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Applied Physiology of Diving

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Summary

Recreational diving is a popular sport, although human ability to stay in and under water is severely limited physiologically. An understanding of these limitations enhances safety and enjoyment of sports diving.

Breath-hold diving involves head-out water immersion, apnoea and submersion, exercise, cold stress, and pressure exposure. Each of these components, by itself, elicits prominent and specific physiological effects. Combination of these factors produces a unique and interesting physiological response generally known as diving reflex. Humans display weak diving responses, but exhibit no oxygen conservation function. Nevertheless, application of diving-induced physiological changes is now finding its way into clinical practice.

Apnoea, face immersion, and head-out water immersion all show promise of clinical

application. There are several spin-offs from diving research worth noting. Diuresis, enhancement of cardiac performance, and redistribution of blood flow, all produced by head-out water immersion, have been shown to be clinically useful, besides providing physiological data useful to space travel. Results from investigations on apnoea have been shown to be relevant to the following: treating some forms of cardiac arrhythmias; understanding drowning, sudden infant death syndrome and sleep apnoea; and confirming hyperventilation as the major cause of drowning.

In comparison to marine mammals, humans are poor divers because of severe physiological constraints which limit their breath-hold time, diving depth, and ability to conserve body heat. Although under special circumstances humans can achieve unusually long breath-hold time and reach exceptional depth with a single breath, the sustainable working time and depth are only about 1 minute and 5 metres, respectively. Hypothermia inevitably results in divers working in the ocean. Without thermal protection, the intolerable limit of 35°C is reached within 30 minutes in winter (10°C) water and within 60 to 90 minutes in summer. Nevertheless, effective harvest work can be performed by humans in the ocean, and recreational benefits enhanced when these physiological limitations are respected. An unusual circulatory state exists during head-out water immersion in that there is a sustained increase of stroke volume. This results in 30% increase in cardiac output when the subject is resting in thermal neutral water, indicating a substantial overperfusion for the oxygen requirement. Furthermore, animal experiments showed that the elevated blood flow is preferentially channeled to the liver, fat, and the organs in the splanchnic region. Moreover, head-out water immersion enhances the ability of kidney to eliminate excess body fluid.

Breath-hold and scuba diving are generally termed 'sports diving'. Breath-hold diving (free diving, skin diving) is an ancient practice which offers divers freedom and simplicity. These advantages are offset, however, by the inability of humans to stay under water for extended periods. Humans have practised breath-hold diving as a profession for centuries and it is still practised today, though only in a few places. The advent of artificial materials rendered shell harvesting along the northern Australian shores economically uncompetitive, scuba diving displaced old ways of gathering sponges in the Aegean Sea, and the cultured-pearl eliminated high risk pearl diving in the Tuamotu archipelago. Undoubtedly, industrialisation, and with it new job opportunities, pollution, depletion of resources, and general social attitude, all contribute to the continuing decline of breath-hold diving as a profession.

In 1965, a symposium held in Tokyo (Rahn & Yokoyama 1965) called attention to the original work of Teruoka (1932); the first scientific description of diving equipment, diving pattern, and gas exchange in professional breath-hold divers in Japan. Studies on divers in Japan (ama, sea-women; katsugi, sea-men) and Korea (hae-nyo, sea-women) during the last 30 years have contributed significantly to our knowledge of the physiology of breath-hold diving. Combined results of these studies and laboratory investigations represent our present understanding of human limits in diving. These studies form the basis of this review.

Diving with respiratory aids, as in scuba or surface supplied diving, extends the time of submergence, making possible extended underwater exploration, inspections, installations and repairs. Scuba also offers the diver complete freedom and mobility, and thus seems to be an ideal solution to the shortcomings of breath-hold diving. However, overextending the dive time and/or depth could be dangerous when combined with faulty equipment; ill preparation, poor practice, or lack of common sense can be deadly. Many excellent sources of reference for scuba diving (Erickson 1972; Miller 1979; Petersen 1984) are available.

Bert's work (1870) on animals led to much further research which concentrated mainly on the comparative aspects of animals' ability to with-

stand lack of oxygen, their 'diving reflexes', and the oxygen-conserving effect of breath-hold diving. Scholander (1940, 1963) and others (Hong & Rahn 1967) pointed out the drastic physiological changes that occur during a breath-hold dive. Several reviews have appeared since then (Elsner & Gooden 1983; Hicky & Lundgren 1984; Hong 1976, 1987; Lin 1982, 1986, 1987b; Strauss 1970). This review will firstly focus on the physiological limits of humans as divers, then briefly review the physiology of breath-hold diving, and finally discuss its clinical implications.

With the increasing popularity of recreational diving, accidents occur more frequently (Craig 1961a,b, 1976). Those associated with scuba diving are complex, involving problems with equipment, inert gas narcosis, oxygen toxicity, and, the most troublesome, decompression sicknesses. Treatments are specific, depending on the nature of the affliction, and many accidents could be avoided by good diving practices. Accidents associated with skin diving, in contrast, are mostly physiological, and usually involve drowning or near drowning.

1. Limitations of the Human as a Diver

1.1 Diving Time

As mentioned earlier, humans can only stay underwater for a brief time. Professional divers perform repetitive dives, to a limit of 60 seconds in Korea (Hong et al. 1963; Park et al. 1982), and also in Japan (Teruoka 1932; Shiraki et al. 1985). In the laboratory, where breath-hold, exercise, and water immersion can be separately studied, much longer breath-hold time is demonstrated.

Theoretically, breath-hold time can be calculated from oxygen supply-demand relationships (Klocke & Rahn 1959), as:

Breath-hold with oxygen:

$$BHT = \frac{VC\ (BTPS)}{\dot{V}O_2\ (STPD)} \times \frac{P_b - 47}{863}$$

Breath-hold with air:

$$BHT = \frac{TLC\ (BTPS) \times FAO_2}{\dot{V}O_2} \times \frac{P_b - 47}{863}$$

where BHT represents breath-hold time in minutes; VC and TLC, the vital capacity and total lung capacity in millilitres at BTPS (body temperature and pressure, saturated with water vapour) respectively; $\dot{V}O_2$, the oxygen consumption in ml/min at STPD; FAO_2, the fractional alveolar concentration of oxygen; $P_b - 47$, the barometric pressure (in torr) less the water vapour pressure at 37°C; and 863, the constant for converting a gas volume from BTPS to STPD. VC instead of TLC is used in the oxygen breath-hold experiment. The assumption here is that residual volume limits the shrinkage of the lung. In air breath-hold, TLC × FAN_2 is greater than residual volume, regardless of breath-hold time. Estimated from the oxygen supply in the lung alone, a breath-hold time of 4 minutes with air and 16 minutes with oxygen should be possible for an average resting man at sea level (vital capacity, 4.78L; residual volume, 1.19L; and $\dot{V}O_2$, 250 ml/min). Breath-hold time should be even longer if the calculation includes usable oxygen in the blood, and in fact such times have been demonstrated in subjects breathing air (Hong et al. 1970, 1971) and breathing oxygen (Klocke & Rahn 1959; Schneider 1930). A student of Schneider's held his breath for 15 minutes 13 seconds after a period of hyperventilation with air, followed by 3 deep breaths of pure oxygen (Schneider 1930). Mithoefer (1965) noted that under the same conditions another student at the same university held his breath for 20.1 minutes (table I).

Table I. Published breath-hold records

Breath-hold with oxygen		
20′05″	Frechette	Schneider (1930)
15′13″	A student	Schneider (1930)
14′00″	Subject HR	Klocke & Rahn (1959)
13′48″	Subject MT	Klocke & Rahn (1959)
13′00″	Subject SH	Klocke & Rahn (1959)
Breath-hold with air		
4′30″	Subject SKH	Hong et al. (1970)
4′00″	A student	Hong et al. (1971)

Although oxygen supply-demand relationships accurately predict breath-hold breaking points, they can only be achieved by using all factors known to prolong breath-hold time, and by overcoming

Table II. Alveolar pCO_2 at physiological breaking point of breath-hold with oxygen, at rest

BHT (sec)	$pACO_2$ (mm Hg)	$paCO_2$[a] (mm Hg)	Note	Reference
80	48.8			Kobayashi & Sasaki (1967)
37	49.3	47.6		Agostoni (1963)
68	48.3	46.9	High $\dot{V}_E$	Agostoni (1963)
62	48.4	47.1	80%VC	Agostoni (1963)
66	49.3	47.7	20%VC	Agostoni (1963)
104	48.6	47.9		Lin et al. (1974)
	48.6			Douglas & Haldane (1909)

a Arterial carbon dioxide pressure estimated from $pACO_2$ with correction of sampling delays.

