Diving is a science.
Russian roulette & Bungee Jumping are not!!!
Please take some time to understand the theory if you wish to partake in Technical Dives.

Saturation

A tissue that has absorbed nitrogen to its fullest extent and can hold no more in solution, at an ambient pressure, is said to be saturated.

Henry's Law states that the human body will dissolve nitrogen in proportion to the surrounding pressure. At the surface, the body is saturated, meaning it contains as much nitrogen as it can hold in solution at surface pressure. As pressure increases, such as when the diver descends, the body isn't saturated any longer because more nitrogen from the breathing gas can go into solution in the tissues. If a diver were to remain at a given depth long enough, eventually the gas going into solution in the divers body would reach equilibrium with the surrounding pressure. The body would be saturated again.

Super Saturation

A body tissue submerged in water will eventually be at equilibrium with its environment. It will be saturated to that depth. If the depth is reduced the gas pressure within the tissue will be higher than the surrounding (ambient) pressure and this reduction in pressure will cause the dissolved gas to diffuse out of the tissue. This higher than ambient pressure and consequent pressure gradient and diffusion is the condition of supersaturation.

Henry's Law tells us that gas dissolves into our tissues, from alveolar air and blood plasma, when the ambient pressure is increased. Upon descent more gas enters the tissues than leaves, producing a net increase. This continues until the tissues are at ambient pressure or the ascent is started.

J.S.Haldane observed that divers making dives shallower than 10m of sea water (2ata) could be brought directly to the surface (1ata) without contracting the bends. This observation implied tissues in equilibrium at 10msw could surface without incident. Surfacing from 10msw is a pressure reduction of 50%. From this, Haldane reasoned tissues could always withstand a pressure reduction of 50% to the surface. This is the famous "Haldane ratio" of 2:1 pressure reduction.

As long as the calculated pressure in any tissue was no more than twice the pressure at the next depth, the diver could be allowed to proceed to the next dive step. Haldane proposed a method of "stage" decompression, where the diver would ascend to the shallowest stop possible without exceeding the 2:1 ratio.

Therefore Haldane's decompression model is based upon ascending the diver to a depth where the tissues are supersaturated, waiting for the process of off gassing, via the pressure differential (diffusion gradient) to remove inert gas and then raising the diver again and again to another supersaturated depth until the diver reaches the surface. The balance between a case of bends and tolerable levels of supersturation have been the focus of diving table/computer research and development ever since

Half Times

In absorption (on gassing) or elimination (off gassing) the time it takes for a tissue to change from being fully saturated to halfway saturated and then halfway between half saturated and clear, and so on, is termed the half time.

Theoretically, saturation occurs exponentially, that is, it takes much less time for tissues to reach 50% saturation than to go from 50% saturation to saturated. Physiologists call the time required to reach 50% saturation in a theoretical tissue the tissue halftime, in that it takes one halftime for a theoretical tissue to absorb half the nitrogen remaining to reach saturation.

The rate tissues absorb nitrogen is not the same in all tissues, thus different tissues have different half times. Some tissues, such as blood, absorb rapidly and contain relatively little nitrogen before they saturate. These are (theoretically) fast tissues. Other tissues, like fat, absorb a lot of nitrogen and therefore have long half times. These are (theoretically) slow tissues. The amount of circulation a tissue receives also affects how quickly or slowly it absorbs nitrogen.

Compartments

These are a series of theoretical models used to represent the on/off gassing of nitrogen in the body for the purpose of calculating times for nitrogen absorption and the times for nitrogen elimination. These models or compartments are used to theoretically replicate different speeds of absorption and elimination to create algorithms used for dive tables, dive computers and desk top decompression software

J S Haldane used the original theory of nitrogen elimination to develop a decompression model which became the basis for the first dive tables published in 1907. In theory the model prevented decompression sickness by calculating nitrogen levels in the diver's body and controlling the critical ratio.

