

tation Medicine and various specialist departments of the Tan Tock Seng Hospital. We aim to give all the DCS patients the complete management that they deserve.

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BREATH-HOLD DIVING

Michael Davis

Introduction

There is a beautiful photograph by Flip Nicklin in the December 1984 issue of the National Geographic magazine of a sperm whale sounding. This epitomises for me the wonder and majesty of the diving mammals, that diverse group of animals of widely varying structure, function, habitat and behaviour who grace our oceans (Table 1). This paper briefly reviews the physiology of these animals and aspects of human breathhold diving, whilst the bibliography provides a selection from the literature rather than an exhaustive list.

Asphyxia is a progressive process which begins at the moment that external gas exchange ceases. The cessation of respiration leads to hypoxia, hypercapnia and acidosis, the

TABLE 1
SOME ADAPTATIONS TO APNOEIC DIVING IN AQUATIC MAMMALS

Respiratory

Tolerance of thoracic squeeze

lung collapse
elastic chest wall
mechanically tough tracheo-bronchial tree

Protection from decompression sickness

lung collapse (loss of gas exchange)
Decreased sensitivity to hypoxia and hypercapnia
Increased ventilatory/gas exchange efficiency

Cardiovascular

Blood shift into thoracic cavity
Venous spinclters and sinuses
Large spleen
Diving Reflex CVS components

Biochemical

Increased blood (haemoglobin) and tissue (myoglobin) oxygen stores
Diving Reflex Switch to anaerobic metabolism

Hypothermia/Insulation

Efficient locomotion

triad of asphyxia. The successful mammalian diver can postpone the inevitable functional collapse that follows the cessation of breathing by virtue of three main mechanisms:

- 1 Enhancement of the oxygen stores in the body
- 2 Acid buffering of the products of metabolism
- 3 Circulatory reduction and redistribution, leading to metabolic conservation.

Regulation of these responses is essentially identical to that governing the protective reaction to asphyxia in terrestrial animals, the observed differences being quantitative rather than qualitative.

Scientists have approached the study of these remarkable animals' adaptation to asphyxia both in the laboratory and in their natural environment. Although some laboratory work has been criticised, field and laboratory research are, in fact, complimentary, and not conflicting. "Exploring what an animal is capable of is not the same as asking how it normally behaves."

Adaptations in diving mammals

Animal behaviour is determined by ecological constraints and physiological limits. In diving mammals these limits are defined by the amount of oxygen carried, the rate of oxygen consumption and the maximum pressure the animal is capable of withstanding. For instance, harbour seals have been recorded whilst diving under the Antarctic ice for more than one hour and to depths greater than 600 m. The physiological and anatomical adaptations involved vary considerably from species to species, each adopting different strategies. Therefore, not all the features described below are shared by all.

ANATOMY

Quite apart from the sleek, well-insulated body habitus that allows high swimming efficiency and protection from hypothermia, numerous anatomical features are present in the diving mammals to facilitate tolerance to the challenges of the underwater environment. Many have a floating, compliant rib cage, with a very obliquely inserted diaphragm. The respiratory tract tends to be short, wide and mechanically tough. In some whales, but not in pinnipeds, the respiratory tract can be occluded by special anatomical structures in the blow-hole, spiracle chamber and glottis. In some species these and other features may improve tolerance to chest compression ("thoracic squeeze") during deep dives.

Dolphins have terminal respiratory bronchioles that take part in gas exchange and the structure of the pulmonary microvasculature allows a reduced diffusion barrier. Also present are rete mirabile and arterio-venous shunts in certain organs, including the lining of the airways in the sperm whale. In the deep diving species, a very large inferior vena cava with a precordial sphincter, and a large spleen are present, whilst harbour seals also possess an aortic bulb. All these features may be important either for gas exchange or in the oxygen delivery to vital organs, but in many cases their contributions remain ill-understood.

LUNG VOLUMES

Long-duration diving whales have small lungs which collapse during dives and are not used as an oxygen store, e.g. the bottlenose whale has 25 ml/kg body weight. Short duration divers tend to have lung volumes similar to terrestrial mammals and dive following inspiration. Thus they appear to use the lungs as an oxygen store, e.g. man and porpoise have 70 ml/kg body weight. Seals dive following partial expiration which also results in lung collapse during descent. Although lung size may vary, diving mammals tend to have large tidal volumes, low end-expiratory volumes and a breathhold ("skip") breathing pattern. Minute ventilation is high during surfaced periods between hunting dives. For instance it has been measured at greater than 60 l/minute in the harp seal.

