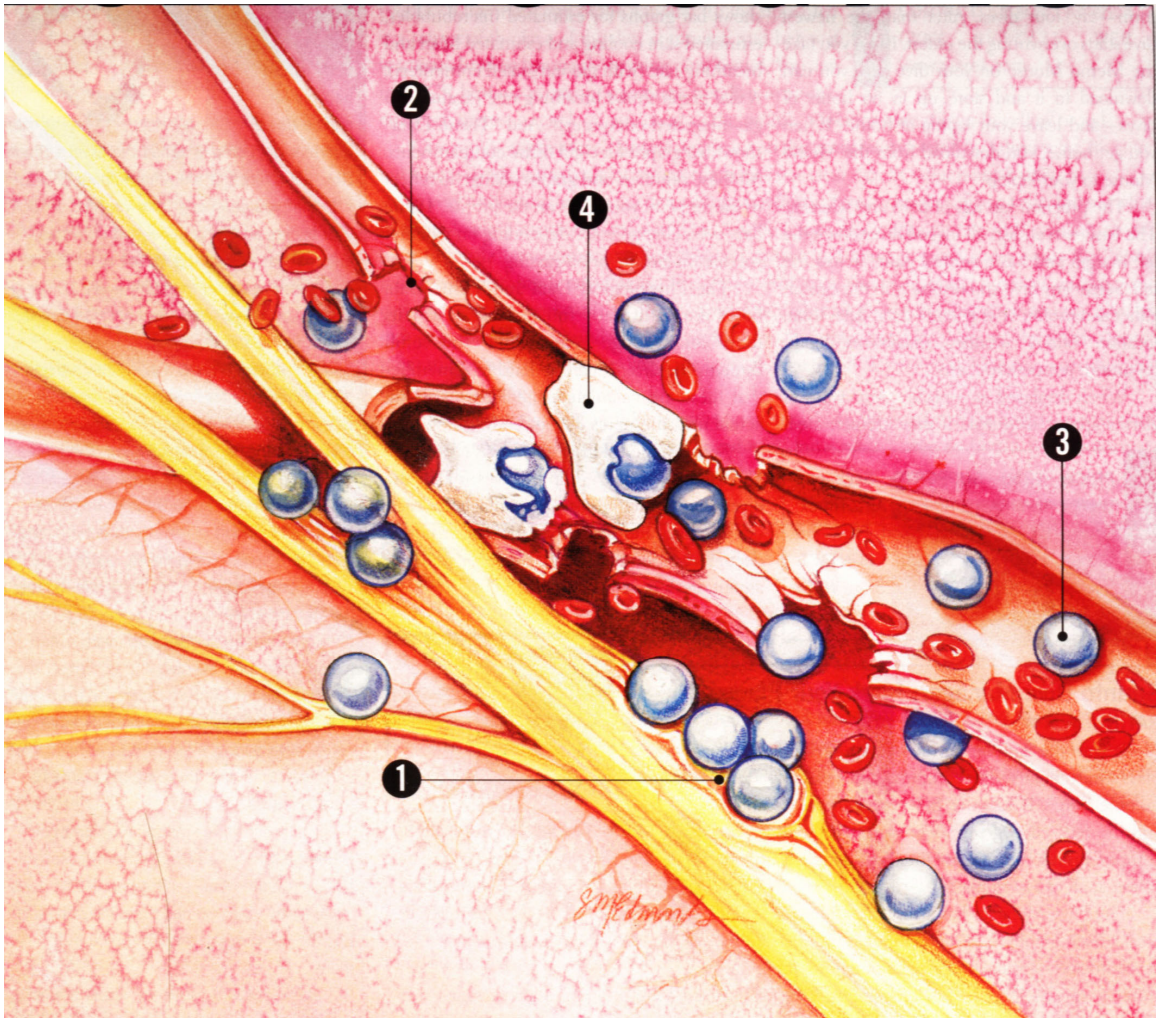
Diving Physiology



As nitrogen bubbles clump together or expand in response to decreasing pressure they can press on nerves (1) and tear blood vessels (2) bubbles can slow down the flow of blood (3). Theory holds that white blood cells can attack the bubble (4) causing even bigger obstructions.

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Abstract

Although recreational scuba diving was born in the late 1940s and early 1950s, the first recorded incidence of decompression illness occurred about 100 years earlier. During the 1840s, workers in pressurized French mines exhibited DCI symptoms after emerging from the mines. SCUBA diving has evolved tremendous in the last 50 years. As aquanauts push the limits of what was thought to be impossible we are discovering new physiological challenges. DCI (Decompression Illness), nitrogen narcosis, barotrauma, arterial gas embolism (AGE), oxygen toxicity are just a few of this aliments discussed in this report. Science is constantly refining models of the human body in order to predict how the humans will react under hyperbaric pressure.

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1 Properties of gas and pressure

Why are snorkels not 50m long? The answer becomes apparent as soon as one tries to breathe through a snorkel when one is more than 1 meter beneath the surface of the water. It becomes almost impossible to inhale at 1-2 meters due to the water pressure. The deeper underwater a diver swims the greater the water pressure becomes. At sea level the diver is exposed to approximately 101300 Pascal of air pressure (0) this is often referred to 1 atmosphere or 1 atm. This pressure is a result of the weight of the air in Earth's atmosphere. Gravity is pulling the gas molecules them toward Earth giving them weight. A column of air that is one square cm in cross section, extending from sea level to the edge of the atmosphere has a combined weight of about 2.66kg. Since the density of water is much greater then air water pressure increases at depth quickly in comparison.

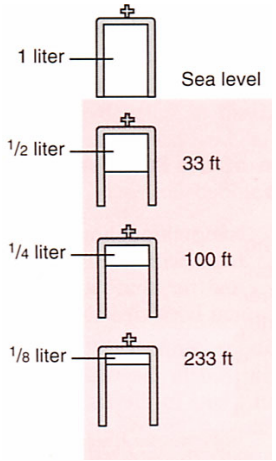

Fig. 1 Air compression at different depths. Guyton

1.1 Boyles Law

Boyle's law states that the pressure (P) of an ideal gas at a constant temperature varies inversely with its volume (V). That is pressure increases as volume decreases and vice versa. The pressure of a gas is the amount of force per unit of area that the gas exerts on its container. The volume of the gas is the three dimensional size of its container. According to Boyle's law the product of an ideal gas's pressure and its volume is constant. The mathematical expression of Boyle's law is PV = constant. If a balloon is filled with volume of 4 liters of air at the surface and then is brought down to 10 meters the balloon's volume will shrink to 2 liters. At 20 meters the air will be twice as compressed as it was at 10 meters so the volume will be just 1 liter of air. This is an exponential decrease (Fig. 1). If a plastic bag is filled with air and sealed at 10 meters and then is allowed to float back to the surface it will explode. This is because the volume of air in the bag will double in the bag every 10 meters on ascent. The same is true for a diver's lungs. Many diving associated traumas are associated with Boyle's law.