While it's accepted that different tissues have different characteristics with respect to nitrogen absorption and elimination, it's important to distinguish between the concept of "tissues" in decompression physiology and in decompression modeling. Some tissues absorb more nitrogen than others and therefore take longer to release nitrogen. To mathematically predict how various tissues throughout the body would absorb and release nitrogen, Haldane developed a model of theoretical "tissues," or theoretical "compartments" based on theoretical half times. While Haldane believed there was some relationship between the halftimes he assigned and the body, he didn't intend for any particular theoretical tissue to correspond to any particular body tissue. Rather, Haldane was simply trying to build a model that replicated the fact the body doesn't absorb and release nitrogen on a single time scale.

Haldane's original model had 5 compartments with halftimes ranging from 5-75 minutes. Contemporary models have calculated as many as 20 or 30 compartments with half times longer than 600 minutes.

M VALUE

The M in M-value stands for "Maximum." For a given ambient pressure, an M-value is defined as the maximum value of inert gas pressure (absolute) that a hypothetical "tissue" compartment can "tolerate" without presenting overt symptoms of DCS. M-values are representative limits for the tolerated gradient between inert gas pressure and ambient pressure in each compartment.

In formulating his tables, J.S.Haldane assigned the critical ratio 2:1 nitrogen pressure. In the late 1930s, the US Navy revised this relationship. US Navy research showed that different model compartments have different critical ratios; fast compartment could tolerate much more supersaturation than supposed by Haldane, and slow compartments a bit less. Later, research in the 1950s demonstrated that critical ratio not only varies with each tissue compartment, but may vary within the compartment itself depending on the depth.

In 1965, Dr Robert D. Workman, a Captain in the US Navy, developed the concept of the M value as an easier way of calculating maximum allowable supersaturation pressures for each compartment. Workman recognized that Haldane's original ratio of 2:1 (based on air) was really 1.58.1 if you considered only the partial pressure of the inert gas in air-nitrogen. The M-value expresses the tissue compartment's maximum allowable pressure in meters of sea water absolute. This streamlines the mathematical formulation of dive tables by defining the maximum amount of excess pressure in the compartment over ambient pressure by the need to calculate variable ratios. With the Navy's changes, one specific tissue, called the controlling tissue or more properly controlling compartment, sets the limits for a specific dive and dominates the schedules the dive computer or table recommends. The controlling compartment depends upon the depth; at deeper depths the fast compartments rapidly absorb nitrogen but at shallower depths the M-value of a fast compartment may be higher than the depth. In this case, the fast compartment can never control the dive, and the task passes to slower ones.

Pressure Gradients

The difference between the partial pressure of gases in contact with a liquid and the gas tension within the liquid is referred to as the pressure gradient.

When the pressure gradient is high, the rate of absorption of gas into the body will be great. As the number of molecules continues to dissolve, the gradient decreases, and the rate at which the molecules dissolve slows.

Eventually the gas tension within a liquid will reach equilibrium with the partial pressure of each gas in contact with the liquid, and no net exchange of gas will occur(although equal numbers of molecules will continue to pass in and out of the liquid). At this point the liquid is said to be saturated.

Absorption and Elimination

The processes by which gas in solution is transferred or absorbed into and out of the body tissues. The mechanism that governs this process is perfusion and diffusion based upon blood flow and differing pressures of dissolved gases inside and outside the tissue.

Not all tissues absorb gas at the same rate. This is because tissues vary in their degree of permeability (ability to let gas pass across the tissues). The absorption rate also depends on the blood circulation to various tissues, which may in turn be affected by variables such as temperature or exercise. In the body different gases have differing solubility in different tissues, and different tissues dissolve gases more readily than others. For example, given an equal quantity of blood tissue and fat tissue, when equilibrated to surface pressure the fat tissue holds more nitrogen molecules in solution, though both have the same tissue pressure. Therefore, some tissues can be described as fast, on and off gassing quickly, such as the brain and some tissues can be described as slow, on and off gassing slowly, such as the bone.

Inert gas nitrogen in inspired air is transported from the lungs around the body in solution via the blood vessels. Through the process of diffusion via a gradient the inert gas in solution passes into the tissues until the tissues are saturated to an ambient pressure. These are the phases of N2 absorption/on gassing. When the ambient pressure drops the pressure gradient is reversed and the N2 diffuses out of the tissues back into the blood. The venous system carries the blood back to the lungs via the heart. The pressure differential between the inspired air and the N2 in the pulmonary capillaries causes the diffusion of the N2 out of the blood and into the lungs for exhalation. These are the phases of N2 elimination/off gassing.