OXYGEN DELIVERY

Oxygen may be stored in the lungs, blood and muscle. These stores are considerably greater in diving mammals than terrestrial animals:

Weddell seal	60 ml/kg body weight
California sealion	40 ml/kg body weight
Man	20 ml/kg body weight

Haemoglobin

Adaptation of haemoglobin-oxygen affinity parallels the modifications in lung volumes in that, where the lung does not act as an oxygen store, oxygen affinity is low. This leads to maximal unloading of oxygen at the tissues (i.e. a shift of the haemoglobin-oxygen dissociation curve to the right). For instance, seals and some whales extract 7-10 vol% of oxygen from the blood, compared to man at around 5 vol%. In those animals in which the lungs act as a store, affinity tends to be high. This ensures maximal oxygen uptake in the lungs (i.e. the dissociation curve is shifted to the left).

Blood stores of oxygen are one to three times that of terrestrial mammals. Both blood volume and haemoglobin concentration may be high. In Weddell seals, the red cell mass and haemoglobin concentration actually increase during a dive by about 8 g/l/min during the first 10 minutes of the dive. This extra red cell mass probably comes largely from venous pooling and sequestration of oxygen-rich blood in the spleen which has the highest weight as a percentage of body weight of any reported mammal. It is probable that the precordial sphincter in the inferior vena cava periodically relaxes to give a pulsed delivery of this oxygen-rich blood.

Interestingly, in seals resting on the surface, arterial oxygen tension is actually lower (70-80 mm Hg) than most terrestrial mammals, but can be rapidly raised to over 110 mm Hg by hyperventilation just before the dive. During the dive, oxygen tension rises to a maximum of about 230 mm Hg and falls to 25-35 mm Hg by the end. The lowest in-dive oxygen tension recorded in Weddell seals in Antarctica was 18 mm Hg.

Myoglobin

Oxygen stores in myoglobin vary from one to nearly ten times that of terrestrial mammals. Like haemoglobin, this parallels the dive duration capabilities of the species concerned.

CARDIOVASCULAR MODIFICATIONS

As the animal dives deeper and the gas volume in the lungs is compressed, the rib cage gradually collapses, as has been dramatically illustrated in photographs taken at depth. In addition, there is a major shift in blood volume into the lungs to take up some of the additional space, thus preventing lung rupture. As well as this increase in pulmonary blood

volume, the coronary vascular blood volume tends to be high during bradycardia. However, coronary blood flow demonstrates large fluctuations from virtually no flow to periods of high perfusion during a dive. This may allow the myocardium to derive maximum benefit from both its oxidative and anaerobic metabolic reserves by permitting periodic wash-out of metabolic products, and reperfusion with oxygen-rich blood.

AEROBIC DIVING

The calculated aerobic breathhold times based on estimated body oxygen stores and oxygen uptake fit very well with metabolic evidence from free-diving seals. For instance, in the Weddell seal the aerobic breathhold limit is about 20-30 minutes which is the same as for this animal's usable oxygen stores under normal aerobic metabolic conditions. Observations on Weddell seals and several other species suggest that more than 90% of dives are within the aerobic limits so that metabolic requirements are satisfied by the body's oxygen stores with minimal increase in lactate.

Short aerobic dives have a brief recovery time since they require restoration of oxygen stores only. Anaerobic dives, however, require metabolism of lactate and other metabolic products that takes much longer to complete. A series of short duration dives increases hunting efficiency compared with a few long dives by allowing a much greater proportion of time underwater. For instance, in harp seals about 88% of the time is spent diving when feeding, with an average dive time of about five minutes. During such "bout" diving there is virtually no disturbance to metabolic homeostasis except the rise in haematocrit seen during dives.

THE DIVING REFLEX, ANAEROBIC DIVING

The diving reflex is triggered by face immersion and breath-holding. It is mediated by both the parasympathetic and sympathetic nervous systems to produce some intense physiological and biochemical changes (Table 2). The components of the diving reflex have been seen best in captive laboratory dives. However, field work with seals and dolphins has shown that this response is not an all-or-none phenomenon, but extremely variable. There is now strong evidence that diving mammals can anticipate the dive duration and control both the degree of bradycardia and the extent of blood flow redistribution needed for a given dive. Animals also anticipate surfacing and their heart rate is seen to increase shortly before the end of the dive.

Once the anaerobic threshold is reached, some major metabolic adaptations come into play. Muscle glycolysis is an impressive anaerobic machine even in non-divers, but the diving mammals have refined it in a number of ways:

- a) Steady state concentrations of a few glycolytic enzymes are high, so increasing the capacity to maintain NAD/NADH (Redox Potential) ratios under anoxic stress.

