Diving injuries - Pressure and its effects in diving

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Abstract

The number of recreational divers is increasing rapidly. While many programs exist to make the sport safer, the potential divers have to deal with cases of decompression sickness and arterial gas embolism which increased. There is a clear need for divers to be aware of the signs, symptoms and treatment of diving-related injuries and decompression illness. The important thing is acquiring a basic ability to recognize decompression sickness and arterial gas embolism and provide treatment to injured divers. Initial recognition and proper treatment of an injured diver can save life.

Key words: *diving, pressure, injuries*

Rezumat

Numărul de scafandri amatori este în creştere rapidă. Deşi există multe programe care fac ca practicarea sporturilor să fie mai sigură, potențialii scafandri se confruntă cu o creştere a incidenței afecțiunilor determinate de decompresie şi a embolismului gazos arterial. A apărut, astfel, necesitatea ca aceştia să fie informați cu privire la semnele, simptomele şi tratamentul afecțiunilor legate de scufundare şi decompresiune. Un aspect important este acela de a dobândi abilitatea elementară de a recunoaşte afecțiunile de decompresiune şi embolismul gazos, precum şi de a indica tratamentul potrivit. Recunoaşterea precoce şi tratamentul corect al acestor afecțiuni pot salva viața.

Cuvinte cheie: *scufundare, presiune, afecțiune*

History of Scuba Diving

Jacques Cousteau was quoted as saying "It will happen my friends, surgery will affix a set of artificial gills to man's circulatory system–right here at the neck–which will permit him to breathe oxygen from the water like a fish. Then the lungs will be by-passed and he will be able to live and breathe in any depth for any amount of time without harm. It will happen, I promise you."

It appears as if Jacques Cousteau's vision came true with the development of scuba diving. Actually, scuba diving or the idea of diving has been around for quite some time.In the 1500's Leonardo Da Vinci designed the first known scuba diving apparatus. His drawings of a self-contained underwater breathing apparatus appears in his Codex Atlanticus.

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There is no record of Da Vinci ever following through with his design though. However, you can go back even further. Aristotle discussed the possibility of developing what eventually came to be known as a diving bell. A diving bell was a cable-suspended airtight chamber. It looked much like its name sake "the bell". As the bell was lowered under the water the pressure of the water would keep the air trapped inside the bell. Hoses fed down from the surface would send in compressed gases. His not only allowed the person to breathe but compensated for the gases that were being released from the bottom of the bell. Without this compressed gas, the bell would partially fill with water. The diving bell was one of the earliest inventions for under water exploration. Around 1531, an Italian explorer Guglielmo de Loreno developed the first true diving bell which he used for exploring sunken ships (1-4).

S.C.U.B.A. stands for Self Contained Underwater Breathing Apparatus. So we can see why it is so much easier to say you're going Scuba Diving.

People have been exploring the ocean since the beginning of time. Man ventured into the ocean for fish and other marine life. People have been practicing holding their breath underwater for as long as we can remember. The ocean and what it holds has always captivated us. Everything from a food source to simple curiosity over marine life and sunken ships, it's all fascinating. In earlier days, reeds were use to breathe underwater. This however was limited since you could only go right below the surface. An attempt was also made to breathe from an air filled bag. The only problem was that divers were breathing back in carbon dioxide.

Advances were being slowly made in the diving world and during the 16th century, France and England, had both created leather diving suits and with the aid of manual air pumps divers were able to go to depths of about 60 feet. The 19th century brought great advances in underwater exploration. With the research completed by Paul Bert from France and John Scott Haldane from Scotland we learned more about the effects of water pressure on the body. Their work also help to define safe limits for compressed air diving.

Technological advancements in the areas of compressed air pumps, carbon dioxide scrubbers, regulators and more made it possible for divers to stay under the water for longer periods of time. There are typically two types of scuba diving, open scuba diving and closed scuba diving. Open diving allows the diver to breathe air from a cylinder and the air blown out goes into the water and rises up to the surface. Closed diving is where the diver breathes in from a tank and then the blown air is released back into the tank where it is recycled to breathe again. This is also known as using a re-breather (5,6).

