suffered a fatal CAGE during a controlled ascent. In case SC 96/10 the victim, certified as an "advanced diver" had managed to run out of air twice during nine training dives. This warning of incompetent air management was not schooled out of him. On the fatal dive he made the same mistake again and this time unfortunately failed to survive. His attempt to "buddy breath" from his buddy's BCD implies that during training he had heard of this unusual procedure, one likely to be of little practical value compared with closer attention to his contents gauge.

There were 3 experienced divers in this series of fatalities. In case SC 96/3 it was the apparent failure to connect his BCD inflation system before entering the water which led to the need for him to attempt (unsuccessfully) to inflate his vest orally. This, combined with the strong surface current, negative surface buoyancy from wearing excessive weights, a possibly tight neck seal to his dry suit and losing of his hold on the mermaid line led to his death. In case SC 96/6 the victim was solo and the presumption is that he was concentrating so much on catching a crayfish that when he lost his regulator from his mouth he was in a position which prevented him from putting it back in his mouth and from making an immediate ascent. Case SC 96/9 is discussed above.

Health factors noted were temporary (fatigue SC 96/9, leg cramp SC 96/8), potential (the cold water asthma history in SC 96/7, personality factors in SC 96/2), or actual but unknown to the victim (myopathy SC 96/1). Depths of 30 m or greater bring nitrogen narcosis into consideration as a factor affecting the responses of the diver to his or her situation, while strong currents affected the course of the dives in cases SC 96/2 and SC 96/3.

Acknowledgments

This investigation would not be possible without the understanding and support of the Law, Justice or Attorney General's Department in each State, the Coroners and the police when they are approached for assistance.

Readers are asked to assist this safety project (PROJECT STICKYBEAK) by contacting the author with information, however tenuous, of serious or fatal incidents involving persons using a snorkel, scuba, hose supply or any form of rebreather apparatus.

All communications are treated as being medically confidential. The information is essential if such incidents are to be identified and avoided in future. Please write to Dr D G Walker, PO Box 120 Narrabeen, NSW 2101.

Dr D G Walker is a foundation member of SPUMS. He has been gathering statistics about diving accidents and deaths since the early 1970s. He is the author of REPORT ON AUSTRALIAN DIVING-RELATED DEATHS 1972-1993 which was published in 1998. His address is PO Box 120, Narrabeen, New South Wales 2101, Australia. Fax + 61-02-9970-6004

SNORKEL DIVING
A REVIEW

Carl Edmonds

Key Words

Barotrauma, breathhold, deaths, decompression illness, hyperventilation, hypoxia, recreational diving, unconscious.

Introduction

Snorkel diving is related to breath-hold diving and free diving.

The earliest evidence of breath-hold diving is attributed to shell divers, around 4500 B.C.

Traditional breath-hold divers include: the female shell divers of Japan (Ama) and Korea (Hae-Nyo); the sea-men (Katsugi) of Japan; sponge divers of Greece; pearl divers of the Tuamotu archipelago and Bahrain, and the underwater warriors of Xerxes.[1,2]

The abalone and paua divers of the USA and New Zealand and spear fishermen world wide use snorkels to simplify the surface phase of breath-hold diving. Submarine escape tank operators of USA, Europe and Australia have adapted breath-hold diving to modern applications.

The number of professional breath-hold divers of Korea and Japan have remained steady at about 20,000. The pearl divers of the Tuamotu archipelago, the Middle East and the Torres Strait, as well as the sponge divers of Greece, no longer have a viable industry. The abalone and paua divers have remained fairly constant, probably only a few hundred, because of the dwindling supply of this natural resource in shallow, accessible waters.

Compressed air diving, including scuba and hookah (surface supply from a compressor), have eroded the occupational activities associated in the past with breath-hold diving.

The recreational snorkellers of Australia are now a major part of the tourist industry of the Great Barrier Reef. Similar explosions of population are seen in the Caribbean, IndoPacific Islands and the Mediterranean.