1.2 Partial Pressure and Dalton's law

It is important to remember that it is the volume of air that is decreased and not the amount of air that is in the container in figure 1 as it descends into the depths. In other words the concentrations of the components in the air are concentrated as the bag descends. Air is composed of about 78% nitrogen, 21% oxygen and about 1% various other gases.

Daltons law states that the total pressure of a gas mixture (like air) is the sum of all the gases in the mixtures partial pressures together. At the surface the partial pressure of nitrogen is 0.78 and O2 is 0.21. In diving applications partial pressure is expressed relative to the surface pressure. It is tempting to assume that a 0.21 oxygen partial pressure is referring to 21% oxygen but this is not the case. At the sea level the partial pressure of nitrogen is about 0.78. As the container in figure 1 is brought down to 10 meters the atoms of nitrogen will be twice as close to each other. The partial pressure of the nitrogen at 10 meters is now said to be 1.56. This is not 156 % nitrogen. Oxygen becomes toxic at about a partial pressure of 1.8 as discussed later under oxygen toxicity. A diver breathing air at 85.7m is risking oxygen toxicity.

1.2 Henry's Law

Henry's Law states that "The amount of a given gas that will dissolve in a liquid at a given temperature is a function of the partial pressure of that gas in contact with the liquid" What this means for divers is that gas molecules will dissolve into the blood in proportion to the partial pressure of that gas in the lungs (5).

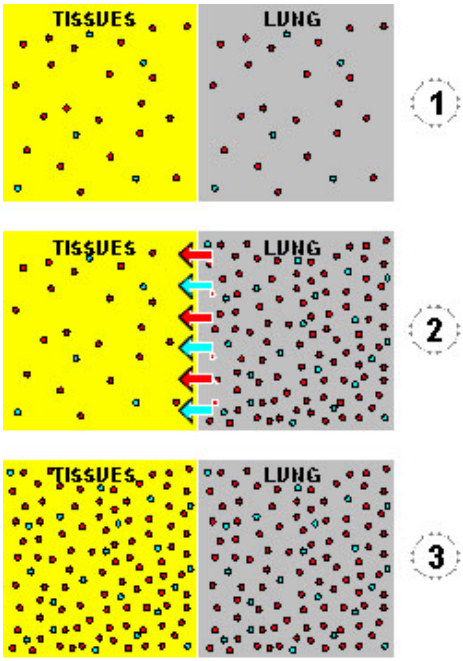

Fig. 2 Henry's law. Tissue loading

Fig. 2 represents the boundary between the lungs and the blood/tissues of a diver. At sea level, the dissolved gases in the blood/tissues and lungs are in equilibrium. As the diver descends into the depths water pressure increases. The pressure of the gases inside the lungs increases likewise. Because the partial pressures of the gases in the lungs are now greater than the dissolved partial pressures of these gases in the blood and tissues the gas molecules begin to defuse from the lungs into the blood and tissues as seen in the middle picture. Ultimately the concentration of the dissolved gases in the blood and tissues will be proportional to the partial pressures in the breathing gas. In SCUBA the breathing gas always has the same pressure as the ambient water pressure. This will continue until a new state of equilibrium is reached for the new depth. The tissue is now said to be "loaded" Some tissues will absorb the inert gases faster then others. The CNS and Fats of the body will for example absorb nitrogen much faster then bone. This is why the body is modeled by tissue compartments as described later in this report.

2 The Haldanean Decompression Model

In 1906, the British Royal Navy assigned physiologist John Scott Haldane to investigate the causes of, and seek a remedy for, DCI. Haldane drew upon the work of Paul Bert, a researcher who had identified dissolved nitrogen as the culprit in DCI, but Haldane was the first to attempt to predict and prevent DCI in a systematized manner. Working with a team of researchers, Haldane experimented initially with goats in pressure chambers. He was on of the most courageous scientists ever. Haldane and his team preformed tests on themselves and volunteers - *Exploding teeth, burst eardrums, seizers and countless minor DCI hits were all in a day's work for the team. Anyone who could prove that they had suffered a DCI hit was given a day out of the pressure chamber* (1).It was said that Haldane could be always be found by following the blood soaked tissues that he used for his frequent nose bleeds resulting from his experiments (1).

Haldane's research allowed him to speculate a decompression model that he made dive tables based on. He verified that his tables would work with human test divers in water. In 1908 their research, model and tables were published in the landmark 1908 paper, "The Prevention of Compressed Air Illness" in the Journal of Hygiene. Virtually all dive tables and dive computers calculate no decompression limits and decompression stops (see decompression diving) based on the original Haldanean decompression model. There have only been small refinements based on recent research and specific applications and the newer decompression models differ little practically from Haldane's original model.

2.1 Haldanean Model Structure and Operation

Based on the previous work of physicists and Paul Bert and on his experiments Haldane founded his model on these concepts:

• Upon descent to a given depth, nitrogen pressure in breathing air is higher than the pressure of nitrogen dissolved in the body. Nitrogen dissolves from the air (higher pressure) into body tissues (lower pressure) (2).

• Nitrogen continues to dissolve into the tissues until, given enough time, the body saturates and absorbs no more nitrogen at that depth. The nitrogen pressure in the breathing air and in the body have equilibrated (2).

• Upon ascent, nitrogen dissolved in the body (tissue pressure) is now higher than surrounding pressure. Nitrogen dissolves out of the tissues (high pressure) into the breathing air (lower pressure) and is exhaled from the body (2).

• The difference between the dissolved nitrogen pressure and the surrounding pressure (whether ascending or descending) is called the pressure gradient (2).

• On ascent, tissues can tolerate some gradient of high tissue pressure without DCI. Nitrogen in solution dissolves harmlessly out of the tissues (2).

• If the gradient exceeds acceptable limits, then dissolved nitrogen comes out of solution faster than the body can eliminate it through respiration and circulation. Nitrogen

bubbles form, causing DCI (2).

