THE PHYSIOLOGY OF BREATH-HOLD DIVING

Undersea and Hyperbaric Medical Society Workshop

Edited by

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and
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THE PHYSIOLOGY OF BREATH-HOLD DIVING

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Special thanks go to Miss Pamela Caron for her great efforts in transcribing the discussions from tape recordings. Thanks are also due to Ms. Ann Barker of the Undersea and Hyperbaric Medical Society for helping to prepare these proceedings for publication.

INTRODUCTION

This publication brings together the presentations and discussions that were part of the two-day workshop "The Physiology of Breath-hold Diving". The workshop was held at the State University of New York at Buffalo on October 28-29, 1985, almost exactly 20 years after the last major meeting on the same subject. That was the Symposium on the Physiology of Breath-Hold Diving and the Ama of Japan, which took place in Tokyo, Japan, in conjunction with the XXIII International Congress of Physiological Sciences.

In 1965 it was probably felt that the diving women of Japan and Korea - the only major group of people resorting to breath-hold diving for a living- were soon to disappear. It is surprising that at this time, 20 years later, these remarkable divers are holding their own and even probably increasing in number, as the market value of their catches continues to rise. Furthermore, in the last 40 years the maximal depths reached by men and women holding their breath have increased dramatically to more than 100 meters. While the small group of these record-setting divers is highly skilled and trained, the very nature of its pursuit puts these persons at the limit of what is physiologically feasible and medically safe. Indeed, there is evidence that sometimes the limit has been surpassed. Nonetheless, like many other hazardous sporting activities, including spear-fishing competitions, these record attempts continue.

While we have included a presentation and a film featuring deep breath-hold diving in the workshop program, we do not endorse this activity. However, as scientist, one is obliged to try to understand the mechanisms that determine the limits, and also to identify the risks, of deep breath-hold diving. For this reason, at this workshop we appreciated the opportunity to draw on the experience of Mr. Maiorca, who is one of the record-holding breath-hold divers.

As public's interest in diving has expanded over the years, millions of people around the world have enjoyed the underwater scenery, either breath-hold diving or simply snorkeling at the surface. Recreational divers span a wide spectrum of diving proficiency, from the highly skilled spear-fishermen in South African waters to the snorkelers on the beaches of temperate and tropical coasts. Add to these the many persons who become involuntary breath-hold divers in drowning incidents, and the number of people affected by by our knowledge, or lack thereof, becomes impressive.

The purpose of this workshop was to review the current status of breath-hold diving physiology. Generous support for this project was provided by the New York Sea Grant Institute (Contract no. NA85AADSG021) . The costs for printing of these Proceedings were covered by the Undersea and Hyperbaric Medical Society, Inc. The presentations and discussions are presented as they occurred in the course of the workshop. However, two additional contributions have been included because they contain information that was frequently referred to in the discussions and presentations. One is a reprint of an article by Y.S. Park, D.R. Pendergast and D.W. Rennie: Decrease in

body insulation with exercise in cool water. Undersea Biomed. Res. 11:159-168. The other one was kindly written by Dr. H. Rahn specifically for this publication and it is entitled: Alveolar Gas Composition and Performance. All presentations were printed from camera-ready manuscripts provided by the authors and the discussions were transcribed from tape recordings. These transcripts were subject to some editing and then reviewed by the speakers.

We hope the reader will enjoy these Proceedings as much as we enjoyed the workshop, and we thank all the participants who contributed with their interesting presentations and lively discussions.

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Breath-hold Diving: A Brief History

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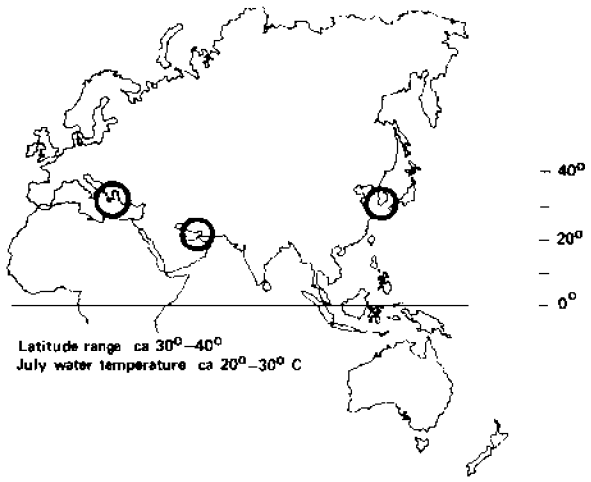
Historical accounts of breath-hold divers can be traced back more than 2000 years, describing the activities among the sponge divers of Greece, pearl divers in the Persian Gulf and India, and the seafood divers of southern Korea and Japan (Fig. 1).



In each region the skills developed independently but are basically similar, namely, head-first descent in shallow or deep dives, the latter being assisted by a weight. Ascent from deep dives was also assisted by pulling up the diver with a rope. As Davis (1934) wrote, breath-hold diving was also a recognized profession in naval warfare, divers being employed to destroy boom defenses in harbors during the siege of Tyre (333 B.C.) and Syracuse (415 B.C.) According to Chris Lambertsen (pers. commun.) the Frogmen of the U.S. Navy's Underwater Demolition Teams operated as breath-hold swimmers throughout World War II. Even today breath-hold diving is still part of the training of underwater demolition teams.

The modern scientific era of breath-hold diving might be said to have begun in the 1920's when Gito Teruoka, Director of the Institute for Science of Labor in Japan, (Fig. 2) became concerned about the occupational health, hazards, and strenuous physical exertion of some 25,000 women who earned their living by diving, not for pearls, but for various types of seafood, to depths of up to 20 meters. His meticulous observations of diving times, patterns, depth, and alveolar gas concentration following

these dives were published in 1932 in Arbeitsphysiologie. I think it is fair to say that his work was promptly forgotten and only rediscovered 25 years later when Dr. S. K. Hong began his studies of the Korean Ama.

Interest in breath-hold diving re-emerged in the 1940's and 1950's with increased public awareness in swimming, snorkeling, and scuba diving as well as their associated hazards. Scientific interest began to focus on the physiological consequences of voluntary apnea, particularly when submersed, the changes in lung gases, chest mechanics, and cardiovascular systems as well as the role played by the O_2 and CO_2 stores of the body. In 1965 the first International Breath-hold Symposium was held in Japan and dedicated to Professor Teruoka (Rahn and Yokoyama, 1965). It provided the first comprehensive account of diving patterns and physiology of the Ama in Japan and Korea, the first estimations of the work of diving, the first hints that repeated dives could result in bends, and a new focus on diving bradycardia and the importance of water temperature and body insulation.

In the intervening years much attention has been focused on the cardiovascular changes during submersion and lung compression, depth records, and associated changes in lung mechanics and gas stores, diving patterns, energetics and thermoregulation, and a better understanding of the circumstances that lead to fatal accidents in competitive sports. Unfortunately for the physiologist the breath-hold diver during descent or ascent becomes rather inaccessible to simple measurements of his cardiovascular and pulmonary system. In other words, he becomes a black box. However, the more recent techniques pioneered by Kooyman (1981) and Zapol (1986) on free diving Weddell seals in Antarctica promise a new tool which could also be applied to man in obtaining blood samples, heart rate, and continuous records of diving velocities during repeated dives as well as at the surface. The other new tool is that of treating the diver as a closed black box and using the computer to calculate the O_2, CO_2, and N_2 changes in the lung, blood, and tissues during descent and ascent. Such

results will be discussed in this Symposium.

Today marks the second International Symposium devoted to breath-hold diving, and this will bring us up to date on all the new insights that have been gained during the last twenty years.

References

Davis, R. H. 1934. Deep diving and underwater rescue. J. Roy. Soc. Arts 82: 1032-1047.

Kooyman, G. L. 1981. Weddell Seal: Consummate Diver. Cambridge University Press, Cambridge.

Rahn, H., and T. Yokoyama. 1965. Physiology of Breath-Hold Diving and the Ama of Japan. Public. 1341. Nat. Acad. Sci., Nat. Res. Counc., Washington, DC.

Teruoka, H. 1932. Die Ama und ihre Arbeit. (Eine Untersuchung über die japanischen Taucherinnen). Arbeitsphysiol. 5: 239-251.

Zapol,W. M. 1986. Diving physiology of the Antarctic Weddell Seal. In: Environmental Physiology (L. E. Farhi and C. V. Paganelli, eds.) Springer Verlag, NY (in presss).

VON TRIELEBEN'S DIVING BELL

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At the beginning of the 17th century Sweden controlled most of the Baltic Sea and occupied much of the surrounding land. King Gustav II Adolf of Sweden expressed the view that "Next to God the welfare of Sweden depends on her Navy. Building small ships is only a waste of young trees." With these concepts in mind he ordered many ships, one of which was the Vasa. This ship, which was launched in 1627, was 50 m (165 feet) long and displaced 1300 tons (Fig. 1). After being fitted, she set sail on August 10, 1628 against a "light breeze from the south south-west." In a few hundred meters "a sudden squall forced her to heel over so far to port that the water flooded in through the lower cannon ports... The Vasa went down with sails up and flags flying" (1). In 1956 Anders Franzen located the remains of the ship. Its restoration is an interesting story, in itself, and a visit to the specially constructed Vasa Museum in Stockholm is a rewarding experience.

At the time of the sinking and for several years thereafter the location the Vasa was well known. In 1664, 36 years after the catastrophe, Hans Albreckt von Treileben of Sweden and Andreas Peckell, a German salvage expert, recovered most of the 64 cannon on board. The heavier cannon weighed between one and two tons. An Italian priest, Francesco Negri, traveling through the area on his way to the North Cape, described the procedure in his book, Viaggio Settentrionale published in 1700 at Padua. The divers wore leather suits and descended in a diving bell (Fig. 2). They used various tools on the ends of 2 m poles to free the cannon and to secure lines with which they were hauled to the surface (Fig. 3). The usual diving time was 15 minutes.

A reproduction of the diving bell constructed from descriptions given by Marten Triewald in his book Kosten att lefwa under vatn (2) is on display at the Vasa Museum in Stockholm. It is conical, measures 1.22 m in height, and has a bottom circumference of 1.06 m.

Fig. 1. The Warship Vasa (1).

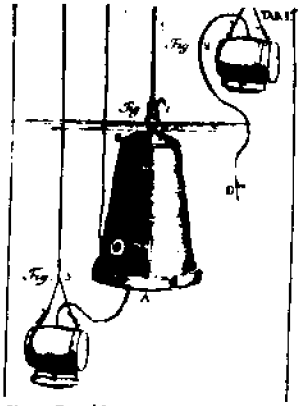

Fig. 2. Von Treileben's diving bell (2).

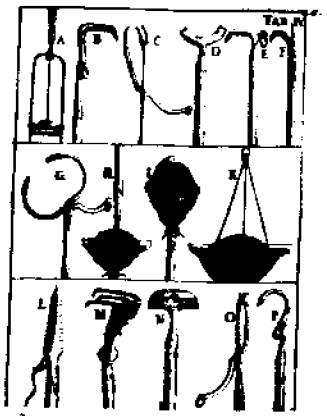

Fig. 3. Implements used for salvage work (2).

The total air volume of the bell is 533 l. If it were assumed that the diver was 1.5 m tall, the top of his head would be just below the top of the bell. As he was lowered to the wreck which lay at 30 m, the level of water would rise to just over half the height of the bell (Fig. 4). It is difficult to imagine how the divers did any useful work with the tools depicted. In addition there is little or no light at the bottom of the silted Stockholm Harbor, and the temperature is a constant 4 C throughout the year. Nevertheless, almost all of the cannon were recovered.

From the physiologic view this type of diving can be considered breath-hold diving. The usual breath-hold diver makes an inspiration, suspends ventilation, and goes below the surface. In effect, the diver takes a limited supply of air for use during the period of apnea. Although the supply of air is larger for the worker in the bell than that for the usual breath-hold diver, it is limited. Assuming that the worker's oxygen consumption was about 1 l/min, the exchange ratio was .8, and that all of the CO2 produced would be expired, it can be calculated that the P CO2 in the bell would rise to about 80 torr at the end of a 15 minute work period at 30 m. However, as the P CO2 increased some of the CO2 would be stored and would not appear in the air space of the diving bell. Therefore, it is unlikely that the P CO2 would rise to 80 torr.

It might be interesting to repeat this 250 year old type of breath-hold diving and to utilize some of our modern capacities for gas analysis and simulated diving. Varying the depths and the external volumes should provide results with which to test some of the recent mathematical models which predict gas exchanges and partial pressures.

It is tempting the conclude that the increase of the CO2 might have been the major factor limiting the diving time under these conditions. On the other hand, in 4 C water the diver would lose considerable body heat even in 15 minutes. Kang et al (3) found that the Korean Ama's working time during the winter months when the water was 10 C averaged 16 minutes. At all times of the year the working time related directly to the water temperature. For the Ama the limits of the working time were dictated by the loss of body heat, and the same might have been true for the salvage workers on the Vasa.

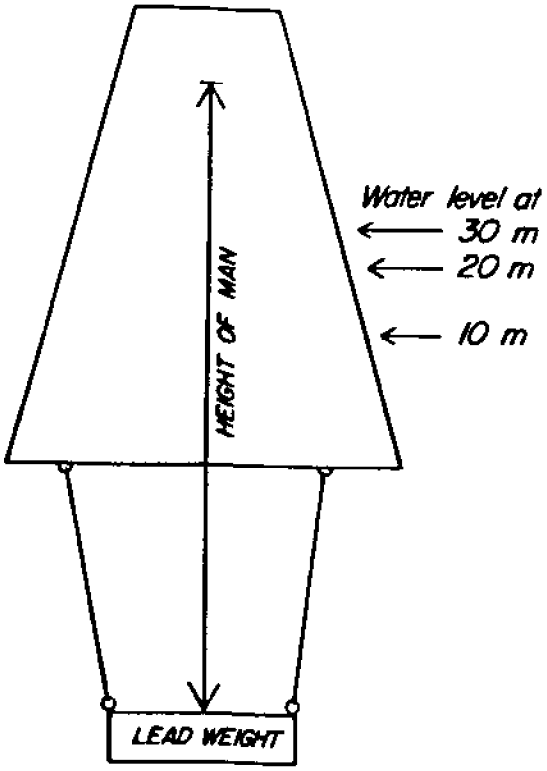

Fig. 4. Calculated level of the water inside the diving bell at different depths.

Using a diving bell to salvage the cannon from the Vasa was a tremendous feat. In the Ama Symposium in 1965 and the current meeting there have been many papers concerned with gas exchanges during breath hold diving. However, most discussions have been limited to the diver who makes a large inspiration and has this air supply for the dive. Reconsideration of the older techniques which employed the diving bell might be an interesting extension of breath-hold diving to explore.

REFERENCES
1. Franzen, A. The Warship Vasa (5th ed.) Stockholm: Norstedts/Bonniers 1966.
2. Triewald, M. Konsten att lefwa under vatn. Stockholm 1734.
3. Kang, D.H., S. H. Song, C. S. Suh, S. K. Hong. Changes in body temperature and basal metabolic rate of the Ama. J. Appl. Physiol. 18: 483-488, 1963

Discussion following Dr. Craig's presentation

Dr. Rahn: You had about 100 liters of oxygen in that bell at the surface and you were using it up at 1 liter per minute according to your protocol, so there was more than enough oxygen at 4 atmospheres. They were actually breathing inspired oxygen tensions equivalent to 80 percent, and so it seems to me there was no problem in terms of oxygen.

Dr. Craig: I didn't see any problem.

Dr. Rahn: And if I make similar calculations for CO_2, there really isn't much of a CO_2 problem.

Dr. Craig: Well, if you assume that all the CO_2 is expired in the atmosphere, it might get up to 70 torr, but you have to partition the CO_2 between two compartments: the body and the atmosphere, and I think this is an interesting problem.

Dr. Elsner: There is also a third compartment, that is the CO_2 dissolved in the water.

Dr. Rahn: So you think the problem was body temperature.

Dr. Craig: I don't know. I just opened the problem.

Dr. Rahn: Any indication of the lights that they used?

Dr. Craig: No indications of any lights. In the Stockholm harbor when you stir up the silt you get zero visibility. I just don't see how they did the job. But they brought up almost all of the cannon. During the salvage procedures they never used lights for anything. They did everything by feel.

Dr. Hong: It is inconceivable that they could stay 15 minutes in $4^{o}C$ water. The Ama tolerate $10^{o}C$ water for 16 minutes.

Dr. Craig: They had some clothing protection.

Dr. Siesjo: Was there any special reason why they worked in water of $4^{o}C$? Even in Sweden there are a few weeks when the temperature gets higher.

Dr. Craig: Not at 30 meters of depth.

Dr. Elsner: How did they actually do the work? Did they take a breath inside the bell and go outside and work, then go back inside the bell, or did they manage to stay inside the bell and still perform the outside work?

Dr. Craig: I don't know. The book that describes this is in old Swedish and this information may be in that book.

Discussion following Dr. Craig's presentation

Dr. Ferrigno: Dr. Craig, you showed an elephant seal. Does this diving mammal have a sphincter in his inferior vena cava?

Dr. Elsner: There is a very well defined muscular sphincter in the inferior vena cava just above the diaphragm and from some other studies we did in a different species we have some idea of the occurrence of a partial constriction of that sphincter during diving. It appears that the flow of blood from the inferior vena cava passing through that sphincter takes place at a very sluggish rate during the dive.

Dr. Lanphier: In preparing for this workshop I went through my old files on the subject and I discovered among many other things a letter that I had written to Dr. Craig shortly after publication of his "Fennology" paper, trying desperately to call his attention to the fact that residual volumes as ordinarily measured are essentially irrelevant with regard to the depth of breath-hold diving. I don't think that there is time to discuss the matter at the moment, so we will let it stand as an apple of discord on the table and hope we can get back to it. Because I think it is awfully important.

Dr. Craig: May I make a comment about that? I do remember the letter. The residual volume that we used was measured by the technique Dr. Rahn et al. and I think that it was a pretty good technique. In addition, we tried to express further air after expiring to minimal volume. One method of doing it was taking one of the surgical abdominal binders and putting it around the thorax, and then having two strong students stand on either side and pull until the subject raised his hand to stop. We could get out about 200 ml more. An alternative method was to breath out to residual volume and hold a tube in the mouth that was connected to a water/air system. With this you could suddenly exert a negative pressure of 20 cm H_2O at the mouth and see if you got out anymore volume. Now I know you are going to say that the airways were closed, but we could under those conditions get out only 200 ml. So we made an attempt to see if we were at residual volume. Now of course I realize what you would say, "those techniques are not good because of the airways closure and you can't get the air volume out but you can still compress it".

Dr. Lanphier: Yes, that is exactly it. I admire your attempt to get around it, but there isn't a way to get around it, except by measuring it by a method involving compression.

Dr. Craig: Which is the body box.

Dr. Lanphier: Yes, under increased pressure, and I don't think anybody has done that.

Dr. Craig: Well, with the body plethysmograph the defined residual volume is the same as with other methods.

Dr. Lanphier: You do that by going to FRC and substracting what you can exhale, but that is limited by airway closure and so on. I think

it is an area of discord and we don't have time to talk about it.

Dr. Hong: What is the maximal amount of blood that can be shifted into the thorax during diving? It seems to increase every year as indicated by the increase in diving depths. Also, can we train divers to increase the volume of blood shift into the thorax?

Dr. Craig: I would like to think that they haven't yet reached the limit of the amount of blood that can be shifted. Some techniques can be useful. For instance, if I were going to attempt a record breath-hold dive, I would start out of the water, not in water. And also remember that at those depths the changes in volume become relatively smaller. I think the limit will be reached when one of these record breath-hold divers comes up coughing blood.

Dr. Arnold: If, as Dr. Craig suggested, we have a blood exchange in the thoracic cavity explaining the ability to tolerate the great depths, the blood in a seal would be in the inferior vena cava, which is well adapted to have a quick venous return. However, in a human there may be the bronchial arterial blood which is not as readily available to venous return. If that's true and if thoracic pressure increases quickly, venous return may be substantially compromised.

Dr. Lundgren: I would like to draw your attention to the fact that the work that Dr. Ferrigno will present addresses precisely this problem, that is, the importance of intrathoracic pressure changes with depth for cardiac function.

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DEPTH LIMITS of BREATH-HOLD DIVING

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In 1951 the record depth of a breath-hold diver was 35 m. Competition in this arena by Croft, Maiorca, Mayol, and others resulted in an increase of the depth to 65 m by 1967. These feats indicated that our concepts of the depth limits of breath-hold diving had to be extended.

According the Boyle's Law the volume of gas in the body must be compressed during descent and the internal and ambient pressures must be essentially equal. Otherwise, pressure differences would result in barotrauma which in the thorax would cause pulmonary edema and/or hemoptysis. Rahn (7) indicated that it is necessary to consider both the rigid and collapsible volumes of the airways. The former spaces are represented by the trachea, upper airway, inner ear, etc. The collapsible air containing spaces are in the thorax. "The residual volume (minus the dead space) is assumed to be the minimal alveolar (volume) which can be tolerated, since we have no experimental data to the contrary."

If a breath-hold diver's residual volume (RV) were 20% of the the total lung capacity (TLC), it was predicted that the maximal depth of a dive would be about 40 m. (Fig. 1). Below that depth the diver's lungs and thorax would act like a rigid system and would be subject to a "lung squeeze".

The physiologic conditions of a diver at this depth were simulated under laboratory conditions by beginning a dive at RV (2). Before each dive the subject hyperventilated for about 30 seconds and breathed 100% O2 during the last 3-4 breaths. These conditions insured that the dives were not limited by hypoxia or hypercapnea (1). The difference in pressure between an esophageal balloon and another one strapped to the lateral thoracic wall was recorded. The pressure at the later site also indicated the depth of the dive (Fig. 2). The first dives were performed cautiously in a laboratory tank the depth of which limted to dives of 1.5 m. As the pressure difference after rapid descent was no more negative than might be expected from

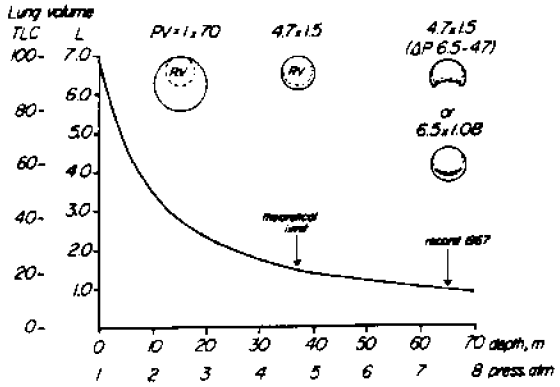


Fig. 1. Presure volume relationships during breath-hold diving. The ordinate indicates lung volume as a per cent of TLC or as liters for a diver whose TLC is 7.0 l. The theoretical limit as indicated by an arrow is defined by the RV:TLC. At the depth record in 1967 2 alternatives were considired. One indicates the the development of a transthoracic pressure difference of 1.8 amt. which would kill the diver, and the other further gas compression.

the static recoil of the lungs and the thorax, deeper dives were conducted in swimming pools and finally in a lake. Even after a dive to 4.75 m the pressure difference was not great enough to produce a "lung squeeze" (Fig 3). However, at this depth the diver did report vague and mild substernal pain.

The diver's RV, determined repeatedly over several years, was 2.0 l. The estimated gas volumes in the lungs during these dives are shown in Fig. 3. These calculations neglected both the transfer of gas into the non-collapsible portion of the airway and changes due to gas exchanges with the circulation. Therefore, they reflect the maximal volume to which the lungs might be compressed. At 4.75 m the beginning gas volume of 2.0 l must have been compressed to at least 1.4 l, a difference of 600 ml. Using these observations, it was possible to

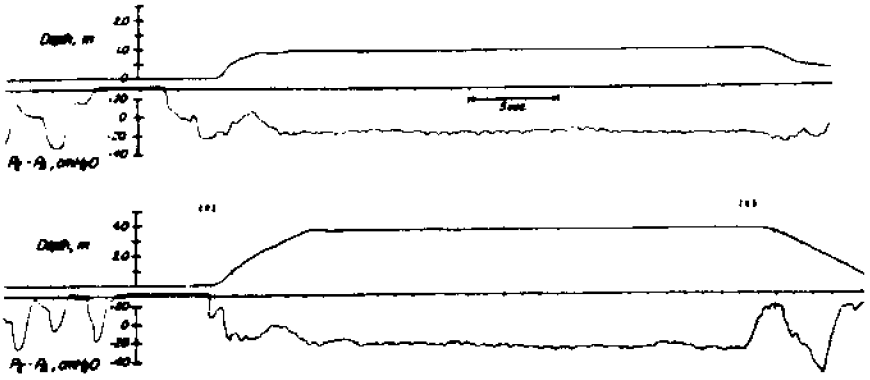


Fig. 2. Photographs of 2 records during dives. The upper half of each shows the pressure, expressed as depth, in the balloon straped tro the lateral thoracic wall. The lower part is the record of the pressure difference between the outer and the esophageal balloon.

reconstruct a dive in which a subject with a TLC at the surface of 7.0 l and a RV of 1.5 l would descend to 65 m, which was the record in 1967. Under these conditions the lung volume would have to be compressed to 570 ml less than the RV. It was suggested that if the RV represented the minimal anatomical volume of the thorax, the transfer of blood from the external to the internal thoracic regions would be a mechanism by which the lung gas volume might be further compressed (Fig 4).

The current depth record of breath hold diving held by Maiorca is 100 m. As there was no evidence of barotrauma in that dive, it is apparent that the gas volume at that depth must have been 9% of the surface volume. Using the beginning conditions mentioned above for a 65 m dive, a plunge to 100 m would require a minimal gas compression to 870 ml less than the RV.

Lanphier has indicated (personal communication) that measurement of the RV by any of the existing techniques only measures the minimal lung volume to which a subject can expire using a maximal muscle force. He suggested that during breath-hold diving the alveolar volume could be compressed farther without supposing the transfer of blood. After discussing this possibility, we attempted to compress our subject's thoracic volume to less than RV by applying the maximal external force which the suoject could tolerate. Under these conditions it was possible to express about 200 ml from the airways into a spirometer. Other attempts to remove gas from the airways after expiring maximally were made by rapidly applying a negative pressure of 20 cm H20 at the mouth for 10 seconds with care to keep the glottis open. In five subjects an average of 250 ml was withdrawn from the airway. From our observations adding an external or an internal force to that produced by the muscles did not change the RV appreciably.

During a dive the compression of the air in the lungs must be associated with a physical change in the size of the thorax. When capacity of this mechanism is reached, some other means of air compression such as an increase in the blood volume within the thorax must occur. Transposition of blood in

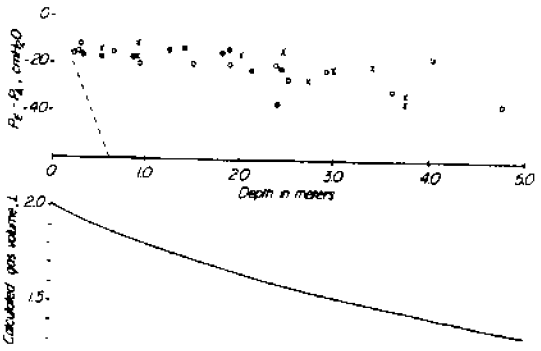


Fig. 3. Observed transthoracic pressure differences during three different series of dives are shown in the upper part. The circles indicate dives performed in two different pools, the crosses in a lake. In the lower part the calculated change in gas volume in this subject whose lung volume at the beginning of the dive was RV and equal to 2.0 l is indicated.

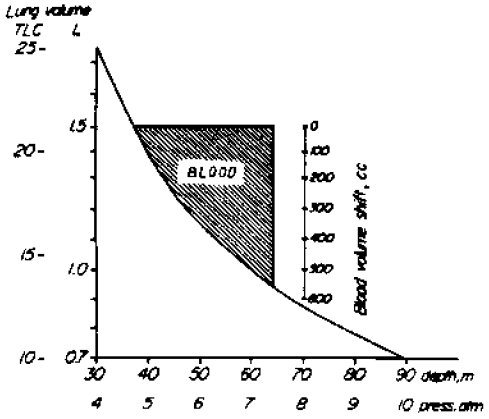


Fig. 4. The cross hatched area indicates the blood volume which would be required to maintain pressure equalization assuming that the diver started from the surface after inspiring maximally. Changes in volume related to gas exchanges with the circulation have been neglected.

and out of the thorax is well known in some other situations. During positive pressure breathing as much as 830 ml of blood could be displaced from the thorax to the lower parts of the body in one subject identified only as Rahn (4). It is also known during negative pressure breathing the thoracic blood volume is increased (7).

Such a shift of blood in other parts of the body has been postulated. The transfer of blood into the venous channels and sinuses in the middle ear of sea lions and seals has been suggested as a way of providing pressure compensation across the tympanic membrane (5,6). It was noted that the elephant seal has a large inferior vena cava which extends from below the diaphram to the distal portion of the abdominal cavity (3). In one large male this vessel contained 20 l blood which was about 25% of the total blood volume as measured by exsanguination. During this same study another male seal was contained in a cargo net, lowered to 300 m, and brought to the surface without apparent ill effects. At that depth the gas in the air containing spaces must have been compressed to 3% of the surface volume. Even though the elephant seal's ribs are completely cartilagenous, it is difficult to conceive of a mechanical change to 3% of the surface volume. It was suggested that the venous resevoir might represent a readily available source of blood which could be transfered to the thoracic region and would provide additional gas compression.

This proposed shift in blood volume as a mechanism which enables human breath-hold divers to achieve depths beyond that accounted for by reduction in the size of the thorax, poses some practical considerations. It is known that immersion up to the neck results in negative pressure breathing which causes a shift of blood into the thorax (7). If a diver attempts a record dive, it would seem reasonable that he should remain out of the water prior to diving and also be in the upright position. Such conditions would minimize the thoracic blood volume and maximize the blood in the periphery. Theoretically, this preparation would optimize the amount of blood which could be shifted into the thoracic regions to provide pressure compensation.

If the depth limit of breath hold diving is associated with such transfers of blood, it would be predicted that the ultimate record for such diving will be signaled by the development of hempotysis and/or pulmonary edema. I hope that such a person has minimal and transient disorders which will indicate to others that the physiolgic limits of breath-hold diving have been reached.

Permission to reprint Fig. 1-4 was obtained from the publishers of Resp. Physiol.

REFERENCES

1. Craig, A. B., Jr. Causes of loss of consciousness during underwater swimming. J. Appl. Physiol. 16:583-586, 1961.
2. Craig, A. B., Jr. Depth limits of breath hold diving (An example of Pennology). Resp. Physiol. 5: 14-22, 1968.
3. Elsner, R.W., P.F. Scholander, A.B. Craig, E.G. Diamond, L. Irving, M. Pilson, K. Johansen, and E. Bradstreet. A venous blood oxygen resevoir in the diving elephant seal. Physiologist 7:124, 1964.
4. Fenn, W.O., A.B. Otis, H. Rahn, L.E. Chadwick, and A.H. Hegnauer. Displacement of blood from the lungs by pressure breathing. Am. J. Physiol. 151:258-269, 1947.
5. Mohl, B. Seal ears. Science 157:99, 1967.
6. Odend'nal, S. and T.C.A. Poulter. Pressure regulation in the middle ear cavity of sea lions: a possible mechanism. Science 153:768-769, 1966.
7. Rahn, H. The physiolgic stresses of the Ama. In: Physiology of Breath-Hold Diving and the Ama of Japan. Ed. H. Rahn. Nat. Acad. Sci. and Nat. Res. Council (U.S.A), Publication No. 1341, 1965.

Depth and Time In Relation to Gas Exchange

A.Olszowka

As an explanation for the occurrence of accidental drowning in breath-hold dives Craig[1] has proposed that the hyperventilation prior to such dives could reduce CO_2 sensitivity and thus allow a profound hypoxia to develop leading to unconsciousness. On the other hand Paulev has reported on cases of decompression sickness[2] as well evidence of increased CO_2 tensions[3] in some tissues following repeated breath-hold dives.

In order to explore in more detail the changes of O_2, CO_2, and N_2 that occur in a breath-hold dive a computer model was developed which permits one to simulate the changes in the tensions of all 3 gases in the lungs, blood, and tissues.

MODEL DESCRIPTION.

Figure 1 shows the outlines of the model used to analyze gas exchange during a breath-hold. It consist of a series of stores forming a closed loop.

Lung stores

Both oxygen and nitrogen stores consist of a gas phase only while carbon dioxide is assumed to have a tissue component which has an effective volume (ELTV) of 1.5 liter.

Blood stores

The blood stores are broken into an arterial, a venous, and an "effective" reserve volume.The size of the arterial and venous volumes are set to the prebreath-hold cardiac output multiplied by the respective transit times, which are assumed to be 5 liters/min ,6 seconds ,and 12 seconds respectively. These volumes are each subdivided

into smaller volumes of equal magnitude in each of which perfect mixing is assumed to exist and changes in concentration are proportional to the difference between the mass of gas entering the volume minus the amount leaving it.

The size of the "effective" reserve volume was assigned a value which yielded an alveolar Po_2 versus time profile which best fitted the breath-hold data of Hong et als[4].

Tissue stores

Since oxygen is poorly soluble and the venous oxygen tensions in the analysis is substantially above the P50 of myoglobin,oxygen tissue stores are assumed to be nonexistent.

Nitrogen stores were assumed to be made up of four compartments:

1)Compartment A having a volume of 0.3 liters receiving 24% of the resting cardiac output and representing kidney.

2)Compartment B having a volume of 5.5 liters receiving 48% of the cardiac output and representing brain,heart,liver and the G.I. tract.

3)Compartment C having a volume of 48.1 liters receiving 18% of the resting cardiac output and representing muscle, skin and the connective tissue. In those simulations where the cardiac output increased during the dive it was assumed that all the increase in flow went to this compartment. Similarly when the oxygen consumption increased during the dive it was assumed that all the increase also occurred in this tissue.

4)Compartment D having a volume of 50 liters receiving 10% of the resting cardiac output and representing fat. Factored into its large volume is an assumed nitrogen fat tissue blood partition coefficient of 5.

Changes in the tissue Carbon Dioxide stores were computed by assuming that end-capillary Pco_2 represented tissue Pco_2 and that the CO_2 volume of a compartment equals the total CO_2 store times the fraction of the total body volume representd by that compartment. The total CO_2 store was assigned a volume which yielded an alveolar Pco_2 versus time relationship which best fitted the breath-hold data of Hong et als[4].

COMPUTATIONS.

Alveolar gas composition prior to the breath-hold was computed using the classic alveolar equations. Pulmonary end capillary gas concentrations are then computed by means of algorithms described elsewhere[5] assuming alveolar and end capillary equilibrium. These concentrations are then used to compute the arterial and mixed venous ones assuming respectively the values 0.2 and 5.0 L/min for the right to left shunt fraction and pre-breathhold cardiac output. For all gases tissue partial pressures are assumed to be in equilibrium with the venous blood draining the tissue and at the beginning of the breath-hold the composition of the blood reserve volume is set equal to that of the venous blood.

When exchange in the unsteady states is analysed a set of mass balance equations are used to quantify the gas transport in each compartment during a small time interval DT. For the lung,blood, and N_2 tissue stores this is expressed as as an equality between the change in the amount of gas in the store and the amount of gas removed from or added to the blood perfusing that store during the interval DT. A similar expression is used for the O_2 and CO_2 tissue stores except that

the amount of O_2 removed or CO_2 added by metabolism is added to one side of the equation.

Starting with the lung, and then proceeding in turn to the arterial, tissue, blood reserve and venous compartments one can by solving the resulting set of equations use the concentrations at time T to obtain the concentrations at time T+DT. Setting T = T + DT ,the process can be repeated starting again with gas exchange in the lung stores.

Model Testing. Before applying this model to a breath-hold dive it simulated the alveolar O_2 and CO_2 tensions during a four-minute breath-hold at the surface using the lung volumes and oxygen consumption rates recorded by Hong and associates[4] for 9 subjects, average age 30 years,TLC (BTPS) = 5450, R.V. = 1470, and O_2 consumption = 247 ml/min, who held their breath after a full inspiration without prior hyperventilation. Every 30 seconds they expired into a small bag to obtain an alveolar gas sample and reinspired.

In those experiments a significant increase in uptake of oxygen from the lung was noted in the first minute of the breath-hold which is consistent with an increase in cardiac output. Therefore in addition to adjusting the blood reserve volume we also adjusted the magnitude of this cardiac output increase so as to produce an anveolar Po_2 vs time relationship which best fitted the above data. In figure 2 are plotted the changes in alveolar Po_2 and Pco_2 during a 240 sec breathold assuming blood reserve volume of 3.2 liters,a tissue CO_2 space of 40 liters and an increase in cardiac output of 1 liter per minute. The alveolar O_2 and CO_2 tensions are shown by crosses. The continuous line represents the integration of the alveolar O_2 and CO_2 tensions presented by the model.

Except for the starting values at zero time (not shown) the agreement appears to be reasonable.

It is worth noting in this figure that except for the first 30 seconds, the change in alveolar Co_2 is somewhat less than the change in alveolar O_2. The final alveolar Co_2 attained is highly depended on the size of the Co_2 stores. Using an earlier version of the model in which only one Co_2 compartment was present the resulting size of the Co_2 store that produced the best fit to the data was about half that required using the present model. This difference is due to the presence in the present model of 2 large compartments(muscle and fat) that receive relatively small fractions of the cardiac output and consequently have proportionately less impact on the changes in Co_2 concentrations of the blood than the other campartments. Indeed Farhi[6] has pointed out that in experiments performed to estimate the size of the body Co_2 stores, the reported size of the store tends to be related to the duration of the experiment -a reflection of the fact that compartments with large "time constants" have more of an effect on alveolar gas composition in longer expirements than shorter ones.

Having been tested against data obtained during breath-holds at sea level, the model was then used to simulate the changes in lung, blood, and tissue gas concentrations that would occur in a brathhold dive to a depth of 10 ATM - a feat that has been performed by E. Maiorca one of the participants in this workshop. In these simulaions we assigned to the various blood and tissue compartments the same size as that used in the sea-level simulation. For the TLC we used 7.2 liters a value appropriate for Mr. Maiorca, this information having kindly given to us by him through correspondence with Dr. Ferrigno. Furthermore since Mr.

Maiorca hyperventilates before he dives we arbitrarilay set the predive alveolar Co_2 to 20 Torr.

In such deep dives the compression of the lung can so encrease the partial pressure of the gases that are present, that a significant transfer of gas from lung to the blood and tissue can occur during the descent.In figure 3 are plotted the changes vs time of the volume of O_2 in the lung and blood as well as their sum. The duration of the dive was set to 220 seconds half of which was used for descent and half for ascent back to the surface.Both the rate of descent and the rate of ascent were assumed to be constant.Oxygen consumtion was assumed to be 300 ml per minute.

The slope of the line representing total O_2 stores represents the oxygen consumption. Note that the transfer of O_2 from lung to blood during the descent is of such magnitude that the volume of O_2 in the blood rises during the descent despite the fact that the tissue is absorbing O_2 for metabolism. However at the end of the dive the blood O_2 volume falls at a rate faster than that due to metabolism and during that period the lung O_2 volume is increasing. This implies that there is then a transfer of O_2 back from the blood to the lung.

The reverse in the transfer of O_2 that occurs at the end of the dive is better illustrated in figure 4 in which is plotted the alveolar and mixed venous O_2 partial pressures during the last 30 seconds of the dive. Note that near the end of the dive the mixed venous Po_2 is higher than the alveolar Po_2.

The profile of the O_2 changes that occur in a dive will obviously be affected by O_2 consumption and the Lung volume at the beginning of the dive.In figure 5 are plotted the alveolar Po_2 profiles during such a dive when the O_2 consumtion is 300,350, and 400 ml/min respectively. TLC

is again set at 7.2 L.

In figure 6 are plotted the alveolar Po_2 profiles in such a dive when the TLC is set to 6.0, 6.6, and 7.2 L respectively. O_2 in all three cases is set at 300 ml.

In such deep dives one can also get significant transfers of nitrogen between lung and tissues. In figure 7 are plotted the changes in the combined nitrogen volumes stored in the blood and tissues as well as that stored in the lung. Note that at the end of the dive, not all the nitrogen that was transferred away from the lung is returned to it, resulting in a net increase of 700 ml in the amount of N_2 in the blood and tissues.

While breath-hold dives to 10 ATM are interesting to simulate, of more practical interest is the gas transport in working breath-hold dives to much shallower depths. In figure 8 are plotted the computed alveolar and mixed venous Po_2 during a 1.5 ATM dive. O_2 consumtion during the dive was assumed to be 1 L/min and cardiac output 10 L/min. Descent, bottom, and ascent times were 15, 30, and 15 seconds respectively. Note that in this relatively prosaic dive the possibility of a reverse alveolar-mixed venous gradient exists.

In its present form the model can be used to analyze gas exchange during a single breath-hold dive. During such a single dive it predicts the accumulation of nitrogen in the blood and tissues as well as a reversal of the alveolar-mixed venous O_2 gradient. Of more interest is the accumulated effects in a repeated series of breath-hold dives. To do this the model will be modified to include the gas exchange between the lung and the environment that occurs in the interval between dives.

REFERENCES

1. Craig, A. B. and Harley, A. D. (1961). Underwater swimming and loss of consciousness. Jour. Amer. Med. Assoc. 176: 255-259.

2. Paulev, P. (1965). Decompression sickness following repeated breath-hold dives. J. Appl. Physiol. 20: 1028-1031.

3. Paulev, P. and N. Naeraa (1967). Hypoxia and carbon dioxide retention following breath-hold diving. J. Appl. Physiol. 22: 436-440.

4. Hong, S. K., Y. C. LIN, D. A. Lally, B. J. Yim, N. Kominami, P. W. Hong, and T. O. Moore (1971). Alveolar gas exchange and cardiovascular functions during breath holding with air. J. Appl. Physiol. 30: 540-547

5. Olszowka, A. J., and L. E. Farhi (1968). A system of digital computer subroutines for blood gas calculations. Respir. Physiol. 4:270-280.

6. Farhi, L. E. (1964). Gas stores in the body. Handbook of Respiration Volume II 873-885.

FIGURE LEGENDS

Fig. 1. Outline of model used to analyze exchange in lung, blood, and tissues during a breath-hold. See text for details.

Fig. 2. Alveolar Po_2 and Pco_2 during breath-hold of 240 seconds at sea level. Solid lines are the values produced by the model while the crosses represent average values of 9 breath-holding subjects reported by Hong et als[4].

Fig. 3 Behavior of oxygen stores during a breath-hold dive to 10 ATM. Lower 2 lines represent stores in lung and blood. Tissue stores are assumed to be negligible. Top line represents the total of the oxygen stores.

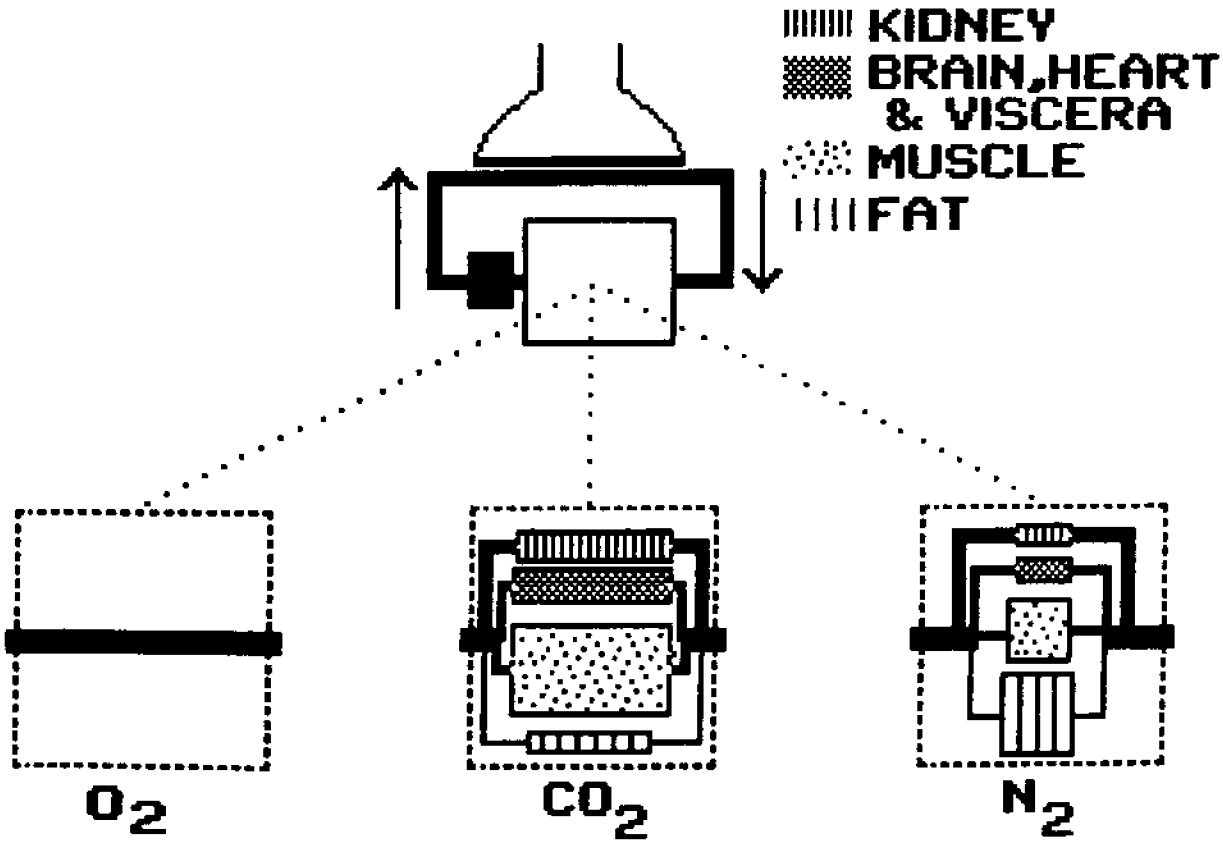
Fig. 4 Alveolar (A) and mixed venous (V) Po_2 during the last 30 seconds of a breath-hold dive to 10 ATM.

Fig. 5 Alveolar Po_2 during a breath-hold dive to 10 ATM when oxygen consumption is 300, 350, and 400 ml per min. The dashed line shows the alveolar Po_2 during a breath-hold at sea level.

Fig. 6 Alveolar Po_2 during a breath-hold dive to 10 ATM when TLC is 6.0, 6.6, and 7.2 Liters.

Fig. 7 Behavior of nitrogen stores during a breath-hold dive to 10 ATM.

Fig. 8 Alveolar Po_2 during a breath-hold dive to 1.5 ATM. Oxygen consumption is 1000 ML per min.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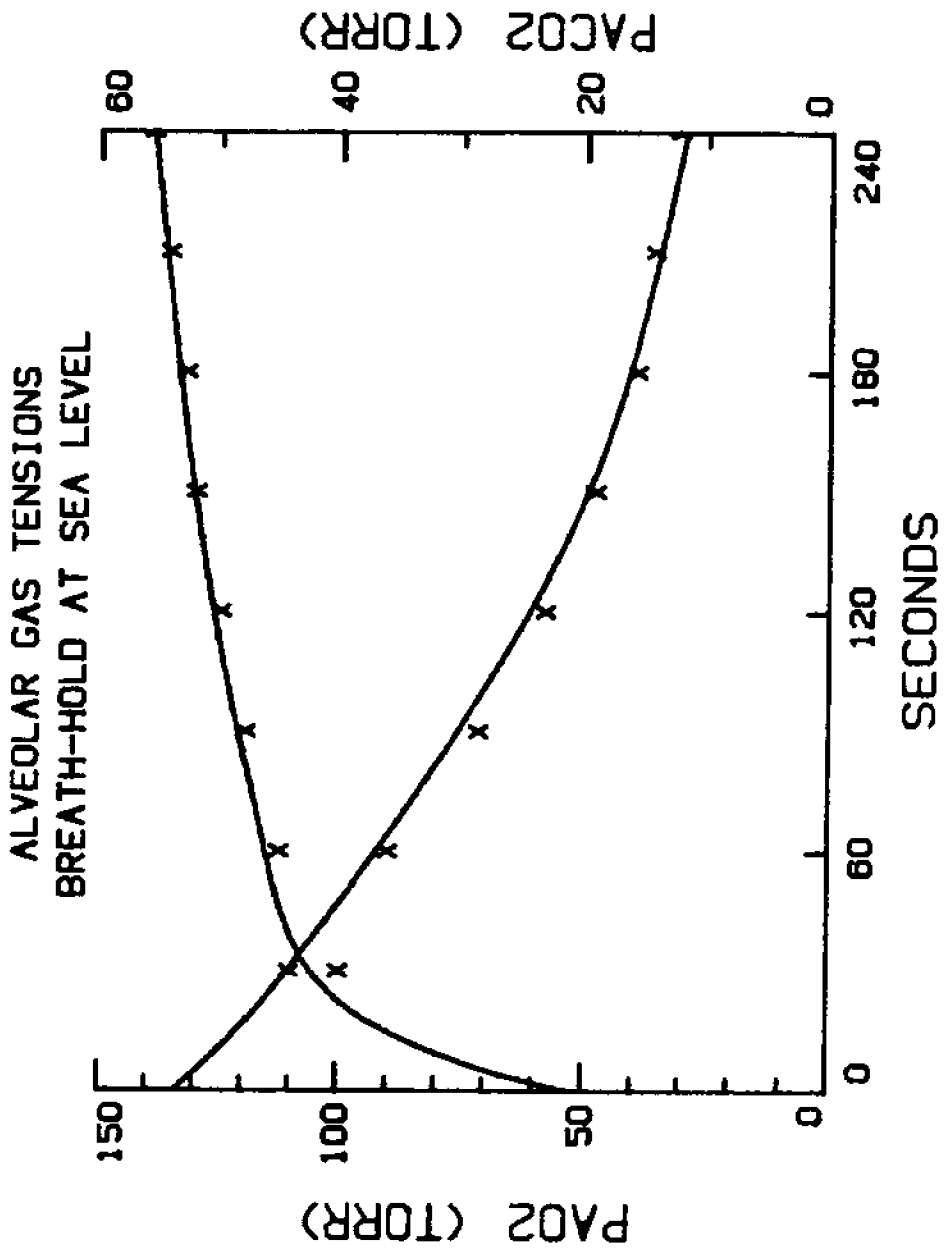
BRAIN,HEART
& VISCERA
MUSCLE
FAT
O2
CO2
N2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BREATH-HOLD AT SEA LEVEL
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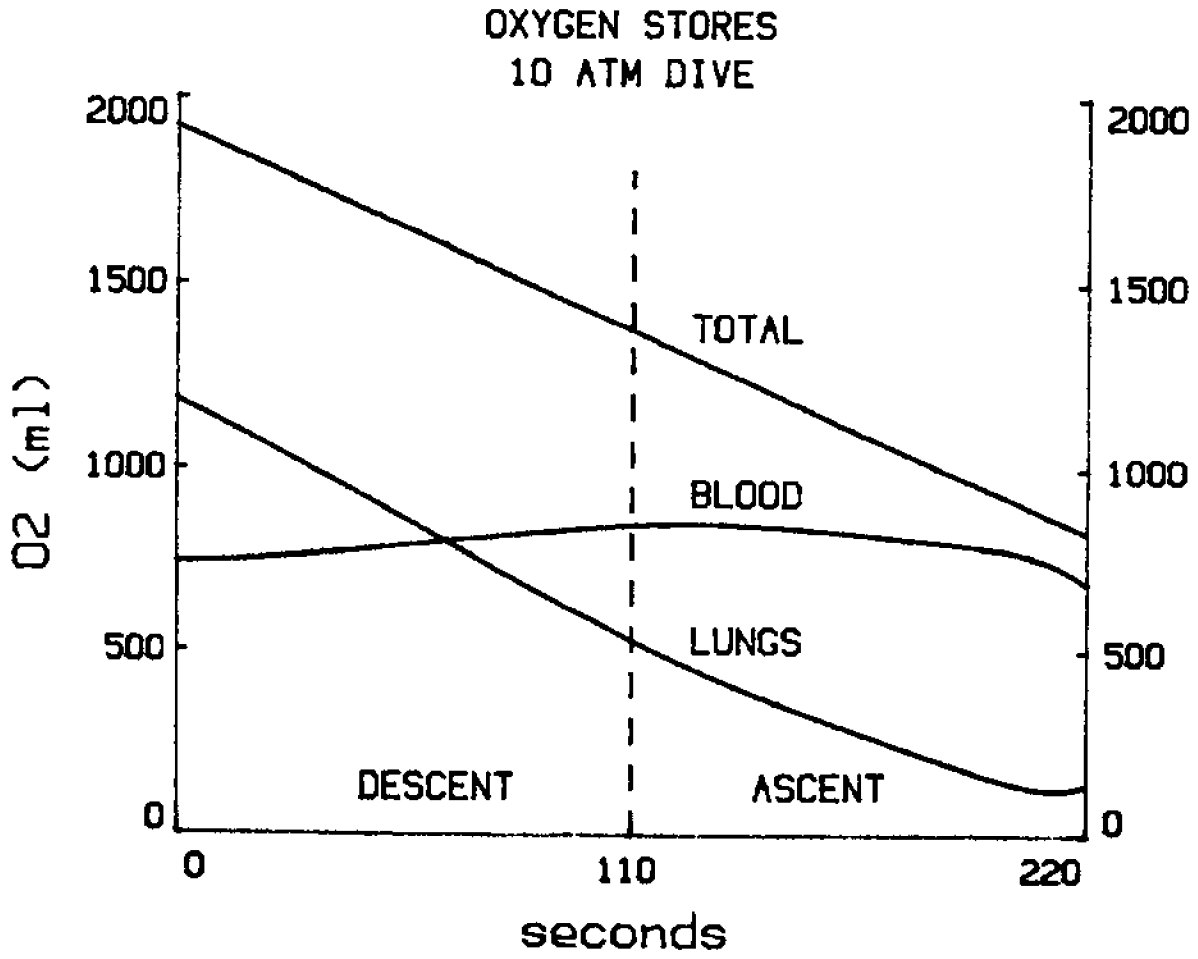
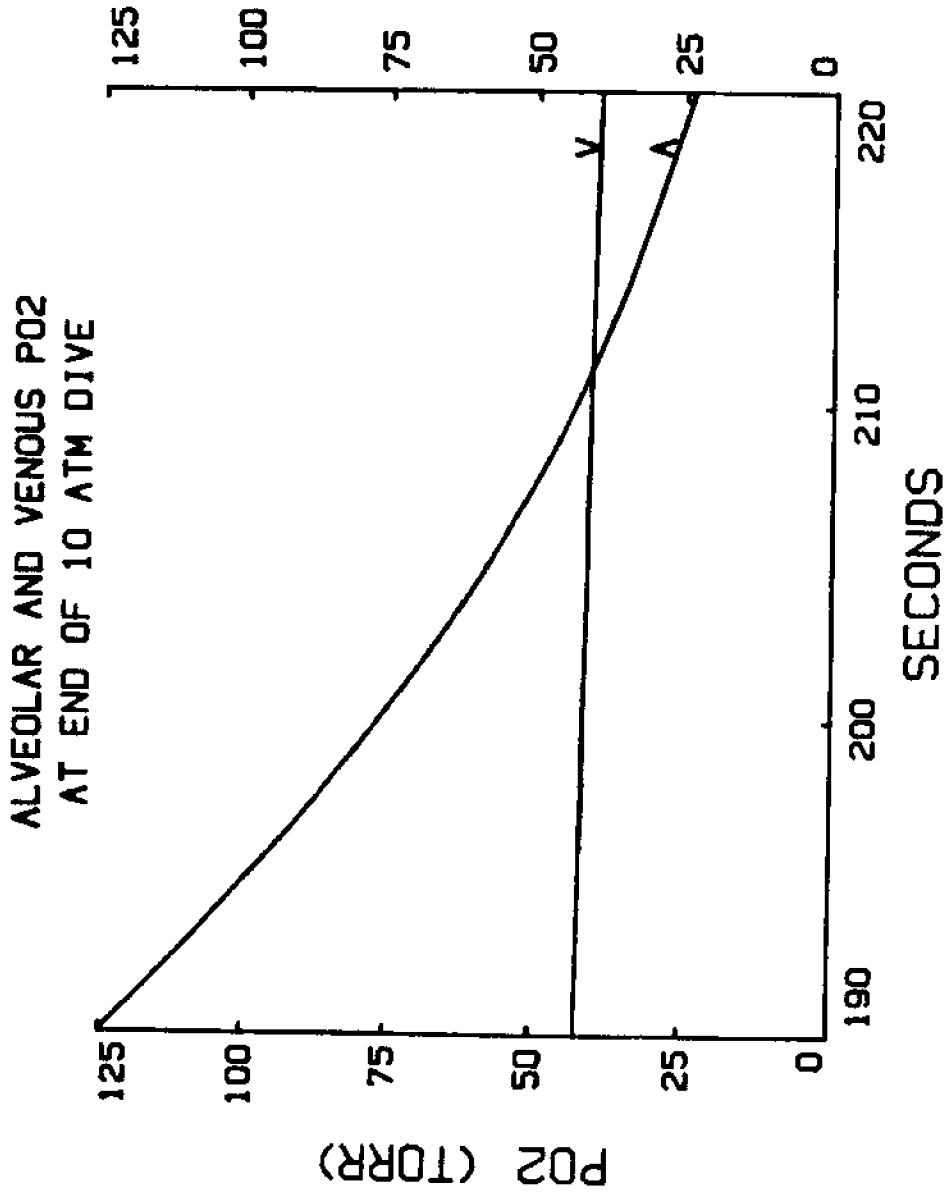
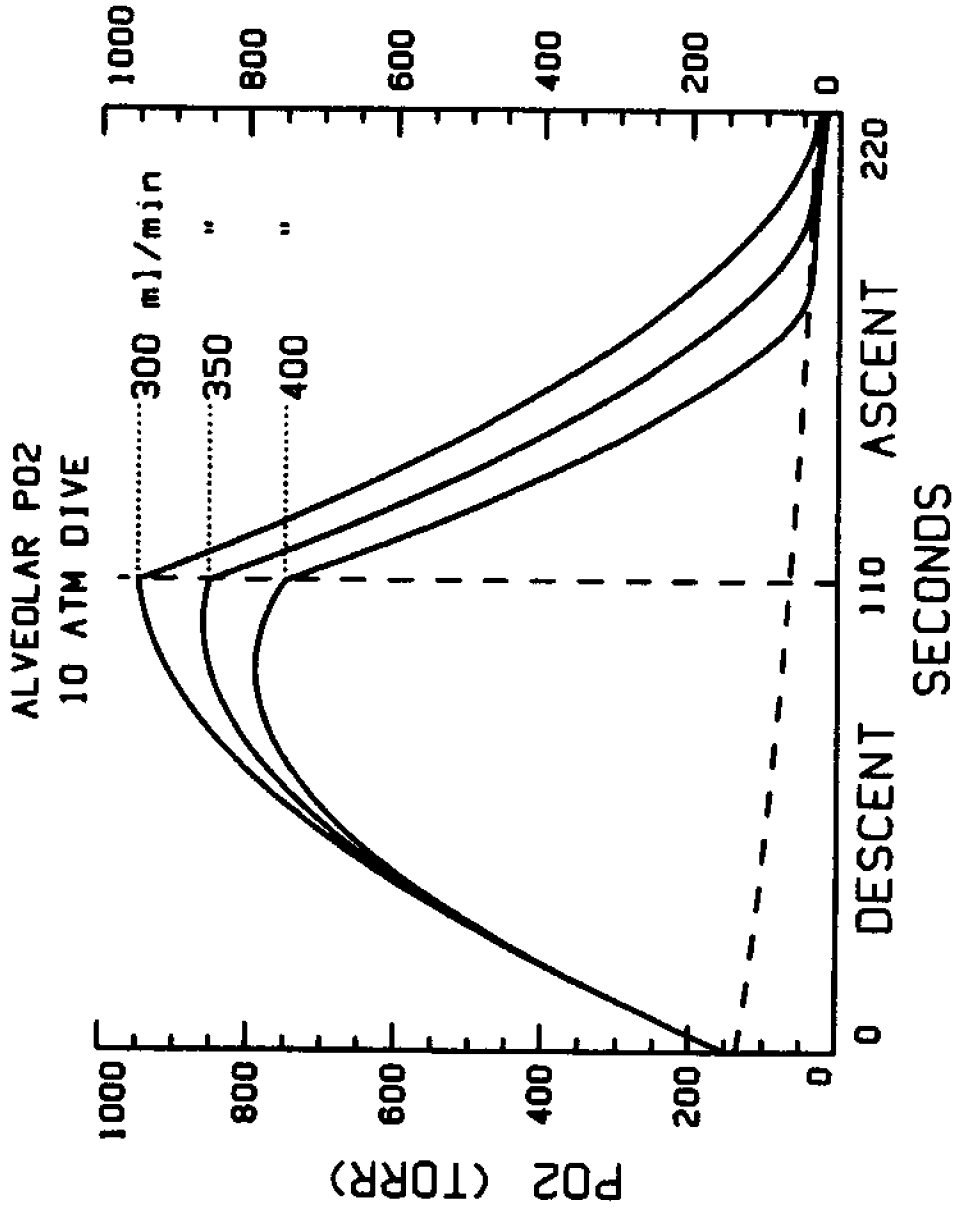
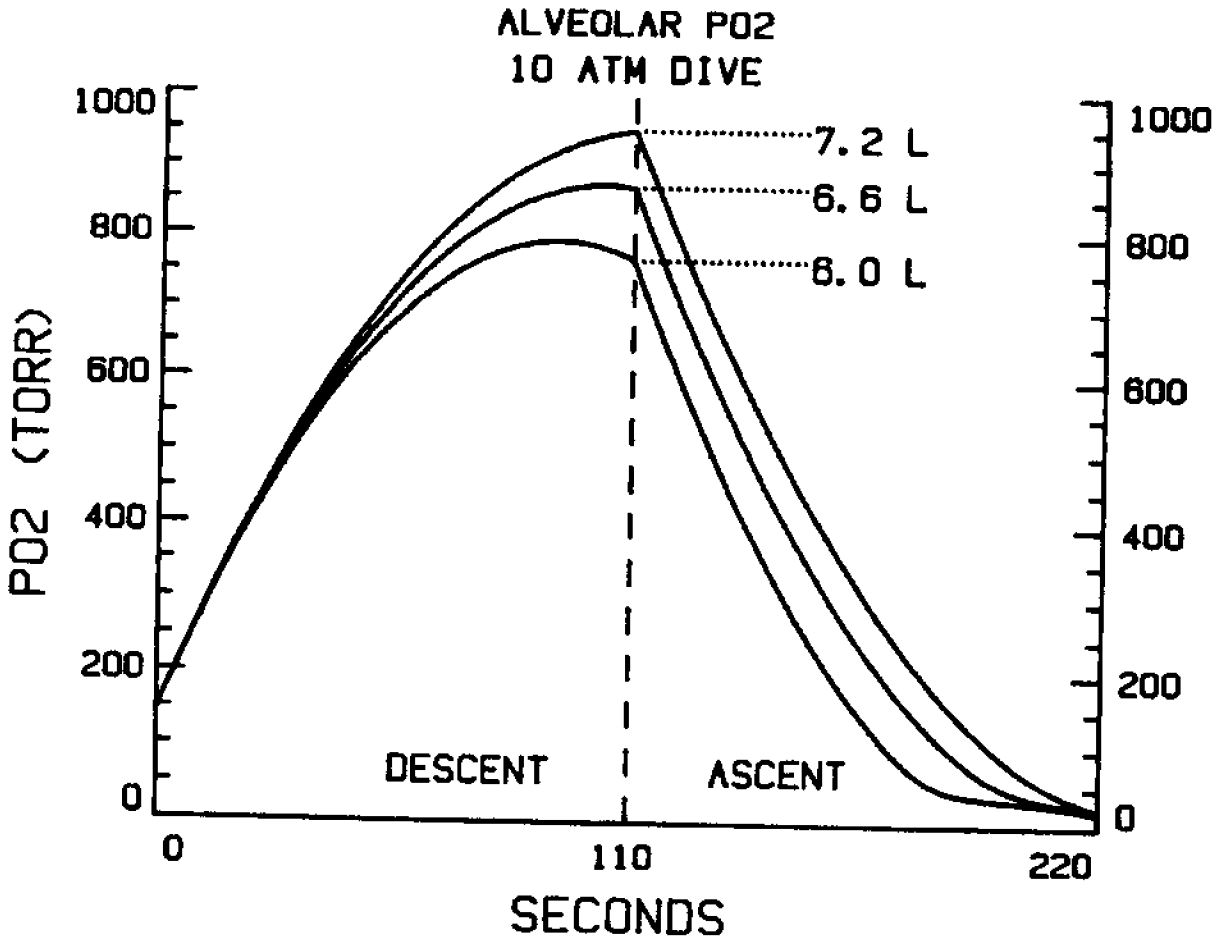
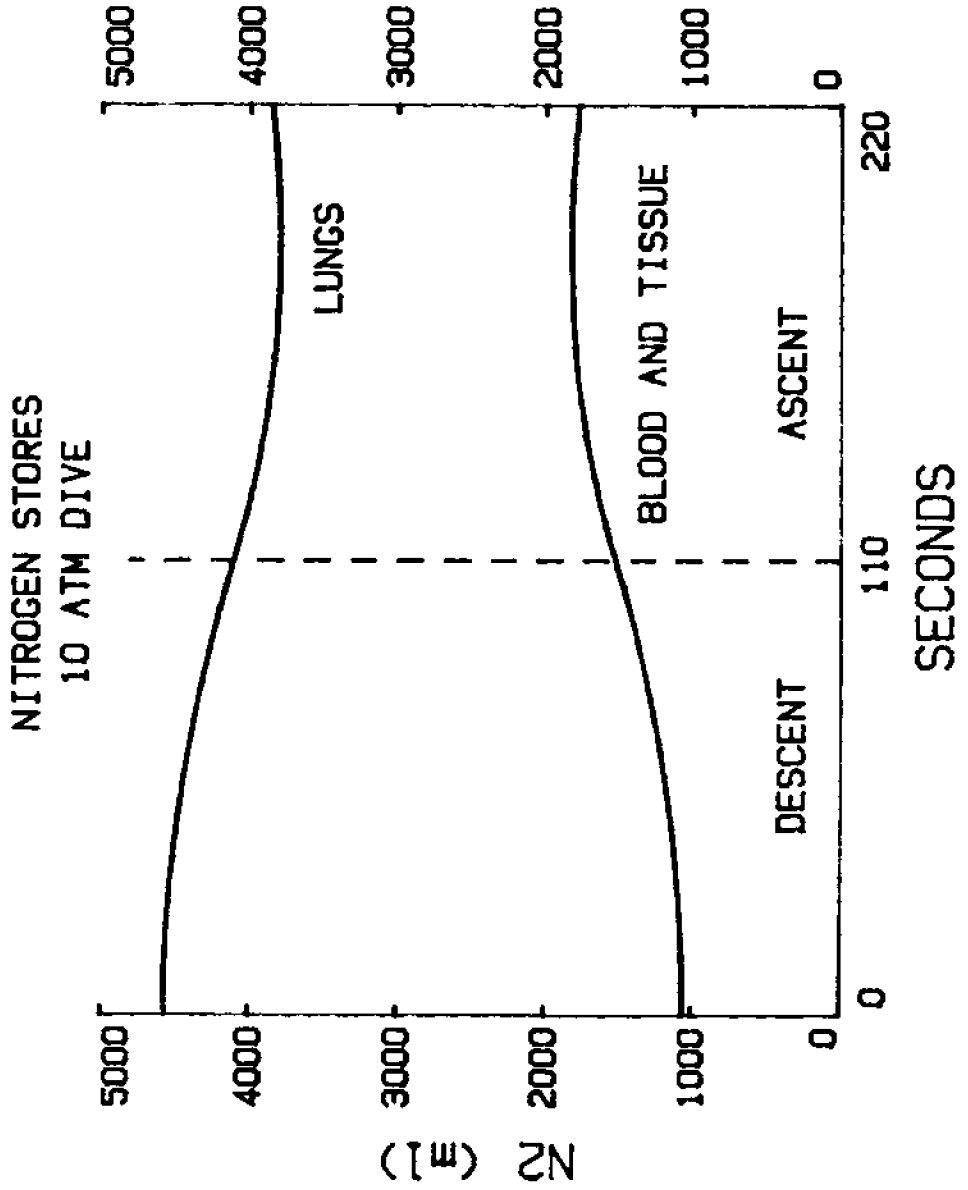
100
50
0
PACO2 (TORR)
60
40
20
0
SECONDS
0
60
120
180
240