TABLE 2

COMPONENTS OF THE DIVING REFLEX

Bradycardia

Intense peripheral vasoconstriction

Reduced cardiac output

Redistribution of cardiac output to vital organs

Metabolic shift to anaerobic glycolysis

Decrease in body temperature due to decrease in metabolic rate

b) Concentrations of fructose-diphosphatase which is a key regulatory step in the glycolytic pathway are amongst the highest reported in animals. This appears to amplify the cyclic-AMP signal for glycolytic activation.

c) Pyruvate kinase activity is enhanced. This enzyme is regulatory to the glycolytic pathway in that it is highly sensitive to both feed-forward activation by fructose-1,6 diphosphate and feedback decrease by ATP, alanine and citrate. This is thought to help particularly in switching to and from the aerobic to the anaerobic state.

d) Very high titres of aspartate and alanine transferases are present. These enzymes are very important in the control of the Krebs cycle.

Diving mammals can tolerate high lactate loads in their tissues. Plasma lactate concentration does not rise much until immediately after the dive, when metabolic products are washed out of the non-perfused tissues once the diving reflex ceases. The amount of lactate produced increases exponentially as dive time goes beyond the aerobic limit. During long dives, seals are able to sustain work loads more than twice their maximum aerobic capacity with little or no increase in oxygen uptake. To support this, both skeletal and heart muscle are rich in myoglobin (see above) which has important buffering functions under anaerobic conditions.

The brain and the heart of diving mammals have considerable anaerobic capacities and can produce large quantities of lactate toward the end of a long dive. In addition, the brain of diving mammals has a much greater tolerance to decreased oxygen compared with terrestrial mammals. Cerebral integrity is retained down to oxygen tensions as low as 8-10 mm Hg, whereas in humans this is about 25-30 mm Hg.

Organs such as the kidney, have also been shown to tolerate asphyxia far better than those of terrestrial mammals. For instance, the rise in intracellular sodium and fall in potassium in anoxic harbour seal kidney slices is consistently less than in rat kidney, especially at low pH. Also, the seal kidney functions differently in that water conservation occurs with immersion unlike the diuresis seen in man.

Hochachka has written extensively on the molecular mechanisms of defense against hypoxia. He believes that three main mechanisms are important:

- 1 Arrest of oxidative metabolism and electron-transfer system functions (i.e. reduced metabolic rate)
- 2 Arrest of glycolytic activation
- 3 Arrest of ion-specific channel functions

He hypothesises that hypoxia-tolerance depends on an exceptionally tight regulation of energy demand and supply. Such close regulation of ATP turnover as the tissue becomes hypometabolic is only possible if at least one regulator signal switches down ATP utilisation and synthesis rates simultaneously. It is not known what this regulator might be nor even if it exists. This concept has been called the "membrane channel arrest" hypothesis.

DECOMPRESSION SICKNESS AND THE DIVING MAMMALS

There are several potential mechanisms for limiting nitrogen loading in the tissues during long deep breathhold dives by reducing the nitrogen stores in the lungs:

- a) Dive at low lung volumes
- b) Produce a profound fall in pulmonary blood flow early in the dive
- c) Allow lung parenchyma to become gas-free with compression, the residual volume shifting into the airways.

It is the latter mechanism for which there is most evidence. Recent work has been done on Weddell seals during dives averaging about 23 minutes duration and 230 m average depth. This has shown that plasma nitrogen tension rises rapidly during the early part of the dive, peaking at 2,000-2,400 mm Hg at about 40 m depth. At this depth (shallower than previously believed) the lungs collapse. Nitrogen tension then slowly falls to about 1500 mm Hg near to the time for surfacing. Thus the seal is protected from decompression sickness and nitrogen narcosis by limiting nitrogen uptake and redistributing nitrogen during dives. Part of this redistribution may be into the red cell mass entering the circulation from the spleen during the dive. This would also protect against oxygen toxicity.

Other factors could also play a part in protecting the animal from decompression sickness. For instance, repeti-

tive breathhold dives might force gas micronuclei into solution, thereby decreasing any tendency to bubble seeding. Finally, the possibility has been raised that the acoustic echo-location system of whales and dolphins, which is fat-rich, may provide an early-warning bubble detection system.

Human breath-hold diving

Free-diving is an ancient and widespread human activity. In communities such as the Ama of Korea and Japan it became part of the economic basis of society. In the twentieth century it has become a popular sport, whilst free diving for a living amongst indigenous peoples has generally either dwindled markedly or they have moved on to scuba techniques.