Recreational snorkelling has become one of the most widely embraced sports of the latter part of the 20th century but the risks associated with this type of diving are not well documented.

A small group of adventurers has extended the depths, as well as the techniques and parameters of deep breath-hold diving. Sometimes the descents and/or ascents are assisted by weights, floats etc. Sometimes the breathing gases or techniques are modified to extend the depth/duration envelope of breath-hold diving. Currently the depth limit exceeds 150 metres and the duration can be extended by various methods to over 10 minutes. With this complexity comes added risks, outside the scope of this review.

Fatality statistics

The mortality and morbidity incidence in recreational snorkel swimming/diving is unknown, as is the number of the snorkelling population. There is little pertinent data available in the general medical literature, even on the deaths from this activity.

A series of 60 Australian snorkelling deaths (1987-96)[3] were compared to a previous series of 90 Australian deaths (<1987).[4] In the 1987-96 decade the average age had risen from 30 to 45 years. Females had increased from 2% to 25% and were mostly in the drowning group. Spear fishing had reduced from 73% to 25% and by now most of the snorkellers were recreational swimmers engaged in organised boat operations.

The three major causes of death in the latest survey were drowning (45%), cardiac (30%) and hypoxia from breath-holding after hyperventilation and/or ascent, producing unconsciousness and drowning (20%). The incidences in the earlier survey were 52%, 3% and 18% respectively.

Some deaths resulted from marine animals and trauma. Other causes, not obvious from these figures, included epilepsy, cerebro-vascular accidents, asthma, aspiration of vomitus, and entanglement. All these were relatively less frequent.

Recreational snorkellers were mainly surface swimmers who occasionally dived. The breath-hold divers were often spear fishermen and collectors of shellfish.

Other characteristics of the 1987-96 survey, which did not differ significantly from those of the previous one, showed that; only 7% had an allocated “buddy diver” accompanying them at the time, 7% were separated by the incident, 47% separated prior to the incident, 40% were solo snorkelling from the start.

This is reflected in the absence of rescue attempts in 42%. In 58% a rescue attempt was made, usually by an unassociated swimmer. Rescue was attempted in under 5 minutes (when resuscitation is feasible) in only 17%, and in 38% it took between 5 and 15 minutes before being initiated.

Many factors contributed to the three major causes of death.

Tourists were over-represented in both the drowning and the cardiac groups. Inexperience, medical and physical unfitness, equipment and environmental factors contributed to the deaths in these two groups.

DROWNING

Drowning cases were frequently inexperienced, medically or physically unfit, inadequately equipped (often without fins, to assist in propulsion) thereby increasing the danger of adverse sea conditions. Supervision was inadequate and therefore rescue and resuscitation were delayed.

It occurred frequently in non-English speaking tourists who may not have understood either the documentation required by commercial boat operators, or the safety instructions from snorkelling supervisors.

CARDIAC

Cardiac deaths were analogous to those in middle aged males while scuba diving.[2,5] They died quietly, usually on the surface. There are many aquatic factors that can precipitate a cardiac event, when superimposed on an underlying cardiac disorder, which was often evident in the medical history.

Inexperience, poor physical fitness, poor swimming ability and the absence of efficient propulsion, all increased the effort required for snorkelling.

The extreme difficulty of achieving adequate resuscitation with this presentation, has been noted with scuba.[6]

HYPOXIA

Hypoxia from hyperventilation and/or ascent,[3,7-10] during breath-hold diving, occurs predominantly in spear fishermen. The hypoxia develops because of the increasing breath-hold time possible after hyperventilation, due to the production of hypocapnoea. With the hyperventilation induced hypocapnoea, there is no proportional increase in oxygen carrying capacity, and therefore these breath-hold divers are able to extend the "breaking point" to such a degree that hypoxia and therefore unconsciousness may result. In these cases there is little or no warning.