These processes are affected individually by personal physiology but generally by how deep we dive, how long we dive and how fast we ascend.

Isobaric Counter Diffusion

In the early to mid sixties commercial diving in the North Sea depended upon a lot of heliox bounce diving as opposed to saturation. The tables devised for this procedure called for a switch from heliox to air at eighty feet. At this switch some divers would show signs of loss of balance and extreme vertigo and vomiting. The symptoms where identical to those of someone suffering from an infection in the inner ear, specifically the semi-circular canals which are involved in control of balance. For many years it was thought that the bubbles causing the damage occurred in the vestibule part of the ear, hence the name given to that specific type of decompression sickness (vestibular bend). It is now thought that the bubbles do not form in this region but in the cerebellum of the brain. This part of the brain controls muscles and receives the impulses from the semi-circular canals of the ear, hence the symptoms displayed. Wherever the bubbles occur the result is the same, severe disability which can leave survivors quadriplegic. It is also thought that the symptoms shown are only the most noticeable and in fact the brain is suffering massive trauma with huge bubble formation. The divers in the North Sea where decompressing in a chamber and so did survive, anyone suffering such an occurrence in the water, especially on SCUBA, would be in afar worse situation.

Physiologically consider the course of events after a gas switch from a high percentage helium mix to a high percentage nitrogen mix during ascent, from the point of view of an interneuron (i.e. nerve cell), from which a large proportion of the cerebellum is composed. A high concentration of nitrogen is carried in solution, by bulk flow in the bloodstream. Rapid diffusion occurs via the aqueous medium into the extra cellular fluid surrounding the interneuron. A situation then exists whereby two aqueous compartments are separated by the cell membrane, one containing a high nitrogen concentration (extra cellular), and one containing a high helium concentration (intracellular). The mechanism by which equilibrium is reached is by passive diffusion through the membrane. (i.e. generated only by the concentration gradient) until the concentrations in both compartments equalize. The rate at which any molecule can pass through the cell membrane is given by its permeability constant. This will depend on factors such as the size of the molecule, its diffusion coefficient and its partition coefficient between the lipid and aqueous phases of the cell. So even though the helium atom moves three times as fast as the nitrogen molecule in terms of its diffusion rate, the nitrogen molecule can cross the cell membrane more rapidly as it has a higher permeability constant.

The flux of molecules across the cell membrane therefore results in more nitrogen molecules entering the cell cytoplasm than helium atoms leaving. Since the partial pressure of any component in a mixture of gases above a solution is directly proportional

to the number of molecules of that gas dissolved in the solution, it follows that as the number of molecules of nitrogen in the cytoplasm increases, then the partial pressure required to prevent them coming out of solution also increases. On ascent the pressure is decreasing so bubbles will form.

Carbon Monoxide and how it affects you in diving (smoking)

In diving, carbon monoxide poisoning generally originates from a contaminated air supply, where it may be unnoticed by a diver because carbon monoxide lacks both odor and taste.

Hemoglobin bonds with carbon monoxide more than 200 times more readily than with oxygen, but does not unbond as easily. Once carbon monoxide enters the bloodstream, it can take 8-12 hours for the circulatory system to eliminate it. In addition, carbon monoxide bonds with enzymes in the blood.

As a diver breathes air contaminated by carbon monoxide, blood hemoglobin reaching the alveoli bonds with the carbon monoxide, forming carboxyhemoglobin; this locks the hemoglobin molecule, making it incapable of carrying oxygen. The strong bond between the carbon monoxide and hemoglobin keeps carbon monoxide bonded as the blood circulates through the tissues, unlike oxygen. As the diver continues to inhale carbon monoxide, more and more hemoglobin bonds with it, so as circulation continues, fewer and fewer uncontaminated red blood cells are available to carry oxygen. Unchecked, this causes hypoxia despite continuing circulation and respiration because the blood can no longer supply oxygen to the tissues.

