin. Sensations that are described include pruritis (itching), formication (sensation of insects crawling on the skin), mild stinging or pin pricks (paresthesias), hot or cold feelings that may or may not alternate in occurrence, and sometimes numbness. These symptoms may be associated with a fine scarletiform rash, which is caused by the movement of nitrogen through sweat glands, and may be transient and migratory in nature. Rarely, a crepitus sensation caused by subcutaneous emphysema can occur. These skin symptoms are usually quite transient, and generally require no treatment.

Intravascular bubble formation which impedes skin and underlying tissue bed circulation may cause some skin areas to turn pale and eventually exhibit a mottled, cyanotic appearance, or marbling. The skin is often raised and has a dark blue discoloration which blanches when pressed, and may be intensely pruritic, progressing to an achy soreness. It often begins as isolated patches on the larger, flat surfaces, such as the back, abdomen, or thigh, which then merge into larger confluent areas. This is referred to as *cutis marmorata* (marbling). These manifestations range from being local and transient, to generalized and persistent. *Cutis marmorata is a bona fide form of DCS which should be treated by recompression on a treatment table 6.* Therapy usually leads to complete resolution of signs and symptoms. Marbling is often a harbinger of more serious forms of DCS, since gas bubbles are present in both tissues and blood vessels. The existence of *Cutis Marmorata*, without any other physical or symptomatic findings, is only considered Type I DCS.

(2) ***Lymphatic Manifestations*** . Pitting edema is caused by blockage of the lymphatics by bubbles. It is fairly uncommon and has been reported as a secondary finding in the vicinity of a painful joint which may have been the presenting symptom. It occurs less commonly in the absence of other manifestations, such as edema of a limb or the face. Though specific treatment is not necessary for this condition, hyperbaric therapy may be used to help relieve pain, decrease swelling, and possibly reduce more serious manifestations from developing later. It should be noted that peripheral swelling may disappear completely during recompression, but usually it takes days or weeks for swollen lymph nodes to subside after treatment.

(3) ***Musculoskeletal Manifestations*** . Pain in and around the joints or, less commonly, in large muscle masses, is called the bends. It is the most common manifestation of DCS. Often the pain is described as being "deep" and throbbing. With time the pain tends to radiate or extend along the limb. Severity ranges from barely perceptible to severe. There is a tendency for the person suffering from the bends to work the joint, or soak it in hot water, attempting to ease the pain. This generally is not effective and often worsens the pain.

Bends pain, as with other DCS manifestations, generally worsens with time at altitude. Pain may decrease with descent, but in some cases the pain may begin and worsen after return to ground level. It is rare for the initial symptoms to occur greater than 24 hours after the altitude exposure. Symptoms beginning after the 24 hour point should be investigated for another cause, however, since bubbles may persist if untreated for up to 3 – 5 days, recompression may produce symptomatic relief up to a week after exposure.

Unfortunately, the amelioration of pain occurring on descent frequently convinces the crewmember to continue the mission and not to report the incident after landing. Although the bends is not commonly perceived as having the same degree of consequences as the more systemic forms of DCS, (e.g. CNS disorders), the pain is often progressive and can lead to total debilitation. In severe cases, the pain effectively prevents movement, with obvious consequences to aircraft operations. Severe cases can lead to collapse, either directly by the effects of pain, or by other DCS manifestations occurring at the same time. Furthermore, while bends pain may be the most prevalent and recognizable manifestation of DCS, other manifestations may also be present at a sub-clinical level and silently developing. Some contend that a single episode of untreated bends involving certain critical areas may produce dysbaric osteonecrosis 10 to 15 year later.

Susceptibility of the joint area may be a consequence of the anatomy vis-à-vis circulation and constituency of connective tissue. The joints consist of relatively non-distensible tissue. Bubble formation may be more likely to impinge on sensory nerves contributing to the perception of pain. Additionally, perfusion of these areas is relatively poor, and this may preclude the removal of nitrogen via diffusion gradients established when blood flow is as good as in other tissues. Thus, a perfusion limitation may exist.

Type I DCS of this kind refers to "pain only" symptoms. Joint pain is by far the most common presenting symptom, seen in up to 90% of all cases of DCS, but other types of pain may occur which do not involve joints. The shoulder is the most common site of joint pain, but any other joint may be involved. Active and passive motion of the joint tends to aggravate the discomfort. The application of local pressure by means of a sphygmomanometer cuff may result in considerable relief and may be useful as a rudimentary means of diagnosis. This is referred to as the *pressure cuff test* and is only helpful in making a diagnosis of DCS when the pain is relieved by pressure, however due to the 40% false negative rate, does not permit the clinician to "rule out" DCS if the test is negative. The characteristic pain of Type I DCS has been described as a deep pain, sometimes a dull ache, but rarely a sharp pain, with intensity ranging from an awareness (a "niggle") to an excruciating pain. When joint pain occurs, it is not uncommon to have aching pain in the muscles around the joint.

A sharp, knife-like pain that shoots down an extremity or encircles the body trunk (radicular pain), vague thoracic or abdominal pain, or pain that moves from one area to another should be treated as arising from CNS involvement and treated as Type II symptoms. Pain may mask other, more significant symptoms, and therefore should not be treated with drugs in an effort to make the patient more comfortable. The pain may be the only way to localize the problem and monitor the progress of treatment. Aspirin is not used as prophylaxis, nor treatment, for DCS.

## **Type II DCS**

In the early stages, symptoms of Type II DCS may not be obvious and Type I symptoms may or may not be present at the same time. Type II DCS comprises serious symptoms or signs involving the central or peripheral nervous system, or the cardiopulmonary system. Any aviator experiencing Type II DCS requires rapid treatment to prevent permanent injury or death.

