

## Shallow Water Blackout

*By Dr. Duke Scott*

Most of us began our diving experience as skin divers...mask, snorkel, fins, a body of water and a curious mind. No fancy equipment or cumbersome tanks were needed. We had total freedom, just another aquatic animal at one with the environment. We quickly learned to suppress our urge to breathe, thereby increasing our maximum time and depth underwater. I have to confess that breath-hold diving is still my favorite diving activity. It gives me a great deal of exercise and requires a considerable amount of skill. I often wonder in this era of high-tech diving, if we as instructors fail to emphasize breath-hold diving to our dive classes? Do we fail to teach the physics and physiology of such diving? Do we offer a thorough explanation of the potential dangers of hyperventilation and the resultant Shallow Water Blackout? In my estimation, we probably do not. Therefore, this article will give you a better understanding of breath-hold diving and its pitfalls.

Breath-holding diving (freediving) is used to describe the activity of skin divers when they hold their breath and dive underwater. The maximum amount of time an individual can hold his breath before safely resuming breathing is called breath-hold time. An individual's maximum breath-hold time is called the breaking point. Training, motivation and a thorough understanding of the physiology of breath-hold diving increase one's maximum breath-hold time.

There are anatomical, physiological and psychological factors that determine maximum breath-hold time.

1. Anatomical factors: An individual's lung volume greatly influences his ability to excel at breath-hold diving. This is genetically determined in each of us and is influenced by environmental factors such as smoking, asthma and chronic lung disease. The healthier the lung means the greater extent for utilizing lung volume.

2. Physiological factors: These factors revolve around the effects of changes in the partial pressure of oxygen (PO<sub>2</sub>) and carbon dioxide (PCO<sub>2</sub>) during breath-hold diving. They result from changes in ambient pressure, variations in metabolic rate and differences in gas-perfusion rates of the body's tissues. The primary stimulus for respiration is increasing PCO<sub>2</sub>, which stimulates the peripheral chemoreceptors located in the internal carotid arteries (carotid bodies) and in the wall of the aortic arch (aortic bodies). It is also the major stimulus of the chemoreceptors located in the base of the brain (medulla). Decreasing O<sub>2</sub> is a secondary stimulus to these chemoreceptors.

3. Psychological factors: The individual's will and training determine psychological factors. It depends on the diver's ability to learn to suppress his response to the urge to breathe, or his ability to ignore rising levels of PCO<sub>2</sub> in his arterial blood.

When we hold our breath on the surface (1 ATM), several simultaneous factors influence our urge to breathe. Initially, the partial pressure of the alveolar gases will approximately equal air at 1 ATM (PO<sub>2</sub> = .21, PN<sub>2</sub> = .79, and CO<sub>2</sub> = .003). Normal metabolism produces a decrease in PO<sub>2</sub> and an increase in PCO<sub>2</sub>. The volume of O<sub>2</sub> consumed essentially equals the volume of CO<sub>2</sub> produced. The amount of energy expended by the individual drives the rate of this production. The increasing PCO<sub>2</sub> and the decreasing PO<sub>2</sub> in the aveolus of the lung and subsequently in the arterial blood determines the breath-hold breaking point. By holding our breath, we allow CO<sub>2</sub> to build within our body. The initial values at the onset of breath holding are approximately 40 mmHg for PCO<sub>2</sub> and 100 mmHg for PO<sub>2</sub>. Once the PCO<sub>2</sub> in the arterial blood increases to 60 mmHg or the PO<sub>2</sub> drops to 30 mmHg, the chemoreceptors become stimulated and the breaking point is reached. But, with dropping PO<sub>2</sub> and increasing PCO<sub>2</sub> occurring simultaneously, a value of 50 mmHg for both creates a marked urge to breathe. In short, that urge is primarily triggered by hypercapnia (elevated levels of CO<sub>2</sub>) and by hypoxia (decreasing levels of O<sub>2</sub>). It

represents a synergistic relationship.