TABLE 3

DIVING DATA FOR MAN AND AND DIVING MAMMALS

	Breath-hold Time (min)	Maximum Depth (m)
Man	2-4	? 100
Dolphin	6	300
Orca	12	?
Weddell Seal	75	600
Sperm Whale	? 75	? 1000 +

When I first learnt to dive in the early 1960s, I was taught two concepts about breathhold diving. The first was that the maximum depth for human breathhold diving was about 33 m (100 ft). This was because, as the lung gas volume diminished with increasing pressure, a point was reached where lung tissue would rupture and pulmonary haemorrhage occur. This was called "thoracic squeeze".

We now know the above concept is not valid. Thoracic squeeze probably does not occur in breathhold diving in man under most circumstances because there is a major shift of blood volume into the pulmonary vascular bed with compression. The second concept of much more practical importance to man than thoracic squeeze was that dive duration was limited to only one or two minutes by "shallow water blackout".

The longest human breathhold times recorded on air are about 4 to 5 minutes. The deepest dives recorded are those of Robert Croft to 240 ft in the 1960s, and Jacques Mayol to well over 300 ft in the 1970s. The pattern of diving activities has been extensively studied for groups such as the Ama, but less so for intensive sport divers such as underwa-

ter hockey players or competitive spearfishermen. The Ama dive 30 to 45 times an hour for 30-45 seconds with 45-90 seconds rest between dives for many hours, spending 1/3-2/5ths of their total time underwater. In contrast, the dive duration for underwater hockey players over a 30-40 minute playing period, averages only 8-10 seconds per dive, but recovery periods are only 3-12 seconds. Thus they spend 2/5-2/3 of the total time underwater (personal observations).

Such efforts pale into insignificance compared to the diving mammals! However, many physiologists have been interested to observe whether human breathhold divers demonstrate any adaptations in the ways seen in the diving mammals. The classic studies on the Ama make absorbing reading and the reader is recommended the proceedings edited by Rahn.

FACTORS INFLUENCING BREATHHOLD TIME IN MAN

TABLE 4
CONDITIONS FAVOURING
LONG BREATHHOLD TIMES

- Large O₂ stores
- Diving Reflex
- Pre-breathhold hyperventilation
- Increased diving depth
- Thermoneutral immersion

LUNG VOLUME

Record divers all have big lungs. Geoff Skinner, Australian spearfishing champion for many years, demonstrated an air breathhold of 3 minutes 8 seconds during the talk at Port Vila. He has a vital capacity of well over seven litres. Ama were shown to have an increased vital capacity compared with non-diving women in their community, but as long as 20 years ago, Hong questioned whether this was due to diving per se or rather to physical fitness. Recent studies suggest the lungs of breathhold divers are indeed no bigger than other athletes. However, studies of breathhold times have been conflicting. Dry-land athletes and divers performed the same in one study, but the divers had longer times in another. Breathhold times in children correlate with age in proportion to their lung size.

Thus, our primary oxygen stores for diving are in the lungs, as for the short duration diving mammals.

HYPERVENTILATION

By decreasing the CO₂ tension, respiratory drive is decreased. The increase in alveolar oxygen concentration increases the oxygen stores in the lung slightly but only minimally increases oxygen content in the blood.

OXYGEN BREATHING

Oxygen breathing increases the oxygen stores in the lungs by up to several litres. Following oxygen breathing for three minutes, Geoff Skinner's breathhold time at the Port Vila meeting increased to just under ten minutes.

METABOLIC RATE (VO₂)

There is an inverse relationship between breathhold time and VO₂. However, oxygen consumption varies with the type of diving being undertaken. For instance, a dive to 5 m depth without swim-fins and lasting 30 seconds has a mean VO₂ of about 0.8 litres, whereas a 10 m dive for 30 seconds uses 1.2 litres O₂. Oxygen consumption is increased when the descent is active rather than passive. Swimfins reduce oxygen uptake slightly at comparable swimming speeds. In underwater hockey, a higher VO₂ is likely because of the high swimming speeds whilst breath-holding. Since the total oxygen stores of an average 70 kg man with normal lung size are only approx 1.5 L breathing air, unconsciousness is likely to occur in approx 80 seconds at a VO₂ of 1.0 l/min (about that of moderate fin swimming). There is no evidence in man of a switch to anaerobic metabolism with the induction of the diving reflex.

