APNEA

Apnea means "suspension of breath", is the most instinctive and immediate way to go underwater, using air stored in the lungs.
While diving in apnea (no breathing), the circulatory and respiratory systems modify their operation to fit the new condition.
This adaptation is already evident with the simple immersion of the body in the water, in fact the heart rate drops considerably.

APNEA AND GAS EXCHANGE

As we know, the atmospheric air that we breathe when diving in apnea, is composed of 78% of N2 (nitrogen) to 21% O2 (oxygen) and 1% other gases including CO2 (carbon dioxide) for the 0.03%. In the pulmonary alveolus, because of gas exchange, these percentages are different, in fact, the O2, this 21% in arterial blood, drops to 15% in the venous. CO2 instead varies from 5.1% in arterial blood to the 5.6% in the venous circulation, while the N2 (inert, not used in any way), remains the same percentage.

APNEA AND HYPOXIA

With the breath act, the air present in the pulmonary alveolus, is constantly ventilated.
In apnea diving, with no breath acts, the air in the lungs is the initial one, so that gas exchange in the lung alveolus continue to be ever present, but by exploiting the same air, in particular O2 continues to be withdrawn, while the CO2 continues to increase.

When the O2 concentration falls below 10%, the metabolic processes are no longer guaranteed and will enter into **hypoxia** (low oxygen).
As said above, being the brain the organ most sensitive to hypoxia, there is loss of consciousness **(syncope),** if this condition continues there will be serious consequences for the brain, until death.

APNEA AND HYPERCAPNIA

As for CO2, the excessive increase is called **hypercapnia** (excess carbon dioxide) that, **at concentrations of 7%, it causes "air hunger,"** and warns the chemotactic bulbar centers that control respiratory muscles, which in turn cause spasmodic contractions of the musculature, and in particular of the diaphragm.
Around 8% of CO2 we have: obstacle to the functioning of the heart, lower muscular and mental efficiency, disappearance of diaphragmatic contractions (as the bulbar centers, first stimulated by CO2, with higher concentrations, are desensitized).
Around 10-11% of CO2, we have: loss of consciousness, block of the functioning of the bulbar centers resulting in respiratory paralysis, cardiac arrest and death.

Limit of the apnea: as seen above the apnea duration **depends on two basic points, which are hypoxia and hypercapnia** (which establish the duration of apnea) **and by several other factors, said complementary** (which modify the duration of the apnea).

KEY FACTORS:
Hypoxia: lack of oxygen to below 10%, there are no symptoms or warning signals;
Hypercapnia: too much CO2 in the blood, around 7% trigger air hunger with symptoms and warning signs as "diaphragmatic contractions", which disappear soon after this limit is exceeded.

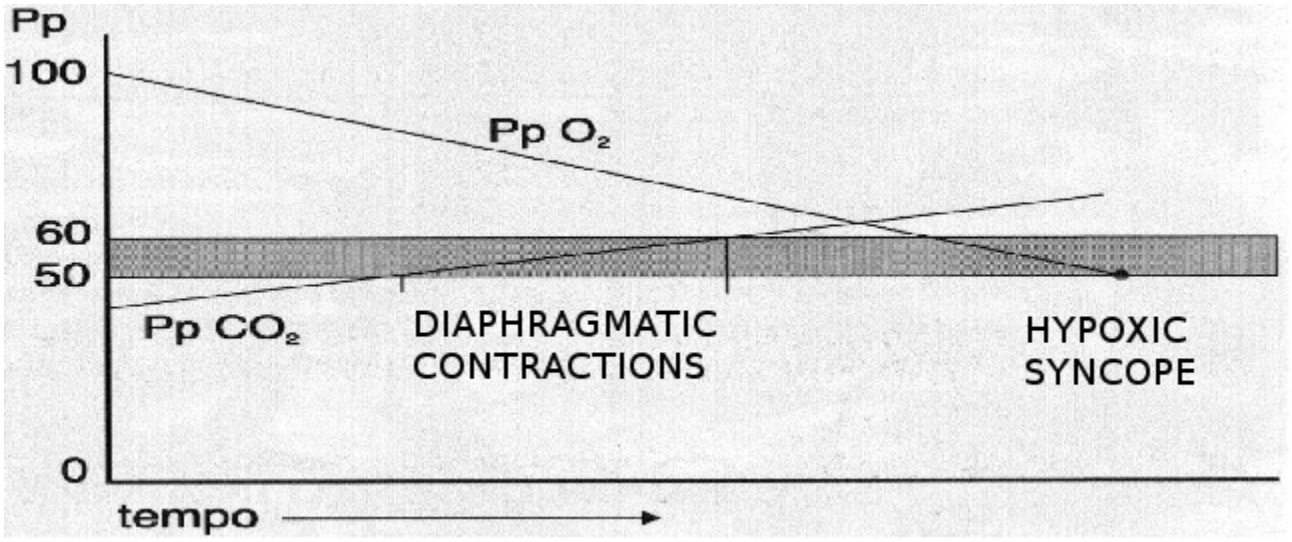
ADDITIONAL FACTORS:
Vital capacity: is different from subject to subject. The greater, the greater the duration of the apnea;
Physical activity: Intended as efforts and movement in the water (the movement increases the consumption of O2 and produces more CO2);
Environmental situation: the cold increases the consumption of O2;
Psychological condition: it is the most important complementary factors, **Anxiety and Fear** are huge obstacles to the duration of apnea;
Depth: the deeper you go, the greater the consumption of O2 and the greater the concentration of CO2;
Hyperventilation: It change the balance between O2 consumption and CO2 production, by changing the concentration of these gases in the blood.

