DIVER EDUCATION SERIES

Physiology of Breath-hold Diving

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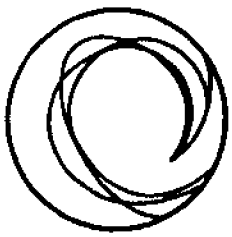

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PHYSIOLOGY OF BREATH-HOLD DIVING

Lee H. Somers, Ph.D.

From the beginning of recorded history man has attempted to extend the confines of his terrestrial environment. This is evident in Greek mythology. Records of human diving experiences are noted in Homer's Illiad and Herodotus' writings. Aquatic mammals such as the porpoise have been revered since Aristotle's time. However, Paul Bert in 1870 appears to be among the first to study diving vertebrates [1]. Irving's classical review in 1939 discussed many of the physiological adjustments that account for the extraordinary diving ability of some aquatic mammals [2]. Subsequently, it was recognized that humans, to a limited extent, could adapt to the aquatic environment.

The diving abilities of mammals reflect physiological adaptations and acclimatization. The most profound and generalized changes are observed in the cardiovascular and respiratory systems. Strauss presents an excellent review of these adaptive changes and their underlying physiological mechanisms [3]. Special emphasis is placed on the presence or absence of these adaptations in the human breath-hold diver. Much of the information presented here is summarized from Strauss.

Aquatic mammals exhibit multiple changes or adjustments in their respiratory system during dives. Several of these changes can also be observed in the conditioned human diver. The most notable respiratory changes in all aquatic mammals are: (1) adaptations to resist the effects of thoracic squeeze, (2) changes which reduce or eliminate the possibility of developing decompression sickness, (3) adaptations which make the organism less responsive to the effects of hypoxia and hypercapnia (carbon dioxide build-up), and (4) those changes which improve the efficiency of ventilation during the surface interval and recovery phase following a breath-hold dive.

The cardiovascular changes observed in aquatic mammals are exceptionally remarkable. In contrast to the respiratory adaptations which tend to be instrumental in protecting the mammals from problems associated with pressure, the cardiovascular changes are responsible for improving dive duration. The immersion bradycardia reflex, marked slowing of the heart, is the most well documented of these responses in all aquatic animals including humans. Bradycardia alone cannot account for the long dive durations observed in aquatic mammals. Bradycardia, in itself, would be detrimental to the organism. There are four major compensating factors which complement the bradycardia response. They are: (1) profound peripheral vasoconstriction and a preferential shunting of blood from the

extremities to the great vessels, heart, lungs, and brain; (2)morphological changes in the vascular system; (3) increased ability for the noncritical tissues to function anaerobically and remain isolated until the dive is completed; and (4) increased ability to carry, store, and utilize oxygen.

PULMONARY CHANGES DURING BREATH-HOLD DIVES

Adaptations to Resist Thoracic Barotrama

As a diver descends while holding his breath, the flexible portion of the thorax is compressed and the diaphragm elevated. Consequently, the air within the lungs and airways is compressed and the system assumes a more "expiratory" position. Classical diving physiology literature has stated that no difficulty is experienced until the position of maximal expiration is reached; then the volume of air equals the residual volume of the lungs plus the volume of the airways. Beyond this point, further descent while breath-holding may result in pulmonary congestion, edema, and hemorrhage in the lungs. The diver may experience a sensation of chest compression, breathing difficulties, and possible chest pain. Further descent may result in collapse of the chest wall [4]. This condition is generally called pulmonary barotrauma of descent or **thoracic squeeze.** Although classical diving textbooks and literature make reference to breath-holding thoracic squeeze, actual clinical documentation appears to be limited.