Abbreviations: pCO_2 = carbon dioxide pressure; BHT = breath-hold time; $pACO_2$ = alveolar carbon dioxide pressure.

physical and psychological discomfort and noxious sensations. The most effective means of extending breath-hold time is hyperventilation before breath-hold, which lowers alveolar pCO_2, and hence delays the feeling of needing to resume breathing. Severe hyperventilation prior to breath-hold is the single most dangerous practice in open water, causing blackout during the ascent phase. Various factors that affect breath-hold time have been reviewed by Mithoefer (1965) and Lin (1987a). Note that these long breath-hold times were achieved in the laboratory at a resting state and that breath-hold time is inversely proportional to oxygen consumption (Klocke & Rahn 1959; Lin et al. 1974). Therefore, such times cannot be achieved in a dive, which involves increased oxygen consumption. However, these findings show that humans can survive without breathing for 15 minutes or longer, provided oxygen consumption is lowered. In fact, survivals after 40 minutes' submergence in cold water have been reported. This emphasises that aggressive cardiopulmonary resuscitation should be attempted in drowning victims, especially when cold water is encountered.

Mechanisms leading to the termination of a breath-hold are complex. Researchers have identified 2 breaking points. The *conventional breaking point*, or simply the *breaking point*, is reached when one feels breath-hold can no longer be continued. The time taken to reach this point is the breath-hold time mentioned above. However, by this criterion there are wide inter- and intraindividual variations in breath-hold times, mainly because of the involvement of subjective judgement. Breaking point varies from less than 20 seconds (Schneider 1930) to 270 seconds (Hong et al. 1970) when subjects are told to 'expire once as deeply as possible, then inspire fully and hold the breath as long as possible', i.e. without hyperventilating. Psychological factors are recognised as the major determinants of conventional breaking point (Hill & Flack 1908; Schneider 1930; White 1920), and undoubtedly contribute to the wide range of breath-hold times reported in the literature.

In contrast, another breaking point, the *physiological breaking point*, is sharply defined by chemical stimulus alone. At such a time the desire to breathe returns, but breath-hold can still be continued by conscious effort. Physiological breaking point, in contrast to the conventional breaking point, varies within a narrow range (Lin et al. 1974). Involuntary ventilatory activities occur when the physiological breaking point is reached, but voluntary inhibition of glottis opening is still possible. A number of terms have been used to identify this stage, such as the end of the 'easy-going phase' (Dejours 1965), the 'desire to breathe' (Douglas & Haldane 1909), 'want of oxygen' (Hill & Flack 1908), 'diaphragm contraction' (Agostoni 1963; Kobayashi & Sasaki 1967; Noble et al. 1971), 'involuntary ventilatory activity' (Lin et al. 1974), or simply the sensation of 'air hunger'.

Physiological breaking point is the signal divers should pay close attention to. Although breath-hold

can still be continued by conscious effort, drowning may occur during the ascent phase of the dive as a result of extreme hypoxia. The desire to breathe occurs at an alveolar pCO_2 which is remarkably similar in various breath-hold conditions. The critical arterial pCO_2 of the brain stem has been estimated to be between 47 and 48 torr (Lin 1987a; table II), and is reached after 30 to 40 seconds. Professional breath-hold divers in Japan and Korea accomplish much useful work in this time. An earlier survey (Hong et al. 1963) of women divers who wore simple cotton suits, and 2 new studies (Park et al. 1983b; Shiraki et al. 1985) of divers in wetsuits showed that only about half of the diving time is available for bottom activity for a 5-metre dive, while only a quarter of the time is available in a 10-metre dive, with the remainder spent on ascent and descent (table III).

The diving pattern described above pertains to a sustained period of repeated dives. Longer dive times of 90 seconds (Scholander et al. 1962), 118 seconds (Teruoka 1932), and 155 seconds (Cross 1965) in open sea have been reported.

It is clear, then, that humans are severely limited in their ability to stay underwater. Nonetheless, water activities are popular; hence, there is a need for a proper understanding of the physiology of diving.

1.2 Diving Depth

Brief diving time is obviously one factor preventing divers from reaching great depth. Professionals dive most frequently to a depth of less than 10 metres, and even with assistance diving depth rarely exceeds 20 metres. Tuamotu pearl divers (men) are the deepest working breath-hold divers known, regularly reaching depths greater than 30 metres (Cross 1965). They also have a lot of diving accidents, indicating the increased depth is, in fact, counterproductive. They are the only breath-hold divers who experience a decompression sickness known as taravana (literally 'went crazy'). By far its most common symptoms are, according to Cross (1965), vertigo, nausea, and, less frequently, mental anguish. Paulev (1965) demonstrated in a laboratory setting that it is possible to induce decompression sickness by repetitive breath-hold diving when the dive time-depth product exceeds certain limits.

Just as humans can achieve astonishingly long breath-hold time under some unusual circumstances, so can they reach great depths with a single breath. It was predicted that the depth limit for humans should be about equal to the total lung capacity/residual volume ratio (in atmospheres). If total lung capacity is allowed to compress down to residual volume during a dive, the depth limit (D, in metres) should be:

$$D = P_b \times (TLC/RV - 1) \times 10$$

where P_b is the barometric pressure expressed in atmospheres, and 10 represents the depth (in metres) equivalent sea water of 1 atmosphere.

Table III. General patterns of diving to 5- and 10-metre depth by wetsuit divers

Dive time	Korean women[a]		Japanese men[b]
	10m dive	5m dive	5m dive
Single dive time (sec)	43 (100%)	32 (100%)	39 (100%)
descent time (sec)	19 (44%)	9.3 (29%)	8 (20%)
ascent time (sec)	12 (28%)	8.0 (19%)	8 (20%)
bottom time (sec)	12 (28%)	16.5 (52%)	23 (60%)
Single surface time (sec)	85	46	42
No. of dives/h	28.1	46.2	44.4

a Park et al. (1983b).
b Shiraki et al. (1985).

Depth limit should be in the range of 30 to 40 metres, according to this formula. However, when Craig (1968) reviewed breath-hold diving depths up to 1967 he found that the record of 64.8 metres considerably exceeded this estimate. This is possible, as was found later, because the residual volume diminishes (increasing the total lung capacity/residual volume ratio) during breath-hold dive by the chestward displacement of blood. Over the years, depth records have increased, and it now looks as though the limit may have been nearly reached. By 1976, Mayol reached 86m or 284ft (Ricci & Marroni 1976). Later Maiorca made an 87m record, and his latest record is now 91m (Maiorca, personal communication).

1.3 Hypothermia

Another limitation on human in-water activity is the fact that hypothermia inevitably follows prolonged immersion. The water temperature at which the human body neither loses nor gains heat is called 'thermoneutral temperature'. This temperature is 35°C, which is substantially higher than that of ocean water, even in mid-summer. Thermal drain continues as long as a person is in water. Hypothermia affects the performance of all organ systems, and its contribution to drownings is well known (Keatinge 1969). Insulation is the only protection against hypothermia, as heat flow is dictated by the thermal gradient and property of the fluid environment, and the thermal conductivity of water is 25 times that of air, causing rapid heat loss along the thermal gradient. In addition, body movement in water reduces the thermal insulation of the body shell, thus facilitating heat loss from the core to water during diving (Keatinge 1961; Park et al. 1984; Rennie et al. 1980; Veicsteinas & Rennie 1982). Cardiac irregularities, impairment of rational thinking, sensory and motor degradation, loss of consciousness, and drowning all occur when body temperature falls below 35°C (Webb 1976). Because of this, professional breath-hold divers stop harvesting when body temperature reaches 35°C (Kang et al. 1965) and allow their bodies to warm up before resuming. Diving time was increased with the introduction of the wet suit (Kang et al. 1983; Park et al. 1983b; Shiraki et al. 1986a). As a result, the previous practice of breaking up dives into 1-hour shifts in summer, or much shorter shifts in winter, has been replaced by 2- to 3-hour shifts, which are not limited by hypothermia.

Besides increasing the risk of hypothermia, cold water shortens breath-hold time. Sterba and Lundgren (1985) and Hayward et al. (1984) reported a direct correlation between breath-hold time and water temperature, and claimed that reduction of breath-hold time in cold water corresponds closely to elevated oxygen consumption. Lessons on the thermal properties of water and their consequences should therefore be a part of the education of sports diving.