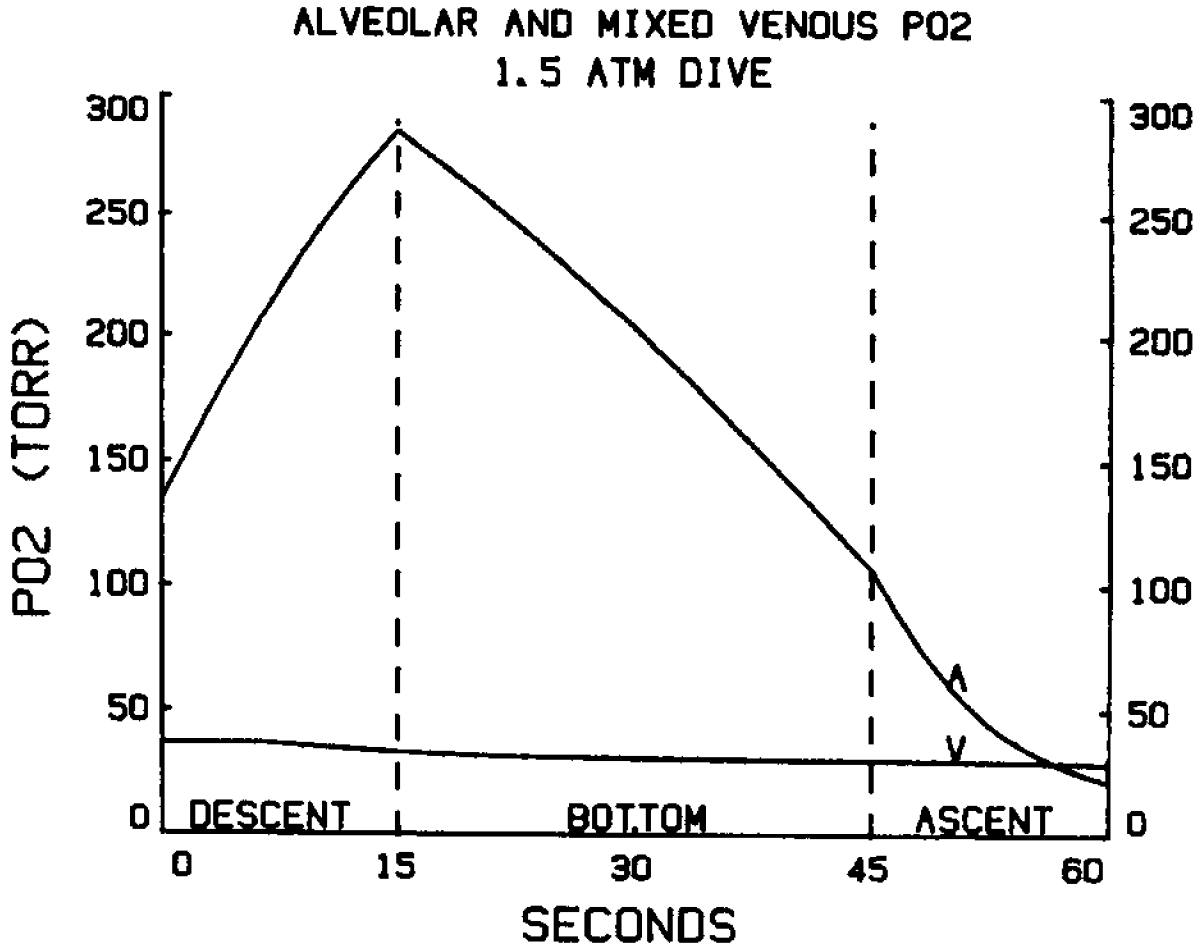

OXYGEN STORES
10 ATM DIVE
2000
1500
1000
500
0
O2 (ml)
TOTAL
BLOOD
LUNGS
DESCENT
ASCENT
0
110
220
seconds


ALVEOLAR AND VENOUS PO2
AT END OF 10 ATM DIVE
PO2 (TORR)
125
100
75
50
25
0
190
200
210
220
SECONDS
V
A


ALVEOLAR PO2
10 ATM DIVE
300 ml/min
350
400
PO2 (TORR)
1000
800
600
400
200
0
DESCENT
110
ASCENT
220
SECONDS


ALVEOLAR PO2
10 ATM DIVE
1000
800
600
400
200
0
PO2 (TORR)
7.2 L
6.6 L
6.0 L
DESCENT
ASCENT
0
110
220
SECONDS


NITROGEN STORES
10 ATM DIVE
LUNGS
BLOOD AND TISSUE
DESCENT
ASCENT
N2 (ml)
SECONDS
5000
4000
3000
2000
1000
0
110
220


ALVEOLAR AND MIXED VENOUS PO2
1.5 ATM DIVE
PO2 (TORR)
300
250
200
150
100
50
0
A
V
DESCENT
BOTTOM
ASCENT
0
15
30
45
60
SECONDS

Discussion following Dr. Olszowka's presentation

Dr. Craig: Dr. Olszowka, you didn't mention CO_2. Would it be a factor during the descent?

Dr. Olszowka: In terms of the effect on oxygen exchange any rise in CO_2 would shift the oxygen dissociation curve a bit and therefore would facilitate the transfer of oxygen between blood and tissues and also -near the end of the dive - between venous blood and the alveoli. Dr. Hong has stated that in his experiments arterial P_{CO2} was in the low 50's or high 40's. In the 10 Atm breath-hold dive simulation arterial P_{CO2} did not go up significantly. In computing CO_2 exchange per se I have assumed that the sizes and flows assigned to the stores to fit the breath-hold data at sea level also apply to the diving situation. This might not be valid in the diving situation because of shifts in blood flow not accounted for in the model.

Dr. Craig: But because of the rate of descent and the depth, doesn't the CO_2 go up as the gas volume of the lung decreases?

Dr. Olszowka: The P_{CO2} in the lung does go up and then as you're coming towards the surface, the P_{CO2} drops in a way that is parallel to the drop in P_{O2}. While the alveolar P_{CO2} does get up to the 60's in the exercising diver at the bottom of the 1.5 Atm dive it is prevented from getting too high by the presence of tissue stores. Then as you are ascending the lungs expand at a rate faster than the rate at which CO_2 can get back into the lungs. As a result alveolar P_{CO2} falls to a value that is not substantially higher than the value at the beginning of the dive.

Dr. Craig: The reason I mention the CO2 is that when we were diving in Hawaii we were going only 10 meters but going down very fast, and when we reached the bottom -where we calculated the P_{CO2} to be very high- we had the sensation that we had reached the breaking point and didn't know how we were going to stay there. Dives to this depth (100 meters) and at this speed must have resulted in values of P_{CO2} that were fairly high.

Dr. Olszowka: I recall that in the 1.5 Atm simulation the values are in the 60's and thus are consistent with the experience you describe. I know, however, that during rebreathing experiments one can easily tolerate P_{CO2} values in the 60's. However, if one could not expand one's lungs, that is to perform respiratory movements, such levels would probably be very uncomfortable.

Dr. Rahn: I would like to interject something about the interesting thing I noticed as I looked over some of Dr. Olszowka's calculations. When you come up after 3 o 4 minutes there has been almost no change in the CO_2 content of the lungs, which did not surprise me because of the big riddle in Teruoka's publications 50 years ago. He had these women go down to 20 meters for approximately 1 minute and when they came to the surface he would get their very first expiration as alveolar samples, which had almost normal CO2 values. Presumably, their excess CO2 was all in the tissues. So the amazing thing to me is that even with the simulation of the 100 meter dive with the Maiorca

pattern, the CO_2 tension at the end of the dive is not very high.

Dr. Lundgren: One comment as far as the practical execution of these dives goes, and Mr. Maiorca can tell you more about it, and perhaps the most impressive illustration will be the excerpts of films that have been taken of Mr. Maiorca's deep dives. They clearly show that these very deep record-breaking dives are always preceded by rather vigorous hyperventilation. The tendency for the alveolar CO2 to rise to high levels during the dive is obviously minimized by that maneuver.

Dr. Olszowka: I should have mentioned that. We simulated the hyperventilation of Mr. Maiorca by assuming that the alveolar CO_2 was only 20 torr before the start of the dive. I now recall that, as a result of this, the highest alveolar P_{CO2} reached during the simulation of this dive was in the low 40's.

Dr. Hong: In your calculations and model, how do you deal with the lung volume during the dive to 100 m?

Dr. Olszowka: I assumed that the gas in the lung was compressible and the resulting volume changes were adjusted only by the net transfer of gas between the alveoli and the blood. I did not address the problem of whether or not the lungs could be compressed. I just assumed they could be.

Dr. Rahn: The program shows that at a depth of 100 m the total lung volume is 400 cc and if you take another 150 for dead space, you haven't got much left. I would like to point out something that I found interesting. You didn't mention anything about the dashed line in figure 5, where you had plotted alveolar PO2 during the 100 meter dive. The dashed line is based on what Dr. Hong did on divers breath-holding at the surface. You can see that at 30 seconds you are below 100 torr and you keep on gradually becoming hypoxic and ending up approximately at 20-25 torr - that' s where unconsciousness sets in. That's why I find this dashed line particularly interesting because in the computations based on Mr. Maiorca's lung volume, going to depth of 100 meters, at similar times you always have a higher alveolar P_{O2} except during the last few seconds, compared to the surface breath-hold.

Dr. Lanphier: What was the oxygen consumption on the dashed line?

Dr. Rahn: 247 ml per minute compared to 300 ml per minute for the 100 meter dive.

Dr. Lanphier: In other words, it was lower than that associated with the continuous lines.

Dr. Rahn: It is very interesting what you can do with pressure to saturate your hemoglobin.

Dr. Lundgren: If I can make a brief comment while we have this slide on, it's true that while Dr. Hong said that the dashed line represents a non dive situation, I would be comfortable in making the

extrapolation to the situation of an underwater swimmer who swims the length or perhaps several lengths of a swimming pool. Although he is now working admittedly at a higher oxygen consumption than that associated with the figure, he represents the surface breath-hold situation more than the diving one because he does not have the benefit of compression. If I may briefly shift to another aspect of Dr. Olszowka's presentation, that of nitrogen uptake, I do not recall the magnitudes of the shifts in nitrogen volumes. But I tried to catch the numbers as they flashed by and it seemed to me that although the total amount of nitrogen taken up isn't terribly large, the fact that a lot of that nitrogen might be in a tissue phase, where the nitrogen pressure is pretty high, could be of consequence. It seemed to me that you would have a tissue phase where, if I did my calculations correctly, you might have a nitrogen pressure of between 4 and 4.5 times the atmospheric pressure. That is an oversaturation in the order of 4 to 1 and the question inevitably comes up whether that is enough to cause a threat of bubble formation.

Dr. Olszowka: At the end of the dive the compartment representing heart, brain and viscera has the highest oversaturation ratio, and it's about 4 to 1.

Dr. Lanphier: I haven't really looked into this question on a single deep dive, but I have no indication that single breath-hold dives have ever caused decompression sickness.

Dr. Olszowka: We do plan to simulate the effect of repeated dives where eventually the additional nitrogen accumulated after each dive might produce tissue N_2 pressures at the surface large enough to produce decompression sickness.

Dr. Lundgren: Certainly we don't know of decompression sickness occurring in these deep dives but the clinical diagnosis of decompression sickness with all its imperfections may not catch bubble formation that actually can do damage. Although it isn't immediately obvious as the diver emerges, it could be something that will show up years later on a cumulative basis.

BREATH-HOLD AND ASCENT BLACKOUT

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The physiology of breath-hold (BH) diving gains importance from associated deaths. The more we can understand about the course of events in these deaths, the more specific and credible our educational efforts will be in helping to prevent them.

Even momentary loss or impairment of consciousness in or under water is enough to result in drowning. Cerebral hypoxia is presumably the cause of impaired consciousness in virtually every death in breath-hold diving. Hypoxia may result from events such as cardiac arrhythmia, but the predominant cause is respiratory: simply staying underwater too long. A basic question is: "Why didn't air hunger force the diver to come to the surface to resume breathing -- or why didn't it do so sooner?"

We need to recognize a somewhat arbitrary distinction among respiratory cases: those in which an attempt to reach the surface from some depth appears to be involved and those in which ascent was probably not a factor. I suggest adopting breath-hold blackout (BB) as an overall term and ascent blackout (AB) for those cases where ascent seems to have been important. "Shallow water blackout" is inappropriate because that term belongs to an entirely different condition described by Barlow and MacIntosh (1) in 1944 and because BB can probably occur at any depth.

BREATH-HOLD BLACKOUT.

BH blackout (BB) is most commonly recognized in mishaps that occur in swimming pools and other relatively shallow water. Often, the swimmer or diver made no attempt at all to reach the surface. The same sequence can presumably occur at any depth, but few cases other than those observed from the surface would be recognized and attributed to BB. Al Craig (2) deserves gratitude for describing BB and bringing it to the attention of physicians and the aquatic public.

I like Hermann Rahn's way of depicting the physiological basis of BB. Fig 1 was borrowed from him. It is based on an O_2-CO_2 diagram. The "CO_2 reservoir" on the right includes a

small column representing the lung. The blood-and-tissue reservoir is much larger; but it can be depleted by hyperventilation, and the input from CO_2 production during a BH may not bring it back even to a normal level. As the lower arrow on the graph suggests, subsequent gas exchange may carry the PO_2 well into the hypoxic zone before the combined effect of falling PO_2 and rising PCO_2 would produce significant air hunger.

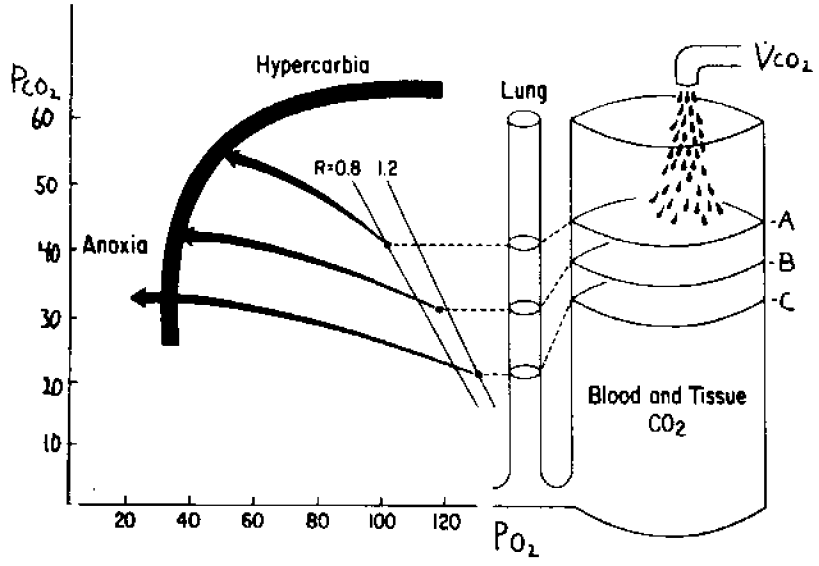

Fig. 1. The course of PO_2 and PCO_2 during breath holding after normal breathing and two degrees of hyperventilation. (From Hermann Rahn.)

It is unrealistic to suggest avoiding hyperventilation entirely. Sciarli (3) proposed a method of keeping it within safe limits, but Hill (4) seems to have torpedoed that approach. Certainly, hyperventilation should not be carried to the point of symptomatic hypocapnia.

ASCENT BLACKOUT.

Ascent blackout (AB) is an insidious hazard. A diver who starts ascent in good condition and at a seemingly appropriate time may lose consciousness and drown before he can resume breathing.

The basic difference between AB and typical BB is a factor that I call "borrowed oxygen." Increased pressure at depth permits a diver to consume more oxygen from his alveolar gas than would be available at normal pressure. The mortgage comes due when he ascends. As ambient pressure drops, the PO_2 of expanding alveolar gas drops in proportion, and the final alveolar and arterial PO_2 may not be compatible with consciousness.

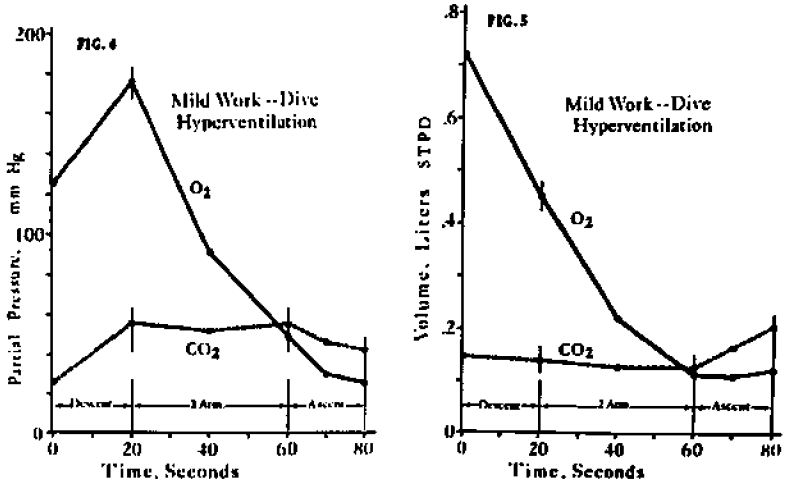

Fig. 2. O_2 and CO_2 pressures and volumes during a simulated BH dive to 2 ATA during mild exertion. From Lanphier and Rahn (6).

If the alveolar PO_2 falls below the mixed venous oxygen tension in the process, the lung will extract oxygen from the blood. In their recent computer modeling of breath-hold dives, Olszowka and Rahn (5) have refined our understanding of this process especially for deeper dives, but I will use a more common sort of example.

Twenty-five years ago, Hermann Rahn and I conducted a series of simulated BH dives in the original chamber at the University of Buffalo. We sampled alveolar gas at intervals by asking the subject to exhale into a small bag to clear his dead space, pulling a small sample for Scholander analysis, and having the subject re-inspire and hold until the next sampling period.

Fig 2 is taken from that study, which we reported in 1963 (6). The graph at the left indicates the values of alveolar PO_2 and PCO_2. The graph at the right shows calculated STPD volumes of O_2 and CO_2 in the lung at corresponding points.

These values are averages from two almost-identical dives by subject DC: Deputy Donald Chamberlin of the Erie County Sheriff's Underwater Division. We chose Don's values, and I am using them now, because he was close to the limits of tolerance for air hunger at depth, and his surfacing values might well have resulted in drowning had he been in the water. Upon surfacing, he was cyanotic and very clearly impaired.

Since these were "dry" dives, we must assume that the effect of immersion on CO_2 storage described by Lundgren (7) would, if anything, have made the CO_2 values lower.

After mild work during 20 sec of descent and 40 sec at 2 ATA, DC's PAO_2 was slightly below 50 torr just before he began ascent. It dropped to about half that value upon his return to 1 ATA. The volume plot indicates that although exertion continued, uptake of oxygen from the lung was decreased even before ascent and ceased in the first part of ascent. Finally, according to our calculations, there was a small reversed transfer of O2 from the blood to the alveoli.

Much greater reversals of O2 transfer are predicted for deeper dives by Olszowka and Rahn (5), and this may occur relatively early in ascent. Reversed O_2 flux is not necessarily indicative of the risk of hypoxia. It often reflects a large amount of O_2 transferred from the lung to blood storage early in the dive.

Rough calculations indicate that DC borrowed and consumed about 175 ml more O_2 than would have been available at PO_2 = 50 torr at 1 ATA. This amount would have met his needs for only about 15 sec, but it is mainly responsible for his close approach to AB.

When ascent began, the PCO_2 started dropping at once and hovered slightly above its normal value. Subjects in this study often commented on the relief of air hunger that they experienced upon ascent. We wondered how many divers have responded to this relief by dallying on ascent and have died as a result. This appears to be a serious hazard. Another critical period is the time at the surface immediately following the first breath. Lin et al.(8) have called attention to the importance of the 5 sec or so in which the

PO_2 of blood reaching the brain will continue to fall. Albano (9) discussed these hazards in graphic terms.

Excessive hyperventilation before a dive can probably lead to quiet loss of consciousness at depth just as it does close to the surface. Perhaps lesser degrees of hyperventilation allow a diver to overstay his time at depth but still try to return to the surface. If the surface is close, neither BB nor AB follows. Deputy Chamberlin had hyperventilated for one minute before his dive, and that was apparently enough to place him at the verge of catastrophe. Deeper depth would have encouraged him to "borrow" even more O_2; and harder work, more time at depth, or more time in ascent all could have pushed him over the edge. It is truly surprising that there are not more fatalities from AB and deep BB than apparently occur.

BREAKPOINTS AT DEPTH

If Don Chamberlin had not been constrained by our protocol, he would have started his ascent a little sooner. If the dive had been a real one in open water, that would have been none too soon. What factors gave DC appropriate air hunger? What factors beside hyperventilation might have caused him to go beyond the point of no return?

Effects of elevated PO_2 at depth.

Maintaining an elevated PO_2 can greatly extend breath-holding time with a corresponding increase in PCO_2 at the ultimate breakpoint. Ferris and Engel and their associates (10-12) were not the first to describe this phenomenon, but they published three papers concerning it in 1946. Probably best known is a 1974 paper by Davidson, Whipp, Wassermann, et al.(13). In Fig 3, I have replotted their values for normal individuals who held their breath after inspiring various concentrations of O_2.

Fig 3 is an O_2-CO_2 diagram, and the values are from alveolar gas collected at the end of breath holds. When the PO_2 is somewhere between 100 and 200 torr, time and PCO_2 both seem to approach a plateau; and further increases in PO_2 have relatively little influence. Stroud reported similar findings in 1959 (14). It was his impression that high PO_2 blocks the ability of the peripheral chemoreceptors to respond to CO_2, leaving only the central response.

A 1951 report by CDR Harry J. Alvis from the Medical Research Laboratory in New London (15) involved breath-holding with air at various depths. I have added Dr. Alvis's values to Fig 3. The experiments were conducted at steady ambient

pressures in a recompression chamber, so they were not simulated dives; but they offer several important lessons.

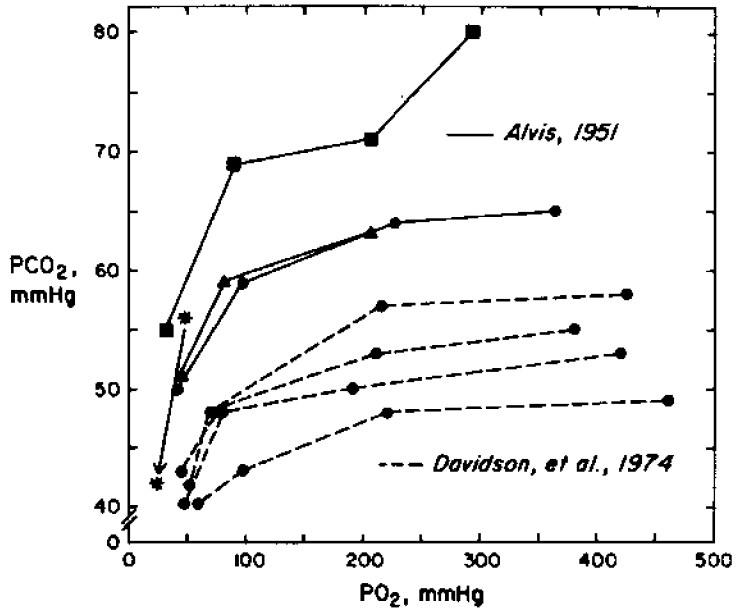

Fig.3. The influence of final PO_2 upon alveolar PCO_2 at BH breakpoint. Values from Davidson et al.(13), Alvis (15), and Lanphier and Rahn (6).

Alvis's subjects were "experienced divers," and perhaps that is why they seem to be a breed apart from Davidson's. Even among the divers, one individual stands out. Presumably, the ability of such divers to tolerate high PCO_2 would put them at exceptional risk of AB.

Alvis's two highest PCO_2 values do not line up very well, but each represents the mean of 6 breath holds, and the S.D.'s are both less than 2 torr. Alvis achieved remarkable consistency as well as remarkable values, and how he did that is part of the story.

CDR Alvis had observed that when involuntary contractions of the diaphragm began, they could be quelled temporarily by swallowing. So he instructed his subjects to hold their breath as long as possible with the help of at least one period of swallowing. Some of his subjects used swallowing

repeatedly, but apparently in a consistent way.

Papers by both Agostoni (16) and Lin, et al.(8) call attention to the considerable time that may elapse between the first involuntary efforts and the ultimate breakpoint. A diver who really wanted to be safe could probably achieve that by starting up when the first involuntary contractions began. By the same token, maneuvers that effectively suppress involuntary activity and air hunger may be responsible for some fatalities. Rigg, Rebuck, and Campbell (17) found that even a strong inspiratory effort or an isovolume maneuver could be as effective in extending time as an actual respiratory excursion.

We see what combinations of PO_2 and PCO_2 caused Davidson's and Alvis's subjects to break their breathholds. Where were Deputy Chamberlin's values in relation to these? I have added two large asterisks to Fig 3 to show them. The upper one indicates Don's PO_2 and PCO_2 just before starting ascent; the lower one indicates his final values upon surfacing.

Even at depth, DC reached a lower PO_2 than any of Davidson's subjects, and his PCO_2 was higher than that of any of Alvis's divers when they held their breath with air at normal pressure. The lower asterisk shows the drop in PCO_2 with ascent. DC's surfacing PO_2 is the lowest on the graph. That supports the impression that such a level is not likely to be reached voluntarily, at least not without very excessive hyperventilation.

What would Deputy Chamberlin's values have been if he had made a real dive but had not hyperventilated at all? We can't be sure. His PCO_2's might have been just about what we see here; certainly, such values would have been reached sooner, and the PO_2 would not have fallen to this extent.

Even though DC's PO_2 was high early in the dive, elevated PO_2 is not likely to have made much difference in his breakpoint. By that time, his PO_2 was unusually low. But that observation does not rule out an unfavorable effect of elevated PO_2 in some dives: a diver who goes to 100 ft (30 m) or deeper ought to begin ascent while his PO_2 is still well above 100 torr. At such levels, his "CO_2 tolerance" probably will be increased, and we may wonder what sensations could possibly tell him that the time for ascent has arrived.

Lung volume.

I have found only one respiratory factor that might reduce comfortable breath-holding time at depth. That is the

reduction of lung volume by compression. Unfortunately, there seems to be no study that directly applies.

Starting a breathhold at a lower lung volume certainly leads to shorter time. With oxygen, low lung volume will increase the rate of rise of PCO_2. With air, it will hasten both the rise of PCO_2 and the drop in PO_2. These changes may explain most of the differences; but the mechanism that is associated with respiratory excursion is probably involved also.

Breath holding with oxygen at normal pressure is probably most nearly comparable to breath-hold dives with air in the sense that falling PO_2 is not a major factor. According to Mithoefer (18), final $PACO_2$ is still more than 80% of the maximum even at a final volume close to RV.

In breath holding after hyperventilation with oxygen, Klocke and Rahn (19) reported such prodigious figures as Hermann Rahn's 14-min time and another subject's final $PACO_2$ of 91 torr. Although they all started at maximum inspiration, some subjects encroached upon their residual volume before breaking. In terms of lung volume, Klocke and Rahn's subjects were certainly comparable to divers approaching maximum feasible depth. It is difficult to support the view that low lung volume, per se, would cause much earlier air hunger except perhaps at an extreme.

SUMMARY AND CONCLUSIONS.

1. Breath-hold blackout (BB) is proposed as the overall term for loss of consciousness due to apneic hypoxia in BH diving. Ascent blackout (AB) would then distinguish cases in which ascent appears to be an important factor.

2. Typical BB is adequately explained by excessive pre-dive hyperventilation. The diver's PO_2 falls to a hypoxic level, and consciousness may be impaired or lost before significant air hunger develops.

4. Hyperventilation is probably involved in most cases of AB also, but other factors are also implicated. Increased ambient pressure permits oxygen to be "borrowed" from alveolar gas, and subsequent ascent may result in PaO_2's incompatible with consciousness.

5. Alveolar PCO_2 drops on ascent, and this may confer relief from air hunger. If relief causes the diver to tarry, his chance of survival is reduced. Another critical period occurs immediately after surfacing.

6. Reversed transfer of O_2, from the blood to the alveoli, may occur upon ascent especially from deep BH dives.

7. Elevated PO_2 can markedly extend BH time, and this may be a factor in AB at greater depths.

8. Certain individuals and groups, especially those with reduced sensitivity to CO_2, may be at unusual risk of BB and AB.

9. Maneuvers such as swallowing and inspiratory effort may suppress involuntary diaphragmatic contractions or otherwise encourage longer BH times. They may be responsible for some cases of BB or AB.

10. Reduction of lung volume by compression of alveolar gas at depth may tend to shorten BH time, but its influence is probably small compared to that of factors tending to increase the duration of BH.

11. Actions or circumstances that appear to invite BB or AB can be summarized:

a. Excessive hyperventilation

b. Unusual depth

c. Failure to heed air hunger

d. Maneuvers to forestall contractions or air hunger

e. Delays during ascent

f. Carelessness upon surfacing

Acknowledgement

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REFERENCES

1. Barlow HB, MacIntosh FC. Shallow water black-out. Royal Naval Physiological Laboratory Report R.N.P. 44/125 UPS 48(a), 1944.

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

3. Sciarli R. Un test simple de controle de l'hyperventilation pour le plongeur en apnee. Bull Med Sub Hyp 1970; 2:26.

4. Hill P McN. Hyperventilation, breath holding and alveolar oxygen tensions at the breaking point. Resp Physiol 1973; 19:201-209.

5. Olszowka AJ, Rahn H. Breath-hold diving. In: Sutton JR, Houston CS, Coates G, eds. Hypoxia and cold. Praeger, 1985.

6. Lanphier EH, Rahn H. Alveolar gas exchange during breath-hold diving. J Appl Physiol 1963; 18:471-477.

7. Lundgren CEG. Breath hold diving mammals. In: Hypoxia, Exercise, and Altitude: Proceedings of the third Banff international hypoxia symposium. New York: Alan R. Liss, 1983:365-381.

8. Lin YC, Lally DA, Moore TA, Hong SK. Physiological and conventional breath-hold breaking points. J Appl Physiol 1974; 37:291-296.

9. Albano, G. Principles and observations on the physiology of the scuba diver. ONR Report DR-150. Arlington: Office of Naval Research, 1970:301-323.

10. Stevens CD, Ferris EB, Webb JP, Engel GL, Logan M. Voluntary breathholding. I. Pulmonary gas exchange during breathholding. J Clin Invest 1946; 25:723-728.

11. Engel GL, Ferris EB, Webb JP, Stevens CD. Voluntary breathholding. II. The relation of the maximum time of breathholding to the oxygen tension of the inspired air. J Clin Invest 1946; 25:729-733.

12. Ferris EB, Engel GL, Stevens CD, Webb J. Voluntary breathholding. III. The relation of the maximum time of breathholding to the oxygen and carbon dioxide tensions of arterial blood, with a note on its clinical and physiological significance. J Clin Invest 1946; 25:734-743.

13. Davidson JT, Whipp BJ, Wasserman K, Koyal SN, Lugliani R. Role of the carotid bodies in breath-holding. N Eng J Med 1974; 290:819-822.

14. Stroud RC. Combined ventilatory and breath-holding evaluation of sensitivity to respiratory gases. J Appl Physiol 1959; 14:353-356.

15. Alvis HJ. Breathholding breakpoint at various increased pressures. Medical Research Laboratory, USN Submarine Base, New London, Report No. 177, Vol 10:110-120, 1951.

16. Agostoni E. Diaphragm activity during breath holding: factors related to its onset. J Appl Physiol 1963; 18:30-36.

17. Rigg JRA, Rebuck AS, Campbell EJM. A study of factors influencing relief of discomfort in breath-holding in normal subjects. Clin Sci Mol Med 1974; 47:193-199.

18. Mithoefer JC. Breath holding. In: Fenn WO, Rahn H, eds. Handbook of Physiology Section 3: Respiration Vol II. Washington: American Physiological Society, 1965:1011-1025.

19. Klocke FJ, Rahn H. Breath holding after breathing of oxygen. J Appl Physiol 1959; 14:689-693.

Discussion following Dr. Lanphier's presentation

Dr. Zannini: Is it better to exhale during the last few meters of the ascent?

Dr. Lanphier: I have heard that but I can't think of any reason why that would be desirable. I consider that dangerous.

Dr. Rahn: Aren't you throwing oxygen away if you expire?

Dr. Lanphier: Yes.

Dr. Lundgren: I think the idea of getting some relief from expiring is probably a correct one. Blowing out would be something that, because of sheer movement of the chest and lungs, might give some relief. You may recall the experimental background for this notion in Fowler's old work (Breaking Point of Breath-Holding. J. Appl. Physiol. 6:539-541, 1954.) where he showed that you could hold your breath longer if you rebreathed your own foul lung air into a rubber bag. So what it could provide, I presume, is a relief from the urge to breath which would otherwise make you drown. Whether that would help you in terms of gas exchange is very questionable because, as you say, you would throw away oxygen and reduce the space in which you could deliver CO_2. But by allowing you not to inhale water it would help. In the same vein, the relief that Dr. Lanphier referred to during ascent may perhaps come from the mere expansion of the chest and the lungs.

Dr. Craig: Dr. Lanphier, you mentioned excessive hyperventilation as an undesirable factor that would increase the chances of losing consciousness. I think we should point out that we think of a vigorous hyperventilation as being more dangerous than mild hyperventilation, but we should also consider that the time course of hyperventilation is very important. In fact, if you were hyperventilating moderately for a long period of time, you would be much worse off than with a short period of vigorous hyperventilation because of the time flow-limiting aspects of the CO_2 elimination.

Dr. Lanphier: The only thing is important here is how much you deplete the CO_2 reservoir, and I think it is more effectively done by long periods of mild hyperventilation. I think we are going to hear later that if you try to overdo it, you may increase your CO_2 production enough to offset elimination.

CNS TOLERANCE TO ASPHYXIA

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Although asphyxia leads to both a reduction in arterial oxygen tension and to carbon dioxide retention it is the hypoxic component which usually causes CNS dysfunction. In fact, some results demonstrate that at certain degrees of hypoxia, hypercapnia improves the function of the brain. The objective of this article is to provide an overview of the tolerance of the brain to asphyxia. I will begin by discussing functional effects of arterial hypoxia and proceed by considering the modulating influence of hypercapnia. Following this, I will enquire into the cellular and molecular mechanisms by which hypoxia disrupts normal integrated brain functions. Finally, I shall discuss mechanisms of brain damage as this evolves after severe hypoxia/ischemia. More detailed accounts of these subjects can be found in recent review articles (Siesjö 1978, 1981, Siesjö and Wieloch 1985a, Siesjö and Ingvar 1985).

1. Brain dysfunction in hypoxia

Extensive information exists on signs and symptoms of cerebral oxygen deficiency, whether due to mild, moderate, or severe arterial hypoxia. This is because the subject has been of considerable interest to research workers in the fields of high altitude physiology and in aviation medicine (see e.g. Lutt 1965). Fig. 1 lists some signs and symptoms of arterial hypoxia, as these have been observed at different simulated altitudes, and gives the equivalent inspired oxygen concentrations. The figure shows that certain visual functions, notably the acuity of the dark adapted eye, fail at very mild degrees of hypoxia, while it takes slightly more severe hypoxia to affect short term memory, or the accomplishment of complex tasks. Serious symptoms, such as loss of critical judgement, usually occur first when PaO_2 falls below about 40 mmHg, and consciousness is lost first when PaO_2 falls to 30 mmHg, or lower.

Observations in man during exposure to hypoxia revealed that even moderate hypoxia elicits a compensatory increase in ventilation with a reduction in $PaCO_2$ (see Fig. 1). If occurring in isolation, such ventilatory hypocapnia would be expected to reduce cerebral blood flow (CBF). However, early measurements in man demonstrated an increase in CBF (Kety et al. 1948, Cohen et al. 1967). Obviously, the reduced oxygen delivery must cause cerebral vasodilation. The possibility remains, though, that the hypocapnia accompanying mild to moderate degrees of hypoxia curtails the rise in CBF for a given reduction in PaO_2 (see below).

Although animal experiments cannot give comparable information on the functional effects of mild to moderate hypoxia, they allow precise measurements of physiological variables. In this context, it is of interest that when PaO_2 is reduced in steps from normal values (about 90 mmHg) CBF is gradually increased until, at a PaO_2 of about 50 mmHg, the rate of increase is suddenly enhanced (McDowall 1966, Borgström et al. 1975, Jóhannsson and Siesjö 1975). We conclude from this that cerebral resistance

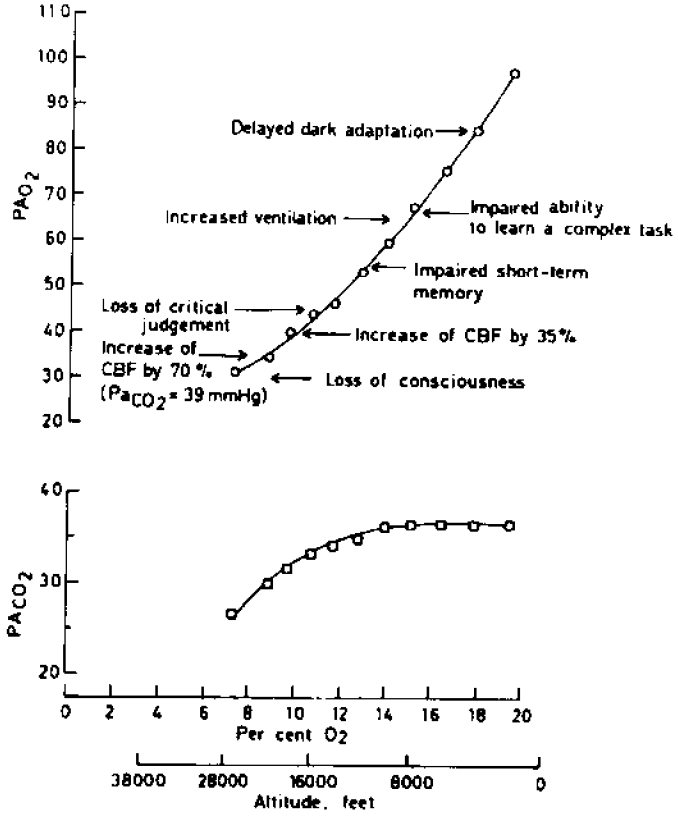

Fig. 1

Relationship between inspired oxygen concentration, altitude, and physiological responses of man to hypoxia. The left hand panels give alveolar PO_2 and PCO_2, and the upper curve signs and symptoms of hypoxia at decreasing PaO_2 values. Reproduced with permission from Siesjö et al. (1974) Brain Dysfunction in Metabolic Disorders (Ed. F. Plum). Res. Publ. Assoc. Nerv. Ment. Dis. Vol. 53, pp. 75-112, Raven Press, New York.

vessels are moderately sensitive to changes in PO_2 around the normal, and that the hypoxic threshold at PaO_2 values below 50 mmHg must reflect a more dramatic perturbation of cerebral metabolism.

2. Modulating influence of hypercapnia

Hypercapnia has by itself profound influences on brain functions (see Woodbury and Karler 1960, Wyke 1963). Like hypoxia, it stimulates lung ventilation and increases cerebral blood flow. However, none of these alterations is detrimental in the sense that the integrity and viability of brain neurons are endangered. Thus, ventilated animals may be exposed to CO_2 concentrations of 50 % without showing signs of energy failure at tissue level (Folbergrová et al. 1975, see also Siesjö 1978).

It was early recognized that signs and symptoms of mild to moderate hypoxia could be ameliorated or prevented if CO_2 was added to the hypoxic gas mixture (Gibbs et al. 1943, Otis et al. 1946). Two possible explanations emerge: (a) the low PCO_2 accompanying hypoxia affects the function of cerebral neurons, and (b) hypocapnia curtails the compensatory rise in CBF. Evidence has been presented that an increase in PCO_2 enhances CBF during hypoxia (Kety and Schmidt 1948, Cohen et al. 1967). It should also be recalled that when the PaO_2 of experimental animals is lowered to

below 30 mmHg an increase in PCO_2 to hypercapnic values (about 65 mmHg) does not affect cerebral energy state, and it significantly curtails the rise in tissue lactate content (MacMillan and Siesjö 1972a, see also Gottesfeld and Miller 1969). We conclude that when asphyxia threatens the proper functioning of the brain, or the viability of its cells, it is the hypoxic component which is responsible.

3. Neuronal dysfunction in asphyxia: cellular and molecular mechanisms

At first sight, it would seem likely that hypoxia (or asphyxia) disrupts brain function by curtailing cerebral energy production. However, there is no evidence that signs and symptoms of hypoxia are due to ATP failure, unless the hypoxia is severe enough to cause loss of consciousness, i.e. coma. This can be illustrated by measurements of cerebral energy state at various degrees of arterial hypoxia (Fig. 2). Rats exposed to hypoxia with PaO_2 values of below 50 mmHg show behavioural and EEG changes. However, as Fig. 2 demonstrates tissue concentrations of ATP,

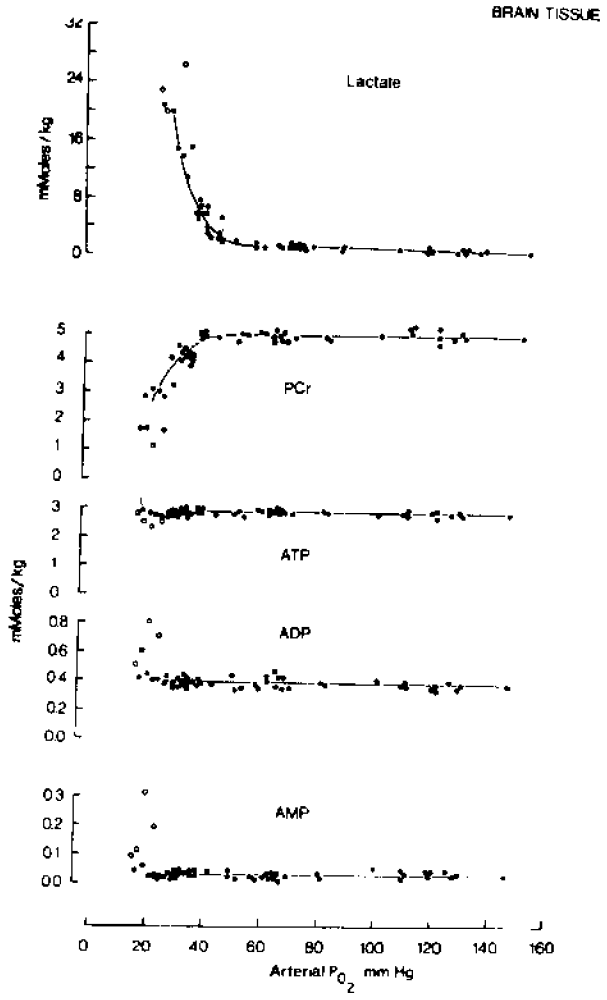

Fig. 2

Brain contents of lactate, phosphocreatine, ATP, ADP, and AMP in rats maintained for 15-30 min at different arterial PO_2 values before the tissue was frozen in situ. Unfilled circles denote animals in which blood pressure fell below 120 mmHg (to 80-105 mmHg). Data from Siesjö and Nilsson (1971). Reproduced with permission from Siesjö et al. (1974). (See Legend to Fig. 1).

ADP, and AMP remain unaltered even if the PaO_2 is reduced to 20 mmHg; at that PaO_2, though, even a relatively moderate reduction in blood pressure precipitates cellular energy failure. The results obtained in these experiments were subsequently confirmed by workers using even more sensitive analytical techniques, and analysing small brain areas (Duffy et al. 1972, MacMillan and Siesjö 1972b, Norberg and Siesjö 1975a).

Although moderate hypoxia does not measurably alter tissue concentrations of labile phosphates it perturbs carbohydrate metabolism. Thus, at PaO_2 values of below 50 mmHg tissue lactate content rises, with an associated increase in the lactate/pyruvate ratio. Enhanced glycolysis with increased production of lactic acid reduces intra- and extracellular pH (MacMillan and Siesjö 1972c). Since the creatine kinase reaction is pH-sensitive, the PCr concentration falls even though the ATP/ADP ratio is not altered; in fact, the reaction was used to calculate changes in pH (MacMillan and Siesjö 1972c). Also the lactate dehydrogenase reaction is pH sensitive; however, reduced pH could not explain the whole change in the lactate/pyruvate ratio. Accordingly, a redox change must be present in hypoxia, with a rise in the $NADH/NAD^+$ ratio. This redox change, and the increased pyruvate concentration, can be made responsible for the alterations observed in levels of citric acid cycle intermediates, and of associated amino acids (Duffy et al. 1972, Norberg and Siesjö 1975b).

Obviously, other mechanisms than energy failure must be responsible for neuronal dysfunction in mild to moderate hypoxia. The problem of defining these mechanisms is similar to that confronting workers enquiring into the cellular mechanisms of altered brain functions caused by anesthetic and sedative drugs. Two types of mechanisms have been discussed: (a) failure of synaptic transmission by alteration of transmitter synthesis or release, and (b) hyperpolarisation of postsynaptic membranes, with reduced synaptic transmission.

a. Hypoxia and transmitter synthesis/release. It has been known for many years that the synthesis of catecholamines and indole amines from the precursor amino acids (tyrosine and tryptophan) requires the participation of molecular oxygen, also that the appropriate K_m values are in the physiological range of PO_2 values. Davis and Carlsson (1973, see also Davis et al. 1973) observed that even relatively moderate hypoxia reduced the rate of synthesis of the amines, and postulated that a small transmitter pool with a fast turnover was important to function in monoaminergic circuits. Later results suggested that the synthesis of serotonin was strictly O_2-dependent, while the K_m for oxygen in the tyrosine hydroxylase reaction seemed to change in stressful situations (Davis 1976). Nonetheless, the data obtained suggested that part of the symptomatology of hypoxia was due to failure of function in monoaminergic neurons. For example, the symptoms were ameliorated by administration of L-DOPA (Brown et al. 1974).

Results also exist demonstrating failure of synthesis/release of acetylcholine in hypoxia (see Gibson et al. 1981, Gibson and Blass 1983). Acetylcholine is supposed to be synthesized from a small pool of pyruvate via reactions which are sensitive to lack of oxygen or glucose. If newly

synthesized acetylcholine is what is preferentially released one could argue that hypoxia leads to failure of cholinergic transmission. In support, results demonstrate that mild-to-moderate hypoxia reduces the rate of acetylcholine synthesis (Gibson and Duffy 1981). However, even more recent results hint that the fall in the rate of synthesis could be the result rather than the cause of the reduced synaptic activity (Peterson and Gibson 1982). These new results suggest that mild hypoxia retards Ca^{2+} influx into pre-synaptic terminals, thereby reducing release of transmitter and, secondarily, its synthesis. We recognize that such an effect, if existing, could possibly explain failure of transmission at other synapses as well.

b. Hypoxia and neuronal hyperpolarisation. Hypoxia, hypercapnia and metabolic inhibitors such as 2,4-dinitrophenol cause hyperpolarisation of neurons and decrease the resistance of their membranes, probably by an increase in K^+ conductance (Speckmann and Caspers 1974, Krnjevic 1975, Krnjevic et al. 1978, Hansen et al. 1982). As discussed by Hansen (1985) these events would be expected to block synaptic transmission by clamping the postsynaptic membranes at hyperpolarised values, thereby rendering the membranes less prone to depolarisation.

It this were the mechanism of hypoxic neuronal dysfunction, what causes the hyperpolarising efflux of K^+ along conductance channels? K^+ channels are usually considered to be voltage-dependent or sensitive to chemical perturbation, notably to a raised Ca^{2+} concentration. Thus, if Ca^{2+} enters the cell or is released from intracellular binding or sequestration sites, K^+ conductance should increase. There are no results demonstrating that moderate hypoxia causes Ca^{2+} influx into neurons since such influx is a late phenomenon, occurring at very low PO_2 or blood flow values (Hansen and Zeuthen 1981, Harris et al. 1981). In theory, though, Ca^{2+} could be released from intracellular binding/sequestration sites, either by a lowering of pH_i (e.g. Meech and Thomas 1980) or by agonist-receptor interactions at the membrane surface, activating phospholipase C (Berridge 1984).

Whether or not Ca^{2+} is a second messenger it is conceivable that an increased K^+ conductance during ischemia, or indeed any permeability change contributing to the functional "block", occurs in response to phosphorylation/dephosphorylation of membrane proteins by the appropriate kinases and phosphatases (Neary and Alkon 1983, Nestler and Greengaard 1983, see also Hansen 1985). This is a speculative deduction, though, and we neither know the first nor the second messenger in this hypothetical chain of events.

4. Ischemia

If the arterial hypoxia is sufficiently severe cardiovascular function usually suffers, with a fall in cerebral perfusion pressure. Since hypoxia abolishes cerebrovascular autoregulation, hypotension is synonymous with a reduction in CBF. As long as CBF does not fall below normal (remember that it is increased during hypoxia) the condition is one of hypoxia with relative ischemia (or restricted hyperemia). As we have seen, cerebral

energy state may then deteriorate (see Fig. 2 above). Such energy failure must endanger the viability of brain cells, especially neurons. For these reasons, a reduction of blood pressure during severe arterial hypoxia can have devastating consequences.

Under normoxic circumstances, CBF can be reduced to about 18 ml·100 g^{-1}·min^{-1} before spontaneous or evoked electrical activity fail, and to about 12 ml·100 g^{-1}·min^{-1} before membrane failure ensues with massive release of K^+ from cells, and uptake of Ca^{2+} (Branston et al. 1977, Astrup et al. 1981, Harris et al. 1981). This depolarisation, and the influx/release of Ca^{2+}, cause an avalance series of metabolic reactions with enhanced lipolysis, proteolysis, disassembly of microtubuli and protein phosphorylations, reactions which may be responsible for the final cell damage, once the ischemia has surpassed the revival time, i.e. the shortest ischemic period compatible with full restitution (see Siesjö 1981, Siesjö and Wieloch 1985a).

