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NAUI #2710

NOTES
NAUI HIGH ALTITUDE DIVING CONFERENCE

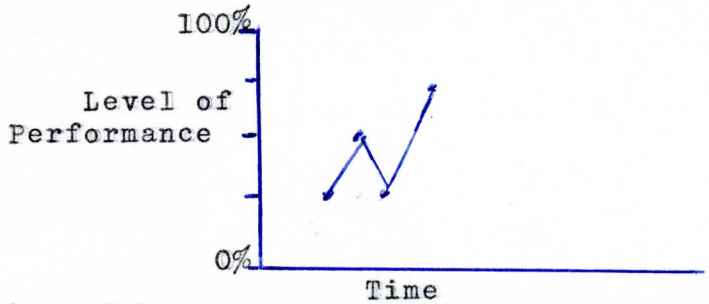
BOB TOLAR, CONFERENCE SPONSOR:

The objectives of this conference are: 1. To understand the nature of decompression sickness, 2. To understand the causes of problems associated with diving at altitude, and 3. To arrive at a concensus of how to teach high altitude diving or objectives for the second conference on high altitude diving (this being the first).

Dr. Al Behnke, PHYSIOLOGY OF DECOMPRESSION SICKNESS

In an aside at the beginning of his talk Dr. Behnke spoke on smoking, saying it was a prime problem in submarines as the particle size of smoke is small enough to form an aerosol with water which absorbs anything toxic and becomes a perfect vehicle to get these substances into the lungs of others. Also, withdrawal from smoking (nicotine) leads to decreased creativity for approximately one year, but this can be overcome by using nicotine in chewing gum, which increases productivity and reduces withdrawal symptoms after quitting smoking. This allows a gradual withdrawal from nicotine after quitting smoking altogether.

The new (1974) Navy Diving Manual has a lot of new material, hopefully including the basis for their derevation of the dive tables. Speaking of dive tables, a person cannot extrapolate with the tables without running risks, but they can be interpulated. Also, the imperical experience of trained indivuals is invaluable, although measurements are difficult. Feelings are important, and the responces can be graded. Individuals can express it accurately within 25% (trained individuals, that is). Individuals can mentally



do incredibly accurate performances. Baseball players, for instance, can catch a fly ball by instantly taking into account many factors, such as arc, speed of the ball, wind, etc., which would make catching it impossible if they had to calculate all the factors. Therefore, imperical evidence is very important, and after dive tables are calculated, they are modified, largely because of imperical evidence.

Pressure per' se:

To build up pressure in a vessel, the gas pressure or number of molecules must be increased which leads to an increased density. Animals are now tested using fluid breathing.

The pressure buildup can cause direct problems to enclosed air spaces in the diver; the ears, sinouses and lungs are subject to squeezes which can cause trauma. The ear can be subject to incredible trauma without too much damage. If problems occure, and there's no infection, resolution (of fluid) is usually spontanious. Moral, stay away from otologists. A serious problem associated with ear injuries is vertigo. Alternobaric vertigo results when the round window in the ear ruptures and fluid is lost from the cloclia. The result is permanent vertigo (not dizziness) which can be classified into two types: 1. when the world rotates around the individual and 2. when the individual seems to rotate (the world around him seems stable). The second type can be diagnosed by jerky eye movements. Alternobaric vertigo is a permanent disability which will not resolve itself. However, Otologists can now repair the round window and restore the individual to normal.

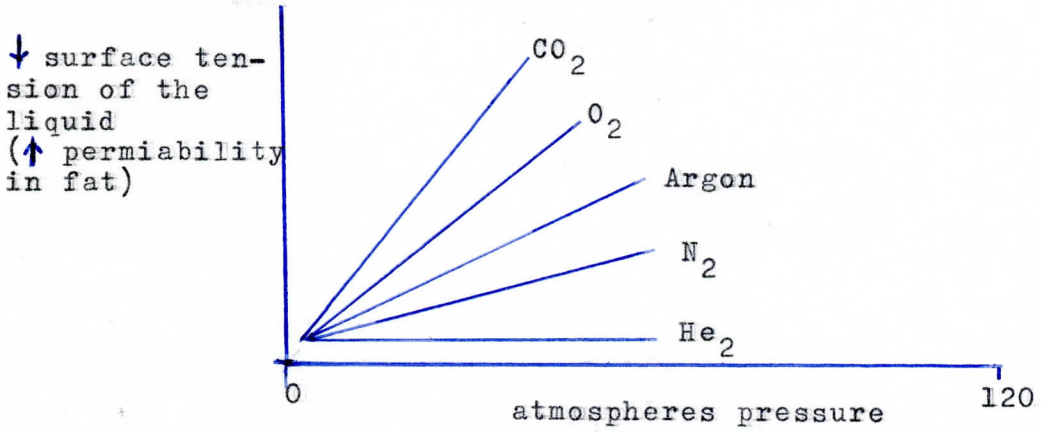
Pressure itself can cause problems. Trained individuals can go to 600 feet (and have) in 30 sec, and ascend to surface pressure in 30 sec and be safe. They can accomodate both to pressure and nitrogen for this short time. However, at around 300-400 feet divers experience arthralgia and complain of pain in their joints, "dry" joints, and crackling sounds from the joints. This is a result of fluid displacement from the joint capsules as the gases move in. Descents are now made very slowly (4 feet/hour) to accomodate the gas movement into the joint and maintain fliud in the capsules.