(1) ***Pulmonary Manifestations*** . Chokes is caused by an overwhelming evolution of bubbles from body fluids and tissues which then embolize the pulmonary vessels. Reduced gas exchange and circulatory embarrassment can occur as a direct result of such embolization. The flooding of the systemic arterial circulation by bubbles forced through pulmonary shunts can be an indirect serious consequence of chokes. The most specific early symptom of the chokes is a substernal pain made worse by deep inspiration. The affected person commonly has a feeling of suffocation with an obvious concurrent apprehension. The individual often is pale and sweating and feels fatigued and faint. A dry, progressive cough is frequently present. Total collapse may occur. The classic triad of dry, nonproductive cough, substernal chest pain, and a burning pain on inspiration (dyspnea) must be differentiated from the burning inspiratory pain often felt after breathing dry oxygen for prolonged periods (pulmonary oxygen toxicity, sometimes called false chokes). Substernal chest pain always requires the clinician to consider angina and MI, however if the patients recent history includes decompression, then chokes must also enter the differential.

Chokes is often confused with air embolism. Air embolism (AGE) can be caused by a breach in the alveoli, permitting gases to enter the circulation. Holding one's breath while ascending in diving is the most common cause of air embolism, but it also may occur if the breath is held during a decompression to a higher altitude. Ascent and subsequent expansion of alveolar air can easily and painlessly burst the alveoli in as little as 4 feet of seawater near the surface. Symptoms of AGE typically present within 5 minutes of the rapid decompression.

Chokes that are allowed to continue without treatment typically results in progressive dyspnea with shallow respiration, cyanosis, loss of consciousness, and circulatory collapse. Progression may be fast or slow, occurring over 30 – 60 min, or over several hours. Left untreated, death may follow in either case. Immediate hyperbaric therapy is required.

(2) ***Neurological Manifestations*** . These manifestations appear to be associated with disturbances within the brain or the spinal cord. Neurological DCS may be manifested by a wide range of signs and symptoms involving the central or peripheral nervous system, with signs and symptoms appearing in virtually any area of the body. About 25 percent of all DCS cases include signs of neurologic involvement. Central nervous system involvement includes both the brain and

spinal cord. In aviators the brain is most commonly affected, while lesions in divers often involve the spinal cord. When the spinal cord is involved, the lower thoracic or upper lumbar segments are most frequently affected, producing motor and sensory disturbances like paraplegia, monoplegia, spasticity, loss of bladder and rectal control, muscular weakness, altered reflexes, and paresthesias (collectively from numbness, tingling, and decreased sensation to touch and pain). Some cases of spinal cord DCS begin with girdling abdominal or thoracic pain, which precedes the onset of sensory and motor deficits. Within 30 minutes of onset, the entire clinical picture of a partial or complete transverse spinal cord lesion may be noted.

Cerebral DCS is one of spotty sensory and motor signs and symptoms not attributable to a single brain locus. Visual disturbances are common manifestations from altitude exposure, including blurring, scotomas, tunnel vision, diplopia, and other field defects. Other manifestations frequently noted are headaches, spotty motor or sensory deficits, unilateral paresthesias, confusion, and changes in affect or personality. The deficits may be very subtle, and may not be recognized by the individual affected. Seizure activity, dizziness, vertigo, nausea, vomiting, and unconsciousness may also occur. When the cerebellum is involved, ataxia, tremor, nystagmus or a lack of coordination may be present.

Inner ear or labyrinthine DCS ("staggers") is an uncommon manifestation of central nervous system involvement. It generally only occurs following deep dives, and is very rare from altitude exposure. In these cases, either the cochlea or the vestibule may be involved. The presenting symptoms include tinnitus, deafness, vertigo, nausea, vomiting, and ataxia. Physical examination may reveal nystagmus. Involvement of either the hearing or the vestibular system may predominate. Inner ear DCS is a true emergency, and it must be treated immediately if permanent damage is to be avoided. Because the nutrient arteries supplying the inner ear are small, a rapid reduction in bubble size is important.

Permanent neurologic deficits result from spinal cord DCS and are most feared by divers. Even with proper and rapid treatment, approximately 15% of patients who have suffered spinal cord DCS show some degree of permanent neurologic deficit, ranging from minor sensory and motor losses to complete paraplegia.

(3) ***Circulatory Manifestations*** . Circulatory collapse is a rare form of DCS. Generally, circulatory impairment is manifested as shock following the development of chokes, severe bends pain, or severe neurological impairment. Circulatory collapse without other symptoms preceding the development of shock has not been reported. Possible mechanisms of circulatory collapse include direct involvement of the vasomotor regulatory center, overwhelming release of vasoactive substances, or massive vessel endothelial damage by the bubbles, with subsequent loss of intravascular fluid. Hematocrits as high as 70% have been recorded in some cases. Whatever the course, a hallmark of this grave sign is its failure to respond to fluid replacement, as fluids are third-spaced as fast as they can be administered. This refractory circulatory collapse is similar to the response commonly seen in a severe head injury that results in a central "sympathectomy." Treatment should consist of hyperbaric therapy and fluids, along with vasopressor agents. Although recovery is not assured even with prompt therapy, failure to treat the patient within 6 to 24 hours after symptom onset is likely to result in death.



## **FACTORS AFFECTING DCS INCIDENCE AND SEVERITY**

Certain physiological and environmental factors pertaining to both altitude and diver DCS are thought to increase the likelihood or severity of this syndrome. Most of these factors influence the blood supply to tissues, and therefore, the speed of gas uptake or release. In general, analyses of these factors do not predict the probability that a person will suffer DCS, but are believed to indicate individual susceptibility in a large unselected group.