Clearly, if arterial oxygen tensions are low the ischemia need not be that dense to cause membrane failure and cell injury/death. It seems warranted to discuss two important issues: ischemic revival times, and selective neuronal vulnerability.

a. Revival times. It is common clinical experience that cardiac arrest lasting longer than 4-5 min invariably leads to irreversible brain damage. There may be several reasons for these very short revival times. Most importanly, patients with cardiac arrest probably have poor tissue perfusion in the immediate recirculation period, with prolongation of the ischemia into the period of resuscitation. Experimentally, one can achieve square wave ischemia and, by pressor agents, secure prompt recirculation of the ischemic tissue. Under such circumstances, some complex physiological and metabolic functions can return, at least for many hours, following ischemic periods of as long as 60 min (Hossmann 1982, 1985). Long-term recovery of neurological function, with little or no histopathologic damage, has been achieved after ischemic periods of 16-20 min (for literature, see Siesjö 1978, Hossmann 1982).

b. Selective vulnerability. While it may be pssible to identify a revival time for the function of the brain as a whole, or in terms of neuropathologically defined brain damage, the term becomes ambiguous when one considers recovery of individual brain neurons, or groups of neurons. This is because a selective vulnerability exists, with pronounced differences in susceptibility to incur damage following hypoxia/ischemia (e.g. Brierley 1976). Recent results have provided some challenging concepts. First, neuronal necrosis following short periods of ischemia can "mature" over hours and days, and may appear after a delay during which function is transiently resumed (Kirino 1982, Pulsinelli et al. 1982, Pulsinelli and Duffy 1983, Suzuki et al. 1983). Second, under seemingly optimal experimental conditions unequivocal brain damage has been observed after such short ischemic periods as 5 min (Kirino 1982, Smith et al. 1984). Typically, such damage then affects pyramidal cells in the hippocampus, notably in the subiculum, CA1, and CA4 sectors, and neurons in some other selectively vulnerable areas (Kirino 1982, Pulsinelli et al.

1982, Smith et al. 1984). On scrutiny, one observes that neurons injured by short periods of ischemia belong to the limbic system (Wieloch 1985). This fact, and the slow maturation, suggest that limbic seizures may contribute to the final brain damage. With slightly longer ischemic periods, other regions are involved, such as the neocortex and the caudoputamen. However, also in these regions ischemia affects certain selectively vulnerable cells (see Pulsinelli et al. 1982, Smith et al. 1984).

5. Mechanisms of brain damage

I will confine the discussion to selective neuronal necrosis and abstain from discussing infarction, a pan-necrosis accompanying more prolonged ischemia, especially under conditions of hyperglycemia (see Siesjö 1981, Siesjö and Wieloch 1985, for further literature).

Pyramidal cells in the neocortex and hippocampus belong to the selectively vulnerable cells in the brain. Fig. 3 depicts probable synaptic

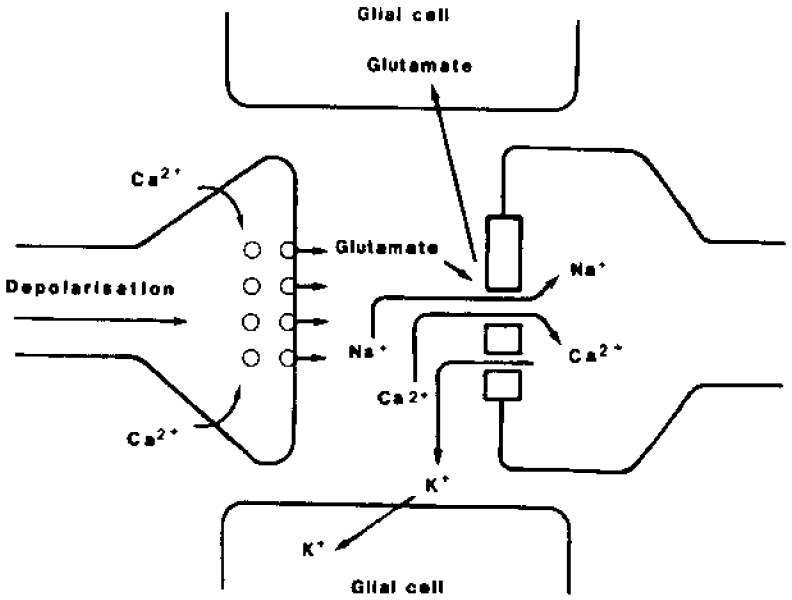

Fig. 3. Schematic diagram illustrating Ca^{2+}-dependent transmitter release at presynaptic terminals, and agonist-dependent influx of Na^+ and Ca^{2+} at postsynaptic sites. The excitatory transmitter (glutamate) is assumed to activate Na^+ channels, leading to depolarisation and Ca^{2+} influx. The latter is shown to enhance K^+ permeability, causing an outward K^+ current. The diagram also indicates that part of the uptake of transmitter and of K^+ occurs into glial cells. Modified after Collins et al. (1983). Reproduced with permission from Siesjö and Wieloch. Cerebrovascular Diseases (Ed. F. Plum and W. Pulsinelli). Raven Press, New York, 1985.

events leading to excitation of such cells (Collins et al. 1983). An excitatory transmitter (glutamate or aspartate) is believed to open Na^+ channels, the influx of Na^+ then depolarising the postsynaptic membrane, this in turn causing Ca^{2+} influx. Apart from contributing to depolarisation, the influx of Ca^{2+} can cause lipolysis, proteolysis, and protein phosphorylation, i.e. enhance calcium-activated or calcium-dependent reactions.

The events depicted in Fig. 3 illustrate features of two current hypothesis on mechanisms of neuronal necrosis. Most importantly, the figure focusses attention on calcium and excitatory amino acids. It had previously been assumed that uncontrolled calcium influx, and/or its release from intracellular binding or sequestration sites, contributed to cell necrosis in other tissues than the brain. The application of the calcium hypothesis to the brain was inspired by the observation that selectively vulnerable neurons appeared identical to those identified as having high dendritic calcium conductances, making these cells prone to epileptogenic firing (see Siesjö 1981, Siesjö and Wieloch 1985 a and b).

It now seems that such a simple calcium hypothesis ("vulnerable cells have high calcium conductances") does not suffice to explain selective neuronal vulnerability. Previous work on the neurotoxicity of excitatory amino acids led to the hypothesis of excitotoxic neuronal damage, and its association with seizure states (Olney 1978, Collins et al. 1983). This hypothesis has now been extended to ischemia (Jørgensen and Diemer 1982, Simon et al. 1984, Wieloch 1985). The hypothesis receives strong support from observations that vulnerable neurons are innervated by glutamatergic (or aspartergic) afferents, and that lesions of such afferents seem to ameliorate ischemic damage (Wieloch 1985).

According to this hypothesis, neurons innervated by excitatory amino acids are vulnerable because ischemia leads to massive release of the amino acids from presynaptic endings (see Benveniste et al. 1984), and because agonist-receptor interaction then leads to uncontrolled influx of Na^+ and/or Ca^{2+} into postsynaptic elements. This is, essentially, unbalanced excitation. However, it has been recognized that the outcome of the ischemic insult must depend on inhibitory tonus as well. For example, lesions of the noradrenergic system originating in the locus coeruleus, a system conveying inhibition, has been found to aggravate the ischemic neuronal damage (Blomqvist et al. 1985). Clearly, agonist-receptor interaction must play an important role in the development of ischemic neuronal necrosis.

6. Summary and conclusions

The results discussed make it clear that CNS tolerance to asphyxia must be discussed at two levels. The first concerns changes in brain function at mild to moderate degrees of hypoxia (or asphyxia). Since such changes can cause disturbing and threatening failure of intellectual and emotional functions (e.g. loss of visual acuity, failure to accomplish complex tasks, loss of critical judgement) it is justified to explore the mechanisms involved. Energy failure at the cellular level does not seem

responsible. Possibly, reduced rates of synthesis of monoamines and of acetylcholine could contribute. However, it seems even more likely that the metabolic perturbation leads to the appearance of a second messenger, this in turn influencing membrane permeability by phosphorylation of ionic channels. Such a sequence of events could underlie an observed hyperpolarisation of neurons, with reduced resistance due to enhanced K^+ permeability, events that could induce blockade of synaptic transmission.

The second level of hypoxia is one sufficiently severe to endanger the viability of brain cells. Since these cells are quite resistant to pure arterial hypoxia overt cell damage requires that hypoxia is complicated by hypotension, or circulatory collaps. Under such conditions (hypoxia-ischemia) damage is incurred by certain selectively vulnerable cells, their vulnerability residing in special agonist-receptor interactions. Specifically, vulnerable cells seem innervated by excitatory amino acids, whose interaction with post-synaptic sites causes influx of Na^+ and Ca^{2+}. Probably, the toxicity of these amino acids is due to such influx, and to enhanced firing of neurons innervated by them.

REFERENCES

Astrup J, Siesjö B K, and Symon L (1981) Thresholds in cerebral ischemia - the ischemic penumbra. Stroke 12:723-725.

Benveniste H, Drejer J, Schousboe A, and Diemer N H (1984) Elevation of the extracellular concentrations of glutamate and aspartate in rat hippocampus during transient cerebral ischemia monitored by intracerebral microdialysis. J Neurochem 43:1369-1374.

Berridge M J (1984) Inositol trisphosphate and diacylglycerol as second messengers. Biochem J 220:345-360.

Blomqvist P, Lindvall O, and Wieloch T (1985) Lesion of the noradrenergic locus coeruleus system aggravate neuronal necrosis after transient cerebral ischemia. Neurosci Lett. In press.

Borgström L, Jöhannsson H, and Siesjö B K (1975) The relationship between arterial PO_2 and cerebral blood flow in hypoxic hypoxia. Acta Physiol Scand 93:423-432.

Branston N M, Strong A J, and Symon L (1977) Extracellular potassium activity, evoked potential and tissue blood flow. Relationships during progressive ischaemia in baboon cerebral cortex. J Neurol Sci 32:305-321.

Brierley J B (1977) Cerebral hypoxia. In: Greenfield's Neuropathology, ed. by W. Blackwood and J.A.N. Corsellis. London: Arnold, p. 43-85.

Brown R M, Davis J N, and Carlsson A (1974) Dopa reversal of hypoxia-induced disruption of the conditioned avoidance response. J Pharm Pharmacol 25:412-414.

Cohen P J, Alexander S C, Smith T C, Reivich M, and Wollman H (1967) Effects of hypoxia and normocarbia on cerebral blood flow and metabolism in conscious man. J Appl Physiol 23:183-189.

Collins R C, Lothman E W, and Olney J W (1983) Status epilepticus in the limbic system: Biochemical and pathological changes. In: Advances in Neurology. Vol. 34: Status Epilepticus (Delgado-Escueta A V, Wasterlain C G, Treiman D M, Porter R J, eds.). Raven Press, New York, p. 277-288.

Davis J N, and Carlsson A (1973) The effect of hypoxia on monoamine synthesis, levels and metabolism in rat brain. J Neurochem 21:783-790.

Davis J N, Carlsson A, MacMillan V, and Siesjö B K (1973) Brain tryptothan hydroxylation: dependence on arterial oxygen tension. Science 182:72-74.

Davis J N (1976) Brain tyrosine hydroxylation: alteration of oxygen affinity in vivo by immobilization or electroshock in the rat. J Neurochem 27:211.215.

Duffy T E, Nelson S R, and Lowry O H (1972) Cerebral carbonydrate metabolism during acute hypoxia and recovery. J Neurochem 19:925-934.

Folbergrová J, Norberg K, Quistorff B, and Siesjö B K (1975) Carbonydrate and amino acid metabolism in rat cerebral cortex in moderate and extreme hypercapnia. J Neurochem 25:457-462.

Gibbs F A, Gibbs E L, and Lennox W G (1943) The value of carbon dioxide in counteracting the effects of low oxygen. J Aviat Med 14:250-261.

Gibson G E, Pulsinelli W,Blass J P, and Duffy T E (1981) Brain dysfunction in mild to moderate hypoxia. Am J Med 70:1247-1254.

Gibson G E, and Blass, J P (1983) Metabolism and neurotransmission. In: Handbook of Neurochemistry, edited by A. Lajtha, New York: Plenum, vol. 3, p. 633-651.

Gibson G E, and Duffy T E (1981) Impaired synthesis of acetylcholine by mild hypoxic hypoxia or nitrous oxide. J Neurochem 36:28-33.

Gottesfeld Z, and Miller A T (1969) Metabolic response of rat brain to acute hypoxia: influence of polycythemia and hypercapnia. Amer J Physiol 216:1374-1379.

Hansen A J, and Zeuthen T (1981) Extracellular ion concentrations during spreading depression and ischemia in the rat brain cortex. Acta Physiol Scand 113:437-445.

Hansen A J, Hounsgaard J, and Jahnsen H (1982) Anoxia increases potassium conductance in hippocampal nerve cells. Acta Physiol Scand 115:301-310.

Hansen A J (1985) Effect of anoxia on ion distribution in the brain. Physiol Rev 65(1):101-148.

Harris R J, Symon L, Branston N M, and Bayhan M (1981) Changes in extracellular calcium activity in cerebral ischemia. J Cereb Blood Flow Metabol 1:203-209.

Hossmann K.-A. (1982) Treatment of experimental cerebral ischemia. J Cereb Blood Flow Metabol. 2:275-297.

Hossmann K-A (1985) Post-ischemic resuscitation of the brain: selective vulnerability versus global resistance. In: Progress in Brain Research, Vol. 63, edited by K. Kogure, K.-A. Hossmann, B.K. Siesjö and F.A. Welsh. Elsevier Science Publishers B.V., Biomedical Division, Amsterdam, pp. 1-15.

Jóhannsson H, and Siesjö B K (1975) Cerebral blood flow and oxygen consumption in the rat in hypoxic hypoxia. Acta Physiol Scand 93:269-276.

Jørgensen M B, and Diemer N H (1982) Selective neuron loss after cerebral ischemia in the rat: Possible role of transmitter glutamate. Acta Neurol Scand 66:536-546.

Kety S S, and Schmidt C F (1948) The effects of altered arterial tensions of carbon dioxide and oxygen on cerebral blood flow and cerebral oxygen consumption of normal young men. J Clin Invest 27:484-491.

Kirino T (1982) Delayed neuronal death in the gerbil hippocampus following ischemia. Brain Res 239:57-69.

Krnjević K (1975) Coupling of neuronal metabolism and electrical activity. In: Brain Work, edited by D.H. Ingvar and N.A. Lassen. Copenhagen: Munksgaard, 65-78. (Alfred Benzon Symp.)

Krnjević K, Puil E, and Werman, R (1978) Significance of 2,4-dinitrophenol action on spinal motoneurones. J Physiol London 275:225-239.

Luft U C (1965) Aviation physiology - the effects of altitude. In: Handbook of Physiology. Respiration, edited by W.O. Fenn and H. Rahn. Washington, DC:Am Physiol Soc, sect 3, vol. II chapt. 44, p. 1099-1145.

McDowall D G (1966) Interrelationships between blood oxygen tensions and cerebral blood flow. In: Oxygen Measurements in Blood and Tissues, edited by J.P. Payne and D.W. Hill. London: Churchill. p. 205-219.

MacMillan V, and Siesjö B K (1972a) The effect of hypercapnia upon the energy metabolism of the brain during arterial hypoxemia. Scand J Clin Lab Invest 30:237-244.

MacMillan V, and Siesjö B K (1972b) Brain energy metabolism in hypoxemia. Scand J Clin Lab Invest 30:126-136.

MacMillan V, and Siesjö B K (1972c) Intracellular pH of the brain in arterial hypoxemia, evaluated with the CO_2 method and from the creatine phosphokinase equilibrium. Scand J Clin Lab Invest 30:117-125.

Meech R W, and Thomas R C (1980) Effect of measured calcium chloride injections on the membrane potential and internal pH of snail neurones. J Physiol London 298:111-129.

Neary J T, and Alkon D L (1983) Protein phosphorylation/dephosphorylation and the transient voltage-dependent potassium conductance in Hermissenda crassicornis. J Biol Chem 258:8979-8983.

Nestler E J, and Greengaard P (1983) Protein phosphorylation in the brain. Nature London 305:583-588.

Norberg K, and Siesjö B K (1975a) Cerebral metabolism in hypoxic hypoxia. I. Pattern of activation of glycolysis, a reevaluation. Brain Res 86:31-44.

Norberg K, and Siesjö B K (1975b) Cerebral metabolism in hypoxic hypoxia. II. Citric acid cycle intermediates and associated amino acids. Brain Res 86:45-54.

Olney J (1978) Neurotoxicity of excitatory amino acids. In: Kainic as a Tool in Neurobiology (E G McGeer, J W Olney, P L McGeer, eds.) Raven Press, New York. pp. 95-121.

Otis A B, Rahn H, Epstein M A, and Fenn W O (1946) Performance as related to composition of alveolar air. Am J Physiol 146:207-221.

Peterson C, and Gibson G E (1982) 3,4-diaminopyridine alters acetylcholine metabolism and behaviour during hypoxia. J Pharmacol Exp Ther 222:576-582.

Pulsinelli W A, Brierley J B, and Plum F (1982) Temporal profile of neuronal damage in a model of transient forebrain ischemia. Ann Neurol 11:491-498.

Pulsinelli W A, and Duffy T E (1983) Regional energy balance in rat brain after transient forebrain ischemia. J Neurochem 40:1500-1503.

Schmahl F W, Betz E, Dettinger E, and Hohorst H J (1966) Energiestoffwechsel der Grosshirnrinde und Elektroencephalogramm bei Sauerstoffmangel. Pflügers Arch ges Physiol 292:46-59.

Siesjö B K (1978) Brain Energy Metabolism. John Wiley & Sons, Ltd. The Pitman Press, Bath.

Siesjö B K (1981) Cell damage in the brain: A speculative synthesis. J Cereb Blood Flow Metabol 1:155-185.

Siesjö B K, and Nilsson L (1971) The influence of arterial hypoxemia upon labile phosphates and upon extracellular and intracellular lactate and pyruvate concentrations in the rat brain. Scand J Clin Lab Invest 27:83-96.

Siesjö B K, Jóhannsson H, Ljunggren B, and Norberg K (1974) Brain dysfunction in cerebral hypoxia and ischemia. Res Publ Assoc Res Nerv Ment Dis, 75-112.

Siesjö B K, and Ingvar M (1985) Ventilation and brain metabolism. In: Handbook of Physiology, to be published by American Physiological Society. In press.

Siesjö B K, and Wieloch T (1985 a) Brain injury: Neurochemical aspects. In: Central Nervous System Trauma - Status Report (Becker D, Povlishock J, eds.) William Byrd Press Inc., Richmond, VA. pp. 513-532.

Siesjö B K, and Wieloch T (1985 b) Molecular mechanisms of ischemic brain damage: Ca^{2+}-related events. In: Cerebrovascular Dieseases, edited by F. Plum and W. Pulsinelli. Raven Press, New York. pp. 187-200.

Simon R P, Swan J H, Griffith T, and Meldrum B S (1984) Blockade of N-methyl-D-aspartate receptors may protect against ischemic damage in the brain. Science 226:850-852.

Smith M-L, Auer R N, and Siesjö B K (1984) The density and distribution of ischemic brain injury in the rat following two to ten minutes of forebrain ischemia. Acta Neuropathol (Berl). 64:319-332.

Speckmann E-J, and Caspers H (1974) The effect of O_2- and CO_2-tensions in the nervous tissue on neuronal activity and DC potentials. In: Handbook of Electroencephalography and Clinical Neurophysiology, edited by A. Remond. Amsterdam: Elsevier, vol. 2, pt. C, p. 71-89.

Suzuki R, Yamaguchi T, Choh-Luh L, and Klatzo I (1983) The effects of 5-minute ischemia in mongolian gerbils: II. Changes of spontaneous neuronal activity in cerebral cortex and CA 1 sector of hippocampus. Acta Neuropathol (Berl) 60:217-222.

Wieloch T (1985) Neurochemical correlates to selective neuronal vulnerability. Progress in Brain Res. vol. 63. In press.

Woodbury D M, and Karler R (1960) The role of carbon dioxide in the nervous system. Anesthesiology 21:686-703.

Wyke B (1963) Brain Function and Metabolic Disorders, Butterworths, London.

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Discussion following Dr. Siesjo's presentation

Dr. Arnold: Have you considered the use of calcium channel blockers after clinical transient ischemic attacks or cardiac arrests and revival?

Dr. Siesjo: Yes, there are at least three experimental studies which have shown that you can ameliorate the damage if you give the calcium entry blockers after transient ischemia. They have also been used in middle cerebral artery occlusion and seem to have effect on the associated damage. Clinical trials are now being initiated, one of them in Scandinavia. They are double blind control studies and their results are not ready yet.

Dr. Hong: What is the mechanism for the entry of calcium across the post-synaptic membrane?

Dr. Siesjo: It brings in calcium by opening voltage-dependent calcium channels.

Dr. Hong: What about the sodium-calcium exchange mechanism?

Dr. Siesjo: The primary effect of the excitatory transmitter is probably to open conductance channels for Na+. The depolarization associated with sodium influx is supposed to open voltage-dependent calcium channels.

Dr. Rahn: Voltage dependent?

Dr. Siesjo: Yes. And both ions carry a current across the membrane. A depolarizing current.

Dr. Hong: Is it possible that hypoxia-induced increase in cell sodium could be responsible for the increase in cell calcium via inhibition of sodium-calcium exchange mechanisms?

Dr. Siesjo: Most people would assume that in the majority of cells you have both a sodium-calcium exchange, which is not ATP-dependent, and an ATPase which would pump calcium out of the cell.

Dr. Hong: This is another mechanism. Sodium-calcium exchange itself is not related to calcium ATPase.

Dr. Siesjo: No, but there are probably two mechanisms for calcium extrusion, sodium-calcium exchange being one.

Dr. Hong: You are not eliminating the possibility of the involvement of sodium calcium exchange.

Dr. Siesjo: Oh no, the reason why I am talking about the calcium influx along conductance channels is that neurophysiologists consider many pyramidal cells to fire repetitively by calcium-dependent mechanisms, that is, the calcium influx contributes to excitation by depolarizing cells.

Dr. Arnold: I was very interested in your mention of the limbic system being specifically sensitive to hypoxic damage. With respect to underwater blackout, there are two main possibilities: cardiogenic or just a purely chronic blood gas change. You didn't mention anything about watershed areas of the brain being specifically sensitive, which would be due to a cardiogenic as opposed to long term gas exchange mechanism.

Dr. Siesjo: I didn't because time didn't allow me to do that, but if you have a situation with a reduced perfusion pressure and when you reach the critical levels of perfusion pressure, which would be around 30 mm Hg, the first areas to suffer would be the watershed areas. That's quite clear, but I was discussing circumstances where you have another type of selective neuronal vulnerability, i.e., those in which the decrease in perfusion pressure is to lower values. But clearly in an asphyxtic situation where there would be cardiac involvement, you could very well have lesions which reflect an inhomogeneous perfusion of the ischemic tissue.

Dr. Rahn: Dr. Lundgren and I have been having discussions before your arrival about warning signs before becoming unconscious under water. Dr. Craig, a long time ago, pointed out that we do not have warning signs when we are under water, but we do have warning signs when our cardiac output is reduced to a point where our brain no longer gets sufficient blood flow and, in other words, during hypotension. What is the difference between a blackout under water with presumably normal blood flow to the brain but low oxygen, versus hypotension wherein the oxygen tension falls in your brain due to lower blood pressure?

Dr. Siesjo: I really don't know but I wonder if I could respond with a question. Is anything known about the response to the two situations in terms of plasma adrenaline concentrations?

Dr. Rahn: I don't know.

Dr. Craig: One of the things we found when we reviewed a number of cases of loss of consciousness associated with underwater swimming and diving was that in many situations the subject continues doing what he has been doing, continues swimming normally and is unconscious. I've never known whether this is a retrograde amnesia secondary to hypoxia, or there is anything in biochemistry of brain function which suggests different levels of consciousness.

Dr. Siesjo: I don't know of any neurochemical sensor - or response - which would tell you why you have this difference.

Dr. Craig: There is also the classical physiology experiment when you write a series of numbers and you are asked to remember the last one and, as a result of hypoxia, the remembered number always precedes the last one on the list.

Dr. McDonough: Some observations in clinical medicine, especially cardiology, might lend some dimension to the question of how much time one has before losing consciousness when the cardiocirculatory system

undergoes a sudden change.

Dr. Rahn: In terms of flow or are we looking at oxygen delivery?

Dr. McDonough: No, in terms of flow.

Dr. Rahn: There may be a difference.

Dr. McDonough: Yes. For instance, in a situation of sudden ventricular fibrillation, when flow becomes almost zero suddenly, and when posture is supine (having observed this several times in patients in the coronary care unit), there is a gradual loss of consciousness over a period of perhaps 6 or 8 seconds and a patient is aware that there is a pending loss of consciousness. In the erect individual who undergoes ventricular fibrillation and is being monitored, there may be a duration of 3-6 seconds from onset of ventricular fibrillation until sudden collapse and fall. My experience with one such subject being ECG-monitored in a gymnasium exercise program and suddenly collapsing and successfully resuscitated, was that this individual had absolutely no warning of the impending loss of consciousness. So that posture may be an important factor here.

Dr. Lin: In a tilt table experiment, the subject can always detect an impending fainting, some kind of warning that tells him it is coming, presumably due to the decreased cardiac output. People who are standing erect and motionless in hot temperature also know the impending fainting, presumably due to decreased blood flow to the brain.

Dr. Rahn: So I suppose that there is a difference in terms of blood flow decrease which leads to low oxygen and normal blood flow to the brain with reduced oxygen.

Dr. Ferrigno: I know that Mr. Maiorca has suffered a total of seven episodes of syncope, during horizontal underwater swimming and during deep breath-hold dives. So I would like to ask him if he experienced any warning symptoms before losing consciousness.

Mr. Maiorca: No, I never had any warning symptoms before losing consciousness.

Dr. Elsner: It may be interesting to think about what happens to certain seals during dives. There are several species that we know living in the polar regions of the world, south and north. Their habitat for a long period of the year is in regions having a continuous ice cover, and they move from one breathing hole to another. In the antarctic the Weddell seal is capable of a breath-hold time of approximately 60 minutes, and we know that they are able to go from a breathing hole to make a long excursion of a couple of kilometers and come back to the same breathing hole. Not only do they have to find their way back to the breathing hole but they have to sense the half time of the stored oxygen to get back.

Dr. Siesjo: That's remarkable. One thing which could be very rapid, of course, would be a response of an intracellular messenger system

which would reduce synthesis or release of agonists like acetylcholine. There is evidence that hypoxia reduces synthesis and/or release of transmitters such as catechole and indolamines, as well as acetylcholine. Clearly, that could alter synaptic function and, thereby, mental and motor performance. The data which I showed from the literature show that the loss of critical judgement occurs at about 40 mm Hg of alveolar oxygen tension. I guess that a classical symptom of hypoxia is loss of judgement.

Dr. Lundgren: I would like to ask Dr. Siesjo if he has had an opportunity in his type of analysis to look at acclimated animals. Acclimated man may show a remarkably increased tolerance to hypoxia and the same thing could be true in animals. Is the acclimation understandable in terms of energy states and so forth?

Dr. Siesjo: No, it isn't. We haven't done it, but it has been done. The mechanisms are not clear, though.

Dr. Arnold: Dr. Siesjo, while we are on the subject of blackout, and you're the brain expert, do you think that seizure could be a possible mechanism in underwater blackout?

Dr. Siesjo: Yes I think so. The ideas about what happens to the neurons in terms of their polarization is that first you would get a slight hyperpolarization, which would reduce or block synaptic transmission, and then you would get depolarization. In the depolarization stage you could easily have synchronous discharge of neurons which would give you a seizure type of functional activity. Then, of course, you block ordered brain functions. I guess that the CO_2 tension attained will greatly influence whether you would get seizure discharge or not.

Dr. Rahn: On that note I would like to ask Dr. Craig whether we have good evidence for seizure underwater?

Dr. Craig: There has been a number of observations, one of them several years ago. I won't mention the source, but he said "we use to put everyone through an underwater swimming". The usual description was to keep on swimming. If the swimming movements became somewhat uncoordinated and then they stopped swimming, we pulled them out. There is no evidence of anyone undergoing seizure. On the other hand, there is the well known hypoxic seizure but it's just a couple of movements, and that is different from the epileptic type of seizures.

Dr. Siesjo: Can I ask if someone has specifically looked for the presence of limbic seizures.

Dr. Arnold: I have seen very few of them, but one would <u>not</u> look for a typical tonic-clonic, epileptic if you're looking for a lymbic seizure.

Dr. Siesjo: What would you test for if you suspected that limbic seizures occur?

Dr. Arnold: Well, first, one would check for memory deficits. There is often emotional lability during that period of time.

Dr. Zannini: I observed a seizure in my buddy while we were breath-hold diving in a swimming pool. He lost consciousness underwater and we took him out of the water over the pool's rim, where he suffered a typical tonic-clonic seizure. When he finally regained consciousness, after about twenty minutes, he said that two months earlier he had suffered a head trauma. An EEG examination revealed fronto-parietal abnormalities.

Dr. Siesjo: I should perhaps, before I forget it, mention that for those of you who are interested in the symptoms of complete anoxia in the brain, there is a remarkable and fascinating article by Rossen et al. from 1943. (Rossen, R., H. Kabat, and J.P. Anderson. Acute arrest of cerebral circulation in man. Arch. Neurol. Psychiat. 50: 510-528, 1943). The authors took volunteers, I think prisoners who volunteered to do this, put a cuff around the neck and recorded the symptoms of these individuals second by second, and described everything which happened up to 2.5 minutes of anoxia. That's in man.

GENERAL DISCUSSION

Dr. Arnold: Just once again, if we could make our best summary right now as to whether seizure is a likely contributor or main candidate of underwater blackout. In your areas that were susceptible, the reticular activating system in the brain stem seem to be better protected than the limbic system and a limbic seizure would cause emotional changes, but should not alter consciousness unless it generalizes. When it generalizes, then it will look tonic clonic, so if we've observed, as Dr. Craig has observed, lots of different cases and there was no tonic clonic activity other than the one with trauma, I would say it's probably a less likely candidate for the actual underwater blackout, although limbic seizures might be possible.

Dr. Siesjo: I should perhaps mention that it is discussed at present whether short periods of ischemia give rise to a longlasting state of increased excitability at certain synapses. When, for example, glutamate receptors are bombarded with their agonists, as would occur during a transient ischemic attack, the ensuing calcium influx is supposed to activate calcium-dependent proteases which break down cytoskeletal proteins. The result of that is that you increase the number of receptors at the synapse so now the post-synaptic receptors are supposed to emerge with an increased sensitivity to their own transmitter. Then, even if you release normal amounts of transmitter in the hours or days to come, the response will be brisker than it was before. Conceivably, therefore, one could have a hyperexcitable state lasting for hours and days after a calcium transient influx of this type.

Dr. Lundgren: In John West's description of the last American Himalayan expedition, in an issue of Science in 1984 (West, J.B., Science 223: 784-788, 1984), he mentiones that 13 out of 16 climbers had measurable signs of cerebellar dysfunction that still persisted one year after the expedition. Can you in any way place that in the context of your observations of selective vulnerability?

Dr. Siesjo: Experimentally, one can grade the ischemia to its density and length so that the only area in the brain which would be hit, and eventually more or less completely destroyed, would be the CA1 area in the hippocampus. It can be expected that the animals afterwards have a defect in short term memory. There are patients described with similar lesions and, as far as I know, they have memory deficits. A recent case illustrates the point. That patient had had a few minutes of cardiac arrest during an anesthesia accident and survived and lived with a surprisingly good intellectual capacity and died for some other reason two or three years afterwards. On pathology, it turned out that the only damage in the brain was a complete destruction of the CA1 sector in the hippocampus. There are patients of this type who can come into his or her physician, start the interview and appear absolutely intact in their intellectual functions, then the doctor can leave the room, come back in, and the patient will stand up and introduce himself or herself. Presumably, such patients have hippocampal damage.

Dr. Arnold: Siebke (Siebke, et al. Lancet 1: 1275-1277, 1975)

reported a case of a five-year-old in Norway, who probably has the record of survival after being forty minutes under the ice. He gradually had a return of memory function which was slower than his motor function and it appeared as if his limbic system had the slowest recovery. He was also starting from a five-year-old system which was more adaptable to recovering than an adult.

Dr. Rahn: I'd like to ask Dr. Lanphier to reflect upon the observations reported by Cross on the divers in the Tuamotu Archipelago.

Dr. Lanphier: We're talking here about the condition called Taravana and it was described classically, I think, in an article in Skin Diver in 1962 by E.R. Cross, who also gave a paper at the 1965 Tokyo symposium and it's a real puzzle because these people apparently surface successfully, but then they undergo some drastic occurrence and fall back into the water. Hypoxia could be an explanation, we were particularly intrigued with the possibility that it represented decompression sickness involving probably the fastest tissues, (the blood, perhaps) with bubbles that immediately obstructed cerebral vessels. I don't have the answer to what's happening to these people. The way to weasel it out would be to say that sometimes it's decompression sickness and sometimes it's hypoxia, but I think that is very unlikely. It's got to be one or the other and, it would be fascinating to find out which.

Dr. Rahn: These were dives to 30 or 40 meters

Dr. Lanphier: They were as deep and long as you could do; and I think that the observations by Dr. Lin and his associates point out that there's about a five second delay between the first breath and the brain actually seeing additional oxygen, and that would almost be enough to explain these cases.

Dr. Rahn: But would it explain what looked like spinal hits which resolved in most cases after two or five days. Was there severe spinal damage?

Dr. Lanphier: I had forgotten about that one - that of course would make it look more like decompression sickness.

Dr. Craig: Wasn't there also some type of impairment and some people experienced residual damage?

Dr. Rahn: Yes, in terms of vision and in terms of hearing.

Dr. Lanphier: After what we've heard just now, I think it could be late hypoxia. But I prefer to have decompression sickness as the explanation.

The Diving Response and Breath-holding Capacity in Man

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The diving response has traditionally been looked upon as an important oxygen conserving mechanism in natural divers such as seals, whales, and ducks. Peripheral vasoconstriction combined with bradycardia and lowered cardiac output may reserve the oxygen content of the blood primarily for the heart and CNS allowing the animals to perform long duration breath-hold dives lasting as much as 30-60 minutes in the large diving mammals. This mechanism has also been suggested to have contributed to the survival of persons who have been accidentally submerged in ice water for up to about 40 minutes (1). Not surprisingly, there is no experimental support for this notion. More remarkable is that there seems to be very little in terms of animal experimentation to directly test the importance of the diving response or the dive duration in diving animals.

In recent years there has even been a de-emphasis of the importance of the diving response since it was found that the response, at least in terms of bradycardia, often is absent in freely diving birds and seals. However, its presence under more strenuous conditions such as forced diving in the laboratory or very deep dives in the Weddell seal suggest that it is of biological value (2).

In conjunction with studying the circulatory responses in ducks whose beaks were forcibly submersed under water, Hollenberg and Uvnas (3) found that chemoreceptor denervation abolished the circulatory response. Although the study was not aimed at recording maximal tolerance to asphyxia, one of three denervated ducks unexpectedly succumbed after 2 to 3 min of asphyxia which was much less than non-denervated ducks were able to tolerate (3).

The diving response may be reliably produced in man performing voluntary breath-holds while exposed to cool water. The possibility that the diving response may be of importance for man's ability to breath-hold has recently been explored in a couple of studies (4,5). In our laboratory a series of experiments were performed with subjects submerged in water at different temperatures (5). They made repetitive breath-holds of maximal duration at a standardized lung volume of 85% of vital capacity, the breath-holds being separated by 4 minute rest intervals. In Fig 1 are shown the results of such series of breath-holds performed by one of the subjects while submerged, on different days, in water at temperatures ranging from 15°C to 35°C. Each series began with several breath-holds while the

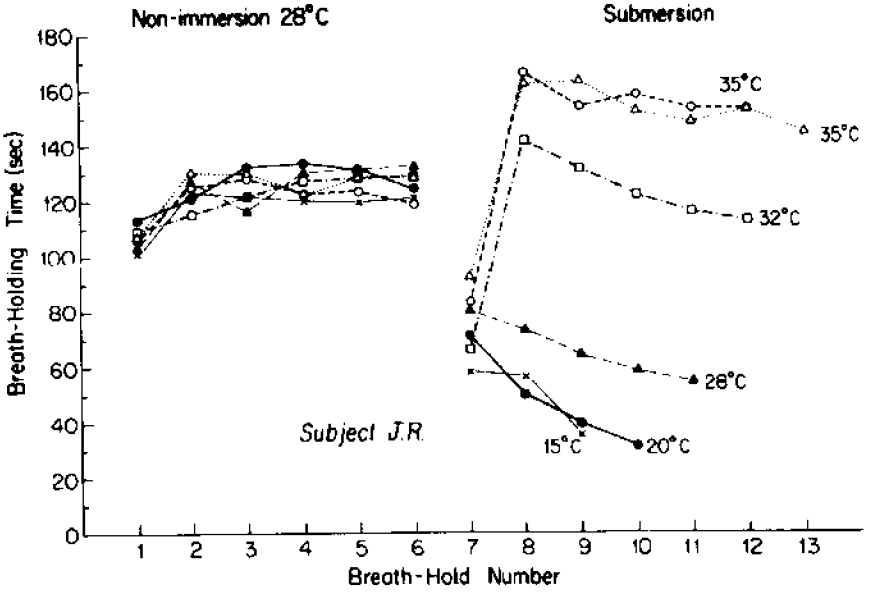

Fig 1 Maximal breath-holding time in series of up to 13 breath-holds recorded on six different days in a resting subject during non-immersion in an ambient air temperature of 28°C followed by submersion in water temperatures between 15°C and 35°C (two series of breath-holds). Resting intervals between breath-holds lasted 4.0 min. From (5) with permission.

subject sat non-immersed in a thermoneutral (28°C) environment. These first breath-holds always showed an increase in duration which is known as the short term training effect (6). After that, reproducibility was excellent - the range of variation in this subject was less than 6% between different days. The results of breath-holds in different water temperatures are shown in the right hand panel of Fig 1: the cool water temperatures were connected with markedly shortened breath-holding times while thermoneutral water (35°C) yielded somewhat increased breath-hold durations, and the durations in water at 32°C were similar to those during the control experiments in the non-immersed condition.

The averaged results in five subjects are shown in Fig. 2 in which breath-holding times are expressed in percent of control values. The decrease in maximal breath-hold duration in water at 20°C was about 55% and the increase in water at 35°C was 26%. These results should be compared with the diving response under the corresponding experimental conditions.

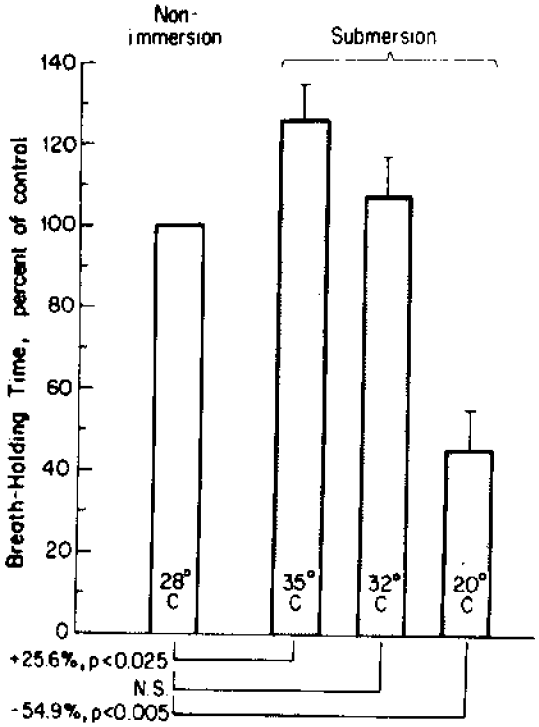

Fig 2 Maximal breath-holding times during submersion in water at 35°C, 32°C, and 20°C expressed as percent of breath-holding time under control conditions, i.e., nonimmersion, ambient air temperatures 28°C. Values are means ± SE of two series of breath-holds in each of 5 subjects; statistics were made by paired comparisons and t-test. From (5) with permission.

The diving response expressed as a lowering of the heart rate is illustrated in Fig 3 which shows average values for normalized heart rates. These were based on measurements taken 30 sec into the breath-holds when typically a stable heart-rate had been established.

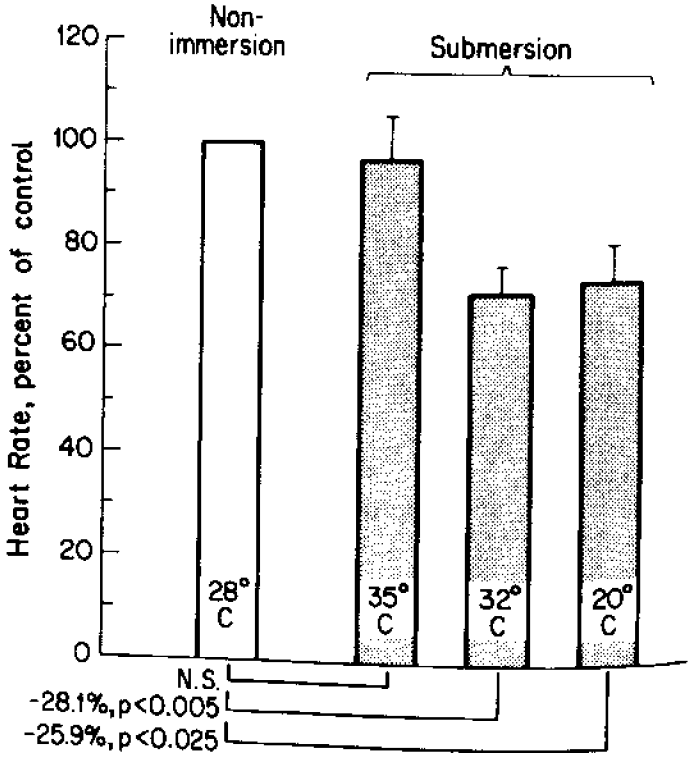

Fig 3 Heart rates 30 s into breath-holds during submersion in water at 35°C, 32°C, and 20°C expressed as percent of heart rate during breath-holding under control conditions, i.e., nonimmersion, ambient air temperatures 28°C. Values are means ± SE of two series of breath-holds in each of 5 subjects; statistics were made by paired comparisons and t-test. From (5) with permission.

In water at 35°C there was no change in heart rate while in cooler water there was a relatively marked diving bradycardia, i.e. the heart rate was reduced by 28% in water at 32°C and by 26% at 20°C. Yet, as mentioned above, the breath-hold durations were substantially reduced. These results were obtained with pre-breath-hold CO_2-tensions in alveolar air which, in the cool water experiments, were slightly lower than in the non-immersed control situation (Fig 4), and therefore should have favored the breath-holding capacity. The alveolar CO_2-tensions at the breaking point were the same in all experiments (Fig 4)

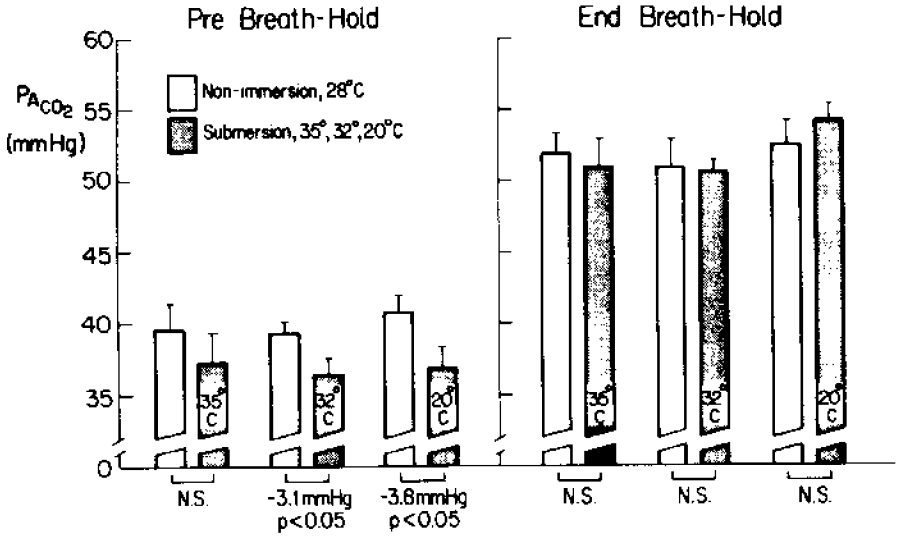

Fig 4 Alveolar carbon dioxide tensions (PA_{CO2}) pre breath-hold and at end of breath-holds of maximal duration during nonimmersion in air at 28°C and during submersion in water at 35°C, 32°C, and 20°C. Values are means ± SE of two series of breath-holds in each of 5 subjects; statistics were made by paired comparisons and t-test. From (5) with permission.

indicating a stable CO_2-sensitivity throughout. The shortening of the breath-holds in cool water seems to have depended at least in part on an increase in metabolism. We measured oxygen consumption and carbon dioxide elimination during eupenic breathing after the breath-hold series (Fig 5). With an increase

in metabolism by over 250% in water at 20°C, it is only to be expected that the breaking point should be reached earlier. Another important respiratory stimulus may be generated by the stimulation of skin cold receptors (7,8).

The effect of cold water exposure was particularly well demonstrated in a study by Hayward and co-workers (4). Their subjects were catapulted down a slide into water of different temperatures ranging from 0°C to 35°C. This was done in order to simulate an accident such as driving off a pier in a car. The time the subject could hold the breath was recorded. Heart rate was recorded by ECG. The breath-hold durations are shown as

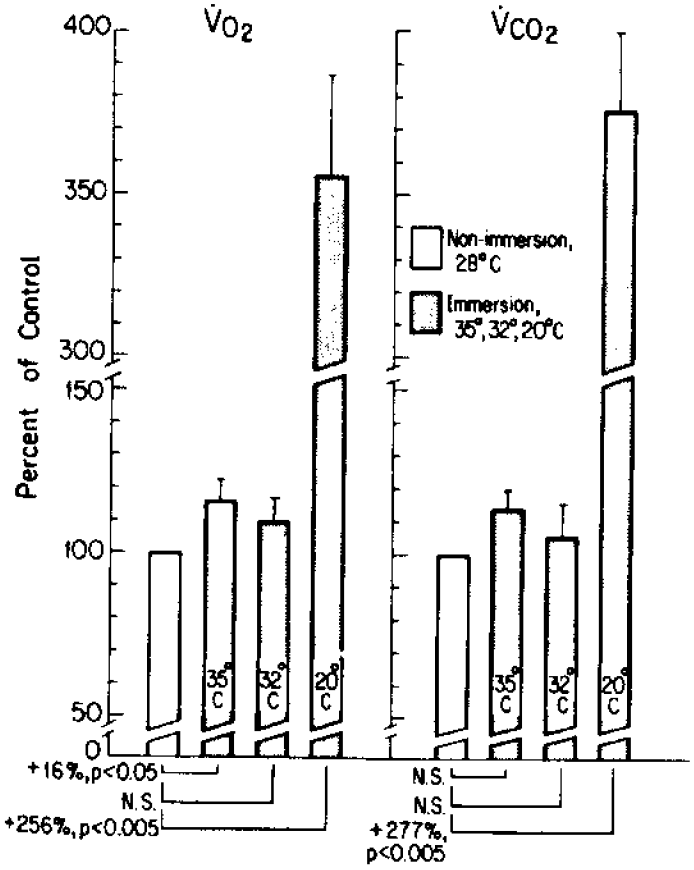

Fig 5 Oxygen uptake (VO_2) and carbon dioxide elimination (VCO_2) during resting, breathing, and immersion to the neck in water at 35°C, 32°C, and 20°C expressed as percent of values recorded in air at 28°C (control experiments: mean VO_2 = 264 ± 20 ml STPD/min, VCO_2 = 222 ± 18 ml STPD/min). Values are means ± SE from 5 subjects; statistics were made by paired comparisons and t-test. From (5) with permission.

"first submersion" in Fig 6. The striking feature is, again, that breath-holding times were always shorter than during pre-immersion despite the fact that there was a solid diving bradycardia throughout the range of water temperatures (Fig 7).

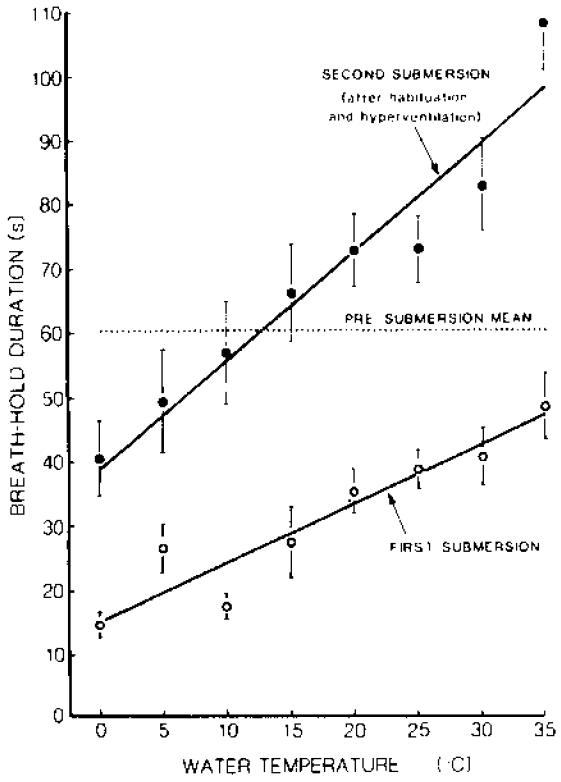

Fig 6 Effect on breath-hold duration of humans subject to a first submersion in water of different temperatures and a second submersion two minutes later after a head-out rest in the water and a brief hyperventilation; each pair of data points from groups of 20 different subjects. Values are means ± SE. From (4) with permission.

After the first submersion and breath-hold the subjects of Hayward et al (4) rested with the head above water for 2 minutes and performed a hyperventilation during the last 10 seconds.

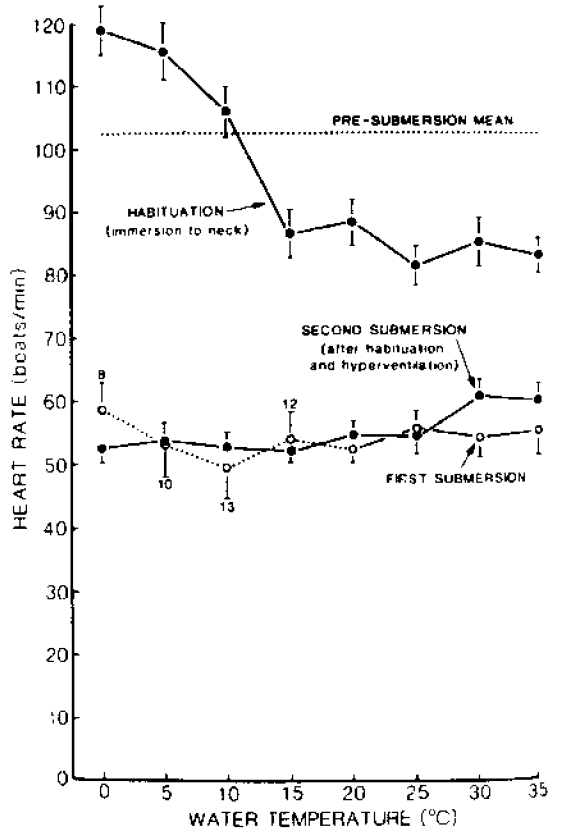

Fig 7 Effect of water temperature on minimum heart rate observed during diving bradycardia of humans. Comparisons with presubmersion heart rate and that during 2-min period of habituation between submergences is provided (cf Fig 6). Dotted lines in first submersion join means for which subject numbers were reduced from 20 due to insufficient breath-hold duration for full development of diving bradycardia. Vertical lines indicate SE. From (4) with permission.

This was followed by a second breath-hold. This phase of the experiment was designed to simulate a situation in which a person in a submerged car is able to breathe from an air pocket before attempting to escape. Again, the subjects developed a marked diving bradycardia across the range of water temperatures (Fig 7). That they did so as much in water at 35°C as at colder temperatures is at odds with many other investigators' findings (e.g. 9, 10, 11, 12, 13, 14, 15, 16, 17), including our own (5). At any rate, the duration of the breath-holds (Fig 6) after habituation were still below control levels in waters colder than 15°C and the increase, above control, in breath-holding time in water that was warmer than 15°C (4) may have been due to a number of factors including hyperventilation.

Because of the possibility that cold receptor stimulation and/or increased metabolism may mask any effect of the diving response in man who is not a natural diver we designed experiments in which the diving response was elicited by breath-holding and wetting only the face with cold water (18). The diving response was further strengthened by performing the breath-holds in combination with light exercise on a bicycle ergometer. In these experiments the diving response was monitored in terms of cardiac output, heart rate and limb blood flow.

The strengthening of the diving response that we were able to achieve by flushing the face with cold water consisted of a roughly 25% depression of cardiac output, mostly due to bradycardia and an almost 50% reduction of forearm blood flow. Despite these relatively pronounced circulatory changes, all being part of the diving response, there was no measurable change in the breath-hold durations.

Our conclusion from these various observations is that the diving response in man, at least when it consists of reducing limb blood flow by half and cardiac output by up to 30%, whether elicited in conjunction with whole body submersion or face immersion only, is of no consequence for man's breath holding ability.

As for our observation in subjects submersed in thermoneutral (35°C) water that the breath-holding time may substantially exceed that observed in the non-immersed control situation, we have observations indicating that their ability to store CO_2 is enhanced in this situation (19). Purely as a speculation, we have considered that, if the increase in cardiac output during immersion were to lead to an increase in perfusion of the chemoreceptors, this could perhaps slow the buildup of respiratory drive. Further details are being worked out in ongoing experimentation.

REFERENCES

1. Nemiroff, M.J., Salz, G.R., Weg, J.G. Survival after cold-water near-drowning: the protective effect of the diving reflex. Am. Rev. Respir. Dis. 115(suppl):145, 1977.

2. Kooyman, G.L., Campbell, W.B. Heart rates in freely diving Weddell seals. Leptonychotes wedelli. Comp. Biochem. Physiol. 43A:31-36, 1972.

3. Hollenberg, N.K., Uvnas, B. The role of the cardiovascular response in the resistance to asphyxia of avian divers. Acta Physiol. Scan. 58:150-161, 1963.

4. Hayward, J.S., Hay, C., Mathews, B.R., Overweel, C.H., Radford, D.D. Temperature effect on the human dive response in relation to cold water near-drowning. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 56:202-206, 1984.

5. Sterba, J.A., Lundgren, C.E.G. Diving bradycardia and breath-holding time in man. Undersea Biomed. Res. 12:139-150, 1985.

6. Heath, J.R., Irwin, C.J. An increase in breath-hold time appearing after breath-holding. Respir. Physiol. 4:73-77, 1968.

7. Keatinge, W.R., Nadel, J.A. Immediate respiratory responses to sudden cooling of the skin. J. Appl. Physiol. 20:65-69, 1965.

8. Cooper, K., Martin, S., Ribea, P. Respiratory and other responses in subjects immersed in cold water. J. Appl. Physiol. 40:903-910, 1976.

9. Craig, A.B.,Jr. Heart rate responses to apneic underwater diving and to breath holding in man. J. Appl. Physiol. 18:854-862, 1963.

10. Hong, S.K., Moore, T.O., Seto, G., Park, H.K., Hiatt, W.R., Bernauer, E.M. Lung volumes and apneic bradycardia in divers. J. Appl. Physiol. 29:172-176, 1970.

11. Kawakami, Y., Natelson, B.H., DuBois, A.B. Cardiovascular effects of face immersion and factors affecting diving reflex in man. J. Appl. Physiol. 23:964-970, 1967.

12. Kobayasi, S., and Ogawa, T. Effect of water temperature on bradycardia during nonapneic facial immersion in man. Jpn. J. Physiol. 23:613-624, 1973.

13. Moore, T.O., Lin, Y.C., Lally, D.A., Hong, S.K. Effects of temperature, immersion, and ambient pressure on human apneic bradycardia. J. Appl. Physiol. 33:36-41, 1972.

14. Paulev, P.E. Cardiac rhythm during breath-holding and water immersion in man. Acta Physiol. Scand. 73:139-150, 1968.

15. Song, S.H., Lee, W.K., Chung, Y.A., Hong, S.K. Mechanism of apneic bradycardia in man. J. Appl. Physiol. 27:323-327, 1969.

16. Speck, D.F., Bruce, D.S. Effects of varying thermal and apneic conditions on the human diving reflex. Undersea Biomed. Res. 5:9-14, 1978.

17. Whayne, T.F.,Jr., and Killip, T., III. Simulated diving in man: comparison of facial stimuli and response in arrhythmia. J. Appl. Physiol. 22:800-807, 1967.

18. Sterba, J.A., Lundgren, C.E.G. Breath-hold duration in man and the diving response induced by face immersion. Unpublished.

19. Sterba, J.A., Lundgren, C.E.G. Unpublished observations.

Discussion following Dr. Lundgren's presentation

Dr. Hong: What is the relationship between the breath-holding time and the oxygen consumption? In general, there is an inverse relationship between the VO_2 and the breath-holding time. Have you considered this relationship as a factor for determining the breath-holding time in your experiments?

Dr. Lundgren: The best person to give you a quantitative answer is Dr. Lin. We have not correlated oxygen consumption and breath-holding time in a quantitative manner.

Dr. Hong: The oxygen consumption is expected to be high during cold water immersion, which should cut down the breath-holding time.

Dr. Lundgren: Yes, I'm saying that that is probably the explanation, plus cold receptor stimulation. Those are the most likely candidates to explain the shorter breath-holding time in the cold water. In 35°C water, which I didn't discuss much today, we observed a prolonged breath-holding time.

Dr. Craig: Along the same lines about the 35°C water and the other water temperatures, how long were your subjects immersed before you made measurements?

Dr. Lundgren: Well, the first immersed breath-hold was immediately upon immersion, and then they sat for six to seven breath-holds in the water with four minute rest periods in between, so say that each breath-hold took two minutes and a rest period of four, we're talking something like half an hour for the last breath-hold.

Dr. Craig: We had subjects in 35°C water for about an hour and the response to the breath-hold was that the heart rate changed over the whole hour, first from a slight bradycardia to tachycardia within the hour, so the time period does make a difference.

Dr. Lundgren: It might well make a difference, however, for any one of these series, whether it's in 32, 20, or 35 degree C water, we have comparable conditions between the eleventh and the twelfth breath-holds, so in each series, they are comparable in that phase.

Dr. Siesjo: Dr. Lundgren, I was wondering about one thing you said. Normally we would believe that brain blood flow depends on two factors, perfusion pressure and vascular resistance. There is normally a very efficient vascular autoregulation. You stated that an increase in cardiac output would lead to an increase in blood flow, what is the evidence for that?

Dr. Lundgren: Well, I cannot speak for the brain, I can speak for the rest of the body in that, in our catheterized subjects whom we put in an identical situation, we did indeed see a rather drastic fall in peripheral resistance in conjunction with head-out immersion (Arborelius, M., Jr., U.I. Balldin, B. Lilja, and C.E.G. Lundgren. Aerospace Med. 43(6): 592-598, 1972). It almost completely balanced the increase in cardiac output.