The gases which play a role in deep diving are Nitrogen, oxygen, CO_2 and helium. High Pressure Nervous Syndrom (HPNS) was at first thought to be an effect of high pressure helium. Now it, and Microsleep are known to not be a gas effect, or a narcotic effect, but an effect of pressure itself, and acts as an adaptive mechanism to the increasing pressure. There has been useful work done at 1600 feet, especially after a slow descent. No symptoms of HPNS were observed until after ascending from 1600 to around 800-900 feet. ~~HPNS~~ Narcotic effects can be observed in the chambers using a steadiness test and in the laboratory using a two plane rotation platform. Several steadiness tests can be used under field conditions. The Brownberg test requires the subject to stand feet together with shoes and balance himself for 60 sec with his eyes closed. The Sharpmen/Romberg test requires standing in the same manner with the feet tandom. Another requires the subject to stand with eyes closed for thirty seconds on one foot, and nan be used to see if one or the other leg is affected by a narcotic drug, gas, etc. Narcotics affect the balance center.

Nitrogen Narcosis is the only narcotic whose effects are completely reversable. Alcohol has a parallel affect. Narcotics can be used to separate stable individuals from those who are emotionally unstable. An unstable individual is completely distracted while under the effects of a narcotic. In one case a diver who was deep on air and had at one time been a football player (quarterback) began calling plays and completely ignored his underwater work. Stable individuals are slowed down, and must think each individual process out (pick up the wrench, place it on the nut, etc.) but can get the job done. One peculurality: with the wrench, nut business, the diver inevitably turns the wrench the wrong way; reason unknown.

The narcotic effect of nitrogen is related to its soluability in fat and fatty tissues. The Meyer/Oberson Law states that all substances which are soluable in fat are narcotic, and that the degree to which it is soluable indicates the degree to which is is also narcotic. An exception is helium. Based on fat soluability helium is predicted to become narcotic at 1000 feet, but this doesn't occure. It does cause tremors in animals at the 1000 foot level, however, and HPNS can be prevented using small amounts of air (nitrogen) in the breathing mixture.

Gases can be scaled on their narcotic potancy, using in vetro diffusion studies, and are shown below:

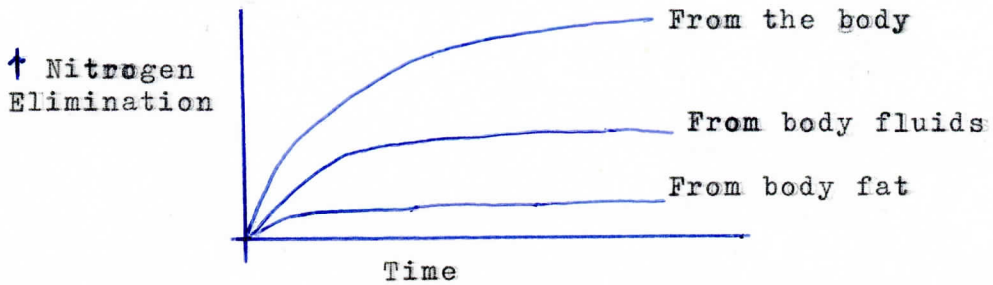


Oxygen also produces seisures, and this effect is felt before its narcotic effect. In a few individuals, O_2 doesn't produce the seisures, but the individuals go to sleep or loose consciousness. This is known as the Rare Bird Syndrom.

The effects of narcotic substances are additive. Two substances combined will produce twice the effect. This is not a problem with nitrogen narcosis while diving at altitude however. Since the pressrue is actually less, diving on air at altitude won't increase the likelyhood of nitrogen narcosis.

The half-life of nitrogen in the tissues varies with the tissue-type. It's approximately 15 minutes in the circulatory system and from 90-120 minutes in fat tissue. This becomes important in figuring dive tables for deep dives. Individual divers can also be classified by the amount of body fat they have:

1 unit individual--10% body fat
2 " " --20% " "
3 " " --30% " "



Use of oxygen and decompression sickness:

The use of oxygen in treatment of decompression sickness in the chamber virtually assures that the person suffering from the "bends" will suffer no residual effects. Data from about 120 cases of decompression sickness involving tunnel workers, seven with bad neurological symptoms, in which O_2 was used in the final stages of recompression for 5 hours elapsed time proves this out. The problems encountered with sport scuba divers are that chambers are not available and there's a deplorable length of time between symptoms' appearance and treatment.

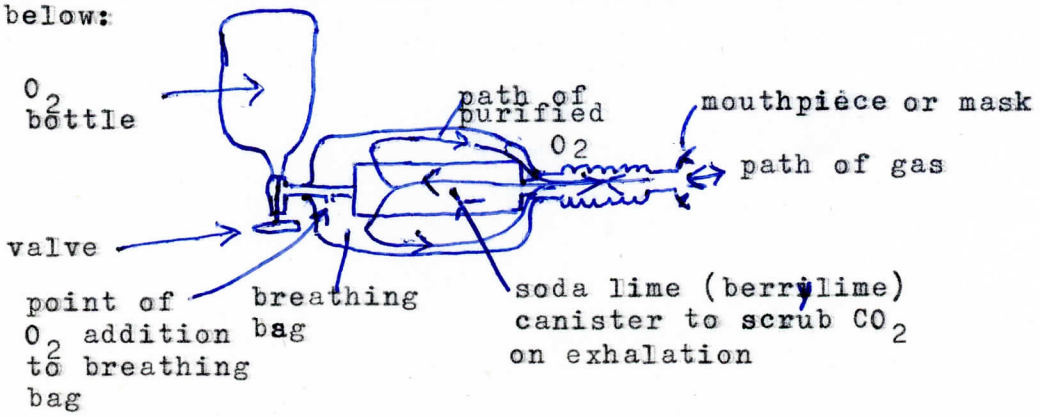
Question: Can O_2 be used effectively in the water during decompression?