***Altitude*** . Increasing the altitude contributes to an increased incidence of DCS. A higher altitude increases the degree of supersaturation which increases the probability of bubble formation. A flyer whose body tissues are in equilibrium at sea level achieves a condition of supersaturation during decompression to altitude, during the loss of aircraft pressurization or during simulated flights in an altitude chamber. Considerable debate surrounds the minimum, or threshold altitude for DCS. Recent evidence indicates DCS is possible as low as 15,000 feet, but it is exceedingly rare unless an extended period of time is spent at the altitude. Although no discrete boundary exists, the most commonly cited minimum "bends altitude" is 25,000 feet. Based on decades of unpressurized flight in Air Force trainers to FL 250, this appears to be a reasonable supposition, although low symptom severity, resolution on descent, reporting bias and a slow ascent rate may be important cofactors. The occurrence of DCS with altitude exposures of less than 18,000 feet is very rare, unless there was a precipitating factor, such as recent exposure to compressed gas breathing (scuba diving) within 24 hours. After scuba diving, DCS onset may occur at altitudes of 5,000 feet or less. Aircraft pressurization systems have greatly decreased DCS incidence because they effectively limit the crewmembers exposure to safe altitudes (cabin altitude). However, at some flight altitudes, e.g., greater than 40,000 feet, the cabin altitude may still be in the DCS danger area. More commonly, a loss of pressurization because of a system failure may rapidly expose the crewmember to a hazardous outside pressure.

***Time at Altitude*** . Increased time at altitude contributes to a greater incidence of DCS. It would seem logical that if one remains at altitude long enough, nitrogen stores would be depleted and the DCS probability would decrease. However, this is not the case. Altitude exposure itself decreases the denitrogenation rate. And more importantly, bubble formation and its pathological effects causing pain and incapacitation, usually occur before tissue nitrogen levels decrease to the point that bubbles can no longer form or grow. Most probably, this is because increased time at altitude permits growth of bubbles that are otherwise innocuous at a smaller size. A rapid descent after a decompression and particularly after experiencing any manifestation of DCS, is critical to minimizing the complications of DCS. In most cases, descent and landing as soon as possible eliminates the pain of simple limb bends.

***Rate of Ascent*** . A faster rate of ascent to altitude contributes to a greater incidence DCS. A slow ascent permits excess nitrogen to be transported in solution from the tissues to the lungs so that exceeding critical supersaturation is less likely. Rate of altitude ascent probably has been overemphasized in the past. Generally, a rapid decompression on the order of tens of seconds has

about the same incidence as a decompression taking a few minutes. If the decompression is slower (several hours), the risk decreases significantly.

**Exercise.** Exercise during altitude exposure increases DCS incidence. Several factors that may contribute to this effect include: *the sliding movement of one tissue against another*, such as joints and muscles, which cause a shearing action and encourage bubble formation, and *contracting skeletal muscles*, which may cause local areas of blood cavitation and turbulence, encouraging bubble formation. For this reason, persons exposed to high altitude should minimize exercise. Furthermore, if DCS does develop, any additional movement especially of the limb (in the case of bends) may worsen the condition.

Some studies have shown that submaximal exercise prior to or during the prebreathing period may decrease DCS incidence during the subsequent exposure. The suggested mechanism is that increased blood flow to the muscles accelerates nitrogen offloading. This remains unproven, however has been accepted and utilized in the high flight community.

The effect of exercise after return to ground level following an altitude exposure also remains unproven. Numerous anecdotal accounts, however, suggest that postflight exercise increases the probability of delayed DCS. Furthermore, postflight exercise-induced injury may be confused with or mask DCS pain. Finally, if asymptomatic bubbles caught in the lungs later pass through to the arterial circulation as exercise induced cardiac output increases, then very serious DCS symptoms may arise. For these reasons, persons exposed to high altitude, scuba and dry chamber diving, should be discouraged from strenuous exercise immediately after exposure.

**Body Fat.** Earlier studies have emphasized that an increased proportion of body fat increases the probability of DCS. This theory was based on the fact that nitrogen is about five times more soluble in fat than in water and therefore serves as a reservoir for the gas. Recent studies have not confirmed the correlation between body fat and DCS in persons who have met USAF weight standards, i.e., those persons not significantly different from average weight for a given height.

There is no scientific evidence to validate whether or not obesity increases the probability of diving DCS. However, when body fat, rather than body weight, is examined in relation to DCS, it is clear that obesity does increase the probability of DCS, especially after long, deep dives. The reason is that adipose tissue is slow to take up or eliminate inert gas because of its relatively low blood perfusion and increased inert gas solubility. In summary, the crewmember slightly above average in body fat content but who meets current weight standards is not at a measurable increased risk.

**Previous Injury.** There are numerous accounts of bends pain occurring preferentially in areas that have been previously injured. No objective data are available for analysis to support this theory. However, injury may cause blood perfusion changes or deposition of scar tissue. It is possible that these changes decrease nitrogen washout rates and predispose bubble formation in these areas. In addition, flight often involves extended periods in cramped conditions. Pain not

actually associated with bubble formation may mimic bends pain. Since diagnosis under these conditions is difficult, such pain must be carefully investigated when reported.

**Injury.** Recent local injury may predispose a diver to DCS at or near the site of the injury. Such DCS is manifested as localized pain. Although the mechanism which accounts for the predisposition is unclear, it has been suggested that changes in local perfusion, local inflammatory changes, and an increase in gas micronuclei formation in the injured tissue may be responsible for this effect.

**Age.** Before the age of about 40 years, no correlation between age and DCS incidence is clearly demonstrated. After 40 years, DCS incidence increases with increased age. This increase occurs in both compressed air workers and aviators, with a three-fold increase in incidence between the 19-25 year old and 40-45 year old age groups. The mechanism underlying this phenomenon is not understood but may result from changes in circulation due to aging. Factors contributing to this effect might be an increased deposition of fat within connective tissues, and changes in capillary density and permeability.