• DCI can be avoided by keeping the gradient within acceptable limits. This is done by calculating the theoretical pressure of nitrogen dissolved into the body and controlling the ascent so the gradient between theoretical tissue pressure and the surrounding pressure doesn't exceed critical limits. If the gradient will be too great by having a diver surface directly, the diver ascends in stages (decompression stops) that allow nitrogen to dissolve out of the body without ever exceeding the gradient limit (2).

Haldane found that a diver whose "tissues" were saturated by breathing air at a depth of 10 meters could ascend directly to the surface (sea level) without experiencing symptoms of DCI. Because the ambient pressure at 10m depth is twice that at sea level, Haldane concluded that a ratio of 2:1 for tolerated overpressure above ambient could be used as the ascent limiting criteria (3). This approximate ratio was used by Haldane to develop the first decompression tables.

3 Tissue Compartments

From his experiments Haldane also found that different parts of the body absorb and release dissolved nitrogen at different rates. To account for this Haldane constructed a mathematical model consisting of multiple theoretical tissues. *The theoretical tissues do not correspond to any particular body tissues to 100% but simply account for the fact that the body doesn't absorb and release nitrogen on a singular time scale.* Generalizations can be drawn on what tissues the compartments represent only. Because of this, it is more appropriate to call these compartments or tissue compartments rather than "tissues." Haldane's original model employed five compartments but modern versions may have 16 or more compartments. It's theoretically possible to create models with thousands of compartments, but on the practical side there's little benefit to doing so(2).

3.1 Halftimes

To each compartment Haldane assigned a halftime which is the rate it absorbs and releases nitrogen. The halftime is the time in minutes for a certain compartment to go halfway from its beginning tissue pressure to equilibrium (saturation) at a new depth. This is based on Henry's Laws as described earlier. The compartment dissolves in or out half the remaining inert gas for each half time creating an exponential progression so that a compartment is 50 percent equilibrated after one halftime, 75 percent after two, 87.5 percent after three and so on. After six haiftimes the compartment is practically 100 percent equilibrated.

Each compartment has an assigned halftime in minutes. A five minute halftime compartment would equilibrate in 30 minutes, and a 60 minute halftime compartment would equilibrate in 360 minutes. Haldane's original model had

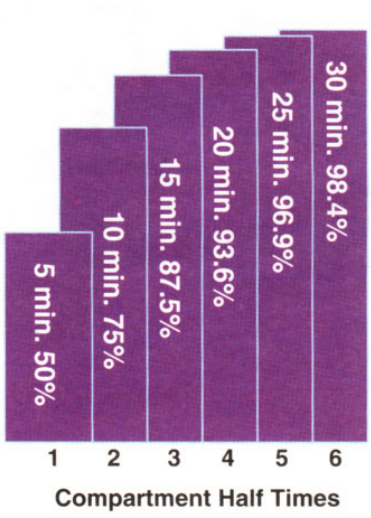

Fig. 3
Half times for compartments.
PADI

halftimes ranging from five minutes to 75 minutes; modern models range from three minutes to more than 600 minutes. Compartments with short halftimes are sometimes called “fast tissues” or “fast compartments” and those with longer halftimes are sometimes called “slow tissues” or “slow compartments.”

4 M-values

4.1 Workman M-values

Robert Workman began a systematic review of the decompression model including prior research that had been carried out by the U.S. Navy. Workman extrapolated some important conclusions. Workman acknowledged that Haldane's original ratio of 2:1 (based on compressed air) was actuality a ratio of 1.58:1 (3) considering only the partial pressure of the inert gas in air nitrogen. At this time it was known that oxygen was not a significant factor in DCI since it is metabolized. It was the inert gases like nitrogen and helium that were the cause of DCI. In his assessment of the research data Workman established that the "tissue ratios" for tolerated overpressure varied by halftime compartment and by depth. The statistics showed that the faster halftime compartments tolerated a greater overpressure ratio than the slower compartments. For example the 5 minuet compartment will never reach its M-value at 25 meters while the slower ones will. This is shown in fig. 4. Workman also showed that for all compartments the tolerated ratios became less with increasing depth. Instead of using ratios Workman described the maximum tolerated partial pressure of nitrogen and helium for each compartment at each depth as the "M-value."

The "M" in M-value represents "Maximum." For a given pressure an M-value is the maximum amount of inert gas pressure that a hypothetical "tissue" compartment can tolerate without symptoms of DCI due bubbles coming out of solution. M-values are representative limits for the tolerated gradient between inert gas pressure and ambient pressure in each compartment. Other terms used for M-values are "limits for tolerated overpressure," "critical tensions," and "supersaturation limits." The term M-value is commonly used by decompression modelers.

If the diver exceeds the M-value in any compartment, there’s an unacceptable risk of decompression sickness.

In no decompression diving, we need only concern ourselves with the surfacing M-value, sometimes written as the “M_0-value” pronounced "M naught". the faster the compartment, the higher its M-value filling at a deeper depth with slower compartments having lower M-values filling at shallower depths. Decompression designers base M-values on test dives and dive data which show what dive profiles do and do not result in DCI or in micro bubbles that do not cause DCI but are detectable by Doppler ultrasound in the circulation. The designer sets the M-value for each compartment so that time limits determined by the model remain consistent with the data. The trial and error technique was much the way Haldane worked.

4.2 Bühlmann M-Values

Bühlmann's technique for decompression calculations was similar to the one that Workman had worked out. This included M-values which expressed a relationship between water pressure and tolerated inert gas pressure in the tissue compartments. The
main difference between the two models was that Workman's M-values were based on *depth pressure* such as diving from sea level and Bühlmann's M-values were based on *absolute pressure* such as diving at altitude. Workman was only concerned with the diving actions of the U.S. Navy performed at sea level. Bühlmann was concerned with diving activities in the high mountain lakes of Switzerland. Since divers will dive in anywhere possible such as the Dead Sea Dive with its sea level of -394 meters to mountain lakes at 4000 meters computers today try to accommodate various types of diving and thus generally use Bühlmann's algorithm.

5 Haldanean Model in Action

As mentioned modern variations of the Haldanean model work basically the same way. The model works by determining how much each compartment absorbs for a given depth and time. When any compartment reaches its M-value the dive ends in so called “recreational diving” or becomes a decompression dive. This is discussed later.

On deeper dives the faster (with shorter half times) compartments reach their M-values first. The M-values are higher but the half-times are so short that the compartments quickly reach their limits. This can be a problem on bounce dives where the diver dives very deep and back up again in a short period of time.