Answer: Yes, the use of oxygen during decompression would be a good preventative measure. It has also been used in the chamber when symptoms of decompression sickness were experienced at a depth equivalent of 60-70 feet. Administration of O_2 for fifteen minutes at thirty feet eliminated the symptoms.

Question: Can O_2 administration on the surface be an effective emergency measure in the treatment of decompression sickness?

Answer: Again yes. Breathing oxygen at the surface up to 3-5 hours should be considered essential in the treatment of decompression sickness cases while traveling to the chamber. Breathing oxygen, either during decompression or at the surface, increases the <u>"oxygen window"</u> and allows more nitrogen to be eliminated from the body through the lungs. It should be remembered that while a person is on oxygen, breathing as such is only necessary to eliminate CO_2, providing 100% O_2 is used. Another thing, <u>prebreathing oxygen</u> can be considered a good preventative measure before making decompression dives as it eliminates nitrogen in the body at 1 atmosphere. It therefore takes longer for nitrogen to attain saturation at depth and, if normal air decompression tables are used, provides a good safety factor.

Getting back to surface treatment of decompression sickness by administering O_2, Dr. Behnke later stated that he considered <u>oxygen availability a necessesity for all diving activities</u>, including sport scuba diving. He had been working on a simple rebreather system which will allow a small container of oxygen to last a long time on the surface. Such a system would greatly increase the safety of deeper diving and diving at altitude where chambers are not available. As described by him, a drawing of the apparatus is below:



Recent studies have shown that after many dives there are "silent" bubbles, or bubbles which are small and don't cause symptoms. It takes many bubbles to cause symptoms, and one of the first signs is fatigue. After deep dives (some 3-4 hours afterwards) in the chamber they've had divers say they felt tired and then sleep for 11-12 hours. This is apparently especially evident on repeditive dives.

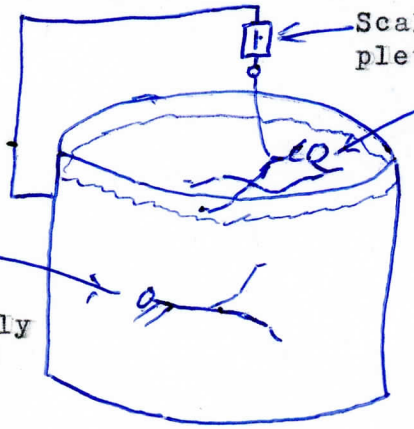
There are two degrees of decompression sickness, Type I, where the classic symptoms are noted and Type II, which also involves neurological symptoms. (Someone asked if there was a Type III, and Dr. Behnke stated "we don't talk of that one."). Eighty percent of the decompression sickness cases are Type I. In both cases all symptoms can be eliminated if treatment is given quickly. It is also necessary to check for neurological symptoms prior to releasing a case from the chamber as there is a risk of a reoccuring symptom without it. He later stated that one should never hesitate to recompress an individual with symptoms even 1-7 days after the incident, as the symptoms can be releaved even after this time interval. This is especially true of cases which involve the spinal cord. Nerve tissue after bubble occlusion is usually viable, but not functional, so that treatment usually brings complete recovery even a week later.

In the making and modifying of recompression tables, Dr. Behnke stated the Golden Rule for this operation. You don't try out anything on anyone which you don't try on yourself. In this way, mistakes aren't repeated!

While conducting the Squalus operation they had very few accidents. This was attributed to the way they conducted the operation. The individuals (divers) were well rested, rehearsed the techniques used underwater on the surface, took their time doing the work underwater, and when each individual operation (task) was completed, they came up.

Body fat:

Body fat can be measured by weighing the individuals in water. Any body of water will do, even a swimming pool. Some come equipped with a six foot deep tank and a scale for this purpose. To be accurate, a measurement of the residual volume in the lungs must also be made.