Hypoxia is aggravated during ascent because of the expansion of gas (Boyle's Law) in the breath-hold diver's lungs, reducing the partial pressure of oxygen and increasing the likelihood of hypoxic unconsciousness.

This is a younger, fitter, group of divers, very experienced and with good equipment, undertaking a hazardous diving technique which should be discouraged.

TABLE 1

PROFILES OF DEATHS

Cause of death	Drowning N=27	Cardiac N=18	Hypoxia N=12
Male: Female	15: 12	15: 3	12: 0
Average Age (SD)	44.7 (18.7)	55.6 (14.6)	35.2 (14.9)
Site of death	Surface	Surface	Depth/ascent
Equipment	Inadequate	Inadequate	Complete
Nationality	48% Foreign esp. Japanese	50% Foreign esp. USA	100% Australian
Experienced	30%	50%	100%
Buoyancy	Positive	Positive	Negative

Marine environments

Like all other divers,[2] breath-hold divers are susceptible to the hazards of the marine environment. These include injuries from marine animals, infections and envenomations. They include exposure to water temperatures less than thermo-neutral (35°C) as well as the various drowning syndromes, including salt water aspiration and near-drowning. Motion sickness is a common problem, as is trauma (ocean currents, rocks, boats etc.) and entrapment.

Equipment problems

These hazards include a variety of problems due to the actual equipment being worn by the free diver, i.e. mask, snorkel, fins etc., together with the problems of entrapment, the use of spear guns, floats, boats etc. They are no different in principal from those encountered by scuba divers, but the disadvantage for the free diver is that a plentiful supply of air is not available to him. One of the increasing dangers is entrapment and entanglement in lines (floats, spears, etc.). Some of the modern filament fishing lines are not able to be snapped nor cut by a knife. A scuba diver has much more time available to cope with such difficulties.

Common equipment problems include flooding of the face mask and restriction to snorkel breathing with exertion. The reduction in maximum voluntary ventilation and increase in the work of breathing produces dyspnoea when the respiratory demands are great.

Barotrauma

Barotrauma of descent [2] is more common in free divers than scuba divers, because of the rushed nature of the activity. Free divers have so little time that they have to descend more rapidly and often without much attention to the various symptoms that may be caused by barotrauma. They also undertake more ascents and descents, producing more barotrauma. The areas affected are; face, ears, sinus, dental and the gastrointestinal tract.

Following barotrauma of descent, there is often an associated barotrauma of ascent. This is especially seen with otological, sinus, dental and gastrointestinal barotraumas. Ascent cannot be delayed, nor even slowed, and so the manifestations cannot be diminished, as they are with scuba.

Pulmonary barotrauma of descent (lung squeeze),[11,12] occasionally occurs in breath-hold divers.

With descent, the gas volume in a breath-hold diver's lungs will contract in accordance with Boyle's Law. Thus a diver with a total lung volume of 6 litres (and residual volume of 1.5 litres) on the surface (1 ATA) will be able to descend to 4 ATA (30 m) before the lung volumes will equate with the residual i.e. ($P_1 V_1 = P_2V_2$), 6 x 1 = 1.5 x 4.

Initially it was believed that further descent would lead to lung pathology (haemorrhages, oedema etc.). In practice this is avoided by the negative pulmonary pressure resulting in engorgement of the pulmonary vessels. This reduces the residual volume, replacing air spaces with intravascular blood from the periphery.

Breath-hold descents, without inducing "lung squeeze", have been undertaken to depths in excess of 150 m.

Decompression sickness

Decompression sickness has also been postulated as a result of intensive free diving.[13-16]

Cross described an illness called Taravana (tara = to fall, vana = crazily), in the pearl divers of the Tuamotu archipelago.[13,14] The dives were to 30-40 m, lasting 1.5 to 2.5 minutes each, over a 7 hour period. The illness, which was characterised by vertigo, nausea, paresis, unconsciousness or death, could be due to decompression sickness in some of the cases. Perusal of the original cases would indicate that many could have been due to a variety of other disorders, e.g. inner ear barotrauma, salt water aspiration, near drowning causing hypoxic encephalopathy and drowning.