Dr. Siesjo: Was there any evidence that that happened to the brain?

Dr. Lundgren: We did not measure brain blood flow.

Dr. Siesjo: I would be surprised if there were a change. There is an article by Scheinberg and Stead from 1949. (Scheinberg, P. and E.A. Stead (1949). J. Clin. Invest. 28. 1163-1171). They looked at the effect of head-up tilting on cerebral blood flows using Scheinberg's modification of the Kety-Schmidt technique. There were no changes in blood flow.

EFFECT OF O_2 AND CO_2 ON BREATH-HOLD BREAKING POINT*

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Workers in the field of breath-hold (BH) studies recognized from the start the complexity of BH breaking point and the inadequacy of chemical stimuli in predicting BH breaking point. Simple observations warrant such conclusion in that BH terminates at a variety of alveolar gas compositions; rebreathing can be tolerated much better than BH at a similar alveolar gas composition; and that BH time varies greatly from person to person and from time to time in the same individual under an identical condition.

A variety of factors, including subjective ones, contribute to bring about the **conventional BH breaking point**, that is, to terminate BH when one feels BH can no longer be continued. This subjectiveness contributes to the great variability of BH time which defies prediction based on chemical and mechanical factors. On the other hand, chemical stimuli alone determine a physiologically definable BH end point. At such time during a course of BH, specific physiological responses occur, signifying desire to resume breathing, but BH may still be continued by conscious effort. This "breaking point" is termed **physiological breaking point** which, in contrast to the conventional breaking point, varies within a narrow range. This review examines the effect of PO_2 and PCO_2 on BH breaking points and draws attention to the multitude of factors controlling them.

TIME COURSE OF A BREATH-HOLD

The time course of breath-hold (BH) can be characterized by 1) voluntary inhibition of respiratory muscular activity, with the glottis closed the intrathoracic pressure is stable and slightly above the ambient; 2) the onset of involuntary inspiratory activity with the glottis closed, accounting for the subatmospheric and cyclic intrathoracic pressure which intensifies as BH continues; and, finally, 3) opening of the air way. This sequence of events corresponds, respectively, to the event markings A, B, and C (Fig. 1).

PHYSIOLOGICAL BREAKING POINT

At event B, involuntary ventilatory activities (IVA) occur while inhibition of glottis opening is still possible. It signals the end of the "easy-going phase" (12); and identifies the onset of "desire to breathe"

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(13), "want of oxygen" (18), "diaphragm contraction" (1, 23, 32), or "involuntary ventilatory activity" (25).

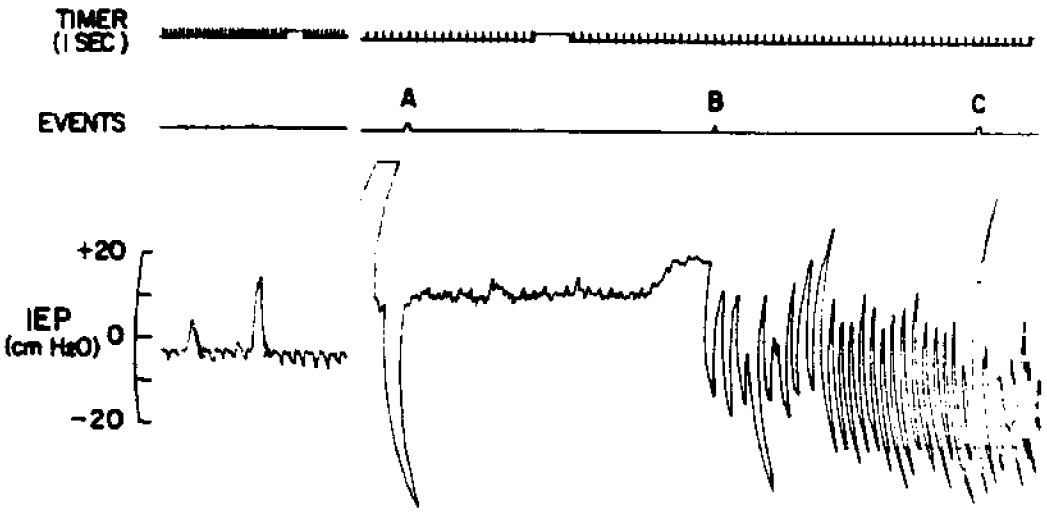

Fig. 1. A recording of intraesophageal pressure (IEP) during a course of breath hold. Events are A, beginning of breath-hold (BH); B. onset of involuntary ventilatory activity; and C, termination of BH. The large upward and downward deflections of IEP just prior to BH represent maximal expiration and maximal inspiration. The subject was exercising at a rate of 167 kgm/min while breath holding. A similar result was observed during BH at rest, but with a much longer BH time (Modified form Lin et al, 1974).

Critical PCO_2

The initiation of involuntary ventilatory activity (IVA) occurs at an alveolar PCO_2 which is remarkably similar in various BH conditions (Table 1). For this reason, it is reasonable to call it the physiological breaking point. When hypoxic effect was removed by BH with O_2, the IVA begins when the $PACO_2$ reached 48-49 torr (Table 1).

TABLE 1 ALVEOLAR PCO_2 AT PHYSIOLOGICAL BREAKING POINTS*

REFERENCE	BHT sec	$PACO_2$ mmHg	$PaCO_2$** mmHg	NOTE
Kobayashi & Sasaki, 1967	80	48.8	-	
Agostoni, 1963	37	49.3	47.6	
"	68	48.3	46.9	High VE
"	62	48.4	47.1	80%VC
"	66	49.3	47.7	20%VC
Lin et al, 1974	104	48.6	47.9	
Douglas and Haldane, 1909	-	48.6	-	

*BH with oxygen at rest
**Estimated from $PACO_2$ with correction of sampling delays

The arterial PCO_2 ($PaCO_2$) in the respiratory centers at the onset of diaphragm contraction had been estimated from alveolar values by correcting for sampling (t_1) and circulatory transport (t_2) delays:

$$PaCO_2 = PACO_2 - (t_1 + t_2)dPACO_2/dt$$

The sampling delay was short (1 sec) and constant. The transport delay from the lung to the ear has been estimated to be 5-13 s with various methods (3, 7, 25, 45). The critical $PaCO_2$ during BH with O_2 was reported to be between 46.9 and 47.6 torr (1, 25). This is a remarkably narrow range considering the wide variation in BHT (Table 1).

Interaction of Hypoxia and Hypercapnia

With presence of hypoxia when BH with air, PACO2 is slightly lower than BH with O_2 at physiological breaking point. At rest, the $PACO_2$ was lowered from 48.6 torr BH with O_2 to 45.8 torr BH with air; and during exercise, it was lowered from 54.0 torr BH with O_2 to 47.9 torr BH with air (25). When PAO_2 is high the $PACO_2$ differed little at the onset of IVA. The regression for this relationship was $PACO_2 = 0.00903PAO_2 + 43.8$ (Fig. 2). The slope differed insignificantly from zero. However, when the PAO_2 falls during BH with alveolar $PACO_2$ also decreases (1, 13, 25).

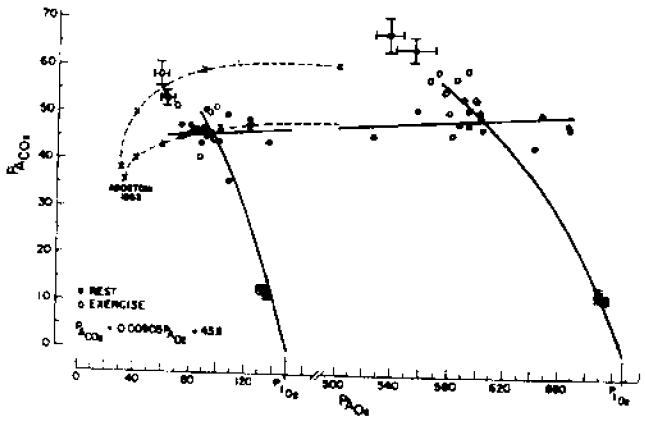

Fig. 2. Alveolar gas composition at the onset of involuntary ventilatory activity and at the conventional breaking point. Solid dots indicate BH at rest, and the circles BH during exercise at 167 kgm/min. Data for the conventional breaking point are grouped for clarity. Mean values at the lower portion of the graph are the calculated gas composition at the beginning of BH. Crosses (x) and broken lines represent Agostoni's data (1963). He obtained these curves for the onset of diaphragm activity (lower curve) and the conventional breaking point (upper curve) by varying the inspired gas mixtures. Douglas and Haldane (1909) and Otis et al. (1948), also by varying inspired gas composition, presented a similar curves superimposable onto the physiological breaking curve shown above (Reproduced with permission from Lin et al., 1974).

Involuntary Ventilatory Acitivity

Esophageal pressure creeps up slightly just prior to the initiation of involuntary respiration (Fig. 1). Contrary to popular belief, the subject does not engage in active Valsalva maneuver. The intrathoracic pressure falls after the initiation of IVA. Similar to this, the intrathoracic pressure (as reflected in the right atrial pressure) also rises just prior to the initiation of involuntary inspirations in the anesthetized dog having the trachea clamped at the end of an inspiration (Fig. 3). The similarity between these two cases indicates that the transient increase in the intrathoracic pressure is not a result of conscious effort. On the contrary, we detect no such response when the dog is allowed to terminate a voluntary snout immersion freely (Fig. 4A). The conscious dog simply does not allow BR to continue long enough to reach the physiological breaking point (29). The invariate intrathoracic pressure, as indicated by the end-diastolic ventricular pressure, represents absence of respiratory activity during the course of a BR (Fig. 4B).

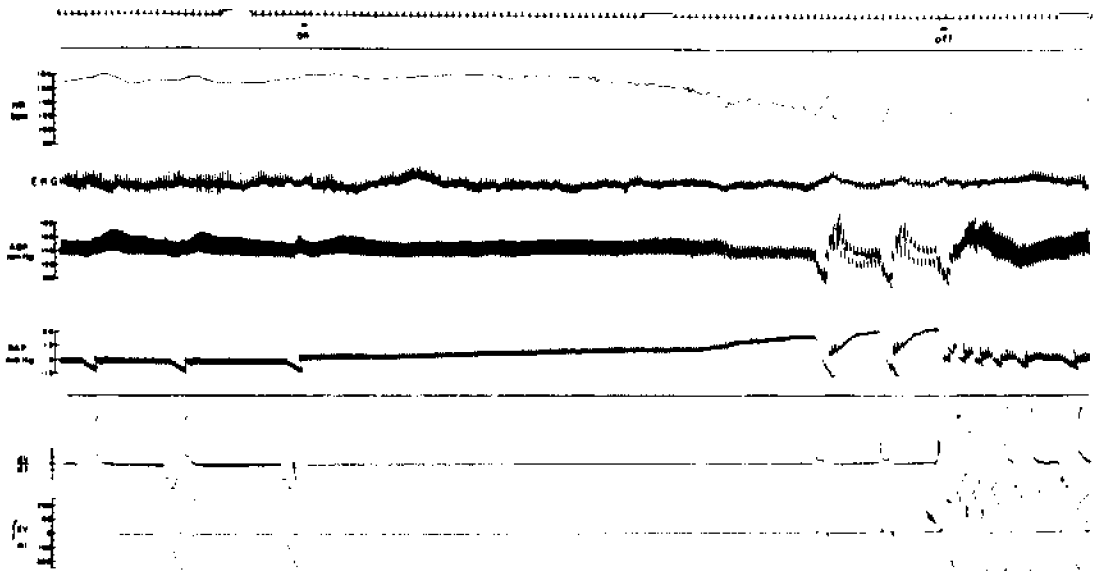

Fig. 3. Typical response of the anesthetized dog to apnea. "On" and "Off" on the top trace signal the start and the end of apnea. Note that right atrial pressure (RAP) rose just prior to initiation of inspirations with trachea clamped for a duration of 80 sec. The bottom two traces represent the tracheal air flow (dV/dt) and tidal volume ($\int dV$) with downward deflection showing inspiration (Adopted from Lin et al., 1975).

Intrathoracic Pressure

According to Agostoni (1), the diaphragmatic contractions account for the involuntary respiration during a BR, and muscles of the abdominal wall contribute little. Since the action of the diaphragm is not balanced

by an activity of its antagonist muscles, the intrathoracic pressure falls with each contraction. Both the frequency and intensity increase as BH continues.

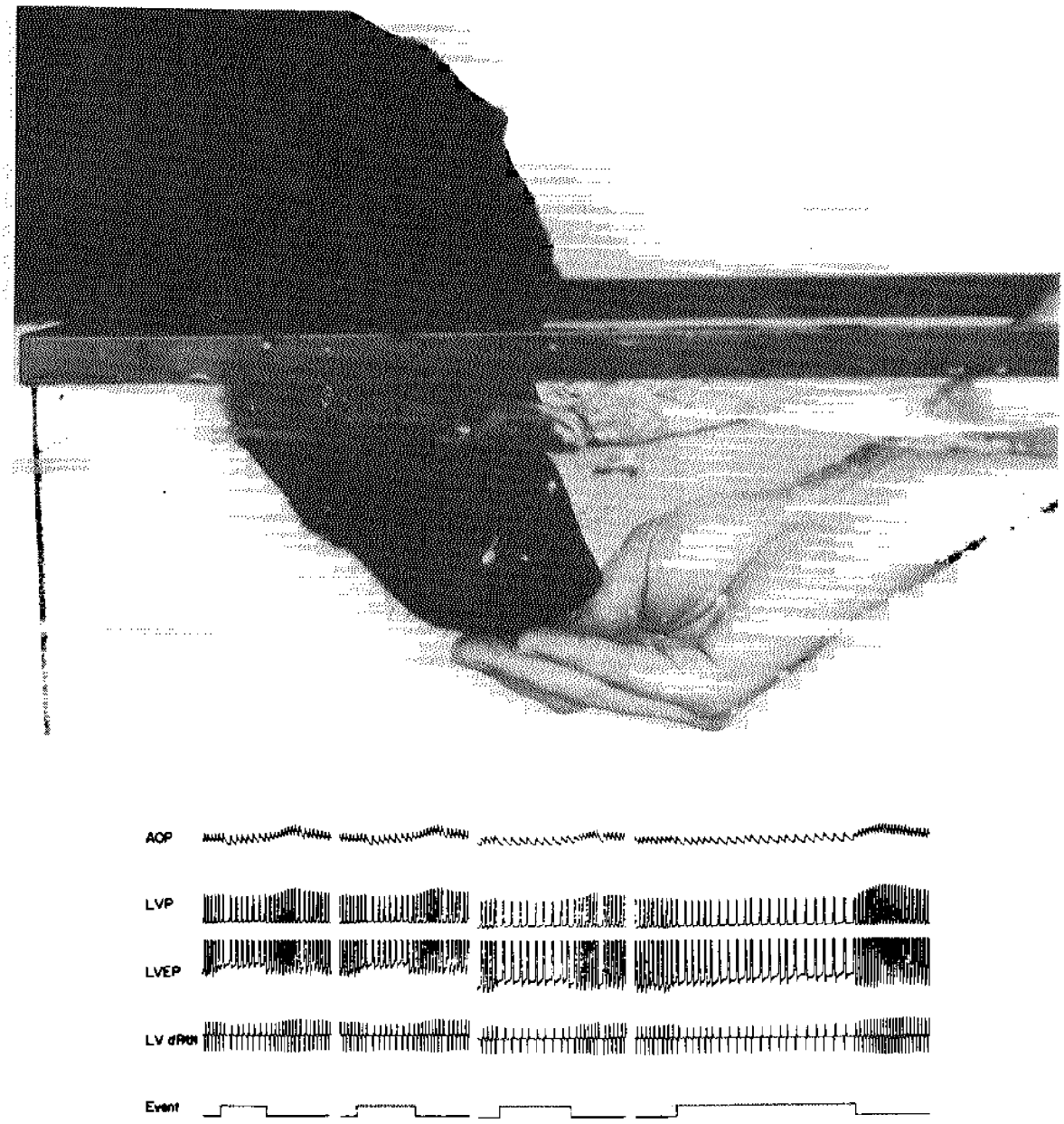

Fig. 4. Top panel. The dog trained to voluntary immersion of snout, on command, up to eye level. Bottom panel. Circulatory responses of a conscious dog to voluntary snout immersion with apnea durations ranging from 5-25 sec. There was no involuntary inspiration during the course of a BH as indicated by the unchanging left ventricular end diastolic pressure (LV EP). AOP and LVP are the aortic and left ventricular pressure. The event trace at the bottom marks the initiation and termination of a voluntary BH. (Modified from Lin, 1983)

In a series of experiments in 5 males, Lin et al. (25) recorded esophageal pressure during BH either with air or oxygen both at rest and during exercise (2-3 times resting $\dot{V}O_2$). Esophageal pressure starts at a level slightly above the ambient, but falls continuously after the onset of IVA to between 10-15 torr below the ambient at the conventional breaking point.

Exercise

Elevation of rate of oxygen consumption shortens duration to both physiological and conventional breaking points. This is not surprising, since the rate of rise of alveolar $PACO_2$ increases during exercise, and should shorten BHT correspondingly. Although exercise elevates $PACO_2$ at IVA at a given PAO_2, it is possible that part of the $PACO_2$ elevation could be related to exaggerated sampling delay and transport delay (7).

CONVENTIONAL BREAKING POINT

Following its onset, the frequency and intensity of IVA increase with continued BH. Nevertheless, conscious suppression of glottis prevails. Eventual opening of the glottis occurs when ventilatory stimulus is too strong to be resisted further by conscious effort. The event at C (Fig. 1) ends an active BH and is termed variously as the "conventional breaking point", the end of the "struggle phase" (12), or simply the **breaking point**. The duration between B and C is highly variable. This variability accounts for BH time ranging from 20 s (37) to 270 s (20) on an identical command: "Exhale maximally, without prior hyperventilation, followed by maximal inhalation and hold the breath as long as possible." Obviously, it involves non-chemical stimuli.

Breath-hold Time

A student of Schneider at the Wesleyan University held his breath for 15 min 13 sec after a period of hyperventilation with air followed by three deep breaths of pure oxygen (37). Mithoefer (31) mentioned in his review that under the same condition, another student at the same university held his breath for 20.1 min. BHT of this magnitude have also been reported following hyperventilation with O_2 by Klocke and Rahn (22). One of their subjects, H. R., performed a BH for 14 min, 13.6 min, and 14.4 min on three occasions. To my knowledge, the longest published record of BH with air without prior hyperventilation is 270 sec (20). BHT of 240 sec under the same conditions are routinely demonstrated by Hong and his colleagues at the University of Hawaii (21). These unusually long BHTs are in accord with O_2 supply and demand relationships. However, the great majority of subjects demonstrate ability to BH only a fraction of the BHT cited above. Fig. 5 shows BHT with air without prior hyperventilation. Such variability in BHT indicates clearly the involvement of non-chemical factors.

Oxygen Supply and Demand

The factors that contribute to the eventual termination of BH have

been examined in terms of alveolar gas composition, lung volume, oxygen consumption, mechanical intervention, such as rebreathing during a BH, psychological factors, and interactions among these factors. Factors that, accountable through supply and demand of oxygen, are summarized in the following equations (22):

$$\text{BH with O2} \quad BHT = \frac{VC(BTPS)}{\dot{V}O_2(STPD)} \times \frac{P_B - 47}{863}$$

$$\text{BH with air} \quad BHT = \frac{TLC(BTPS) \times FAO_2}{\dot{V}O_2} \times \frac{P_B - 47}{863}$$

where BHT represents BH time in minutes, VC and TLC, repectively, the vital capacity and total lung capacity in ml BTPS, $\dot{V}O_2$ the oxygen consumption in ml STPD/min, FAO2 the fractional alveolar concentration of oxygen, $P_B - 47$ the barometric pressure less water vapor pressure at 37°C, and 863 the constant for converting a lung volume from BTPS to STPD. VC instead of TLC is used in the O_2 BH experiment. The assumption here is that residual volume (RV) limits the shrinkage of the lung. TLC is used in air BH experiments because TLC x FAN_2 is greater than RV, regardless of BHT. Estimating from the O_2 supply from the lung alone, BHT of 4 min with air and 16 min with O_2 should be possible for a standard resting man at sea level (VC, 4.78 L; RV, 1.19 L; and $\dot{V}O_2$, 250 ml/min). The BHT should be even longer, if the calculation included usable O_2 in the blood. These unusually long BHTs have indeed been demonstraed in man breathing air (20,

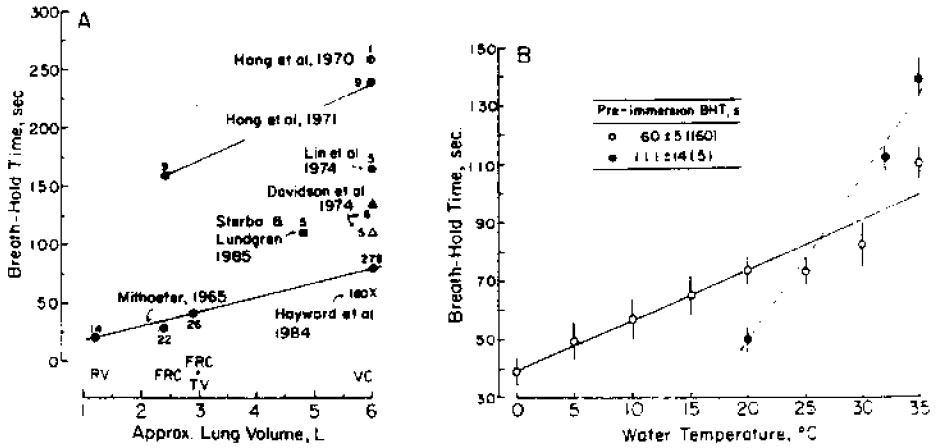

Fig. 5. Effect of lung volume (A) and water temperature (B) on conventional BH breaking point. Hayward et al. (1983) and Sterba and Lundgren's (1983) data are reproduced respectively by circles (o) and dots (•) in B. In A, number of subjects studied and the source of the data points are indicated. Davidson et al. (1974) presented BHT for 5 controls (△) and 6 carotid body resected patients (▲). These patients demonstrated no greater ability to BH comparing to those in many other studies.

21), and breathing oxygen (22, 37). These heroic results can only be achieved by overcoming discomfort, noxious sensations, as well as psychological uncertainty.

The above equations predict prolongation of BHT by raising oxygen supply, reducing $\dot{V}O_2$, or both. These have been demonstrated by the following:

Hyperventilation

Hyperventilation exerts effective prolongation of BHT by raising O_2 supply as well as lowering alveolar PCO_2. This procedure, however, is a double-edged sword (6). With lung supply of O_2 increased by breathing O_2 and reduced alveolar PCO_2 by hyperventilation, BHT can be extended considerably, from 3.1-8.5 min to 6-14 min (22). They recorded a wide range of $PACO_2$ at the breaking point ranging from 51 to 91 torr. Linear relationship exists between BHT and $PACO_2$ ($PACO_2 = 7.69BHT - 10.8$) for BH with O_2, breathing normally. Following hyperventilation, additional BHT is gained by the lowered starting $PACO_2$. Total absorption of available lung O_2 accounts for the increased BHT following hyperventilation with O_2.

Hypobaria and hyperbaria

Hyperbaria prolongs and hypobaria shortens BHT. The above equations readily predicted these results. Linear increases in BHT (as % of 1 atmosphere values) with increasing ambient pressure have been demonstrated in both BH at TLC (35), and at FRC (2, 16). In contrast, a decrease in BHT is found at high altitude. In addition, BHT decreases progressively during exposure to altitude, a phenomenon relating to the loss of alkaline reserve (34, 38).

Oxygen Consumption

It is not surprising that accelerated use of O_2 reduces BHT. Lin et al. (25) described BHT as a function of $\dot{V}O_2$ both for the physiological and conventional breaking points. Their data fit the equation in the form of:

$$BHT = a(1/\dot{V}O_2) + b$$

where a and b are constants. The b in the above equation is small and affects BHT insignificantly. Therefore, a good approximation would be: $BHT \times \dot{V}O_2 = a$. The constant a is the available amount of O_2. The inverse relationship between BHT and $\dot{V}O_2$ has been observed by many investigators previously (3, 7, 8, 10, 35).

Sterba and Lundgren (40) and Hayward et al. (17) reported a direct relationship between BHT and water temperature. Reduction in BHT in cold water, according to these authors, relates closely to the elevated oxygen consumption. These two papers reported a qualitatively similar, but quantitatively diverse, BHT relating to water temperature. Again, it points to the inability of chemical factors to account for the outcome of conventional breaking point.

Breath-Hold Time and Alveolar Gas during Diving

Diving involves physical activity which depresses BHT. Professional BH divers limit their diving time to 60 sec or less. Hong et al.(19) reported a contest for 20 ama and recorded a maximal diving time of 82 sec. Diving duration of 90 sec (39), 118 sec (42), and 115 sec (9) are recorded occasionally. The experienced divers also limit their hyperventilation prior to a dive to a minimum. They know that hyperventilation increases diving time, but they also aware of the danger of extreme hypoxia on ascent.

Alveolar gas composition has been analyzed in the field (6, 19, 42) and in the laboratory with simulated dive (24). The course of alveolar gas exchange differs greatly from that in ordinary BH. Both alveolar PO_2 and PCO_2 at the breaking point is lower than in comparable BH at the normal ambient conditions. Extreme hypoxia could, therefore, occur during the ascent phase.

NON-CHEMICAL FACTORS

Psychological factors have long been recognized as the major determinants of conventional breaking point (4, 18, 37, 43), and undoubtedly contribute to the wide range of BHT reported in the literature (27, 30). Fig. 5 demonstrates clearly the involvement of factors other than the availability of O_2. Prolongation of BHT occurs when respiratory movements are allowed, such as rebreathing without improving alveolar gases (5, 14, 23, 44). Furthermore, reviews show that rigid concepts of chemical and mechanical thresholds are inadequate for interpreting BH breaking point (15, 44), suggesting the importance of subjective factors. Since psychological factors play such an important role and, since it is unreasonable to assume the constancy of these factors within and between individuals, caution should be exercised on regulatory mechanisms based on conventional breaking point (41). Physiological breaking point would be suited better for such purpose.

SUMMARY

Two breath-hold (BH) breaking points have been identified: **physiological** and **conventional**. The former constitutes the initiation of involuntary ventilatory efforts with glottis closed, and the latter coincides with eventual opening of glottis. A variety of factors influence the ability to BH, including psychological ones, which account for the great variability in BH time. within as well as between individuals. The involvement of psychological factors render rigid concepts of chemical and mechanical thresholds inadequate in interpreting conventional breaking points. However, experimental subjects could reproduce consistently the time to initiation of involuntary respiration, i.e. the **physiological breaking point** which relates closely to arterial levels of PCO_2 and PO_2. Control of the **Conventional breaking point** is more complex. O_2 and CO_2 are but two factors in a set of complex mechanisms that govern the BH breaking points.

REFERENCE

1. Agostoni, E. Diaphragm activity during breath holding: factors related to its onset. J. Appl. Physiol. 18:30-36, 1963.

2. Alvis, H. J. Breath-holding break point at various increased pressure. U. S. Navy Medical Research Laboratory Report #177, 10:110-120, 1951.

3. Astrand, P. O. Breath-holding during and after muscular exercise. J. Appl. Physiol. 15:220-224, 1960.

4. Bartlett, D., Jr. Effects of Valsalva and Mueller maneuvers on breath-holding time. J. Appl. Physiol. 42:717-721, 1977.

5. Cain, S. M. Breathing point of two breath holds separated by a single inspiration. J. Appl. Physiol. 11:87-90, 1957.

6. Craig, A. B. Jr. Causes of loss of consciousness during underwater swimming. J. Appl. Physiol. 16:583-586, 1961.

7. Craig, A. B. Jr., and S. A. Babcock. Alveolar CO_2 during breath holding and exercise. J. Appl. Physiol. 17:874-876, 1962.

8. Craig, F. N., and E. G. Cummings. Breath-holding during exercise. J. Appl. Physiol. 13: 30-34, 1958.

9. Cross, E. R., Taravana. Diving syndrome in the Tuamotu diver. In: Physiology of Breath-Hold Diving and the Ama of Japan, edited by H. Rahn, Washington, D. C.: National Academy of Science, Publication #1341, 1965, p. 207-219.

10. Cummings, E. G. Breath-holding at beginning of exercise. J. Appl. Physiol. 17:221-224, 1962.

11. Davidson, J. T., B. J. Whipp, K. Wasserman, S. N. Koyal and R. Lugliani. Role of the carotid bodies in breath-holding. N. Engl. J. Med. 290:819-822, 1974.

12. Dejours, P. Hazards of hypoxia during diving. In: "Physiology of Breath-Hold Diving and the Ama of Japan", edited by H. Rahn. Washington, D. C.: Natonal Academy of Science, publication #1341, 1965, p. 183-193.

13. Douglas, C. G., and J. S. Haldane. The causes of periodic or Cheyne-Stokes breathing. J. Physiol. 38:401-419, 1909.

14. Fowler, W. S. Breaking point of breath-holding. J. Appl. Physiol. 6:539-545, 1954.

15. Godfrey, S., and E. J. M. Campbell. The control of breath holding. Resp. Physiol. 5:385-400, 1968.

16. Hesser, C. M. Breath holding under high pressure. In: Physiology of Breath-Hold Diving and the Ama of Japan, edited by H. Rahn. Washington, D. C.: National Academy of Science, publication #1341, 1965, p. 165-181.

17. Hayward, J. S., C. Hay, B. R. Matthews, C. H. Overweel, and D. D. Radford. Temperature effect on the human dive response in relation to cold water near-drowning. J. Appl. Physiol. 56:202-206, 1984.

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

19. Hong, S. K., H., Rahn, D. H. Kang, S. H. Song, and B. K. Kang. Diving pattern, lung volumes, and alveolar gas of the Korean diving woman (ama). J. Appl. Physiol. 18:457-465, 1963.

20. Hong, S. K., T. O. Moore, G. Sato, H. K. Park, H. R. Hiatt, and E. M. Bernauer. Lung volumes and apneic bradycardia in divers. J. Appl. Physiol. 29:172-176, 1970.

21. Hong. S. K., T. O. Moore, G. Seto, H. K. Park, Hiatt, and E. M. Bernauer. Lung volumes and apneic bradycardia in divers. J. Appl. Physiol. 29:172-176, 1970.

22. Klocke, F. J., and H. Rahn. Breath-holding after breathing of oxygen. J. Appl. Physiol. 14:689-693, 1959.

23. Kobayashi, S. and C. [illegible]ki. Breaking-point of breath holding and tolerance time in rebreathing. Japn. J. Physiol. 17:43-56, 1967.

24. Lanphier, E. H., and H. Rahn. Alveolar gas exchange during breath-hold diving. J. Appl. Physiol. 18:471-477, 1963.

25. Lin, Y. C., D. A. Lally, T. O. Moore, and S. K. Hong. Physiological and conventional breath-hold breaking points. J. Appl. Physiol. 37:291-296, 1974.

26. Lin, Y. C. T. O. Moore, J. J. McNamara, and S. K. Hong. Oxygen consumption and conservation during apnea in the anesthetized dog. Resp. Physiol. 24:313-324, 1975.

27. Lin, Y. C. Breath-hold diving in terrestrial mammals. In: "Exercise and Sport Sciences Reviews", Vol. 10. Edited by R. L. Terjung. Philadelphia: Franklin Press, 1982, p. 270-307.

28. Lin, Y. C. Hemodynamic changes during voluntary snout immersion in chronically instrumented dogs. Chinese J. Physiol. 26:1-10, 1983.

29. Lin, Y. C. E. L. Carlson, E. P. McCutcheon, and H. Sandler. Cardiovascular reactions during voluntary apnea in dogs. Am. J. Physiol. (Reg. Intg. Comp. Physiol.) 245:R143-R150, 1983.

30. Hithoefer, J. C. The breaking point of breath holding. In: Physiology of Breath-Hold Diving and the Ama of Japan, edited by H. Rahn, Washington, D. C.: National Academy of Science, Publication #1341, 1965, p. 195-205.

31. Hithoefer, J. C. Breath holding. In: Handbook of Physiology, Respiration. Washington, D. C.: Am. Physiol. Soc., 1965, 2:1011-1025.

32. Noble, M. I. M., J. H. Eisele, H. L. Frankel, H. Else, and A. Guz. The role of the diaphragm in the sensation of holding the breath. Clin. Sci. 41:275-283, 1971.

33. Otis, A. B., H. Rahn and W. O. Fenn. Alveolar gas exchanges during breath hold. Am. J. Physiol. 152:674-686, 1948.

34. Rahn, H., H. T. Bahyson, J. F. Muxworthy and J. M. Hagen. Adaptation to high altitude: chagnes in breath-holding time. J. Appl. Physiol. 6:154-157, 1953.

35. Robard, S. The effect of oxygen, altitude and exercise on breath-holding time. Am. J. Phsyiol. 150:148-152, 1947.

36. Schilling, C. W., R. A. Hansen, and J. A. Hawkins. The effect of increased air pressure on vital capacity, expiratory force and breath-holding ability. Am. J. Physiol. 110:616-619, 1935.

37. Schneider, E. C. Observation on holding the breaath. Am. J. Physiol. 94:464-470, 1930.

38. Schneider, E. C. Respiration at high altitude. Yale J. Biol. Med. 4: 537-550, 1932.

39. Scholander, P. F., H. T. Hammel, H. LeMessurier, E. Hemingsen and N. Garey. Circulatory adjustment in pearl divers. J. Appl. Physiol. 17:184-190, 1962.

40. Sterba, J. A. and C. E. G. Lundgren. Diving bradycardia and breath-holding time in man. Undersea Biomed. Res. 12:139-150, 1985.

41. Stroud, R. C. Combined ventilatory and brath-holding evaluation of sensitivity to respiratory gases. J. Appl. Physiol. 14:353-356, 1959

42. Teruoka, G. Die Ama und ihre Arbeit. Arbeitsphysiol. 5:239-251, 1932.

43. White, P. D. Observations on some tests of physical fitness. Am. J. Med. Sci. 159:866-874, 1920

44. Whitelaw, W. A., B. McBride, J. Arnar, and K. Corbet. Respiratory neuromuscular output during breath-holding. J. A. Physiol. 50:435-443, 1981.

45. Williams, M. H. Jr. A simple method for measuring lung-to-ear circulation time with an ear oximeter. J. Appl. Physiol. 16:15-20, 1961.

Discussion following Dr. Lin's presentation

Dr. Rahn: I have a question for Dr. Hong. When you were doing those four minute breath-holds mentioned by Dr. Lin, were you also delivering alveolar samples?

Dr. Hong: Yes, that was the trick!

Dr. Lundgren: The point has just been made, and yet I think it should be emphasized even more: we may be talking about different things when we talk about breath-holding. There is a big difference between actually holding your breath and re-breathing into a bladder. The terminology here is a little hazy, when we talk of breath-holding.

Dr. Rahn: You mentioned also some ways to prolong breath-holding, and it doesn't make any difference whether you exhale into a bag and reinhale or whether you squeeze a tennis ball. It seems to me there is a big difference.

Dr. Lin: But that just simply affirms that the chemical factor is not an important determinant of breath-hold time. The discomfort, physical and/or psychological, is the major determinant. If you do anything to divert from discomfort you could prolong the breath-hold.

Dr. Rahn: Yes, but is blowing into a bag and reinhaling the same diversion as squeezing a ball ?

Dr. Lin: No, wait a minute, I didn't say that they prolong a breath-hold in the same way. There are many ways one can prolong a breath-hold, such as, exhale, inhale, swallowing, and even squeezing a tennis ball will help.

Dr. Craig: When you get the involuntary contractions, you accentuate them by pumping in and out with the involuntary contractions. Why that helps, I don't know. It seems to be the most common way for divers to prolong the breath-hold period.

Dr. Zannini: With regard to the breath-hold breaking point, I would like to report some observations on breath-holding times in a group of thirty-seven subjects during training for SCUBA diving. They performed a total of 1800 breath-holds, both before and after a month of training. Those of them who had been training and practicing breath-hold diving even before the course achieved the longest breath-holding times in every condition, i.e., either dry or in the water, with preceding hyperventilation or with resistance to diaphragmatic contractions, at rest or during work. The only condition where they did not differ much was in the dry breath-hold at rest. One month of physical work-out and underwater training did not allow the subjects, who previous to the course had not been breath-hold divers, to do better than the trained ones or to substantially improve their own breath-hold times. That could be due to several factors, among which the increased resistance to hypercapnia and to neuromuscular stimuli, or it could be due to a stronger breath-hold diving reflex in the previously trained subjects.

RESPIRATORY NEUROMUSCULAR OUTPUT IN BREATHHOLDING

by

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Although breathholding is commonly thought of as voluntary suppression of respiratory effort, most subjects studied in the laboratory and experienced physiologist breathholders, in particular, have involuntary rhythmic contractions of inspiratory muscles that begin shortly after the beginning of breathholding and increase in amplitude and frequency until the breakpoint. To avoid breathing in spite of these contractions, the breathholder must voluntarily seal off his glottis or pharyngeal airway, and the breakpoint occurs when some sensation associated with prolonged breathholding persuades him to open his airway and allow the inspiratory efforts he has been making all along to actually draw in some air. Such contractions were reported by Agostoni in 1960 (1) and he and others (2) have used the time when contractions first appeared as the true or "physiological" breakpoint of breathholding. Such experiments showed that the physiological breakpoint was influenced by oxygen and carbon dioxide tensions in much the same way as the breakpoint defined in the more usual manner, but of course the "physiological" breathhold times were considerably shorter--of the order of a third or a quarter the time the subject could keep from breathing by sealing his upper airway.

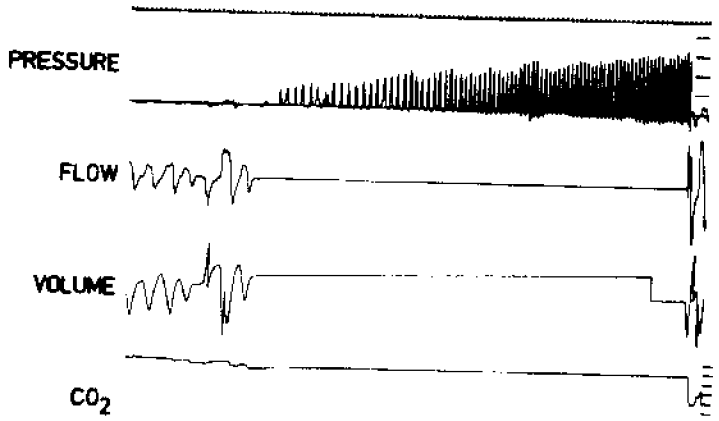
The act of breathholding itself somehow constitutes a stimulus to breathe or a source of discomfort over and above the sensation due to altered blood gas tensions resulting from apnea. This was shown in 1908 by Hill and Flack (3), who compared breathholding with rebreathing from a small bag, which produces the same stimulus of asphyxia. Rebreathing could be tolerated several times longer than breathholding. Fowler's experiment in 1954 suggested the same thing (4). Near the breakpoint of a voluntary breathhold he permitted subjects to take two or three breaths, but from a bag containing a gas mixture identical in composition to alveolar gas at that moment. Without any change in gas tension, these subjects were able to resume breathholding and postpone the breakpoint by many seconds.

Campbell and Godfrey, in a series of papers, developed the hypothesis that the contractions themselves were the source of the extra discomfort that finally obliged subjects to take a breath (5,6,7). The most striking experimental evidence they put forward came from an experiment in which voluntary breathholding at functional residual capacity (FRC) was compared with apnea induced by curare in the same subjects. When respiratory muscle efforts were abolished by curare, most of the discomfort of breathholding disappeared (5).

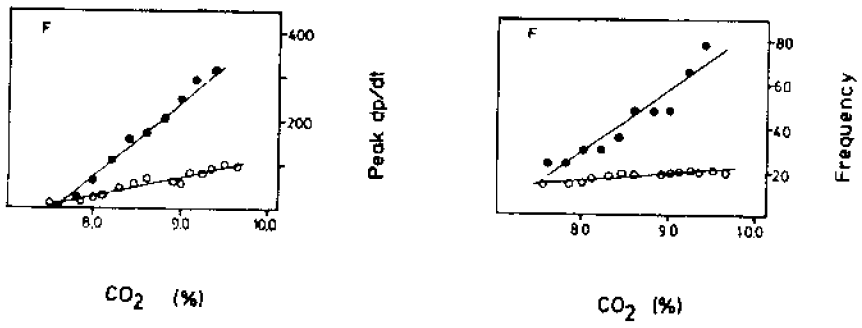
We have done a series of breathholding experiments to test the Campbell-Godfrey hypothesis and examine mechanisms that govern the generation of respiratory muscle contractions and how they might give rise to the discomfort of breathholding. We considered that if the contractions were directly responsible for making the subject abandon breathholding, there should be a correlation between some measurable feature of the contractions and the breakpoint.

All the experiments are performed in the same way. Breathholds are performed by normal subjects sitting in a chair who begin taking a few deep breaths from a small bag containing 8% carbon dioxide in oxygen. At the altitude of our laboratory this mixture has a P_{CO2} close to mixed venous, an alveolar plateau is established, and from then on P_{CO2} rises nearly linearly with time at a rate which is nearly constant, independent of minute ventilation and nearly equal to the rate at which it rises during a breathhold (6,8). Respiratory muscle contractions are monitored by

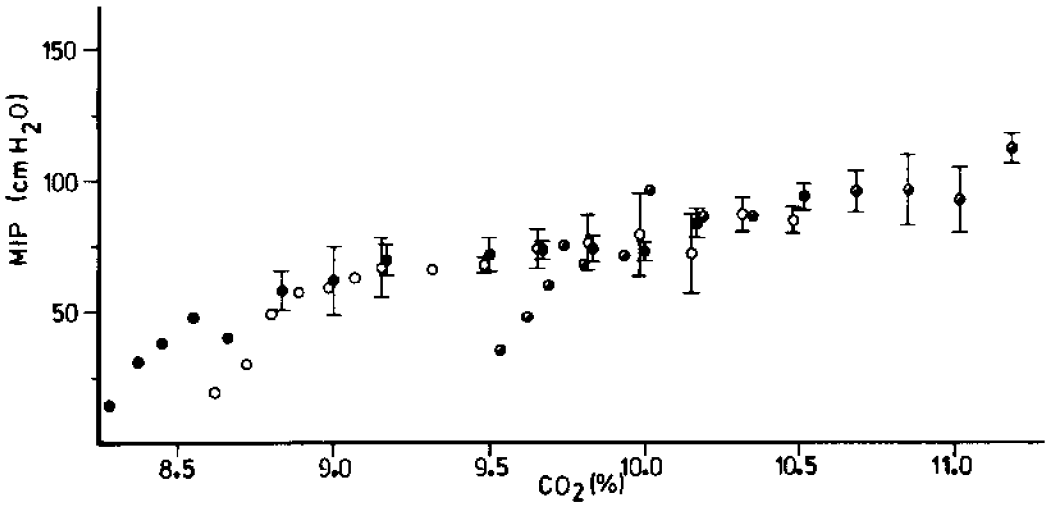
measuring pressure in the airway or in the pleural space. In breathholds at FRC, the mouthpiece is occluded by a valve and pressure measured at the airway opening, but at volumes above FRC the subjects usually close their upper airway, making it necessary to monitor intrathoracic pressure with an esophageal balloon. During a breathhold, the pressure is essentially an "occlusion pressure" and gives a reasonable estimate of global respiratory neuromuscular output (9). At the end of the breathhold the subject exhales to give a measurement of alveolar P_{CO2}, and P_{CO2} within a breathhold is calculated by linear interpolation between the beginning and breakpoint values (10).



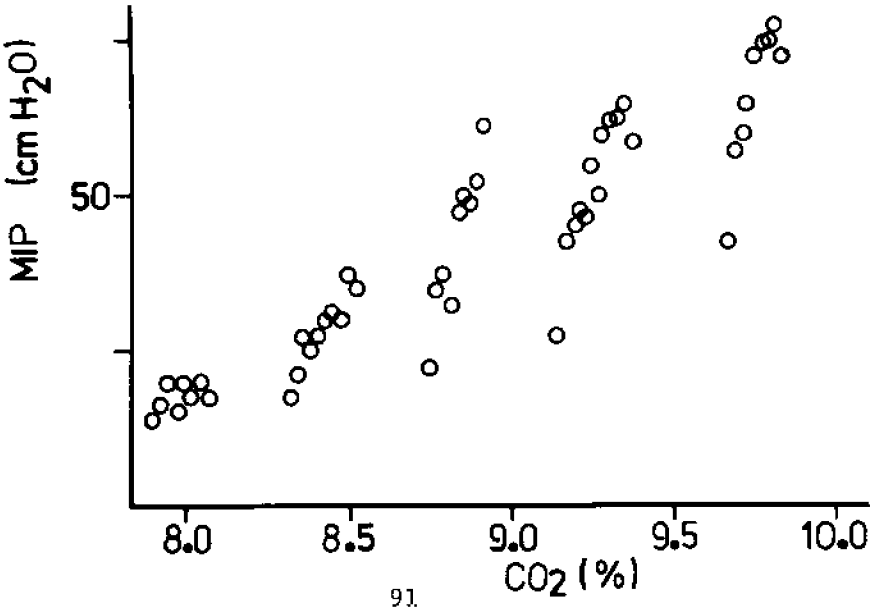
A typical tracing from an FRC breathhold is shown in Figure 1 where negative pressure is up, and the scale shows gradations of 20 cm H_2O. The time marker shows seconds. Shortly after the beginning of the breathhold, negative pressure waves appear and increase in amplitude and frequency until the breakpoint, when peak negative pressure is about 70 cm H_2O and frequency is 70/minute. These pressures and frequencies are several fold greater than those generated by the same subject during an ordinary rebreathing experiment, as shown in Figure 2, where occlusion pressure [measured as $\Delta P/\Delta t$ in cm H_2O/sec at the beginning of inspiration in the left panel (open circles)] and frequency in breaths/min) are plotted in the right panel against percent CO_2 for breathholding (solid circles) and rebreathing (open circles).



In a second kind of experiment, subjects began breathholds at FRC with a variety of starting values of P_{CO2}. This was achieved simply by having them rebreathe from the small bag for different periods before beginning the breathhold. The results of such an experiment are shown in Figure 3. Peak inspiratory pressure in cm H_2O is plotted against CO_2. Different symbols represent different breathholds. Means from all the contractions in a 10 sec interval are shown.

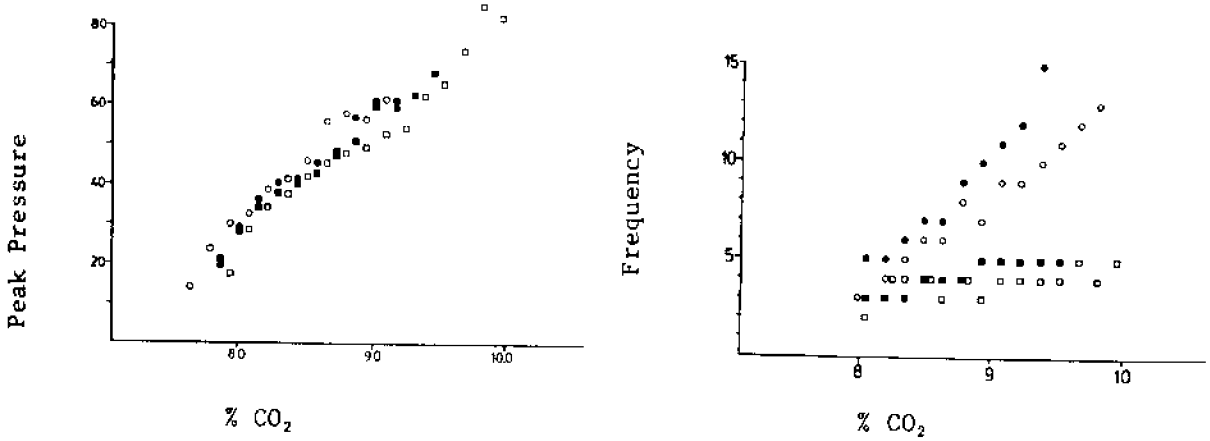


Starting from a low P_{CO2} (solid circles) peak negative pressure of the contractions rose, quickly at first, then more slowly to breakpoint. Starting from a higher P_{CO2} (open circles and split circles) pressure rose quickly, then slowly again, but at the breakpoint had reached a higher value, at a higher P_{CO2} (and at a higher frequency, not shown) than in the previous breathhold. In an experiment like Fowler's, where the subject was permitted to take a few breaths from the bag at intervals, the magnitude and frequency of contractions dropped immediately each time he did so, then rose rapidly again to a high level (Fig. 4).



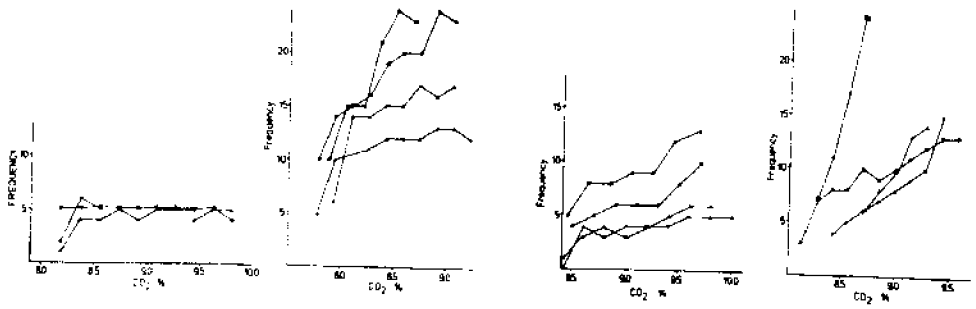
These experiments show that the breakpoint is not set by a certain threshold magnitude or frequency of contractions. If the contractions are directly responsible for the discomfort that determines the breakpoint, it must be in some complicated way, related to their time course before the breakpoint (11).

In another set of experiments (12), subjects held their breath at FRC while cool gas was circulated in through the nose and out the mouthpiece at rates comparable to normal inspired flow. This had a marked effect on the frequency and inspiratory time of contractions without changing their amplitude (Fig. 5). The left panel shows peak inspiratory pressure, in cm H_2O against CO_2%. The right panel shows frequency of the contractions as number per 10 seconds. Different symbols are for different runs, two with cool air flowing in the nasal-oral circuit (squares) and two with no flow (circles).



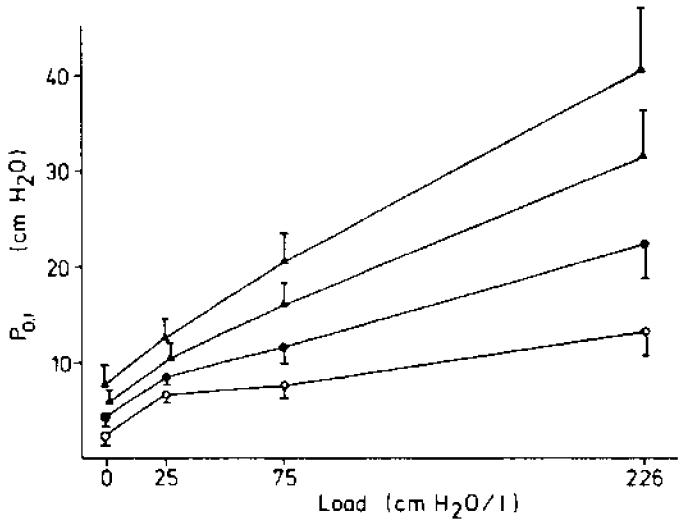
The inhibition was greater with lower temperature of the gas and depended on the phase of respiration, being negligible if the flow was applied only in the expiratory phase of contractions. This suggested that part of the nonchemical stimulus to respiration in breathholding might arise from deprivation of an inhibitory influence due to flow or temperature receptors in the upper airway. As in other experiments, a decrease in the contractions, in this case in their frequency, was associated with prolongation of breathhold time.

Breathholding at volumes above FRC is known to be easier, with longer times to breakpoint. Subjects with gastric and pleural balloons holding their breath at residual volume (RV)FRC, total lung capacity (TLC) and a volume intermediate between the last two were found to have the highest frequency of contractions at RV and the lowest at TLC (Fig. 6). In this figure frequency of contractions during breathholds at four volumes is plotted. Solid triangles TLC, open triangles about 1 litre above FRC, solid circles FRC, open circles RV.



The pressure generated by contractions was also lower at TLC, but the decline in inspiratory pressure with volume could be accounted for by mechanical factors (force-length and geometry, which make inspiratory muscles ineffective as pressure generators at high volume).

Seeking an explanation of the very high pressures and frequencies of inspiratory muscle contractions in breathholding, we considered the similarity between this pattern of respiratory muscle activity and that observed in subjects obliged to breathe against elatic loads. A set of experiments explored this possibility by applying large elastic loads during rebreathing and comparing the response to breathholding. The pressure and frequency of contractions in breathholding could be predicted as an extrapolation of the data from elastic loads, breathholding being considered as an elastic load in which respiratory muscles generate a small tidal volume by decompressing thoracic gas (Fig. 7).



Rate of rise of occlusion pressure (P0.1) is plotted against load in unloaded breathing, with elastic loads of 25 cm H_2O/L and 75 cm H_2O/L breathholding (where the load is calculated according to Boyle's Law). Each line joins points measured at iso-CO_2, the values of CO_2 running from 8% (lowest line) to 9.5% (highest line). Mechanisms responsible for the loading response (13) may thus explain the large pressures and frequencies.

Finally, we wondered if fatigue of inspiratory muscles, particularly the diaphragm, could be the principle that linked contractions with unbearable discomfort at the breakpoint. Such a hypothesis might explain data like that of Figure 3, where long bursts of intermediate level activity or short bursts of high intensity activity were equally able to bring a subject to the breakpoint. Analysis of the pressures and wave forms generated showed that pressure and timing of the contracitons were in the range that can cause eventual diaphragm fatigue (14). Diaphragm EMG obtained in some subjects showed spectral shifts (high/low ratio, 15) associated with fatigue. After repeated breathholds, a decrease in maximal

transdiaphragmatic pressure was noted. It thus seems possible that contractions of the magnitude seen in breathholding are capable of eventually causing diaphragm fatigue, and that the intolerance of longer breathholds is related to the need to avoid fatigue or to afferent information detecting incipient fatigue.

In summary, breathholding studies in the laboratory find a strong association between the number, frequency and itnensity of involuntary inspiratory muscle contractions and the breakpoint. The relation is not a simple one, but the principle of avoiding diaphragm fatigue is one plausible link. Unexpected stimuli, the example being cold in the upper airway may have an effect on the contractions and prolong breathholding, as can other maneuvres known to delay the breakpoint.

The application of this kind of experiment to breathhold diving is not certain. It is not known whether experienced divers suppress the contractions or whether they occur and constitute an important urge to return to the surface. Occasional normal subjects are able to suppress contractions for a long time, but most find this very difficult and can hold their breath much longer by sealing the upper airway while contractions occur. Our experiments were conducted after oxygen breathing. Breathholds on air waould likely produce more vigorous contractions, and hypoxia increases diaphragm vulnerability to fatigue (16). If contractions of this nature are important, such things as this time course of gas tensions in a dive, and the ambient water temperature could have an important influence on the urge to breathe and the tolerable duration of the dive.

REFERENCES

1. Agostoni, E. Diaphragm activity during breathholding: factors related to its onset. J. Appl. Physiol. 18:30-36, 1963.
2. Lin, Y.C., D.A. Lally, T.O. Moore and S.K. Hong. Physiological and conventional breathhold breaking points. J. Appl. Physiol. 37:291-296, 1974.
3. Hill, L., and M. Flack. The effect of excess of carbon dioxide and of want of oxygen upon the respiration and the circulation. J. Appl. Physiol. London 37:77-111, 1908.
4. Fowler, W.S. Breaking point of breathholding. J. Appl. Physiol. 6:539-545, 1954.
5. Campbell, E.J.M., S. Freeman, T.J.H. Clark, J.G. Robson and J. Norman. The effect of muscular paralysis induced by tubocurarine on the duration and sensation of breathholding. Clin. Sci. 32:425-432, 1967.
6. Godfrey, S. and E.J.M. Campbell. Mechanical and chemical control of breathholding. Q.J. Exp. Physiol. 54:117-128, 1969.
7. Godfrey, S. and E.J.M. Campbell. The control of breathholding. Respir. Physiol. 5:385-400, 1968.
8. Read, D.J.C. A clinical method for assessing the ventilatory response

to carbon dioxide. Australas. Ann. Med. 16:20-32, 1967.

9. Whitelaw, W.A., J.P. Derenne and J. Milic-Emili. Occlusion pressure as a measure of respiratory centre output in conscious man. Respir. Physiol. 14:181-199, 1975.

10. Mithoeffer, J.C. Mechanisms of pulmonary gas exchange and CO_2 transport during breathholding. J. Appl Physiol. 14:706-710, 1910.

11. Whitelaw, W.A., B. McBride, J. Amar and K. Corbet. Respiratory neuromuscular output during breathholding. J. Appl. Physiol. 50:435-443, 1981.

12. B. McBride and W.A. Whitelaw. A physiological stimulus to upper airway receptors in humans. J. Appl. Physiol. 51:1189-1197, 1981.

13. Cherniack, N.S. and M.D. Altose. Respiratory responses to ventilatory loading. In: Regulation of Breathing, Part II. (ed.) T.F. Hornbein, Marcel Dekker, 1981.

14. Bellemare, F. and A. Grassino. Effect of pressure and timing of contraction on human diaphragm fatigue. J. Appl. Physiol. 53:1190-1195, 1982.

15. Gross, D. A. Grassino, W.R.D. Ross et al. Electromyogram pattern of diaphragmatic fatigue. J. Appl. Physiol. 46:1, 1979.

16. Roussos, C.S. and P.T. Macklem. Respiratory muscle fatigue: a cause of respiratory failure? Clin. Sci. Mol. Med. 53:419, 1977.

Discussion following Dr. Whitelaw's presentation

Dr. Arnold: Are there any calculations of how much energy respiratory muscles consume while you're between the onset of involuntary diaphragmatic contractions and blackout?

Dr. Whitelaw: We haven't calculated that but you can make a guess at it because there are measurements now that relate the tension-time index to the oxygen consumption of respiratory muscles, the diaphragm, anyway, and these would certainly be consuming lots of oxygen. Fatigue is probably due to an imbalance between supply of oxygen and oxygen demand and it's aggravated when cardiac output is low and perfusion is less, when there's hypoxia, and when there's hypercapnia, and all of these things could actually be going on in a breath-hold as well. So it's not easy to see how they interact in detail.

Dr. Rennie: This may not be an appropriate time to ask this question, but with gastric pressure going up to plus 20 to 30 torr at the same time as the esophageal pressure is decreasing by 20 or 30 torr, all the conditions are set for a diaphragmatic circulatory pump that may augment somehow the cardiac pump under these conditions. Now, what do we know about the interrelationship between the diaphragm and the heart during breath-hold as an augmented circulatory pump?

Dr. Whitelaw: I don't know, don't know at all.

Dr. Rahn: You were asking about the work done by the diaphragm. Could that be calculated from the volume and pressure changes, rather than from extrapolating to tension of the diaphragm itself?