Scale--measurements made after complete exhalation

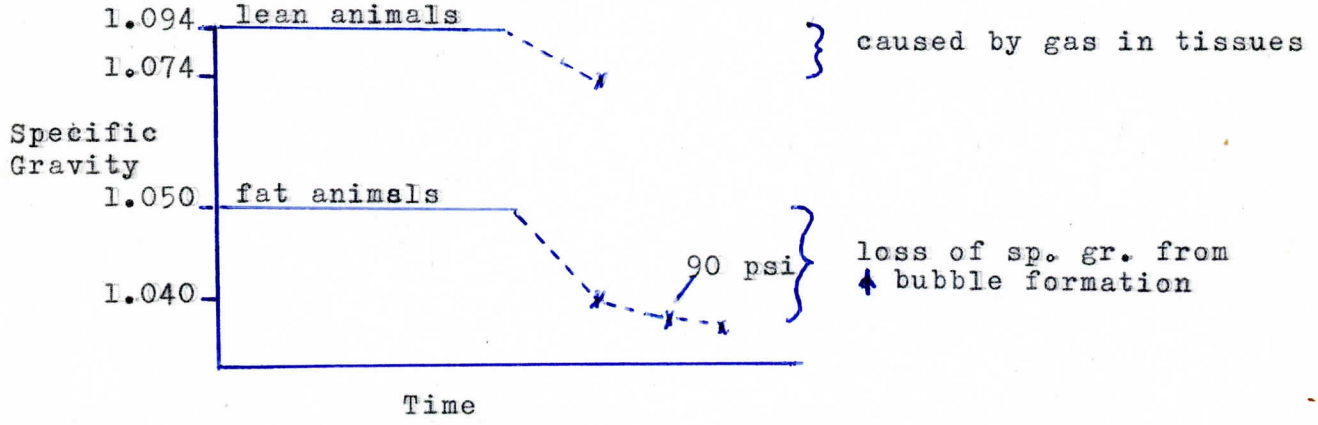
Fat individual
Specific gravity--1.02
% body fat--40%
Can withstand 1 hour exposure to cold water

Thin individual
Sp. Gr.--1.09
% body fat--5%
Can withstand only 10 minutes in cold water

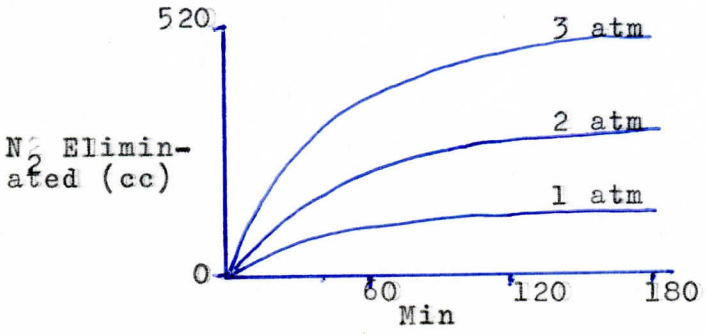
It is not the number of calories an individual looses in cold water which counts, but the amount by which this caloric loss lowers the core body temperature. For this reason a fat individual can afford to loose more calories than a thin individual. A channel swimmer (the only one to do this) swam across the Strait of Jaun de Fuca, a nineteen mile swim. To do this he added 65 lbs to his normal 240 lbs as insulation. So fat can be an advantage in scuba diving as protection from cold; it is a disadvantage in deep diving for reasons already discussed.

Man in cold water adapts in the same way aquatic mammals adapt. Skin temperature decreases to around 65°F, blood flow to the skin is cut off to prevent chilling of the blood, and although the individual may shiver, he doesn't feel cold until exposed to the air (even warm air). This is caused not so much by evaporation as by blood being chilled as it is reshunted to the skin during a slow rewarming. Core body temperature will drop about 3°F while in the water and can then be secondarily dropped another 3°F during a slow rewarming as the blood is chilled by the skin. The best measure is to use a rapid rewarming (say a 100°F shower) after the dive. However, it must not be too warm as this will lead to a decrease in circulatory effeciency.

Fatty animals, however, can have problems with decompression sickness. An experiment was conducted in which animals were compressed then rapidly decompressed, and their specific gravity taken and compared before and after (as a measurement of bubbles in the tissues) and it was found that lean animals had less bubble formation than fat animals.



A number of dogs have been decompressed from 4 atmospheres to 1 atm in 30 seconds, then given surface O_2 treatment. Surprisingly, the all-or-nothing law applied. They either made complete recoveries or died. They also found that N_2 elimination on the surface was proportional to the pressure at which they were saturated.



Air embolism:

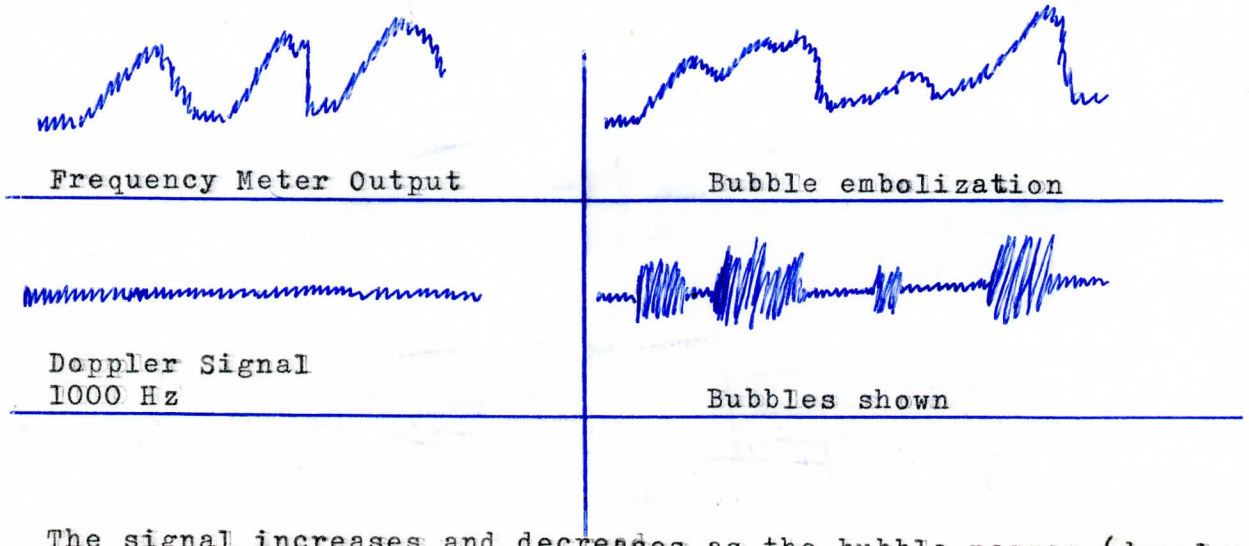
There are differences between what the helmet diver and the scuba diver experience in the water. The helmet diver is enclosed in air, and therefore his lungs experience ambient pressure. However, the scuba diver is always in immenant danger of drowning as he's exposed to the water. In the vertical position, his lungs experience a negative water pressure due to the length of the respiratory passages. Some years ago there was a death in a training tank in which a diver made a free ascent from ninty feet. He blew most of his air out, and was ascending so slowely that the safety divers pushed him toward the surface. He was okey going past the two safety stations, but went unconscious near the surface and died shortly thereafter. Throughout, the ascent had been slow and he'd been exhaling, so they reported that it could not possibly be a case of air embolism. Recently, the case was reviewed and the conclusion drawn that he had in fact died of air embolism. What had apparently happened was that the complete exhalation at the beginning of the ascent had compressed the lower lobes of the lungs, causing an entrapment of air in the aveoli which lead to the embolism upon ascent. Moral: don't completely exhale during free ascent.

Decompression sickness:

There are visual disturbances associated with decompression sickness, but these are usually transient. The bubbles pass through the arteries due to high arteriol pressure and accumulate at the low pressure side in the veins and venioles. Large accumulations of bubbles leads to chocks and the classic symptoms around the joints, & substernal distress upon deep inspiration (an early symptom). Greek sponge divers look for two signs: 1. Skin discoloration due to bubbles in the cutanious tissue (the marbling effect), and 2. paroximal caughing upon inhalation of cigarette smoke (a person with decompression sickness is intolerant of lung contaminents). If these symptoms occure, they put him back into his hard hat suit and push him overboard, keeping him at 50 feet for the night.

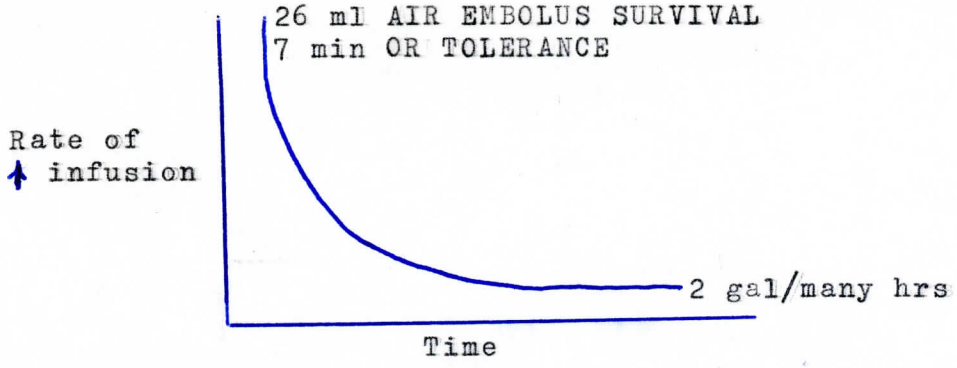
Two other general comments: increased CO_2 in the blood stream tends to enlarge the bubbles and the administration of O_2 decreases bubble formation (probably both rate and size).

Bubble formation can now be monetored using high frequency sound. More on this later, but graphs of the bubbles look like this:

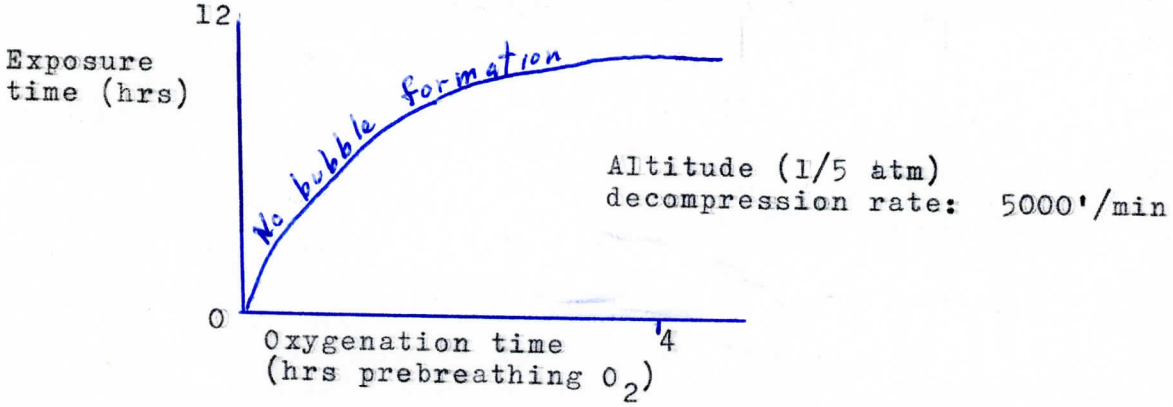


The signal increases and decreases as the bubble passes (doppler

effect) and is recorded on the graph. It is possible to use this device to decompress an animal releasing pressure slowely until "silent" bubbles appear, then stopping, etc. Animals have a tolerance to bubbles if its spread over time, as is graphically shown below:



Another experiment, using animals exposed to 1/5 atm after varying O_2 prebreathing times, clearly shows the value of prebreathing.