Speed of Descent

The speed with which the scuba diver can safely descend depends on his quickness of equalizing the pressure in the head cavities, and his training. It is more difficult to equalize in the first ten meters that at a depth of 100 meters. For example, diving from 100 m to 110 m is like diving from the surface to 1 m. After careful equalization in the first ten meters, the diver can reach 30-40 meters for one minute. Of course, this speed rests with the individual' s ability and his healthy condition.

Depth of Diving

The scuba diver breathes air under pressure from the aqualung. That is why the permitted depth of diving depends on the type of the breathing apparatus and the gas with which it is filled (7).

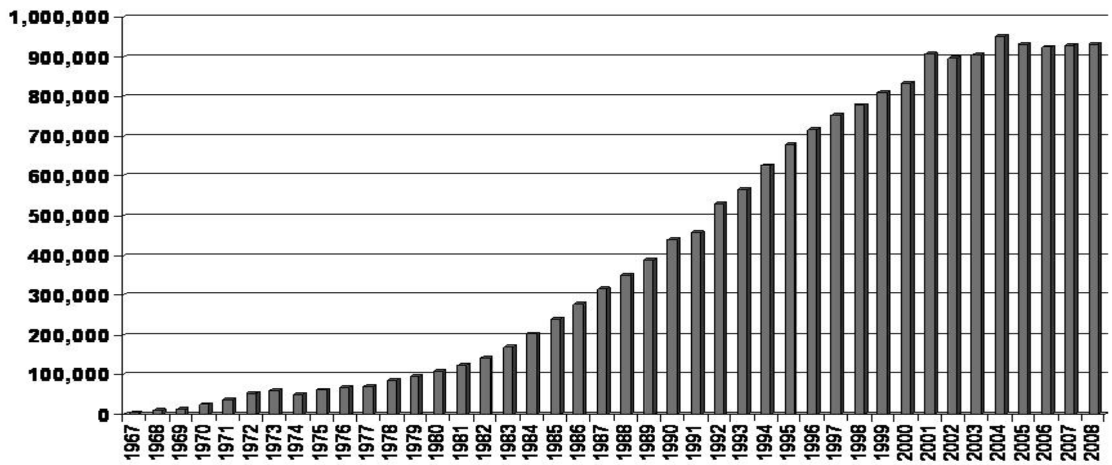

Figure 1. Total world entry level and continuing education diving certifications for all PADI (**P**rofessional **A**ssociation of **D**iving **I**nstructors) Offices combined; divers may have multiple certifications (9)

Improper use or lack of knowledge lead to problems - diving with pure oxygen. In hyperbaric conditions (conditions of increased pressure), oxygen changes its effect on the organism. From a gas of vital necessity at normal pressure, it turns into a toxic gas below a particular depth (13 m if breathing pure oxygen – 100% purity, and 105 m if breathing air) and stay of 20 minutes. In such case, there occurs oxygen toxicity which develops rapidly. It is assumed that hyperbaric oxygen destroys some key processes in the nerve cells. This results in overexcitation in the central nervous system leading to convulsions and death. Because pure oxygen is toxic below 13 m, it is obligatory that the percentage of oxygen in deeper dives be decreased. For example, the breathing mixture of dives below 80 meters consists of 90/95 % helium and 10/5 % oxygen respectively. The small percentage of oxygen is compensated by its raised partial pressure which normally ranges from 120 mm Hg to 350 mm Hg (tables I-III and figure 2) (8).

- 7 m – safe depth for diving with pure oxygen
- 13 m – the limit of diving with pure oxygen
- 40 m – danger of nitrogen narcosis
- 60 m – the limit of diving with compressed air; diving can continue with a cocktail
- 350 m – danger of HPNS.

Decompression

Air consists of nitrogen and oxygen. One part of oxygen is consumed and another is replaced by carbon dioxide. That is why the gas that concerns diving and decompression most is nitrogen.