With this basic understanding of surface breath-hold physiology, we tackle the more complex interactions associated with breath-hold diving. Once the breath-hold diver begins his descent, the increasing ambient pressure compresses the lung and thereby increases the partial pressure of the alveolar gases. These effects are clearly defined by Boyle's and Dalton's Laws. The increasing ambient pressure effects the lung by decreasing its volume, as previously discussed. This causes an increase in the partial pressure of the alveolar gases - primarily O<sub>2</sub>, CO<sub>2</sub> and N<sub>2</sub>. Understanding the effect this has on breath-hold divers is the major concern, so let's examine the components individually and how they interact.

Increasing CO<sub>2</sub> is the main source causing the urge to breathe during descent. With oxygen consumption, there is an equal production of CO<sub>2</sub>. During breath-hold diving, CO<sub>2</sub> builds up, and as the ambient pressure increases, the PCO<sub>2</sub> in the aveolus increases to the point that the normal outward perfusion of CO<sub>2</sub> is reversed and CO<sub>2</sub> is driven into the body's tissue and fluids. But, because of the increased solubility of CO<sub>2</sub> in these tissues, the increase of the PCO<sub>2</sub> in the alveolar and the arterial blood is not as great or as rapid as expected. Dr. Suk-Ki Hong showed that during a typical breath-hold dive to 30 feet, the PCO<sub>2</sub> increased from 29 mmHg upon leaving the surface to a PCO<sub>2</sub> of 42 mmHg on reaching the bottom. This is far below the required PCO<sub>2</sub> of 50-60 mmHg needed to trigger the urge-to-breathe mechanism. On the surface the PCO<sub>2</sub> increases more rapidly and triggers the mechanism more quickly. As the dive continues and more energy expends, the PCO<sub>2</sub> increases. When the breaking point is reached, the diver is alerted to begin his ascent. Once the diver begins his ascent, the ambient pressure decreases. With decompression the lung volume increases and the PCO<sub>2</sub> decreases. This initial decrease in PCO<sub>2</sub> gives the diver a momentary sense of well being and decreases his sense of urgency to reach the surface. But as the PCO<sub>2</sub> perfusion gradient reverses the CO<sub>2</sub> flows out of the tissues into the lung and again increases the urge to breathe. Dr. Hong showed that the diver has an average immediate post-dive PCO<sub>2</sub> of 42 mmHg. This demonstrates that although the elevated PCO<sub>2</sub> alerts the diver to ascend, its influence on his urge to breathe may actually diminish as he rises toward the surface.

The ambient pressure increases with depth. Correspondingly, the PO<sub>2</sub> in the aveolus steadily increases with depth. So, although the diver's oxygen store is consumed by metabolism, this decrease in oxygen is not correctly reflected. The increasing PO<sub>2</sub> in the aveolus and therefore in the arterial blood continues to provide adequate oxygenation of the brain and other vital organs as long as the diver remains at depth or deeper. Of course, this depends on the fact that he does not completely deplete his oxygen supply or reach a state of critical hypoxia (PO<sub>2</sub> ≤ .10ATM). The increasing PO<sub>2</sub> also suppresses the oxygen component of the urge to breathe. The deeper the diver goes, the greater the arterial PO<sub>2</sub> and the longer the urge to breathe are suppressed. This occurs despite the fact our diver is expending a considerable amount of energy and is quickly using his oxygen stores. Dr. Hong showed that during a typical breath-hold dive to 30 feet, the PO<sub>2</sub> increases from 120 mmHG to 149 mmHG on reaching the bottom. This is far above the oxygen breath-hold breaking point of 30 mmHG. So, obviously during descent, the PO<sub>2</sub> is not a factor in triggering the urge-to-breath mechanism. Upon ascent the ambient pressure steadily decreases. This causes an increase in lung volume and a decrease of the PO<sub>2</sub> in the aveolus and arterial blood. Also, our diver continues to consume oxygen. Therefore, as he ascends he develops a progressively sever hypoxia. So, during ascent the PO<sub>2</sub> becomes a major factor in the urge-to-breath equation. Our diver should have just enough oxygen to safely reach the surface. Obviously the degree of hypoxia is more profound than experienced during a comparable breath hold on the surface. Therefore, a good rule of thumb is never breath-hold dive longer than you can hold your breath on the surface.