Rahn, however, suggested that during breath-hold dives to greater depths, blood is forced into the thorax, replacing air and resulting in a significant decrease in residual volume [5]. Impedance plethysmograph measurements of thoracic blood volume displacements during breath-hold dives to depths of 130 fsw confirmed that a significant shift in blood volume into the thorax does take place [6]. Furthermore, Robert Croft, a U.S. Navy diver, and Jacques Mayol successfully dived to depths of 240 fsw and 231 fsw, respectively. These are considerably greater depths than could be predicted on the basis of total lung volume/residual volume ratios. Mayol's total lung volume and residual volume is 7.22 and 1.88 liters, respectively. Based on total lung volume/residual volume ratios, Mayol's depth threshhold would have been 90 fsw. Theoretically, a blood shift of 980 ml into the thorax was necessary, with a corresponding replacement of air and reduction of his residual volume to approximately one-half that measured. Underwater photographs taken during the 240 fsw dive show pronounced caving in of the thorax, compression of the abdomen, and skinfolds flapping around the chest. More recently, Mayol successfully completed a breath-hold dive to 316 fsw [7].

Post-dive breathing patterns may be significant in compensating for intrathoracic blood volume changes. After

surfacing, the Ama divers of Japan breathe slowly and whistle during the exhalation phase. The valsalva-like effect of the whistling may partially displace the intrathoracic blood volume and, thereby, counteract the effect immersion has in reducing the total lung volume [5].

Increased lung capacity is also another important factor which increases the theoretical depth to which a breath-hold diver can descend. Schaefer observed increases in total lung volume and a relative decrease in residual volume of the lungs in subjects exposed to repeated breath-hold dives [8].

Adaptations to Prevent Decompression Sickness

Aquatic mammals have made a number of adaptations to eliminate the occurrence of decompression sickness. Most mammals dive after full exhalation and, thus, reduce the available nitrogen for tissue saturation by 80 to 90 percent. This may also facilitate diving bradycardia and peripheral vasoconstriction responses as well as aid in achieving negative buoyancy for descent [3]. The Ama of Japan also dive after inhaling to only about 85% of their total volume [9].

Adaptations to Carbon Dioxide Build-Up

Aquatic mammals are less sensitive to low alveolar oxygen concentrations and high carbon dioxide levels than nonaquatic mammals. Analogous adaptations have been observed in trained human breath-hold divers [10]. Schaefer observed that trained and conditioned breath-hold divers had significantly decreased ventilatory response to 10.5% carbon dioxide, better oxygen utilization, acceptance of larger oxygen debt, and increased tolerance to elevated tissue carbon dioxide levels as compared to non-diving humans. These responses disappeared three months after diving was discontinued. This suggests that physical conditioning and repeated breath-hold diving exposures are necessary to develop and maintain these adaptations.

Adaptations Which Improve Recovery Time

Aquatic mammals have respiratory adjustments which improve the efficiency of air exchange and reduce the recovery time following a stressful dive. Human respiratory exchange function is relatively inefficient when compared to other mammals. The seal and porpoise have markedly slower respiration rates, much greater oxygen utilization percentages and larger tidal volumes than humans. As the tidal volume approaches the total lung capacity, ventilation becomes increasingly efficient and compensates for the lower respiratory rate. The respiratory rate in the porpoise and seal is 2 to 4 and 3 to 4 ventilations per minute as compared to the human rate of 15 per minute.

The effects of these characteristics are fourfold [3]. First, the lower respiration rate and large air exchange volume with each cycle reduces the energy expenditure required for ventilation. Second, the elevated oxygen utilization increases the efficiency of each breath and eliminates the need to increase respiration rate. Schaefer reported significant increases in inspiratory reserve volumes, tidal volumes, vital capacities, and total lung capacities in well-trained and experienced human breath-hold divers [8]. These changes signify improved efficiency in respiratory exchange. High and low ventilatory response groups can be defined in humans [11]. The low ventilatory response group is characterized by large tidal volumes, lower respiratory rates and improved tolerance to elevated alveolar carbon dioxide tensions. Third, the fully inflated lung assists in buoyancy during surface swimming and resting, thus reducing energy expenditure. Fourth, improved efficiency in respiration relates to conservation of body heat.

CARDIOVASCULAR CHANGES DURING BREATH-HOLD DIVES

Remarkable cardiovascular changes are observed in aquatic mammals during immersion. The well documented **dive reflex** plays a major role in oxygen conservation and extending dive duration. The dive reflex consists of a reflex bradycardia, peripheral vasoconstriction with preferential shunting of blood from extremities, morphological changes in the vascular system, increased ability for the "noncritical" tissues to function anaerobically, and increased ability to carry, store, and utilize oxygen.