2. Physiology of Breath-Hold Diving

Breath-hold diving consists of water immersion up to the neck, breath-hold, submersion, and underwater exercise. It involves only modest pressure, unless carried out to extremes, which nonetheless elicits pronounced physiological changes, as do all components of a dive.

2.1 Water Immersion

Water immersion to the neck precedes breath-hold diving, and in fact divers spend most of the time floating on the surface of the water (table III). Head-out immersion in thermoneutral water produces a variety of prominent physiological changes which become even more complex in cold water. The physiological effects of water immersion have been reviewed recently, with regard to respiration (Lundgren 1984), circulation (Krasney et al. 1984; Lin 1984), body fluid regulation (Epstein 1976, 1978, 1984), and temperature regulation (Hong 1984; Nadel 1984; Park et al. 1984).

2.1.1 Cardiovascular System

Head-out water immersion causes blood shift toward the upper body, resulting in an increased central blood volume which, in turn, causes en-

gorgement of the vasculatures in the thoracic region. Central venous pressure rises as a consequence. Various estimates indicate that central blood volume increases by 300 to 700ml (Arborelius et al. 1972; Hong et al. 1969). Reasons for its increase include: (a) hydrostatic compression, which reduces venous capacity in the lower extremities, as well as displacing abdominal contents chestward; (b) the fact that the density of sea water is similar to that of human tissues, which effectively renders the diver weightless (or neutrally buoyant); and (c) a negative transthoracic pressure of about 14 to 20mm Hg, which also promotes redistribution of circulating blood toward the upper body. Part of the increased central blood volume resides within the heart and associated great vessels. By x-ray technique the increase in heart volume has been estimated to range from 180 to 247ml (Lange et al. 1974; Risch et al. 1978).

Dimensional expansion of atria, pulmonary arteries and veins is known to: (a) activate cardiac mechanoreceptors that, via vagal afferents, inform the hypothalamus of hypervolaemia where in fact, the total blood volume remains unchanged; (b) encroach on pulmonary air space and alter respiratory mechanics; and (c) enhance ventricular diastolic loading. These changes cause functional changes in the circulatory and respiratory, as well as the renal systems.

Stroke volume rises, on average, by about 30% during head-out immersion in or near thermoneutral water (Lin 1984). This increase has been demonstrated by dye-dilution (Arborelius et al. 1972; Hood et al. 1968), acetylene rebreathing (Bazett 1937; Begin et al. 1976), CO_2 rebreathing (Farhi & Linnarsson 1977; McArdle et al. 1976; Rennie et al. 1971), and impedance cardiography (Matsuda et al. 1981; Shiraki et al. 1986b). Review of the literature suggests that altered preload is responsible for the increased stroke volume, and that afterload and myocardial contractility play insignificant roles in water-immersion (Lin 1984).

Heart rate changes little during immersion in thermoneutral water. Review of 13 reports involving 100 male subjects indicates that it is either unchanged or decreases only slightly (Lin 1984). Although cold water depresses it (Keatinge & Evans 1960; Knight & Horvath 1987; Rennie et al. 1971), several opposing factors act together to determine heart rate during immersion. Other factors being equal, the rising central blood volume and pressure should trigger a tachycardial response through the Bainbridge reflex, but concurrent elevation of arterial pressure, by the increased stroke volume and cardiac output, acts against an increase in heart rate. Cold water reduces heart rate, but increased metabolic demands during diving (staying afloat in place, replacing heat loss, and exercise) cause it to rise. The predive heart rate in open sea is higher than the resting value in air (Hong et al. 1967; Irving 1963; Scholander et al. 1962).

Cardiac output increases to a similar extent as stroke volume in thermoneutral water. However, when the water temperature falls, so does heart rate, leading to only a slight increase in cardiac output, or maybe even a decrease (Lin 1984; Rennie et al. 1971).

As noted above, cardiac output is increased and redistributed during head-out immersion. The redistribution pattern is of particular interest; studies in the dog have shown a marked increase in blood flow in the gastrointestinal tract, liver, heart, skin, and respiratory muscle during head-out immersion in thermoneutral water (Hajduczoc et al. 1985; Krasney et al. 1982). Of these changes, the increase in liver blood flow may prove clinically useful.

2.1.2 Respiratory System

Lung Volume

Immersion up to the neck does not affect tidal volume or residual volume, but vital capacity is reduced by about 5 to 10%, mainly because of a decrease in expiratory reserve volume. Several factors contribute to decreased lung volume during immersion; among these, the chestward pooling of blood and chestward displacement of the diaphragm appear most important (Agostoni et al. 1966; Buono 1983; Dahlback 1975; Hong et al. 1969).

Respiratory Work

The increased central blood volume during immersion engorges and stiffens lung tissues, which decreases lung compliance; compresses small airways, elevating airway resistance; and increases air trapping and closing volume. Together, these alterations increase the load on respiratory muscles. Hong et al. (1969) estimated that resting respiratory work during head-out immersion was elevated by 65% over that at rest in air.

Gas Exchange

Head-out water immersion apparently has no effect on gas exchange at rest or during exercise (Dressendorfer et al. 1976), as opposing factors prevent marked changes. One might expect an increase in gas exchange during water immersion because of the increase in central blood volume and elimination of blood flow dependence on gravity, thus promoting homogeneous perfusion of the lungs. Although the same mechanism does not operate for gas distribution within the lungs, a reduction in alveolar ventilation to blood flow maldistribution was nevertheless noted. This advantage, however, is offset by increased intrapulmonary shunt (Cohn et al. 1967), trapped gas, closing volume, and lung stiffness (see above).

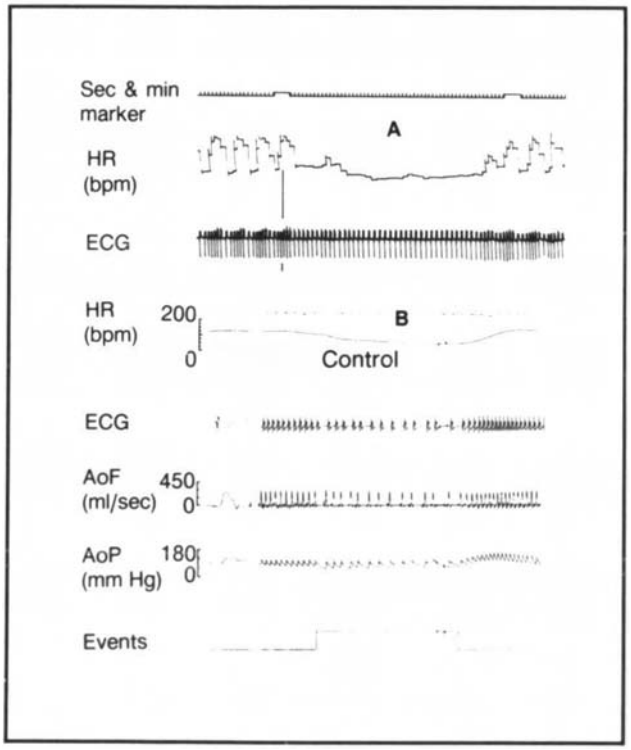

Fig. 1. Comparison of bradycardial response of a human volunteer (A) and a dog (B) trained to immerse the snout on command (from Lin 1983a). HR = heart rate; ECG = electrocardiogram; AoF = aortic blood flow; AoP = aortic pressure.

2.1.3 Renal Function

Extensive reviews have established the diuretic effect of water immersion (Epstein 1976, 1978; Gauer & Henry 1976). It suffices to say that consequent to the increased urine output, plasma volume falls. Secondary effects, such as decrease in work capacity, orthostatic intolerance and other disturbances in circulatory function could result. Dehydration is a potential problem during a prolonged stay in water, even at thermoneutral temperature.

2.1.4 Thermal Regulation

Thermoregulation in water has been extensively reviewed, although the topic is beyond the scope of this review. However, it should be pointed out that exercise drastically alters thermoregulation in water. Body temperature may fall, even when heat production is increased by 10 to 15 times above the basal temperature, as in intense swimming, if water temperature is sufficiently low and insulation is inadequate (Keatinge 1969; Nadel 1984; Park et al. 1984; Veicsteinas et al. 1982). Thermal regulation, adaptation, and deadaptation after the introduction of the wetsuit, have been investigated in Korean divers (Kang et al. 1965, 1983; Park et al. 1983b; Rennie et al. 1962; Rennie & Hong 1985).