HYPERVENTILATION

Hyperventilate means to breathe deeper and faster. During normal ventilation a volume of about 8 liters of air passes through the lungs every minute and this quantity can be increased in two ways:
1) Increasing the volume of air ventilated: By breathing out to the max and inspiring to the maximum (in doing so you make the lung lavage);
2) Increasing the frequency of breathing: Not efficient because performing this procedure involves a greater muscular effort resulting in consumption of O2 and CO2 increase;
3) With both methods.
In practice hyperventilating it completely eliminates the presence of CO2 from the air contained in the lungs, and then from the tissue, with a consequent reduction of its partial pressure. This does not result in a greater absorption of O2, and it effects on the O2 Pp in the blood are negligible.

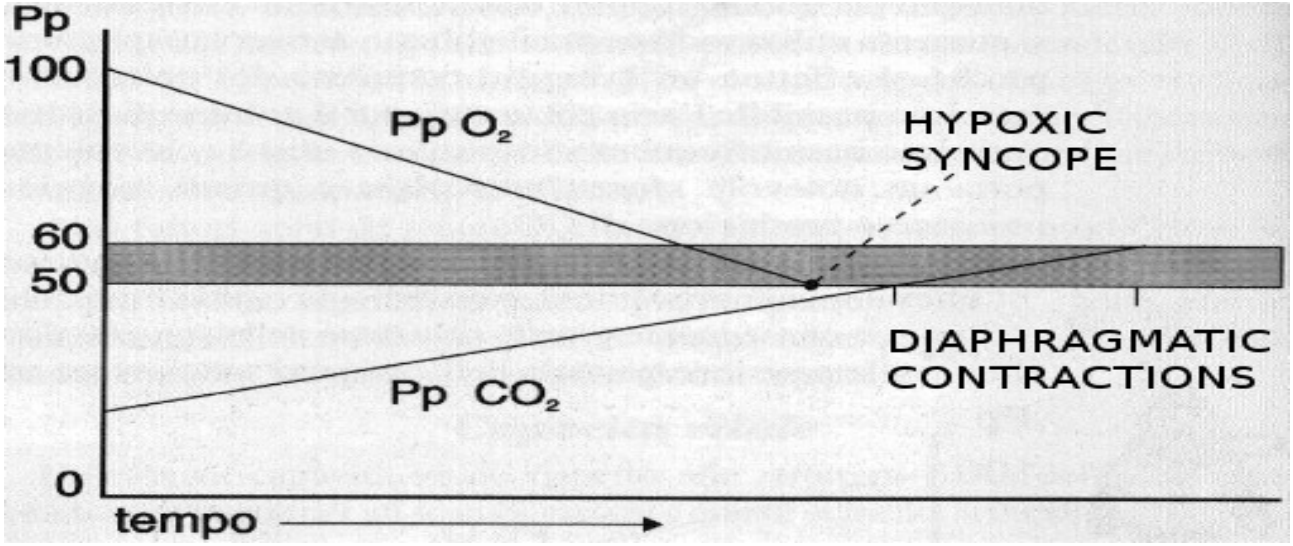
WHY HYPERVENTILATING INCREASES APNEA TIME?

Hyperventilating increases the length of apnea because, having performed the complete washing of CO2, **the appearance of hypercapnia (concentration 7%), is delayed.**
Nell'apnea practiced without hyperventilation, increasing the concentration of CO2, which starts from normal levels, reaches the concentration of 7% and determines the appearance of the diaphragmatic contractions (stimulus to breathe), before the O2 concentration is decreased to a dangerous level (syncope). Hypercapnia is the limiting factor.