On shallower dives the maximum pressure that can load into any compartment even after saturating is less than the M-value of the faster compartments. This means the fast compartments can never reach their M-values on shallow dives so slower compartments set the dive’s limits or control the dive. On a deep or shallow dive the compartment that reaches its M-value first and therefore ends the dives is called the controlling compartment.

In the fig. 4 we see 3 different scenarios the first one is nitrogen

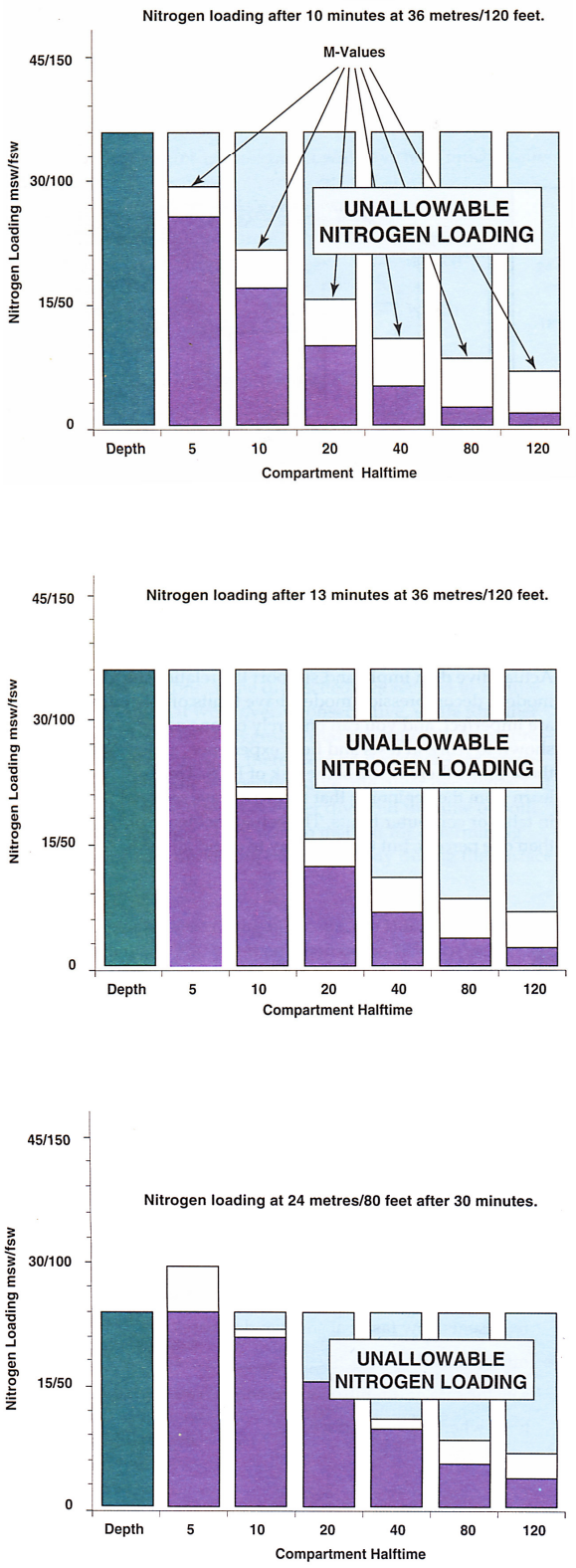

Fig. 4
Compartment halftimes on X axis and M-values on the Y axis.

loading after 10 minutes at 36m. No compartment has reached its M-value

In the 2ed table in fig. 4 we see nitrogen loading after 13 minutes at 36m. The 5 minute compartment has reached its limit, making it the controlling compartment

In the 3ed table in fig. 4 we see nitrogen loading after 30 minutes at 24m. The 5 minute compartment has equilibrated however at 24m the 5 minute compartment can never reach its M- value control the dive.

5.1 Basis for Haldanean Models

As mentioned Haldane based his model on experiments just as revisions to the models over the years have also been supported by test and field information.
There is no *direct relationship* between the decompression model and the body.
Dive data imply and support the relationship but like all models decompression models have limits of reliability. Models are imperfect and one can only rely on one as far as it has been shown to work in tests and field experience and even within those limits there is still some risk of DCI. This is why divers learn from the beginning that there's always some risk even within table or computer limits. The actual incidence of DCI is less than one percent following the tables but the only way to avoid all risk is to not dive.

5.2 Other algorithms

Newer and more complex models are now being used with great success. The Reduced Gradient Bubble Model (RGBM) is a quantum leap in diving technology (12). Unfortunately the mathematics is extremely advanced and makes it difficult for submergible diving computers to be able to use the model in its fullness in real time. The Variable Permeably Model (VPM) is another algorithm in use but it is very experimental and there have been fatalities with it.

6 Decompression diving

If a diver surpasses the M-values by staying at depth for a long period of time the diver will have to ascend slowly to vent out inert gas safely. In practicality this is done in steps. The diver ascends and the tissue tension will rise accordantly. In other words ascending will cause a pressure gradient for the inert gas in the loaded tissues to rise. This is due to the high amount of inert gas in the tissue and the lower pressure the diver is exposed to on ascending. The diver stays at this new depth until the excess inert gas is expelled from the lungs and the tissue tension lowers. The diver repeats this method of ascending and stopping until at the surface (see Summing up a dive profile). Nitrogen will have ample time to defuse out of the tissues slowly enough so that that tissue tension will not become so high that the inert gas will form bubbles and cause DCI.

6.1 Accelerated decompression

A diver decompressing will want to have as little nitrogen in the lungs as possible when decompressing to reduce decompression time. As dictated by the laws of entropy a high concentration of a certain gas will defuse into a lower concentration of that same gas. If the body has a high load of nitrogen and the diver is breathing a much lower concentration of nitrogen in the mix the tissue load will defuse out into the lungs much faster. See fig. 2 but this time the diffusion of nitrogen is the going into the lungs because of the low nitrogen concentration there. Optimally the diver could breathe 100% oxygen at depth. This is not possible because of the partial pressure 1.8 limit (see oxygen toxicity). Decompression divers will try to keep a oxygen partial pressure of 1.6 (1.8 is too dangerous). This amounts to 50% oxygen when the diver is at about 21 meters and 100% oxygen when the diver is at 6 meters.