Nitrogen is an inert gas in the organism. It does not take part in the exchange of substances and respiratory processes. There is about 1 liter of nitrogen which is dissolved in fluids and cells. During compression, nitrogen is intensively absorbed by the blood and dissolved in the organism until the partial pressure inside and the ambient pressures are equalized (saturation). During decompression, the reverse process takes place. Because of the decrease in water pressure with ascent, the partial pressure of nitrogen in the organism becomes higher and the gas is released by the cells and tissues (desaturation).

Table I. Clinical aspects of diving with pure oxygen

Diving with pure oxygen			
Disease	**Cause**	**Symptomes**	**Treatment**
Hypoxia (lack of oxygen); the partial pressure of oxygen falls below 120 mm Hg	Improper handling of pure-oxygen apparatus; If oxygen is not 100 % pure because of a mistake, nitrogen will remain after the consumption of O2. The diver cannot realize the difference and loses consciousness.	Good mood, jauntiness without reason; Later – dizziness, suffocation, blackout	The patient should be given pure oxygen to breathe; artificial respiration, outer irritation (slaps, amonia under the nose)
Hyperoxia(oxygen excess); partial pressure of oxygen exceeds 360 mm Hg	7 m – the process of receiving O2 and releasing CO2 is hindered; 13 m – this process stops so that cells cannot accept oxygen despite of its abundance;	at first – discomfort, nausea, spasms of lips and eyelids, disturbances in eyesight, sleepiness; later – stiffness of muscles, convulsions (similar to the epileptic ones), blackout, death	The diver should be taken out to breathe fresh air.
Hypercapnia (CO_2 toxicity) - the partial pressure of CO_2 is higher than normal;	Improper use of pure-oxygen-apparatus; not working absorber	Rapid pulse rate, shortness of breath and rapid heart beat, rapid respiration, headache, blackout	Artificial respiration; Access to fresh air;

Table II. Clinical aspects of diving with compressed air

Diving with compressed air			
Disease	**Cause**	**Symptomes**	**Treatment**
Nitrogen narcosis Disturbances which occur diving with compressed air at a depth of over 4 at	It is assumed that at increased pressure, N connects with body's fats, thus destroying the normal physiological functions of the cell and leading to disturbances in the central nervous system. Factors – stress, heavy work and CO2 retention.	First phase – "alcoholic intoxication", euphoria – jauntiness and high spirits. Second phase – fatigue, sleepiness, loss of consciousness and drowning	Go back several meters and there will be nothing left of this disease.
CO toxicity. It is not a common disease because carbon monoxide is not found in the at-mosphere.	Carelessness and lack of knowledge during the compre-ssion of air in bottles.	Headache, noise in the ears, dizziness; Later– shortness of breath, loss of consciousness, death	Access to fresh air; artificial respiration

Table III. Clinical aspects of diving with gas mixtures

Diving with gas mixtures			
Disease	**Cause**	**Symptomes**	**Treatment**
HPNS (High pressure nervous syndrome);	This is due to overexcitement of the central nervous system caused by the increased water pressure. It occurs at depths exceeding 350 meters.	Dizziness, nausea, tre-mors, loss of dexterity and memory	Prevention is the best treatment. Stage compression with long pauses; Add N to the breathing mixture to become trimix (He, O2, N)

Table IV. Specific medical contraindication for diving

Contraindication	Specific Examples or Adverse Effects
Lung disorder	Active asthma, COPD, cystic fibrosis, bronchiectasis, interstitial lung disease, history of spontaneous pneumothorax
Cardiovascular disorder	History of significant ventricular arrhythmias, intracardiac shunt, heart failure, significant coronary artery disease
Psychologic disorder	Panic or phobia
Structural disorder	Unrepaired inguinal hernia
Neurologic disorder	Seizure disorder, syncope
Metabolic disorder	Insulindependent diabetes mellitus, gross obesity
No equilibration of gases between body compartments	Lung cysts, perforated tympanic membrane, upper respiratory infection, allergic rhinitis
Pregnancy	Increase in incidence of birth defects and fetal death
Habitual air-swallowing	Gastro-intestinal overinflation during ascent due to swallowing pressurized air at depth
Poor exercise tolerance	—
Severe gastroesophageal reflux	Aggravated by loss of gravity effect on abdomen when submerged
Children under age of 10 years	—