When bubbles form in the circulation of the lung, the animal (or man) can get a maledy known as a micro-lung, a constriction of the bronchioles and resultent decrease in lung efficiency. The mechanism is shown below:

PHYSIOLOGIC EFFECT	CAUSE	RESULT
Catecholamines released Histamine Bradykinin Serotropin (these are released into the blood stream)	Pulmanary Embolism	
	↓	
	Vasoconstriction	Dyspnea (Tachypnea)
	↓	
	Pulmanary artery Hypertension	
↓ Plasma lipids	↓	Bradycardia
	Anoxemia	
Cell (RBC) Clumping	↓	Coronary Pulmenale
	Hemoconcentration	
Platlet aggregation	↓	Fall in arterial blood pressure
	Pulmonary Shock	

The treatment for micro-lung was not covered, but it is assumed by JCR that administration of 100% O_2 and recompression as soon as possible would be the only recourse. Dr. Behnke did say that the increasing tendency by physicians to use fluids must be avoided as this can overload the system and cause death.

Aseptic Bone Necrosis and bone lesions:

Lesions in the bone apparently result after repeated insult, and are caused by a blockage of the bone's vessels. This can occure with and without symptoms; e.i. can be caused by "silent" bubbles. Individual tolerance varies, as about 25% of those who should get lesions don't while others do. (Japanese shellfish divers have an 80% incidence of bone lesions by age 40, but they don't decompress.)

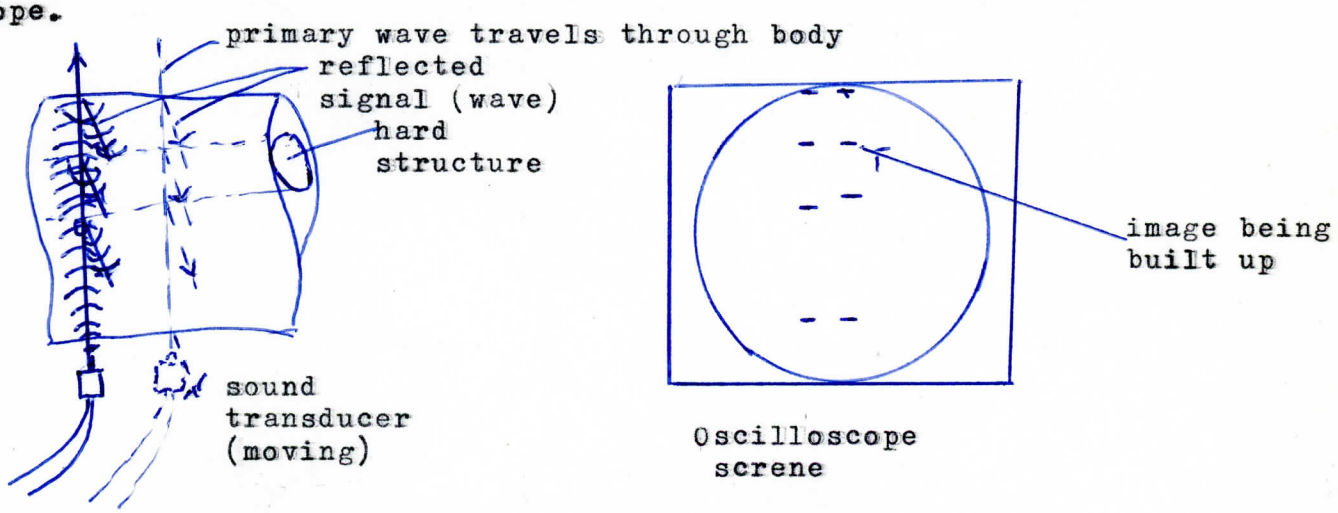
Mixing gases for recompression:

At 165 feet (58psi) a mix of 50-50% O_2N_2 gives the advantage of having enough pressure to saturate the nitrogen bubbles back into the blood without accumulating more nitrogen in saturation.

DR STUART MACKAY: DIVE TABLE DESIGN

Asides on ultrasonics, biomedical telemetry and liquid breathing:

We can now "see" bubbles in a diver's body and decompress accordingly. How can one watch bubbles as they form in decompression sickness? Using ultra sound, we can now build up images of the inside of the body. This is done by scanning an area with a sound transducer, recieving the reflection and building up an image on an oscilloscope.



The sound transducer uses 5000 cycles/second frequency and this is shown on the oscilloscope at 20 scans/second.

The interface between a gas and a liquid provides a very good reflective surface for sound waves. Individual bubbles as small as one micron can be seen.

Different tissue types have different <u>half times</u>, or <u>characteristic times</u> for saturation, desaturation and bubble formation.