At depth, this condition can be further complicated because increased pressure dissolves oxygen into the blood plasma. Although at the surface blood plasma does not carry sufficient dissolved oxygen to support the body's tissues, higher oxygen partial pressure while breathing at depth greatly increases oxygen dissolved in the plasma. This action helps meet the tissue oxygen requirements and delays the onset of symptoms warning the diver. When carbon monoxide poisoning symptoms do occur – headache, confusion, narrowed vision – the diver ascends, blacking out from hypoxia at shallow depths because there is no longer sufficient pressure to dissolve adequate oxygen into the plasma.

Carbon monoxide poisoning may cause a victim's lips and nail beds to turn bright red. Hemoglobin bonded with oxygen appears red, and hemoglobin bonded with carbon monoxide appears even redder than usual. Contaminated blood is highly visible as it flows through capillaries of the lips and nails, which are close to the surface of the skin.

Although carbon monoxide rarely contaminates a diver's air supply, it should be noted that smoking is another source. Physiologists have found that smoking raises normal carbon monoxide levels in the blood 3-12 times, which impairs oxygen transport and carbon-dioxide elimination. Circulation increases so uncontaminated red blood cells can meet tissue gas exchange requirements, raising blood pressure and heart rate. This is why smoking stimulates the heart. It takes 10-12 hours for gas exchange to return to normal after smoking.

Blood Circulation – Path

The heart, arteries, veins and capillaries make up the cardiovascular system. Basically, the heart moves the blood, which travels through the arteries to tissues throughout the body. Arteries branch into the tiny capillaries, in which the blood and tissues exchange gases and material. The capillaries lead to the veins, which return the blood to respiratory system and heart to begin again.

The human heart is essentially a four chambered organic pump composed of muscle tissue and divided longitudinally. The upper chambers on each side called atria receive blood coming into the heart and pump it into ventricles, the chambers below. The ventricles pump blood away from the heart.

Oxygen rich blood coming from the respiratory system enters the left side of the heart (left heart), which pumps the blood into the aorta, the largest artery in the body. Because the left side of the heart supplies the entire body with blood, it is larger and stronger than the right side (right heart).

The carotid arteries branch off the aorta almost immediately and supply blood to the brain. Tracing the arterial system away from the heart, the arteries continue to branch into smaller arteries until they reach the capillaries, the actual site of gas and material transfer with the tissues. Capillaries are microscopic, with diameters so small that blood cells pass through single file and walls so thin that gases and materials diffuse readily through them to and from other body tissues.

Capillaries lead through the tissues they supply and then branch together into the veins, which branch together returning oxygen poor blood to the right side of the heart. The right side of the heart pumps blood into the pulmonary arteries, which lead to the pulmonary capillaries inside the lungs. The oxygen bonding with the hemoglobin and carbon dioxide release from the blood actually occurs in the pulmonary capillaries. Oxygen rich blood leaving the pulmonary capillaries flows into the pulmonary veins and back to the left atrium to begin another cycle.

Gas embolism AGE Barotraumas

A condition in which gas bubbles enter the arterial system and cause damage by blocking blood flow to vital organs, most commonly the brain. This is generally caused by air passing through the walls of the alveoli into the bloodstream.

The most serious injury from lung over pressurization develops if air enters the bloodstream through ruptured alveoli into the pulmonary capillaries, causing an air embolism or Arterial Gas Embolism (AGE). An embolism is any foreign body in the blood stream that can block its flow; an air embolism is such a body composed of air (i.e. a gas bubble), and an AGE is a bubble on the arterial side of the circulatory system.

Air entering the bloodstream in the lungs flows through the pulmonary vein to the heart, through the left side of the heart into the aorta and the arterial system. The air bubbles can lodge virtually anywhere in the body's circulatory system, and can – in a manner similar to Type II decompression sickness – cause severe damage by stopping blood flow to tissue.

The first main arterial branches off the aorta are found in the aortic arch above the heart. These include the carotid arteries, which supply the brain. If bubbles travel to the carotids, which is likely, they will go to the brain and cause cerebral arterial gas embolism (CAGE).