Dr. Whitelaw: I agree - I jumped to oxygen consumption and bypassed the work question, but sure you can calculate the work problem.

Dr. Rahn: Well, all you'd have to know is what the volume was at the pressure point.

Dr. Whitelaw: Well, only the pressure because the volume is calculated from the pressure swing and the starting volume (FRC).

Dr. Lundgren: Were you thinking of the expansion of the gas?

Dr. Rahn: Yes. Because that would be easy to do, wouldn't it? It could give you some idea of how much energy you are losing.

Dr. Craig: You're wasting a lot of energy.

Dr. Ferrigno: You mentioned the Hering-Breuer reflex. I was thinking about the difference between dry and wet breath-hold dives. How does immersion affect the Hering-Breuer reflex? Does it affect it through intrathoracic blood pooling?

Dr. Whitelaw: It could, but first of all immersion drops lung volume, so that would have an effect, and thoracic blood volume might have an effect on a vagal reflex, it would not be the Hering-Breuer reflex. I

don't really know the answer to this question at all - we thought we would do this experiment under water once just to have some data for the meeting, but we didn't manage to get it.

Dr. Arnold: I think the breaking point is very important to people who are going to dive more than once and enjoy it. I think another message from the last couple of papers is that people, who have been able to survive near drowning, have gone through an experiment testing the physiology and giving us all sorts of information. I'm sure the breath-holding time for a given individual is going to be shorter with lower temperature, but near drowning victims seem to survive better if the temperature is lower. To a certain extent, I think it's a benefit if a given individual goes under the ice water and goes unconscious sooner without going through a lot of this energy consumption and if they're able to do that without inhaling water, the heart rate is still maintained.

Dr. Lundgren: I have a comment that's related to the question of how we deal as breath-holders with these diaphragmatic movements and it is kind of anecdotal but some of you, I'm sure, have made the same observation. Those of you who have hyperventilated on oxygen and held your breath for a long time, may have noticed how there is a period of some air hunger, if you will, fairly early in the breath-hold and then it goes away. The question is: what is this adaptive mechanism? Another, and certainly anecdotal, piece of information that I want to offer you has to do with some experiments that we did a long time ago in Sweden, when we were interested in ventilation without respiratory movements. We used a Thunberg barospirator (Thunberg, T. Der Barospirator, ein neuer Apparat fur kunsliche Atmung. Skan. Arch. Physiol. 48:80-94, 1926), a steel cylinder in which you could place a subject and which was connected to a huge piston pump that oscillated back and forth and gave pressure swings in the barospirator. In that way you actually achieve pulmonary ventilation without respiratory movements. Now, we did this, and it was very hard to relax and not to breathe, despite the fact that our CO_2-meter indicated that we were, if anything, hyperventilated. With training, we could relax completely and we observed, objectively, lack of movement. There was a very distinct training effect and the reason I'm mentioning this is that, perhaps with training in breath-holding, you become less sensitive to the need for respiratory movements as a way of satisfying your respiratory regulation.

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Decrease in body insulation with exercise in cool water

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Park YS, Pendergast DR, Rennie DW. Decrease in body insulation with exercise in cool water. Undersea Biomed Res 1984; 11(2):159–168.—Steady-state body insulation was measured in 7 healthy male subjects during rest and exercise for 3 h in water of 28°C–32°C. At rest, maximal body insulation increased as a linear function of mean subcutaneous fat thickness by an amount approximately 4-fold what would be predicted from the physical insulation of fat alone. With arm plus leg exercise, body insulation declined as an exponential function of the exercise intensity, reaching approximately 25% of the resting value at work loads above $\dot{V}O_2$ = 1.2 liters · min $^{-1}$. During exercise the relationship between overall body insulation and mean subcutaneous fat thickness was almost identical to that predicted from fat insulation alone. These results suggest that 75% of maximal body insulation in resting subjects is achieved by use of skeletal muscle as an insulative barrier and that the muscle component is increased with increasing fat thickness. This muscle insulation shell is lost during exercise. As a practical consequence, heat generated by muscular exercise in water colder than critical water temperature cannot offset cooling unless the exercise intensity is great.

body insulation
water temperature
exercise
skeletal muscle

The indirect evidence seems quite good that the thickness of subcutaneous fat is only one of several factors slowing the rate of central body heat loss in cold water. There is general agreement that subcutaneous fat provides an insulative barrier to the loss of body heat during immersion in cool water (1–3). Carlson et al. (4), Keatinge (5), Sloan and Keatinge (6), Rennie (7), Burton and Edholm (8), and Pugh and Edholm (9) have shown for subjects resting in cold water that more obese persons with greater subcutaneous fat thickness cool at slower rates at the same water temperature. Second, the ratio of surface area to mass must be considered as a factor when comparing cooling rates of individuals having substantially different body shapes and sizes, e.g., men vs. women or adults vs. children (9, 10). For man in water a third factor, and possibly the most important, is the insulative shell beneath the skin and subcutaneous fat. Sloan and Keatinge (6) pointed out that lean subjects cool much less rapidly than would be predicted from their ratio of surface area to mass, "probably because deep tissues provided appreciable insulation to supplement that of skin and subcutaneous fat." Rennie et al. (11) reported that the linear regression of maximal body insulation on subcutaneous fat thickness,

when extrapolated to fat thickness of zero, yielded a positive intercept on the insulation axis; therefore they postulated the existence of an in-series insulative muscle shell based on the concepts of insulative cold acclimatization developed by Carlson et al. (4). Exercise in water cooler than 25°C has been shown to actually accelerate body cooling above rates at rest (5, 12), probably due to increased muscle perfusion and hence reduced insulation. However, there has been no quantitative assessment of the actual magnitude of muscle insulation during progressive increments of exercise, primarily because most studies have been done in water so cold as to preclude achievement of the thermal balance needed to calculate overall body insulation.

The current study represents an attempt to quantify the relative insulative value of subcutaneous fat vs. muscle during prolonged immersion in water of 28°C–32°C by measuring the steady-state body insulation during the last hour of a 3-h immersion of subjects at rest and during each of several levels of combined arm and leg exercise.

METHODS

To study the effects of long-term exercise on body insulation in water, 7 healthy male volunteers were used with a range of subcutaneous fat thickness from 1.4 to 7.4 mm. None of the subjects were in rigorous physical training. Their physical characteristics are summarized in Table 1.

Skinfold thickness was measured with a Lange caliper (Cambridge Scientific Inc., Cambridge, MD) at each of 10 positions (chin, cheek, back, chest, midlateral thorax, umbilicus, lateral waist, triceps, knee, and calf), and the weighted mean subcutaneous fat thickness (SFT) was estimated after correction for 4-mm double-skinfold thickness for each position (11). Percentage of total body fat was estimated from the relationship between skinfold thickness and adiposity as described by Allen et al. (13).

Establishment of critical water temperature. Critical water temperature (T_{cw}), defined as the lowest temperature a resting individual can tolerate for 3 h without shivering, was determined for each subject in a series of preliminary experiments by immersing them to the chin in vigorously stirred water ($v = 0.25 \text{ m} \cdot \text{s}^{-1}$) as previously described (11). Subjects were clothed in swim suits and seated motionless for up to 3 h. Water temperature was regulated to within 0.05°C at each selected temperature. The $\dot{V}O_2$ was measured directly at 20-min intervals using a closed-circuit 13L Collins spirometer (Warren E. Collins, Inc., Braintree, MA). If the three final readings exceeded the initial hour's $\dot{V}O_2$ by more than 10%, the water temperature was considered too cold and was raised 1°C for the subsequent test. Usually 2 but occasionally 3 immersions were necessary. Table 1 summarizes the T_{cw} of the subjects.

Exercise in water of critical temperature. A modified Collins bicycle ergometer was mounted over a water tank 3 m long, 1.5 m wide, and 3 m deep. Subjects were seated comfortably on the ergometer seat with legs extended horizontally to the foot pedals. A rope with hand loops passed through an adjustable friction ring mounted on the ergometer frame so that active flexion of first one arm and then the other could be accomplished over the full range of arm flexion in synchrony with leg pedaling. After the subject was strapped comfortably to the seat the entire frame was lowered into the water to the level of the subject's chin. An exercise intensity that could be continued for 3 h was established empirically for each subject by modifying the frequency of simultaneous cycling and arm flexion and the drag. The $\dot{V}O_2$ was

TABLE 1

PHYSICAL CHARACTERISTICS OF SUBJECTS

Subject symbol	Age, yr	Ht, cm	Wt, kg	LBM, kg	BSA, m_2	SA/Mass, m^2/kg	SFT, mm	Body Fat, %	T_{cw}, °C	I_{max}, °C/kcal · m^{-2} · h^{-1}
AV (•)	34	179	53	47	1.67	0.032	1.4	10.4	32	0.110
GVL (□)	18	175	66.7	60	1.8	0.027	1.5	10.6	32	0.110
YP (•)	38	160	50	42	1.5	0.03	3.7	16.8	30	0.150
AF (○)	21	168	70	58	1.78	0.025	4.1	17.6	30	0.150
GK (▲)	34	184	88.4	72	2.1	0.024	4.6	18.7	29	0.175
BD (△)	20	188	86.2	68	2.1	0.024	5.6	20.7	29	0.190
MG (×)	23	180	91	79	2.0	0.022	7.4	23.8	28	0.235

LBM, lean body mass; BSA, body surface area; SA/Mass, ratio of surface area to mass; SFT, subcutaneous fat thickness; T_{cw}, critical water temperature at rest; I_{max}, maximum insulation.

measured at 20-min intervals and the level of exercise was expressed in terms of kcal · m $^{-2}$ · h $^{-1}$, where 1 kcal = 0.207 liters O_2 (STPD).

Calculation of body insulation. To calculate overall body insulation (I) the following equation was used (11):

$$I\ (°C/(kcal \cdot m^{-2} \cdot h^{-1}) = (T_{re} - T_w)/(0.92\ \dot{M} \pm S) \quad (1)$$

where T_{re} is rectal temperature measured by thermistor at a depth of 20 cm, T_w is water temperature 20 cm from the subject, $\dot{M}$ is metabolic heat production in kcal · m $^{-2}$ · h $^{-1}$, and S is the loss or gain of body heat stores (ΔT_{re} · 0.83 · 0.6 · body wt) during the final hour of immersion, in kcal · m^{-2} · h^{-1}. Respiratory heat loss was assumed to be 0.08 $\dot{M}$ at rest and during exercise and was subtracted from $\dot{M}$ to give overall skin heat flux of 0.92 $\dot{M} \pm S$.

RESULTS

Figure 1 summarizes the time course of T_{re} (top) and $\dot{M}$ (bottom) for the 7 subjects at rest in critical water temperature and while performing leg and arm exercise at $\dot{M}$ = 73 (Exercise 1) and $\dot{M}$ = 120 (Exercise 2) kcal · m^{-2} · h^{-1} at this same water temperature. The T_{re} of the resting subjects dropped exponentially over 3 h from an initial value of 37.35°C ± 0.14 (SE) to 36.68°C ± 0.08. A similar change in T_{re} was observed during light exercise (Exercise 1). During moderate exercise (Exercise 2) there was no significant change in T_{re}: 37.30°C ± 0.07 at rest vs. 37.25°C ± 0.10 after exercise. The value of $\dot{M}$ rapidly rose during exercise (bottom of Fig. 1) and remained constant for the entire 3 h of immersion, thus assuring a steady state of heat flux.

Table 2 summarizes the average steady-state value for core to skin temperature difference ($T_{re} - T_w$), metabolic rate ($\dot{M}$), and body insulation (I) at rest and during exercise in the 3rd h of immersion. Since the $T_{re} - T_w$ gradient was increased at most from 6.48°C ± 0.85°C at rest to 7.08°C ± 0.89°C during exercise level 2, the overall body insulation decreased on the average from 0.151°C + 0.029°C/kcal · m^{-2} · h^{-1}) at rest to 0.064 ± 0.008 during Exercise 2 (see Table 2).

Figure 2 depicts the value of overall body insulation as a function of exercise level in 7 subjects. Each point depicts the value for I_{tot} and $\dot{M}$ measured during the final hour of the 3-h exercise, when a new steady state was established. The most obese subject, MG (•), had the largest overall insulation at rest, I = 0.23°C/(kcal · m^{-2} · h^{-1}), and underwent the greatest decline to a value of 0.073 at an exercise level of $\Delta\dot{M}$ = 100 kcal · m^{-2} · h^{-1}. Subjects GVL (□) and AV (■) had the smallest overall insulation at rest, 0.11°C/(kcal · m^{-2} · h^{-1}) and underwent the least decline to a value of 0.04 at $\Delta\dot{M}$ = 100 kcal ·m^{-2} · h^{-1}. The proportionate decrease in I was virtually the same in each subject, however.

Figure 3 depicts each steady-state value of overall body insulation as a function of subcutaneous fat thickness (SFT) for all subjects at various levels of exercise ($\Delta\dot{M}$ = 0 at rest, 25, 50, and 100 kcal · m^{-2} · h^{-1}), estimated from Fig. 2. A series of linear regressions of I on SFT was observed with the resting subjects having a slope almost 4-fold that measured for in vitro fat (1), or in vivo fat of subjects in T_{cw} (2). The slope of the regressions and their intercepts decreased progressively as exercise increased until at $\Delta\dot{M}$ = 100 kcal · m^{-2} · h^{-1} the slope was not significantly different from that for unperfused fat, depicted as the dashed line in Fig. 3.

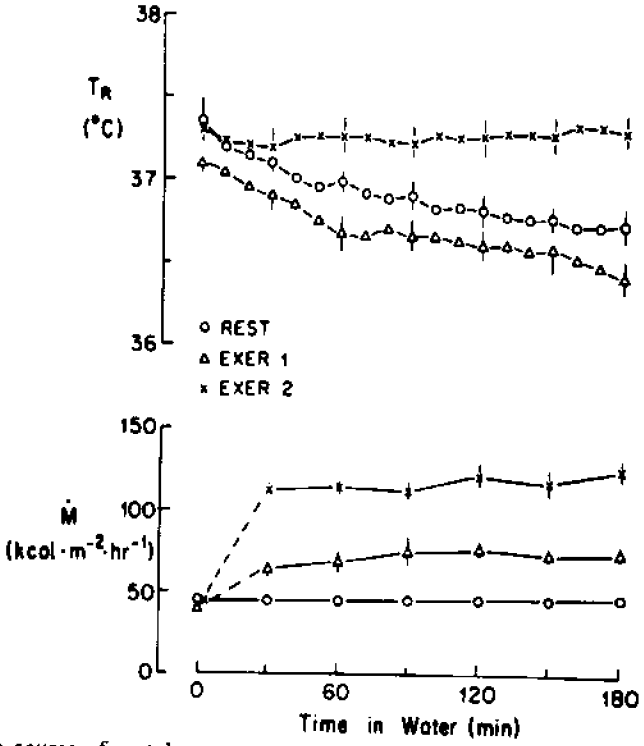

Fig. 1. Time course of rectal temperature, T_{re}, *(top)* and metabolic rate, $\dot{M}$ *(bottom)* during 3 h of immersion in water of critical temperature (28°C–32°C). Values are mean ± SE for 7 subjects at rest (o) and while performing combined arm and leg exercise at $\dot{M}$ = 75 kcal · m^{-2} · h^{-1} (Exercise 1, Δ) and at $\dot{M}$ = 125 kcal · m^{-2} · h^{-1} (Exercise 2, ×).

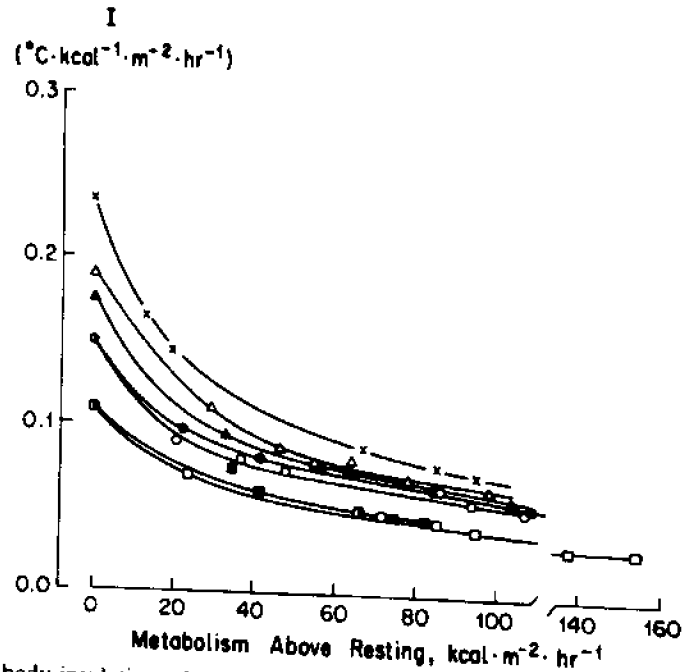

Fig. 2. Overall body insulations, I, of 7 subjects, measured during final hour of a 3-h immersion in water of critical temperature (28°C–32°C) and plotted as function of metabolism above resting, $\Delta\dot{M}$. The I values of resting subjects are shown at the *far left* where $\dot{M}$ = 0. Each subject was studied during 3-h of exercise on 4 or more occasions, and the I values for each subject are connected by a curvilinear line drawn by eye.

TABLE 2
$T_{re} - T_{cw}$, METABOLIC RATE, AND BODY INSULATION DURING 3RD H OF IMMERSION

	At Rest	During Exercise*	
		1	2
$T_{re} - T_{cw}$ (°C)	6.48 ± 0.85	6.37 ± 0.75	7.08 ± 0.89
$\dot{M}$ (kcal · m^{-2} · h^{-1})	47 ± 3.59	72.04 ± 2.43	123 ± 6.25
S** (kcal · m^{-2} · h^{-1})	−3.6 ± 0.76	−6.5 ± 3.0	1.47 ± 3.51
$I = \frac{T_{re} - T_{cw}}{0.92\,\dot{M} \pm S}$ (°C/(kcal · m^{-2} · h^{-1})	0.151 ± 0.029	0.095 ± 0.011	0.064 ± 0.008

*Exercise 1: $\Delta\dot{M}$ = 25 kcal · m^{-2} · h^{-1}. Exercise 2: $\Delta\dot{M}$ = 76. **Change in body heat storage.

DISCUSSION

For many years investigators have intimated that factors in addition to subcutaneous fat thickness must determine body cooling rates or body insulation in water (2–7, 10, 11). The present study focuses on the hypothesis that skeletal muscle acts as an insulative barrier in resting subjects. Although we claim no originality for this concept, which dates back at least to Carlson et al. (4), the design of this study does provide for the first time a quantitative estimate of the importance of muscle, and hopefully it will be a stimulus for basic studies on the circulation of skeletal muscle in immersion hypothermia.

The results depicted in Figs. 2 and 3 indicate that exercise progressively diminished overall body insulation to values that can be accounted for by unperfused subcutaneous fat and skin alone (2). Figure 4, which is derived from data in Fig. 2, depicts the decrease of body insulation for each individual exercise period as a percentage of its value at rest. On the average, the insulation decreased to approximately 50% of I_{max} at $\Delta\dot{M}$ = 50 kcal · m^{-2} · h^{-1}, to 35% at $\Delta\dot{M}$ = 100, and to 27% at $\Delta\dot{M}$ = 150. Thus skeletal muscle appears to provide as much as 75% of total body insulation in subjects resting in water of critical temperature, with subcutaneous fat and skin accounting for the remainder (see Fig. 4). A similar conclusion has been drawn in another study (2), in which superficial shell (fat and skin) insulation was estimated from direct measurements of fat and skin temperatures and skin heat flux. This suggests that for subjects immersed in moderately cold water, heat loss is controlled largely by blood flow to skeletal muscle with unperfused subcutaneous fat and skin providing a less important role than commonly supposed.

Measurements of skeletal muscle blood flow in different parts of the body during immersion have not been reported. However, resting blood flow in the entire forearm is reduced to only 10% of control when the temperature of water immersion changes from 35°C to 30°C (14, 15). It therefore seems likely that the blood vessels of muscle constrict in response to whole-body immersion in water that feels tepid, thereby providing an insulative layer underneath the superficial tissues. The signal for this severe vasoconstriction deserves to be an object of study, since neither central nor skin temperatures are greatly different from these during comfortable exposure in air. This additional insulation would naturally be removed at least in part by exercise hyperemia.

In the present study, as well as in other studies (7, 16, 17), the maximal body insulation attained during rest at critical water temperature was linearly correlated with the subcutaneous

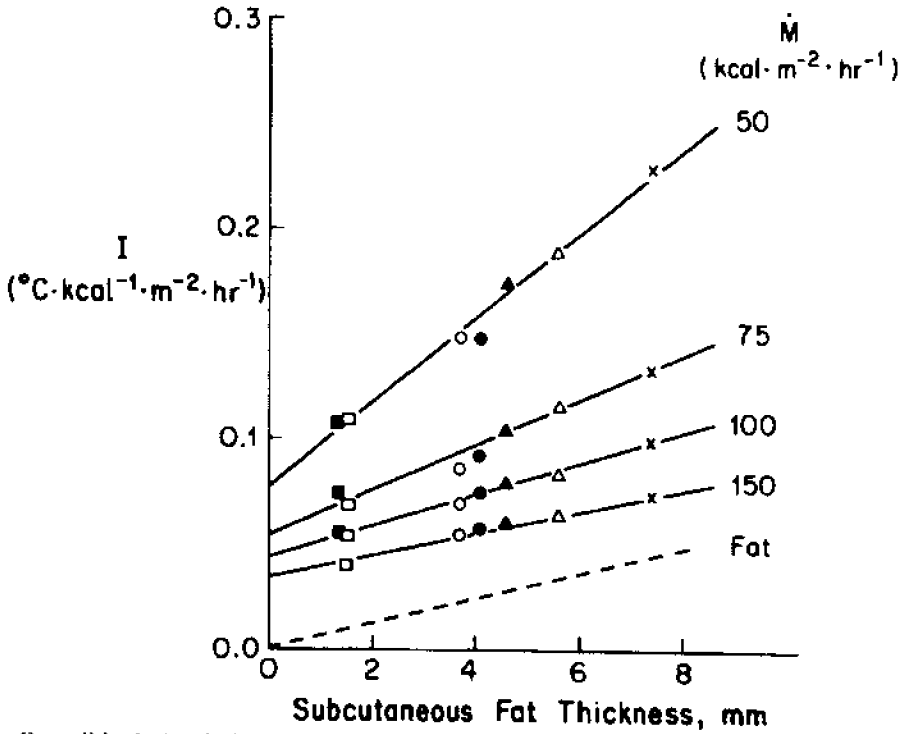

Fig. 3. Overall body insulation, I, plotted as a function of subcutaneous fat thickness in 7 subjects. Maximal I values during 3rd h of immersion in water of critical temperature are depicted by the *top line*, M = 50 kcal · m^{-2} · h^{-1}. Each of the *lower solid lines* depicts I during 3rd h of exercise at 75, 100, and 150 kcal · m^{-2} · h^{-1}. *Dashed line* depicts physical insulation of fat alone, i.e., 0.058°C/(kcal · m^{-2} · h^{-1}). See Refs. 1 and 2.

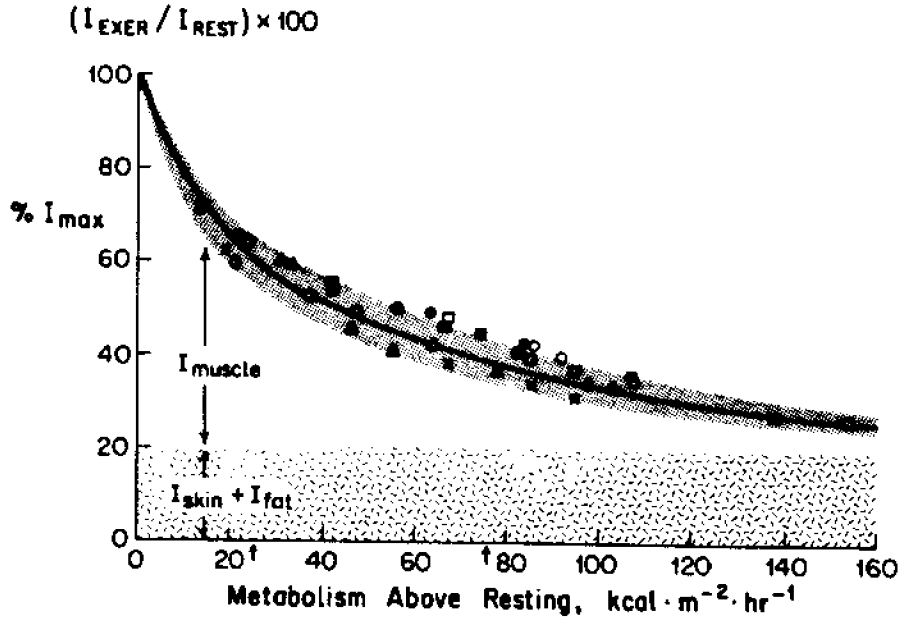

Fig. 4. Percentage of maximal insulation at rest, % I_{max}, is plotted for all 7 subjects as a function of metabolism above resting. At ΔM = 50 kcal · m^{-2} · h^{-1}, % I_{max} = 48. The asymptote for % I_{max} approaches 25% when ΔM exceeds 150 kcal · m^{-2} h^{-1}.

fat thickness with a slope 3- to 4-fold greater than that for fat alone (1, 2). This augmentation of overall insulation suggests that the insulative layer of muscle increases in persons with greater fat thickness. A schematic representation of this is depicted in Fig. 5 for subjects resting at T_{cw}. Since the insulations of skin and fat layers remain maximal at water temperatures below 32°C (2), the insulation due to skin would be independent of the obesity of the subject, whereas the insulation due to fat would increase in direct proportion to the thickness of the fat layer. Assuming a value of 0.03°C/(kcal · m^{-2} · h^{-1} · cm^{-1}) for the insulation of unperfused muscle (1), the equivalent thickness of skeletal muscle needed to account for the slope of I versus fat thickness in Fig. 5 would be 32 mm in subjects with 2 mm of fat thickness and 60 mm for subjects with 8 mm of fat thickness (Fig. 5 *inset*). This suggests that muscle blood flow is more severely restricted in fat subjects than in thin subjects in water of critical temperature. The critical water temperature, and hence the skin temperature during immersion, decreases as the skinfold thickness increases (Table 1 and Refs. 7, 11). One hypothesis to explain the increased muscle vasoconstriction, therefore, is that the activity of cutaneous cold receptors may be greater in fat subjects than in thin subjects in T_{cw}, and this may cause more complete muscle vasoconstriction in an obese person. An additional factor, not mutually exclusive of the above-proposed mechanism, is the larger and lean body mass of the more obese subjects in this study (see Table 1). The same intensity of muscle vasoconstrictor tone could lead to a larger mass of unperfused muscle in the obese subjects. Regardless of interpretation, it is apparent that subcutaneous fat retards body heat loss in water of critical temperatures not only due to the physical insulation of fat itself but also due to its amplification of body insulation brought about by an increased thickness of the muscle shell.

Exercise-induced decreases in body insulation, as observed in the present study, have some practical consequences. In Fig. 6 the hypothetical relationship between critical water temperature and metabolic heat production is depicted for subjects having overall body insulations of 0.025°C, 0.10°C, and 0.20°C/(kcal · m^{-2} · h^{-1}) (the series of straight lines intercepting at 36°C on the temperature axis). This relationship is calculated by the equation: $T_{cw} = T_{re} - 0.92\,\dot{M} \cdot I$ (a rearrangement of Eq. *1*) where 36°C is the minimal steady-state T_{re} chosen for effective performance of exercise (3). If one uses a person's resting maximal I value for predictive purposes, it is clear that an increase in $\dot{M}$ will predict a decrease in the hypothetical water temperature of immersion without incurring a drop in T_{re} below 36°C. The higher the I value, the lower will be the hypothetical water temperature for a given $\dot{M}$.

Also plotted in Fig. 6 are relationships actually observed between $\dot{M}$ and T_{cw} for two subjects: GVL (▫) of this study and JZ in the study of Pugh and Edholm (9). GVL had a resting I of 0.11°C/(kcal · m^{-2} · h^{-1}) in T_{cw} of 32°C. However, with progressive increases in $\dot{M}$, his I value decreased to 0.025 as an asymptote (see Fig. 2). Thus, there would be no drop in T_{cw} until $\dot{M}$ exceeded 200 kcal · m^{-2} · h^{-1}. Subject JZ (*Z* in Fig. 6) had an overall body insulation that we calculated to have been 0.17°C/(kcal · m^{-2} · h^{-1}) when "resting" in 17°C water with $\dot{M}$ = 134 kcal · m^{-2} · h^{-1}. Presumably, his T_{cw} at rest in the conditions of the present study ($\dot{M}$ = 50 kcal · m^{-2} · h^{-1}) would have been almost the same. We calculate his overall body insulation dropped to 0.07°C/(kcal · m^{-2} · h^{-1}) when exercising for long periods in water of 16°C, as shown in Fig. 6. We can only speculate that at still higher levels of $\dot{M}$, the T_{cw} for JZ would have decreased along the hypothetical linear relation for I = 0.07 as depicted in Fig. 6.

In practice, therefore, one must allow for the predictable decrease in I (shown in Fig. 4) when estimating the coldest water temperature for long-term muscular exercise. Presumably, this principle will apply as well to man in wet suits, but that will be an object for further study.

We would like to thank the 7 subjects who willingly participated in this rather rigorous study and Mr. Donald Wilson for his expert technical services. This work is supported by HL-14414 and HL-28542.—*Manuscript received for publication February 1984.*

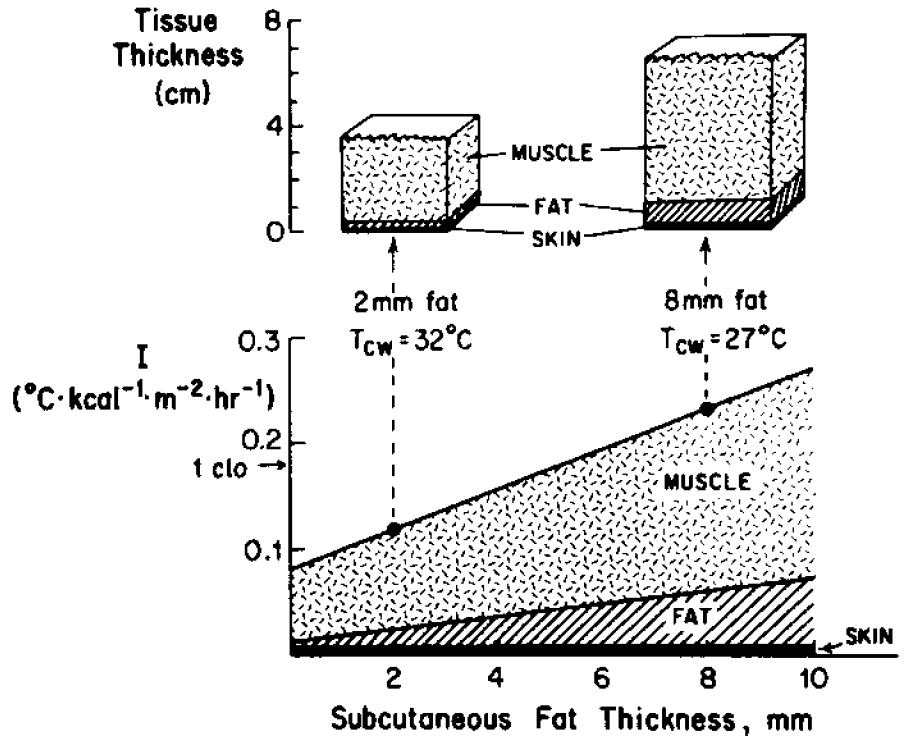

Fig. 5. Schematic representation of the shell thickness *(top)* of unperfused skin, fat, and skeletal muscle that would account for the observed relation between maximal insulation at rest, I_{max}, and subcutaneous fat thickness *(bottom)*. For a subject with mean fat thickness of 2 mm the relative thickness of skin, fat, and muscle would be 2, 2, and 32 mm, respectively, based on thermal conductivity of dead tissue. For a fat thickness of 8 mm, the respective tissue thicknesses would be 2, 8, and 58 mm. See Ref. 1.

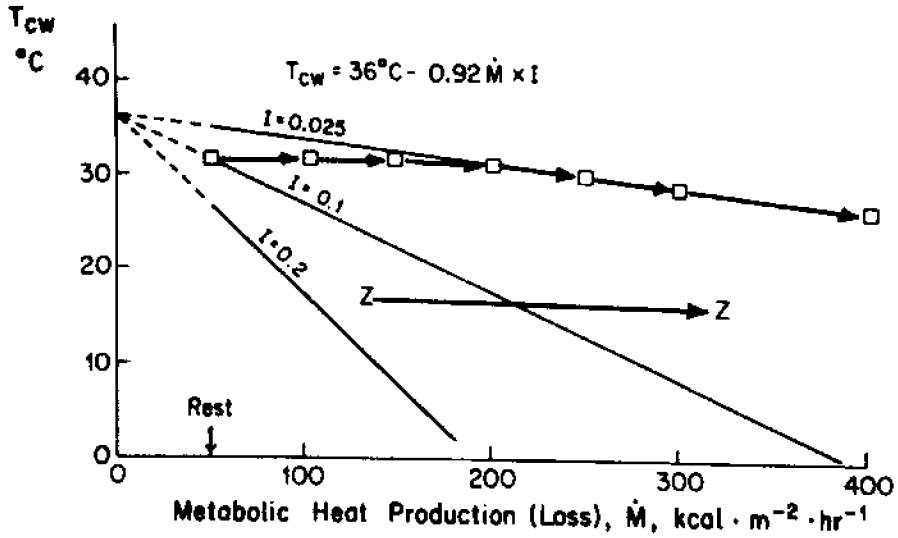

Fig. 6. Relationship between critical water temperature, T_{cw} (ordinate), and metablic heat production, $\dot{M}$ (abscissa). Straight-line isopleths are hypothetical relationships based on the equation $T_w = T_{re} - 0.92\ \dot{M} \cdot I$ for I values of 0.025, 0.10, and 0.20°C/(kcal · m^{-2} · h^{-1}) and for a T_{re} = 36°C, which was selected as the lowest rectal temperature for effective performance (3). Values for subject GVL (□) from this study and JZ from the study of Pugh and Edholm (9) indicate that T_{cw} cannot be predicted from this simplified equation until $\Delta\dot{M}$ exceeds 150–200 kcal · m^{-2} · h^{-1}. At lower levels of $\Delta\dot{M}$ the decrease in I offsets the increase in $\dot{M}$ so that no decrease in T_{cw} occurs.

Park YS, Pendergast DR, Rennie DW. Diminution de l'isolation corporelle durant l'exercice en eau froide. Undersea Biomed Res 1984; 11(2):159–168.—L'isolation corporelle à l'état stable fut mesurée pendant 3 h chez 7 sujets mâles en santé au repos et à l'exercice dans de l'eau à 28°C–32°C. Au repos, l'isolation corporelle maximale augmente linéairement en fonction de l'épaisseur moyenne du tissus adipeux sous-cutané d'une valeur d'approximativement 4 fois celle prédite par l'isolation physique de la graisse seule. Au cours de l'exercice avec les bras et les jambes, l'isolation corporelle diminua exponentiellement en fonction de l'intensité du travail, atteignant environ 25% de la valeur de repos aux charges de travail dépassant une $\dot{V}O_2$ = 1.2 litres · min $^{-1}$. Durant l'exercice, la relation entre l'isolation corporelle dans son ensemble et l'épaisseur moyenne du tissus adipeux sous-cutané était presque identique à celle prédite par l'isolation de la graisse seule. Ces résultats suggèrent que 75% de l'isolation corporelle maximale chez les sujets au repos est achevée par l'emploi des muscles squelettiques comme barrière isolante, et que la composante musculaire est augmentée par l'accroissement de l'épaisseur de graisse. Cette envelope d'isolation musculaire est perdue pendant l'exercice. Comme conséquence pratique, la chaleur produite par l'exercice musculaire dans de l'eau à une température supérieure à une valeur critique ne peut pas contrecarrer le refroidissement à moins d'effectuer un travail à une intensité plus grande.

isolation corporelle
muscle squelettique
température de l'eau
exercice

REFERENCES

1. Hatfield HS, Pugh LGC. Thermal conductivity of human fat and muscle. Nature 1951; 168:918–919.
2. Veicsteinas A, Ferretti G, Rennie DW. Superficial shell insulation in resting and exercising men in cold water. J Appl Physiol: Respir Environ Exercise Physiol 1982; 52:1557–1564.
3. Bullard RW, Rapp GM. Problems of body heat loss in water immersion. Aerosp Med 1970; 41:1269–1277.
4. Carlson LD, Hsieh ACL, Fullington F, Elsner RW. Immersion in cold water and body tissue insulation. J Aviat Med 1958; 29:145–152.
5. Keatinge WR. The effect of work and clothing on the maintenance of body temperature in water. Q J Exp Physiol 1961; 46:69–82.
6. Sloan REG, Keatinge WR. Cooling rates of young people swimming in cold water. J Appl Physiol 1973; 35:371–375.
7. Rennie DW. Thermal insulation of Korean diving women and non-divers in water. In: Rahn H, ed. Physiology of breath-hold diving and the Ama of Japan. Washington, DC: National Academy of Science, National Research Council, 1965:315–324.
8. Burton AC, Edholm OG. Man in a cold environment. London: Edward Arnold Publishing Ltd., 1955.
9. Pugh LGC, Edholm OG. The physiology of channel swimmers. Lancet 1955; October:761–767.
10. Kollias J, Bartlett L, Bergsteinova V, Skinner JS, Buskirk ER, Nicholas WC. Metabolic and thermal responses of women during cooling in water. J Appl Physiol 1974; 36:577–580.
11. Rennie DW, Covino BG, Howell BJ, Song SH, Kang BS, Hong SK. Physical insulation of Korean diving women. J Appl Physiol 1962; 17:961–966.
12. Cannon P, Keatinge WR. The metabolic rate and heat loss of fat and thin men in heat balance in cold and warm water. J Physiol 1960; 154:329–344.
13. Allen TH, Peng MT, Chon KP, Ruang TF, Chang C, Fang HS. Prediction of total adiposity from skin folds and curvilinear relationship between external and internal adiposity. Metabolism 1956; 5:346–352.
14. Rennie DW. Body heat loss during immersion in water. In: Yoshimara H, Weiner JS, eds. Human adaptability and its methodology. Tokyo: Japan Society for the Promotion of Sciences, 1966:36–41.
15. Bacroft H, Edholm OG. The effect of temperature on blood flow and deep temperature in the human forearm. J Physiol 1943; 102:5–20.
16. Hong SK. Pattern of cold adaptation in women divers of Korea (ama). Fed Proc 1973; 32:1614–1622.
17. Hanna JM, Hong SK. Critical water temperature and effective insulation in scuba divers in Hawaii. J Appl Physiol 1972; 33:770–773.

Discussion related to the article "Decrease in body insulation with exercise in cool water"

Dr. Arnold: Dr. Rennie, in a hypothermic patient who has been out of the water or otherwise, when one worries about rewarming the limbs quickly, the thought being that one ends up getting a dumping of very cold blood back from the limbs into the ventricles causing ventricular fibrillation, it may very well be that the temperature of the muscles and the periphery is lower than the rectal temperature and therefore your whole periphery seems to be a better insulator for your core.

Dr. Rennie: That's right. If in fact there is a severe sustained vasoconstriction of skeletal muscle as a consequence of cool water stress, then you would imagine there could be a progressive loss of stored heat from a poorly perfused limb, or lengthening of the thermopathway providing a progressively longer physical pathway for the conduction of heat through that muscle. You're quite right, that muscle would cool below the temperature of the central core providing a heat sink. In the case of rewarming you possibly do run the risk of cold blood going back into the core and still further contributing to the drop in core temperature. That's kind of a controversial question.

Dr. Elsner: It would seem to suggest from what you have done and from the earlier discussions that if you were in a survival situation and you are dumped into cold water, you may choose to try to swim to the shore. Your chances would improve if you are large and fat. However, if you're in a situation in which there is no hope of swimming to the shore, it would perhaps be better to be skinny and small and loose consciousness and depend upon CNS cooling until rescued. We have seen recent examples of this response in children immersed in cold water for periods up to 40 min.

Dr. Rahn: To continue this argument in another way, what would be the best procedure after falling overboard in the middle of the Atlantic?

Dr. Rennie: In the middle of the Atlantic? Stay with the boat, stay with your clothes, remain immobile, go to the fetal position.

Dr. Rahn: In other words, don't exercise

Dr. Rennie: Don't exercise. Now, if this is a skeletal muscle vasoconstriction problem and is cold induced, then it is still an open question what exercise hyperemia will do under those conditions. Will it break through the cold induced vasoconstriction completely, or only in part? And if it's only in part, then this could even affect your swimming performance. It would be like swimming with a partial tourniquet on, which is still another reason for remaining immobile, or at least not striking out for a distant shore at a high rate of speed.

Dr. Rahn: What about the breath-hold diver?

Dr. Rennie: Dr. Elsner has got data from years ago on the effects of breath-hold on human calf blood flow. I think he interpreted that as

vasoconstriction in the calf. I don't remember if it was in the muscle or not.

Dr. Elsner: In some subjects the flow shut right down so it has to be muscle ischemia as well as skin vasoconstriction.

Dr. Rahn: What about a real situation, for example, Dr. Hong's Ama in ten degrees water, diving daily for 15 minutes at the most.

Dr. Rennie: Well, the interpretation that I would have, of course, is that the functional insulation in the real world is so much less than the insulation studied under conditions of critical water temperature, that there is no comparison between the two whatsoever. Combined shivering and exercise so reduce the insulation, that it is immaterial whether they develop a slight increase in the insulation of the periphery in water of critical temperature. They may truly have developed that increase in insulation. But whether it was of survival value, practical value, I would say not anymore than an increase in basal metabolic rate could reasonably be argued to be a survival factor in the Eskimo.

Dr. Rahn: You know what the diving endurance is at 10 degrees C and at 25 degrees C in water. Can you on the basis of these time differences, and knowing the rectal temperatures, go back and reinterpret and predict the duration?

Dr. Rennie: Yes you can. Everything you gain by increasing metabolic heat production by exercise, you loose by the decrease in insulation, so that any increased metabolic heat produced by exercising has no effect on the minimal operational water temperature in which one works. That is, there is no effect at the levels of metabolic rate that they use for the 35-45 minutes that they are working, which is only about three times greater than rest.

Dr. Van Liew: Does still water act as an insulator?

Dr. Rennie: All our studies were done with vigorously stirred water, so that the additional stirring effect of exercise, for example, would be minimized.

Dr. Van Liew: But in the real world, the amount of stirring is an important complication.

Dr. Rennie: In the real world that's still a further complication, that's correct.

Dr. Lundgren: The amount of reduction in blood flow that you saw and that we saw, in the face immersion studies that I mentioned earlier, where the fall in forearm blood flow was 50%, has to involve muscle to a substantial degree, because in order to cut the blood flow in half for the whole limb, given the high proportion of muscle tissue relative to skin tissue, a lot of the reduction has to be taken out of muscle blood flow.

Dr. Elsner: It's very curious that in our studies some years ago one

of the subjects who showed extremely low calf blood flow during face immersion is a fellow who, characteristically, complained of leg cramps when he was surfing.

THERMOREGULATION IN WET SUIT DIVERS

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Maintenance of normal body temperature is a critical problem for divers working in cold water. Because of the high heat conductivity and heat capacity of water, body heat is rapidly lost from the skin to the adjacent layer of water. This drain of body heat is the principal problem of divers and, in fact, the dive time of unprotected divers is primarily determined by this loss of body heat (1,2).

As reported elsewhere (3) the thermal cost of diving in Korean women divers decreased remarkably since they adopted wet suits probably due to additional insulation provided by the suit. In breath-hold diving, divers repeat the cycle of a dive and surface recovery each lasting for 30 - 40 sec in the case of Korean women divers (4). During surface recovery, divers are resting in a head-out water immersion, whereas during dives they exercise and are exposed to various degrees of hydrostatic pressure. As discussed by Rennie in the preceeding paper, insulation of the human body undergoes a dramatic reduction during exercise in water. Moreover, physical insulation of the wet suit changes with pressure due to compression of trapped air (5). Therefore, overall thermal insulation (and hence heat balance) of breath-hold wet suit divers will be changed cyclically during the course of diving work.

In the present paper, we will first present data of heat exchanges obtained in Korean women wet suit divers during their natural diving work, and then describe effects of exercise and pressure on the thermal insulation of wet suit divers in cold water.

A. Thermal Balance During Breath-hold Diving

In 4 Korean women wet suit divers the heat exchange was studied while they were working in the sea (4 - 5 m depth) (3). In order to evaluate the effect of wet suits on the thermal balance, the subjects wore wet suits (5 mm thick neoprene jacket, pants, hood and boots) in one series and cotton suits (traditional diving suits of previous Korean women divers) in the other.

Fig. 1 compares the time course of rectal (T_R) and the mean skin (T_S) temperatures of wet suit and cotton suit divers during diving work in summer (22.5°C water) and in winter (10°C water). In cotton suit divers, the T_R declined to about 35°C in 30 min in winter and in 1 hr in summer, at which time divers did not want to continue diving work any longer. The $\overline{T}_S$ dropped

to 24°C in summer and 13°C in winter at the end of the work period; hence the reduction in mean body temperature ($T_B = 0.6\ T_R + 0.4\ \bar{T}_S$) was 8.4°C in winter and 6°C in summer. The loss of body heat content ($\Delta S = \Delta T_B$ x body wt x 0.83) was calculated to be 240 and 363 kcal in summer and winter, respectively. These results confirm the earlier notion that the most important factor determining the working time in unprotected divers is deep body cooling (1,2).

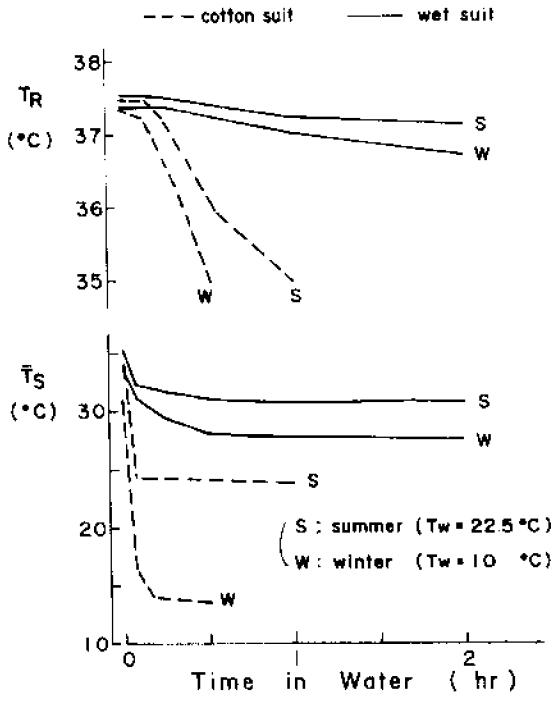

Fig. 1. Average time course of rectal (T_R) and mean skin ($\bar{T}_S$) temperatures of 4 women divers during wet suit and cotton suit divings in summer (S) and winter (W). Data based on Kang et al. (3).

During wet suit diving, divers did not experience such a hypothermia. The fall in T_R in 2 hr was only 0.4°C in summer and 0.6°C in winter. Thus T_R was of no major importance in the determination of work period. The $\bar{T}_S$ was also maintained at a level significantly higher than that in cotton suit divers, but it is important to notice that $\bar{T}_S$ in winter was still well below the comfortable range (30 - 33°C). Values of $\bar{T}_S$ and T_B at the end of 2 hr work period were 31 and 35°C in summer and 28 and 33°C in winter, respectively. The calculated body heat debt was 28 and 119 kcal in summer and winter, respectively.

Fig. 2 summarizes average hourly values of heat production ($\dot{M} = 4.83\ \dot{V}O_2$) and heat loss ($\dot{H} = \dot{M} \pm \Delta T_B$ x Body wt x 0.83) of 4 divers during the work shift. In summer, heat production and heat loss of wet suit divers were

approximately equal (about 85 kcal/m^2·hr), indicating that the subjects were in thermal steady-state. However, in cotton suit divers the heat production (105 kcal/m^2·hr) was only 60% effective in offsetting the heat loss (167 kcal/m^2·hr). The amount of excess heat production in cotton suit divers over that in wet suit divers was about 20 kcal/m^2·hr, which may represent shivering thermogenesis. It is evident that even this small amount of shivering accelerated heat loss more than heat production in unprotected divers, and that its elimination by wearing wet suits greatly improved the thermal economy of divers.

As expected, when water temperature decreased in winter, heat production (148 and 210 kcal/m^2·hr in cotton suit and wet suit divers, respectively) and heat loss (153 and 335 kcal/m^2·hr in cotton suit and wet suit divers, respectively) increased in both wet suit and cotton suit divers. However, as in summer, the heat loss was nearly (95%) compensated by the heat production in wet suit divers, but only 60% compensated in cotton suit divers.

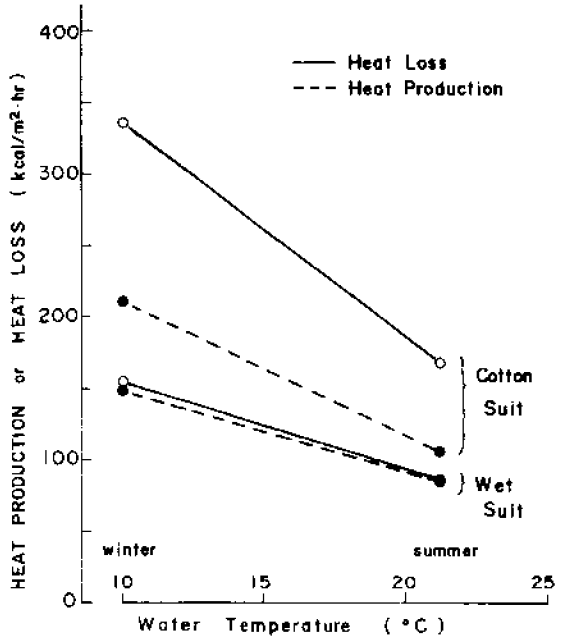

Fig. 2. Average heat production and heat loss of 4 women divers during wet suit and cotton suit divings in summer and winter. Data based on Kang et al. (3).

The estimated shivering thermogenesis in winter was 64 and 126 kcal/m^2·hr in wet suit and cotton suit divers, respectively. It is of interest to note that the degree of shivering was greater in wet suit divers in winter than in cotton suit divers in summer (64 vs 21 kcal/m^2·hr) although the T_R and $\bar{T}_S$ were higher in the former than in the latter (see Fig. 1). Since the present subjects (also other Korean women divers) did not wear protective gloves even in the cold season, we speculated that the

exposure of the hands to cold (10°C) water potentiated the shivering response in wet suit divers. In fact, Van Someren et al. (6) have observed that selective cooling of hands and feet stimulates heat production in subjects immersed in 29°C water. Regardless of the mechanism, the effectiveness of shivering in maintaining thermal balance in wet suit divers is clearly evident in Fig. 2.

Evidently, the reduction of heat loss by wearing wet suits was due to additional insulation provided by the suit. As illustrated in Fig. 3, the total insulation estimated from the rectal to water temperature difference and skin heat loss ($I_{total} = (T_R - T_W)/\dot{H}_S$) appeared to be 2.1 to 2.8 times greater in wet suit divers in summer and winter, respectively (0.170 vs 0.193°C/kcal/m^2•hr in summer vs winter) than in cotton suit divers (0.081 vs 0.068 in summer vs winter). However, the tissue insulation ($I_{body} = (T_R - \bar{T}_S)/\dot{H}_S$) was not significantly different between the two conditions (approximately 0.07°C/kcal/m^2•hr). Consequently, the additional insulation afforded by wet suits ($I_{suit} = I_{total} - I_{body}$) was 0.10 - 0.13°C/kcal/m^2•hr. Since the physical insulation of wet suits may vary with the depth of diving and the physiological insulation provided by wet suits decreases with exercise (see below), the values of suit insulation obtained in working breath-hold divers, as described above, may only represent the average insulation during diving work. In any event, such a value of extra insulation is equivalent to the insulation that can be provided by the 17 mm fat layer (0.1°C/kcal/m^2•hr÷0.006°C/kcal/m^2•hr per mm fat = 17 mm fat). Such an increase in subcutaneous fat insulation would reduce proportionately the heat loss of divers for a given temperature difference between the central body and water.

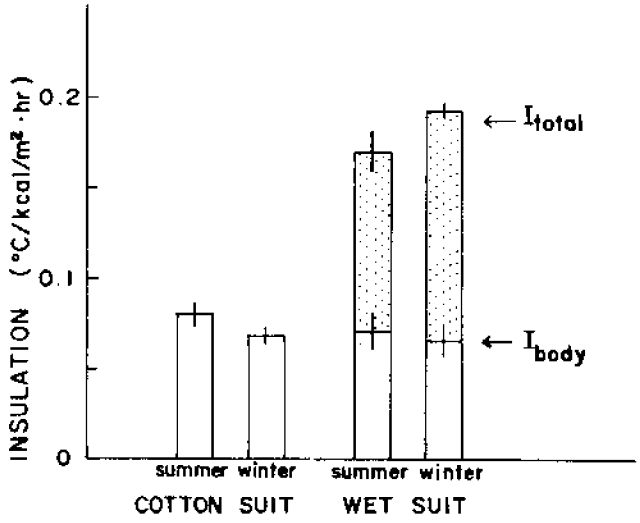

Fig. 3. Total and body insulations (I) during diving work in wet suit and cotton suit divers in summer and winter. The stippled portion of the bar represents the insulation provided by wet suits. Each value represents the mean (±SE) of 4 women divers. Data based on Kang et al. (3).

B. Effect of Exercise on Thermal Balance of Wet Suit Divers

It is well known that exercise in cold water increases heat loss more than heat production in an unprotected individual (7,8). In order to evaluate the effect of exercise on the thermal balance of wet suit divers we have measured heat exchanges in 4 Korean women wet suit divers at rest and during exercise in cold water. Subjects were clad in their personal wet suits (jacket, pants and boots of 5 - 6 mm thick) and were immersed up to the neck in a circulating water bath. The subject rested for 3 hr in a seated position or exercised for 2 hr at a constant intensity using a bicycle ergometer submersed in the bath. In order to prevent convective heat loss from under the suit (9) we taped the suit at the ankles and the wrists. For each subject the water temperature was adjusted to the critical temperature (Tcw, the lowest water temperature a resting subject could tolerate for 3 hr without shivering) in order to evaluate the thermal insulation at the maximal degree of peripheral vasoconstriction. The average Tcw with wet suits was 16.5 $\pm$ 1.2 (SE)^{o}C.

Fig. 4 depicts average skin heat loss ($\dot{H}_S$) and rectal temperature (T_R) over the final 1 hr at rest or during exercise. The exercise intensity was expressed as a percentage of the resting metabolic rate. The $\dot{H}_S$ at rest was on the average 45 kcal/m^2•hr, but it increased linearly with the exercise intensity. However, the T_R was not different whether the subjects were resting or were exercising at the various levels tested (less than 3.5 Met). These results indicate that thermal insulation decreased inversely with exercise intensity. As shown in Fig. 5, the calculated overall insulation

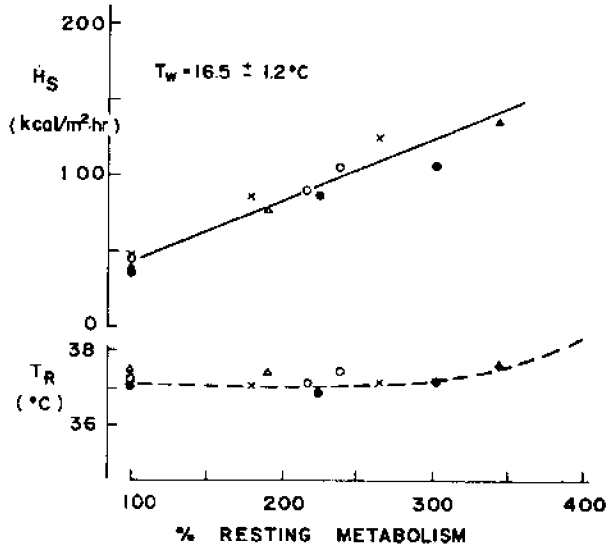

Fig. 4. Steady-state skin heat loss ($\dot{H}_S$) and rectal temperature(T_R) of 4 female wet suit divers as a function of exercise intensity (% resting metabolism) in water of critical temperature (T_{cw}). Each symbol represents an individual diver. Dotted line represents T_R calculated using the following formula: $T_R = T_W + (I_{total} \times 0.92M)$.

($I_{total} = (T_R - T_W)\dot{H}_S$) decreased from about 0.5°C/kcal/m^2•hr at rest to approximately one half at 2 Met and to one third of the resting value at 3 Met. This decrease in I_{total} appeared to be due in part to the reduction in body insulation (I_{body}) and in part to the decrease in insulation afforded by wet suits. The I_{body} declined exponentially from 0.23°C/kcal/m^2•hr at rest to 0.06 at 3 Met. The apparent suit insulation estimated from the difference between the total and body insulation ($I_{suit} = I_{total} - I_{body}$, stippled area) was on the average 0.27°C/kcal/m^2•hr at rest, but it decreased gradually with exercise intensity until it reached approximately 0.1°C/kcal/m^2•hr at above 3 Met. The latter value of I_{suit} is similar to the physical insulation of 5 mm neoprene wet suits obtained using a copper manikin (10). Since there was no apparent reason for change in thickness (and hence physical insulation) of wet suits between rest and exercise we speculate that the unexpectedly high functional insulation of wet suits in resting subjects is a consequence of physiological regulations in cold water.

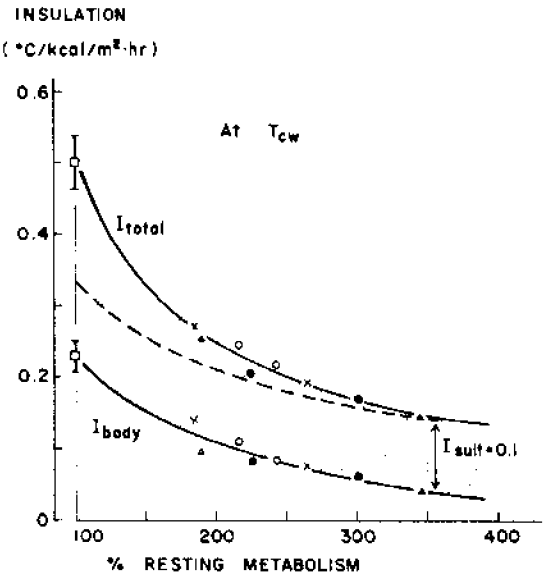

Fig. 5. Steady-State insulations (I) of 4 female wet suit divers as a function of exercise intensity in water of critical temperature. In each subject, values of I_{total} and I_{body} were normalized against the corresponding mean value at rest. Each symbol represents an individual diver. Stippled area represents the insulation afforded by wet suits (I_{suit}). Dashed line represents the I_{total} calculated by adding an I_{suit} value of 0.1 to I_{body} at various exercise levels.