**Gender.** A great deal of controversy exists regarding the possible differences in susceptibility to DCS between men and women. There is some information which suggests females may be 3 to 4 times more prone to DCS than males exposed to similar altitude conditions. Women may also be at increased risk of DCS at the time of menses. The mechanism for this observation is unknown and definitive studies have yet to be published.

**Temperature.** No correlation exists between the frequency of DCS and the ambient air temperature in the range of 21.1° C, to 34.3° C. However, at an ambient temperature of -23.3° C, the incidence of DCS is twice that at 21.1° C, with a larger ratio of serious cases to mild cases. Temperature, then, may be an important factor in cold water diving activities (similar effects may exist if a diver is externally warmed), but is of little or no consequence to altitude-induced DCS.

**Denitrogenation .** Decreasing total body nitrogen before decompression reduces DCS incidence. Breathing 100% oxygen establishes a diffusion gradient to offload nitrogen from the blood to the lungs. In turn, this establishes a gradient to offload nitrogen from the tissues to the blood. Denitrogenation with oxygen, also called "prebreathing", is the current prophylaxis for DCS. The longer the time spent in prebreathing, the lower is the body's residual nitrogen stores, and the lower the probability of DCS. From empirical studies, it is apparent that some tissues are very slow in eliminating nitrogen. Although the total body reservoir of nitrogen may be one liter or less, and no nitrogen can be detected by mass spectrometer in the exhaled gas after 30 – 60 minutes of oxygen prebreathing, there is sufficient residual nitrogen in some of the slower tissue compartments to produce DCS given sufficient exposure.

Prebreathing 100% oxygen for 30 minutes prior to initiating ascent to altitude significantly reduces the incidence of DCS for short exposures (10-30 minutes) up to moderate altitudes (18,000 - 35,000 feet). This denitrogenation process eliminates nitrogen from the body tissues. This then keeps the supersaturation ratio below the critical threshold for bubble formation during subsequent altitude exposure because the value for  $P_{N_2}$  has been reduced.

***Prebreathing at Altitude*** . Breathing 100% oxygen during ascent or at altitude is just as effective in eliminating nitrogen as prebreathing at ground level for an equal time up to an altitude of 16,000 feet (Webb, et al, 2000). Additional studies are underway at the Armstrong Laboratory to determine safe oxygen prebreathing schedules at various altitudes, which will have operational significance, especially during wartime missions, when decreasing the time needed to prepare for missions may be extremely important.

***Break in Prebreathing Schedule*** . It was once thought that if a person breathed air during prebreathing, the break in prebreathing could be compensated by extending the prebreathing period by an equal amount of time. Recent studies have shown this assumption to be false. Breaks in denitrogenation usually occur when there is an equipment failure, the oxygen regulator is accidentally reset to the NORMAL (less than 100%) oxygen setting, the mask is removed, or the mask fit is unacceptable.

Since denitrogenation is an exponential process, a great amount of the total body stores of nitrogen is offloaded early in the pre-breathe period. The partial pressure of nitrogen decreases rapidly during the early part of prebreathing. If prebreathing is interrupted with even short periods of air breathing, the diffusion gradient strongly favors nitrogen diffusion back into the tissues. Since this onloading rate is faster than the offloading rate, an equal amount of time added to the prebreathe period does not return the tension of tissue nitrogen to a level equal to the tension had the prebreathe period not been interrupted.

Estimates have been made of the additional time that must be added to the total denitrogenation period to compensate for breaks in prebreathing. These formulas are not practical in the training arena. Although some new internal tables have been generated to compensate for a break in prebreathe (Zwart, 2001), they have not yet been experimentally validated. A good general rule to follow is, if the air breathing period exceeds a few breaths, it is best to restart from the beginning.

***Repeated Exposure*** . There is controversy concerning the effects of repeated exposure, that is, two or more altitude exposures in succession. It appears that exposures occurring in rapid succession within minutes or a few hours of the previous exposure increase the incidence of DCS during the subsequent exposure. This is presumably because some bubbles may remain from the previous exposure and bubble growth is now more likely. If the exposures occur on successive days, there is no increase in the incidence of DCS, but the time of first appearance of symptoms is decreased in the subsequent exposure. Although there is an increased risk from multiple exposures, when the risk of a single long altitude exposure is compared to the risk of several shorter exposures having intervening surface intervals but the same cumulative time at altitude, the single long exposure has a higher risk of DCS. Thus it is better to repressurize between high altitude air drops, than to leave the plane depressurized.

***Diving*** . Safe decompression limits vary with the time and the depth of the dive. A diver may exceed these safe limits either through missed decompression stops to the surface or through multiple ascents during a dive, thus producing or enlarging inert gas bubbles (typically nitrogen). If a diver re-exposes himself to increased partial pressures of inert gas within 12 hours (2.2 days

for some advanced decompression calculators – Zwart, Nobendem 1998) of a previous dive (i.e. a repetitive dive), the residual nitrogen remaining within the tissues must be accounted for or there will be an increased likelihood of DCS. Mild or insignificant cases may become worse.

***High Altitude Diving*** . Diving in mountain lakes and other bodies of water that exist at altitudes well above sea level creates special problems for the diver. Sea-level decompression tables become invalid as altitude increases. Several decompression methods for altitude diving have been proposed (Buhlman, Cross correction, Nobendem), but few have been extensively tested in human research protocols (Buhlman tables have reportedly undergone some testing). Special decompression schedules have been provided to the Peterson AFB hyperbaric facility because of its elevation above 6000 feet.

***Flying After Diving*** . SCUBA diving followed by altitude exposure vastly increases the incidence of DCS. Furthermore, the procedure decreases the minimum altitude at which DCS manifestations begin. This can be demonstrated even during flight on aircraft equipped with excellent pressurization systems such as commercial airlines, whose cabin altitudes are 1524 - 2438 meters (5,000 to 8,000 feet).