In much the same manner as with cerebral decompression sickness, the bubbles dent the brain tissue oxygenated blood, which causes a stroke. The symptoms include dizziness, confusion, shock, personality change, paralysis, unconsciousness and death. Compared to decompression sickness, the effects of cerebral air embolism and other lung expansion injuries tend to be rapid and dramatic. Decompression sickness tends to be somewhat delayed.

If an air embolism victim should be fortunate enough to have bubbles miss the carotid arteries, the emboli can still cause damage and symptoms in other areas in the body. If bubbles were to block the coronary artery, for example, the restricted blood flow could result in heart attack.

Type I – Type II DCS

The diversity of DCS symptoms makes diagnosis complex. Yet despite the different symptoms, manifestations of decompression sickness tend to share some characteristics. DCS tends to be delayed after the dive, and may take as long as 36 hours to manifest. About half of all DCS cases appear within an hour after the dive and about half take longer. Also, DCS tends to get worse with time until treated.

Physiologists often group decompression sickness into Type I – skin and pain only symptoms or Type II – with more significant, sometimes life threatening symptoms. Generally, cutaneous (skin) and joint pain DCS are regarded as Type I, with all others regarded as Type II. Both may be possible simultaneously, depending on where bubbles form or accumulate in the body.

Cutaneous DCS: bubbles coming out of solution in skin capillaries can cause cutaneous DCS, which may be characterized by a red rash in patches, usually on the shoulders and upper chest. Although cutaneous DCS is not considered serious by itself, its presence indicates decompression problems and the possibility of more serious symptoms.

Joint and limb pain DCS: joint or limb pain occurs in approximately 75% of DCS cases. Physiologists believe bubbles growing around or within the tendons, ligaments and related muscles is the immediate cause. Symptoms may be found in more than one place on the same limb, such as the shoulder and elbow; bisymmetrical symptoms are unusual. Like cutaneous DCS limb pain DCS is considered serious primarily because it may indicate significant decompression problems.

Neurological DCS: effects on the nervous system produce some of the most serious cases of DCS. Because the nervous system reaches throughout the body, neurological DCS can affect movement or touch, and life support functions like breathing and heartbeat.

Besides bubbles growing within nervous tissue and in some cases bubbles in the venous system may block blood outflow, backing up the system and reducing the arterial flow to the affected area. Spinal cord related DCS may be related to this type of blood flow restriction. Neurological DCS involves the spinal cord most frequently, commonly causing numbness and paralysis in the lower extremities that creep upward. In a relatively short time, victims may become paralyzed from the neck down.

Bubbles can also travel to the brain (Cerebral DCS) causing stroke as they block blood flow. These symptoms may be very similar to those caused by AGE and include blurred vision, headache, confusion, unconsciousness and death. Symptoms depend upon where bubbles end up in the brain; the similarity between cerebral DCS and AGE makes it probable that bubbles pass through the lungs and enter the carotid arteries supplying the brain.

Pulmonary DCS: DCS manifesting itself in lung capillaries signals the possible onset of life threatening symptoms. Silent bubbles and micro bubbles reaching the pulmonary capillaries normally diffuse into the alveoli, or in rare cases, if present in large quantities, may travel into the arterial system and cause neurological DCS. If bubbles accumulate faster than they diffuse or travel through the pulmonary capillaries, they block and back up blood flow to the lungs. With less blood moving through the lungs, the left side of the heart receives less blood causing the heart rate to rise and the blood pressure to drop. Assuming the circulatory system continues to function two possibilities exists. Either blood begins traveling around the blockage through unobstructed pulmonary capillaries and thus maintains circulation until the bubbles diffuse into the alveoli, or the bubbles continue to accumulate faster than they can diffuse, interfering with gas exchange in the lungs. This simultaneously reduces oxygen to the tissues and nitrogen elimination. Pulmonary DCS creates breathing pain, commonly associated with a short irritated cough. The victim often feels air-starved, which has given pulmonary DCS the nickname "the chokes." Symptoms tend to progress rapidly and may lead to shock.