For example a 170 minutes decompression time will be shorted to 68 minutes using the described oxygen scheme if following an unmodified Bühlmann's (ZH-L16B) table. There are risks by do this. If the diver is breathing 100% oxygen at 6m the partial pressure of oxygen is 1.6. If that diver for some reason descends just 40 cm while decompressing the partial pressure will become 2.0 and the diver very likely will have a seizer and drown.

7 Diving related injuries

7.1 Pulmonary barotrauma

Pulmonary barotraumas is a lung over expansion injury resulting when a diver holds their breath or can not exhale gas out of their lungs and ascends. This is described under Boyle's law and fig. 1. Holding ones breath and ascending only a few meters is often enough to cause a pulmonary barotrauma. Other physiological conditions such as a ventilation obstruction like a chest cold or an asthma attack can also cause a barotrauma to the lungs.

As the reader recalls it is impossible to inhale air though a long hose to the surface. This is due to the intense water pressure around the body that presses the gas out of the lungs when at depth. So how does a SCUBA diver inhale when underwater? The diver is able to fill their lungs with gas at depth because diver is breathing through a regulator. Gas is force into the diver's lungs with the same pressure that the water has. Hence the name breathing compressed air and breathing though a regulator. The result is that the diver can breathe as easily underwater as on the surface since the 2 forces will cancel each other out.

It is now realized that the gas the diver is breathing is compressed and thus has the potential to expand if the diver ascends. For example if the diver hold their breath at 10 meters and has 4 liters of air in her lungs the air will expand to 8 liters on surfacing. This will cause the lungs to explode. This often happens when a diver panics since there is a reflex to hold your breath and swim to the surface as fast as possible when underwater. An asthma attack at depth is another reason for compressed air not to be able to exit the

lungs. This often happens if has asthma and the water is cold or the diver exerts themselves.

In either case the expanding air can rupture the lung releasing bubbles into the body tissues leading to four distinct possible injuries:

7.2 Air embolism (AGE)
The most serious and most common of the four is air embolism also called arterial gas embolism (AGE). Expanding air is forced though the alveoli into the pulmonary capillaries. Air bubbles travel in the blood stream though the heart into the arterial blood supply then to the body tissues where they block blood flow. Respiratory or cardiac arrest is common. More often then not the air will travel directly into the brain though the carotid arties. The blood flow to the brain is blocked and brain tissue is deprived of oxygen. Death often occurs swiftly. If death does not follow the victim is often brain damaged.

7.3 Mediastinal emphysema
Occurs when the expanding air get trapped in the chest cavity between the lungs. As the diver continues to ascend the air constricts the heart and lungs. This often leads to shock and cyanosis.

7.4 Subcutaneous emphysema
Similar to mediastinal emphysema but this time the air gets trapped under the skin around the neck and collar bone. The skin around the neck and shoulders tends to blow up like a balloon due to the over pressurization. A “crunching sound” it both felt and heard when the traumatized area is touched.

7.5 Pneumothorax.
Pneumothorax means air from an overexpansion forces its way into the space between the lung and the pleural cavity. Often the lung will collapse totally or in less sever cases partly.

7.6 Ear Squeeze

The outer ear canal leads to the eardrum (tympanic membrane). Ambient water pressure has direct contact with the ear drum (6) .The space behind the ear drum (middle ear air space) will be compressed at depth unless pressure is equalized. This is generally not a problem since the diver is breathing compressed

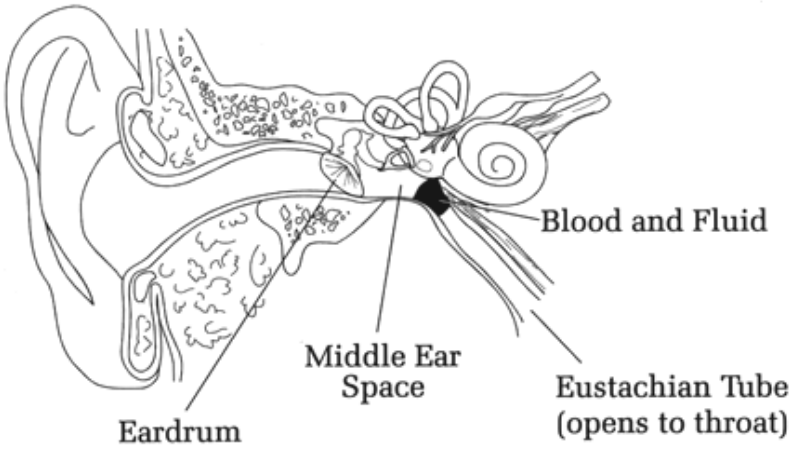

Fig. 5
Inner ear with squeeze

gas that has the same pressure as the water puts on the eardrum. The compressed gas travels up the Eustachian tube and the 2 pressures cancel each other out. The effect is the eardrum is not affected.

If the Eustachian tube is blocked gas cannot enter the middle ear space and the ear drum will bulge inward causing pain and possible trauma. The ear drum can rupture if the diver descends without equalization. If the diver continues to descend without equalizing fluids and blood from surrounding tissue will be forced into the middle ear space.

The diver has to continuously equalize to maintain the Eustachian tube is open on descent. If the diver does not equalize then the Eustachian tube will seal shunt when the pressure gradient across the Eustachian tube (nasopharynx to middle ear) exceeds about 90mm Hg or about 1.5 meters.

There are a few ways equalizing is done. The Valsalva technique is accomplished by a forced exhalation with the nose pinched, the lips shut against mouthpiece, and glottis open. The Frenzel technique is accomplished with nose pinched and lips sealed against the mouthpiece. The back of tongue is pushed against soft palate pushing air through the Eustachian tubes. The Frenzel technique is very similar to just swallowing.