Persons who have been diving usually have elevated nitrogen stores, i.e., are supersaturated, after return to the surface. This elevated tissue nitrogen predisposes the diver to bubble formation if further decompression occurs. In addition, subclinical bubbles may form during a dive, fail to resolve on return to the surface, and expand while ascending to altitude. Some special military operations limit the maximum cabin altitude of flight to be determined by the maximum depth and time at depth. In practice, however, this is difficult to control. The USAF policy for all except special operations is to forbid flight within 24 hours of a compressed air exposure.

Flying after diving produces bubble formation by increasing the nitrogen supersaturation ratio beyond the critical threshold, even though decompression to sea level was uneventful. There are enough cases of DCS to substantiate this occurrence. Although various methods have been proposed (Nobendem method, PADI Repet Group "D", and others) absolute limits for the interval between diving and flying based on specific hyper/hypobaric conditions have not been experimentally confirmed. Therefore, the present conservative approach of the Air Force is to specify 24 hours between any dive and subsequent altitude exposure. Recent Navy experience with divers using saturation tables has shown that DCS can result 4 days after surfacing if the diver is exposed to altitude. Thus, for exceptional exposure dives, the 24-hour nonflying rule is too short. Until more conclusive research is accomplished in this area, the 24-hour restriction appears to be a practical solution for Air Force aviators.

***Individual Variability*** . There is great variability in individual susceptibility to DCS. Under the same conditions, some persons will develop DCS while others show no signs or symptoms. There is also day-to-day variability for each individual, but this is smaller than the variability seen between individuals. There may be a tendency for a person who develops DCS under given conditions to again develop DCS later under similar conditions. Individual susceptibility was the basis of the effort to screen "bends-prone" flyers during World War II.

However, since the factors predisposing DCS are extremely complex, a single case of bends in an individual is not indicative of consistently recurring problems during future exposures. Individuals who suffer bends are often generally affected in the same area upon subsequent exposure. This suggests that there are specific anatomic factors predisposing DCS in those areas.

**General Notes .** DCS is a normal response to an abnormal condition. If you subject an individual to conditions sufficient to produce DCS often enough, he or she will oblige you by becoming symptomatic. This has recently been recognized and formalized in an interim change to AFI 48-123 as follows:

A7.30.1.1. Any episode of decompression sickness (DCS) which produces residual symptoms after completion of all indicated treatment, or 2 weeks, whichever is shorter. All episodes of DCS require a minimum of 72 hours DNIF. Consultation with USAFSAM/FEH (Hyperbaric Medicine) and concurrence of MAJCOM/SG is required before RTFS in rated individuals. The local RAM (or flight surgeon plus MAJCOM/SG) may clear non-rated flight crew after consultation with USAFSAM/FEH. In cases of DCS with neurological manifestations, a normal examination by a neurologist is required before RTFS. DCS cases having persistent residual symptoms should be submitted for waiver at MAJCOM/SG level if not operationally significant; AFMOA/SGZA level if there is potential operational impact.

**Note:** The reasoning for removing the requirement for waiver for recurrent DCS is to not “force” a waiver upon someone for a normal response to an abnormal environment. As long as there are no residual symptoms, waiver is not required for single or recurrent DCS episodes. However any episode of DCS that results in persistent residual requires waiver (at the level stated above).

## **TREATMENT OF DCS (refer to website at <http://www.brooks.af.mil/web/hyper>)**

### **Initial Response to DCS Onset During Flight**

Decompression sickness continues to occur during altitude chamber training exposures at a rate of 2-3 per 1000. Prebreathing before ascent significantly lowers the incidence of DCS, but many hours of denitrogenation are required for total protection. The same is true for operational flying. Whether or not unpressurized operations are planned or occur accidentally, the development of DCS symptoms must not be taken lightly. All cases of DCS should be treated as an emergency. If a trainee or crewmember is suspected of developing DCS, the following procedures should be used:

1. ***Descend and use 100% oxygen from an aviators oxygen mask during the entire descent.*** The increased pressure on descent serves to compress existing bubbles. Oxygen serves to establish a diffusion gradient to further reduce the size of bubbles. Often the pain associated with DCS decreases or completely disappears on descent. Resolution of pain does not cancel the emergency.

2. ***Land (or return to ground level in a chamber) as soon as possible and remain on oxygen.*** Surface Level O<sub>2</sub> may be an adequate therapeutic option depending on symptoms.

3. ***Get qualified medical help .*** Contact the supervising Flight Surgeon immediately. Delayed DCS manifestations are possible after return to ground level whether or not

manifestations were present at altitude. In the case of altitude chamber induced DCS, students are no longer under the supervision of an aerospace physiologist after they depart the altitude chamber, thus, they should be instructed to be aware of the possibility of postflight symptoms.

### **Medical Management of DCS**

The following procedures should be adhered to in all suspected cases of DCS (Type I and Type II):

a. ***Place the patient in a supine position and immediately provide 100% oxygen by a tight fitting aviator's mask*** . The use of 100% oxygen enhances nitrogen elimination by increasing the diffusion gradient of nitrogen and relieves tissue hypoxia. Avoid using other hospital-type oxygen masks and nasal cannulas. Non-rebreathers are also inadequate.

b. ***Intravenous fluids (normal saline, Ringer's lactate) should be used to restore intravascular volume*** since hemoconcentration can occur with loss of volume from the vascular compartment. An infusion rate of 250 cc/hour is recommended for the first few hours. One should not be overly concerned with overhydration in a young, otherwise healthy aviator. The need for an IV depends on the severity of the DCS hit.

c. ***Obtain a complete history and perform a complete physical exam*** , including a detailed neurologic exam. Do not ignore seemingly minor symptoms. They can quickly become major. If multiple symptoms occur, treat for the most serious condition.