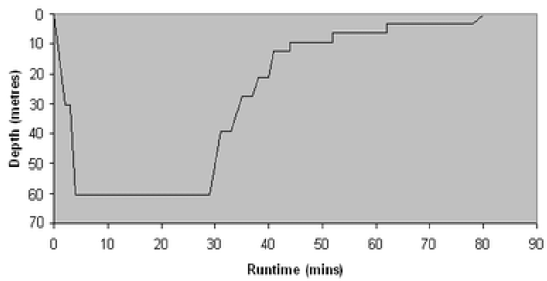

Figure 2. Sample Dive Profile of a 25 minute dive to 60m breathing Trimix showing decompression stops from 39m to 3m on Nitrox and Oxygen

This principle concerns helium, hydrogen and other inert gases too. The speed of desaturation is a major problem in diving medicine. If the diver ascends too fast and before complete desaturation, nitrogen begins to form bubbles in the organism. These bubbles are very dangerous because they can plug small arteries and cause serious damage by stopping the blood flow to a particular organ. From its first observation by the French Physiologist Paul Bert in 1878 through to Haldane's 1905 research, Bühlmann's 1990's research, and the latest RGBM work by Bruce Weinke at Los Alamos Labs in the USA - A key feature of them all is to attempt to accurately predict the human body's response to working at depth and then ascending to the surface

The earliest tables were entirely empirical, based on measurements on what caused a military diver pain.

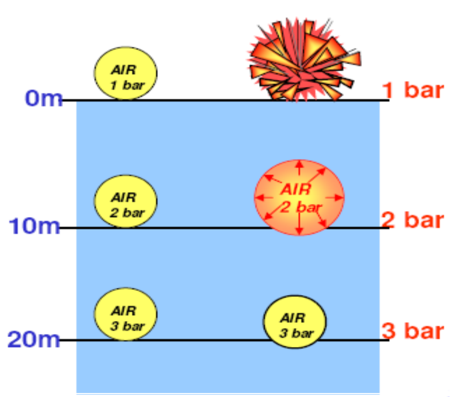

Figure 3. The air expansion

Navy divers are notoriously tough, so one can imagine what pain level had to be reached before they complained of being 'bent'. It's not the sort of pain level that you and I would like to endure on every recreational dive.

The first serious attempt at reducing the pain threshold or 'barotrauma insult' to the body to sub-clinical levels by actual measurements of bubbles flowing around the body was produced by Buhlmann. His vast database of bubble grade measurements from divers enduring hundreds of hours of chamber dives with mixed gases produced a massive table of 'half times' and 'm-values' The introduction of Buhlmann tables derived from these measurements still represents a watershed in decompression research, and marked a move away from 'pain based' research.

Further work still on newer models is proceeding with an almost entirely mathematical approach. The use of animals for medical research is well known. Early deco research used goats in hyperbaric chambers.

A hundred years ago, 'tech diving hungry goats' were taken for simulated deep dives in a chamber and subsequently decom-pressed 'to the surface' to test table limits. If the goat walked out of the chamber following the dive, it was assumed to be OK. If it couldn't or wouldn't get up to walk out, it was assumed to be bent. Later work within the last few years has used salmon or even gel samples in Petri dishes to research deco tables. The test of any good deco theory is how well it performs against the known database of the human body's tolerance to pressure changes. No matter how elegant the theory, if divers get hurt applying it's predictions, it is of questionable use for its intended purpose.