Bradycardia

The immersion bradycardia reflex involves significant reduction in heart rate. In humans the onset is usually gradual and after 30 seconds the rate may slow to 50% of normal [12]. Temperature is important; a 20% greater bradycardia was observed in Ama divers during the winter than in summer [13]. This temperature effect has also been reproduced in the laboratory with facial wetting [14]. Reduction in human heart rate to 8 to 10 beats per minute has been documented [15]. The mechanisms of immersion bradycardia are summarized by Strauss [3]. Among several hypotheses proposed to explain the immersion or diving bradycardia reflex, Andersen suggests that the reflex is initiated by stimulation of the trigeminal nerve [16]. This mechanism is consistent with the rapid initiation of the response, its independence from anoxia and pressure, and the importance of facial immersion.

Peripheral Vasoconstriction and Preferential Shunting of Blood

Bradycardia alone cannot account for the long dive durations observed in aquatic mammals. Strauss summarizes four major

compensations which complement the bradycardia response [3]. First, there is a profound peripheral vasoconstriction and preferential shunting of blood from the extremities to the great vessels, heart, lungs, and brain. This permits the oxygen stored in the blood to be used almost exclusively to perfuse the heart and brain. The heart's oxygen requirements are reduced. Incisions through muscle and skin of aquatic mammals do not bleed during submergence, however, profuse bleeding does occur immediately after surfacing [17] and limb blood flow in humans falls "nearly to zero" during breath-hold dives [18, 19]

Morphological Changes

Secondly, morphological changes in the aquatic mammals vascular system involve stretching of the highly elastic blood vessel walls and a bulbous enlargement of the aorta [20, 21]. These changes help maintain blood pressure and provide for adequate perfusion of the brain.

Anaerobic Function in Non-Critical Tissues

Third, there is an increased ability for non-critical tissues in aquatic mammals to function anaerobically and remain isolated during the immersion. Muscle tissue has the ability to function anaerobically in the absence of oxygen. As the anaerobic metabolic process continues, an oxygen debt is incurred. This debt must be corrected during the recovery period. Metabolism decreases exponentially as the breath-hold dive progresses [22]. Schaefer reports that the lactic acid (substance produced during anaerobic metabolism) content of breath-hold divers' blood increased from an average pre-dive level of 9 mg % to 55 mg % one minute after a dive to 90 fsw was completed [23].

Increased Oxygen Storage Capacity

Fourth, diving mammals have a 50% greater oxygen storage capacity than terrestrial animals of corresponding size [24]. Higher blood volumes, enriched stores of hemoglobin or myoglobin, diminution in the size of the erythrocyle and elevated hematrents, and decreased pH of the arterial blood secondary to lowered buffering capacity all contribute to a greater affinity for oxygen transport, storage and utilization.

Summary of Physiological Changes

In summary, it should be noted that there are three categories of changes [3]. Changes that are universal to the animal kingdom, and occur spontaneously with anoxia, include peripheral vasoconstriction, bradycardra and decreased blood pH. The rapidity of onset, degree of change, and reversibility distinguish aquatic from non-diving mammals. Next, there are a

group of adjustments that are highly developed in aquatic mammals, but can be developed in non-diving mammals, especially man, with practice or exposure. These include ability of the blood to transfer and carry more oxygen, increased ventilation efficiency and improved ability to tolerate oxygen debt. Finally, those changes peculiar to the aquatic mammals include capability to collapse the lungs during submergence, increased flexibility and compressibility of the thoracic wall, highly elastic vasculture, decreased size of the erythrocyles, dilation of the aorta, and diving after full exhalation.

It should be emphasized that snorkel breathing while the face is immersed was found to abolish bradycardia [12]. Consequently, this also suggests that the scuba diver should not experience bradycardia.