OXYGEN AND CARBON DIOXIDE CHANGES DURING BREATHHOLD DIVES

Changes in O₂ and CO₂ are very important in the control of breathing, in determining the "breaking point" for a breathhold, and in the aetiology of shallow water blackout.

a) **Oxygen**

During most of a dive the relationship between VO₂ and oxygen tension is linear. However, once the "alveolar stores" in the lungs are exhausted, the "blood stores" of oxygen are used and the relationship becomes non-linear with rapid desaturation of haemoglobin. Thus, the rate of fall of PaO₂ below 100 mm Hg is highly dependent on the haemoglobin concentration and on circulating blood volume.

The alveolar-arterial oxygen difference is similar throughout the dive at approximately 10 mm Hg. If an ascent is needed at the end, there is a rapid decrease in PAO₂ and in very long dives this could result in an actual reversal of oxygen transfer across the alveolar membrane resulting in even more rapid desaturation. Towards the end of long dives, in fact, mixed venous and

arterial oxygen tensions approach each other. In the many studies by Craig, Hong and Paulens, oxygen tension at the end of most dives tended to be around 60 mm Hg but was quite variable, and in some dives levels as low as 30 mm Hg were observed.

b) Carbon Dioxide

Changes in CO₂ during a dive are more complex than those for oxygen:

1 Approximately half the CO₂ enters the alveoli during a dive in the first few seconds as a consequence of the acute fall in the CO₂ tension (P_ACO₂). This is due to dilution into the large inspired volume immediately prior to the dive.

2 With the initial descent, the PACO₂ increases with lung compression to approximately 50-60 mm Hg. This leads to an increased P_ACO₂ and therefore a decreased gradient for tissue off-loading. Tissue off-loading could be further reduced by decreased muscle perfusion as part of the diving reflex as in diving mammals. However, there is no metabolic evidence for this in man (see below).

3 The alveolar volume cannot serve as a CO₂ store due to its rapid compression with descent. During a dive this rapid compression is analogous to a rebreathing test and equilibrium between alveolar and arterial PCO₂ should occur rapidly. Indeed, it does so after less than a minute, after which the alveolar-arterial CO₂ gradient may actually reverse and alveolar CO₂ fall slightly. During the remainder of a dive after the appear to be related to the length of the dive and is non-linear.

In Craig's studies, PACO₂ tended to be approximately 60 mm Hg suggesting tissue CO₂ tensions close to this. This could explain the subjective experience of approaching the breaking point immediately after a rapid descent. This sensation then passes, only to return towards the end of the dive. During ascent the urge to breathe is often relieved again as the P_ACO₂ falls, enhancing CO₂ off loading into the alveoli.

4 The PACO₂ at the end of a dive is much less than after an equivalent period of breathhold exercise on dry land. Values are typically 48-50 mm Hg, but in Paulev's study they were normal in many subjects. Hong calculated that during a 4-minute oxygen breathhold, the lung supplied 700 ml oxygen, but only gained 160 ml CO₂.

Why does the PACO₂ rise initially then plateau during a dive? Where is the CO₂ stored, since there is no evidence of a shift to anaerobic metabolism in man? Exercise studies at 30% of VO₂ max

suggest a storage capacity for CO₂ in the body of 1.83±0.55 ml/kg/hr, this is very large and is due to the high solubility of CO₂ in body tissues. Thus it would appear that the tissues act as the buffer for P_ACO₂ changes during a dive and as a store for CO₂.

c) Shallow Water Blackout

The non-linearity of the relationship between oxygen tension and blood oxygen content at the end of a breathhold dive is very important to understanding the mechanism of shallow water blackout. To illustrate this, let us consider the two dives in Figure 1. The first graph is derived from a dive in Shaeffer's earlier work. This illustrates a 9 m dive divided into three equal 20 second periods for descent, bottom time and ascent.

During descent the PaO₂ increases, then gradually falls during the working part of the dive. Then, on ascent, this fall becomes steeper. If, however, we look at oxygen saturation in the blood during this period, it remains nearly fully saturated for virtually the whole dive, only dropping toward the end of the ascent. In the first dive the O₂ saturation never reaches dangerous levels at which a diver might lose consciousness.

If we now extend this dive by a further 20 seconds working time (Figure 1), then at the end of one minute the P_AO₂ is still relatively high and the S_aO₂ only just starting to fall. However, because the lung oxygen stores are almost used up at this stage, the diver starts to draw on his blood stores which, as we said earlier, are of limited capacity. During this ascent, saturation falls far more precipitously and the point of loss of consciousness is reached very rapidly. On the graph this point is arbitrarily set at about 29 mm Hg or the P₅₀ for the normal haemoglobin-oxygen dissociation curve, since consciousness is lost in the range 25-30 mm Hg.