Effect of repeat divings

Divers are expected to do a work shift each day which means that excursions are repeated with approximately 16 hours between each. Any bubbles remaining from the previous excursion would be compressed on the return to the deeper depth and the gas leaving the bubbles would add to the gas load in solution. When the previous excursion has lasted 8 hours and the second excursion is to the same depth, for the same time, the gas load at the end of the second excursion, in all except the slowest tissues, is the same as that for the first excursion because most tissues are saturated by an 8 hour excursion. The exception is the slowest tissue, the fat. The first 8 hour excursion brings this tissue to 99.81% saturation whereas the addition of the remaining gas load to the uptake of the second excursion brings it to 99.96% saturation. This results in an increase in gas in bubbles, following the second excursion, of less than 0.02% The difference between first and second excursions, the risk of build up of gas as the number of excursions increase, would be greater than quoted above if the excursion duration were less than 8 hours. However the gas levels can only increase to saturation so the gas in bubbles will not go above the values quoted in this report by more than 0.02% (3).

Diving accidents are caused, most often, unknowledging thorough pressure effects on the human body (figure 3) and the rules of diving off of what these effects, insufficient knowledge of the equipment they have, improvisation equipment and procedures, overestimation opportunities that are available during the performance of diving, unknowledging environment and lack of exercise and training.

Diving accidents can be classified into four main groups: Accident due to mechanical effects of pressure, known physical and mechanical injury (pulmonary overpressure, barotrauma and diver`s colics);

- Accident due to the effects of biophysics pressure called accidents biophysics;
- Accident due to biochemical effects of pressure, called accidents biochemical (nitrogen narcosis, hiperoxic crisis, intoxication with carbon dioxide and hypoxia)

Physical and mechanical accidents are accidents due to mechanical effects of pressure and primarily affects the body cavities and organs in them; lungs, ear average, sinus and the digestive tract, with the question of variations of gas volume in body cavities, determined variations in pressure (9).

The following are general elements of accidents that may occur in sinking air:

A. Pulmonary overpressure

Pulmonary overpressure (figure 3) is a diving accident that occurs due to gas expansion blocked in the lungs over the limit of their elasticity, while raising the water surface.

Although sinking autonomous rule is maintaining a continuous game of breathe in and breathe out during surface climbing, it is not respected in the most extreme cases, when panic overtakes the diver unschooled. Despite all signes some divers stop breathing, preventing exhaling (in some cases on a voluntary basis, in other cases the reflex by a spasm of the glottis caused by fear), which lead to rapid growth in the volume of gas contained in the lungs once with decreased pressure (depth). Continuing to surface water boarding, the gas into the lungs increase so much that exceed the maximum amount of lung tissue and beating the limit of their elasticity. This fact leads to breaking and the appearance among others of a pneumotorax spontaneous, often suffocating and gas embolism into the pulmonary circulation. Accident is the most serious as the blocking exhale is closer to the water surface, where the decrease of depth lead to large variations in volume. The consequences of pulmonary overpressure are:

- *Barotrauma*

Barotrauma is tissue injury caused by a change in pressure, which compresses or expands gas contained in various body structures. Barotrauma refers to injury sustained from failure to equalize the pressure of an air-containing space with that of the surrounding environment. The most common examples of barotrauma occur in air travel and scuba diving. Although the degree of pressure changes is much more dramatic during scuba diving, barotraumatic injury is possible during air travel. Barotrauma can affect several different areas of the body, including the ear, face and lungs.

Here we will focus on barotrauma as it relates to the ear.

Symptoms of barotrauma include "clogging" of the ear, ear pain, hearing loss, dizziness, ringing of the ear (tinnitus), and hemorrhage from the ear.

Dizziness (or vertigo) may also occur during diving from a phenomenon known as alternobaric vertigo. It is caused by a difference in pressure between the two middle ear spaces, which stimulates the vestibular (balance) end organs asymmetrically, thus resulting in vertigo. The alternobaric response can also be elicited by forcefully equalizing the middle ear pressure with the Politzer maneuver, which can cause an unequal inflation of the middle ear space.

Lung over-expansion injury falls under the category of Decompression Illness in diving medical terms. Lung over-expansion injury can also occur in pilots on assisted breathing, and patients on a ventilator or respirator. Lung over-expansion injury occurs when a SCUBA diver holds their breath when ascending underwater. This can be as little as 1.5 meter ascents near the surface. When using SCUBA equipment underwater, the diver is breathing compressed air at a pressure equal to that of the water around him/her. This means the air entering their lungs is at the same pressure as the water. When the diver ascends, according to Boyle's Law, the air expands due to reducing hydrostatic pressure, (the shallower the depth, the less the pressure) causing the lungs to over-inflate.