A diver with atherosclerotic heart disease or abnormalities of the heart's conduction system is at some risk while diving [15]. Even healthy young adults have experienced syncope and died on sudden immersion in cold water. With the strong vasoconstriction and ischemia of extremities and kidneys, divers with peripheral vascular or renal disease may be at increased risk of damage to these organs.

Dircks suggests that all divers should have an electrocardiogram in order to detect possible congenital or acquired heart disease [15]. He also recommends that the diver observe the following precautions to protect himself/herself from "exceptional" and possible adverse effects of the dive reflex [15]:

* Avoid rapid cold water entry followed by immediate full breath-hold diving;

* Enter the water slowly and splash cold water in the face prior to the dive;

* Exercise briefly on the surface before diving;

* Wear protective mask or hood over the face in cold water;

* Avoid deep breath-holding while scuba diving;

* Don't perform a maximum inspiration maneuver before diving into the water;

* Avoid diving when experiencing anxiety or fear;

* Avoid vertical, head first immersion;

* Do not perform the valsalva maneuver while entering the water; and

* Avoid excessive hyperventilation and excessive duration breath-holding.

IMPAIRED CONSCIOUSNESS DURING BREATH-HOLD DIVING

Hypoxia and Carbon Dioxide Retention

Prolonged voluntary breath-holding while swimming underwater can result in a loss of consciousness and subsequent drowning. Craig studied cases of near drownings and deaths resulting from loss of consciousness while swimming underwater and found that during such circumstances diving accidents were explainable by loss of consciousness due to hypoxia [25, 26]. Hyperventilation is a common practice among underwater swimmers, i.e., skin divers, sponge and pearl divers, etc. By hyperventilating, the swimmer can significantly deplete the carbon dioxide (CO_2) stores of the body. The partial pressure of CO_2 (pCO_2) in the nerve tissue regulating respiration appears to be the primary stimulus to respiration, with comparatively little stimulus derived from low oxygen partial pressures (pO_2). While swimming underwater, the diver uses O_2 and produces CO_2; however, since the CO_2 is used for replacement of the subnormal body CO_2 stores, there is insufficient CO_2 stimulus for respiration. When the oxygen consumption is increased, as in the first few seconds of exercise, the pO_2 may decrease to a degree incompatible with cerebral function before the rise in pCO_2 commands the diver to surface for air. Loss of consciousness can result from hypoxia (or anoxia, which has about the same meaning) with little specific warning. The victim may actually continue his activity between the time of loss of consciousness and final collapse.

Controlled experiments to study the mechanism of hypoxia and carbon dioxide retention during breath-holding dives revealed that this condition is further complicated by increased ambient pressure and ascent from depth [27, 28]. During these dives the alveolar oxygen tension (pAO_2) decreased linearly, but remained high enough to reoxygenate the blood quite completely during most of the dive. However, staying on the bottom longer than 90 seconds yielded significantly low pAO_2 and arterial O_2 tension (paO_2). The pAO_2 and the Bohr effect (the greater the $paCO_2$, the lower the hemoglobin O_2 saturation) resulted in a significant fall in O_2 saturation; thus, the blood could not carry as much O_2 from the lungs to the tissue as before. Some divers have been reported to have lost consciousness at the bottom; possibly they have contracted the dangerous combination of a low pAO_2 and a very high $pACO_2$.

Shortly after reaching the bottom, a diver may experience a subjective "breaking point" approach sensation due to increased $paCO_2$ stimulus, plus stimuli elicited from smaller lung volume. This sensation is easily overcome by the willpower of trained breath-hold divers. The expert skin diver can actually "condition" himself to voluntarily or involuntarily ignore the breaking point sensation (or urge to breathe) and over a period of time becomes inured to the subsequent pCO_2 buildup that would drive the average person to the surface for air. During ascent, a relief of the breaking point sensation is experienced because

the lung volume increases and the $pACO_2$ falls, even though oxygen may actually diffuse from the alveoli to the blood at a slower rate due to $pACO_2$ decrease. Since the pAO_2 may fall below the venous pO_2, the possibility for O_2 transfer from the blood to the lungs is present. Blood oxygen stores may be depleted rapidly. If during ascent blood deprived of oxygen arrives at the cerebral cortex, the diver may lose consciousness with little or no warning before or just as he reaches the surface. Unconsciousness during ascent when the diver is below the "buoyancy point" is a potentially fatal condition. Ironically, many competitive skin divers wear lead weight belts, making them negatively buoyant for effortless diving.