When exposed to cold, the human body increases thermal insulation through peripheral vasoconstriction. As schematically illustrated in Fig. 6, this regulation of thermal insulation is mostly accomplished in the extremities and not in the trunk (8,11,12). Infrared thermography studies by Hayward et al.(13) indicated that the highest skin temperatures following

prolonged resting in water are lateral thorax, upper chest and groin. The constriction of limb blood vessels in cold water may occur also in wet suited subjects. Since the physical insulation of foamed neoprene will decrease as the curvature of surface increases (14), the insulative value of wet suits will be much smaller in the limb than in the trunk. Furthermore, the design of wet suits is such that most of the trunk surface is covered by double sheets (pants and jacket) and the limbs by a single sheet. Thus wet suits provide good insulation to the trunk surface, but poor insulation to the limbs. As a consequence, the skin temperature underneath the suit will become much lower in the extremities than in the trunk during immersion in cold water (31.3°C, chest vs 26°C, leg in the present study). In other

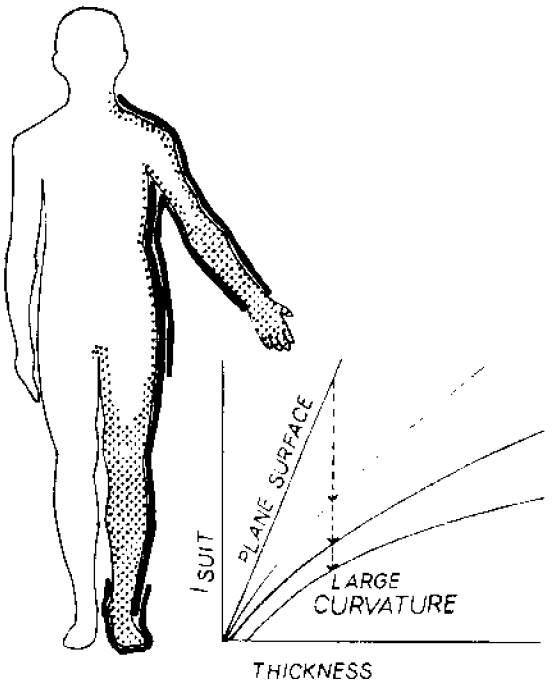

Fig. 6. A schematic illustration of the insulative shell (stippled area) of a wet suited subject in cold water. Note that the trunk is covered by double sheets (pants and jacket) and the limbs by a single sheet of the suit. Inset illustrates schematically changes in clothing insulation as a function of thickness and curvature (after Van Dilla et al. (13).

words, immersion with wet suits is analogous to exposing the limb to water colder than that exposing the trunk. This will lead to strong vasoconstriction in the extremities. Restriction of limb blood flow will greatly reduce the surface area for heat exchange and most of the heat exchange between the body core and water will take place at the trunk surface where suit insulation is relatively high. For these reasons, wet suits provide far greater physiological insulation at rest than during exercise. The exercise hyperemia reduces not only the thermal gradient from

the deep tissues to the skin but also thermal insulation down the length of the limb. Therefore, much of the heat produced in the skeletal muscle is dissipated through the large surface area of limbs rather than returning to the body core. Since the insulative value of wet suits is relatively low in the limbs because of its design (single sheet) and the high curvature, the effect of exercise is to increase the area for heat exchange over a poorly insulated region in parallel to the trunk. Added to this is increased convective heat loss from the escape of heated water at wrist, ankle and neck seals (9). Estimation of the heat exchange area ($A = I_{suit} \times 0.92\ \dot{M}/(T_S - T_W)$, $\dot{M}$ is metabolic rate in kcal/hr), assuming that I_{suit} is constant at $0.12^{\circ}C/kcal/m^2 \cdot hr$, indicates that the area at rest is only 0.66 m^2, which is equivalent to 40% of the total suit surface area (Fig. 7). The heat exchange area increases with exercise and becomes almost identical to the actual suit surface area at $\dot{M}$ = 200 kcal/hr. This analysis clearly indicates that the relatively high apparent suit insulation in the resting subject is due to reduced surface area for heat exchange.

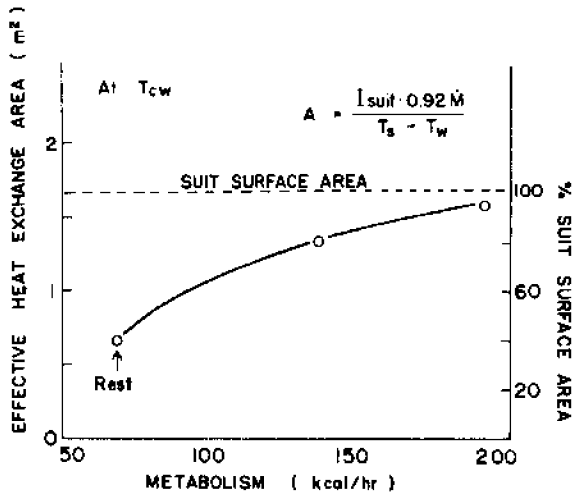

Fig. 7. The effect of heat exchange area of wet suits (A) at rest and at two levels of exercise. The formula for A is shown in the inset. The value of I_{suit} was set at $0.12^{\circ}C/kcal/m^2 \cdot hr$, which was observed for the 5 mm neoprene wet suit using an electrically heated manikin (10).

C. Effect of Pressure on the Thermal Insulation of Wet Suit Divers

Having established the pattern of thermal exchange in wet suit divers at the surface of the water, we next investigated the effect of hydrostatic pressure on the thermal balance of divers.

Three male wet suit divers were immersed in 15 - 16°C water in the wetpot of a hyperbaric chamber up to the neck. The chamber pressure was maintained at 1, 2 or 3 ATA air. Subjects rested for 3 hr or exercised for 2 hr using bicycle ergometer. The I_{total}, I_{body} and I_{suit} were estimated

as described above when the body temperature change was minimal during the final hour of immersion. The actual thickness of suit reduced from 5 mm at 1 ATA to 3.5 at 2 ATA and 2.6 at 3 ATA. Thus, the physical insulation of the suit might be decreased with pressure, and consequently skin surface underneath the suit was cooled more as the pressure increased. The $\overline{T}_S$ at rest was approximately 27°C at 1 ATA, 24.5°C at 2 ATA and 24.2°C at 3 ATA. Fig. 8 illustrates thermal insulations at rest and during exercise in water at 1, 2 and 3 ATA air. The degree of exercise was expressed as the metabolism above resting. At all pressures, insulations declined inversely with exercise intensity. The I_{total}, either at rest or during exercise, decreased as the pressure increased. On the other hand, I_{body} increased slightly at pressure than at the surface (1 ATA). Consequently, the difference between I_{total} and I_{body} (i.e., I_{suit}) decreased as pressure increased (inset). The relatively high I_{body} at 2 and 3 ATA as compared with 1 ATA may be attributed to more intensive peripheral vasoconstriction induced by the lower skin temperatures at pressure.

A practical implication of these findings is that if a wet suit diver is in a situation when escape from the cold water is not possible, he is better off to move to the surface and hold still than to swim if wasting of energy is to be prevented. Even with wet suits, exercise increases heat loss as much as heat production in cold water.

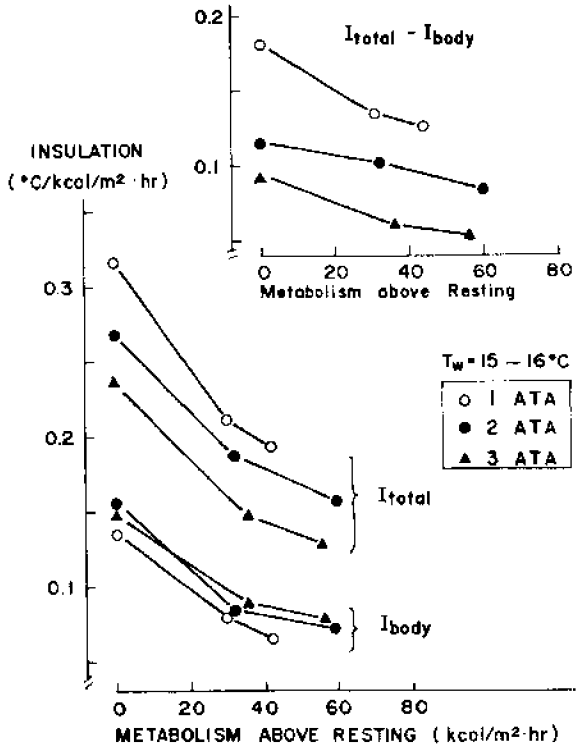

Fig. 8. Insulations (I) of wet suit divers at rest and during exercise in 15 - 16°C water at 1, 2 and 3 ATA air. Values represent the mean of 3 male divers.

D. Changes in Wet Suit Insulation with Surface to Dive Time Ratio in Breath-hold Diving

Since the functional insulation of wet suits changes with exercise and pressure, as described above, the protective effect of wet suits in a breath-hold diver will increase as the surface to dive time ratio increases. In fact, a preliminary observation in 2 Korean women divers in summer (23°C water) indicated that the apparent I_{suit} increased from 0.126 to 0.153°C/kcal/m^2•hr as their surface to dive time ratio was changed from 1/1 to 2/1. Thus, by adjusting the surface to dive time ratio one can prolong the working time without increasing heat loss during breath-hold diving in cold water. This wisdom of behavioral adjustment of diving pattern (depending on water temperature) is actually observed in male divers of Tsushima Island, Japan, as will be presented by Shiraki in the following paper.

REFERENCES

1. Kang BS, Song SH, Suh CS, Hong SK. Changes in body temperature and basal metabolic rate of the ama. J Appl Physiol 1963; 18: 483-488.

2. Kang DH, Kim PK, Kang BS, Song SH, Hong SK. Energy metabolism and body temperature of the ama. J Appl Physiol 1965; 20: 40-50.

3. Kang DH, Park YS, Park YD, Lee IS, Yeon DS, Lee SH, Hong SY, Rennie DW, Hong SK. Energetics of wet suit diving in Korean women breath-hold divers. J Appl Physiol 1983; 54: 1702-1707.

4. Park YS, Rahn H, Lee IS, Lee SI, Kang DH, Hong SY, Hong SK. Pattern of wet suit diving in Korean women breath-hold divers. Undersea Biomed Res 1983; 10: 203-215.

5. Beckman EL. Thermal protection during immersion in cold water. In: Underwater Physiology Vol II Lambertsen CJ and Greenbaum LJ eds. Pub 1181, National Academy of Sciences - National Research Council, Washington, D.C. 1963; pp 247-266.

6. Van Someren RNM, Coleshaw SRK, Mincer PJ, Keatinge WR. Restoration of thermoregulatory response to body cooling by cooling hands and feet. J Appl Physiol 1982; 53: 1228-1233.

7. Crittenden G, Morlock JF, Moore TO. Recovery parameters following underwater exercise. Aerospace Med 1974; 45: 1225-1260.

8. Keatinge WR. Survival in Cold Water. Blackwell Scientific Pub. Oxford and Edinburgh, 1969.

9. Wolff AH, Coleshaw SRK, Newstead CG, Keatinge WR. Heat exchanges in wet suits. J Appl Physiol 1985; 58: 770-777.

10. Goldman RF, Breckenridge JR, Reeves E, Beckman EL. "Wet" versus "dry" suit approaches to water immersion protective clothing. Aerospace Med 1966; 37: 485-487.

11. Burton AC, Edholm OG. Man in Cold Environment. Hafner Pub. Co., New York and London, 1969.

12. Cannon P, Keatinge WR. The metabolic rate and heat loss of fat and thin men in heat balance in cold and warm water. J Physiol 1960; 154: 329-344.

13. Hayward JS, Collis M, Eckerson JD. Thermographic evaluation of relative heat loss areas of man during cold water immersion. Aerospace Med 1973; 44: 708-711.

14. Van Dilla M, Day R, Siple PA. Special problem of hands. In: Physiology of Heat Regulation and the Clothing. Newburgh LH ed.

Discussion following Dr. Park's presentation

Dr. Craig: In one of your slides you showed the insulation index for subjects not in the wet suit, and then you showed the increase in the insulation index with the wet suit. You said the insulation index of the body itself was not different. This is somewhat different from our point of view, this would imply that your skin temperature with the wet suit was not different from your skin temperature without the wet suit.

Dr. Park: During diving work in the sea the average skin temperature of divers was much higher with the wet suit than without the wet suit (by about 0.7°C in summer and by 14°C in winter), however, the skin heat flux was much lower with the wet suit than without the wet suit. Consequently, the insulation index of the body calculated by dividing the rectal to skin temperature difference with the skin heat flux appeared to be similar between the two conditions.

Dr. Rahn: I want to ask you about the assumption that the insulation of the wet suit itself remains constant, and it seems to me that the boundary layer - you're going to have a boundary layer around the wet suit - (will make a difference) and as your metabolism increases, your work rate increases and therefore your boundary layer is going to become reduced.

Dr. Park: It may well be, but probably not much so in vigorously circulating water. We tried to eliminate the boundary layer outside by stirring water very vigorously.

Dr. Rahn: Yes, that's fine, but when you sit in the suit and you're going to have very vigorous stirring, you're still going to have boundary layers and now, when you work harder and harder, you will reduce those boundary layers and I was wondering whether that curve that you showed for the suit insulation (figure 5) is not the real story, because you had to invent a new kind of insulation. I'm just wondering whether the difference in the suit insulation between resting and exercising subjects represents the changes in the boundary layer due to excessive work.

Dr. Park: Partly, probably. But like I said, water circulation was vigorous.

Dr. Rahn: Ah, but I'm telling you that, no matter how vigorous it is, I'm going to have pockets all over here and there that will produce a boundary layer. Then when I exercise I will reduce those boundary layers, at least I think, what do you think?

Dr. Rennie: Well there's also a degree of convective heat loss from inside.

Dr. Park: Inside of the suit, now you're talking about outside or inside?

Dr. Rahn: I'm talking about outside.

Dr. Rennie: The water circulation inside the suit complicates matters also.

Dr. Rahn: But it seems to me you can never wipe out the boundary layer no matter how much current you have because man is not a single beautiful egg.

Dr. Park: Certainly so, but like Dr. Rennie said, convection underneath the suit could also have an effect. When we did this experiment, we taped the suit at the ankles and the wrists to prevent heat convection from under the suit as much as possible. But still we probably had some convection underneath the suit, which reduces the external insulation. One more thing I want to point out is that the value of the suit insulation measured during heavy exercise was very similar to that obtained by Goldman et al. (Aerospace Med. 1966; 37: 485-487). In their study they measured the wet suit insulation using an electrically heated copper mannequin with and without circulating water. The difference was surprisingly small, indicating that the effect of boundary layer on the physical insulation of the suit is rather small.

Diving pattern and thermoregulatory responses of male and female wet-suit divers

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Along the coast and islands of Japan and South Korea there live more than 30,000 breath-hold divers who harvest professionally the ocean floor, gathering abalones, snails, sea urchins and sea weeds. While this profession belongs exclusively to females in Korea (Hae-Nyo), both sexes are engaged in diving work in Japan (Ama). According to the statistics compiled in 1977 by the Ministry of Agriculture, Forestry and Fisheries of the Japanese Government, there are approximately 13,000 full-time divers in Japan, of whom 8,500 (65%) are males; moreover, the number of male divers remain the same while that of the female decreased by 20% since 1965. It is also significant to note that male divers are responsible for 75% of the annual harvest by all divers in Japan. Since these divers start their profession at the teenage and continue until they reach more than 60 years old, they have been the subject of many physiological studies. Most of these studies have been focused on female divers in whom diving pattern, cardiovascular, respiratory and thermoregulatory functions have been extensively investigated (1-3). In contrast, male divers so far received little attention from physiologists despite their dominance in numbers and quantity of the harvest over the female counterpart. While this review is focused on comparing physiological functions between male and female divers (Ama), special emphasis is laid on the sex difference of thermoregulatory mechanisms of breath-hold divers.
Investigations of male divers were carried out at Tsushima Island, located between the island of Kyushu, Japan and the Korean peninsula (24 20 N, 129 20 E), and the data for the female counterpart were obtained from earlier studies on Korean women divers.

Methods

Experimental methods were matched in divers of both sexes, and the experiment was conducted in two seasons; once in summer and another in winter. However there were indispensable differences in the way of diving and wet suits between Japanese male and Korean female divers. Male divers in Tsushima Island used boats to reach the area where they dived, on the contrary female divers in Pusan area dived in the sea close to the shore (approximately 10 m from the shore). All divers of both sexes wore neoprene wet suits. During winter season, male divers wore a thicker wet suit (5.5-6.0 mm) and also were protected with a hood, gloves and boots made of foam neoprene, however during the summer season, they wore a thinner wet suits (5 mm) with cotton gloves and socks and no hood. On the other hand, Korean women divers wore a neoprene wet suit of same thickness regardless of the season; hood and boots of neoprene and cotton

gloves were used in summer and winter. Thus, the physical insulation external to the body was obviously increased in winter for male divers.

Measurement of diving pattern The depth of diving was recorded on a small aneroid gauge connected by a 30-m teflon tube to a rubber balloon of 500 ml capacity. The balloon was protected by a perforated polyethylene bottle and attached to the diver's belt. During actual diving operations the pressure changes were monitored either by reading a pressure gauge manually (4) or by recording through a pressure transducer automatically (5). The duration of each dive and the interval between two successive dives, i.e surface time, were measured during their natural work shifts.

Measurement of heat exchange during diving work. The body temperature was measured with thermocouples (copper-constantan). Skin temperature of 7 points (forehead, chest, forearm, hand, thigh, calf, and foot) in male subjects and 4 points (chest, forearm, thigh and calf) in female divers were measured by thermocouples taped on the skin and mean skin temperature (Tsk) was calculated by the formula of Hardy and Dubois (6) for the 7 measurements and by Ramanathan (7) for the 4 measurements. Rectal temperature (Tre) was measured by a thermocouple probe sealed in polyethylene tubing inserted 10-15 cm beyond the anal sphincter. Mean body temperature (Tb) was calculated by the following equation: $Tb = 0.6Tre + 0.4Tsk$. Oxygen consumption was measured by collecting expired gas into a meteorological balloon. The gas was collected immediately prior to the diving while resting in air and at 5 to 15 min intervals in water while the subject was on the surface between dives. Heat production (M, kcal/m^2/h) was calculated indirectly from oxygen consumption and respiratory quotient. Total heat loss (H, kcal/m^2/h) was calculated as follows: $H = M + (dTb * BW * 0.83)$, where dTb represents the change in the mean body temperature in water ($^{\circ}C/h$), BW is body weight (kg), and 0.83 is the specific heat of the body (kcal/$^{\circ}C$/kg).

Measurement of thermal insulation The tissue insulation (I-tissue) during diving work was estimated by the following formula: $I\text{-tissue} = (Tre - Tsk)/Hsk$, where Hsk represents the skin heat flux (kcal/m^2/h), which was computed by the following formula: $Hsk = H - 0.08 \cdot M$, where 0.08 represents the fractional loss of M through the respiratory tract (8). The total insulation (I-total) provided by the shell and wet suits in the diver was estimated by the following formula: $I\text{-total} = (Tre - Tw)/Hsk$, where Tw represents water temperature.

Results and discussion

Diving pattern. The average rate of ascent and descent for male and female divers is summarized in Table 1. The average descent rate of shallow dives was similar for both sexes. However, the rate of descent for deep dives in male divers was almost 2-folds of the shallow dives and of female deep dives. Male divers used lighter counterweights than female divers (4) and thus the increased rate of descent can not be explained by differences in buoyancy. Actual underwater observations

of descending pattern indicated that male divers used legs and fins more extensively than the female to achieve a fast rate of descent. The rate of ascent was quite uniform in all divers. The lower value of the ascent rate for deep dives as compared to the descent rate in male divers indicate that they were not vigorously using their legs and fins for ascent as they did for descent. In shallow dives, the measured ascending rate of 0.27 m/sec was slower than that of the female diver. The slower rate of ascending was apparently related to the fact that the male divers in their ascent moved slowly toward the shallower region as they seeked their harvest. These results indicate that the rate of descent and ascent for shallow dives was not much different between Tsushima male divers and Korean women divers. On the other hand, the male divers were able to markedly increase the rate of descent for deeper dives unlike the women divers.

Table 1. Rate of ascent and descent of male and female divers

	Rate of descent and ascent		
	Descent (m/sec)	Ascent (m/sec)	Dive time (sec)
A. Shallow dives (depth<10 m)			
Male (n=2)	0.63	0.27	88
Female (n=5)	0.54	0.84	35
B. Deep dives (depth>10 m)			
Male (n=4)	1.12	0.77	56
Female	0.53	0.83	43

Frequency of dive and surface times and ratio of surface to dive time of Tsushima male divers in summer and winter seasons were compared in Fig.1. The seasonal difference of diving profiles of the divers, i.e. longer surface time and shorter diving time during the winter dive than the summer dive is clearly demonstrated. The average dive time, surface time (interval between two successive dives) and the ratio of surface time to dive time for male and female divers are shown in Table 2. The average dive time for male divers was 39 s which is longer than that for female divers, but the average surface time for male divers was 42 s which is slightly shorter than the female counterpart. The average ratio of surface to dive time was 1.30 for male divers in contrast to 1.49 for female divers. If one assumes the rate of ascent for male divers to be the same as that of descent for shallow dives (0.63 m/s), then the combined time for descent to and ascent from 5 m depth and bottom time were

calculated as shown in Table 3. The diving pattern was quite similar among male and female divers. On a daily basis, out of 237 min of total working time in water, male divers spend 114 min (48%) for diving and 123 min (52%) floating at the surface, on the other hand female divers dive for 74 min (41%) and float for 106 min (59%) out of 180 min daily working time in water. The Tsushima male divers spend 67 min on the bottom while the Korean female counterpart spends 37 min. In other words, the male divers have a daily bottom time twice longer than that of the female counterpart. These results indicate that, whereas the basic diving pattern was similar in both male and female breath-hold divers, the overall efficiency of diving (in terms of the rate of descent and the bottom time) appeared to be superior in male divers.

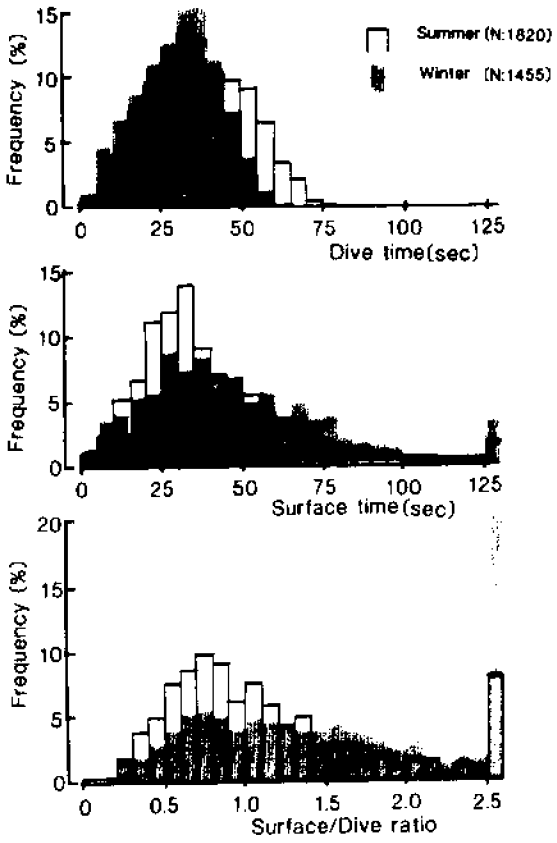

Fig. 1. Seasonal difference of percent frequency of individual dive time (duration), surface time (interval between two successive dives), and the ratio of surface to dive time of Tsushima male divers.

Table 2. Average dive and surface times of male and female divers (Average depth of dive ranged from 3 to 10 m)

		Dive time (sec)	Surface time (sec)	Surface/dive
Male (n=10)	Summer	38.6	42.3	1.30
	Winter	33.1	47.1	1.65
Female	Summer	31.8	45.7	1.49

Table 3. Comparison of general pattern of diving to 5-m depth between male and female divers (summer)

	Male	Female
Single dive time (sec)	39	32
Time for descent (sec)	8	9.3
Time for ascent (sec)	8	6.0
Bottom time (sec)	23	16.5
Single surface time (sec)	42	46
No. of dives/h	44.4	46.2
Total daily working time (min)	237	180
Total diving time (min)	114 (48%)	74 (41%)
Total surface time (min)	123 (52%)	106 (59%)
Total bottom time (min)	67	37

Heat exchange during diving work. The reduction of Tre in summer season during 2-h work period in water was similar in male (0.38°C :37.68°C before to 37.30°C after) and female (0.4°C :37.6°C to 37.2°C). On the other hand, the reduction of Tre during winter dive for male (water temperature, Tw of 14°C) was 0.79°C (from 37.67°C to 36.88°C) in 90 min and 0.6°C in 120 min for female divers (Tw = 10°C). This finding leads us to assume that female divers can maintain their body temperature longer in cold water, which may be explained partly by the thicker subcutaneous fat layer (3) as shown in Table 5. Also the duration of daily work shift in women divers was longer by 30 - 50 % (Table 4) in both summer and winter. In any case, it is reasonable to think that the divers perhaps recover their body temperature during the pause between work shifts.

Metabolic heat production for male divers increased to 125% of predive value after 5 to 10 min of diving work and then remained elevated until the termination of the diving work in summer and winter. On the other

hand female divers increased their metabolic heat production only 70 % in summer and 140 % in winter. The cumulative extra heat loss (i. e., net thermal cost of diving work) as a function of working time is shown in Fig. 2. The net thermal cost of diving work was calculated by summing the extra heat production (extra M = M during work - M during rest) and the reduction on body heat content (estimated from Tb change) in water. The cumulative extra heat loss increased rapidly in female divers in winter but not in male divers. To estimate the total thermal cost of diving work during a natural work, we extrapolated the steady state portion of the curve in Fig. 2 to the total work time in a day of an individual and the findings are summarized in Table 4. Daily total energy used for diving was 250 kcal/m^2 (summer) to 160 kcal/m^2 (winter) higher in male than female. The measured daily energy expenditure was identical to the estimated daily caloric intake of both sexes demonstrating that male and female divers were able to maintain their body weight and fat content almost constant in winter and summer seasons.

Thermal insulation during diving work. Steady state thermal insulation of divers during working in the sea is summarized in Table 5. Insulative values of female divers were significantly higher than those for male divers. I-total, I-tissue and I-suit for male divers were significantly increased in winter, however there was no such seasonal difference in female divers. For a subject submerged in cold water, the shell insulation is provided by the tissue and suit. The tissue insulation (I-tissue) is mainly attributed to the subcutaneous fat and non-perfused muscle layers. The muscle insulation, which accounts for at least 75% of the maximal I-tissue at rest in water critical temperature, is gradually diminished in exercise in proportion to work intensity (10). The subcutaneous fat insulation, on the other hand, is not altered during exercise in water (11). Thus, overall I-tissue of the subject exercising in cold water is expected to be higher in a fat subject than in a lean subject.

Table 4. Energy cost of daily diving work in summer and winter of both male and female divers

	No. of work shift	Duration of work shift (min)	Surface/dive ratio	Total working time (min)	Total extra energy cost (kcal/m^2)
Tsushima male diver (N=10)					
Summer	2	138±4	1.30±0.18	276±8	425±12
Winter	3	80±2	1.97±0.18*	240±4	405± 6
Korean female diver (N=4)[a]					
Summer	1	180	1.49±0.09	180	179
Winter	1	120	....	120	245

[a] Data from Kang et al(9). Values are means±SE. *$p<0.05$ vs summer value.

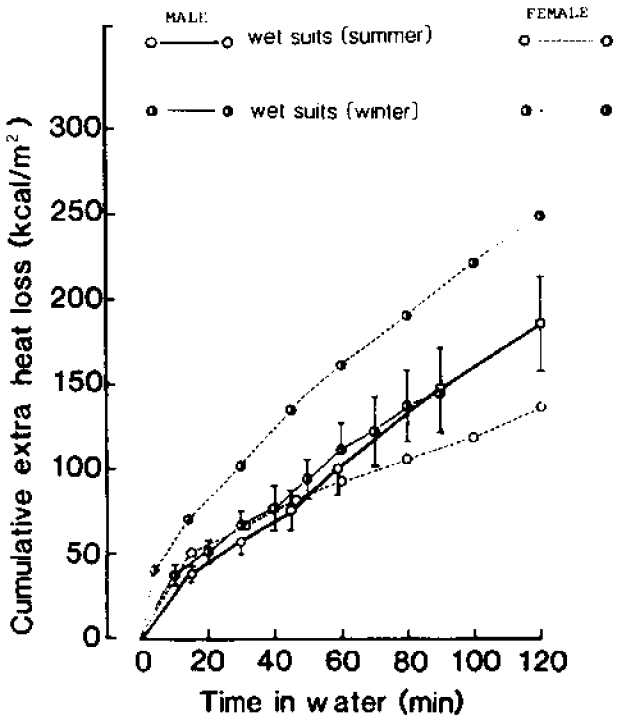

Fig. 2. Cumulative extra heat loss during the diving work in male and female divers. Points and vertical bars represent mean +SE. Data of the females are replotted by using the report of Kang et al. (9).

Table 5. Seasonal changes in thermal insulation ($^{o}C.m^{2}.h/kcal$) of male and female divers

	Summer				Winter			
	I-total	I-tissue	I-suit	body fat(%)	I-total	I-tissue	I-suit	body fat(%)
Male	0.065	0.028	0.037	14.7	0.135*	0.053*	0.082*	15.3
Female+	0.170	0.071	0.099	25.8	0.193	0.066	0.127	25.4

* $p<0.05$ vs corresponding summer value.
\+ data from Kang et al (9)

A significantly higher I-tissue during winter ($p<0.05$) in male divers indicates a further vasoconstriction in male divers, since the body fat content was identical in summer and winter. No further increase in I-tissue occurred in female divers indicating that they were in a state of near maximum vasoconstriction in summer and winter. The insulative value of wet suits (I-suit) significantly increased in male divers but not in female divers. The difference would be partly attributed to the thicknss of wet suits and the body area protected with the suit (refer the method section). Thus, the physical insulation external to the body was obviously increased in winter only in male divers. This may not fully account for the increased change in I-suit, because Goldman et al. (12) have reported that the difference of physical insulation of 4.76 and 6.35 mm neoprene wet suits was slight (0.128 vs. 0.139 $^{o}C.m^{2}.h/kcal$). Since the insulative value of wet suits was reported to decrease as the work intensity in water increased (8), then the lower value of I-suit in summer male divers may be accounted for by the higher work intensity in water. Also bypass heat flow due to convective water exchange through the wet suit (13) could account for the reduction in I-total, especially in male divers who do not wear the hood in summer. The convective heat flow from the neck portion of the wet suit would be high in water, leading to an underestimation of the I-total. Another important reason for the change in I-suit is a change in diving pattern between summer and winter in male divers. Since the diver stayed longer on the water surface in winter, as indicated by the increased surface/dive time ratio, and a foam neoprene suit is compressed less, the physical insulation of the wet suit should be higher in winter.

In the laboratory, we conducted an experiment to test whether or not I-tissue increases in high pressure in water and I-suit decreases in proportion to the depth of the water. A clear increase in I-tissue and reduction in I-suit is illustrated in Fig. 3. To test this phenomenon in actual diving in the field, we conducted an experiment to measure the insulative values on two Korean women divers by fixing the surface/dive time ratio to 1 and 2 and the results are summarized in Table 6. The data clearly suggest that when the surface/dive time ratio was increased from 1 to 2, the insulative values of tissues and wet suits increased considerablly. This finding indicates that divers may maintain their body temperature with less increase in metabolic heat production.

We therefore conclude that professional male divers of Tsushima Island increased I-suit in the cold season by behavioral adjustment of their diving pattern, whereas such behavioral adaptation was not observed in Korean female divers.

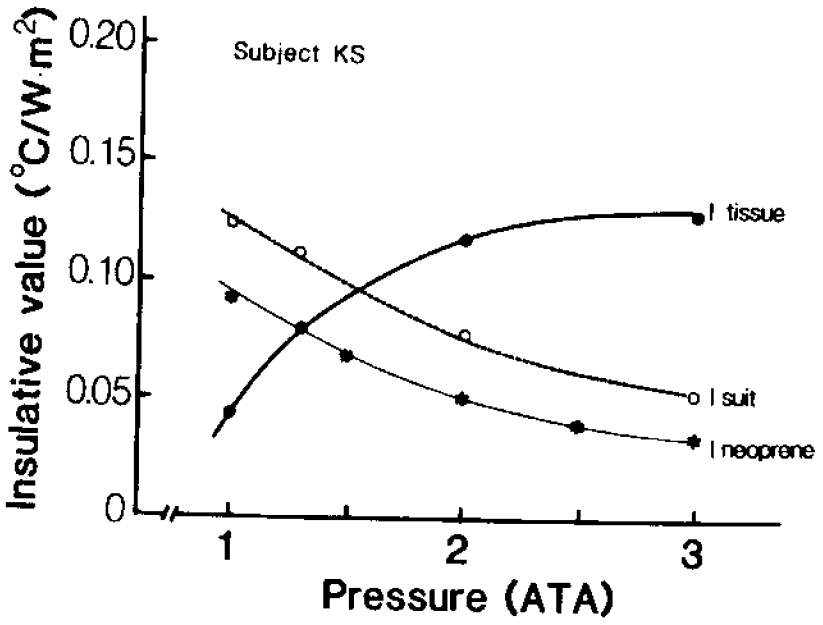

Fig. 3. Relationship between insulative values and water pressures. I tissue and I suit represent the average insulative value of the body surface and wet suit during the steady state of water immersion up to the neck, respectively. I neoprene is the insulative value of neoprene used for the wet suit (5mm).

Table 6. Effect of diving pattern on changes in insulative values of diving women

		M	I-total	I-tissue	I-suit	Dive	Surface
		(kcal/m²/h)	(°C/kcal/m²/h)			(sec)	
Resting (air)	#1	51.3	....	....	....	....	....
	#2	56.0	....	....	....	....	....
Dive (S/D=1)	#1	86.5	0.210	0.067	0.143	32.3	32.6
	#2	97.8	0.164	0.056	0.108	27.2	27.4
Dive (S/D=2)	#1	66.8	0.246	0.070	0.176	37.7	75.7
	#2	70.9	0.214	0.080	0.130	31.6	62.5

References

1. Hong, S.K., H. Rahn, D. H. Kang, S. H. Song, and B. S. Kang. Diving pattern, lung volumes, and alveolar gas of the Korean diving woman (ama). J. Appl. Physiol. 18: 457-465, 1963.
2. Hong, S. K. and H. Rahn. The diving women of Korea and Japan. Sci. Am. 216:34-43, 1967.
3. Hong, S. K. Pattern of cold adaptation in women divers of Korea (ama). Fed. Proc. 32: 1614-1622, 1973.
4. Park, Y. S., H. Rahn, I. S. Lee, et al. Patterns of wet suit diving in Korean women breath-hold divers. Undersea Biomed. Res. 10: 203-215, 1983.
5. Shiraki, K., N. Konda, S. Sagawa, et al. Diving pattern of Tsushima male breath-hold divers (Katsugi). Undersea Biomed. Res. 12, 1985 (in press).
6. Hardy, J. D., and E. F. DuBois. The technique of measuring radiation and convection. J. Nutr. 15:461-475, 1938.
7. Ramanathan, N. L. A new weighting system for mean surface temperature of the human body. J. Appl. Physiol. 19:531-533, 1964.
8. Rennie, D. W., B. G. Covino, B. J. Howell, et al. Physical insulation of Korean diving women. J. Appl. Physiol. 17:961-966, 1966.
9. Kang, S. H., Y. S. Park, Y. D. Park, et al. Energetics of wet-suit diving in Korean women breath-hold divers. J. Appl. Physiol.:Respirat. Environ. Exercise Physiol. 54:1702-1707, 1983.
10. Rennie, D. W., Y. S. Park, A. Veicsteinas, and D. Pendergast. Metabolic and circulatory adaptation to cold water stress. In: Exercise Bioenergetics and Gas Exchange, edited by P. Cerretelli and B. Whipp. Amsterdam, Netherland: Elsevier/North-Holland, 1980, p. 315-321.
11. Veicsternas, A., G. Ferretti, and D. W. Rennie. Superficial shell insulation in resting and exercising men in cold water. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 52:1557-1564, 1982.
12. Goldman, R. F., J. R. Breckenridge, E. Reeves, and E. L. Beckman. "Wet" versus "dry" suit approaches to water immersion protective clothing. Aerospace Med. 37:485-487, 1966.
13. Wolff, A. H., S. R. K. Coleshaw, C. G. Newsted, and W. R. Keatinge. Heat exchange in wet suit. J. Appl. Physiol. 58:770-777, 1985.

Discussion following Dr. Shiraki's presentation

Dr. Craig: If the male has a longer diving time, does the male produce more or make more money than the female?

Dr. Shiraki: Well, the situation in Korea and in Japan is quite different, but I can say that the male divers make two or three times more.

Dr. Hong: You said that the breath-hold divers dive deeper during winter, but you did not give the reason why they dive deeper in winter than in summer. Are there economic reasons for that?

Dr. Shiraki: During the summertime they pick up abalones while during the wintertime they pick up sea urchins, and also sea cucumbers, which live deeper than the abalones.

Dr. Elsner: I would like to ask a question about the Funado divers, particularly about the two men in the boat, is it possible that the man who gives her the weight is her husband, and the fellow who pulls her up is her boyfriend?

Dr. Lundgren: A question that is related to some earlier discussions we've had. The information that I'm looking for may be in your data. For those, I think it was the women, who increased their metabolism during the winter, does that influence breath-hold diving time?

Dr. Hong: If you take the grand average of nearly two thousand dives, single dive times decreased during winter.

Dr. Lundgren: Is it reasonable to relate that to an increase in metabolism? The male divers did not increase their metabolism during the winter, right? Does their winter dive time also change, or does it stay the same?

Dr. Shiraki: The dive time in the winter is shortened.

Dr. Lin: Dr. Park and Dr. Hong's study indicated that insulation is insufficient in Korean women in the winter, because the body temperature fell noticeably, but in the summer, the suit provides sufficient insulation.

Energetics of Breath-Hold Diving

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Abstract

The O_2 consumed and CO_2 produced during a breath-hold dive is important in determining the time of a breath-hold and the depth of the dive. A typical dive consists of a combination of descent, swimming at the bottom and ascent. The energy cost (E) of movement in water is directly proportional to the external work (W) and inversely proportional to the net mechanical efficiency (e), $E = W/e$. The W is proportional to the total resistance to movement (D) and the distance moved (d). The D during descent and ascent is a combination of the drag of the body (D_b) and the difference between weight in water and lung volume D_B. During bottom swimming, D would be equal to D_b and would be described by the equation, $D_b = KV^n$, with the constant K reflecting primarily the physical characteristics of the diver and the exponent n the hydrodynamic characteristics. The n can vary from ~1.2 to 1.85 and is dependent on depth and speed and K can vary from ~3 to 9 and is dependent upon equipment, swimming style, body composition and to a degree, speed. The D_B can vary from +6 to -6 Kg and is dependent on the weight in water, lung volume, and depth of the diver. Due to the differences in body composition and lung volume, the D for men and women is different. The D can be altered by the use of weights during descent and ascent. The value of e can vary from ~2 to 15%. The actual value is dependent on the speed, equipment used and swimming style. The energy supplied would be 50 and 70% aerobic for a .5 and 2 min dive, respectively, with the balance of the energy being supplied by, primarily, high energy phosphates in the shorter dives and glycolysis leading to lactic acid production in the longer dives. As long as there is a 3 min recovery between shorter dives, minimal net lactic acid build up is expected. Diving in cold water would increase the E by about 10% due primarily to a decreased e and body cooling of 1-2°C would result in an additional 10% increase in E.

Introduction

Breath-hold diving can take many forms. These could range from repetitive diving to 5-20 m to do a job, to diving deeper than 70 m for the purpose of setting a breath-hold diving record. Although these activities are quite different, they have many similarities. Most specifically, the time of the breath-hold is determined to a great extent by the total lung volume prior to the dive and the rate of oxygen consumption and CO_2 production. Other presentations in this workshop and previous papers have been devoted to the role of lung volume (2,7,8,14) and CO_2 production (8,13). The purpose of this presentation is to discuss the factors that influence the rate of consumption of oxygen during a dive. The VO_2 is important not only because it determines the rate of reduction of alveolar PO_2, but also because it influences the PCO_2 which is critical in setting diving time.

In order to analyze breath-hold diving it must be divided into descent, swimming at the bottom, and the ascent. This is important because the factors that determine energy cost of the three parts of a breath-hold dive may be quite different. The total energy cost (E) of a dive is determined by the external work (W) and net mechanical efficiency (e), see equation 1:

$$E = \frac{W}{e} \qquad (1)$$

The W is a product of the water resistance, termed drag (D), and the distance of progression through the water (d) (equation 2):

$$W = D \cdot d \qquad (2)$$

Combining equations (1) and (2) we can derive the following equation (3):

$$E = \frac{D \cdot d}{e} \qquad (3)$$

This equation can be rearranged further to show that the E to swim a given d is equal to the ratio D to e (equation 4):

$$\frac{E}{d} = \frac{D}{e} \qquad (4)$$

The above equations can be analyzed quantitatively if one assumes that 1 l O_2 = 5 Kcal and 1 Kcal = 427 Kg·m. The ratio of E/d or D/e can be termed economy. It is important to note that both D and e are independently variable and can be influenced by the velocity of swimming (V), body composition and size, sex, swimming style, water depth and water temperature. In order to understand the energetics of breath-hold diving, the factors that influence both D and e will be analyzed.

Previous investigators have measured or estimated the oxygen cost of repetitive diving in the field at periodic intervals to estimate the overall energy balance (7), while other investigators studied Navy divers, in a protected environment, making single dives with passive descents and active ascents (13). In another study (14), the authors estimated the cost of diving from the force-velocity relationship during descent and ascent and measurements of water resistance. This analysis made many assumptions, most importantly that e was 3% and was independent of speed. This latter assumption we know is not correct (4,5,9,10,11).

The best measurements of energy cost of breath-hold diving have been made by Craig (2). He measured the $\dot{V}O_2$ and $\dot{V}CO_2$ during and after the dive, for varying number of repetitive dives, both assisted and unassisted. The dives ranged from 30 to 60 s in duration and they were to depths of 5-10 m. In general he found that a 5 m dive required .8 l O_2 and a 10 m dive 1.2 l O_2. In addition to energy derived from $\dot{V}O_2$, lactic acid built up in the blood after the first dive and decreased thereafter. These data imply that at least part of the E for the dive came from glycolysis leading to lactic acid production (anaerobic metabolism) and, in addition, from high energy phosphate depletion (aerobic metabolism) as indicated by the large $\dot{V}O_2$ during recovery (2). The data from the above studies can be combined and it would appear that the E of breath-hold diving ranges from .8 to 1.4 l O_2 for dives to depths ranging from 5 to 27 m and lasting between 30 and 90 s. The variability of

diving styles and profiles make a quantitative analysis of the data from these studies impossible. Furthermore, no attempt was made to extrapolate the cost of these relatively shallow dives to deeper dives. To this end, I will present data from selected studies concerning factors that could influence E and, therfore, diving depth.

To determine the E of diving, both D and e have to be considered. Inasmuchas they are both variable and influenced by different factors each will be considered separately.

Drag

The drag that must be overcome by a diver is the total of the water resistance per se (1,3) and the net buoyancy of the body (11). For example, to swim at a given speed at the surface, women, who are more buoyant than men, require about 1/2 of the E (4,9). Specifically in the case of diving, the lung volume acts as a buoyant force that must be overcome by propulsion and, therefore, is added to the propulsive force needed to overcome water resistance. At the beginning of a dive, the buoyant force is large (equivalent to total lung volume 4-8 Kg) and as the diver goes deeper, the volume decreases as does the buoyant force. Ironically, as the lung volume is increased, to increase breath-hold diving time, the buoyant force increases. During descent, the propulsive force can be maintained at a constant level and, as the depth of the dive increases, the diver's speed would increase. Once on the bottom, the total lung volume is not critical; however, the density (diver plus equipment) is important, as the attitude of the diver affects the drag (9,10,11). During ascent, the initial period requires the diver to overcome the water resistance plus the effect of the body's sinking force, as the lung volume and buoyant force are low. As the diver approaches the surface, the lung volume increases and the buoyant force assists the

ascent.

The drag resulting from propulsion in water increases exponentially as a function of velocity in water (see equation 5; 1,3,4,5,6,9,10,11,12), with the rate constant K reflecting the characteristics of the diver and the exponent n the hydrodynamic characteristics:

$$D = KV^n \qquad (5)$$

Both K and n are variable and are related to the drag coefficient (C_D), the density of the water (ρ_W) and the subject's effective cross sectional area A. The latter variable is proportional to the subject's mass and inversely proportional to the subject's body density (ρ_M) and height (L). Theoretically, the exponent in equation 5 should range from 1 at low speeds, to 2 at moderate speeds and as high as 4 at high speeds. In fact, the n ranges from ~1.2 to 1.9 with most conditions being around 1.8-1.85, while the K can vary from 3 to 9. From equation 5 an equation for the coefficient of drag C_D (see equation 6) can be derived and can be used to examine the factors that affect the K in equation 5:

$$C_D = 2\,K\,\frac{\rho_M}{\rho_W}\cdot\frac{g}{W}\cdot L \qquad (6)$$

where g is the acceleration due to gravity. Studies in our laboratory have shown that the C_D is about 20% less in women than in men. Furthermore, at the surface, the C_D is 20% greater than it is at 2′ and 20% greater at 2′ than at 4′. These changes can be directly translated into changes in E, and, therefore, the depth of water or in fact swimming within 2′ of the surface will affect the E. Another important aspect of D is that, due to the exponential increase in D with V, E increases exponentially with V. As a further point, the increased E at high speed is not recovered during a "glide" due to the high water resistance. This suggests that selecting the correct swimming speed is critical for breath-hold diving.

The drag discussed in the paragraph above refers to D without subject

movement. As soon as the arms or legs are moved, the D is increased (4,5,9,10,11). The depth of kick of the legs, when combined with the kicking frequency, influences dramatically the D. In studies in our laboratory, D, during swimming with fins, was relatively high at low speeds (~7 Kg), decreased at moderate speeds (~5 Kg) and increased again at high speeds (~10 Kg). These changes in D were a result of the changes in the depth of kicking at the three speeds. Paradoxically, at low and moderate speeds the economy (E/d) of swimming was similar as changes in e offset the changes in D. At speeds greater than .9 m/s, the increase in e was less than the increase in D, and E/d increased dramatically. It should be noted that technical skill of swimming can affect both D and e (4,9,10,11) and swimming with fins can change E/d by about $\pm$20%. Fins alone in surface swimming tend to buoy the legs and decrease the E/d; however, in a vertical descent, this effect could result in a increased E.

The net result of these factors on D during breath-hold diving is obviously very complicated. Being assisted during descent and ascent can lower the drag, especially considering the drag created by swimming. The D is also influenced by buoyant forces and water depth. If a subject produces a propulsive force of 2 Kg during descent, his speed would range from .5 m/s at 5 m to 1.5 m/s at 30 m, while, during ascent, even if the propulsive force were 4.5 Kg at 30 m, the speed would be .4 m/s and would only increase to .65 m/s at 5 m with an additional 2 Kg propulsive force.

Efficiency

As described above, the net mechanical efficiency is equal to the ratio of E to W. Swimming efficiency can vary from 2 to 15% (1,3,4,5,6,9,10,11). The variability depends, to a great extent, on the technical ability of the swimmer. Women have a two times better efficiency than men due to their

greater buoyancy (4,9). In addition, fin swimming has a higher e than swimming without fins. Most importantly, e increases linearly with speed for most types of swimming; however, in fin swimming, e is high at low speeds (~8%), decreases at moderate speeds (~4%) and increases again at high speeds (~10%). As discussed above, these changes are paralleled by changes in D. The e appears to be related primarily to kicking frequency.

Diving

Combining the data from above we can calculate the E for dives to different depths. Assuming that the O_2 available at the maximum is .21 times the total lung capacity and knowing E/d, we can calculate the maximal depth of a dive when swimming without fins, with fins and swimming with minimal drag and maximal efficiency (minimal E/d). Under these circumstances the total available 1 O_2 would be between .75 and 1.5 1 O_2 and be dependent on total lung capacity (4-7 1). The maximal diving depth would range from 5 to 10 m without fins, from 10 to 20 m with fins and from 15 to 30 m in the case of maximal efficiency. It is obvious from this discussion that, for dives to depths greater than 30 m, the diver must be assisted both during ascent and descent.

Effect of Water Temperature

Most breath-hold diving is performed in water colder than thermoneutral. Even if there is no shivering, exposure to water below ~34°C results in metabolic alterations to swimming. Investigators have noticed (10-12) that the E/d is increased during exposure to cold water, even though the exposure is not long enough to decrease core temperature. This increase amounts to 10-20% additional E at any speed. If the exposure is long enough to cause a decrease in core temperature, the E is increased an additional 10 to 20% at all speeds. The increase in E is primarily the result of decreased e, as

there is either increased metabolic activity in muscles antagonistic to swimming or the resistance of the muscles themselves increases when they are cooled.

Summary

The energy cost of breath-hold diving has been measured to be between .8 and 1.4 l O_2 for dives to depths ranging from 5 to 27 m. Although the minimal cost (.8 l O_2) appears not to be too variable, as the depth increases, there is a large variability in energy cost. Many factors can influence this cost as described above. It is clear that more experimental data is needed to quantify the exact cost of diving to various depths, with special consideration to water temperature. At any rate, under optimal conditions, unassisted diving would appear to be limited to ~30 m. More investigation is needed to provide the data necessary to calculate the maximal diving depth during an assisted dive.

References

1. Alley, L.E. An analysis of water resistance and propulsion in swimming the crawl stroke. Res. Quart. 23:253-270, 1952.

2. Craig, A.B., Jr. and W.L. Medd. Oxygen consumption and carbon dioxide production during breath-hold diving. J. Appl. Physiol. 24:190-202, 1968.

3. Karpovich, P.V. Water resistance in swimming. Res. Quart. 4:21-28, 1933.

4. di Prampero, P.E., D.R. Pendergast, D.W. Wilson, and D.W. Rennie. Energetics of swimming in man. J. Appl. Physiol. 37:1-5, 1974.

5. Goff, L.G., H.F. Brubach, and H. Specht. Measurement of respiratory responses and work efficiency of under water swimmers utilizing improved instrumentation. J. Appl. Physiol. 10:197-202, 1957.

6. Holmer, I. Oxygen uptake during swimming in man. J. Appl. Physiol. 33:502-509, 1972.

7. Kang, D.H., P.K. Kim, B.S. Kang, S.H. Hong, and S.K. Hong. Energy metabolism and body temperature of the ama. J. Appl. Physiol. 20:46-50, 1965.

8. Lanphier, E.H. and H. Rahn. Alveolar gas exchange during breath-hold diving. J. Appl. Physiol. 18:471-477, 1963.

9. Pendergast, D.R., P.E. di Prampero, A.B. Craig, Jr., D.W. Wilson and D.W. Rennie. A quantitative analysis of the front crawl in men and women. J. Appl. Physiol. 43:475-479, 1977.

10. Pendergast, D.R., and D.W. Rennie. The energy cost of swimming with SCUBA. Sixth Symposium on Underwater Physiology. (Abstract) 1975.

11. Pendergast, D.R., P.E. di Prampero, A.B. Craig, Jr. and D.W. Rennie. The influence of selected biomechanical factors on the energy cost of swimming. In: Swimming Medicine IV. B. Eriksson and B. Furberg, eds. University Park Press, 1978, pp. 367-378.

12. Puah, L.C.G. and O.G. Edholm. The physiology of channel swimmers. Lancet 2:761-768, 1955.

13. Schaefer, K.E. The role of carbon dioxide in the physiology of human diving. In: Proc. Underwater Physiology Symposium. L.G. Goff, ed. Washington, D.C.: National Academy of Sciences - National Research Council, Publ. 377, 1955, pp. 131-139.

14. Yokoyama, T. and S. Iwaski. Energy cost of diving ama. In: The Physiology of Breath-Hold Diving and the Ama of Japan. H. Rahn, ed. Washington, D.C.: National Academy of Sciences - National Research Council, 1965, pp. 349-365.

Discussion following Dr. Pendergast's presentation

Dr. Boysen: In the study you did there were individuals of different technical skills, from the novice all the way up to experts and professionals. Was that a breath-hold swim?

Dr. Pendergast: No, those were SCUBA swims.

Dr. Boysen: Then my question is: there is a big difference between the expert and the professional. The two groups would be expected to be closer together. You alluded to the fact that you didn't know the distance they went. I don't know whether they were swimming aerobically or anaerobically, but you said you thought the big difference in their performance was technique. To further elucidate that, do you know anything else about their degree of fitness? In other words, their oxygen consumption, their maximum oxygen consumption, in and especially out of water; were they the same or different?

Dr. Pendergast: The swims that I showed you were all sub-maximal aerobic swims and that's why they ended at the velocities that they did. So these were swims these individuals could keep up for six or eight minutes at each speed. So there was no anaerobic component, and secondly, if there were a change in fitness, it would not affect the energy cost per unit distance of swimming. I think this is an extremely important point: fitness does not affect that, while technique does. Now, we did a similar graphical analysis of those divers and the big difference in the divers was the stroke frequency and the depth of the kick and so one can account for the changes in whole cost based on those two variables.

Dr. Hong: Let us get down to the nitty gritty. Are the values calculated by Yokohama in agreement with your data? What is the bottom line?

Dr. Pendergast: Well, I think that Yokohama's estimates were based on Dr. Craig's data and Dr. Craig actually made the measurements. At the two depths the VO2 that they calculated or measured agrees with our data. They were right for the wrong reason, and that is that the drag and efficiency that they used were too low. The bottom line is, as the energy cost is the ratio of drag and efficiency, it turned out to be the same energy cost.

Dr. Arnold: Dr. Pendergast, at the end of your abstract you mentioned that a three minute recovery period between your one minute or so dives was sufficient to take care of the lactic acid build-up.

Dr. Pendergast: Yes, as repeated diving continues, that lactic acid is burned as fuel and no net accumulation ensues, as shown by Craig. Three minutes are sufficient to resynthesize the high energy phosphates.

Dr. Arnold: In 1979, I did a study with Dr. Elsner where we compared facial immersion breath-holds of 30 second duration to determine whether the period of time between two breath-holds affected the

amount of bradycardia elicited by the second. We found that a short period of time between the breath-holds did alter the bradycardia in the second and, in fact, there was more bradycardia or faster onset of bradycardia in the second breath-hold than in the first. We assumed that that might very well have had something to do with either blood gas or lactic acid build-up and that a recovery period of at least two minutes should separate that.

Dr. Pendergast: My comments are based on two things - one is that it's well known now that lactic acid can be burned as a substrate for subsequent exercises. In your experiments, you were resting which makes a considerable difference in the lactate wash-out. We have recently done a study that shows that if you do a set amount of super maximal exercise, if you start with resting or some elevated lactate, at the end of that exercise, you wind up with the same lactate. Now what this means is that the balance of the lactate was burned in that particular exercise if it's an aerobic exercise. So the lactate can in fact be burned off. The other answer would be that, if in fact there is lactate build-up, which there probably is for other reasons, then in fact the lactic acid would serve to drive the heart rate higher and counteract in part your bradycardia.

Dr. Craig: In regard to the lactate, we used very different types of diving patterns. 30 second down then 30 second rest, 5 meters diving, 10 meters diving on different schedules and in some of the more difficult dives we did lactic acid and we never found a significant elevation in lactic acid in repetitive diving.

Dr. Lundgren: I don't know if it's a question or a comment, but, Dr. Pendergast, you made the point that swimming harder will allow energy production by glycolysis and that would not put a penalty on the diver in terms of CO2 production and oxygen consumption, which is kind of obvious, but I suppose you're not proposing that you would thereby be able to dive longer - well longer perhaps in distance, is that what you imply? What is perhaps obvious to everybody is that lactacidemia, for one, is a respiratory stimulus itself and it could also increase PCO2 by reducing the solubility of any CO2 present.

Dr. Pendergast: I guess I would suggest this somewhat tongue-in-cheek because I don't think you can predict whether the increase in drive to ventilation or the small reduction in P_{O2} would offset the tremendous savings that you could have in oxygen consumption as far as the cost of the swimming is concerned. The other important thing is that the cost per unit distance, over the range of speeds that you can do breath-hold diving, is constant, and therefore one conceivably, and I'm saying this to an extent tongue-in-cheek, could cover a much greater distance under that situation than under the other.

Dr. Van Liew: Is it possible to say that there is a given amount of oxygen saved, due to the lactic and alactic debts? That would be independent of speed.

Dr. Pendergast: Yes, there is. The alactic, so called, deficit is somewhere between one and two liters of oxygen total, so in the cost of a dive you can save that much energy immediately with really no net

consequence, as long as there is enough time in between the dives to resynthesize the high energy phosphates. If the time between the dives is too short, then you would have embarrassed your high energy phosphates and you'd begin to produce lactic acid which then perhaps would accumulate over time. The lactic acid component would be equivalent to three milliliters of oxygen per millimole per kilogram body weight and this can account for maximally probably about four liters, but it would take about two to two and a half minutes to build that total deficit and so totally it's estimated that the total amount of oxygen equivalent available from anaerobic sources would be somewhere between four and six liters of oxygen.

THE DIVING RESPONSE: A COMPARISON OF ANIMALS AND MAN

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I think that it is fair to say that we agree with respect to the existence of a set of reflexes, collectively described as the "diving response", in certain marine species of birds and mammals during submergence. The response is an identifiable physiological condition which occurs in dives, especially those of long duration, and results in an adaptation of the animal to the cessation of external respiration thus produced. In many species the success of the response is dependent upon (1) enhanced oxygen storage by large blood volume and high levels of hemoglobin and myoglobin, (2) tolerance of elevated concentrations of metabolic products, such as CO2, H+ and lactate and (3) cerebral and cardiac resistance to hypoxemia. Adaptations which permit seals, ducks and other aquatic diving animals to tolerate long submersions resemble general vertebrate reactions to asphyxia. Neural regulatory mechanisms are also similar. Species vary in the quantitative expression of the responses and in the intensity with which they are regulated. Apnea, bradycardia and redistribution of cardiac output favoring brain and heart are precipitated in varying degrees by aquatic immersion. The display of these events depends upon dive duration and its emotional content. In this presentation I wish to review some of the evidence relating to diving responses in humans and animals. Adaptations of marine mammals to high pressure have been reviewed by Scholander (1940) and Denison and Kooyman (1973).

In his classical work of 1940, Scholander pointed out that a seal freely submerging in water sometimes failed to show diving bradycardia. However, seals reacted to startle and loud noise with sudden heart rate slowing. Experimental diving studies in which animals were trained to dive upon a signal resulted in variable responses in the sea lion, harbor seal and dolphin (Elsner, 1965; Elsner et al. 1966; Elsner, Kenney & Burgess, 1966). There was usually less decline in heart rate during trained immersion than during forced immersion (sea lion and harbor seal), but in dolphins the bradycardia was intensified during trained dives. Kooyman et al. (1980) described dives of up to 20 min in Weddell seals (which are capable of maximum dives exceeding 60 min) in which little or no dependence upon the anaerobic resources required for longer dives was made, although cardiovascular changes, signalled by bradycardia, presumably occurred (Kooyman & Campbell, 1972). Oxygen reserves were apparently sufficient to allow the dives to be made aerobically. If, as seems likely, these reserves are principally contained within the blood and oxygenated myoglobin of skeletal muscle, then the reactions of the voluntarily diving Weddell seals resembled those of restrained diving harbor seals in a previous study in which myoglobin oxygen was rapidly depleted before lactate production commenced in about 10 min, earlier in

seals which struggled (Scholander et al., 1942). One is struck by the similarity of the responses in the two examples, rather than by differences. Authors of both studies remarked upon the observation that the preponderance of free dives in nature are brief and aerobic, indicating that anaerobic responses, which are more exhausting and require longer recovery, are seldom brought into operation.