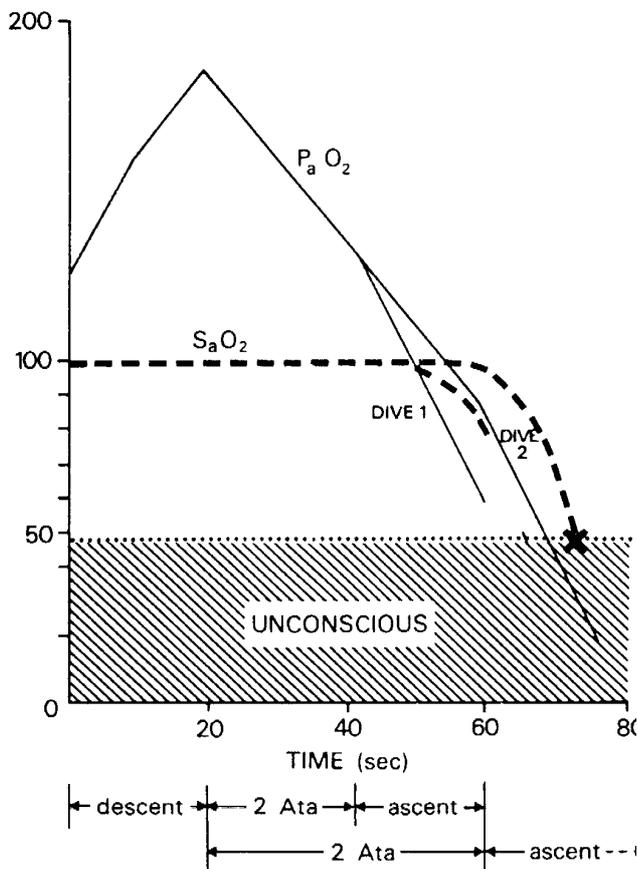
We are all aware that shallow water blackout does occur even without a descent/ascent dive pattern, particularly if respiratory drive is diminished by prior hyperventilation. What I wish to emphasise is the non-linear nature of the rate of change in oxygen saturation, and how narrow is the margin between a "safe" dive and shallow water blackout.

Bove has suggested that those with diminished carbon dioxide and hypoxic responses may be more prone to shallow water blackout. He believes this is partly because the initial decrease in heart rate with face immersion is later potentiated by hypoxia during long breath-holds.

OXYGEN AND CARBON DIOXIDE SENSITIVITY

There has been much work on the ventilatory responses to exercise, hypercapnia and hypoxia in athletes. These studies have demonstrated that the peripheral che-

FIGURE 1



The time course of changes in $P_a O_2$ and $S_a O_2$ during two breathhold dives to 9 m depth, one having a 20 seconds longer bottom time than the other. Note the steepness of the fall in $S_a O_2$ towards the end of the longer dive.

moreceptors in the carotid body are responsible for the respiratory compensations to the metabolic acidosis of exercise. For instance, if the carotid body is absent, both the rate of rise of ventilation and its peak level are diminished during graded exercise.

It is also known that there is marked individual variability in the responses to hypercapnia and hypoxia. High performance athletes tend to show decreased responses at rest, suggesting decreased chemoreceptor function. There appears to be a major genetic component to this in endurance athletes and swimmers, with no evidence that it is altered by physiological training.

The ventilatory response to exercise appears to parallel the hypoxic response at both low and high work levels and the hypercapnic response at heavy exercise. Also any

fall in ventilation with hyperoxia is proportional to the hypoxic response of the individual. It has been reported that breathhold time is inversely proportional to the CO_2 response ($re = -0.89$) in dry-land athletes, but such a relationship has not been the case in studies of breathhold divers.

DO DIVERS HAVE ALTERED SENSITIVITY TO CO_2 AND O_2 ?

It has been suggested for many years that divers may have reduced chemoreceptor sensitivity. What is the current evidence for this?

1 Of the two types of Ama divers, only the Funado have shown any evidence of decreased carbon dioxide responses, yet their breathhold times were the same as non-diving controls. There has been no convincing evidence of a decreased hypoxic response in the Ama.

2 Submarine-escape tower divers: One NMRI report suggested that the CO_2 response decreases during training in successful diving candidates. Schaeffer reported a low CO_2 response in escape tower safety divers, which increased after a five week lay off.

3 Melamed and Kerem reported that O_2 diving did not alter peripheral oxygen chemoreceptor response and there was no change in hypoxia response compared to controls. A sub-group of scuba divers in their study were reported to have a decreased CO_2 response.