Nitrogen is not a major player during breath-hold dives. Although the partial pressure of nitrogen (PN<sub>2</sub>) in the aveolus is slightly increased, the amount transferred from the alveoli to the circulation is comparatively small. Therefore, the danger of developing decompression illness (DCI) is remote during any given breath-hold dive. Some investigators expounded on the potential danger of repetitive dives to great depths with only minimal surface intervals. They postulate this could allow the N<sub>2</sub> to build up sufficiently in the

diver's tissues to allow DCI development. This, in my opinion, is speculative and does not apply to the recreational breath-hold diver.

Now we have a concept of the forces that interact during a breath-hold dive. The main factor that determines how long an individual can stay underwater is his ability to suppress his response to the PCO<sub>2</sub> stimulus to breathe. Fortunately, most of us are not able to suppress that urge long enough to deplete our oxygen to the point of critical hypoxia. This is accomplished by vigorous pre-dive hyperventilation. Almost all breath-hold diving accidents are associated with such hyperventilation and the over-achiever mentality (the individual who always pushes the limits). Hyperventilation increases breath-holding time. During hyperventilation the rate of elimination of CO<sub>2</sub> from the body greatly increases, while the amount of O<sub>2</sub> in the lung essentially remains unchanged. Therefore, pre-breath-hold hyperventilation greatly reduces the amount of CO<sub>2</sub> in the aveolus and the arterial blood. This increases the time before the PCO<sub>2</sub> in the blood reaches the breath-hold breaking point. But, this extension of underwater time produces a further reduction of the PO<sub>2</sub> in the arterial blood, which reflects the severe reduction in oxygen stores. This endangers one's safe return to the surface. If the critical state of hypoxia is reached while the diver struggles to reach the surface, he may slip into unconsciousness and drown. Remember that severe hypoxia leading to unconsciousness and/or death can strike without any warning signs or symptoms. This is the Shallow Water Blackout.

In summary, let's introduce our SCUBA students to the fascinating and challenging world of snorkeling and breath-hold diving. Leave them with a list of dos and don'ts to ensure they pursue it safely.

**Don'ts:**

1. Never hyperventilate vigorously prior to breath-hold diving – no more than four slow and controlled breaths.
2. Never breath-hold dive alone.
3. Never exceed your ability, go deeper or stay down longer than your physical condition and training permits.
4. Never practice breath-hold diving in any body of water without a knowledgeable observer.
5. Never perform recreational breath-hold diving after inhaling pure oxygen, Nitrox I or II or any other gas, except air.
6. Never breath-hold dive in cold or murky water without sufficient, prior training.

**Dos:**

1. Always breath-hold dive with a buddy. One knowledgeable diver should be on the surface as an observer at all times.
2. Always fly a dive flag when breath-hold diving in open water.
3. Always limit pre-breath-hold dive hyperventilation to no more than four breaths and then fill the lung to approximately 80 percent lung volume.
4. Always avoid unnecessary or excessive exertion during breath-hold dives.
5. Always wear a flotation device.
6. Always review the danger of shallow water blackout as part of your pre-dive plan.
7. Always limit your maximum breath-hold dive time to no longer than your surface breath-hold time.

I have not discussed all the aspects of breath-hold diving. Some are still speculative and others pertain to the specialized sport of deep breath-hold diving. Included in this list are such things as the effects of immersion in water to the neck, cold water, face immersion, bradycardia, use of oxygen-enriched gases, peripheral vasoconstriction and the stretch receptors in the lung.

**Obtain these and other such data from:**

1. Hong, S.K., Cerretelli P., Cruz J.E., Ruhn H.: Mechanics of Respiration During Submersion In Water. J. Appl Physiol 27:537-538, 1969.

2. Blue Water Free Divers: 1-800-667-7462. Freedive! (\$45)
3. Local experts: Tec Clark and Glennon Gingo.

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