2.2 Breath-Holding (Apnoea)

2.2.1 Circulatory Changes

Slowing of the heart rate and widespread vasoconstriction stand out as the most pronounced physiological changes brought on by breath-hold. 'Diving reflex' is a term used for describing circulatory changes during the act of breath-holding. During submersion, profound bradycardia occurs in diving mammals, causing a marked fall in card-

iac output, but the intense peripheral vasoconstriction that follows apnoea prevents arterial blood pressure from falling. The consequence is a preferential delivery pattern of blood flow to the brain and the heart. Similar responses occur in animals that are natural divers, as well as in many non-natural diving terrestrial mammals (Lin 1982), and are thought to play an important role in conserving oxygen during a dive. Humans also exhibit diving bradycardia and vasoconstriction, though in a somewhat attenuated form (Hong et al. 1971; Lin 1983a; Lin et al. 1983a,b; Song et al. 1969), and their circulatory responses lack the intensity and promptness that are required for an effective conservation of oxygen during diving (Hong et al. 1971; Lin et al. 1975). For this reason, humans are considered poor breath-hold divers.

The phenomenon of diving bradycardia is by far the most frequently studied of the circulatory changes, both in the field and in the laboratory. Bradycardia develops promptly upon breath-hold in humans, but is slow to reach its lowest point (Lin et al. 1983a; fig. 1). In comparison, maximal bradycardia is attained within a few cardiac cycles following the onset of apnoea in diving mammals (Elsner 1968), as well as non-diving mammals such as dogs (Elsner et al. 1966; Lin 1983b; Lin et al. 1983) and rats (Lin 1974; Lin & Baker 1975).

Figure 2 summarises the survey of the breath-hold induced bradycardia in humans, which may be very intense in some instances. It is evident that bradycardia occurs during breath-hold both at rest and during exercise, although the response is greatly increased in men exercising while holding their breath. Those indicated by an 'a' are single-subject records during exercise. The heart rate levels off at 55 beats per minute during a 50-metre underwater swim (Jung & Stolle 1981). Such activity requires between 5 and 10 times the resting oxygen consumption. In some cases, the decrease may be as much as 90% of pre-breath-hold heart rate (Asmussen & Kristiansson 1968; Irving 1963; Stromme et al. 1970). Arnold (1985) recently reported several equally marked breath-hold bradycardias in human subjects at rest.

Elevation of vagal tone is responsible for diving

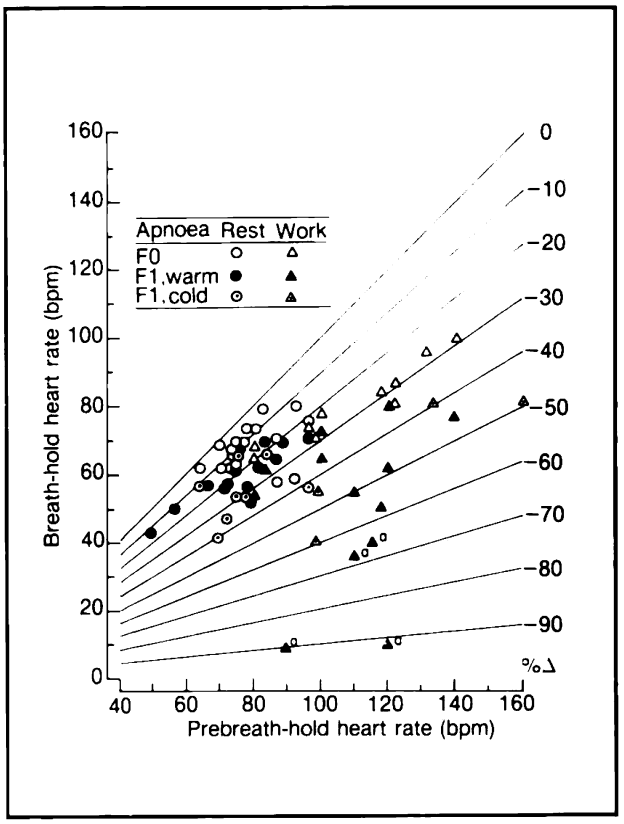

Fig. 2. Bradycardial response in humans at rest and during exercise (from Lin 1982).

bradycardia, as was deduced by differential autonomic nervous blockade (Berk & Levy 1977; Finley et al. 1979; Heistad et al. 1968), while the sympathetic branch of the autonomic nervous system plays a minor role. More convincing evidence is, however, derived from animal data, both in diving (Andersen 1966; Murdaugh et al. 1961; Scholander 1940) as well as non-diving species (Gooden et al. 1974; Lin 1974; Lin et al. 1972, 1983).

Cardiac output changes little during breath-hold and face immersion, as demonstrated by a variety of techniques (Hong et al. 1971; Lin 1982; Lin et al. 1983b). Changes in stroke volume from the pre-breath-hold values are also insignificant in most studies, indicating that the enhanced preload of the heart (low heart rate, high central blood volume and central venous pressure) is offset by peripheral vasoconstriction. Myocardial contractility is probably reduced during apnoea, as was deduced from

systolic time intervals (Ferrigno et al. 1986; Gross et al. 1976).

Humans differ from diving mammals and birds (Blix & Folkow 1983) in that hypertension develops progressively, in proportion to the duration of breath-hold. Since there is no evidence of increased cardiac output during breath-hold in humans (Hong et al. 1971; Lin 1982; Lin et al. 1983b), hypertension indicates that vasoconstriction occurs in most vascular beds. It is known that forearm blood flow decreases by 7 to 50% from pre-breath-hold level (for review see Lin 1982). Whether vasoconstriction during breath-hold results in selective patterns of blood flow, as occurs in diving mammals, is uncertain. There is evidence that peripheral vasoconstriction exists during breath-hold in humans, though not approaching zero flow, as occurs in most organs of the diving species. Calculations from available data shows that total peripheral resistance increases range from 26 to 33% (Hong et al. 1971; Lin et al. 1983b; Whayne et al. 1972). In comparison, 4- to 10-fold increases in total peripheral resistance are reported in diving species (Blix & Folkow 1983). For this reason, it is doubtful that humans are able to effectively conserve oxygen during breath-hold (Hong et al. 1971; Lin et al. 1975).

2.2.2 Pulmonary Gas Exchange

Gas exchange during breath-hold involves temporal changes in alveolar gas concentration and lung volume, neither of which are easily measured in natural diving conditions. In earlier studies only the pre- and post-breath-hold alveolar samples were available. Measurement of alveolar gas composition during breath-hold has been made simpler by a sequential sampling device, which also makes possible the estimation of dynamic changes in lung volume during a dive (Lanphier & Rahn 1963a). By knowing the initial alveolar gas composition and volume and changes thereafter, one can calculate the rate of pulmonary gas exchange during breath-hold.

Breath-Hold at a Constant Ambient Pressure

Breath-hold in the laboratory involves no change in ambient pressure. Immediately before breath-hold, alveolar pO_2 rises, and alveolar pCO_2 falls following a deep inspiration of fresh air. The rate of oxygen disappearance from the lung is approximately linear for the first 2 minutes of breath-hold and then diminishes (Hong et al. 1970, 1971; Lanphier & Rahn 1963a; Tibes & Stegemann 1969). Despite this, oxygen continues to be transferred from the lung into the blood, although the diminished pO_2 in the lungs cannot keep arterial blood fully saturated. Consequently, arterial oxygen content declines progressively in humans (Hong et al. 1971) as well as in dogs (Lin et al. 1975).

Initially, CO_2 is transferred rapidly from the blood into the alveolar space because alveolar pCO_2 is low, and it rises quickly to approach or equal that of the blood. It may continue to rise, exceeding that of the blood, as a result of the concentrating effect of the shrinking lung volume, caused by the continuous transfer of oxygen from the lung to the blood. The result is diminished movement of CO_2 from the blood to the lung, eventually stopping or even beginning to transfer in the reverse direction (Mithoefer 1959). This peculiar gas exchange pattern results in an extremely low alveolar gas exchange ratio during breath-hold (Craig & Harley 1968; Hong et al. 1971; Lanphier & Rahn 1963a).

Breath-Hold During a Compression and Decompression Cycle

In a dive, alveolar pO_2 rises continuously because of the compression effect, and thus remains elevated throughout the descent. Consequently, the transfer of oxygen from the lung into the blood proceeds at a normal rate. For the same reason, CO_2 is likely to be transferred in the opposite direction from normal gas exchange until well into the ascending phase of the dive. The maintained transfer of oxygen and retention of CO_2 in the blood during descent and at the bottom lead to low alveolar pCO_2 and pO_2 at the end of a dive, as the lung expands during ascent. This paradoxical result, originally observed by Teruoka (1932), has

since been confirmed in the field (Hong et al. 1963), in the hyperbaric chamber (Lanphier & Rahn 1963b) and in the US Navy submarine escape training tank (Openshaw & Woodroof 1978). It is during the ascending phase that most accidents occur as a result of hypoxia.