d. ***The USAF Hyperbaric Center physician on call at Brooks AFB, TX (DSN, daytime 240-3281, nights 240-3278) should be notified as quickly as possible*** concerning all known or suspected cases of DCS regardless of where they are to be treated or whether or not they are to be treated. This physician can provide valuable information and advice regarding treatment and transportation of the patient.

e. ***For diving related DCS:*** use TT-6 even if Sx have “greatly abated” with prior therapy. Recurrence rates for treatment with SLO2 or TT-5 are too high.

f. ***For altitude chamber related DCS:*** If joint pain only - absolutely no other Sx - and gets significantly better within 30 min on 100% surface level O2 (SLO2), fully resolved within 2 Hr on O2: recommend at least 1 hour SLO2 after 100% symptom free with a minimum of 2 hours of O2. May observe as outpatient. F/U next day to document stable resolution. Instruct no fly or dive X 3 days, maintain hydration, no sports or exercise or strenuous activity, avoid alcoholic beverages. Return PRN (or in 3 days if needs RTFS).

g. ***Continuing for altitude hits:*** if any residual symptoms after 2 hours SLO2 (sooner at discretion of treating physician - no improvement after 30 min SLO2, sooner if condition worsening), begin hyperbaric treatment. If simple joint pains only, may try TT-5. Complete resolution of all Sx MUST occur no later than 10 minutes into the first O2 period at 60 FSW. Even a “sensation” or “joint presence” requires moving to full TT-6. Most patients who are successfully treated on a TT-5 will report significant resolution of almost all their symptoms during the DESCENT to 60 FSW, even before O2 is applied (multiplace chamber).

h. ***For all DCS:*** ensure a complete neurological exam is conducted! Often, vague suspicions regarding clarity of the patients thought process can be easily confirmed if a solid baseline is established prior to treatment. Serial 3's or 7's; spelling 5 letter words forward and backward; Rhomberg and sharpened Rhomberg; Rinnes and Webers tests; olfactory sensation (use spices in capped pill-bottles); are quick and simple procedures that may reveal hidden

deficits! Neuro exam is doubly important if Pt reports any symptoms related to the CNS such as dizziness, gait disturbances, headache, feeling “fuzzy” or inability to concentrate.

f. ***If a hyperbaric chamber is on site and the patient has continuing symptoms, the patient should be moved to the chamber and immediately treated on the proper hyperbaric treatment table*** . No observation period is warranted at ground level. The effectiveness of treatment decreases as the length of time between the onset of symptoms and the treatment increases.

Aspirin is NO LONGER USED in the treatment of DCS.

Reasons: ASA administration has never been shown to help prevent or treat bubbles  
DCS may induce microhemorrhages in tissues. ASA slows clotting.  
Hard to determine if symptom decrease is due to HBO treatment table or ASA  
This may be a critical treatment decision factor if using TT-5  
Hyperbaric Oxygen is the only necessary and sufficient drug for DCS

Employment of 100% O<sub>2</sub> by tight fitting aviators mask during transportation to HBO chamber

For mild to moderate symptoms with no CNS or spinal neurological involvement:

Use cycles of 50 min of 100% O<sub>2</sub>, followed by a 10 min air break, instead of continuous O<sub>2</sub>

Reasons: Air breaks minimize likelihood of pulmonary and CNS O<sub>2</sub> toxicity  
Rehydrate & feed patient orally during air break  
May obviate need for IV rehydration  
Bathroom privileges during air breaks  
You ever wear a tight fitting oxygen mask for 3 hours straight?  
Patient can call spouse – “I’ll be home late tonight.”

Air breaks are NOT used during SLO<sub>2</sub> (Surface Oxygen) treatment protocols

For severe symptoms, chokes, shock, CNS, spinal, or worsening Sx despite O<sub>2</sub> therapy:

Use continuous O<sub>2</sub> therapy with IV rehydration (0.9% Saline or Ringers, NOT D5W)

In general, do not pass up an otherwise acceptable monoplace facility (with BIBS) in favor of a multiplace facility for treatment of severe DCS or AGE if the additional travel time to the multiplace exceeds 30 minutes. Less than 30 minutes difference, or mild to moderate symptoms, requires the sending physician to consider risks and benefits of treatment delay versus multiplace advantages.

Reasons:

There is no faster way to remove excess Nitrogen than 100% O<sub>2</sub> at 66 FSW (Dick Rutkowski)

Treatable severe injury may become permanent damage through treatment delay  
Monoplace facilities with BIBS (built in breathing system for air breaks) can do TT-6 and

TT-5

TT-6 early may well be better than TT-6a later (TT-6a is relatively dangerous)

Current Navy TT-6a (Diveman 4) starts with TT-6 and reassessment for deeper after 20 min O<sub>2</sub>

Bottom Line: If you pass up a monoplace facility, make sure you reasons are valid



*h. If bends pain is relieved while awaiting transport but symptoms recur, the patient should be transported and treated even if symptoms are relieved again after recurrence.*

*i. Any patient with symptoms or signs of neurological DCS, chokes, or neurocirculatory collapse (shock) should be immediately transported to the nearest chamber regardless of whether or not the symptoms persist.*

*j. Air transportation cabin pressure must be within 1000 feet of, or lower than, the point of origin, but no lower than the destination .* Aircraft used for movement must possess pressurization capability. (Any need to exceed cabin pressure more than 1000 feet higher than the point of origin must be discussed with a hyperbaric physician.) The use of a pressurized aircraft is ideal, but if a helicopter or other unpressurized aircraft must be used, a maximum flying altitude of 800 to 1000 feet above the point of origin should never be exceeded. If the patient is exposed to high altitude, bubbles in the tissues will expand and the medical condition will deteriorate rapidly. During ground transportation over a high altitude area, symptoms may also worsen. This must also be considered when transportation is planned. Oxygen should be administered by a tight fitting mask and used continuously during flight.