The lung does not burst like a balloon when over-inflated, instead it tears. However the tear itself is not so much a problem as the resulting air entering the tissues and bloodstream. The result of such a rupture causes the pulmonary capillaries and alveoli to rupture mixing blood and air into the lungs usually causing the diver to cough up blood (a definite alert sign).

The rupture can also cause one of four injuries:

1. air embolism
2. pneumothorax
3. mediastinal emphysema
4. subcutaneous emphysema.

Air embolism

Increased pressure outside the body is transmitted equally throughout the blood and body tissues, which do not compress because they are composed mainly of liquid. Thus, a person's leg, for example, does not feel squeezed as water pressure increases. However, gases (such as the air inside the lungs, sinuses, or middle ear or inside a face mask or goggles) compress or expand as outside pressure increases or decreases. This compression and expansion can cause pain and damage to tissue.

Ear and Sinus

Ear barotrauma is the most common injury in divers. On descent, failure to equalize pressure changes within the middle ear space creates a pressure gradient across the eardrum, which can cause bleeding or fluid accumulation in the middle ear, as well as stretching or rupture of the eardrum and the membranes covering the windows of the inner ear (figure 5).

Pulmonary

Overinflation of the lungs can result as a scuba diver ascends toward the surface without exhaling. During ascent, compressed gas trapped in the lung increases in volume until the expansion exceeds the elastic limit of lung tissue, causing damage and allowing gas bubbles to escape into one or more of three possible locations: 1) Gas entering the pleural space can cause lung collapse or pneumothorax; 2) Gas entering the mediastinum (space around the heart, trachea and esophagus) causes mediastinal emphysema and frequently tracks under the skin (subcutaneous emphysema) or into the tissue around the larynx, precipitating a change in the voice characteristics; 3) Gas rupturing the alveolar walls can dissect into the pulmonary capillaries and pass via the pulmonary veins to the left side of the heart, where it is distributed according to relative blood flow, resulting in arterial gas embolism (AGE) (2).

While mediastinal or subcutaneous emphysema usually resolves spontaneously, pneumothorax may require specific treatment to remove the air and reinflate the lung. AGE is a medical emergency requiring appropriate intervention, which may include recompression treatment (see below).

Symptoms can include pain, ringing in the ear, vertigo, a sensation of fullness or effusion within the ear, and decreased hearing. Paranasal sinuses, because of their relatively narrow connecting passageways, are uniquely susceptible to barotraumas, generally on descent. With small changes in pressure (depth), symptoms are usually mild and short lived, but can be exacerbated by continued diving. Larger pressure changes, especially with forceful attempts at equilibration (e.g., valsalva maneuver), can be more injurious. Additional risk factors for ear and sinus barotrauma include earplugs, medications, ear and/or sinus surgery, nasal deformity and disease (1).

A diver who may have sustained ear and/or sinus barotrauma should discontinue diving and seek medical attention. Lung overinflation injuries from scuba diving can range from dramatic and life threatening to mild symptoms of chest pain and dyspnea. Although pulmonary barotrauma is relatively uncommon, prompt medical evaluation is necessary, and evidence for this condition should always be considered in the presence of respiratory or neurologic symptoms following a dive.

A *pneumothorax* is the presence of air between the two layers of pleura, resulting in partial or complete collapse of the lung.

Normally, the pressure in the pleural space is lower than that inside the lungs or outside the chest. If a perforation develops that causes a connection between the pleural space and the inside of the lungs or outside the chest, air enters the pleural space until the pressures become equal or the connection closes. When there is air in the pleural space, the lung partially collapses. Sometimes most or all of the lung collapses, leading to severe shortness of breath. A pneumothorax that occurs without any apparent cause in people without a known lung disorder is called a primary spontaneous pneumothorax. Primary spontaneous pneumothorax usually occurs when a small weakened area of lung (bulla) ruptures.