Bond condemns wearing excessive weights and competitive breath-holding exercises and contests, even under the auspices of a good organization [29]. Unfortunately, competitive breath-holding contests are a common occurrence in nearly every American swimming pool. Bond cites one such experience involving a 16-year old male in excellent physical condition participating in a contest conducted in a swimming pool. Wearing a face mask and weight belt, the young man settled to the bottom of the pool and remained there for nine minutes in full view of almost 200 spectators. Finally, he was hauled to the surface in a state of unconsciousness and not breathing. His breathing was successfully revived; but subsequent examination and electroencephalograms revealed no cortical activity. In other words, this young man was now doomed to live the rest of his life with major neurological deficits.

Decompression Sickness

Neurological phenomena, including unconsciousness as a result of decompression sickness, may occur from repeated breath-hold dives to great depths [30, 31]. The increase in the pAN_2 is high at about 20 m depth. Although the volume of N_2 absorbed during each dive may be small, the increase in tissue pN_2 (ptN_2) could account for the occurrence of N_2-containing bubbles in the tissue following many repetitive and rapid alterations in ambient pressure. Bond relates a personal experience in which he was a victim of decompression sickness as a result of seven hours and 20 minutes of continuous breath-hold skin diving to depths of 80-100 feet [29]. Fortunately, such cases are rare, probably because most human divers cannot breath-hold dive deep enough nor often enough to contract decompression sickness.

Cross discussed the dreaded disease of Tuamotus pearl divers, **"taravana"** [32, 33]. These pearl divers are true skin divers; they use no breathing apparatus or air supply for their underwater work. Yet, many of those stricken with "taravana" exhibit symptoms like those of classic decompression sickness -- vertigo, paralysis, unconsciousness, and insanity. These divers may make as many as 6 to 14 dives per hour to depths up to 150 feet and stay submerged an average of one minute and 35 seconds.

This schedule is continued daily throughout the pearl diving season. On one exceptionally good diving day (good weather and seas), Cross observed that 47 divers were stricken with "taravana". Thirty-four of the 37 suffered vertigo, nausea, and dizziness, and 11 surfaced paralyzed or unconscious and were rescued. Of the 11, six were partially or completely paralyzed, two were "mentally affected", and two young men died.

Cross suggests that anoxia is the principal cause of "taravana", with its effects on the central nervous system and the brain accounting for the many and varied symptoms. He points out that Mangareva divers space their dives 15 minutes apart, instead of the 4 to 8 minutes used by the Tuamotus divers, and do not suffer from "taravana" [33].

Certainly anoxia or hypoxia explains many of the symptoms, and it should also be stated that continuous daily and seasonal exposure of brain tissue cells to hypoxia conditions could possibly result in cumulative and irreversible brain damage. However, decompression sickness is also an equally significant explanation, especially when considering the cumulative underwater time and depth [30, 31].

Frequently, loss of consciousness while underwater is referred to as shallow-water or underwater blackout. The U.S. Navy defines shallow-water blackout as an "accident in which a diver loses consciousness, presumably from carbon dioxide excess without an adequate respiratory warning" [34]. Bond considers lowering of oxygen levels of vital organs as a primary cause [29].

For comprehensive physiological studies and reviews of breath-holding, readers are encouraged to consult Strauss [3], Rahn and Yokoyama [35], and Mithoefer [36].

CONCLUSION

Numerous studies support the fact that significant physiological changes occur in the human body during periods of breath-holding and submergence [35-43]. In order to reduce the possibility of adverse effects resulting from these changes, breath-hold skin divers are encouraged to limit pre-dive hyperventilation and submergence time. Studies and experience also show that impaired consciousness without diver awareness is possible during prolonged breath-holding submergence. Team diving procedures are encouraged to enhance the overall safety and enjoyment of breath-hold diving.

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