Butler & Woakes (1979) and Kanwisher, Gabrielsen & Kanwisher (1981) showed that freely diving birds make many short dives without changes in heart rate. Clearly, most of the dives performed by aquatic animals in nature are of short duration, and they apparently depend largely upon oxygen-sustained rather than anaerobic mechanisms. Ducks and seals doubtless find it uncomfortable and exhausting to push their diving habit to the anaerobic extreme of their reserve capabilities. Although that physiological reserve is rarely invoked, it is, nevertheless, a resource upon which survival may depend. From these considerations there emerges the concept of graded responses to diving, varying in intensity depending upon severity of the imposed stress and of the degree to which control is exercised by higher cortical functions.

The understanding of diving biology is enhanced by examining the natural history of diving species. It is equally important that we study the full range of physiological responses of which these animals are capable by submitting them to experimental dives. Controlled laboratory investigations require either the use of trained animals or, more drastically, the interventions of restraint and anaesthesia. These approaches have all been usefully employed in various studies. A major response to most diving situations, natural and experimental, is the selective redistribution of the circulation. Inasmuch as many of the tissues of the body can tolerate oxygen deprivation far longer than can the more vital ones, the heart and the brain, the available blood oxygen could be conserved by its preferential distribution to those vital organs for their immediate needs while the remaining organs, deprived of circulation, survive on anaerobic metabolic processes or, in the case of skeletal muscle, on oxygen bound to myoglobin. Modern understanding of these diving responses rests on the foundation of classical research reports by Irving and Scholander. Some aspects of these adaptive mechanisms exist in many species which are not habitual divers when they are exposed to asphyxia (review: Elsner & Gooden, 1983).

Hypometabolism, a strategic retreat

Restricting the circulation of blood to a tissue or an organ results in a steady decline of the oxygen available for support of oxidative metabolic processes and subsequent dependence upon whatever anaerobic

resources are available. Eventually, a depression of metabolism takes place. This lowering of the rate at which the many complex chemical processes can occur, thus conserving metabolic energy, is, in fact, a central feature of the adaptations to asphyxia. Viewed in this context, the diving response represents but one specialized example of a widespread and general response of many living animals, invertebrate as well as vertebrate, to life-threatening situations. There are abundant examples in nature. Marine intertidal invertebrates at low tide, fish out of water, hibernators and estivators come to mind. Some possible human examples will be examined.

Recognition that such a wide variety of species, from invertebrates to mammals, show the ability to make strategic retreats from environmental threats into states of metabolic depression suggests the fundamental and general nature of this phenomenon. Cardiovascular adaptations leading to a redistribution of the blood flow that favors the more vital structures, thus accomplishing an overall sparing of metabolic energy, play a major role in these examples. Homeostasis is modified but not abandonded. Continued regulation, but with an extension of its range to include adaptive metabolic conservation, is the central theme upon which these variations are played.

The occurrence of reduced metabolic rates during experimental diving asphyxia is supported by several lines of evidence. Scholander (1940) was the first to demonstrate this reaction when he found that the extra oxygen consumption of seals during recovery from quiet dives could not account for the amount predicted if metabolism during the dive was maintained at the pre-dive level. Body temperatures decreased in diving seals along with the decrease in metabolism (Scholander, Irving & Grinnell, 1942; Hammel et al., 1977; Kooyman et al., 1980). Therefore, they could be expected to repay only part of the accumulated oxygen deficit during recovery from the hypoxic diving episode. Experimental evidence indicates that the cardiac oxygen consumption in diving seals declined to 7 - 14% of the non-diving value (Kjekshus et al., 1982). In dogs trained to dive the decrease was 42% (Gooden et al., 1974). In both examples oxygen consumption roughly followed the decline in heart rate.

Human dives

Human experiments in which diving bradycardia was recorded, even during underwater swimming, first appeared in 1940. These were mentioned briefly in a paper describing the respiratory metabolism of the porpoise (Irving, Scholander & Grinnell, 1941), but were not fully reported until 1963 when they became of historical importance (Irving, 1963). Bradycardia has been observed in human subjects during actual and simulated diving.

In one particularly striking case Elsner et al. (1966) observed several pulse intervals equivalent to 13 beats/min in a subject performing a 30-s face immersion. This subject began the procedure with a heart rate of 90 beats/min. The diving response was evoked in similar experiments by simple face immersion with simultaneous breath-holding and it was found that this procedure was as effective in producing diving bradycardia as total body immersion. The subjects for this study were nine young men of varying diving experience. They lay prone and performed face immersion with breath-holding in a bowl of water, temperature 18 to 20 degrees C, for 1 min. Calf blood flow was measured by venous occlusion plethysmography using a mercury-in-rubber strain gauge. The simulated dives were performed after moderate inspiration or expiration without hyperventilation. The limb blood flow consistently decreased more during face immersion than during breath-holding alone.

The reduction in calf blood flow observed was assumed to be the result of vasoconstriction, but proof of this assumption depended upon the simultaneous measurement of the systemic arterial blood pressure and limb blood flow. Heistad, Abboud & Eckstein (1968) measured brachial arterial blood pressure directly by cannulation as well as finger and forearm blood flow by venous occlusion plethysmography during breath-holding with and without face immersion. During 30-s of face immersion breath-holding there was a progressive rise in mean arterial blood pressure to an average of 20% above the control level. Both systolic and diastolic pressures were elevated. At the same time the finger and forearm blood flow decreased by an average of 72 and 50% respectively below the control value. They concluded that vasoconstriction must occur in the forearm vasculature during this form of simulated diving. Breath-holding alone produced similar but consistently smaller changes in pressure and flow. Face immersion in human subjects results usually in a modest increase in arterial pressure, and the bradycardia is more severe when the immersion takes place in cold water (Song et al., 1969). Neither the pressure nor temperature effects are evident in diving seals.

As in seals and ducks, vascular constriction during diving in man may not be limited to the arterioles. Olsen et al. (1962) reported frequent damping of the brachial arterial pressure recording during total immersion of their diver subjects. They suggested that this effect resulted from the constricted arterial wall occluding the tip of the intraarterial needle. Elsner, Gooden & Robinson (1971) found considerable difficulty in withdrawing brachial arterial blood samples during face immersion breath-holding in a subject who had developed a profound reduction in forearm blood flow. Additional evidence that vasoconstriction occurs in human arteries of greater than arteriolar size during diving was obtained by Heistad et al. (1968). During simulated dives, a substantial systolic

pressure gradient developed between the brachial and digital arteries in three out of five subjects, suggesting that vasoconstriction must have occurred in larger arteries upstream from the digital artery.

Functional significance of the diving response

The question arises whether the cardiovascular response to diving in man has any functional significance and in particular whether the human response has an oxygen-conserving role. Oxygen conservation could result in two ways. First, by limiting the blood flow to peripheral tissues such as the limbs and gut, the uptake of oxygen by these tissues would be reduced. The reduction in the heart rate can be expected to result in a modest decrease in myocardial oxygen consumption, although this reaction might be at least partially reversed if arterial pressure is elevated. When Olsen et al. (1962) were studying blood gas tensions in their divers during total immersion, they noted a surprisingly high arterial blood oxygen content at the end of long exercise immersions. This observation suggested to them that there was a reduced extraction of oxygen in the tissues during diving in man.

Elsner et al. (1971) examined the influence on the diving response of varying the arterial gas tensions of oxygen and carbon dioxide before face immersion breath-holding. Brachial arterial blood was sampled from one arm, and forearm blood flow was measured by venous occlusion plethysmography simultaneously in the other arm. Five male subjects hyperventilated on oxygen for 15 min and then perforemd face immersions which lasted an average of 5 min. The mean arterial oxygen tension before face immersion was 548 mmHg, and after 4 min the tension was still considerably elevated in four of the five subjects (mean value, 326 mmHg). A highly significant correlation existed between sequential changes in forearm blood flow and decreases in arterial oxygen tension (Fig. 1). Thus the greater the reduction in flow, the smaller was the decrease in oxygen tension. If this relation in the upper limb is representative of a more widespread phenomenon in the lower limbs, gut and elsewhere, then it seems likely that the diving response in man operates in some circumstances to conserve oxygen. In contrast to these results, no reduction in oxygen consumption could be detected during human breath-holding face immersion in the following studies: Raper et al. (1967), Heistad & Wheeler (1970) and Hong et al. (1971).

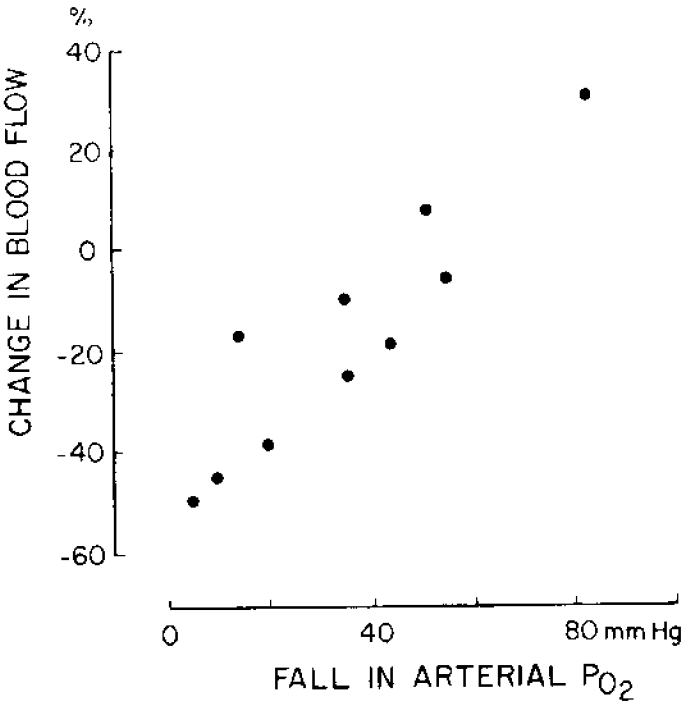

Fig. 1. Percentage change in forearm blood flow and decline in arterial PO2 during 5-min face immersion after oxygen hyperventilation in 5 subjects (from Elsner et al., 1971).

Whatever the extent of oxygen conservation in humans may be, it is unlikely to be ordinarily more than trivial when compared with the capability seen in the aquatic animals. However, the significance of the response in humans may extend well beyond its occurence in breath-hold dives. There are several conditions in which this can be expected to be so, although experimental verification remains uncertain (review: Elsner & Gooden, 1983). It is well known that fetal and newborn infants are able to resist exposure to asphyxia better than can adults. Their survival during birth asphyxia is likely to depend upon adaptations for that once-in-a-lifetime event. Perinatal reactions to asphyxia resemble in some respects the diving response, and the end result is the protection of the central nervous system. The initial events favoring survival in

near-drowning accidents might be related to vigorous diving responses which precede rapid cooling of the central nervous system and consequent reduction in oxygen demand. The clinical utility of face immersion in reversing paroxysmal tachycardia has been established. Observations on experimental animals of the vigor with which lung inflation during diving episodes stimulates cardiac action suggests that activation of pulmonary stretch reflexes during cardio-pulmonary resuscitation may have useful effects on heart function in addition to its respiratory benefits. Insights into the possible causes of sudden infant death syndrome may be provided by consideration of the neural mechanisms governing the diving response. Those mechanisms have been identified in a variety of experimental animals, including both marine and terrestrial species.

Circulatory control of metabolism? Its possible role in asphyxial defense

The basic priority of the circulation is that blood flow should be supplied to tissues in accordance with their needs. This primary role leads one to think of the circulation as the servant of tissue metabolism. The circulation may also act as a controller of metabolic rate, as can be demonstrated in limbs. The effect of decreased blood flow on tissue metabolism has been studied in skeletal muscle. Pappenheimer (1941) found that at flow rates greater than 40 ml/min in a 10 kg dog, the rate of oxygen consumption in the isolated perfused hindlimb of the dog was independent of blood flow. However, below this value the oxygen consumption decreased, reaching 50% of its normal value when the flood flow was reduced to about 10 ml/min. Fales, Heisey & Zierler (1962) studied the dog gastrocnemius-plantaris muscle *in situ* during partial venous occlusion and compared this procedure with the effects of arterial occlusion. They found that, within limits, muscle oxygen consumption was dependent on or limited by blood flow. Such conditions may well have existed in the trained seals in which abdominal aorta blood flow was much decreased, although not to the degree seen in long or forced dives, despite a modest bradycardia (Elsner et al., 1966).

REFERENCES

Butler, P. J. & Woakes, A. J. (1979). Changes in heart rate and respiratory frequency during natural behavior of ducks with particular reference to diving. J. Exper. Biol. 79:283-300.

Denison, D. M. & Kooyman, G. L. (1973). The structure and function of the small airways in pinniped and sea otter lungs. Respir. Physiol. 17:1-10.

Elsner, R. (1965). Heart rate response in forced versus trained experimental dives in pinnipeds. Hvalradets Skrifter, Norske Videnskaps-Akad., Oslo 48:24-29.

Elsner, R., Franklin, D. L., Van Citters, R. L. & Kenney, D. W. (1966). Cardiovascular defense against asphyxia. Science 153:941-949.

Elsner, R. & Gooden, B.A. (1983). Diving and Asphyxia, A Comparative Study of Animals and Man, Monographs of the Physiological Society, Cambridge University Press. 168 pp.

Elsner, R., Gooden, B. A. & Robinson, S. M. (1971a). Arterial blood gas changes and the diving response in man. Aust. J. Exp. Biol. Med. Sci. 49:435-444.

Gooden, B. A., Stone, H. L. & Young, S. (1974). Cardiac responses to snout immersion in trained dogs. J. Physiol. 242:405-414.

Hammel, H. T., Elsner, R., Heller, H. C., Maggert, J. A. & Bainton, C. R. (1977). Thermoregulatory responses to altering hypothalamic temperature in the harbor seal. Am. J. Physiol. 232:R18-26.

Heistad, D. D. & Wheeler, R. C. (1970). Simulated diving during hypoxia in man. J. Appl. Physiol. 38:652-656.

Heistad, D. D., Abboud, F. M. & Eckstein, J. W. (1968). Vasoconstrictor response to simulated diving in man. J. Appl. Physiol. 25:542-549.

Hong, S. K., Lin, Y. C., Lally, D. A., Yim, B. J. B., Kominami, N., Hong, P. W. & Moore, T. O. (1971). Alveolar gas exchanges and cardiovascular functions during breath holding in air. J. Appl. Physiol. 30:540-547.

Irving, L. (1963). Bradycardia in human divers. J. Appl. Physiol. 18:489- 491.

Irving, L., Scholander, P.F. & Grinnell, S.W. (1941). The respiration of the porpoise Tursiops truncatus. J. Cell. Comp. Physiol. 17:145-168.

Kanwisher, J. W., Gabrielsen, G. & Kanwisher, N. (1981). Free and forced dives in birds. Science 211:717-719.

Kjekshus, J. K., Blix, A. S., Elsner, R., Hol, R. & Amundsen, E. (1982). Myocardial blood flow and metabolism in the diving seal. Am. J. Physiol. 242:R79-R104.

Kooyman, G. L. & Campbell, W. B. (1972). Heart rates in freely diving Weddell seals Leptonychotes weddelli. Comp. Biochem. Physiol. 43A:31-36.

Kooyman, G. L., Wahrenbrock, E. A., Castellini, M. A., Davis, R. W. &

Sinnett, E. E. (1980). Aerobic and anaerobic metabolism during voluntary diving in Weddell seals: evidence of preferred pathways from blood chemistry and behavior. J. Comp. Physiol. 138:335-346.

Olsen, C. R., Fanestil, D. D. & Scholander, P. F. (1962). Some effects of apneic underwater diving on blood gases, lactate and pressure in man. J. Appl. Physiol. 17:938-942.

Pappenheimer, J. R. (1941). Blood flow, arterial oxygen saturation and oxygen consumption in the isolated perfused hind limb of the dog. J. Physiol. 99:283-303.

Raper, A. J., Richardson, D. W., Kontos, H. A. & Patterson, J. L., Jr. (1967). Circulatory responses to breath-holding in man. J. Appl. Physiol. 22:201-206.

Scholander, P. F. (1940). Experimental investigations on the respiratory function in diving mammals and birds. Hvalradets Skrifter, Norske Videnskaps-Akad., Oslo22:1-131.

Scholander, P. F., Irving, L. & Grinnell, S. W. (1942a). Aerobic and anaerobic changes in the seal muscles during diving. J. Biol. Chem. 142:431-440.

Scholander, P. F., Irving, L. & Grinnell, S. W. (1942b). On the temperature and metabolism of the seal during diving. J. Cell. Comp. Physiol. 19:67-78.

Song, S. H., Lee, W. K., Chung, Y. A. & Hong, S. K. (1969). Mechanism of apneic bradycardia in man. J. Appl. Physiol. 27:323-327.

Stainsby, w. N. & Otis, A. B. (1964). Blood flow, blood oxygen tension, oxygen uptake and oxygen transport in skeletal muscle. Am. J. Physiol. 206:858-866.

Discussion following Dr. Elsner's presentation

Dr. Hong: There are many interesting comparisons that can be made between diving mammals and humans, but we can reserve those for general discussion.

Dr. Data: What about the pulmonary circulation during breath-hold diving?

Dr. Elsner: Not much is known about it. There is little information. It is very sluggish during the dive, it falls to very low values, but some flow is maintained. Pulmonary pressure is not changed during diving.

Dr. Siesjo: I wonder if there's any information about the oxygen content or oxygen tension and glucose concentration in the plasma of these animals during prolonged dives.

Dr. Elsner: Yes there is, there is in all of those. The blood gases and pH, glucose, lactate, free fatty acids and such things.

Dr. Siesjo: What P_{O2} do you get after prolonged breath-hold dives (in seals)?

Dr. Elsner: At the end of a long dive, a very long dive, a dive that's carried to the point of the beginning of slow wave and high amplitude activity on the EEG, the PO2 gets down to about 9 torr.

Dr. Siesjo: Is it that low?

Dr. Elsner: Yes.

Dr. Siesjo: And at that level, what is the plasma glucose concentration?

Dr. Elsner: I can't tell you off the top of my head. The changes in plasma glucose are not spectacular, there is a small decrease.

Dr. Arnold: If a seal dives for 15 minutes, and it's got blood pooled in the inferior vena cava, do they have a mechanism to avoid clotting?

Dr. Elsner: That's a very good point, and a student of mine, Lori Wickham, is studying that point right now. Yes, the point is that the blood is sitting in those large venous pools, the kind that Dr. Craig talked of yesterday. It's not completely stopped, it's flowing through at a very sluggish rate, but certainly sedimentation is taking place and it turns out that the viscosity of seal blood at low shear rates is especially low so that it could be that there is some mechanism to inhibit the aggregation of red cells in that condition.

BREATH-HOLD BRADYCARDIA IN MAN: AN OVERVIEW

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More than a century ago Bert in France observed a pronounced bradycardia in the diving duck. Since then, this phenomenon has been confirmed in every diving animal. Largely due to the excellent pioneering work of Irving and Scholander, the physiological implication of this fascinating bradycardic response to breath-hold diving has been well documented (1). In essence, Irving and Scholander view this diving bradycardia as a reflex phenomenon which is accompanied by an intense peripheral vasoconstriction and by a drastic reduction in the cardiac output, leading to a significant conservation of oxygen (2-4). However, it may be noted that this cardiovascular reflex appears to be less marked during short, natural dives than during forced dives as used in earlier studies (5). Whether or not human breath-hold divers also display such a cardiovascular reflex has been the topic of exhaustive investigation during last two decades. As Dr. Elsner just described before me (6), there are certain qualitative similarities between diving animals and humans in responding to breath-hold diving but it is still inconclusive as to whether these cardiovascular responses observed in human divers lead to conservation of oxygen. In this presentation, I will focus on the heart rate responses to breath-hold diving, with the special emphasis on the trigger mechanism as well as on various factors modulating the response.

A. Heart Rate During Breath-Hold Diving:

Scholander et al. (7) were the first to demonstrate that human breath-hold divers, native men of the Torres Strait archipelago who dive for trochus shell, show bradycardia while diving for 45-60 sec. They observed that all 19 divers decreased their heart rate to 40-50 beats per min within 20-30 sec, which persisted in spite of considerable exercise under water. This finding has been confirmed subsequently by other investigators (8-11). In studies reported by Hong et al. (11) using Korean women divers, the heart rate was measured under various breath-hold maneuvers including the dive in both summer (27°C water) and winter (10°C water) (Fig. 1). As expected, the heart rate continuously decreased during the first 20 sec of breath-hold diving, after which it leveled off during the rest of the 50 sec diving time. With the termination of diving, the heart rate generally returned to the predive level within 10 sec. However, it is important to note that the degree of diving bradycardia was considerably greater in winter as compared to summer. In other words, the bradycardial response appears to be significantly potentiated during cold water diving. Another important observation was that a virtually identical pattern of bradycardia was observed even during either breath-hold surface swim with the face submerged or a simple breath-holding in water with the face submerged.

These observations support the notion that the diving bradycardia is not necessarily induced by the act of diving per se but by the act of breath-holding. As shown in the bottom panel of Fig. 1, a simple breath-holding at rest while sitting on the boat also induced a bradycardia, although its magnitude is considerably less than when various breath-hold maneuvers were held in water. Evidently, neither the depth of diving nor the swimming exercise seems to contribute to the development of diving bradycardia. This view is also supported by Irving (9) who reported that the onset and degree of diving bradycardia were not reduced by vigorous swimming underwater, and by Craig (8) who reported the independence of diving bradycardia from the diving depth. It appears that the bradycardia is primarily induced by the act of breath-holding per se and is then secondarily potentiated by the immersion of whole body in water. Moreover, the degree of potentiation of the bradycardia is clearly greater in colder water.

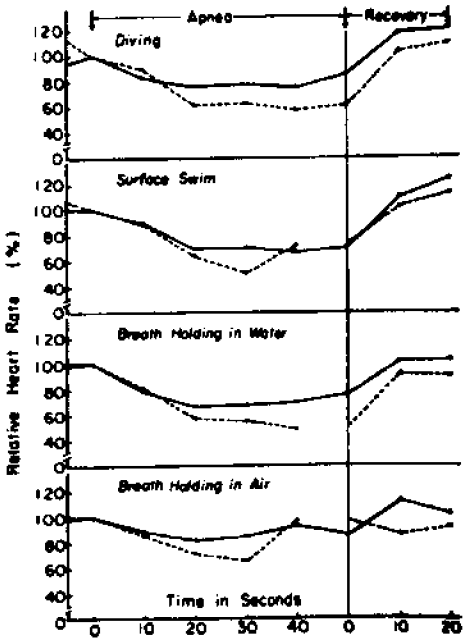

Fig. 1 Changes in the relative heart rate during various breath-hold maneuvers. Solid line (summer), 27°C; broken line (winter), 10°C water temperature. From Hong et al. (11) with permission.

Before I go into the detailed analysis of various factors involved in the development and modification of diving bradycardia, it should be pointed out that the breath-hold bradycardia in man is often associated with various types of cardiac arrhythmias. In Korean women divers, we observed abnormal P waves and nodal rhythms, idioventricular rhythms, premature atrial beats, premature ventricular beats, and others (11).

Moreover, the incidence of these arrhythmias was considerably higher in winter (72%) than in summer (43%). Scholander et al. (7) were also surprised to find that a high incidence of arrhythmia developed during diving. Thirteen out of 18 divers developed anomalies visible on the ECG, such as inversion of T waves, ventricular premature contractions, idioventricular rhythms, and one case of atrial fibrillation. These observations in human divers are in total contrast to those in diving animals in which arrhythmias have not been observed during diving.

As discussed above, the diving bradycardia appears to be triggered by breath holding and is then potentiated by immersion of the body in water (especially in cold water). In fact, Wyss (12) already reported nearly 30 years ago that ECG's showed bradycardia in men while submerged and during apnea. Whatever is the mechanism underlying this bradycardic response, the parasympathetic nerve supply seems to play a critical role because diving bradycardia in man can be blocked by atropine (13). It could be that inhibition of the respiratory center induced by the cessation of respiratory movements excites cardiac vagal motoneurones leading to bradycardia (14).

Although the whole body immersion in water appears to potentiate the bradycardial response to breath holding, it soon became evident that simple face immersion in water alone also similarly potentiates the response. Although Elsner et al. (15) used the latter approach for the first time in 1963 to induce a simulated diving bradycardia, the quantitative similarity of apneic bradycardia induced by whole-body immersion to that induced by face immersion alone was not established until 1972 (16). In order to define if a certain region of the face is involved in potentiating breath-hold bradycardia upon immersion, face immersion experiments were carried out with a nose clip or with a face mask. However, the results obtained from different laboratories are conflicting and thus no definite conclusion can be drawn regarding the particular role played by the stimulation of the skin in the region of the eyes and nose (17,18). Moreover, it is not even unequivocally established that the face needs to be wetted during face immersion to potentiate breath-hold bradycardia. Separating the face from water by thin plastic sheet either attenuated (19,20) or had no significant effect (16) on the breath-hold bradycardia. However, it is generally accepted that thermal receptors in the facial skin surface seem to play an important role (see below).

Despite the lack of the understanding of the mechanism underlying the unique role of face immersion in simulating the diving bradycardia in man, this technique has been widely utilized during last two decades by investigators to study diving bradycardia. In fact, most of the current knowledge of diving bradycardia is derived from such studies.

B. Role of Water Temperature:

As stated above, the magnitude of breath-hold bradycardia in Korean women divers was significantly greater in winter than in summer (11), indicating the important role of temperature in determining the degree of

breath-hold bradycardia. Actually, it was Craig (8) who first observed that the degree of diving bradycardia was greater in water of 22°C as compared to that in water of 29.5 and 34.5°C. Kawakami et al. (21) then demonstrated that the bradycardic response to breath-hold face immersion is also highly temperature dependent. Since then more systematic studies on the relationship between the bradycardic response and the temperature of water into which the face was immersed with breath held were carried out by a French group (22) and in my laboratory in Korea (23) and Hawaii (16,24). As shown in Fig. 2, the bradycardic response is inversely proportional to the water temperature, ranging from ~40% reduction at 5°C to ~15% at 30-40°C. The degree of bradycardia in response to apneic face immersion in water of 30-40°C is not different from that seen during a simple breath holding in air without face immersion. These findings support the view that the facial cold receptors play a very important role, and also lend an additional support to the view that the wetness of the face itself is not the major determinant of the bradycardic response.

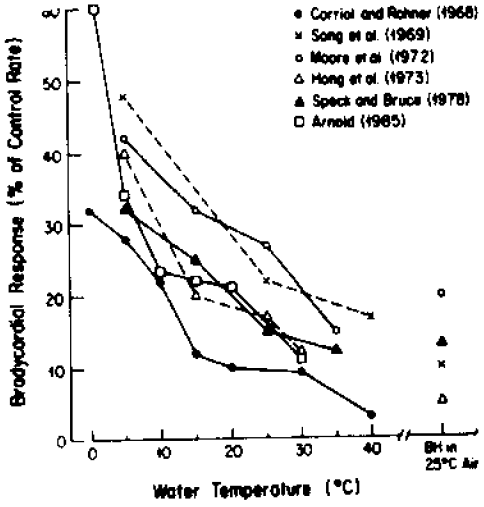

Fig. 2 Bradycardial responses (as % of control rate) as a function of the temperature of water into which the face is immersed. From Corriol and Rohner (22), Song et al. (23), Moore et al. (16), Hong et al. (24), Speck and Bruce (53), and Arnold (52).

Nonfacial cold receptors also seem to play a role, although their contribution to breath-hold bradycardia is less than that of facial cold receptors. It has been shown by Moore et al. (16) that the degree of breath-hold bradycardia is greater during a short (5 min) head-out immersion in water of 25 and 15°C than in water of 35°C; moreover, when this head-out immersion was accompanied by breath-hold face immersion (with or without face mask), the heart rate decreased further in water of 25 and

15°C but not in water of 35°C. However, the heart rate responses in the latter experiment were not different from those observed during breath-hold face immersion alone in water of corresponding temperatures, indicating the dominant role of facial cold receptors. In this connection, it should be pointed out that a face immersion in cold water without breath holding also induces a short, slight bradycardia (21,25-27).

Although it is quite clear that the combination of breath holding and the stimulation of facial cold receptors acts synergistically, the underlying mechanism is not understood. Typically, a breath-hold face immersion in cold water increases the blood pressure (especially the diastolic pressure) and decreases the peripheral blood flow (21,23), but the correlation between the bradycardic response and these hemodynamic changes is not evident. Moreover, the same hemodynamic changes may be induced by immersion of the hands or feet in cold water without breath-holding and/or face immersion and yet the magnitude of bradycardia, if any, is much smaller (21,23). In order to accentuate the degree of cold-induced hemodynamic changes, Moore et al. (16) studied the bradycardic response to breath-hold face immersion in water of 25, 15 and 5°C in human subjects (wearing only swimming trunks) resting in a 25 or 5°C room. As compared to that in the 25°C room, the mean skin temperature was lower (by approximately 5°C) while the diastolic blood pressure was higher (by approximately 10mmHg) in the 5°C room. Moreover, the control heart rate (without any breath-hold maneuvers) was lower by 13 bpm in 5°C than in 25°C room, suggesting a cold-induced increase in vagal tone. During apneic face immersion, the temperature-dependent bradycardic response was observed at both room temperatures, but the relative degree of bradycardic response to a given apneic maneuver was clearly less in 5°C room. However, there was no difference in terms of the lowest absolute heart rate during 60 sec apnea between the two room temperatures. These findings indicate that an increased vagal tone induced prior to an apneic face immersion in cold water by exposing to cold air for 30 min reduces the magnitude of apneic face immersion-dependent vagal activation. In other words, the breath-hold face immersion in cold water alone appears to increase the vagal tone to the same maximal level. Once the vagal tone is increased by means other than breath-hold maneuvers, the magnitude of the breath holding-induced increase in vagal tone decreases correspondingly.

C. Role of Intrathoracic Pressure:

In man, diving is usually preceded by a near maximal inspiration (equivalent to ~85% of the vital capacity) followed by breath holding (28). The inspiratory muscles are then relaxed, and the intrathoracic pressure is increased at least to the extent of the relaxation pressure of the lungs and thorax. Moreover, diving often involves a position change which would alter the pattern of venous return, the cardiac output and certain hemodynamic parameters such as arterial pressure. Any of these diving-induced changes could alter the heart rate. Craig (8,54) observed that maneuvers which increased venous return at the beginning of the breath hold produced a bradycardia during the apnea, and conversely when venous return

was impaired there was a tachycardia. Moreover, he observed that the heart rate during breath holding in air was closely related to the level of the intrathoracic pressure such that even a marked tachycardia was produced when the intrathoracic pressure increased to +40mmHg. Based on these and other observations, Craig stated that the act of breath holding *per se* during diving is not responsible for changes in heart rate, but that the intrathoracic pressure changes associated with relaxation after full inspiration and the application of a Valsalva maneuver may be responsible for the different signal. Furthermore, he proposed that the carotid sinus may be the site of the afferent signal.

In a subsequent study, however, Kawakami et al. (21) found that the heart rate during apneic face immersion always decreased irrespective of esophageal (or intrathoracic) pressure ranging from −5 to +16 cm H_2O. Song et al. (23) also reported that the bradycardia induced by apneic face immersion in 5 and 20°C water was maintained when the intraesophageal pressure was kept below 10–20 cm H_2O, above which a marked attenuation of the bradycardia was observed (Fig. 3). These investigators further observed that for a given level of intraesophageal pressure, the breath-hold face immersion in cold water always resulted in a greater bradycardia as compared to a simple breath holding in air. These observations indicate that the breath-hold face immersion can override the effect of the intrathoracic pressure on the heart rate. In other words, the changes in the intrathoracic pressure can modify the heart rate response to diving but can not initiate or trigger the bradycardic response.

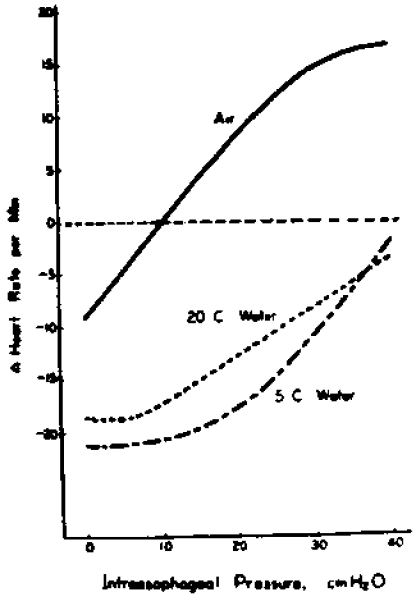

Fig. 3 Changes in the heart rate responses (Δ heart rate) at 30 sec of breath holding in air and of breath-hold face immersion at different levels of the intraesophageal pressure. From Song et al. (23) with permission.

D. Role of Lung Volume:

Elsner et al. (15) originally reported that the cardiovascular response (i.e., bradycardia and peripheral blood flow) to apneic face immersion was accentuated when performed in expiratory position. This finding is interesting in that diving mammals are known to dive in expiratory position. Although Asmussen and Kristiansson (26) reported a similar observation, Kawakami et al. (21) failed to confirm this interesting observation. The latter investigators, in fact, observed that the magnitude of bradycardic response to breath-hold face immersion in cold water tended to be lower when performed in expiratory position (with the functional residual capacity; FRC) than in inspiratory position (with the total lung capacity; TLC). Kawakami et al. (21) further observed that, in simple breath holding in air (without face immersion) at FRC, there was no change in heart rate, whereas a decrease of 10 bpm from the resting level was seen during breath holding at TLC. Similarly, Song et al. (23) reported that the reduction in heart rate during simple breath holding in air is proportional to the initial lung volume. In fact, at the residual volume (RV), the heart rate even tended to increase rather than decrease during breath holding. The reason for these differences in observations regarding the effect of the lung volumes on breath-hold bradycardia among investigators is not obvious. Nevertheless, it may be parenthetically noted that Angelone and Coulter (29) demonstrated an inverse relationship between the heart rate and the lung volume and suggested that stretch receptors located in the lungs or thoracic wall initiate a reflex responsible for this phenomenon. On the other hand, the attenuation of breath-hold bradycardia at FRC or RV, observed by Kawakami et al. (21) and Song et al. (23), may be related to a possible increase in the intrathoracic pressure associated with the act of expiration prior to breath holding (see above).

E. Role of Hypoxia and Hypercapnia:

An act of breath holding is associated with the cessation of respiratory movements and the development of both hypoxia and hypercapnia. It is, therefore, possible that hypoxia and/or hypercapnia could be responsible for the bradycardic response. However, Olsen et al. (30,31) pointed out that diving bradycardia in man occurred before any major changes in blood gas pressure could be expected; moreover, the degree of bradycardia did not correlate with the duration of breath holding. Kawakami et al. (21) also observed that breathing 5% O_2 - 20% CO_2 mixture for 2 min did not cause bradycardia either with face out or with face immersion. It thus appears that asphyxia *per se* does not produce a bradycardia. However, it is still possible that hypoxia and/or hypercapnia might either potentiate or attenuate the breath-hold bradycardia. In fact, it was proposed that hypercapnia followed by hypoxia during breath holding may contribute to the maintenance of the diving bradycardia (21). Based on the experiments in which alveolar O_2 and CO_2 pressures were selectively altered prior to breath holding (with or without face immersion in water), Moore et al. (32) reported that the breath-hold bradycardia was potentiated in the presence of low alveolar PO_2, attenuated under

conditions of high alveolar PO_2, and appeared to be independent of alveolar PCO_2 levels. These observations suggest that the peripheral chemoreceptors can modify the intensity of the diving response.

Recently, Lin et al. (33) designed 5 series of breath-holding experiments to partition the effect of apnea per se, hypercapnia, and hypoxia on the development and maintenance of breath-hold bradycardia (induced by face immersion in 25°C water). According to their analysis, a 31% reduction in the heart rate induced by this breath-hold maneuver represented an arithmetic sum of 19% reduction by apnea, 18% reduction by hypoxia, and 6% increase by hypercapnia (19+18-6=31). These results indicate that the hypoxia which develops during the course of breath holding has a major potentiating effect while hypercapnia has only a minor attenuating effect on breath-hold bradycardia.

F. Effect of Physical Exercise:

Studies conducted in the field using professional breath-hold divers indicated that diving bradycardia persisted in spite of considerable exercise under water (7,9,11,30). These observations led many physiologists to believe that even a severe physical exercise may not be able to override the mechanism leading to diving bradycardia. In fact, Stromme et al. (17) found that breath-hold face immersion during exercise induced a nearly 50% reduction in heart rate, independent of work load over a $\Delta\dot{V}O_2$ (the difference between the resting metabolic rate and the metabolic rate during steady state of swimming) range of 5-30 ml O_2/Kg per min. On the other hand, Craig (8) reported that the typical diving bradycardia was almost completely reversed during underwater exercise (moving flippers up and down at a moderate rate and force for 35 sec) in a diver who hyperventilated using 100% O_2 for about 5 min and then passively descended (while breath holding) to the pool bottom and remained quiet for 1 min. In fact, the heart rate increased to about the same rate as when the subject was at the surface and breathing. However, the recovery period was much shorter when the subject was submerged. The reason for this disagreement between Stromme et al. (17) and Craig (8) is most likely attributed to the difference in work load. According to Paulev and Hansen (34), breath holding produced bradycardia at relatively low rates of steady-state (cycling) work ($\Delta\dot{V}O_2/\dot{V}O_2max$ below 40%) just as during rest, and as relative work rates increased, the bradycardia gradually disappeared. In this connection, it is interesting to note that the heart rate during exercise in apneic divers is dependent on the type of exercise. For instance, decreases in heart rate induced by apneic face immersion (in 15°C water) were much more marked during dynamic exercise than during rest or isometric exercise (35). However, the exact physiological mechanism responsible for the differing influence of isometric and dynamic exercise on the diving bradycardia is obscure at present.

Whether or not physical conditioning (or fitness) modifies the breath-hold bradycardia is still controversial. Originally, Bove et al. (36) reported that a physical training program appeared to augment the bradycardia and, on this basis, suggested that the breath-hold maneuvers

could be used to determine which individuals are in need of physical training. On the other hand, Whayne and Killip (37), Stromme et al. (17), and Stromme and Ingjer (38) found no indication of a possible relationship between the degree of breath-hold bradycardia and the level of physical fitness.

A similar controversy exists concerning the relationship between the degree of breath-hold bradycardia and the level of swimming or diving experience. Irving (9) stated that diving bradycardia occurred with more regularity and to a greater degree in those accustomed to swimming and with a proclivity for aquatic acivity. Similarly, the magnitude of breath-hold bradycardia has been reported to be greater in Korean women divers (unpublished data of B.S. Kang) and in trained breath-hold and/or SCUBA divers than in the control (22,39). However, other studies do not agree with the above results (8,40,41).

G. Other Cardiovascular Changes:

Since Dr. Elsner (6) already discussed and Dr. Ferrigno (42) will also present his own data on cardiac performance during breath-hold diving, I will very briefly summarize other cardiovascular changes induced by breath holding in resting and exercising subjects. As stated above, the breath-hold bradycardia in man is always accompanied by an increase in arterial blood pressure (21,43-48) and a reduction in peripheral blood flow

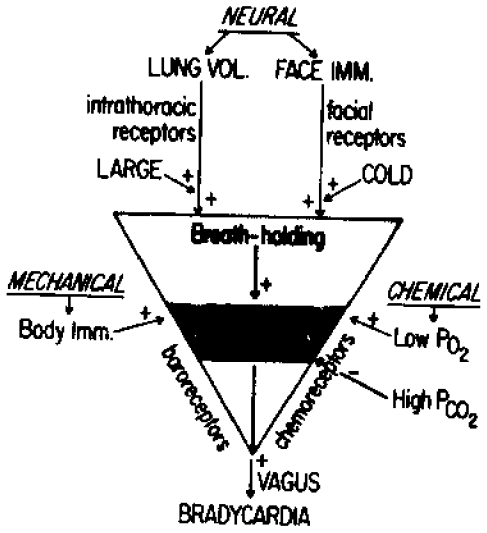

Fig. 4 Schematic model of factors modifying apneic bradycardia. + Indicates potentiation; - indicates attenuation of bradycardic response. Triangle symbolizes the different types of modifying stimuli to the basic bradycardia accompanying apnea, per se. Adapted from Moore et al. (32).

(15,23,44,45,48,49), although the latter changes are not necessarily responsible for the development of bradycardial response (see above). On the other hand, the cardiac output during breath holding with or without face immersion either remained unchanged (24,47), decreased by -20% (21,48) or increased by 14% (50). Therefore, total peripheral resistance has been shown to increase by 26 to 53% (46,48). More recently, Bjertnaes et al. (51) studied cardiovascular responses to face immersion and apnea during steady state muscle exercise and reported that the cardiac output during the last half of breath-hold face immersion was reduced by 49% while systemic vascular resistance increased by 200%. Moreover, these investigators found that myocardial O_2 demand determined by the heart rate - pressure double product decreased by 42% during breath-hold face immersion. However, they also noted that the rise in systemic vascular resistance is not closely adjusted to the reduction in cardiac output, and that the O_2-saving potential is reduced because of rise in cardiac afterloads.

H. Summary:

The breath-hold bradycardia is triggered by the cessation of respiratory movements, but there are many neural, mechanical and chemical factors known to modify the degree of the bradycardia. Based on the data available in the current literature, a modified version of previously published "multiple factor theory" (23,32) is shown in Fig.4.

REFERENCES

1. Anderson HT. Physiological adaptation in diving vertebrates. Physiol Rev 1966; 46: 212-243.

2. Irving L, Scholander PF, Grinnel SW. The regulation of arterial blood pressure in the seal during diving. Am J Physiol 1942; 135: 557-566.

3. Scholander PF. Physiological adaptation to diving in animals and man. Harvey Lect Ser 1961-62; 57: 93-110.

4. Scholander, PF. The master switch of life. Sci Am 1963; 209: 92-106.

5. Elsner R. Heart rate response in forced versus trained experimental dives in pinnipeds. Hvalradets Skrifter, Norshe Videnskaps-Akad, Oslo 1965; 48: 24-29.

6. Elsner R. Diving response: A comparison of animals and man. In: Lundgren C. ed. Proceedings of the Workshop on the Physiology of Breath-hold Diving. Undersea Med Soc: Bethesda, 1985.

7. Scholander PF, Hammel HT, LeMessurier H, Hemmingsen E, Garey W. Circulatory adjustment in pearl divers. J Appl Physiol 1962; 17: 184-190.

8. Craig AB Jr. Heart rate response to apneic underwater diving and to breath holding. J Appl Physiol 1963; 18: 854-862.

9. Irving L. Bradycardia in human divers. J Appl Physiol 1963; 18: 489-491.

10. Sasamoto H. The electrocardiogram pattern of the diving AMA. In: Rahn H, Yokoyama T, eds. Physiology of Breath-Hold Diving and the AMA of Japan. NAS-NRC Publication 1341, 1965; 271-280.

11. Hong SK, Song SH, Kim PK, Suh CS. Seasonal observations on the cardiac rhythm during diving in the Korean AMA. J Appl Phsyiol 1967; 23: 18-22.

12. Wyss V. Electrocardiogram of apneic subjects during immersion in water at various depth. Boll Soc Ital Biol Sper 1956; 32: 503-506.

13. Heistad DO, Abboud FM, Eckstein JW. Vasoconstrictor response to simulated diving in man. J Appl Physiol 1968; 25: 542-549.

14. Daly MB, Angell-James JE. The diving response and its possible clinical implication. Internat Med 1979; 1: 12-19.

15. Elsner RW, Garey WF, Scholander PF. Selective ischemia in diving man. Amer Heart J 1963; 65: 571-572.

16. Moore TO, Lin YC, Lally DA, Hong SK. Effects of temperature, immersion, and ambient pressure on human apneic bradycardia. J Appl Physiol 1972; 33: 36-41.

17. Stromme SB, Kerem D, Elsner R. Diving bradycardia during rest and exercise and its relation to physical fitness. J Appl Physiol 1970; 28: 614-621.

18. Gooden BA, Feinstein R, Skutt HR. Heart rate response of scuba divers via ultrasonic telemetry. Undersea Biomed Res 1975; 2: 11-19.

19. Wolf S, Schneider RA, Grover ME. Further studies on the circulatory and metabolic alterations of the oxygen-conserving (diving) reflex in man. Trans Assn Am Physns 1965; 78: 242-254.

20. Gooden BA, Holdstock G, Hampton JR. The magnitude of the bradycardia induced by face immersion in patients convalescing from myocardial infarction. Cardiovas Res 1978; 12: 239-242.

21. Kawakami Y, Natelson BH, DuBios AB. Cardiovascular effects of face immersion and factors affecting diving reflex in man. J Appl Physiol 1967; 23: 964-970.

22. Corriol J, Rohner JJ. Role de la temperature de l'eau dans la bradycardie d'immersion de la face. Arch Sci Physiol 1968; 22: 265-274.

23. Song SH, Lee WK, Chung YA, Hong SK. Mechanisms of apneic bradycardia in man. J Appl Physiol 1969; 27: 323-327.

24. Hong SK, Moore TO, Lally DA, Morlock JF. Heart rate response to apneic face immersion in hyperbaric heliox environment. J Appl Physiol 1973; 34: 770-774.

25. Brick I. Circulatory response to immersing the face in water. J Appl Physiol 1966; 21: 33-36.

26. Asmussen E, Kristiansson NG. The "diving bradycardia" in exercising man. Acta Physiol Scand 1968; 73: 527-535.

27. Paulev PE. Cardiac rhythm during breath-holding and water immerison in man. Acta Physiol Scand 1968; 73: 139-150.

28. Hong SK, Rahn H, Kang DH, Song SH, Kang BS. Diving pattern, lung volumes and alveolar gas of the Korean diving women (AMA). J Appl Physiol 1963; 18: 457-465.

29. Angelone A, Coulter NA Jr. Heart rate response to held lung volume. J Appl Physiol 1965; 20: 464-468.

30. Olsen CR, Fanestil DD, Scholander PF. Some effects of breath-holding and apneic underwater diving in man. J Appl Physiol 1962; 17: 461-466.

31. Olsen CR, Fanestil DD, Scholander PF. Some effects of apneic underwater diving on blood gases, lactate, and pressure in man. J Appl Physiol 1962; 17: 938-942.

32. Moore TO, Elsner R, Lin YC, Lally DA, Hong SK. Effects of alveolar PO_2 and PCO_2 on apneic bradycardia in man. J Appl Physiol 1973; 34: 795-798.

33. Lin YC, Shida KK, Hong SK. Effects of hypercapnia, hypoxia and rebreathing on heart rate response during apnea. J Appl Physiol 1983; 54: 166-171.

34. Paulev PE, Hansen HG. Cardiac response to apnea and water immersion during exercise in man. J Appl Physiol 1972; 33: 193-198.

35. Bergman SA Jr, Campbell JK, Wildenthal K. "Diving reflex" in man: its relation to isometric and dynamic exercise. J Appl Physiol 1972; 33: 27-31.

36. Bove AA, Lynch PR, Connell JV Jr, Harding JM. Diving reflex after physical training. J Appl Physiol 1968; 25: 70-72.

37. Whayne TF Jr, Killip TK III. Simulated diving in man: Comparison of facial stimuli and response in arrhythmia. J Appl Physiol 1967; 22: 800-807.

38. Stromme SB, Ingjer F. Comparison of diving bradycardia and maximal aerobic power. Aviat Space Environ Med 1978; 49: 1267-1270.

39. Hong SK, Moore TO, Seto G, Park HK, Hiatt WR, Bernauer EM. Lung volumes and apneic bradycardia in divers. J Appl Physiol 1970; 29: 172-176.

40. Harding PE, Roman D, Whelan RF. Diving bradycardia in man. J Physiol 1965; 181: 401-409.

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

42. Ferrigno M. Cardiac performances during breath hold diving. In: Lundgren C, ed. Proceedings of the Workshop on the Physiology of Breath-hold Diving. Undersea Med Soc: Bethesda, 1985.

43. Gross PM, Terjung RL, Lohman TG. Left-ventricular performance in man during breath-holding and simulated diving. Undersea Biomed Res 1976; 3: 351-360.

44. Heistad DO, Abboud FM, Eckstein JW. Vasoconstrictor response to simulated diving in man. J Appl Physiol 1968; 25: 542-549.

45. Heistad DO, Wheeler RC. Simulated diving during hypoxia in man. J Appl Physiol 1970; 28: 652-656.

46. Hong SK, Lin YC, Lally DA, Yim BJB, Kominami N, Hong PW, Moore TO. Alveolar gas exchanges and cardiovascular functions during breath holding with air. J Appl Physiol 1971; 30: 540-547.

47. Lin YC, Shida KK, Hong SK. Effects of hypercapnia, hypoxia and rebreathing on circulatory response to apnea. J Appl Physiol. 1983; 54: 172-177.

48. Whayne TF, Smith NTY, Eger EI II, Stoelting RK, Whitcher CE. Reflex cardiovascular responses to simulated diving. Angiologia 1972; 23: 500-508.

49. Folinsbee A. Cardiovascular response to apneic immersion in cool and warm water. J Appl Physiol 1974; 36: 226-232.

50. Paulev PE, Wettergvist H. Cardiac output during breath-holding in man. Scand J Clin Lab Invest 1968; 22: 115-123.

51. Bjertnaes L, Hauge A, Kjekshus J, Soyland E. Cardiovascular responses to face immersion and apnea during steady state muscle exercise. A heart catheterization study on humans. Acta Physiol Scand 1984; 120: 605-612.

52. Arnold RW. Extremes in human breath hold, facial immersion bradycardia. Undersea Biomed Res 1985; 12: 183-190.

53. Speck DF, Bruce DS. Effects of varying thermal and apneic conditions on the human diving reflex. Undersea Biomed Res 1978; 5: 9-14.

54. Craig AB Jr. Effects of submersion and pulmonary mechanics on cardiovascular function in man. In: Rahn H, Yokoyama T, eds. Physiology of breath-hold diving and the AMA of Japan. NAS-NRC Publication 1341, 1965; 295-302.

Discussion following Dr. Hong's presentation

Dr. Ferrigno: You talked about the relationship between lung volume and heart rate, and you also said that when the subject performed a Valsalva maneuver, the bradycardial response was reduced, and conversely, when he did the Mueller maneuver, the bradycardia was accentuated.

Dr. Hong: Yes. In fact, during the Valsalva maneuver, even a tachycardia develops.

Dr. Ferrigno: Of course even if the subject did not intend to do a Valsalva or Muller maneuver, but he just held his breath at different lung volumes with relaxed respiratory muscles, that itself would have influenced the intrathoracic pressure, so would you comment on the effect of lung volume on the heart rate, independently from the intrathoracic pressure.

Dr. Hong: There are two notions. One notion is that when we start the breath-holding following a maximal inspiration, of course, intrathoracic pressure becomes more negative during the act of maximal inspiration, which could play a role by increasing the venous return. According to Dr. Craig's model, this would potentiate the bradycardial response. The other possibility is that there is a paper indicating the presence of some sort of intrathoracic receptors sensitive to the lung volume which modulate the heart rate.

Dr. Arnold: The effect of lung volume has been studied with measurement of intrathoracic pressure and without, and when intrathoracic pressure is maintained constant, lung volume still has the effect that Dr. Hong mentioned.

Dr. Craig: Dr. Hong, as you mentioned, you can change the heart rate during apnea by changing position. I think an awful lot of results can be explained in terms of baroreceptor response in light of changing arterial resistance and the other aspects of changing venous return and cardiac output.

Dr. Hong: In my last slide I mentioned the mechanical effect involving baroreceptors.

Dr. Craig: I'm not sure we have to look for some triggering mechanism. It would just depend on the usual physiology.

Dr. Lin: We talk about baroreceptor that can modify the response, but not triggering the response, and we determined it in this way. We did some experiments with rats where we depleted their catecholamines and we dunk his head in the water, now his blood pressure goes down, but the bradycardial response still appears. Normally when you dunk the head of the rat in the water, you would have bradycardia and the blood pressure rise, but now you decreased its catecholamines, and doing the same thing, blood pressure goes down, the bradycardia is still there. Therefore, we conclude that the baroreceptor activity has nothing to do with initiation, however this does not deny its modifying role.

Dr. Arnold: The Cold Pressor Test can cause essentially the same amount of peripheral vasoconstriction and the heart rate stays the same.

Dr. Hong: Yes, the blood pressure goes up tremendously, but it alone doesn't necessarily potentiate the breath-hold bradycardia.

Dr. Ferrigno: Dr. Hong, you showed also that the response of the heart rate related to the temperature of the water. Some Japanese researchers (Kobayasi, S. and T. Ogawa. Effect of water temperature on bradycardia during nonapneic facial immersion. Jap. J. Physiol. 23:613-624, 1973) did experiments with water warmer than thermoneutral, which is 35^{o}C at rest and they saw that the trend was inverted at higher temperatures; instead of seeing a smaller bradycardial response, the response was increasing again. Will you comment on that?

Dr. Arnold: I looked at that. On either end of the spectrum, in ice water and above thermoneutral, nociceptors are activated, whereas between about 27 degrees and 5 degrees cold receptors, and between 25 degrees and 35 warm receptors are activated. So nociceptive receptors are probably important afferents in the diving response at low temperatures and at temperatures above thermoneutral.

CARDIAC PERFORMANCE DURING BREATH-HOLD DIVING IN MAN: AN OVERVIEW.

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The classical description of the diving response, elicited in diving animals by apnea and submersion, includes a vagally induced bradycardia, a sympathetically controlled peripheral vasoconstriction and a decreased cardiac output. A diving response, although attenuated or modified, and especially the diving bradycardia, have been described also in man and a review of this topic was recently done by Elsner and Gooden (9), Lin (31), Hickey and Lundgren (22) and by Hong (23).

With regard to the effects of breath-holding on cardiac output in man, earlier studies have shown differing results. Kawakami and his colleagues (27) measured right cardiac output with a N_2O-plethysmographic method in seven subjects during breath-holding with face immersion. In this condition stroke index and cardiac index decreased by 14% and 22%, respectively. The decrease in cardiac output was accompanied by a significant decrease in heart rate, more pronounced when the subjects' face was immersed in cold rather than in cool water. Paulev and Wetterqvist (33), using a dye-dilution technique, showed a 20-40% reduction in cardiac output in four subjects holding their breath with relatively high intrapulmonic pressures. In contrast, cardiac output was unchanged or increased in four other subjects who held their breath with intrapulmonic pressures around zero. Hong and his colleagues (25) did not observe any changes in cardiac output, measured by the Cardio-green dilution method, during 2 minute breath-holds. In their experiments, during which their subjects rebreathed into a bag for sequential alveolar gas sampling, heart rate tended to decrease, while stroke volume tended to increase. Whayne and his colleagues (42) measured, by means of a ballistocardiogram (Beg)-analog computer system, stroke volume and cardiac output in eight subjects who held their breath with a cold cloth (0°C) on their face. After an initial transient increase, cardiac output decreased because of a parallel change in heart rate. Lin, Shida and Hong in 1983 (32) obtained stroke volume with an impedance cardiogram during 90 second breath-holds with face immersion in 22-24°C water, and they observed a 33% increase in stroke volume which was counterbalanced by a 30% reduction in heart rate. One year later, Bjertnaes and his colleagues (3), using the thermodilution technique, observed a 49% reduction in cardiac output with face immersion in ice water and apnea during steady state exercise.

Some of these discrepancies were probaly caused by the presence, or absence, of the cold-induced circulatory components of the diving response (28). Another important consideration is the possibility that differences in the intrathoracic pressures of the subjects in the various studies were responsible for the dissimilar cardiac output measurements during breath-holding.

It is predictable that if a subject takes a deep inspiration and holds his breath with closed glottis and relaxed respiratory muscles, his intrathoracic pressure will increase. This increase in intrathoracic presure is caused by the

inward chest-wall recoil at large lung volume, and it is likely to hinder venous return and therefore reduce cardiac output. However, the situation probably changes during actual diving. Once the subject starts his descent underwater, the increasing hydrostatic pressure surrounding him will compress his chest. Because of this, the inward recoil of his chest will decrease, and, at a sufficient depth, it will even be reversed, thus reducing the subject's intrahoracic pressure. Consequently, his venous return should no more be impeded and his cardiac output should be either restored to, or increased over, pre-breath-hold levels.

Submersion, which is an integral part of breath-hold diving, is also likely to influence venous return (2,10). As you can see in Fig. 1, the water pressure counteracts the blood pooling in the dependent regions, which is present in air and due to the gravity force. The hydrostatic pressure on the outside of the body will redistribute blood into the thorax and the filling of the heart will be increased. Arborelius and his colleagues (2) observed in three subjects, during head-out immersion in 35°C water, a mean increase in central blood volume by 700 ml. In the same conditions, they also showed a 32% increase in cardiac output in ten subjects, with a 35% increase in stroke volume, while heart rate did not change.

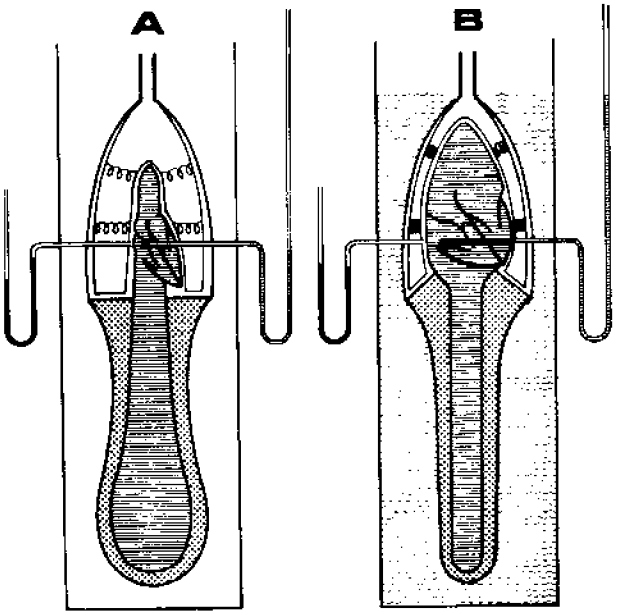

Fig. 1. Schematic illustration of the effect of immersion with the head above water on the distribution of blood between dependent regions of the body and the thorax. The springs indicate the elasticity of the lung tissue. Dotted areas: incompressible but resilient tissues. Hatched areas: blood. The left manometer (black fluid column) indicates the pleural pressure, the right mamometer (hatched fluid column) indicates the right atrial pressure. The difference between right atrial pressure and pleural pressure reflects atrial transmural pressure gradient. A: erect body position in the non immersed situation; blood pooled in the vascular bed below the heart. Atrial pressure is almost zero and pleural pressure is negative. Right atrial transmural pressure gradient is small. B: erect body position during immersion with the head above water; redistribution of blood towards the heart and intrathoracic vascular bed, distending these structures. Atrial pressure is raised and pleural pressure almost zero. Right atrial transmural pressure gradient is increased. From (2) with permission.