The reason decompression sickness can develop with breath-hold diving is that the nitrogen pressure in the lungs increases with depth, and with the greater depths there is a greater nitrogen partial pressure, with nitrogen diffusing from the lungs into the bloodstream and thence to the tissues. If the surface interval is inadequate to eliminate this nitrogen, or if bubbles develop due to the rapid ascent, then it will accumulate with repeated dives throughout the day.

Paulev, a Danish submarine escape tank safety diver in the Norwegian Navy, performed 60 breath-hold dives to 20 m in 5 hours, each lasting about 2.5 minutes with surface intervals of less than 2 minutes. He developed symptoms consistent with decompression sickness.[15] Other submarine escape instructors have suffered similar problems, in both Norway and Australia.[16]

Hypoxic blackout

This is sometimes called breath-hold syncope or "shallow water blackout". As the latter term was first used in 1944 to describe loss of consciousness using closed circuit diving apparatus, it is best avoided in the breath-holding context. "Hypoxic blackout" is a reasonable alternative.

There are two causes for this disorder,[7-10] hyperventilation and ascent, and, as they may occur concurrently, they are often confused. The hyperventilation effect is independent of depth, and may be encountered in 1 m deep swimming pools, often by children trying to swim greater distances under water.

Divers who train to extend their breath-hold time and also dive deep (such as in free diving competitors, spear fishing etc.) risk hypoxia of ascent, with loss of consciousness and subsequent drowning.

With hypoxia there is little or no warning of impending unconsciousness. With increased experience the breath-hold diver can delay the need to inhale by various techniques, without improving his oxygen status. Breath-hold time can be extended (but not with increased safety) by feet first descent, training (adaptation), swallowing, inhaling against a closed glottis, diaphragmatic contractions etc.[11]

One way of avoiding this hypoxia is to inhale 100% oxygen prior to the breath-hold.

HYPERVENTILATION AND HYPOXIA

Craig observed that swimmers who hyperventilated could stay longer underwater, but risk losing consciousness with little or no warning.[7-9] They were often competing, against others or themselves, and often exercising. The hyperventilation extended their breathholding time, because it washed out a large amount of CO_2 from the lungs, often reducing arterial CO_2 to half the normal levels.

The build-up of CO_2 is the main stimulus compelling the swimmer to surface and breath. After hyperventilating it takes much longer for this level (the "breaking point") to be achieved.

The arterial O_2 pressure drops to a level inadequate to sustain consciousness, if breath-hold time is extended and exercise consumes the available oxygen.

One can see the effects of both hyperventilation and exercise in reducing the O_2 level to a dangerous degree, when breath-holding, in Craig's original experiment.[7] Table 2 (page 20) reproduces his results.

When the swimmer is concentrating on some purposeful goal, such as trying to spear a fish, retrieve a catch, or untangle an anchor, then he is more likely to ignore the physiological warning symptoms of an urge to breath (due to the rise in CO_2 level in the blood), and delay the breaking point.

HYPOXIA OF ASCENT

Ascent hypoxia was described first in military divers using gas mixtures, who lost consciousness as they surfaced with low oxygen levels in their rebreathing equipment.[2]

In breath-hold divers, with descent the pressure rises proportionately in the alveoli gases, increasing the

TABLE 2

EFFECTS OF HYPERVENTILATION ON THE BREATH-HOLDING (BH) TIME AND ALVEOLAR GAS PRESSURE AT THE BREAKING POINT IN RESTING AND EXERCISING MAN[7]

MEASUREMENTS	RESTING	
	Without Hyperventilation	With Hyperventilation
BH time (sec)	87	146
End-tidal pCO_2 (mm Hg)		
Before BH	40	21
Breaking point	51	46
End-tidal pO_2 (mm Hg)		
Before BH	103	131
Breaking point	73	58
	EXERCISING	
	Without Hyperventilation	With Hyperventilation
BH time (sec)	62	85
End-tidal pCO_2 (mm Hg)		
Before BH	38	22
Breaking point	54	49
End-tidal pO_2 (mm Hg)		
Before BH	102	130
Breaking point	54	43

available O_2, CO_2 and nitrogen uptake. Some O_2 can be absorbed and utilised, some CO_2 absorbed and buffered, some nitrogen absorbed and deposited in tissues.