APNEA PRECEDED BY HYPERVENTILATION

In the apnea preceded by hyperventilation, having cleaned your lungs completely from the presence of CO2, before reaching the concentration of 7% CO2 (with air hunger), the Pp of O2 is lowered, until the onset of syncope, which is followed by rising CO2 concentration till, reached 7%, triggers diaphragmatic contractions and the subject (being unconscious), performs the actual breaths underwater, with consequent entry of water into the lungs. So **the limiting factor is hypoxia.** This drawback is, however, only present after excessive hyperventilation, which also cause the appearance of: dizziness, tingling in the lips or hands, blurred vision caused by vasoconstriction (narrowing) of the brain caused by hypocapnia (low concentration of CO2, as a result of excessive lung lavage.) **At the onset of these symptoms you should immediately stop hyperventilation.**



BEHAVIOUR IN APNEA

The dive time is the most natural and immediate approach to diving, and the only way permitted by law to perform underwater fishing.

Accidents in apnea are rare but can be deadly.

Devote great care to the equipment: the suit is essential (5 to 7 mm), if the water is so cold as to require wetsuits thicker it is advisable not to dive in apnea unless well trained.

Ballast slightly positive on the surface for deep apnea, and neutral or slightly negative for diving in shallow waters.

Anklets ballasted, for better balancing on the surface and to increase the effectiveness of the finning.

Mask internal volume should be as small as possible, for easy compensation.

Fins with long blade narrow and flexible, giving the maximum thrust possible in case of need.

Knife fixed in position easy to reach (better if with both hands).

Float with “Alpha” flag (the latter is required by law), to indicate the presence of the diver, also serves as a float to hang the equipment (rifle, light, fish stringer etc.) and to hold to in case of fatigue or accident.

Apnea is practiced in pairs, in turn a diver plunges and the other follow him from the surface: the second diver dives only after the friend as started to breathe normally.

Hyperventilation should be no more than three deep breaths.

In deep diving spit the mouthpiece immediately after the upside down, so as to ease breathing once resurfaced, without having to empty the snorkel.

The compensation of the mask and ear, must be decided and accurate, stop the descent in the event of incomplete compensation.

On the bottom reduce the movements to a minimum so as to diminish the consumption of O2

At the first contraction in the diaphragm immediately back to the surface looking up for not "clash" under the boat or other obstacles.

Between a dive and the other has to pass a time long enough to bring the values of CO2 and O2 at normal levels, if the time is too short it will establish a progressive hypercapnia that occurs with hyperacidity (cramps), fatigue, decreased performance.

ADAPTATION of the cardiovascular

Essentially two: 1st Bradycardia and 2nd Blood Shift.

Bradycardia: when we immerse our face in water, to perform immersion in apnea, a reflex occurs with reduction of the heart rate, said "bradycardia" (slow heart), in fact the heartbeat rate at surface is about 65 beats per minute, at a depth of 40 m. is reduced to 40 beats per minute, at 55 mt become 36 beats per minute, and so on. In practice by reducing the heart rate, the body reduces the consumption of O2, for the benefit of the organs most sensitive to hypoxia. There is however the risk that a marked bradycardia (caused by cold or sudden illness), cause a too low flow of blood to the brain with the risk of syncope.

Blood Shift: After a certain depth (- 50 mt.), occurs the collapse of the lungs, this thought arose from an examination of the law of Boyle and Mariotte: if the lung capacity of a diver is about 6 lt. at 50 m (6 atm.), this capacity becomes 1/6 or 6:6 = 1 lt., which is lower than the residual volume of the lungs of 1.350 lt., from here the theoretical collapse of the lungs.
In practice it is not so (just think of the record apnea, far beyond 50 mt.).
In fact, after the 20 mt. begins the phenomenon of Blood Shift, that consists in the ability of the organism, to move the greatest amount of blood in the lungs, so as to make it occupy part of the space present in the lungs, and the blood being incompressible, prevents the crushing of the lungs by the hydrostatic pressure.

But why the blood moves into the lungs?: The Blood Shift occurs for two reasons:
The first reason is due to the fact that the pulmonary vessels can dilate with ease, thus allowing blood to penetrate more easily compared to the vessels of large blood circulation;
The second reason relates, instead, the small depression that is created as a result of the crushing of the rib cage to its maximum allowed, depression that draws blood into the lungs.
The brain, being delivered of blood in a direct way, receives the necessary blood all the same.