7.8 Decompression Illness

If a diver stays at depth long enough the tissue compartments will eventually reach and then surpass their M-values due to inert gas diffusing into the tissue compartments as dictated by Henry's Law. If the diver then ascends quickly to the surface the compartments will have a much higher partial pressure then the surface partial pressures the diver is now exposed to. The tissues *gas tensions* become to great to keep the inert gas dissolved in the tissues comes out of solution forming bubbles that are released into the tissues and circulation. It is only the inert gases

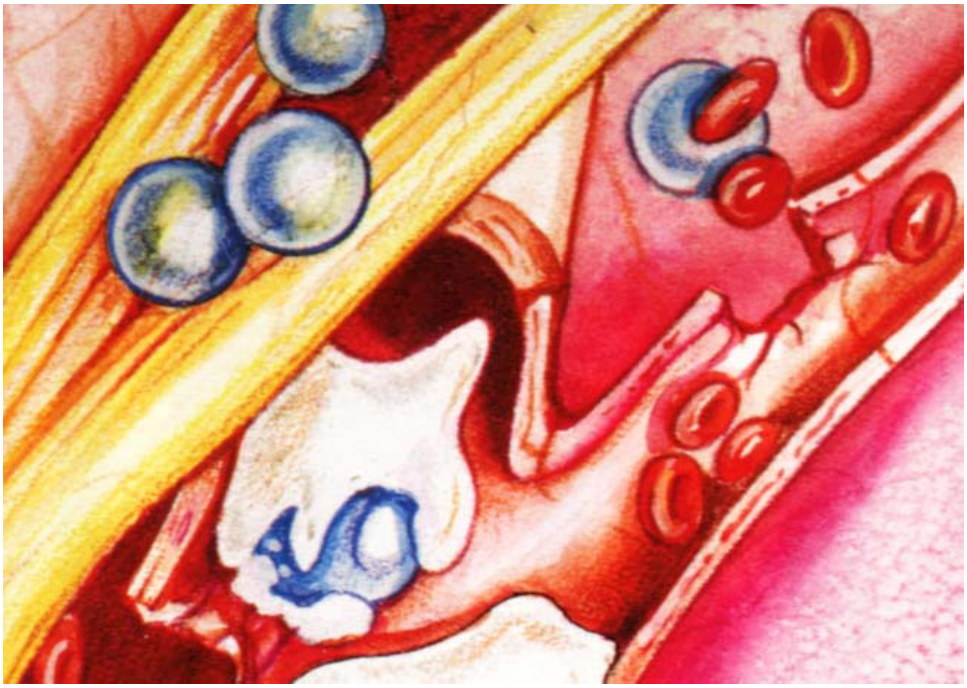

Fig. 6
As nitrogen bubbles clump together or expand in response to decreasing pressure they can press on nerves (1) and tear blood vessels (2) bubbles can slow down the flow of blood (3). Theory holds that white blood cells can attack the bubble (4) causing even bigger obstructions.

that cause DSI because oxygen is metabolized by the body's cells. In vary sever cases (usually supersaturation commercial divers) the blood can become so full of bubbles that the plasma becomes a foam. Since the CNS and expressly the spinal cord absorb nitrogen the fastest with their short half times bubble tend to form there first. As the diver continues to ascend the bubble will expand and cause havoc.

Damage to the spinal cord often looks similar to that of a shot gun wound often leaving the victim a paraplegic or in a worse case scenario a quadriplegic.

The inert bubbles clump together and expand in response to decreasing pressure. They can press on nerves and tear blood vessels, bubbles can slow down the flow of blood. White blood cells can attack the bubble causing even bigger obstructions (7). The secondary effects of inert bubbles in the body activate the inflammatory system since the bubbles are interpreted as foreign objects. The bubbles active thrombocytes, the cascade system and the compliment system. The smaller fragments of the compliment system (particularly C5a) activate granulocytes that in turn will attract white blood cells.

7.9 Thrombocytes and coagulation.
A quick decompression will cause blood trauma. The bubbles will active factor XII and trombocytes (8). The trombocytes become "sticky" adhering to the bubbles and will release substances such as serotonin, phospholipids and platelet factor III. Activation of factor XII will act as a starting vector for the intrinsic path of the coagulation cascade while platelet factor III will form complexes with activated factor X, factor V and Ca2+ that will in turn active thrombin. This results in the bubbles becoming encapsulated in a mesh of fibrin. Red blood cells and more thrombocytes will in turn become trapped by this mesh insuring a lager obstruction of the vessels that cause further depravation of oxygen flow to the surrounding tissues (8).

The anaphylatoxins will recruit leukocytes to the area as mentioned. Neutrophils and macrophages will up the expression of their adhesion molecules that will in turn adhere to the endothelia of vessels in the area. The anaphylatoxin C5a will also cause respiratory burst leading to the production of free radicals that will further damage the tissues of the area. All the above factors of the immune system are will further damage the tissues.

8 Oxygen toxicity

Free radicals are produced as a result of mitochondrial oxireductive processes and also produced by the action of enzymes such as xanthine/urate oxidase at extra-mitochondrial sites, from auto oxidative reactions, and by phagocytes during bacterial killing. These free radicals cause lipid peroxidations, especially in the cell membranes, inhibit nucleic acids and protein synthesis, and inactivate cellular enzymes. Normally, various antioxidant enzymes, e.g., glutathione peroxidase, catalase, and superoxide dismutase (SOD) protect the body from these free radicals, but in hyperoxic situations (diving), there is explosive free radical production leading to swamping of the enzyme systems and as a result free radicals escape
Inactivation (11).

Two oxygen toxicity parameters are typically “tracked” in technical diving calculations. The first is pulmonary oxygen toxicity which primarily concerns the effects to the lungs of long term exposures to oxygen at elevated partial pressures. The second parameter is central nervous system (CNS) oxygen toxicity which primarily concerns the effects to the brain of short to medium term exposures to oxygen at elevated partial pressures. Both oxygen toxicity parameters are a function of the partial pressure of oxygen in the inhaled breathing gas and the time of exposure. CNS oxygen toxicity is generally the parameter of most apprehension and greatest impact in technical diving. CNS toxicity leads to epileptic seizers with no warning. A seizer underwater is considered on a deep dive to be non survivable.

8.1 Pulmonary oxygen toxicity

When the lungs are exposed to high levels of oxygen deterioration of the lung occurs progressively. The first step is an acute influx of fluid into the tissues of the lung. Subsequently there is bleeding between the alveoli and destruction of capillary and type I alveolar epithelial cells. The fluid phase merges into a sub acute phase that is pronounced by production of tissues typically seen in attempts at healing. The result is thickening and scarring of the lung tissue.

The first symptoms are variable but usually occurs after about 12 to 16 hours of exposure at 1 $PP0_2$, 8 to 14 hours at 1.5 $PP0_2$, and 3 to 6 hours at 2 $PP0_2$. Pulmonary oxygen toxicity is generally not a problem in non decompression diving. Divers at risk for this type of toxicity are those who need to complete long decompression times of several hours on high mixtures of high PPO2 (see summing it up) or Nitrox divers doing multiple dives.