2.2.3 Underwater Exercise

There is as yet no reliable estimate of the energy expended by professional divers during their harvesting activity. Such estimates would certainly be made more complicated by diving patterns, equipment, and thermal conditions. Various aspects of swimming, including energetics, have been reviewed recently (Lavoie & Montpetit 1986).

3. Applied Physiology

3.1 Immersion

Water immersion produces an unusual circulatory state, which is characterised by sustained increase in stroke volume, and which leads to a variable increase in cardiac output. Cardiac output is also redistributed preferentially toward the liver, fat, skin, and splanchnic region. Of particular importance is the discovery of a 5-fold increase in hepatic arterial blood flow in dogs during vertical head-out water immersion, prominent diuresis also occurring. Based on the above observations, the following applications of head-out water immersion appear feasible:

1. As a ground-based simulation for weightlessness. Together with bed rest studies, head-out water immersion provides a significant data base to account for physiological changes during and immediately after space travel (Blomqvist 1983; Greenleaf 1984; Sandler 1976).

2. As a diuretic. It has been shown to reduce excess body fluid in decompensated cirrhotic patients (Bichet et al. 1983; Epstein 1978, 1984) in congestive heart failure and even heart transplant patients.

3. Head-out water immersion promotes inert gas elimination by the sustained increase in cardiac output (Balldin & Lundgren 1972).

4. It shortens decompression time by blood flow redistribution to skin and fat tissues.

5. It could be used for preferential delivery or elimination of drugs to or from the liver.

If blood flow to the liver were increased, as occurs during head-out water immersion, the concentration of drugs in the general circulation could be correspondingly reduced in order to achieve the same rate of delivery to the liver. This is especially important in the administration of toxic drugs. Similarly, head-out water immersion, by increasing hepatic blood flow, could accelerate the elimination of toxic substances from the circulation; barbiturates or alcohol for example. These possibilities are currently being tested.

3.2 Breath-Hold

3.2.1 Paroxysmal Supraventricular Tachycardia

Breath-hold induces slowing of the heart via the elevation of vagus activity. Since the vagus nerve innervates the supraventricular region of the heart, increasing vagus activity by breath-hold could be used to suppress tachycardia of supraventricular origin.

Previously methods of treating paroxysmal supraventricular tachycardia involved drugs such as digitalis, propranolol, or quinidine, electroversion, or increasing vagal tone. Vagal tone can be increased by vasopressor agents, carotid massage, the Valsalva manoeuvre, eyeball compression, or gag reflex; all physiological methods. Breath-hold turned out to be the simplest and most effective and specific way of producing increased vagal tone, as shown by a number of studies. Whayne and Killip (1967) reported that simulated diving eliminated for several minutes multiple ventricular premature contractions in one patient. Following this report, the diving reflex has been shown to eliminate paroxysmal supraventricular tachycardia in infants (Hamilton et al. 1979; Heistad et al. 1968; Sperandeo et al. 1983; Whitman et al. 1977), children (Whitman & Zakeosian 1976) and adults (Hunt et al. 1975; Mathew 1978, 1981; Pickering & Bolton-Magge 1975; Wildenthal & Atkins 1979; Wil-

denthal et al. 1975). Breath-hold bradycardia can be effectively induced by face immersion in cold water (about 10°C) or breath-hold with a cold towel applied to the face.

The use of the diving reflex may not be hazard-free, however. Stimulation of the nerve pathways mediating the diving reflex may evoke serious or fatal arrhythmias in patients with pre-existing organic disorders. Wildenthal and Atkins (1979) cautioned that the first treatment should always be made under conditions of careful monitoring, with emergency equipment available.

3.2.2 Near Drowning

The distinctive gas exchange pattern, as summarised above, clarifies the causes of shallow water accidents. During descent and at the bottom of a dive, the compression effect keeps pO_2 high despite decreasing alveolar oxygen concentration, but during ascent the lung expands and oxygen concentration falls markedly. Arterial pO_2 approaches that of venous blood on surfacing in a 60-second dive. In the normal course of events, divers depend only on hypercapnic stimuli as a signal for surfacing (pO_2 is high at the bottom). However, an accident may occur when a person hyperventilates before diving, as under such conditions oxygen depletion can occur, without the diver's knowledge, before the arterial pCO_2 can build up to a level that normally signals the desire to resume breathing. Blackout often occurs without warning during the ascent phase. Shallow water blackout still occurs at an alarmingly high rate (approximately 7000 per year in the US). It continues despite the publication of detailed scientific findings attributing blackout to hyperventilation before diving (Craig 1961a; Hong et al. 1963; Lanphier & Rahn 1963a), and the publication of repeated warnings (Craig 1976; Hong 1976; Hong & Rahn 1967). The perils of excessive hyperventilation should be emphasised in all swimming and diving programmes.

Although there is no evidence of human ability to conserve oxygen while diving, it is important to note that apnoea (a component of diving reflexes) occurs when a person is accidentally dropped into water, thus preventing water inhalation. The cardiovascular response that follows immersion enhances the chance of revival, even after what seems like hopelessly long apnoea. The odds of revival improve when conditions favour rapid chilling of the body, such as cold water, a thin-bodied or very young victim (small body mass). Hypothermia reduces metabolic requirements and redistribution of blood flow, minimising heart and brain damage. Revival is possible even after 40 minutes' submersion (Nemiroff 1977; Siebke et al. 1975). Siebke et al. (1975) reported the revival of a 5-year-old Norwegian boy who regained full cerebral function after having been under ice water for more than 40 minutes. Here again, it must be said that aggressive cardiopulmonary resuscitation (CPR) should be attempted in drowning incidents, especially when cold water is involved.

3.2.3 Cardiac Risk

Cardiac arrhythmia often develops in breath-hold diving, especially in cold water (Hong et al. 1967; Jung & Stolle 1981; Olsen et al. 1962; Sasamoto 1965; Scholander et al. 1962). The most common forms of arrhythmia are those associated with enhanced cardiac vagal activity, such as a shift in the normal pacemaker locus and an altered atrioventricular condition. Occasionally, idioventricular rhythm, premature atrial beats, and premature ventricular beats are also observed. It is, therefore, prudent to obtain health clearance before starting breath-hold diving as exaggerated breath-hold bradycardia, cardiac arrest and syncope may develop in persons with underlying cardiac diseases. Wolf (1964) suspected that diving bradycardia may have been related to the sudden death of an elderly man while he was washing his face.

3.2.4 Integrity of Autonomic Nervous System

Breath-hold or face immersion produces elevated vagal input to the heart and sympathetic activity to the peripheral vessels. It is, therefore, a simple method for assessing the integrity of autonomic nervous system in certain diseases (Bennett et al. 1976; Gooden et al. 1978).

3.2.5 Sudden Infant Death Syndrome

The primary cause of sudden infant death syndrome remains unresolved. Apnoeic spells during sleep are a common problem in preterm infants. Bradycardia occurs promptly upon apnoea, whatever the cause may be. Exposure of the face to cold air and pressure on the eyeballs have been used to test the chronotropic liability of infants at risk (Gandevia et al. 1978; Kahn et al. 1983). These procedures cause trigeminal and vagal-mediated bradycardia similar to that of the diving response. Exaggerated cardiac response following provocation indicates a child at risk of sudden infant death syndrome.

3.2.6 Upper Airway

Studies of both humans and animals have shown that mucosal stimulation of the upper airways can induce apnoea and diving responses. This manoeuvre may therefore place some patients at risk. Such situations may arise during examination of upper airways (for dentistry, laryngoscopy, bronchoscopy); irrigation of the nose, sinuses or pharynx; intubation; aspiration of nasal or tracheal secretions; and application of aerosol through upper airways. Thus, caution should be exercised in carrying out these procedures, especially in patients with underlying cardiac abnormalities.

4. Conclusion

The recreational benefits of human interaction with the water is well recognised. However, man's enjoyment in water depends on his recognition of the limitations of the human as a diver. This knowledge has been gained from the example of professional breath-hold divers in the field, and from the study of volunteers in the laboratory. Avoidance of human tragedy in water relies upon the understanding of the physiology of diving, especially that of breath-hold diving.