Thus if a diver, having 100 mm Hg 0_2 and 40 mm Hg CO_2 in his alveolar gases, was immediately transported to 2 ATA, the lungs would halve their volume (Boyle's Law P1 V1 = P2 V2), the O_2 would be 200 mm Hg, and the CO_2 80 mm Hg. Both would pass into the pulmonary blood circuit, the O_2 to be used and the CO_2 to be buffered. Thus the O_2 and CO_2 pressures in the alveoli would decrease rapidly. By the time they were both back to "normal" levels, with O_2=100 mm Hg and CO_2 = 40 mm Hg, then the diver would appear to be in a satisfactory respiratory status, until he ascended. With an expansion of the lungs to twice their size at depth, the pressures in both gases would halve, i.e. the O_2 would drop to 50 mm Hg (approaching a potentially dangerous hypoxic level) and the CO_2 to 20 mm Hg, if the ascent was immediate.

As ascents do take time, more O_2 will be consumed, extracted from the lungs during the ascent, and the CO_2 would increase towards normal due to the gradient between the pulmonary blood and alveoli. The actual figures would vary with the speed of ascent, the diffusion gradient and rate, and the consumption of O_2.

The drop in O_2 is then able to produce the loss of consciousness, the "syncope" or "blackout", commonly noted amongst spear fishermen. This is now known as hypoxia of ascent. In deeper dives it becomes more likely, and with some very deep dives, the loss of consciousness may occur on the way to the surface, in the top 10 m (probably an explanation for the "seven metre syncope" described by French workers[17]).

OTHERS

Despite the classical causes of hypoxia, as above, probably the commonest cause is the aspiration of salt water, resulting in near drowning and drowning states[3,6].

Cardiac disorders [18-25]

Human breath-hold divers produce a dramatic bradycardia from the diving reflex.[11] It reaches its zenith in 20 to 30 seconds, usually to an equivalent of two-thirds the pre-dive level, but sometimes to less than 10 beats/minute in experienced divers. It bears a linear relationship to the water temperature below 15°C, non-linear above that. The bradycardia might then permit other arrhythmias to develop. The arterial blood pressure seems to increase with the diving reflex in humans.

In humans, unlike most of the diving mammals, free diving is associated with significant cardiac arrhythmias. These can be provoked by common respiratory manoeuvres such as deep inspiration, prolonged inspiration, breath-holding, release of breath-holding, Valsalva manoeuvres etc.[24]

In a study of Korean women divers the incidence of cardiac arrhythmias was 43% in the summer (water temperature 27°C) as compared with 72% in the winter (water temperature 10°C).[18]

There is a high frequency of arrhythmias in association with immersion breath-holding, even without diving. The head-out immersion position increases the workload on the heart, because of the negative pressure effect (the intrapulmonary pressure remains at 1 ATA, while a negative pressure, needed to inhale, is approximately -20 cm H_2O). There is a reduction in the functional residual capacity of the lungs, an increased work of breathing and an increase in the intrathoracic blood volume, with a corresponding dilatation of the heart, and especially the right atrium. The latter may be a major cause of arrhythmias from sinus rhythm. Immersion diuresis and associated loss of sodium may exacerbate cardiac problems. With very deep breath-hold divers about a litre of extra blood can fill the pulmonary circuit and the heart.