<u>Tissue pressure</u>: Bubbles form at ambient pressure and grow as the gas comes out of saturation from the tissue. The tissue pressure can be read by raising the ambient pressure until the bubble stops growing (dissolved gas in the tissue reaches equilibrium or saturation level). The ratio of tissue pressure to ambient pressure is called gamma (γ).

tissue pressure/ambient pressure = γ

Liquid breathing:

Dr. Mackay showed the group several films, one on liquid breathing, in which rats breathed the liquid for several hours without problems. The liquid was not water, but a fleurocarbon or silicone fluid which had these properties:

1. Higher O_2 and CO_2 carrying capasity than whole blood.
2. It's not miscable with body fluid (which prevents hemo-concentration or dilution)
3.

Liquid breathing can be done with water, but it must be isotonic saline and have about nine atmospheres of pressure over it. Even then, a working diver would have trouble eliminating CO_2 (this is true with any of the liquids). Chemicals which control blood acidity (trisk buffers) would also have to be added.

The greatest problem encountered with liquid breathing, however, is the question of how to maintain core body temperature when the huge surface area of the lungs are in contact with a cooler liquid. The advantages of a diver breathing liquid (no worries of decompression sickness or nitrogen narcosis, for instance) are far out weighed by the temperature problem. One should also consider that no aquatic mammal has evolved a system for breathing water. They all breath air.

Body temperature (core temperature) can be monetored by using extremely small radio transmitters which are fed to the animal. The animal is free to do whatever it wants, and therefore the data is realistic. These transmitters are very inexpensive and will transmitt through fresh water and for limited distances through salt water.

Decompression Tables:

All decompression tables are based on a simple model, the Haldane model which states that different parts of the body will take up and release gas at different rates, dependent upon blood profusion into the area. A mathmatical model is constructed by assigning different tissue half times to different "compartments" of the body. One compartment would have a half time of 5 minutes, another of 10 minutes, etc.:

	$\frac{1}{2}$ times
T_1	= 5 min
T_2	= 10 min
T_3	= 20 min
T_4	= 40 min
T_5	= 80 min
T_6	= 120 min

"T" equals various tissue types

$T_i = T_1 + T_2 + T_3 + T_4 + T_5 + T_6$

T_i = 5, 10, 20, 40, 80, 120 min

$$\frac{d\pi_i}{dt} = K_i(P_n - \pi_i), \quad \text{where } K_i \ \frac{.693}{T_i}, \ dt = \text{rate of change}$$

π = partial pressure of inert gas in the tissue

P_n = partial pressure of inert gas in the breathing medium

$P_n = P - 66$ (Continuously variable gas mixture)

P = surrounding ambient pressure

Calculations for air tables are based on N_2 partial pressures. What the equasion says is that the rate of flow of gas into tissue is directly proportional to the ratio of the difference of partial pressure between the gas in the breathing medium and in the tissue. The tissue pressure ratio, then, is all important in bubble formation.

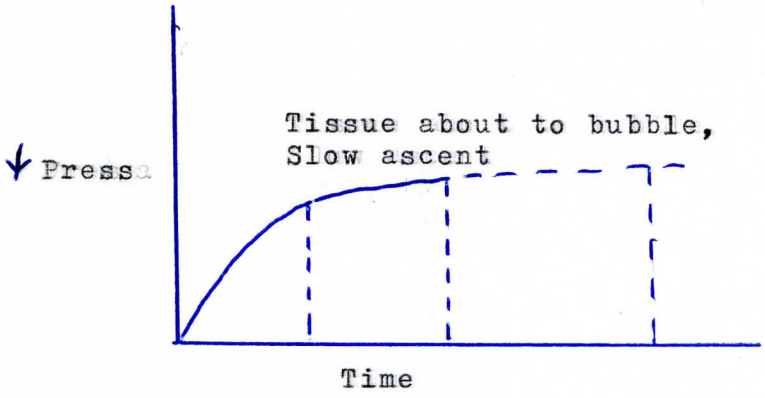
The equasion used for tissue pressure ratios is below:

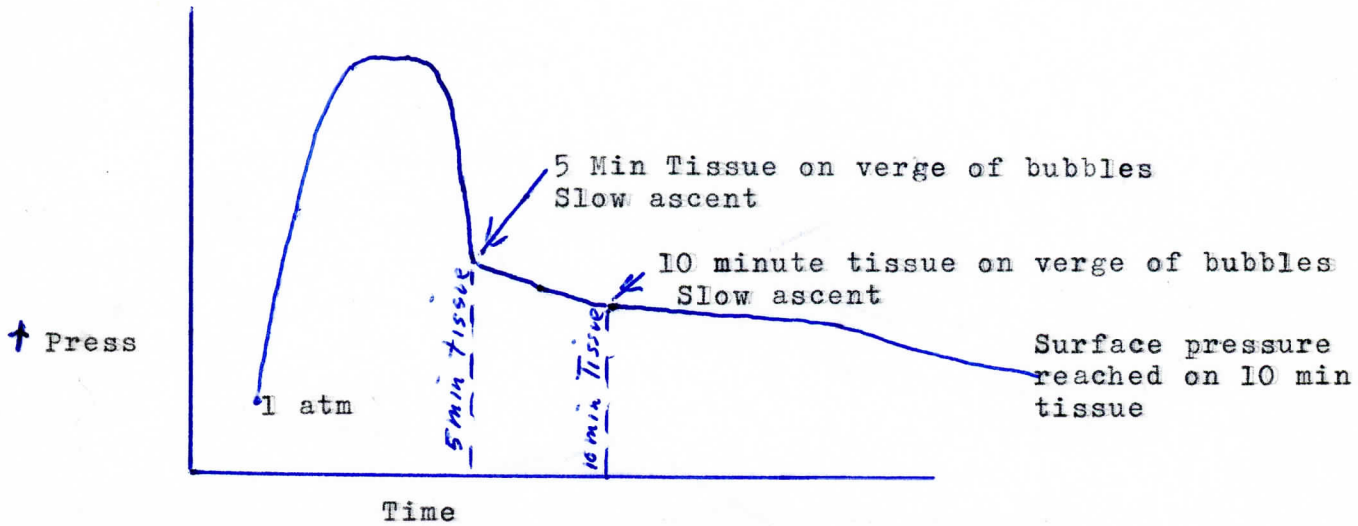
$$\pi_i = \gamma_i P$$

OR

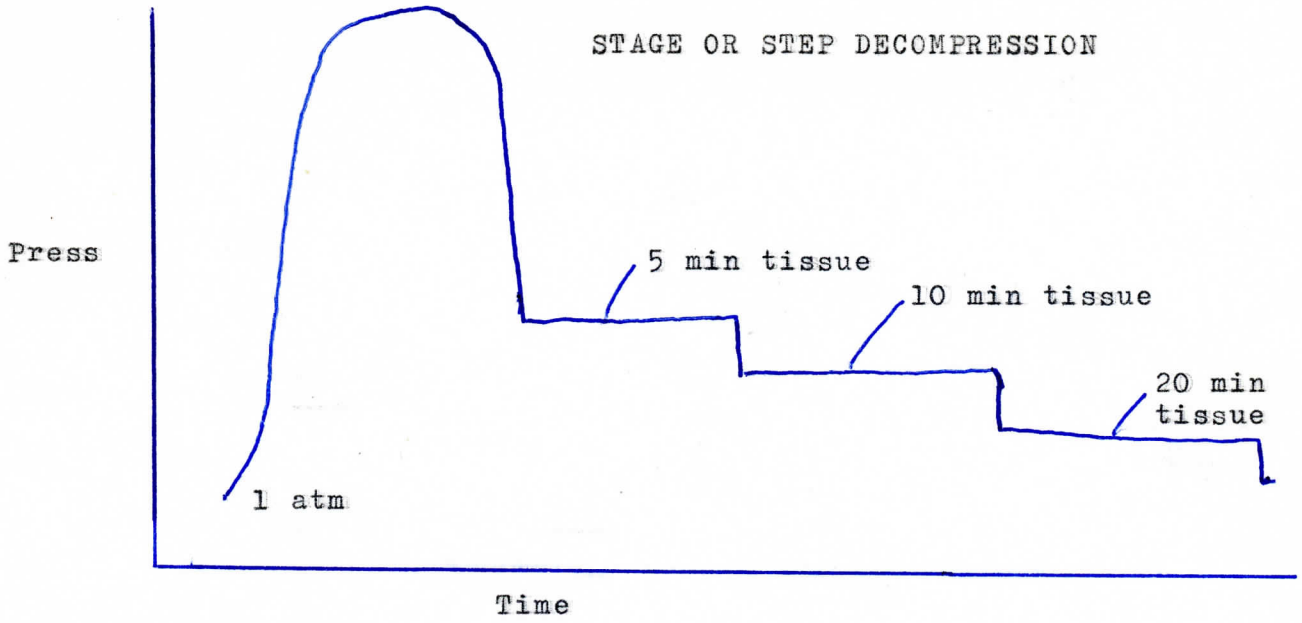
$$\gamma_i = \frac{\pi_i}{P}$$

According to the Haldane model, bubbles will begin to form when γ_i is approximately 2 (The pressure in the tissue is two times the pressure of the breathing medium). The graphs below show how the tables are built by watching tissue pressure ratios.





The above graph represents continuous decompression, which is a speedy method of decompressing and is used in the chambers. This cannot be used in water, however, so another form of decompression, called stage decompression is used. Here, the diver stops his ascent at various levels to allow the tissue pressure ratio (and N_2 saturation level) to drop.



Diving at altitude:

If the diver starts from a lower pressure (and finishes), he can't immediately ascend as far to meet δ and not bubble. And so he must use equivalent depths (the depth equivalent to sea level) when diving at altitude.

$$\text{Depth}' = \text{Depth}\left(\frac{\text{Barometric press for w/ table made}}{\text{Barometric press at altitude}}\right)\left(\frac{\text{Density of } W_s}{\text{Density of } W_f}\right)$$

Where:

Depth' = equivalent depth at altitude

W_s = sea (salt) water

W_f = fresh water

For Lake Tahoe:

BP = 23.80" Hg
sea level BP = 29.92" Hg
Density of W_s = 63.9 lbs/ft^3
Density of W_f = 62.4 lbs/ft^3

A second problem, unrelated to decompression sickness, must be faced at altitude, that of O_2 debt blackout. Oxygen debt blackout may occure when a diver works hard at depth and builds up an O_2 debt, then surfaces. The rarefied atmosphere may not be enough to maintain O_2 levels and an irreplaceable O_2 debt, resulting in unconsciousness, may occure.

ULTRASONIC IMAGING, DECOMPRESSION SICKNESS AND BREATHING LIQUIDS

References containing figures from the films used in the lecture.

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