Studies on gas exchange during breath-hold has shown that shallow water blackout often occurs during ascent when a diver hyperventilates excessively just prior to a dive. This should be brought to the attention of those teaching underwater activities. Protracted water immersion should be avoided since hypothermia inevitably results in open water. The prominent physiological changes that occur during immersion and breath-holding show potential clinical applications. Among these, the suppression of paroxysmal supraventricular tachycardia by breath-hold and face immersion appears to be underutilised at present. Its simplicity and proven safety warrant a large scale clinical trial. Head-out water immersion has been shown to be an important investigative tool as well as being clinically useful. The induction of diuresis, enhancement of cardiac peformance, and increased perfusion of liver by head-out water immersion deserve further investigation, not only into the mechanisms of action but also into their specific clinical applicabilities.

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References

Agostoni E. Diaphragm activity during breath-holding: factors related to its onset. Journal of Applied Physiology 8: 30-36, 1963

Agostoni E, Gurtner G, Rahn H. Respiratory mechanics during submersion and negative pressure breathing. Journal of Applied Physiology 21: 251-258, 1966

Andersen HT. Physiological adaptations in diving vertebrates. Physiological Reviews 46: 212-243, 1966

Arborelius Jr M, Balldin UI, Lilja B, Lundgren CEG. Hemodynamic changes in man during immersion with head above water. Aerospace Medicine 43: 592-598, 1972

Arnold RW. Extremes in human breath-hold, facial immersion bradycardia. Undersea Biomedical Research 12: 183-190, 1985

Asmussen E, Kristiansson NG. The 'diving bradycardia' in exercising man. Acta Physiologica Scandinavica 73: 527-535, 1968

Balldin UI, Lundgren CEG. Effects of immersion with the head above water on tissue nitrogen elimination in man. Aerospace Medicine 43: 1101-1108, 1972

Bazett HC, Scott JC, Maxfield ME, Blithe MD. Effect of baths at different temperature on oxygen exchange and on circulation. American Journal of Physiology 119: 93-110, 1937

Begin R, Epstein M, Sackner MA, Levinson R, Dougherty R, et al. Effects of water immersion to the neck on pulmonary circulation and tissue volume in man. Journal of Applied Physiology 40: 293-298, 1976

Bennett T, Hosking DJ, Hampton JR. Cardiovascular reflex responses to apnoeic face immersion and mental stress in diabetic subjects. Cardiovascular Research 10: 192-199, 1976

Berk JL, Levy MN. Profound reflex bradycardia produced by transient hypoxia or hypercapnia in man. European Surgical Research 9: 75-84, 1977

Bert P. Lecons sur la physiologie comparee de la respiration, pp. 526-553, Vailliere, Paris, 1870

Bichet DG, Groves BM, Schrier RW. Mechanisms of improvement of water and sodium excretion by immersion in decompensated cirrhotic patients. Kidney International 24: 788-794, 1983

Blix AS, Folkow B. Cardiovascular adjustments to diving in mammals and birds. In Shepherd & Abboud (Eds) Handbook of physiology: the cardiovascular system: III, pp. 917-945, American Physiological Society, Washington, D.C. 1983

Blomqvist CG. Cardiovascular adaptation to weightlessness. Medicine and Science in Sports and Exercise 15: 428-431, 1983

Boyer JT, Fraser JRE, Doyle AE. The effects of cold immersion. Clinical Science 19: 539-550, 1960

Buono MJ. Effect of central vascular engorgement and immersion on various lung volumes. Journal of Applied Physiology 54: 1094-1096, 1983

Cohn R, Bell WH, Saltzman HA, Klystra JA. Alveolar-arterial oxygen pressure difference in man immersed up to the neck in water. Journal of Applied Physiology 30: 423-425, 1967

Craig AB. Depth limits of breath-hold diving. Respiration Physiology 5: 14-22, 1968

Craig AB. Summary of 58 cases of loss of consciousness during underwater swimming and diving. Medicine and Science in Sports and Exercise 8: 171-175, 1976

Craig AB, Harley AD. Alveolar gas exchanges during breath-hold dives. Journal of Applied Physiology 24: 182-189, 1968

Craig Jr AB. Causes of loss of consciousness during underwater swimming. Journal of Applied Physiology 16: 583-586, 1961a

Craig Jr AB. Underwater swimming and loss of consciousness. Journal of the American Medical Association 176: 255-258, 1961b

Cross ER. Taravana: diving syndrome in the Tuamotu diver. In Rahn & Yokoyama (Eds) Physiology of breath-hold diving and the ama of Japan, National Academy of Science, publication no. 1341, pp. 207-219, Washington, DC, 1965

Dahlback GO. Influence of intrathoracic blood pooling on pulmonary air-trapping during submergence. Undersea Biomedical Research 2: 133-140, 1975

Dejours P. Hazards of hypoxia during diving. In Rahn & Yokoyama (Eds) Physiology of breath-hold diving and the ama of Japan, National Academy of Science, publication no. 1341, pp. 183-193, Washington, DC, 1965

Douglas CG, Haldane JS. The regulation of normal breathing. Journal of Physiology 38: 420-440, 1909

Dressendorfer RH, Morlock JF, Baker DG, Hong SK. Effects of head-out water immersion on cardiorespiratory responses to maximal cycling exercise. Undersea Biomedical Research 3: 177-187, 1976

Elsner R. Cardiovascular adjustments to diving. In Andersen HT (Ed.) The biology of marine mammals, pp. 117-145, Academic Press, New York, 1968

Elsner R, Franklin DL, Van Citters R, Kenney DW. Cardiovascular defense against asphyxia. Science 153: 941-949, 1966

Elsner R, Gooden B. Diving and asphyxia, Cambridge University Press, London, 1983

Epstein M. Cardiovascular and renal effects of head-out water immersion in man. Circulation Research 39: 619-627, 1976

Epstein M. Renal effects of head-out water immersion in man: implication for an understanding of volume homeostasis. Physiological Reviews 58: 529-581, 1978

Epstein M. Water immersion and the kidney: implication for volume regulation in man. Undersea Biomedical Research 11: 113-121, 1984

Erickson RD. Discover the underwater guide to the art and science of skin and scuba diving, Diver's Co, Santa Ana, 1972

Farhi LE, Linnarsson D. Cardiopulmonary readjustments during graded immersion in water at 35°C. Respiration Physiology 30: 35-50, 1977

Ferrigno M, Hickey DD, Liner MH, Lundgren CEG. Cardiac performance in humans during breath holding. Journal of Applied Physiology 60: 1871-1877, 1986

Finley JP, Bonet JF, Waxman MB. Autonomic pathways responsible for bradycardia on facial immersion. Journal of Applied Physiology 47: 1218-1222, 1979

Gandevia SC, McCloskey DI, Potter KE. Reflex bradycardia occurring in response to diving, nasopharyngeal stimulation and ocular pressure, and its modification by respiration and swallowing. Journal of Physiology 276: 383-394, 1978

Gauer OH, Henry JP. Neurohumoral control of plasma volume. In Guyton & Cowley (Eds) International review of physiology: cardiovascular physiology II, pp. 145-190, University Park Press, Baltimore, 1976

Gooden BA, Holstock B, Hampton JR. The magnitude of the bradycardia induced by face immersion in patients convalescing from myocardial infarction. Cardiovascular Research 7: 239-242, 1978

Greenleaf JE. Physiological responses to prolonged bed rest and fluid immersion in humans. Journal of Applied Physiology 57: 619-633, 1984

Gross PM, Terjung RL, Lohman TC. Left-ventricular performance in man during breath-holding and simulated diving. Undersea Biomedical Research 3: 351-360, 1976

Hajduczoc G, Miki K, Krasney JA. Regional circulatory responses to head-out water immersion in dogs. Federation Proceedings 44: 1200, 1985

Hamilton J, Moodie D, Levy J. The use of the diving reflex to terminate supraventricular tachycardia in a 2-week-old infant. American Heart Journal 97: 371-374, 1979

Hayward JS, Hay C, Mathews BR, Overwhiel CH, Radford DD. Temperature effects on the human dive response in relation to cold-water near drowning. Journal of Applied Physiology 56(1): 202-206, 1984

Heistad DD, Abboud FM, Eckstein JW. Vasoconstrictor response to simulated diving in man. Journal of Applied Physiology 25: 542-549, 1968

Hickey DD, Lundgren CEG. Physiology of Diving: breath-hold diving. In Shilling et al. (Eds) Physician's guide to diving medicine, pp. 206-221, Plenum Press, New York, 1984