4 Several studies have shown low CO_2 responses in underwater hockey players, but this has not consistently been less than for high-class dry-land athletes. Only a small effect of training was seen in one unpublished study (McKenna and Green, personal communication). This suggests that, overall, breathhold divers may have a diminished CO_2 response, but whether this is modifiable with training or whether they demonstrate other differences in ventilatory responses is not known. We have recently been studying in our laboratory the effects of breathhold training on the hypercapnic and hypoxic responses in underwater hockey players, but our data have not yet been analysed.

In summary, no clear relationship between peripheral chemoreceptor sensitivity and breathhold times has been demonstrated in man.

OTHER FACTORS

a) Water temperature: With decreasing water temperature there is a decrease in breathhold time proportional to the increased metabolic rate of cold immersion. This is in spite of an enhanced diving bradycardia in cold water. In other words, metabolic rate and the diving reflex are physiologically independent in man, rather than linked as in the diving mammals.

b) The diving bradycardia (see below): There is no prolongation of breathhold time as diving bradycardia is elicited.

c) Cortical stimuli: In man, motivation is probably the strongest factor in breathhold duration to the conventional "breaking point" (see below).

THE BREAKING POINT

The drive to break a breathhold is both chemical and mechanical.

Two end-points have been used in past studies:

- 1 The conventional breaking point is taken as the time to the first inhalation.
- 2 Physiological breaking point is the time to the onset of involuntary ventilatory muscle activity (usually measured using electromyography). This appears to be PCO₂ and VO₂ dependent, non-subjective and therefore a more precise end-point for laboratory studies.

Several components are important, including the chemoreceptor sensitivity to carbon dioxide and oxygen, signals from pulmonary and chest wall stretch receptors, and motivation. Conventional breathhold time is prolonged by rebreathing in the absence of alveolar O₂ and CO₂ changes. This shows the importance of signals from the chest wall and lung stretch receptors.

BREATHHOLD AND FACE IMMERSION - THE "DIVING REFLEX" IN MAN

Breathhold in man is characterised by bradycardia (Figure 2), a progressive rise in blood pressure, stroke volume, cardiac output and peripheral vascular resistance. There is no evidence for significant metabolic changes or major oxygen-conserving responses such as occur in diving mammals. Therefore, the evidence to date is that habituated divers such as the Ama and underwater hockey players do not show enhanced defences against asphyxia.

The "Diving Reflex" in man is:

- 1 Independent of posture
- 2 Relative to the level of body immersion
- 3 Proportional to lung volume at which breath is held
- 4 A function of intrapleural pressure
- 5 Potentiated by face immersion
- 6 Inversely proportional to water temperature
- 7 Enhanced by dynamic exercise, but not proportional to level of fitness (including Ama divers)
- 8 Enhanced in swimmers compared with non-swimmers
- 9 Diminished by wearing facemask and hood in warm but not cold water
- 10 The same in children in whom breathhold time is long enough to reach full diving bradycardia (approx 25 seconds) as it is in adults.