The relatively high incidence of cardiac deaths during snorkelling[3] (and scuba diving[5]) activities may be partly related to the above findings, and partly due to the excessive workload experienced by novice snorkellers, attempting to overcome the influences of panic, adverse tidal currents and a need to keep the head above water.

Pulmonary disorders

The most common lung problem is the aspiration of sea water, producing either the drowning syndromes or provoking asthma in those so inclined.

The changes in lung volumes with the head-out position during immersion have been described above, with the pooling of blood in the thorax, reducing respiratory capability.

Pulmonary oedema[2,11,24] has been described in association with immersion, as have other causes of dyspnoea, including the coronary artery disease, cardiac arrhythmia and cold induced hypertension.

Gastrointestinal problems

Another pressure gradient associated with the head-out immersion, commonly experienced in free divers between dives, is the increased gastro-oesophageal pressure gradient, which increases from 6 mm Hg in air to 16 mm Hg during immersion. This predisposes to gastric reflux, in those with an inadequate oesophageal sphincter.

This also increases the tendency to vomiting which can be aggravated by other factors such as alcohol intake, sea sickness, otological barotrauma, gastrointestinal barotrauma etc.

Medical check list for snorkellers

Unless there are specific medical or physical disorders, medical examinations are not usually required by snorkellers. Because there are organised commercial snorkelling activities, on the Great Barrier Reef and by schools or other organisations, the minimal medical requirement should be a simple questionnaire likely to pick up most major causes of mortality and morbidity from this activity. A typical one is reproduced here as Table 3.

Conclusion

Despite the paucity of medical documentation on this common recreational activity, some information is available. Combined with case histories[4] and consensus views of experts in this field, there probably is enough evidence to base recommendations on safety aspects of this activity. Medical fitness can be indicated by a medical history questionnaire for potential snorkellers.

TABLE 3

MEDICAL CHECK LIST FOR SNORKELLERS

Have you ever had:			
1	Any cardiovascular disease? (Heart, blood pressure, blood, others).	YES	NO
2	Any lung disease? (Asthma, wheezing, pneumothorax, TB, others).	YES	NO
3	Any fits, epilepsy, convulsions or blackouts?	YES	NO
4	Any serious disease? (Such as diabetes)	YES	NO
5	Serious ear, sinus or eye disease?	YES	NO
6	Any neurological or psychiatric disease?	YES	NO
Over the last month have you had any:			
7	Operations, illnesses or treatment?	YES	NO
8	Drugs or medications?	YES	NO
9	If female, are you pregnant?	YES	NO
Can you			
10	Swim 500 metres without flippers?	NO	YES
11	Swim 200 metres in 5 min or less, without flippers?	NO	YES
12	Equalise your ears when diving or flying?	NO	YES

NAME: (If under 16 years, guardian to sign)

DOB:

ADDRESS:

Note: If the candidate indicates an answer in the left hand column, then medical assessment and advice is required before snorkelling is undertaken.

A standard of physical and aquatic fitness is a reasonable requirement. Buddy responsibility should be encouraged and adequate supervision available, especially if the snorkeller is not experienced in the existing conditions. Supervision is essential during commercial diving operations.With the latter, multilingual facilities may be required.

Hyperventilation should be discouraged as a prelude to breath-hold diving.

References

1 Hong SK. Breath-hold diving. In *Bove and Davis' Diving Medicine. 3rd Edition.* Bove AA. Ed. Philadelphia: Saunders, 1997; 65-74

2 Edmonds C, Lowry C, Pennefather J and Walker R. *Diving and Subaquatic Medicine. 4th Edition.* Oxford: Butterworth/Heinemann, In press