Deep Stops/Micro Bubbles

A deep stop is an intermediate stop halfway between the maximum bottom depth and the first required decompression stop. The DSAT deep stop recommendation time is 2 minutes.

Haldanian decompression models focus on the inert gas, nitrogen that has dissolved into body tissues at pressure. The dissolved gas model controls the rate of release based upon a series of tissue models with individual half times that prevent bubble formation concentrating on the risk of DCS from slow tissues. The theory behind deep stops rests upon the importance of fast tissues (well perfused) and the presence of micro bubbles.

Fast Tissues: The Haldanian model controls DCS by getting the diver into shallower water to release inert gas from the slow tissues. However, moving to shallower water on a continuous ascent ignores the effect of falling ambient pressure on the fast tissues. The fast tissues react to this decrease in pressure by off gassing. However, if the ambient pressure falls too quickly the M values (tissue critical tensions) of the fast tissues will be exceeded, and excessive supersaturation creates a risk of bubble formation causing DCS.

Gas Seeds: In 1951 Bateman and Behnke theorized that small micro bubbles were also forming on many dives. In the 1960s Doppler technology confirmed this theory. Micro bubbles from around gas seeds, created by turbulence/or components within the blood, and these bubbles must be controlled to reduce the risk of DCS. Even if these bubbles do not directly cause DCS they can slow down off gassing because the inert gas becomes locked inside the bubble until it can diffuse back into the tissue. As the diver ascends the dissolved gas in the tissues can also dissolve into the gas bubbles increasing their size and therefore obstructive quality. In dissolved gas mechanics as the diver ascends the speed of release increases. However, bubble release actually slows. Therefore, if you stop briefly to decompress deeper these bubbles, kept small by the pressure at the deep stop, will have a chance to collapse as the gas in them is redissolved.

Consequently a deep stop is an opportunity to reduce the DCS dangers from the off gassing of fast tissues and the creation and elimination of micro bubbles.

Perfusion and Diffusion

Perfusion refers to the rate of gas delivery via blood, while diffusion refers to gas penetration across tissue-blood boundaries.

N2 uptake is possible by the processes of perfusion and diffusion. Good blood flow/highly perfused tissues are neurological tissues such as the spinal cord and the brain. Low blood flow/low perfused tissues are bone tissue such as the joints. The terms fast and slow tissues are determined partly by the blood flow to that tissue and partly by the tissues permeability. (In the field temperature and hydration also need to be controlled to allow the perfusion process to continue as efficiently as possible).

Diffusion occurs between the blood and the tissue based upon a gradient of pressure. Upon descent the increase in ambient pressure creates a pressure differential between the gas absorbed in the tissue at a relatively low PPN2 and the gas, from the lungs, recently dissolved into the blood plasma at a higher PPN2. Therefore the N2 diffuses from the blood plasma into the tissue. On ascent the opposite occurs when the ambient pressure drops and the pressure differential is reversed. N2 diffuses out of the tissue into the blood plasma. (In the field ascent rates also need to be controlled to allow the diffusion from the faster tissues to occur within safe limits).

Dissolved Gas Model Theory and examples

"The decompression model that concerns the diffusion, retention and elimination of dissolved inert gas in the body tissues (slow tissues). This model refers to safe maximum values for reducing the ambient pressure based upon the avoidance of excessive supersaturation."

Dissolved gas models limit degrees of tissue saturation assuming that gas exchange is controlled by circulatory rate of delivery (perfusion) or gaseous diffusion between blood and tissue. The exchange of inert gas is driven by the local gradient which is the pressure differential between dissolved gas in the arterial blood and local tissue tension (dissolved gas pressure within the tissues). Upon ascent this model takes the diver from depth to as close to the surface as possible within M value constraints. Any bubbles are now at their largest permissible cumulative volume and bubble (free phase) size for that depth or M value. The dissolved gas model treats larger volumes and bubble size with pressure in the shallows rather than increasing free phase (bubble) elimination at depth.