## **AEROMEDICAL DISPOSITION FOLLOWING DCS**

All individuals with Type I DCS (joint pain or skin manifestations) should not fly as a crewmember or passenger for 72 hours following complete resolution of symptoms. Deviations from this rule should be discussed with a hyperbaric physician prior to the flight. There is no requirement for a waiver, additional workup, or additional consultation, following prompt and full resolution of symptoms (see AFI 48-123 extract above). Crewmembers sometimes have the belief that three episodes of DCS will result in permanent grounding. This is not true. Individuals with resolved neurologic symptoms may return to flying following a normal neurologic exam by a neurologist, consultation with a hyperbaric physician, and MAJCOM. Even if symptoms persist, or are residual, waiver may be granted by MAJCOM if not functionally significant, or by AFMOA if functional but minor (may require SAM eval).

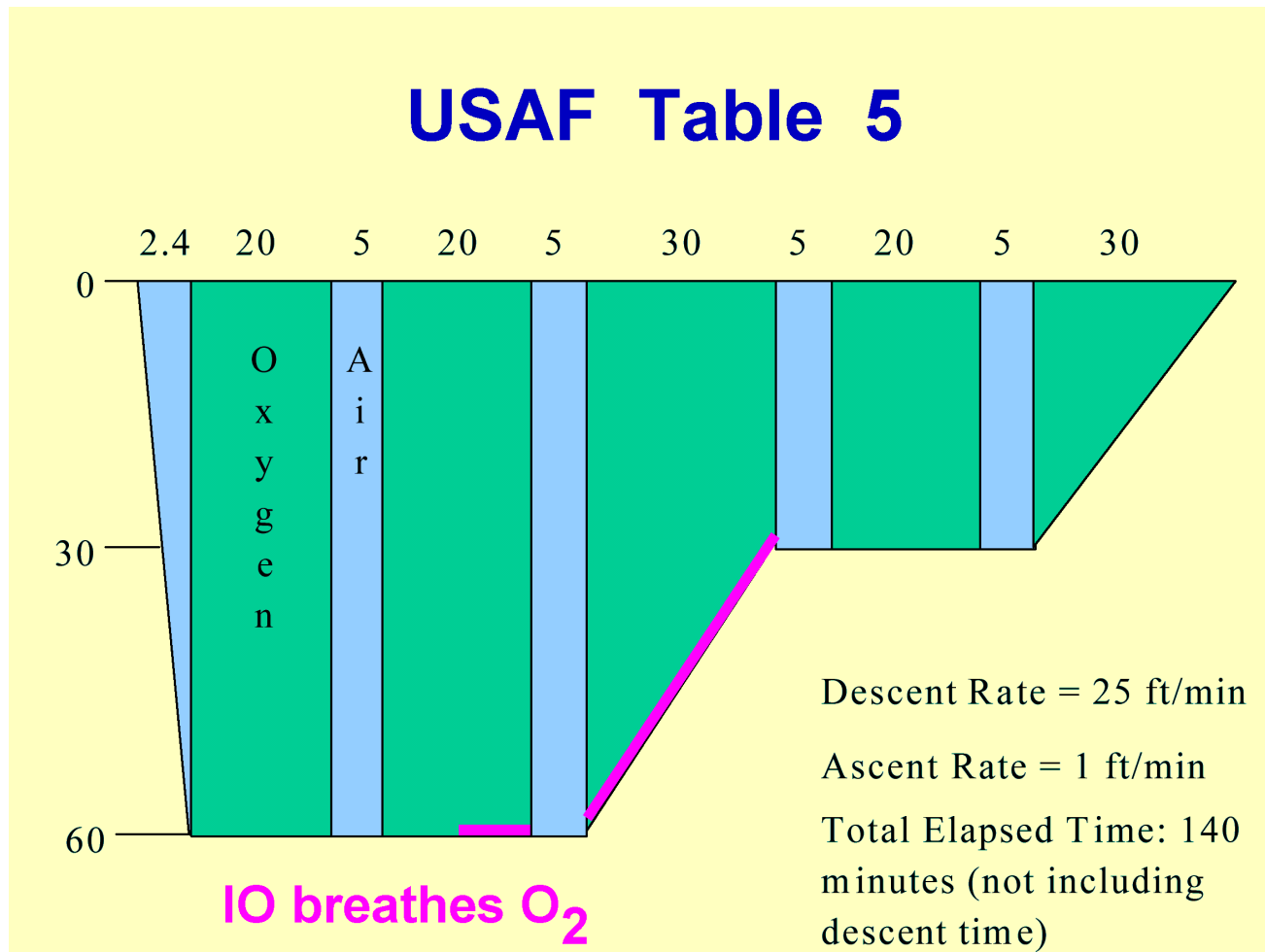
Crewmembers are often reluctant to report symptoms of DCS, resulting in progression of symptoms and increased risk of permanent injury. This is at least partly due to a fear of permanent grounding. Squadron flight surgeons have a duty to educate crewmembers regarding the etiology, signs and symptoms, treatment, and disposition of DCS.