Hill L, Flack M. The effect of excess carbon dioxide and want of oxygen upon the respiration and the circulation. Journal of Physiology 37: 77-111, 1908

Hong SK. Hae-Nyo, the diving women of Korea. In Rahn & Yokoyama (Eds) Physiology of breath hold diving and the ama of Japan, p. 99-111, National Academy of Sciences, National Research Council, Washington, D.C. 1965

Hong SK. Physiology of diving: thermal consideration. In Shilling et al. (Eds) The physicians guide to diving medicine, pp. 153-178, Plenum Press, New York, 1984

Hong SK. The physiology of breath-hold diving. In Strauss RH (Ed.) Diving medicine, pp. 269-286, Grune & Stratton, New York, 1976

Hong SK. Breath-hold bradycardia in man: an overview. In Lundgren & Ferrigno (Eds) The physiology of breath-hold diving, p. 158-171, Undersea and Hyperbaric Medical Society, Bethesda, 1987

Hong SK, Cerretelli P, Cruz JC, Rahn H. Mechanics of respiration during submersion in water. Journal of Applied Physiology 27: 535-538, 1969

Hong SK, Lin YC, Lally DA, Yim BJB, Kominami N, et al. Alveolar gas exchanges and cardiovascular functions during breath-holding with air. Journal of Applied Physiology 30: 540-547, 1971

Hong SK, Moore TO, Seto G, Park HK, Hiatt WR, et al. Lung

volume and apneic bradycardia in divers. Journal of Applied Physiology 29: 172-176, 1970
Hong SK, Rahn H. The diving women of Korea and Japan. Scientific American 216: 34-43, 1967
Hong SK, Rahn H, Kang DH, Song SH, Kang BS. Diving pattern, lung volumes, and alveolar gas of the Korean diving woman (ama). Journal of Applied Physiology 18: 457-465, 1963
Hong SK, Song SH, Kim PK, Suh CS. Seasonal observations on the cardiac rhythm during diving in the Korean ama. Journal of Applied Physiology 23: 18-22, 1967
Hood WB, Murray RH, Urschel CW, Bowers JA, Goldman JK. Circulatory effects of water immersion upon human subjects. Aerospace Medicine 39: 579-584, 1968
Hunt NG, Whitaker DK, Willmott NJ. Water temperature and the 'diving reflex'. Lancet 1: 972, 1975
Irving L. Bradycardia in human divers. Journal of Applied Physiology 18: 489-491, 1963
Jung K, Stolle W. Behaviour of heart rate and incidence of arrhythmia in swimming and diving. Biotelemetry and Patient Monitoring 8: 228-239, 1981
Kahn A, Raizi J, Blum D. Oculocardiac reflex in near miss for sudden infant death syndrome. Pediatrics 71: 49-52, 1983
Kang DH, Kim PK, Kang BS, Song SH, Hong SK. Energy metabolism and body temperature of the ama. Journal of Applied Physiology 20: 46-50, 1965
Kang DH, Park YS, Park YD, Lee IS, Yeon DS, et al. Energetics of wet-suit diving in Korean women divers. Journal of Applied Physiology 54: 1702-1707, 1983
Keatinge WR. Survival in cold water. Blackwell Scientific Publications, Oxford, 1969
Keatinge WR. The effect of work and clothing on the maintenance of body temperature in water. Quarterly Journal of Experimental Physiology 46: 69-82, 1961
Klocke FJ, Rahn H. Breath-holding after breathing of oxygen. Journal of Applied Physiology 14: 689-693, 1959
Knight DR, Horvath SM. Effect of hydrostatic pressure on plasma concentrations of norepinephrine during cold water immersion. Undersea Biomedical Research 14: 1-10, 1987
Kobayashi S, Sasaki C. Breaking point of breath holding and tolerance time in rebreathing. Japanese Journal of Physiology 17: 43-56, 1967
Krasney JA, Hajduczok G, Akiba C, McDonald BW, Pendergast DR, et al. Cardiovascular and renal responses to head-out water immersion in canine model. Undersea Biomedical Research 11: 169-183, 1984
Krasney JA, Pendergast DR, Powell E, McDonald BW, Plewes JR. Regional circulatory responses to head-out water immersion in anesthetized dog. Journal of Applied Physiology 53: 1625-1633, 1982
Lange L, Lange S, Echt M, Gauer OH. Heart volume in relation to body posture and immersion in a thermo-neutral bath: a roentgenometric study. Pfluegers Archives 352: 219-226, 1974
Lanphier EH, Rahn H. Alveolar gas exchange during breath-hold diving. Journal of Applied Physiology 18: 471-477, 1963a
Lanphier EH, Rahn H. Alveolar gas exchange during breath-holding with air. Journal of Applied Physiology 18: 478-482, 1963b
Lavoie JM, Montpetit RR. Applied physiology of swimming. Sports Medicine 3: 165-189, 1986
Lin YC. Autonomic nervous control of cardiovascular response during diving in the rat American Journal of Physiology 227: 601-605, 1974
Lin YC. Breath-hold diving, human imitation of aquatic mammals. In Brubakk et al. (Eds) Diving in mammals and man, pp. 81-89, Tapir Publishers, Trondheim, 1986
Lin YC. Breath-hold diving in terrestrial mammals. In Terjung RL (Ed.) Exercise and sport sciences reviews, Vol. 10, pp. 270-307, Franklin Press, Philadelphia, 1982
Lin YC. Cardiopulmonary physiology of nondiving mammals during breath-hold dives. In Shiraki & Matsuoka (Eds) Hyperbaric medicine and underwater physiology, pp. 25-35, University of Occupational and Environmental Health, Kitakyushu, 1983a
Lin YC. Hemodynamic changes during voluntary snout immersion in chronically instrumented dogs. Chinese Journal of Physiology 26: 1-10, 1983b
Lin YC. Circulatory functions during immersion and breath-hold dives in humans. Undersea Biomedical Research 11: 123-138, 1984
Lin YC. Effect of O_2 and CO_2 on breath-hold breaking point. In Lundgren & Ferrigno (Eds) The physiology of breath-hold diving, Undersea and Hyperbaric Medical Society, Bethesda, in press, 1987a
Lin YC. Human initiation of marine mammals and its clinical significance. In Bachrach et al. (Eds) Underwater physiology IX, Undersea and Hyperbaric Medical Society, Bethesda, in press, 1987b
Lin YC, Baker DG. Cardiac output and its distribution during diving in the rat. American Journal of Physiology 228: 733-737, 1975
Lin YC, Carlson EL, McCutcheon EP, Sandler H. Cardiovascular functions during voluntary apnea in dogs. American Journal of Physiology 245: R143-150, 1983
Lin YC, Lally DA, Moore TO, Hong SK. Physiological and conventional breath-hold breaking points. Journal of Applied Physiology 37: 291-296, 1974
Lin YC, Matsuura DT, Whittow GC. Respiratory variation of heart rate in the Californian sea lion. American Journal of Physiology 222: 260-264, 1972
Lin YC, Moore TO, McNamara JJ, Hong SK. O_2 consumption and conservation during apnea in the anesthetized dog. Respiration Physiology 24: 313-324, 1975
Lin YC, Shida KK, Hong SK. Effects of hypercapnia, hypoxia, and rebreathing on heart rate response during apnea. Journal of Applied Physiology 54: 166-171, 1983a
Lin YC, Shida KK, Hong SK. Effect of hypercapnia, hypoxia, and rebreathing on circulatory response to apnea. Journal of Applied Physiology 54: 172-177, 1983b
Lundgren CEG. Respiratory functions during simulated wet dives. Undersea Biomedical Research 11: 139-147, 1984
Maiorca E. Depth records: practical considerations. In Lundgren & Ferrigno (Eds) The physiology of breath-hold diving. Undersea and Hyperbaric Medical Society, Bethesda, in press, 1987
Mathew PK. Treatment of paroxysmal atrial tachycardia by diving reflex. Lancet 1: 510-511, 1978
Mathew PK. Diving reflex: another method of treating paroxysmal supraventricular tachycardia. Archives of Internal Medicine 141: 22-23, 1981
Matsuda M, Hong SK, Smith RM, Lundgren CEG. Physiological responses to immersion at 31 ATA (SEADRAGON IV). In Bachrach & Matzen (Eds) Underwater physiology VII, pp. 283-296, Undersea Medical Society, Bethesda, 1981
McArdle WD, Magel JR, Lesmes GR, Pechar CS. Metabolic and cardiovascular adjustment to work in air and water at 18, 25, and 33°C. Journal of Applied Physiology 40: 85-90, 1976
Miller JW (Ed.) NOAA Diving manual: diving for science and technology, 2nd ed., US Department of Commerce, National Oceanic and Atmospheric Administration, 1979
Mithoefer JC. Breath-holding. In Fenn & Rahn (Eds) Handbook of physiology, respiration II, pp. 1011-1925, American Physiological Society, Washington, DC, 1965
Mithoefer JC. Mechanism of pulmonary gas exchange and CO_2 transport during breath-holding. Journal of Applied Physiology 14: 706-710, 1959
Murdaugh Jr HV, Seabury JC, Mitchell WL. Electrocardiogram of the diving seal. Circulation Research 9: 358-361, 1961
Nadel ER. Energy exchanges in water. Undersea Biomedical Research 11: 149-158, 1984