CHANGE OF OXYGEN CONSUMPTION

During the immersion in apnea: the consumption of oxygen by the body, increases, and consequently increases the extraction of O2 from air in the lungs forfeited before dive. In practice, with the depth (according to Boyle's law), decreases the volume and increases the pressure, for which (according to the law of Dalton) increasing the total pressure, consequently increases the partial pressure of O2.
It 'important to remember that on the surface the partial pressure of O2 is 159 mm Hg. (Data value from 21% O2 x height 760 mm of mercury (Hg.), or 0.21 x 760 = 159 mm Hg (see the principle of Torricelli) **and the limit in which the O2 is fixed in the blood** (thanks to Henry's Law) **and of 100 mm Hg, below which, within the maximum limit of 60 mm Hg. incurs asphyxia . For the foregoing that we will have a depth of - 30 m. (4 atm.) The Pp. O2 is of 636 mm Hg., value** given by: 4 atm. x 159 mm Hg. (21% of 760 mm Hg.) = 636 mm Hg., **Far superior to 159 mm Hg. the surface, this example suggests how deep gas exchange is facilitated (Boyle's Law + + Henry + Dalton).**

WHY 'THE SYNCOPE PRESENTED IN THE LAST METERS before surfacing?

While staying at the bottom: the skin diving, may consume O2 to Pp. limit of 100 mm Hg., this value, beyond which is no longer guaranteed gas exchange.
An ascent though: the pressure that we have in our body decrease (and for Boyle's law), there will be a consequent increase in the volume of air the lungs.

This decrease of the total pressure, will consequently reduce the Pp O2, which reached a value of 100 mm Hg.

Let's take as example one dive to 30m where the Pp O2 reached 100 mm Hg:

During the descent, already at around 10 m. (2 ata), the Pp of O2 goes from 100 mm Hg (residues to 30 mt./4 ata), to 50 mm Hg present at 10 mt. (2 ata).

That residue of Pp of O2, as one can easily guess, is no longer able to guarantee the gaseous exchanges, therefore, the diver incurs the syncope (this explains why most of the times, when the apnea is "pulled" to the maximum, the syncope is presents around the last meters from resurfacing).

ACCIDENT TO APNEA

The most serious incident that can meet the diver is Syncope.

Syncope: is a loss of consciousness, respiratory arrest and then cardiac arrest.
Cause: lack of cerebral oxygenation (hypoxia).

SYNCOPE PRIMARY AND SECONDARY

The primary syncope: derives from prolonged apnea and occurs when the concentration of O2 in the blood falls below 10%. Under these conditions, there is a "stop" of the functioning of nerve cells that remain in a state of metabolic block, which, if not quickly restored, become unrecoverable.

Syncope warning signs, and its severity depends on whether or not the apnea was preceded by hyperventilation.

The secondary syncope: is not typical of divers, is usually presented after a dip in the water, due to a difference in temperature between the body and the water.

SYNCOPE "Dry" And SYNCOPE "Wet"

Dry Syncope or Damp Syncope, are a further subdivision of Primary Syncope.

Syncope is dry: when there was not hyperventilation and the diaphragmatic contractions arrive before the syncope occur (in this case, even if rendered unconscious, there is no entry of water into the lungs), therefore, the subsequent death is asphyxiation.

Syncope is wet: when there has been hyperventilation, for which the diaphragmatic contractions occur after that the syncope happen (it follows that the diver, lost consciousness, because of the occurrence of diaphragmatic contractions, sucks water in the lungs), therefore, the subsequent Death is due to drowning.

For both syncope loss of consciousness is accompanied by respiratory arrest, while the heart continues to beat and the concentration of CO2 raise because it can not be expelled (because respiratory arrest), that gives the typical cyanotic color (bluish) to the syncopated.

WET SYNCOPE: In Fresh Water, Salt, Polluted.

The wet syncope in fresh water: (eg. pool) is more serious than in salt water because, this being less dense blood, is easily absorbed through the walls of the alveolus, by determining the dilution of the blood, and causing cardiac arrest.

Wet syncope in polluted water: the worst (swimming pool, river, lake), even in the absence of hypoxia (low O2 in the blood), the release of water in the lungs leads to serious infections with chemical damage often fatal.

Therefore, to perform apnea in safety, do NOT:

1) Perform iperventilazioni prolonged (3 deep breaths are more than enough);
2) "Pull" apnea, and diaphragmatic contractions (you only single alarm bell);
3) Diving without the presence of the buddy.

SYMPTOMS AND TREATMENT OF SYNCOPATED

Symptoms of syncopation:
1) Patient is motionless, unconscious;
2) Lack of breath;
3) Rigid body, with jaw contracted;
4) Dark bluish color.

Treatment:
1) Immediate CPR (cardiopulmonary resuscitation) or PR (pulmonary resuscitation);
2) when possible administration of 100% O2.

In the case in which the coloring of the syncopated is pale and whitish, with dilated pupils, without reaction in the presence of light, means that in addition to respiratory arrest there is also a heart arrest (and thus consequent suffering of the brain). Execute immediate cardiopulmonary resuscitation until the arrival of medical personnel.

Thanks to Roberto Brunetti, Instructor Sub and Diver of the Italian Navy for having written this document.