8.2 Cerebral oxygen toxicity

When PPO_2 is about 1.8 PPO_2 there is a serious risk of the diver having a seizer.
The CNS has an arsenal of free radical neutralizing agents. At a certain partial pressure of O_2 the CNS can not deal with the excessive free radicals and the diver becomes a statistic. The exact mechanism of a CNS hit is not know but Glutamic acid decarboxylase is one such enzyme inhibited in the CNS and a reduced level of gamma amino butyric acid (GABA) is seen concomitantly with occurrence of seizures (11).

8.3 Nitrogen Narcosis

Most divers have a love hate realtionship with nitrogen narcosis . Nitrogen narcosis has been called everything from “compressed air intoxication” to “rapture of the deep.” More properly it's "inert gas narcosis" because any inert gas and even some non inert gases can cause it. Nitrogen just happens to be the major inert component in air and thus the main cause of narcosis in recreational diving. At depths as shallow as 30m its effects can be

observed even if the diver is not aware of it. Although narcosis in itself is generely not dangerous it impairs the diver's cognitive functions in its mild form and as depth increases it affects the diver's motor functions. Its effects can be described similar to that of alcohol.

Exactly why compressed inert gas makes divers "drunk" is still a matter of speculation but as far back as 1899 H.H. Meyer noted that the more easily an inert gas is absorbed by fats the more narcotic it is. For example helium is about 1/5 as soluble in fat as nitrogen and about 1/5 as narcotic. Xenon which is used for surgical anesthesia is about 25 times as soluble as nitrogen and about 25 times as narcotic.

"Meyer-Overton Hypothesis" led A.R. Behnke, R.M. Thomson and E.P. Motley to the conclusion in 1935 that it was the absorption of nitrogen by myelin in nerve cells that causes narcosis. The Meyer-Overton hypothesis provides a potential explanation into why this occurs. This hypothesis states that narcosis symptoms occur when gas (N2) penetrates lipids of the brain's nerve cells and interferes with the transmission of action potenials. The nitrogen loading of the cells is directly in proportion to the partial pressure of the nitrogen with is a function of depth. The following table gives a rough estimate of what may happen at depth (9). It is important to bear in mind that the table does not represent factual depth limits. PADI's Deep Technical Dive course teaches dives to regularly dive to 50 m on air and many divers do not feel the described affects from in the following table at 50 m.

DEPTH	WHAT MAY HAPPEN
10-30 m	Mild impairment of performance on unpracticed tasks; mild euphoria
30 m	Reasoning and immediate memory affected more than motor coordination; delayed response to visual and auditory stimuli
30-50 m	Idea fixation; perceptual narrowing; overconfidence; calculation errors; memory impairment
50 m	Sleepiness; illusions; impaired judgment
50-70m	Convivial group atmosphere; possible terror reaction in some; dizziness reported occasionally
70 m	Severe impairment of intellectual performance
70-90 m	Gross delay in response to stimuli; diminished concentration; mental confusion
90 m	Stupefaction; mental abnormalities; almost total loss of intellectual and perceptive faculties
BELOW 90 m	Hallucinogenic experiences; unconsciousness

Table 1
Effects of nitrogen at depth

9 Helium as a substitute for nitrogen

Since nitrogen usually starts causing narcosis at a partial pressure of 3.12 or about 30m dives who want to do deeper dives need to replace the nitrogen in their gas mix with helium. As mentioned helium is not absorbed by the neurons as easily and thus causes much less narcosis. A diver who does a dive to example 100m can substituted a proportion the nitrogen in the gas mix with Helium so that the partial pressure of the nitrogen does not exceed 3.12. A gas mix for 100m often looks like the following:

12% oxygen, At 100m the p.pO2 is 1.32. This is under the 1.8 max.
60% helium
28% nitrogen. At 100m the p.pN2 is 2.31 resulting in a safe nitrogen narcosis.

A mixture of oxygen, helium and nitrogen is called trimix.
This will only give a nitrogen narcosis equal to a partial pressure of 3.08 at 100m (equal to about 30m on air) and oxygen a partial pressure of 1.32. Oxygen can cause a very serious condition called oxygen toxicity (ox-tox) at a partial pressure of about 1.8. The recommended maximum is 1.4 when diving and 1.6 when decompressing. There is generally no warning when hit with ox-tox. The diver goes into sever epileptic seizers usually with no warning signs. The result is almost always death by drowning. If the diver had the "normal" 21% O2 that is in the air the oxygen partial pressure would be 2.31 at 100m likely resulting in a seizer. A problem that arises is that humans need at least a PPO2 0.17 (17% at the surface) to prevent blacking out because of lack of O2. If a diver is breathing a mix with only 12% O2 at the beginning of the dive there is a risk of blacking out. To prevent an early blackout a diver will have several tanks of gas with them and then change the gas mix as the depth increases. In folling case the diver would start with the following mixture and then switch tanks to the

Surface to 60 m
20% oxygen, At 60 m the PPO2 is 1.4. This is under the 1.8 max.
35% helium,
45% nitrogen. 60 m the PPN2 is 3.15 resulting in a safe nitrogen narcosis.

From 60 m to 100 m
12% oxygen, At 100m the PPO2 is 1.32. This is under the 1.8 max.
60% helium
28% nitrogen. At 100m the PPN2 is 2.31 resulting in a safe nitrogen narcosis.

A diver who does a dive to this depth has most defiantly surpassed the described M-values. Helium is also an inert gas and will bubble out of solution just as nitrogen. To swim back to the surface with out decompressing would in this case lead to sever DCI and in all probability kill the diver.

9.1 HPNS

High pressure nervous syndrome (HPNS) is most likely the most significant limitation to the use of helium as a diving gas. The physiological process that creates this syndrome is still not entirely understood.

HPNS is a physical manifestation of a high pressure gas gradient across tissue compartments possibly compounded by breathing helium. It is exacerbated by rapid pressurization to depths of over 120 meters and appears at depths of between 120 and 200 meters, depending on the speed of descent and, to a degree, the physiology of the diver.

The symptoms of HPNS include muscle drowsiness, tremors, loss of appetite, dizziness, difficulty in concentrating, , nausea, vertigo and visual disturbances, such as spots or patterns breaking up the diver's field of vision.

In commercial diving, the effects of HPNS are reduced by slow and staged pressurization and by adding small amounts of nitrogen to "relax" tissues. Divers are pressurized to approximately 10-11 bars and held there for several hours for tissue saturation to take place, and the gas gradient to equilibrate.