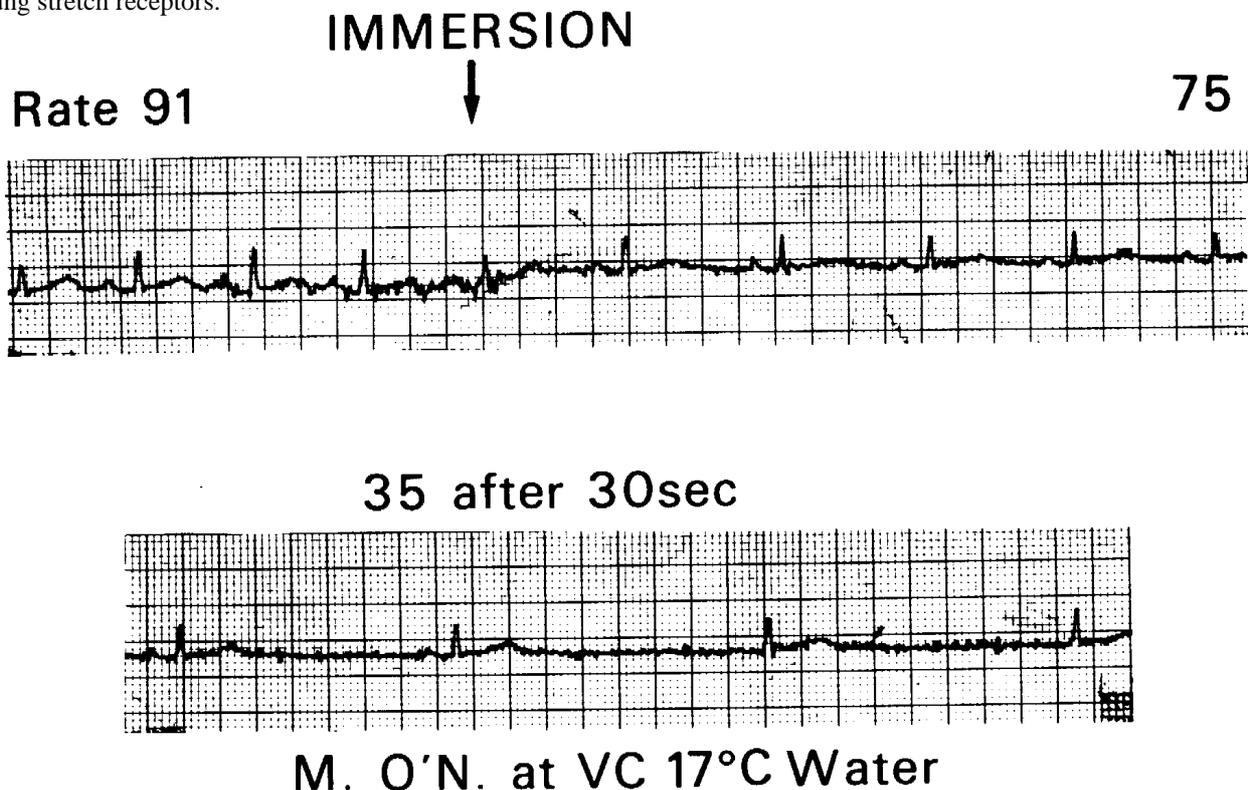


Figure 2 The human diving bradycardia with breathhold and face immersion. Note that the maximal fall in heart rate takes about 30 seconds to be achieved.

In summary, man is a poor diver, reliant on his lung oxygen stores during a breathhold. Whilst the diving bradycardia occurs in man, it is not accompanied by the important metabolic and other circulatory changes that characterise the full "diving reflex" in many diving mammals. In addition, the evidence for physiological adaptation to habitual breathhold diving is inconclusive. An understanding of the non-linear relationship between oxygen tension and content in the blood helps to explain the narrow line between a "safe" dive and one leading to shallow water blackout. Clearly, there is much research yet to be done in this fascinating field of environmental physiology.

UNDERWATER HOCKEY

Underwater hockey is a team water sport, with six players per side in the water, played on the bottom of a 1.8-2.5 m deep swimming pool with swimfins, mask and snorkel. A plastic covered lead or brass disc-shaped puck is pushed or flicked along the bottom of the pool with a short roughly triangular-shaped single-handed stick. A goal is scored when the puck is played into a shallow trough or goal at either end of the playing area. The game is played underwater during repeated breath-holds, and lasts 30-40 min.

At international level, it requires an extremely high level of fitness and breathhold diving skills. However, at all levels of competition it provides extremely good exercise for scuba divers, maintaining water skills and fin swimming fitness. It may thus be regarded as ideal exercise for maintaining scuba diving fitness in all age groups (there are now significant numbers of players over the age of 40 participating very successfully in the sport). Although it obviously will not appeal to all, I believe it should be recommended to divers as an excellent group activity for the maintenance of water skills.

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EDITED QUESTION TIME

Vanessa Haller

How do you feel about asthmatics playing underwater hockey as an alternative to scuba diving?

M Davis

If you want a let out in your response to these patients, swimming is an excellent sport for asthmatics. I cannot think of a reason why underwater hockey should not necessarily be good for them as well. Anybody who is in the water is at risk of aspiration precipitating an asthmatic attack. However, there is good evidence that swimming and water sports apart from scuba diving are often very good for asthmatics. I see no logical reason why asthmatics should not play underwater hockey. The water is not deep, up to 8-9 feet, it is warm and not likely to give cold precipitation of asthma. You have the other advantage of doing competitive exercise with high endogenous adrenalin levels, keeping the airways open! Certainly our top player in Christchurch is a moderately severe asthmatic. He actually controls his asthma by playing underwater hockey and swimming. If he lays off he is into the Ventolin inhaler 3-4 times daily. If he plays regularly he only occasionally needs his inhaler.

V Haller

It is good because it gives them an outlet for their aggression. Hating themselves for having asthma at that age.

V Brand

It is a pity to make a blanket condemnation of hyperventilation. Most spearfishermen and breathhold divers take 3-4 deep breaths before they dive. If they do not, they do not know what they are missing. The dangerous ones are when you seek kids who hyperventilate for a minute or two in order to compete with their peers.

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