In a *mediastinal emphysema* or *pneumo mediastinum* air accumulates in the *mediastinum.* This is the center of the chest between the lungs where the heart resides. Air may press on the heart and major arteries, interfering with circulation. Symptoms are feeling faint and feeling short of breath. However, mediastinal emphysema is far less serious than AGE or pneumothorax.

Figure 4. "You must see me" sign - for attention capture between divers

Figure 5. Pressure equilibration on eardrum

In a *subcutaneous emphysema* air accumulates under the skin in the neck (subcutane means 'under the skin'). Often subcutaneous emphysema results from a mediastinal emphysema, when air seeks its way out of the mediastinum. The way of least resistance is the way into the soft tissue at the base of the neck. Victims experience a fullness of the neck and a change of voice. An odd symptom may be a cracking sound when touching the skin of the neck. In the first table (fig. 6) is a diving after which were small signs of a decompression accident and the second table (fig. 7) is a diving which has not needed a special decompress or to stop in levels.

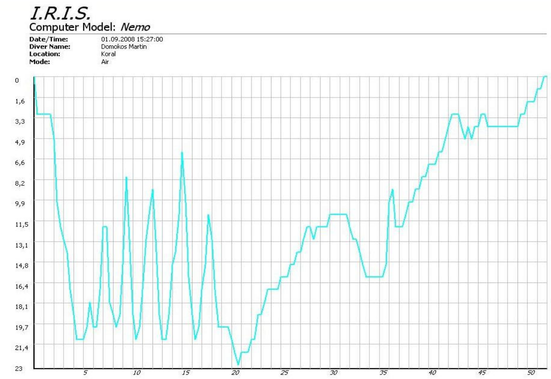

Figure 6. Diving with decompresion`s accident

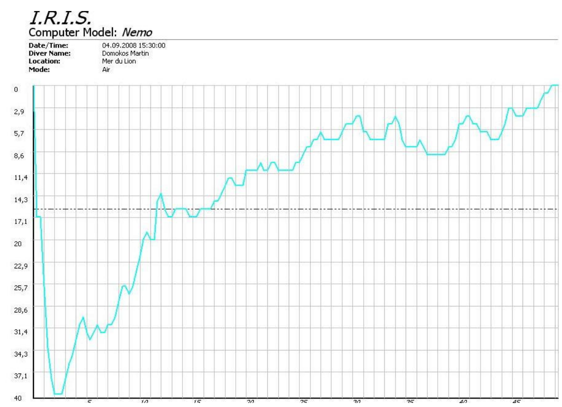

Figure 7. Diving without decompresion`s accident

The mathematical analysis results in the conclusion that the excursions studied, though causing bubbles, are unlikely to cause either detectable bubbles or DCI in the average diver and that detectable bubbles might occur at a level of a few divers in a thousand. The results also show that decompression from saturation is unlikely to cause any bubbles in the average diver though something like 1 in a 1000 may have detectable bubbles. DCI is likely to be a rare event following either an excursion or a decompression from saturation. The risk increases considerably when an excursion is followed by decompression; when a decompression is preceded by an excursion.

This is a significant finding because over the years excursions have been seen as the main cause of problems and as a result depth changes have been restricted. Similarly over the years changes have been made to decompression schedules, in general slowing down the rate of change of pressure in an attempt to reduce the DCI rate, without reference to the fact that the trouble is more likely to have originated with the preceding excursions. This study has shown that excursions and decompression must be considered together. This introduces the possibility that by considering excursions and decompressions together changes can be made which will reduce risk without increasing operational costs. We observe that not the time of diving (which was identical) or maximum depth (which was almost double of the second diving) have led to the problem but repeated and uncontrolled climbings. In conclusion, if during your return to the surface area is taking into account of all factors of safety, the diver can fully enjoy the sensations offered by diving without suffering serious illnesses of the body.

The main conclusion from the work is that neither the excursions currently used nor the decompression procedures are likely to cause decompression problems. The risks result from the combination of excursions followed by decompression before bubbles have totally resolved. Starting decompression whilst bubbles are present is the single significant factor.

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