Nemiroff MJ. Accidental cold-water immersion and survival statistics. Undersea Biomedical Research 4: A56, 1977
Noble MI, Eisele JH, Frankel HL, Else W, Guz A. The role of the diaphragm in the sensation of holding the breath. Clinical Science 41: 275-283, 1971
Olsen CR, Fanestil DD, Scholander PF. Some effects of underwater diving on blood gases, lactate and pressure in man. Journal of Applied Physiology 17: 938-942, 1962
Openshaw PJM, Woodroof GMF. Effect of lung volume on the diving response in man. J Appl Physiol 45: 783-785, 1978
Park YS, Pendergast DR, Rennie DW. Decrease in body insulation with exercise in cool water. Undersea Biomedical Research 11: 159-168, 1984
Park YS, Rahn H, Lee IS, Lee SI, Kang DH, et al. Patterns of wet suit diving in Korean women breath-hold divers. Undersea Biomedical Research 19: 203-215, 1983b
Park YS, Rennie DW, Lee IS, Park YD, Paik KS, et al. Time course of deacclimatization to cold water immersion in Korean women divers. J Appl Physiol 54: 1708-1716, 1983a
Paulev PE. Decompression sickness following repeated breath hold dives. Journal of Applied Physiology 20: 1028-1031, 1965
Petersen DH. Equipment and procedures: scuba diving. In Shilling et al. (Eds) The physician's guide to diving medicine, pp. 625-644, Plenum Press, New York, 1984
Pickering T, Bolton-Magge P. Treatment of paroxysmal supraventricular tachycardia. Lancet 1: 340, 1975
Rahn H, Yokoyama T (Eds). Physiology of breath-hold diving and the ama of Japan, National Academy of Science, publication no. 1341, Washington, DC, 1965
Rennie DW, Covine BG, Howell BJ, Song SH, Kang BS, et al. Physical insulation of Korean diving women. Journal of Applied Physiology 17: 961-966, 1962
Rennie DW, DiPrampero P. Cerretelli P. Effects of water immersion on cardiac output, heart rate, and stroke volume of man at rest and during exercise. Medizin dello Sport 24: 223-228, 1971
Rennie DW, Hong SK. Acclimatization to cold water: evidence from the course of deacclimatization. In Laursen et al. (Eds) Human performance in the cold, pp. 59-72, Undersea Medical Society, Bethesda, 1985
Rennie DW, Park YS, Veicsteinas A, Pendergast D. Metabolic and circulatory adaption to cold water stress. In Cerretelli & Whipp (Eds) Exercise bioenergetics and gas exchange, pp. 315-321, Elsevier, Amsterdam, 1980
Ricci G, Marroni H. Physiological observations during deep breath-hold diving. Cinesiologie 60: 187-193, 1976
Richet C. La resistance des canards a l'asphyxie. Comptes Rendus de Societé Biologie 1: 244-245, 1894
Ridgway SH. Diving by cetaceans. In Brubakk et al. (Eds) Diving in animals and man, pp. 33-59, Tapir Publishers, Trondheim, 1986
Risch WD, Koubenec HJ, Beckmann U, Lange S, Gauer OH. The effect of graded immersion on heart volume, central venous pressure, pulmonary blood distribution, and heart rate in man. Pfluegers Archives 374: 115-118, 1978
Sandler H. Cardiovascular effects of weightlessness. In Yu & Goodwin (Eds) Progress in cardiology, pp. 227-270, Lea & Febriger, Philadelphia, 1976
Sasamoto H. The electrocardiogram pattern of the diving ama. In Rahn & Yokoyama (Eds) Physiology of breath-hold diving and the ama of Japan, pp. 271-280, National Academy of Science, publication no. 1341, Washington, DC, 1965
Schneider EC. Observations on holding the breath. American Journal of Physiology 94: 464-470, 1930
Scholander PF. Experimental investigations on the respiratory function in diving mammals and birds. Hvalradets Skrifter 22: 1-131, 1940
Scholander PF. The master switch of life. Scientific American 209: 92-106, 1963
Scholander PF, Hammel HT, LeMessurier H, Hemingsen E, Garey N. Circulatory adjustment in pearl divers. Journal of Applied Physiology 17: 184-190, 1962
Shiraki K, Konda N, Sagawa S, Lin YC, Hong SK. Cardiac output by impedance cardiography during head-out water immersion. Undersea Biomedical Research 13: 247-256, 1986b
Shiraki K, Konda N, Sagawa S, Park YS, Komatsu T, et al. Diving pattern of Tsushima male breath-hold divers (Katsugi). Undersea Biomedical Research 13: 439-452, 1985
Shiraki K, Sagawa S, Konda N, Park YS, Komatsu T, et al. Energetics of wet-suit diving in Japanese male breath-hold divers. Journal of Applied Physiology 61: 1475-1480, 1986a
Siebke H, Rod T, Breivik H, Lind B. Survival after 40 minutes' submersion without cerebral sequelae. Lancet 1: 1275-1277, 1975
Song SH, Lee WK, Chung YA, Hong SK. Mechanism of apneic bradycardia in man. J Appl Physiol 27: 323-327, 1969
Sperandeo V, Pieri D, Palezzolo F, Donzelli M, Spataro G. Supraventricular tachycardia in infants: use of the 'diving reflex'. American Journal of Cardiology 51: 286-287, 1983
Sterba JA, Lundgren CEG. Diving bradycardia and breath-holding time in man. Undersea Biomed Res 12: 139-150, 1985
Strauss MB. Physiological aspects of mammalian breath-hold diving: a review. Aerospace Medicine 41: 1362-1381, 1970
Stromme SB, Kerem O, Elsner R. Diving bradycardia during rest and exercise and its relation to physical fitness. Journal of Applied Physiology 28: 614-621, 1970
Teruoka G. Die ama und ihre arbeit. Arbeitsphysiologie 5: 239-251, 1932
Tibes U, Stegemann J. Endtidal partial pressures O_2 uptake and CO_2 output following apneic diving and breath-hold in water and in air. Pfluegers Archives 311: 300-311, 1969
Veicsteinas A, Rennie DW. Thermal insulation and shivering threshold in Greek sponge divers. J Appl Physiol 52: 845-850, 1982
Webb P. Thermal stress in undersea activity. In Lamberten CJ (Ed.) Underwater physiology V: proceedings of the fifth symposium on underwater physiology, pp. 705-724, Bethesda, 1976
Whayne Jr TF, Killip III T. Simulated diving in man: comparison of facial stimuli and response in arrhythmia. Journal of Applied Physiology 22: 800-807, 1967
Whayne TF, Smith NT, Eger II EI, Stoelting RK, Whitcher GE. Reflex cardiovascular responses to simulated diving. Angiologia 23: 500-508, 1972
White PD. Observations on some tests of physical fitness. Journal of Medical Science 159: 866-874, 1920
Whitman V, Friedman Z, Berman W, Maissls MJ. Supraventricular tachycardia in newborn infants: an approach to therapy. Journal of Pediatrics 91: 304-305, 1977
Whitman V, Zakeosian GM. The diving reflex in termination of supraventricular tachycardia in childhood. Journal of Pediatrics 89: 1032-1033, 1976
Wildenthal K, Atkins M. Use of the 'diving reflex' for the treatment of paroxysmal supraventricular tachycardia. American Heart Journal 98: 536-537, 1979
Wildenthal K, Leshin SJ, Atking JM, Skelton CL. The diving reflex used to treat paroxysmal atrial tachycardia. Lancet 1: 12-14, 1975
Wolf S. The bradycardia of the dive reflex: a possible mechanism of sudden death. Transcripts of the American Clinical and Climatological Association 76: 192-200, 1964

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