3 Edmonds C and Walker D. Australian snorkeling deaths. *Med J Aust* 1999, In press

4 Walker DG. *Report on Australian Diving Deaths 1972-1993*. Melbourne: JL Publications, 1998

5 Edmonds C and Walker D. Scuba diving fatalities in Australia and New Zealand. *SPUMS J* 1989; 19 (3): 94-104

6 Edmonds C, Walker D and Scott B. Drowning with scuba. In *Near Drowning. The 47th Workshop of the Undersea and Hyperbaric Medicine Society*. Dueker CW and Brown SD. Eds. Kensington, Maryland: UHMS, 1999; 19-33

7 Craig AB. Causes of loss of consciousness during underwater swimming. *J Appl Physiol* 1961; 16: 583-586

8 Craig AB. Underwater swimming and loss of consciousness. *JAMA* 1961; 176: 255-258

9 Craig AB. Summary of 58 cases of loss of consciousness during underwater swimming and diving. *Med Sci Sports* 1976; 8: 171-175

10 Risberg J and Lundgren C. Breath-hold diving. *Proceedings of XXIVth Annual Meeting of European Underwater Biomedical Society Meeting*. Stockholm; 1998; 26-30

11 Lundgren C. *Physiological challenges of breath-hold diving*. Special presentation at 1999 UHMS Annual Meeting, Boston. Available as an audio-tape from UHMS.

12 Lin YC. Physiological limitations of humans as breath-hold divers. In *Man In the Sea. Vol II.* Lin YC and Shida KK. Eds. St Pedro, California: Best Publishing Company, 1990; 33-56

13 Cross ER. Taravana. Diving syndrome in the Tuamotu diver. In: *Physiology of Breath-Hold Diving and the Ama of Japan. National Academy of Science publication #1341*. Rahn H and Yokoyama T. Eds. Washington, DC, 1965: 207-219

14 Cross ER. Taravana and the Tuamotu pearl Divers. The Indigenous Diver Seminar 1997 UHMS Annual Meeting, Anchorage. Kensington, Maryland: UHMS, In press

15 Paulev P. Decompression sickness following repeated breath-hold dives. In: *Physiology of Breath-Hold Diving and the Ama of Japan. National Academy of Science publication #1341*. Rahn H and Yokoyama T. Eds. Washington, DC, 1965: 211-226;

16 Wong RM. Taravana revisited: decompression illness after breath-hold diving. *SPUMS J* 1999; 29 (3): 126-131

17 Boucher L. Le rendez-vous syncopal des 7 mètres. *http://www.worldnet.fr/~lboucher/plongee/rendez-vous.htm*

18 Hong SK, Wong SH, Kim PK and Suh CS. Seasonal observations on the cardiac rhythm during diving in the Korean Ama. *J Appl Physiol* 1967; 23: 18-22

19 Hickey DD and Lundgren CEG. Breath-hold diving. In *The Physicians' Guide to Diving Medicine*. Shilling CW, Carlstrom CB and Mathias RA. Eds. New York: Plenum Press, 1984: 206-221

20 Hong SK. Breath-hold diving. In *Diving Medicine by Bove and Davis. 2nd Edition.* Philadelphia: Saunders, 1990 ; 59-68

21 Park YS, Shiraki K and Hong SK. Energetics of breath-hold diving in Korean and Japanese professional diving. In *Man In the Sea. Vol II.* Lin YC and Shida KK. Eds. St Pedro, California: Best Publishing Company, 1990; 73-86

22 Scholander PF, Hummel H, LeMessurier H, Hemingsen E and Garey W. Circulatory adjustment in pearl divers. *J Appl Physiol* 1962; 17: 184-190

23 Rahn H and Yokoyama T, eds. *Physiology of Breath-Hold Diving and the Ama of Japan. National Academy of Science publication #1341*. 1965

24 Lamb LE, Dermksian G and Samoff CA. Significant cardiac arrhythmia induced by common respiratory maneuvers. *Am J Cardiol* 1958; 2: 563-571

25 Hayward, JS, Hay C, Mathews BR, Overwhiel and Radford DD. Temperature effects on the human dive response in relation to cold-water near drowning. *J Appl Physiol* 1984; 56:202-206

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