The Haldanian dissolved gas model controlled bubble growth using a ratio of tissue critical pressure to ambient pressure. In 1937 the US Navy using this model incorporated M values for theoretical tissues and increased the number of tissue compartments from 5 – 6. Later Professor A.A Buhlmann increased these compartments to 16 and changed the assumption of equal rates of on and off gassing (EE) to an assumed slower rate for the off gassing phase based upon linear calculations (EL). Numerous recreational dive tables utilize this dissolved gas model including Padi as do numerous dive computers such as Uwatec.

Freephase gas Model Theory and examples

The decompression model that is concerned with the formation, consequence and safe elimination of gas bubbles that occur primarily within the blood as a result of gas micronuclei, tissue supersaturation and ascent speed.

Gas micronuclei, known as bubble seeds, that are a third of the size of red blood cells act as a trap for inert gas nitrogen. The absorption and release occur at differing rates based upon the characteristics of the surfactant (bubble film surface) on each gas micronuclei.

As the pressure decreases the dissolved gas at a higher partial pressure begins to diffuse in to the bubble seeds that then increases the internal bubble pressure and causes the bubble to grow. These bubbles need to be controlled and eliminated and a direct ascent will only increase their size and threat. The Freephase models seek to keep a diver at depth to crush the bubbles and squeeze out gas via diffusion across the surfactant. Therefore free phase models seek to control not only the dissolved gas in the tissues but also the bubbles within the blood. This done utilizing deep stops (see Deep Stops) and a diffusion rate for inert gas from the micronuclei and slower ascents to limit micro bubble formation and evolution.

The origins of the free phase models begin in the commercial diving world and the practices followed by pearling divers in Australian and diving fishermen in Hawaii. In the 1980s decompression bubble models where presented by David Yount (Varying Permeability model) and Tom Kunkle (Surfactant Stabilized Model). Brian Hills 1977 book Decompression Sickness explored these concepts further. Currently the most high profile developments to Freephase modeling are associated with Bruce Weinke and the RGBM (Reduced Gradient Bubble Model). Examples of the Freephase model exist in decompression software such as V Planner and the RGBM version in products such as Suunto dive computers and Abyss decompression software. The theory states that:

DESCRIPTION OF INERT GAS ABSORPTION INTO THE BODY THEORY AND PATH

Nitrogen uptake occurs in the lungs. As the ambient pressure increases a consequent pressure gradient occurs that causes N2, via the 300 million alveoli, to diffuse across the cell membrane into your pulmonary capillaries. The N2 in solution is transported via the pulmonary veins to the heart around the body via the aorta. As the PPN2 is of blood nitrogen is higher than cell nitrogen the gas diffuses into the tissues of the body as a process of equilibrium. As the ambient pressure decreases the PPN2 of tissue nitrogen is higher than blood nitrogen and the gas diffuses out of the tissues into the blood. The blood then returns to the lungs via the heart and the pulmonary capillaries for gas exchange across the alveoli.

The body consists of various types of tissue. The rate at which an inert gas is absorbed (on gassed) by each tissue during hyperbaric exposure, and subsequently released (off-gassed) during decompression, depends on several factors. These include the blood perfusion in the tissue and the solubility of the gas in each particular tissue type. A simplified description of tissues is that they can be fast or slow at absorbing and releasing inert gas. The table below gives examples of the 'speed' at which this process can occur for several tissue types exposed to both nitrogen and helium - the two most commonly used inert gasses in diving.

Tissue	Half-time, Nitrogen (mins)	Half-time, Helium (mins)
Spinal Cord	12.5	12.5
Skin, Muscle	37 - 79	14 - 30
Inner Ear	146 - 238	55 - 90
Joints, Bones	304 - 635	115 - 240

Edmonds, Lowry and Pennefather (1991)

This differing rate of absorption depends on the fat content of the tissue and the blood supply to it. Therefore, tissues with a high blood supply and low fat content (e.g. the brain, heart, muscles etc.), will saturate very quickly, whereas those with a high fat content and poor blood flow (e.g. cartilage, tendon etc.) will saturate slowly. N2 becomes more soluble as temperature decreases and exertive activity, which increases the respiratory rate, delivers extra N2 to the body tissues.