Pressurization is then resumed, and the dive halted again after a further increase in pressure, for the process to repeat itself. The transit to the bottom depth may thus take many hours far longer than is possible on open or closed circuit SCUBA, (the diver had their gas with them) with an attendant decompression lasting several days due to the complete saturation of the divers' tissues with the inert gas mixtures involved.

To reduce the effects of HPNS small amounts of nitrogen may be used in the mixture to "relax" different tissues compartments and so reduce the muscle tremors that are typically the earliest and least controllable of the effects. The tremors are postulated to be caused by differential dissolution of gases into the tissues of the myelin sheath surrounding the nerves, causing the nerves to locally spasm. At depths of up to 120 meters HPNS is unlikely to be a problem, though in general, the greater the depth, the more the chance of the syndrome appearing. On the rare occasions that open-circuit divers have descended to greater depths, trimixes containing between 7-11% of nitrogen are thought to have contributed to the partial controlling (though not the elimination) of HPNS.

10 Summing it up

The reader may be wondering how a diver can keep all the above factors in mind when diving. Thanks to modern desktop computer software and computers that are brought on the dive itself diving is made easier. The author (Erik Adler) cut this table for a dive to 100 m with a bottom time of 20 min. The algorithm is Bühlmann M-Values ZH-L16B. The explanation for the Tx (Trimix) 20/30 and TX 12/60 can be found under **Helium as a substitute for nitrogen.** The green dots are stops that the diver must make so the tissue compartments are not overly stressed and release bubbles causing DCI as described under **decompression diving**. As the diver slowly ascends she will want to reduce her

decompression time by changing gas to a O2 50% at 21 m and then a 100% O2 at 6m as described under **Accelerated decompression**. This is done in tight marginal as described under **Oxygen toxicity.** The 16 vertical pillars represent 16 tissue compartments of the body following the **Bühlmann M-Values** ZH-L16B model. The compartments on the left are the faster compartments and the ones on the right are the slower compartments. Since the diver is now on the surface the compartments are showing the tissues tensions the diver has on leaving the water. Compartment 12 is at 99% of is maximum value before being overly stressed. The fast compartments are very permeable to nitrogen and load the fastest. This is true at for the rate they release nitrogen also. By the time the diver is at the surface they are empty. If this diver was to get a DCI hit it would most likely be in a tissue such as bone or cartilage and not in a fatty tissue like the CNS.

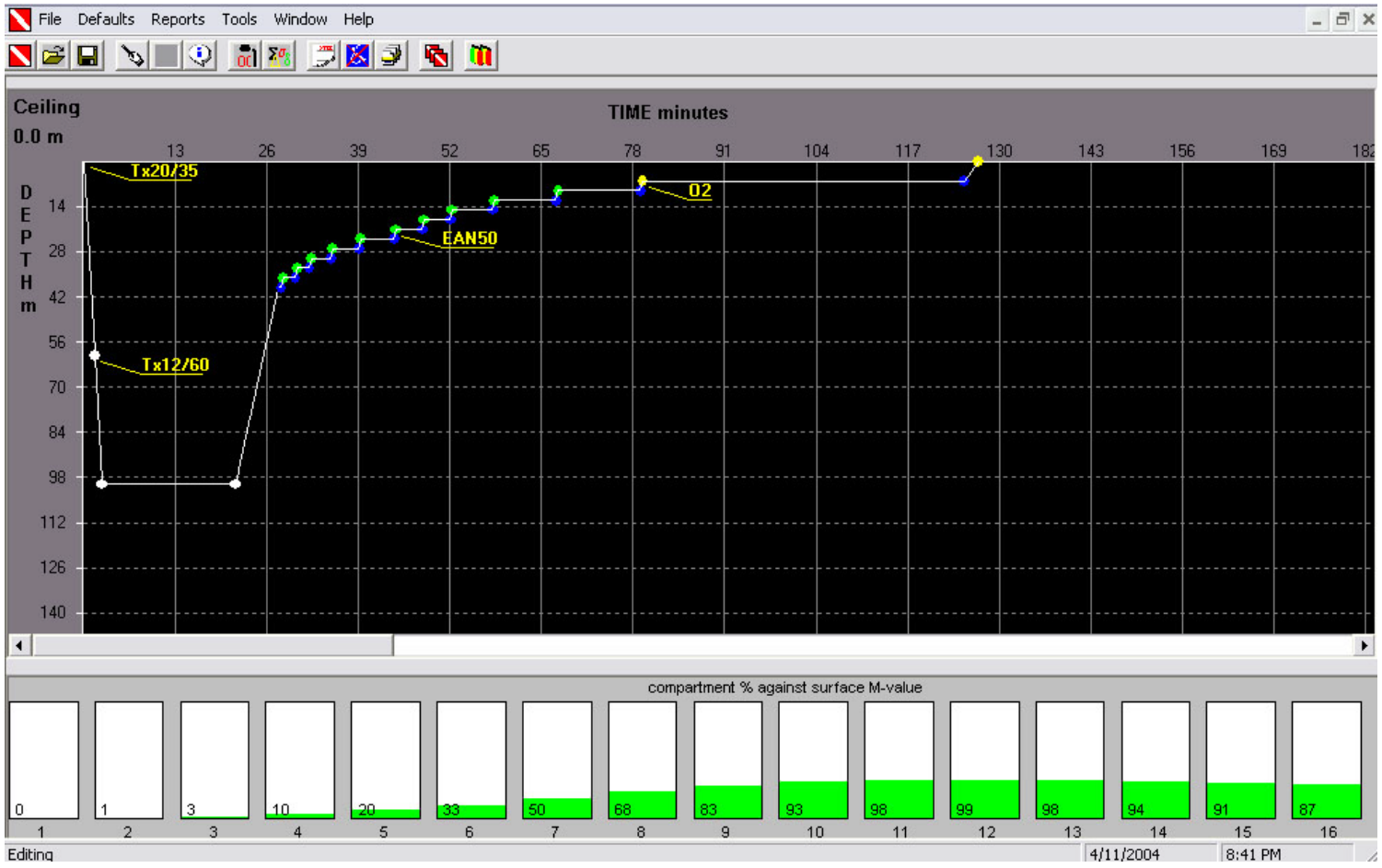

Fig. 7
A decompression dive. The Y axis is the depth and the X axis is the time. The author (Erik Adler) used GAP software to plan a dive to 100 meters with a 20 minuet bottom time. For simplicity deep stops have been omitted. Normally a dive like this has more stops and the decompression times do not stress the tissue tension on the compartments as heavily.

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