## Surface Level O2 (SLO2)

- SLO2 is for Altitude Induced Bends Pain **Only**
  - Pain during, or within 2 hours of the exposure
  - Not for Paresthesias, Neuro, CNS, Chokes
- This Treatment is NOT Recommended for Diving Bends
  - May have recurrence rate of 30% in Diving DCS!
- 100% O2 by Tight Fitting Mask, Hood, or Reservoir System
  - Non-Rebreather is Inadequate
- Old Standard: Successful if Sx Resolved After 2 Hrs of O2
- Current Recommendation: O2 for **at least** 1 Hr after All Sx Gone
  - Minimum of 2 Hours of O2 (Even if Resolved on Descent)
  - Maximum of 3 Hours of O2
  - If Sx Worse or Recur- Begin TT5/TT6 Immediately
  - If No Improvement in 30-60 min, Consider TT5/TT6

## Table 5 Rules

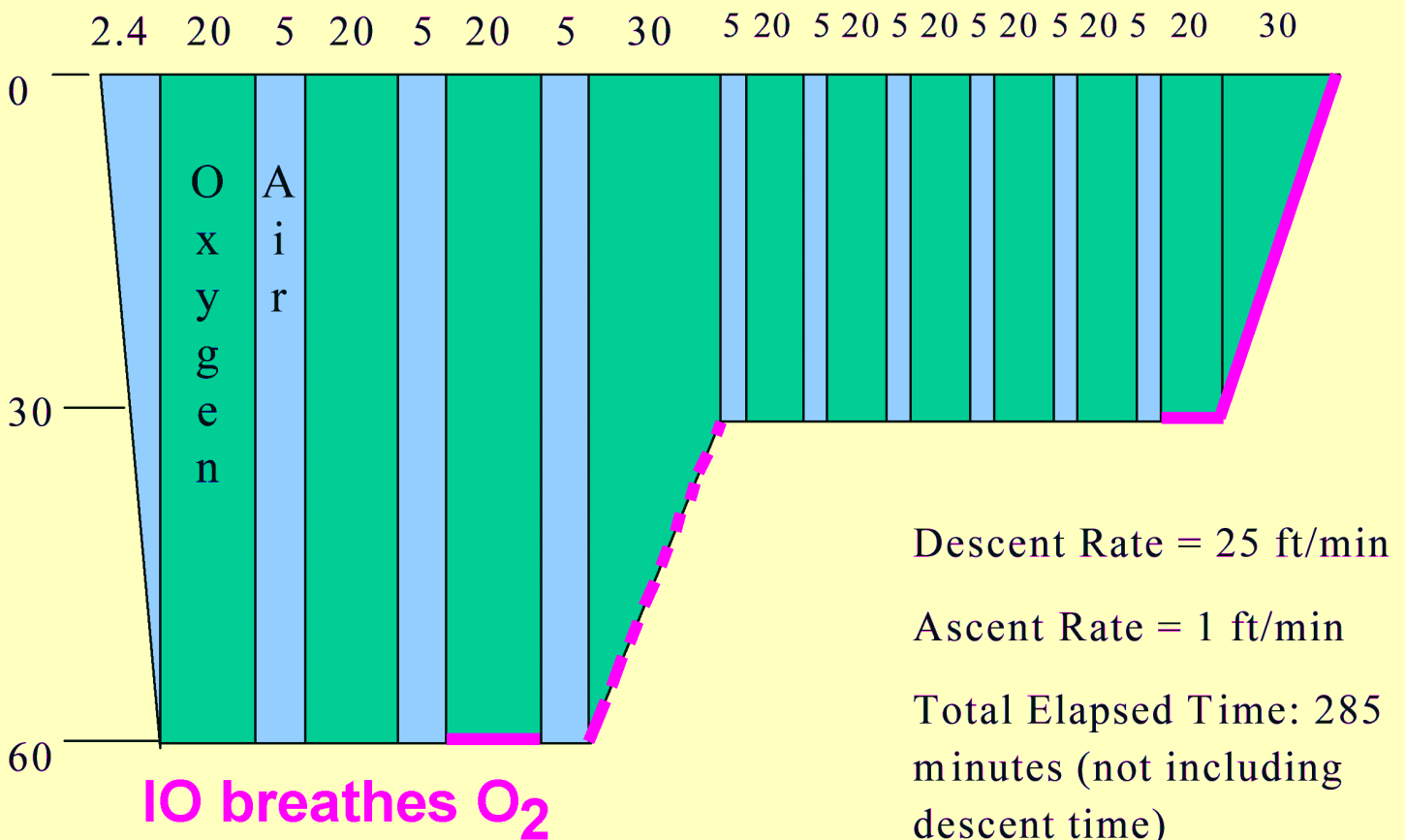
- TT-5 is for Bends Pain Only
  - Altitude Chamber Induced, Within 6 hours of exposure
  - Dive Induced, Within 2 Hours of Surfacing
- Do Not Use This Treatment For Any Other Disorder
  - Any Neurological Symptoms Require TT-6
- If Any Sensation Persists after 10 Minutes @ 60 FSW
  - Go To TT-6
- If Any Discomfort Recurs during TT-5, Go To TT-6
- Inside Observer Takes 10 min O<sub>2</sub> during 2nd O<sub>2</sub> period at 60 FSW, and 30 min during ascent to 30 FSW
- Monoplace Chambers Must Use BIBS for Air Breaks
- Note: Navy TT-5 Omits the Second Air Break at 60FSW and does the Descent on O<sub>2</sub>



## Table 6 Rules

- TT-6 is for Type I and II DCS
- This Table Is Mandatory For Bends Pain More Than 10 Minutes at 60 FSW; All Neurological DCS; Chokes; AGE
- May Extend To A Maximum of 5, 20 min Periods @ 60 FSW
- Minimum of 3, 20 min Periods at 60 FSW
- Extend to One O2 Period Completely ASx, or to Max
- May Extend Up To 2 Additional Hrs of O2 at 30 FSW
- Inside Observer Takes 20 min O2 during 3rd O2 period at 60 FSW, Last 20 min at 30 FSW, and 30 min during ascent to Surface. Any Ext @ 60 FSW - add 30 min during ascent to 30 FSW
- USAF May Use USAF (5/20) or USN (15/60) Schedule @ 30 FSW with a minimum of 2 hrs O2 at 30 FSW (air/O2 time)
- Monoplace Chambers Must Use BIBS for Air Breaks

## USAF Table 6



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