In our laboratory (12) we used an impedance cardiograph to measure cardiac output and systolic time intervals in six subjects, while they were holding their breath at 80 % of their vital capacity, in both the dry condition or submersed in water. In both situations the ambient temperature was maintained thermoneutral, i.e., 28°C air and 35°C water (cf 6), in order to minimize the circulatory aspects of the cold-induced diving response. We also measured intrathoracic pressure with an esophageal balloon. Figure 2 shows cardiac output, expressed as cardiac index, and esophageal pressure, which represents intrathoracic pressure, in the course of 75 second breath-holds, in both the dry and the submersed condition. Cardiac index, which was always larger in the submersed compared to the corresponding dry situation, was decreased throughout breath-holding. These changes in cardiac output were caused by similar changes in stroke volume, as heart rate did not show any modification. On the other hand, intrathoracic pressure was significantly increased while the subjects were holding their breath, and this was probably responsible for the decrease in cardiac output, by hindering venous return. Breath-holding after maximal inspiration has been shown by sonography to cause an enlargement of the inferior vena cava, reflecting an impediment of blood flow into the chest (17). The 20-25% reduction in cardiac index that we observed during breath-holding at large lung volume appears to be in agreement with the 20-40% fall in cardiac output mentioned earlier, which was found by Paulev and Wetterqvist (33) in subjects who induced high intrapulmonic pressures by performing the Valsalva maneuver.

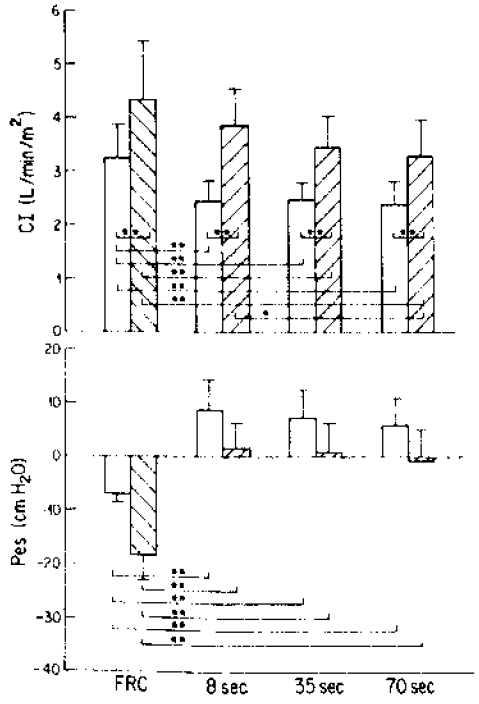

Fig. 2. Cardiac index (CI) and esophageal pressure (Pes) in the control condition (FRC) and at different times during large lung volume breath-holding in the dry (non shaded bars) and in the submersed condition (shaded bars). Values are means ± SD of averages of 3 experiments in each of 6 subjects. * P<0.05; ** P<0.01. From (12) with permission.

Changes in left ventricular function during breath-holding, as reflected by variations of systolic time intervals, have been the subject of five studies (12,15,18,26, 35). The common observation was a depression of left ventricular performance, expressed as a combination of a shortening of left ventricular ejection time (LVET) (12,15,18,35), a lengthening of pre-ejection period (PEP) (12,15 and 18) and an increase of PEP/LVET (12,15,18,26). In our study (12) the depression of cardiac fuction during breath-holding at large lung volume was

also apparent from the above described reductions in stroke volume and cardiac output. Despite the common findings, the interpretations by the various authors of the mechanisms underlying the changes in the systolic time intervals were different.

Table 1, modified from Gross and colleagues (18), shows the factors influencing systolic time intervals and, on the right of the table, the variations of the hemodynamic factors needed to explain the described changes in systolic time intervals during breath-holding, i.e., a lengthening of PEP and a shortening of LVET. Heart rate was not included in the factors influencing the systolic time intervals, as both PEP and LVET were corrected for it, using the regression equations of Weissler et al. (41).

Frey and Kenney (15) and Gross and his colleagues (18) suggested that venous return was increased during breath-holding. They reasoned that the resulting increase of preload should have led to a shortening of PEP and a lengthening of LVET. However, as the observed changes of the systolic time intervals were in the opposite direction, Gross and his colleagues hypothesized that these changes had been caused by a combination of the measured increase in diastolic pressure (18) and of a suggested reduction in the inotropic status of the heart, similar to the one demonstrated in natural divers (11,14). On the other hand, Frey and Kenney suggested a late-occurring decrease in venous return or some other secondary reflex-induced effects (15). A decrease in contractility was also suggested by Jacopin and his colleagues (26), who observed an increase in PEP/LVET in 20 subjects holding their breath with a cold cloth on their face. Such an increase in PEP/LVET has been related to a smaller ejection fraction (16).

Table 1. Modified from (18)

Factors that influence systolic time intervals

Hemodynamic factor	Relationship with:		Response needed to:	
	PEP*	LVET*	↑PEP	↓LVET
Preload *(ventricular filling)*	Varies indirectly	Varies directly	Decrease	Decrease
Afterload *(aortic diastolic pressure)*	Varies directly	Varies directly	Increase	Decrease
Inotropic state	Varies indirectly	Varies indirectly	Decrease	Increase

PEP = preejection period
LVET = left-ventricular ejection time
*Harris 1974

We proposed a differrent explanation for the observed changes in PEP and LVET during breath-holding: the lengthening of PEP and the shortening of LVET indicate, in table 1, a reduced preload which was probably caused by an impediment of venous return, resulting from holding the breath at a large lung volume (12). Breath-holding at about 80 % of vital capacity, which is the lung volume typically used by breath-hold divers such as the diving women of Korea

(24), can on theoretical grounds be expected to generate an intrathoracic pressure of about 6 cm H_2O (Ref 1). This is in agreement with our observation of an esophageal pressure of about 7 cm H_2O (12), and this increased intrathoracic pressure probably impeded venous return, and therefore reduced cardiac output.

Further support to the suggestion that changes in venous return were responsible for the modifications of the systolic time intervals during breath-holding, might be derived from the different temporal pattern of changes, described by Frey and Kenney (15), in what they called the inotropic and the chronotropic response to apneic face immersion. These authors observed that the appearance of the change in left ventricular performance, as shown by an increase in PEP, lagged behind that of bradycardia. We suggested that this temporal separation was the result of the different nature of the two responses (12): the negative chronotropic influence occurred earlier because it was reflex mediated, while it took longer time for the change in PEP to become evident, as it was induced by a gradual reduction in venous return.

In our hyperbaric chamber we also measured cardiac function and intrathoracic pressure in six subjects during simulated breath-hold dives to 20 meters (13), a depth routinely reached by the Japanese female divers (39). Experiments were performed in both the dry and the submersed condition and this is, to our knowledge, the only study of cardiac performance in man during breath-hold diving. Cardiac output, which was decreased by 20-25% during breath-holding at the surface, was restored to control values at depth. Furthermore, the changes in systolic time intervals, induced by breath-holding, had been reversed by compression. We suggested that this restoration of cardiac performance, when the breath-holding subjects were compressed to 20 meters, was the result of the concomitant fall in intrathoracic pressure which was predictable from the compression of the chest to one third of its original volume. Some additional volume reduction probably resulted from alveolar gas exchange at depth (30). Therefore, with the fall in intrathoracic pressure the impediment to venous return, which had been present at the surface, was removed and cardiac output was restored.

It is of interest to consider how the chest wall recoil, responsible for the intrathoracic pressure changes during breath-hold diving, was influenced by the dry and the submersed dives. Figure 3 serves such a theoretical comparison. It shows schematic pressure-volume curves for the chest wall during relaxation in the dry condition (continuous line) and in head-out immersion (dotted line) (modified from Agostoni et al., 1) It also depicts the predicted positions of a subject's lungs on these curves, during breath-hold diving in the dry and in the submersed mode, respectively. An important difference between the dry and the submersed condition is that intrathoracic blood volume is likely to be larger during submersion. The pulmonary air volumes used in designing Fig. 3 were the ones employed in our experiments (13): residual volume plus 80% of "dry" vital capacity in the non-submersed condition and residual volume plus 85% of "wet" vital capacity in the submersed condition. Thus, the difference between dry and immersed vital capacity measurements was considered, and the subject initiated the dry and submersed dives with essentially the same absolute volume of air in his lungs.

For the purpose of the calculations on which Fig. 3 is based, the mean values for lung volumes recorded in our study (13) were used, i.e., a "dry" vital capacity of 5.7 L and a residual volume of 1.5 L. For simplicity, changes

in pulmonary air volume due to gas exchange with the blood during the dives were disregarded. Compression during the dry dives would reduce the air volume in the chest (80% of "dry" vital capacity + residual volume) so as to move the chest wall roughly to the 10% of "dry" vital capacity level. The corresponding drop in esophageal pressure (ΔPes between the two horinzontal markings on the continuous line, Fig. 3) would amount to about 28 cm H_2O. In the case of the submersed dives, the assumption was made that 0.7 L of blood (ΔV_b) had been redistributed from the periphery into the chest as a result of submersion. This figure was based on previously mentioned measurements performed on subjects undergoing head-out immersion at functional residual capacity level (2). The V_b of 0.7 L is probably smaller than the actual blood shift during our submersed experiments, because they involved compression and the intrathoracic pressures generated were considerably lower than those recorded by Arborelius and his colleagues (2). In all likelihood, the lower pressure would cause a larger blood shift into the thorax during the submersed dives. The "wet" vital capacity was taken as 94% of the "dry" (29) and "wet" residual volume was considered to be reduced by 0.15 L (7) during head-out immersion. For the purpose of simplyfing Fig. 3, it was furthermore assumed that the volume of 0.7 L of blood was redistributed into the chest at the beginning of compression in the submersed dives. In reality, a more likely occurrence would be for the blood redistribution to gradually become complete during the descent, as intrathoracic pressure falls due to changing chest wall recoil. However, this makes no difference in the final analysis. In the submersed condition, the chest would have to accomodate the same gas volume as in the dry situation, plus the 0.7 L of extra blood volume, therefore behaving as if the initial lung volume corresponded to 92% of "dry" vital capacity. In this case, the air volume in the lungs, having been reduced to 22% of "dry" vital capacity by the compression to 20 m (3 ATA), would generate a drop in intrathoracic pressure of about 15 cm H_2O (ΔPes between the two horizontal markings on the dotted line, Fig. 3), as opposed to the pressure drop of 28 cm H_2O in the dry dives. The difference of 13 cm H_2O in the pressure drop between dry and submersed dives, arrived at by

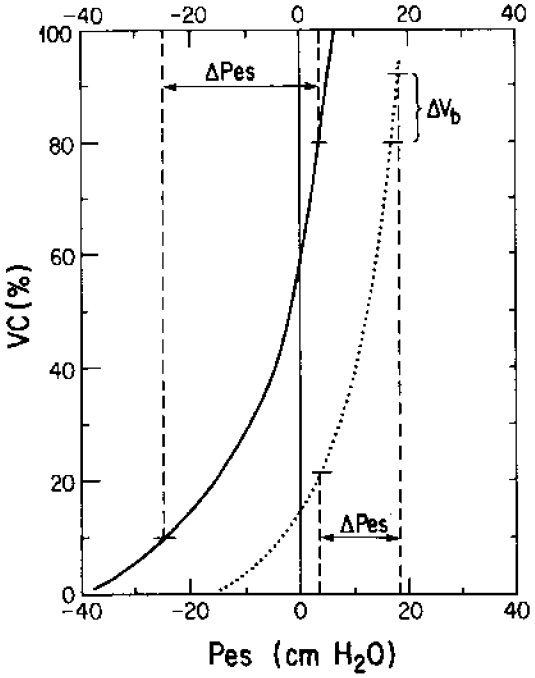

Fig. 3. Predicted changes in lung volume and intrathoracic pressure during breath-hold diving to 20 m. The schematic pressure (Pes)-volume (% VC) curves for non immersion (continuous line) and immersion (dotted line) were adapted from Agostoni et al (1). The upper horizontal markings on the curves denote starting volumes and pressures, and the lower markings indicate volumes and pressures at 20 m. ΔV_b denotes volume of blood redistributed into the thorax during immersion. For details, see text.

these theoretical considerations, should be compared with the difference of 8.2 cm H_2O, which was calculated from our measurements (13). The fact that the latter is somewhat smaller than the predicted difference might be due to the use of a fixed reference pressure in the submersed compression experiments. In the submersed mode, esophageal pressure was referenced to the one obtained with an air-containing balloon placed 14 cm below the sternal notch. This position roughly corresponds to the pressure centroid of the chest at a lung volume equal to "dry" functional residual capacity (cf 40). As the lungs are compressed and the diaphragm elevated, the pressure centroid is likely to shift upward, and this would require adjusting the position of the reference balloon during the dives. However, this was not done because it was impractical (13).

Another important factor that might help to explain the fact that the intrathoracic pressure fall was more pronounced in the dry (about 29 cm H_2O) than in the submersed dives (about 21 cm H_2O), is the possibility of juxtathoracic venous collapse during the dry dives (13). This well known phenomenon (19,20,34) consists in the collapse of the veins entering the thorax, once the intrathoracic pressure falls below zero, and it can be understood in light of the simple fact that it is impossible to suck fluid through a collapsible tube. Because of juxtathoracic venous collapse, any further decrease in intrathoracic pressure will not induce a proportional increase in venous return and in intrathoracic blood pooling. In other words, with less blood pooling in the thorax making up for the reduction of the air volume in the lungs during diving, the chest will be compressed to a smaller volume. At this smaller volume there will be a more forceful outward recoil of the chest-wall and therefore a lower intrathoracic pressure.

Juxtathoracic venous collapse is unlikely during compression of the subjects in the submersed situation (13), because in this case the veins entering the thorax are probably kept open, as the water pressure squeezes blood into the chest (4). Under these conditions, the abdominal (perivenous) pressure near the diaphragm is about 12 cm H_2O higher than atmospheric pressure (1), whereas the downstream pressure in the central veins is even higher (2,8), thus making juxtathoracic venous collapse unlikely. In support of this concept are observations by Schaefer and his colleagues of the effects of simulated breath-hold dives to 90 feet in the dry and in the wet chamber, on blood shifts into the thorax (36). Using an impedance plethysmograph they were able to detect a gradual decrease in "thoracic electrical resistance" during dives in the wet chamber, and they felt that this indicated an increasing intrathoracic blood shift. On the other hand, the dives performed in the dry chamber did not cause any resistance changes.

Schaefer and his colleagues, using again the impedance plethysmograph method, tried to estimate intrathoracic blood volume changes in open-sea breath-hold dives. They reported that at 90 and 130 feet of depth blood was forced into the thorax in the amount of 1047 and 850 ml, respectively (37). These observations are in agreement with the suggestion by Craig that 600 ml of blood had been moved into his thorax during his breath-hold dive to 4.75 m. Craig initiated his descent at residual volume, his dive mimicking, from a physiological point of view, a much deeper dive initiated at total lung capacity (5). His measurement of "transthoracic pressure" and our measurement of intrathoracic pressure in the submersed condition were similar, because in both cases the esophageal pressure was referenced to the pressure of a balloon attached to the thoracic wall. Craig observed that transthoracic pressure was independent of depth during his descent and, therefore, he suggested that

transfer of blood into the thorax had made up for compression of lung air, thus preventing a decrease in intrathoracic pressure. This apparent discrepancy with our observation of an intrathoracic pressure decrease at depth, despite the likely occurrence of intrathoracic blood shift, can probably be explained by the differrent experimental conditions. In particular, our subjects started their breath-hold dives at large lung volume and, therefore, with relatively high intrathoracic pressure (13). Measurements of cardiac performance that we performed during breath-holding at different lung volumes (12) suggest that intrathoracic blood volume is probably larger during breath-holding at small lung volumes.

The possibility of juxtathoracic venous collapse during dry compression leads to important safety considerations in the simulation of breath-hold diving. It may be that the risk of thoracic squeeze is greater when an experimental breath-hold dive is conducted in a dry chamber than when a breath-hold dive is made, to the same depth, but in the submersed condition. In the latter situation there is a smaller intrathoracic pressure fall (13), indicating less chest compression. However, in the submersed dives a larger redistribution of blood into the thorax might cause pulmonary edema and/or dilatation of the heart (cf 38).

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REFERENCES

1. Agostoni, E., G. Gurtner, G. Torri and H. Rahn. Respiratory mechanics during submersion and negative-pressure breathing. J. Appl. Physiol. 21:251-258, 1966.

2. Arborelius, M. Jr., U.I. Balldin, B. Lilja and C.E.G. Lundgren. Hemodynamic changes in man during immersion with the head above water. Aerospace Med. 43:592-598, 1972.

3. Bjertnaes, L., A. Hauge, J. Kjekshus and E. Soyland. Cardiovascular responses to face immersion and apnea during steady state muscle exercise. A heart catheterization study on humans. Acta Phyisol. Scand. 120:605-612, 1984.

4. Bjurstedt, H., G. Rosenhamer, C.M. Hesser and B. Lindberg. Response to continuous negative pressure breathing in man at rest and during exercise. J. Appl. Physiol. 48:977-981, 1980.

5. Craig A.B. Jr. Depth limits of breath-hold diving. Respiration Physiology 1968; 5:14-22.

6. Craig, A.B. Jr. and M. Dvorak. Thermal regulation during water immersion. J. Appl. Physiol. 21:1577-1585, 1966.

7. Dahlback G.O., Jonsson E. and M.H. Linér. Influence of hydrostatic compression of the chest and intrathoracic blood pooling on static lung mechanics during head-out immersion. Undersea Biomed Res 1978; 5:71-85.

8. Echt, M., L. Lange and O.H. Gauer. Changes of peripheral venous tone and central transmural venous pressure during immersion in a thermo-neutral bath. Pfluegers Arch. 352:211-217,1974.

9. Elsner, R. and B. Gooden. Diving and asphyxia. New York: Cambridge University Press, 1983.

10. Farhi, L.E. and D. Linnarsson. Cardiopulmonary readjustments during graded immersion in water at 35°C. Resp. Phys. 30:35-50, 1977.

11. Ferrante, F.L. and D.F. Opdyke. Mammalian ventricular function during submersion asphyxia. J. Appl. Physiol. 26:561-570, 1969.

12. Ferrigno M., Hickey D.D., Linér M.H. and C.E.G. Lundgren. Cardiac performance in humans during breath-holding. J Appl Physiol 60:1871-1877,1986.

13. Ferrigno, M., D.D. Hickey, M.H. Linér and C.E.G. Lundgren. Simulated breath-hold diving to 20 meters: cardiac performance in man. J. Appl. Physiol. 00:0000-0000, 1987.

14. Folkow, B., B. Lisander and B. Oberg. Aspects of the cardiovascular nervous control in a mammalian diver (Myocastor coypus). Acta Physiol. Scand. 82:439-446, 1971.

15. Frey, M.A.B. and R.A. Kenney. Changes in left ventricular activity during apnea and face immersion. Undersea Biomed. Res. 4:27-37, 1977.

16. Garrard C.L. Jr., Weissler A.M. and H. Dodge. The relationship of alterations in systolic time intervals to ejection fraction in patients with cardiac disease. Circulation 1970; 9:455-462.

17. Grant, E., F. Rendano, E. Sevinc, J. Gammelgaard, H.H Holm and S. Gronvall. Normal inferior vena cava: caliber changes observed by dynamic ultrasound. AJR 135:335-338, 1980.

18. Gross, P.M., R.L. Terjung and T.G. Lohman. Left-ventricular performance in man during breath-holding and simulated diving. Undersea Biomed. Res. 3:351-360, 1976.

19. Guyton, AC. Venous return. In: Handbook of Physiology. Circulation. Washington, DC: Am. Physiol. Soc.; sect. 2,vol. 2, chapt. 32; 1963:1099-1133.

20. Guyton, A.C., A.W. Lindsey, B. Abernathy and T.Q. Richardson. Venous return at various right atrial pressures and the normal venous return curve. Am. J. Physiol. 189:609-615, 1957.

21. Harris, W.S. Systolic Time Intervals in the Noninvasive Assessment of Left Ventricular Performance in Man. Chap. 9 In: Mirsky, I., Ghista, D.N. and H. Sandler, eds. Cardiac Mechanics: Physiological, Clinical and Mathematical Considerations. John Wiley and Sons, Inc. 1974:233-292.

22. Hickey, D.D. and C.E.G. Lundgren. Breath-hold diving. Chapter section in: The Physician's guide to diving medicine. Editors: Shilling,C.W., Carlston,C.B. and Mathias,R.A. New York: Plenum Press; 1984:206-221

23. Hong, S.K. Breath-hold bradycardia in man: an overview. In: Lundgren C.E.G. and M. Ferrigno, eds. Proceedings of the Workshop on the Physiology of Breath-hold Diving in Man. Undersea and Hyperbaric Medical Society, Bethesda, MD, 1987.

24. Hong,S.K. Hae-Nyo, The Diving Women of Korea. In: Physiology of Breath-hold Diving and the Ama of Japan. H. Rahn, ed. Washington,D.C.: National Academy of Sciences, 1965; Publication 1341: 99-111

25. Hong, S.K., Y.C. Lin, D.A. Lally, et al. Alveolar gas exchanges and cardiovascular functions during breath holding with air. J. Appl. Physiol. 30:540-547, 1971.

26. Jakopin, J., B. Pohar and B. Sasel. Delovanje srca in ozilja pri prostem potapljanju. [Cardiovascular function in breath-holding]. Pomorska Medicina III, Belgrade, Yugoslavia, Pomorska Biblioteka (Sveska 33), 267-274, 1983. (English Summary).

27. Kawakami, Y., B.H. Natelson and A.B. DuBois. Cardiovascular effects of face immersion and factor affecting diving reflex in man. J. Appl. Physiol. 23:964-970, 1967.

28. Kobayasi, S. and T. Ogawa. Effect of water temperature on the bradycardia during nonapneic facial immersion in man. Jap. J. Physiol. 23:613-624, 1973.

29. Kurss, D.I., C.E.G. Lundgren and A.J. Pasche. Effect of water temperature on vital capacity in head-out immersion. In: A.J. Bachrach and M.M. Matzen,

eds. Underwater Physiology VII. Proceedings of the 7th Symposium on Underwater Physiology. Bethesda: Undersea Med. Soc., 1981, pp. 297-301.

30. Lanphier E.H. and H. Rahn. Alveolar gas exchange during breath-hold diving. J Appl Physiol 1963; 18:471-477.

31. Lin, Y.C. Circulatory functions during immersion and breath-hold dives in humans. Undersea Biomed. Res. 11:123-138, 1984.

32. Lin, Y.C., K.K. Shida and S.K. Hong. Effects of hypercapnia, hypoxia, and rebreathing on circulatory response to apnea. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 54:172-177, 1983.

33. Paulev, P. and H. Wetterqvist. Cardiac output during breath holding in man. Scand. J. Clin. Lab. Invest. 22:115-123, 1968.

34. Permutt, S. and R.L. Riley. Hemodynamics of collapsible vessels with tone: the vascular waterfall. J. Appl. Physiol. 18:924-932, 1963.

35. Rodgers, K.L., P.R. Lynch and B.D. Franks. Effects of apnea and face immersion in water on the time components of the cardiac cycle. Res. Quarterly 47:783-787, 1976.

36. Schaefer K.E., Allison R.D., Carey C.R. and R. Strauss. The effects of simulated breathholding dives in the dry and wet chambers on blood shifts into the thorax. Naval Submarine Medical Research Laboratory: Report No. 729, 26 October 1972

37. Schaefer, K.E., R.D. Allison, J.H. Dougherty, Jr., C.R. Carey, R. Walker, F. Yost and D. Parker. Pulmonary and Circulatory Adjustments Determining the Limits of Depth in Breathhold Diving. Science 162:1020-1023,1968.

38. Stigler, R. Die Kraft unserer Inspirationsmuskulatur. Pflugers Arch 1911; 139:234-254.

39. Teruoka, G. Die Ama und ihre Arbeit. Arbeitsphysiologie 5:239-251, 1932.

40. Thalmann, E.D., D.K. Sponholtz and C.E.G. Lundgren. Effects of immersion and static lung loading on submerged exercise at depth. Undersea Biomed. Res. 6:259-290, 1979.

41. Weissler, A.M., W.S. Harris and C.D. Schoenfeld. Systolic time intervals in heart failure in man. Circulation 37:149-159, 1968.

42. Whayne, T.F. Jr., N.T. Smith, E.I. Eger, R.K. Stoelting and C.E. Whitcher. Reflex cardiovascular response to simulated diving. Angiology 23:500-508, 1972.

Discussion following Dr. Ferrigno's presentation

Dr. Elsner: If I understand correctly, the left ventricular ejection time increased in the dive, is that right?

Dr. Ferrigno: Yes.

Dr. Elsner: That would suggest that there was an increase in contractility.

Dr. Ferrigno: The fact is that the systolic time intervals are not specifically a measurement of contractility, which is only one of the determinants of cardiac performance. In the table I showed you, the coupling of the increase in left ventricular ejection time and the decrease in pre-ejection period observed during compression of the breath-holding subjects, suggests an increase in preload. Getting back to your question, it is just the opposite. An increase in contractility will shorten the left ventricular ejection time, if all the other determinants of cardiac performance are kept constant, because the contraction will be faster. It will also shorten the pre-ejection period.

Dr. Elsner: I asked this because in ducks, in diving seals, in diving dogs, in diving nutria those are the four species we know about, contractility during the dive as measured by dP/dt max is decreased by 20 to 50 percent.

Dr. Ferrigno: Yes, that's a very interesting observation. In diving mammals invasive measurements of contractility have been done and there is sometimes a problem when they measure the dP/dt max: this index should be corrected for the end-diastolic ventricular volume because an increase in preload will cause an increase in cardiac performance but not an increase in contractility.

Dr. Elsner: For the seals we had a better idea of what the volumes were and there's no change in left ventricular end-diastolic pressure until way up at the end of the longest dives.

Dr. Ferrigno: There is also a difference in the vagal innervation of the ventricles in man and in some diving animals. These diving animals have a definite vagal innervation of the ventricles, while in man it doesn't seem particularly pronounced.

Dr. Boysen: I have a question, but let me just say something first. There's a clinical quota of what you've done, that has come out over the last year in patients who are undergoing extra-corporeal shock waves for lithotripty, and these patients are worse when immersed head-out in water. The idea was that they had to be immersed in water to be able to better tolerate the shock waves, and if you do that on patients who have borderline cardiac function, it is reasonable to use a Swan-ganz catheter to monitor their cardiac function during this procedure, and we've done that. And when you immerse these patients, usually up to no more than the suprasternal notch, you see a dramatic increase in pulmonary capillary wedge pressure, as an index of left ventricular diastolic pressure. These

patients would probably go in left heart failure, except that the bath is at 37 degrees C., and so while they had a tremendous increase in preload, they were vasodilated, so at the same time they decreased their afterload. Furthermore, during the procedure over 45 minutes it's not unusual to see diuresis, and presumably this is actually therapeutic for their congestive heart failure. So my question regards water temperature and you alluded to that. If you put the same patients in the cold water and they vasoconstrict, presumably you can cause pulmonary edema.

Dr. Lundgren: I wanted to show this because it bears on what we just heard and it bears on our earlier discussions about the importance of blood redistribution into the thorax to allow deep dives without thoracic squeeze.

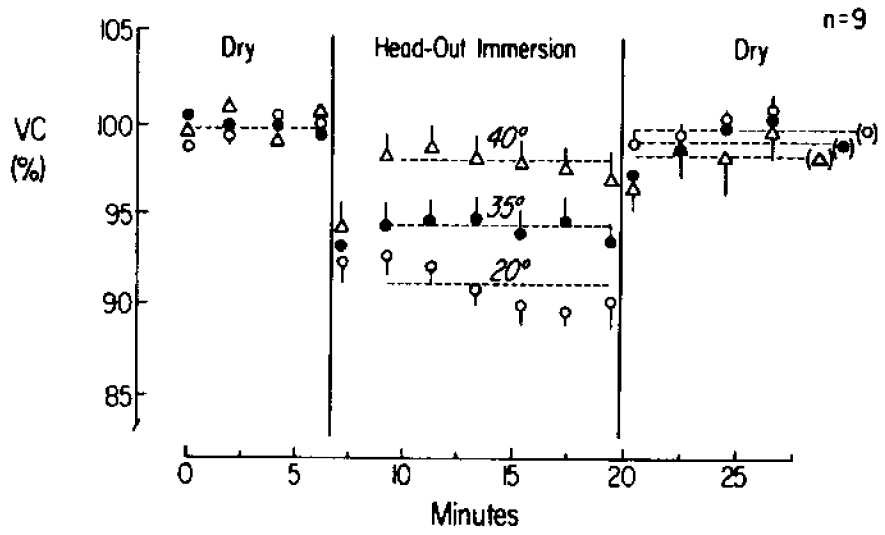

Fig. 1. Vital capacity (VC) in the sitting position during nonimmersion (dry) and head-out immersion in water of 20°, 35°, and 40°C. Results were normalized to means of preimmersion values and means ±SE are given for 9 subjects. Dashed lines indicate mean values for measurements obtained during related time span. Reproduced with permission from Kurss, D.I., C.E.G. Lundgren and A.J. Pasche. Effect of water temperature on vital capacity in head-out immersion. Pp. 297-301 in Bachrach, A.J. and M.M. Matzen, Editors. Underwater Physiology VII. Undersea Medical Society, Inc., Bethesda, Maryland, 1981.

What you see here is the result of some simple measurements of vital capacity under three different conditions, non-immersion, head-out immersion in water of different temperatures and finally back to the non-immersed situation. As you can see, there was not a significant difference in the warm water, 40 degrees, in vital

capacity compared to non-immersion, while in 35 degree water, thermoneutral, you have the classical 5 or 6 percent reduction in vital capacity which has always been ascribed to blood redistribution into the chest. In colder water, 20 degrees C, you have a further decrease in vital capacity averaging some 10 percent. The point here is twofold, one is that it is rather surprising that one can eliminate or reduce the hydrostatic effect of the external water column on your capacitance vessels by warming the water. The other point regards the potential consequence for the fall in intrathoracic pressure relative to ambient pressure during a deep breath hold dive. These observations suggest that you may have a greater intrathoracic pressure drop in warm water than in cold water.

Dr. Boysen: In patients with kidney stones, some are breathing spontaneously with an epidural anesthetic, and I haven't had them breath-hold, but there are some patients that who are under general anesthetic and sometimes it's helpful to give an inflation to fix the position of the diaphragm in order to set up the kidney, so you can focus the stone. This situation is the opposite of breath-hold diving in my view, based on what you just said, because you make the pleural pressure more positive and, although they are at the same time immersed, they are exquisitely sensitive to that technique and their arterial pressure just drops out.

Dr. Craig: In regard to your comment about the response of people with congestive failure and so forth to immersion, I remind you that a number of years ago John Knolls and others looked at the heart rate response to a Valsalva type maneuver in normals and in people with congestive failure. That was a classic paper for that period.

MODIFICATIONS IN THE VASOMOTOR ARM OF THE HUMAN DIVING RESPONSE

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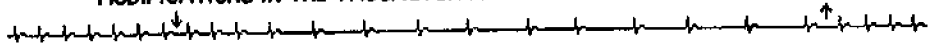


ABSTRACT

Mean arterial pressure is maintained during the human diving response because the vagal bradycardia is accompanied by an intense peripheral vasoconstriction. If the vasomotor component of the diving response of some diving animals is altered, blood pressure regulation is disrupted.

To test the hypothesis that blood pressure regulation takes priority over bradycardia in the human diving response, healthy subjects were exposed to conditions which altered their ability to vasoconstrict. Intense ambient heating gradually attenuated seated diving bradycardia. A change of posture from seated to supine recovered a diving bradycardia. Superimposed thiazide-induced hypovolemia and ambient heating resulted in a rapid elimination of diving bradycardia. The characteristic forearm blood flow decrease that is normally a part of the diving response was reduced by ambient heat exposure. At room temperature, the peripheral vasodilators hydralazine (an arteriolar smooth muscle relaxant) and particularly prazosin (an alpha$_1$-adrenergic antagonist) reduced seated diving bradycardia. These results emphasize the importance of adequate cardiac pre-load and after-load to the bradycardia of the human diving response.

INTRODUCTION

Trigeminal nerve thermoreceptors stimulated by facial immersion and breath hold comprise the primary afferent arms of the diving response. The two efferent arms of the diving response are vagal bradycardia and selective adrenergic peripheral vasoconstriction. As a result of the concommitant vasoconstriction, the diving response is an unique vagotonic maneuver since central mean arterial pressure is maintained, even during extremes in bradycardia[3]. Figure 1 is a diagram of the components of blood pressure. Blood pressure is a function of cardiac output and peripheral resistance. During apneic facial immersion, cardiac output falls in proportion to heart rate while total peripheral resistance rises by selective shifts in vasomotor tone in the parallel vascular beds. Syncope would follow a brief period of hypotension in the cerebral circulation.

If total peripheral resistance is fixed or reduced during apneic facial immersion, there could be at least two possible outcomes depending on the priority of cardiovascular control of the organism. Either blood pressure would fall during bradycardia resulting in light-headedness or syncope, or the preservation of blood pressure would be favored at the expence of diving bradycardia. The following non-invasive experiments were designed to test the hypothesis that blood pressure takes priority over diving

bradycardia in humans.

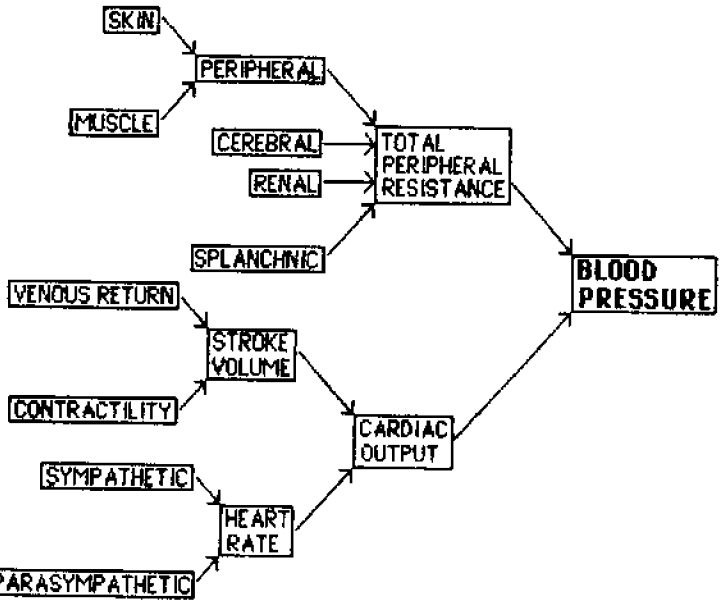


Figure 1. Components of blood pressure.

HEAT AND HYPOVOLEMIA[5]

Seven healthy subjects, five male and two female, aged 23-41, performed serial seated, 30-second breath hold, facial immersions in ice water on two separate days. Prior experience suggested that the degree of diving bradycardia produced by a series of normothermic facial immersions remains constant[7]. The Valsalva maneuver was avoided. The first day was a normovolemic control for the hypovolemic day, which was affected by three days administration of a thiazide diuretic. The degree of hypovolemia (10%) was determined by comparing pre- and post-diuretic hemoglobin concentrations.

On both days, each subject performed dives at room temperature before 38 or more minutes exposure to intense ambient heat in a 68°C chamber. The electrocardiograph was measured 30 seconds before and during each dive from which a pre-dive heart rate was averaged from -15 to -5 seconds and the dive heart rate was averaged from 15 to 30 seconds of dive. Forearm blood flow by venous occlusion plethysmography was recorded 15 seconds before and 15 seconds into each dive. Core temperature was continuously monitored by a thermister passed

naso-esophageally to the level of the right atrium.

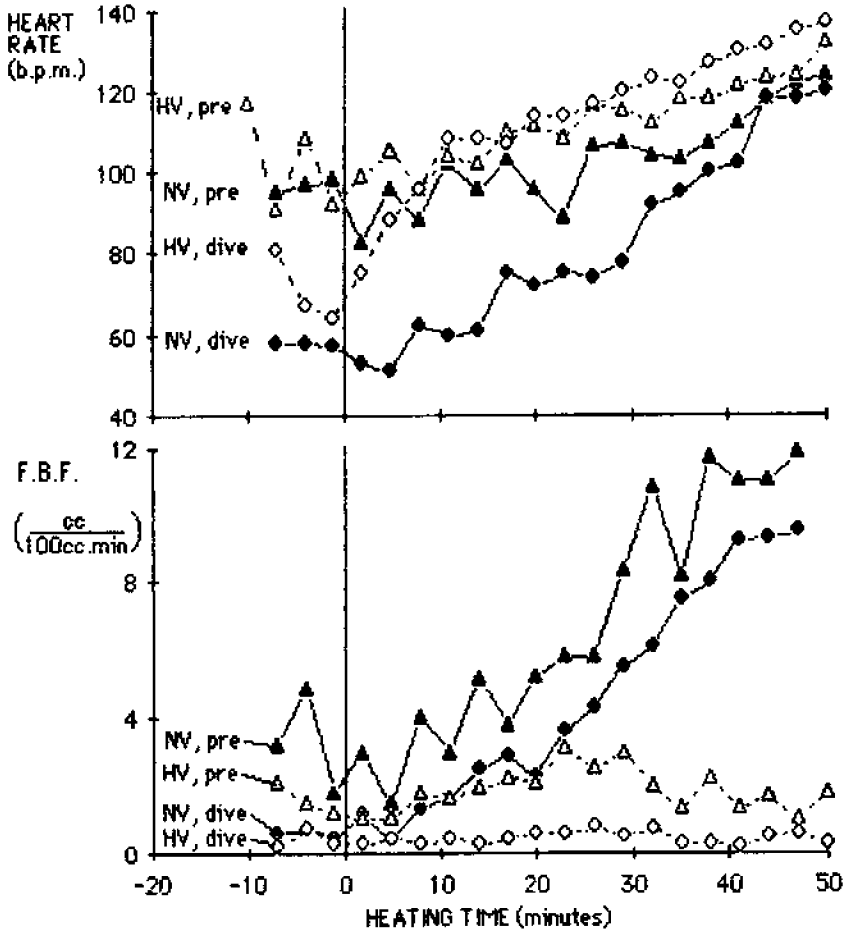


Figure 2. Heart rate and forearm blood flow (F.B.F.) during serial dives before and during exposure to intense ambient heat (68°C) in one subject under conditions of normovolemia (N.V.) and 10% hypovolemia (H.V.).

Normovolemic apneic facial immersion in ice water resulted in a 35% heart rate decrease (from 74±6 to 48±5 b.p.m.) and a 77% decrease in forearm blood flow (from 3.5±1.6 to 0.8±0.3 cc/100cc/min.). 35 minutes of heating produced a gradual increase in resting heart rate and a 4-fold increase in pre-dive forearm blood flow. Apneic facial immersion at 35 minutes heat time produced only an 8% heart rate decrease (98±4 to 90±3 b.p.m.) and a 27% decrease in forearm blood flow (13.5±1.8 to 9.9±1.8

cc/100cc/min.). Superimposed 10% hypovolemia and diving produced a pre-heat bradycardia (-35%) and forearm blood flow decrease (-70%) that were comparable to normovolemic values. However, dive heart rate decreased but 9% (87±6 to 79±6) after only 15 minutes of heating, and only 3% (104±4 to 101±3 b.p.m.) after 35 minutes in hypovolemic subjects. After 35 minutes hypovolemia heat exposure, diving produced a 22% forearm blood flow decrease (5.4±0.6 to 4.2±0.4 cc/100cc/min.). Esophageal temperature rose 0.7°C over 35 minutes independent of hydration state.

Figure 2 shows the heart rate and forearm blood flow responses to serial dives before and during intense ambient heating under normovolemic conditions and during 10% hypovolemia in one subject. Diving bradycardia was gradually attenuated over 40 minutes of normothermic heating. In contrast, diving bradycardia was eliminated after only 8 minutes of hypovolemic heating and further heating resulted in diving *tachycardia*

PERIPHERAL VASODILATORS[6]

On each of four days, five healthy subjects performed seated 30-second breath hold, facial immersions in water at three temperatures: 1°C, 10°C and 25°C. Heart rate was extracted from electrocardiographs monitored during and 15 seconds preceding each dive. Forearm blood flow was measured by venous occlusion plethysmography 15 seconds before and after the beginning of each immersion.

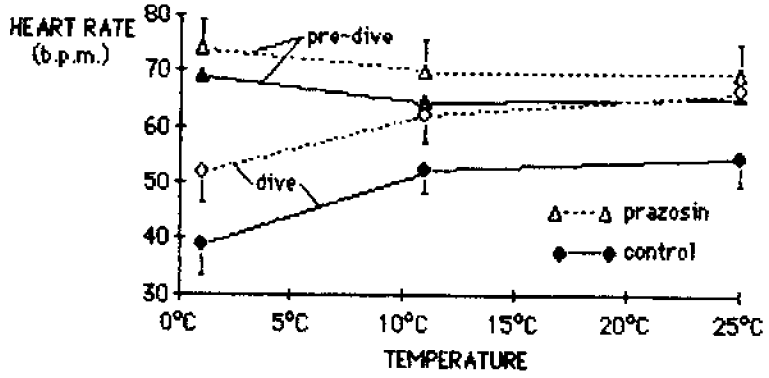
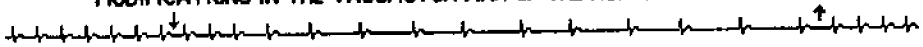


Figure 3. The effect of prazosin on temperature dependent diving bradycardia. Solid figures = control, open-dotted = prazosin, triangles = pre-dive, diamonds = dive, n = 5.

The first day was a control for the three subsequent days, each of



which characterized by administration of a low dose peripheral vasodilator: 50 mg hydralazine by mouth, 2 mg prazosin by mouth and then 15 mg nitroglycerine paste transdermally. Due to the characteristic individual variability of resting heart rates and bradycardia, data are anylyzed by percentage change from baseline using analysis of variance (ANOVA) and paired t-Test.

In the absence of drugs, apneic facial immersion produced a consistent, temperature dependent bradycardia (-34% @ 1°C, -12% @ 10°C, -8% @ 25°C) and forearm blood flow decrease (-62% @ 1°C, -40% @ 10°C, -21% @ 25°C). Nitropaste produced little change in dive heart rate. Hydralazine produced a reduction in diving bradycardia at 1°C (-34% to -28%, $p<.05$). Prazosin produced a reduction in temperature dependent bradycardia ($F_{1,4}=11$, $p<.05$) specifically at 10°C (-12% to -1%, $p<.05$) and 1°C (-34% to -17%, $p<.02$) shown in Figure 3. Doses of peripheral vasodilators was kept sufficiently low to limit changes in forearm blood flow.

HEAT AND POSTURE[4]

One healthy 23 year old male subject performed a series of 30-second breath hold facial immersions in a basin of water at 2-4°C. At least three minutes separated each dive. The Valsalva maneuver was avoided. Electrocardiograph was recorded for the 15-seconds preceding each dive and during the 30-second dive. Three dives were performed at an ambient temperature of 22°C followed by seven dives at 86°C. Most of the dives were performed in a seated posture, however the dive at 26 minutes heating was performed supine. At 32 minutes of heating, a 30-second breath hold without immersion was performed, then the experiment was terminated at the request of the subject.

Figure 4 shows ambient temperature (T_a), immersion water temperature (T_{H_2O}), rectal temperature (T_r), and 15-second pre-dive and dive mean and standard deviation heart rate before and during heat exposure. The 44% pre-heat dive heart rate decrease (from 77±5 to 43±2 b.p.m.) was slightly greater than the average for the previous groups. After only 22 minutes at this high ambient heat, rectal temperature had risen 0.7°C, and the seated diving bradycardia had been eliminated. At 26 minutes of heating, a change of posture to supine reduced pre-dive heart rate and resulted in an 8% diving bradycardia. At 32 minutes of heat, breath hold alone produced a 5% heart rate *increase*.

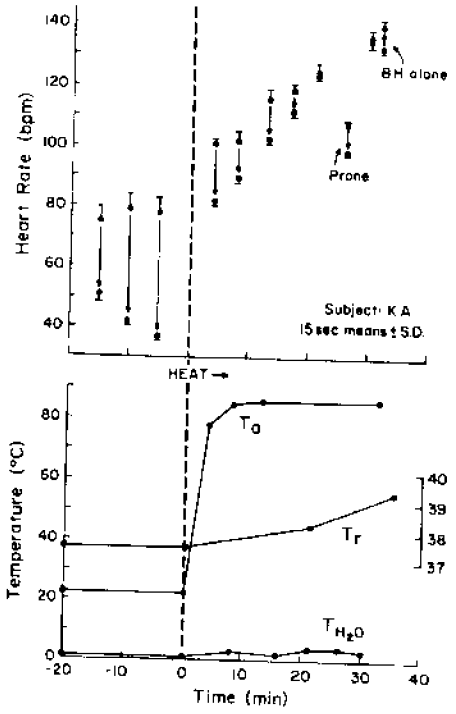
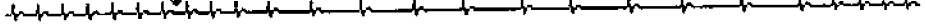


Figure 4. The effect of heat (86°C) and posture on serial diving heart rate. Ta = ambient temperature, Tr = rectal temperature, TH_2O = immersion water temperature, and B.H. = breath hold.

DISCUSSION

Exposure to intense ambient heat gradually attenuates bradycardia due to serial seated apneic facial immersion in ice water. A change in posture from seated to supine recovered bradycardia in a heated, diving subject. Superimposed thiazide-induced hypovolemia accelerated the attenuation due to heat. Normothermic subjects demonstrated a reduction in the temperature dependent diving bradycardia following administration of the low dose arteriolar smooth muscle relaxant, hydralazine and the alpha$_1$-adrenergic blocker, prazosin. No light-headedness nor syncope was encountered in these investigations.

The efferent components of the diving response have been altered in some animals. Guanethedine pre-treatment reduced bradycardia and



induced hypotension in diving ducks. Reserpine pre-treatment resulted in fatal hypotensive diving response in these birds[17]. Reserpine produced diving hypotension in rats, also[18]. When the bradycardia of young diving seals was overpowered by electrical cardiopacing, hypertension occurred and one seal died[20].

The effect on the diving response of such manipulations as exercise, change in posture or intra-pleural pressure, heat and mental stimulation has been studied in humans. One paper reports on the effect of alpha-adrenergic agents in the human diving response. Finley, et al, found relatively little effect of phentolamine on diving bradycardia during a brief, eight second immersion[15]. However, diving produced significant bradycardia from isoproterenol-induced tachycardia.

Diving bradycardia persists during exercise, in fact some of the lowest human dive heart rates have been recorded in exercising subjects[24]. It has been proposed that venous return during exercise permits diving bradycardia[25]. Diving bradycardia is greater in dynamic than isometric exercise presumably because of venous return[10].

Intense ambient heat exposure produces some cardiovascular changes similar to exercise: tachycardia and an increase in core temperature. Relatively few papers address the effect of heat on the diving response. Pulse decreased during shower cooling after sauna bathing but a measure of the effect of such cooling on normothermic subjects was not mentioned[19]. Bachmann, et al, using intra-arterial blood pressure telemetry found a normotensive bradycardia following a transient marked hypertension (300/240 mm. Hg) when a subject was totally immersed in a 15°C pool after sauna[8]. Caputa and Cabanac reported a moderate bradycardia in response to eupneic facial immersion in subjects who had been warmed in a 40°C bath, however they did not include normothermic comparison data[12].

The heart rate decrease during apneic immersion is greater with lower intra-pleural pressures[23]. Lower diving heart rates accompany head-down posture than head up[13]. Venous return is favored by both of these maneuvers.

One of the few factors which reduces human diving bradycardia is mental preoccupation such as performing mental arithmetic during facial immersion[22]. Mental arithmetic produces a dilatation of peripheral

vessels probably by a parasympathetic mechanism[9].

Peripheral vascular activity during the diving response has been approximated by venous occlusion plethysmography. Forearm blood flow, and moreso calf blood flow decrease consistently during facial immersion[11]. The forearm blood flow decrease of the human diving response is temperature dependent[2]. The reactive hyperemia following a period of tourniquet is also reduced by diving[14]. The instantaneous changes in vascular system during the diving response have best been measured by intra- arterial blood pressure catheter monitoring[8]. A gradually increasing systolic and diastolic pressure follow a transient dip during apneic facial immersion[16].

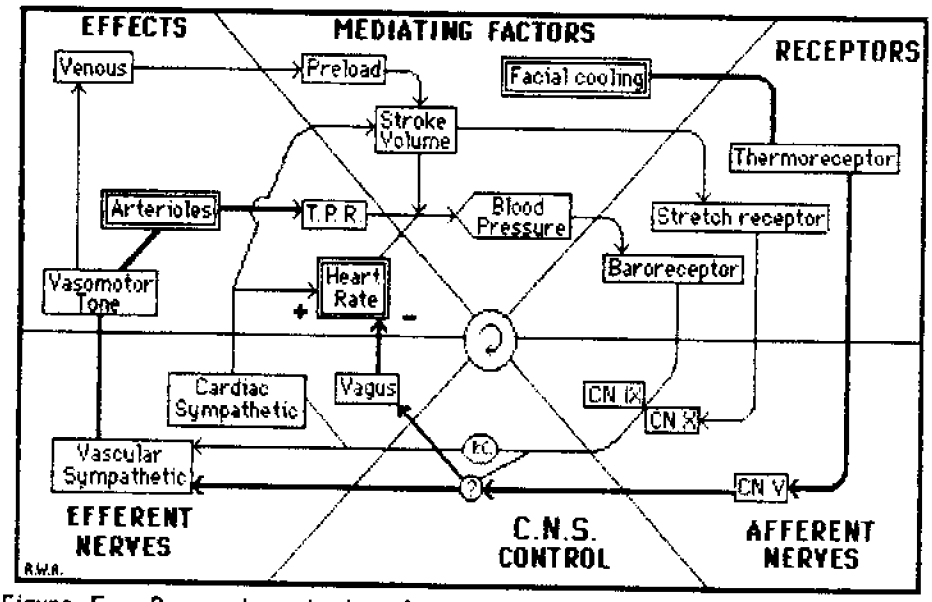
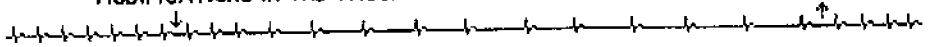


Figure 5. Proposed mechanism for onset and maintenance of the cardio-vascular components of the human diving response. T.P.R. = total peripheral resistance, P.C. = pressor center, CN V = trigeminal, CN IX = glossopharyngeal, and CN X = vagus nerves.

Peripheral veins and arterioles are innervated by adrenergic and some parasympathetic nerves[1]. The control of peripheral vasoconstriction is mediated by the brain stem "pressor center" with feedback input from arterial baroreceptors. Vascular stretch receptors detecting venous return and baroreceptors affect heart rate to regulate blood pressure. Figure 5 gives a proposed mechanism for the control physiology of the



onset and maintenance of the human diving response. The primary afferent is facial cooling mediated by trigeminal thermoreceptors and enhanced by breath hold. The two efferent arms are vagal bradycardia and sympathetic peripheral vasoconstriction. The brain stem control center(s) for the diving response has not been clearly defined. Only instantaneous monitoring of the vascular system can delineate whether bradycardia precedes vasoconstriction or vice versa during the first few seconds after immersion. Vascular stretch receptors and baroreceptors continually monitor cardiac pre-load and after-load respectively, and provide additional afferent input to the maintenance of the diving response. Chemoreceptors, pulmonary stretch receptors, higher cortical centers, and others evidently also provide input to the control of the diving response.

Venous return and peripheral vasoconstriction have a major influence on the degree and duration of the diving response. Adequate cardiac pre-load and after-load are prerequisites to human diving bradycardia. The control mechanism for the diving response in some animals seems to favor bradycardia and vasoconstriction in response to immersion even if blood pressure regulation goes awry. In contrast, it appears that blood pressure maintenance and avoidance of syncope takes priority over bradycardia in the human diving response.

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REFERENCES

1 Abboud,FM., Eckstein,JW. Reflex vasoconstrictor and vasodilator responses in man. Circulation Res. 29: suppl I96-103, 1966.

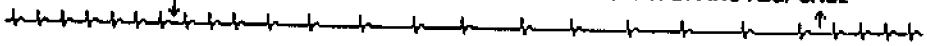
2 Arnold,RW. A vasomotor comparison of the human diving response and the cold pressor test. Alaska Medicine 28: accepted, 1986.

3 Arnold,RW. Extremes in human breath hold, facial immersion bradycardia. Undersea Biomed. Res. 12: 183-190, 1985.

4 Arnold,RW. Modifications in the vasomotor component of the human diving response. M.D. Thesis, Yale, 1984.

5 Arnold,RW., Nadel,EN. The attenuating effect of heat and hypovolemia on the human diving response. Submitted.

6 Arnold,RW., Lister,G., Nadel,EN. The effect of peripheral vasodilators on the human diving response. Submitted.



7 Arnold,RW. The human diving bradycardia and its recovery time. B.S. Thesis, University of Alaska-Fairbanks, 1979.

8 Bachmann,K., Hoffman,H., Gunther,W., Zerzawy,R. Ergebnisse telemitrischer Kreislaufuntersuchen beim Saunabadvorgang. Sauna Archiv. 9: 1-7, 1971.

9 Bancroft,H., Brod,J., Hejl,Z., Hirsjavi,EA., Kitchin,AH. Mechanism of vasodilatation in forearm muscle during stress (mental arithmetic). Clin. Sci. 19: 577-586, 1960.

10 Bergman,SA., Campbell,JK., Wildenthal,K. "Diving reflex" in man; its relation to isometric and dynamic exercise. J. Appl. Physiol. 33: 27-31, 1972.

11 Campbell,LB., Gooden,BA., Lehman,RG., Pym,J. Simultaneous calf and forearm blood flow during immersion in man. Aust. J. Exp. Biol. Med. Sci. 47: 747-754, 1969.

12 Caputa,M., Cabanac,M. Bradycardia during face cooling in man may be produced by selective brain cooling. J. Appl. Physiol. 46: 905-907, 1979.

13 Craig,AB. Heart rate responses to apneic underwater diving and to breath holding in man. J. Appl. Physiol. 18: 854-862, 1963.

14 Elsner,R., Gooden,BA. Reduction of reactive hyperemia in the human forearm by face immersion. J. Appl. Physiol. 29: 627-630, 1970.

15 Finley,JP., Bonnet,JF., Waxman,MB. Autonomic pathways responsible for bradycardia on facial immersion. J. Appl. Physiol. 47: 1218-1222, 1979.

16 Kawakami,Y., Natelson,B., DuBois,AB. Cardiovascular effects of face immersion and factors affecting diving reflex in man. J. Appl. Physiol. 23: 964-970, 1967.

17 Kobinger,W., Oda,M. Effects of sympathetic blocking substances on the diving reflex in ducks. Eur. J. Pharmac. 7: 289-295, 1969.

18 Lin,YC. Autonomic nervous control of cardiovascular response during diving in the rat. Am. J. Physiol. 227: 601-605, 1974.

19 Matousek,J., Pribil,M. Reaction of pulse frequency to various methods of cooling in sauna. Fysiat. Vestn. 56: 208-212, 1978.

20 Murdaugh,HV., Cross,CE., Millen,JE., Gee,JBL., Robin,ED. Dissociation of bradycardia and arterial constriction during diving in the seal, *Phoca vitulina*. Science 162: 362-363, 1968.

21 Olsen,CR., Fanestil,DD., Scholander,PF. Some effects of apneic underwater diving on blood gases, lactate and pressure in man. J. Appl. Physiol. 17: 938-942, 1962.

22 Ross,A., Steptoe,A. Attenuation of the diving reflex in man by mental stimulation. J. Physiol. (London) 302: 387-393, 1980.

23 Song,DH., Lee,WK., Chung,YA., Hong,SK. Mechanism of apneic bradycardia in man. J. Appl. Physiol. 27: 323-327, 1969.

24 Strømme,SB., Karem,D., Elsner,R. Diving bradycardia during rest and exercise and its relation to physical fitness. J. Appl. Physiol. 28: 614-621, 1970.

25 Williams,CI., Bernauer,EM. Diving reflex during exercise in man. Med. Sci. Sports 8: 63-64, 1976.

Discussion following Dr. Arnold's presentation

Dr. Van Liew: I'm very interested in your talk. It seems to me the role of science is to predict and control, and you've described a story that is so complex that I don't think you're really able to predict or control. Experiments done under the circumstances of your study can fit into a scheme all right, but it's hard to use the information for predictions about experiments done under different circumstances.

Dr. Arnold: I agree. I have seen some healthy people with extreme bradycardia while seated. I've used diving response to treat paroxysmal supraventricular tachycardia in older people too. Since the blood pressure is maintained during this vagotonic response, it may be a unique vagotonic maneuver and is very useful and probably quite safe in inducing bradycardia because of that. Carotid sinus massage, Valsalva and other maneuvers are often associated with syncope, because blood pressure is not maintained. In the animals for whom diving response is probably more important for their livelihood than it is for humans, it appears that their diving response takes priority over blood pressure. If they're cardiac paced, or if their vasomotor response is inhibited, they favor the diving response, i.e. bradycardia or vasoconstriction, continues at the expense of blood pressure. I suspect that the human does better maintaining blood pressure at the expense of bradycardia or vasoconstriction in diving response.

Dr. Hong: In humans, the increase in blood pressure is quite substantial and that presents some problem in terms of oxygen conservation. One question that I was interested in is to explore the role of stretch-receptors.

Dr. Arnold: There's A and B and they're frequently found in the atrial walls. It's not clear exactly their role but it's shown in animals that it's off and on, but mainly the B type receptors can induce bradycardia when they're activated. They also regulate urine flow quite a bit, but I haven't been as interested in that.

Dr. Hong: Because in human subjects during head-out immersion a cephalad blood shift takes place and the cardiac stretch receptors are presumably stimulated, heart rate decreases even without breath-holding. That has been our finding.

Dr. Elsner: (You can impose) a thermal load on a diving seal by heating or cooling the hypothalamus, in a technique for looking at the possible conflict between vasodilation of heating and vasoconstriction of diving, and when you do this you find that the seal generally favors the vasoconstriction of diving.

The cardiac response to breath-hold diving at rest and while exercising. (A comparison between man and diving homeotherms)

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The "classical" diving response

A reduction in heart rate below the resting level (bradycardia) occurs in man and in all air breathing vertebrates that habitually dive under water, when they are experimentally submerged in a resting state (Lin 1982; Butler & Jones 1982). Complete submersion is not necessary; merely placing the head under water is all that is required to elicit this response. The significance of the bradycardia in aquatic air breathing vertebrates was elucidated by Irving (1939) and Scholander (1940). Parts of the body that can withstand oxygen lack, e.g. gut, muscles, are effectively eliminated from the circulation by intense vasoconstriction while the oxygen-dependent tissues such as the heart and brain continue to be perfused. An increase in arterial blood pressure, as a result of the selective vasoconstriction, is avoided by a reduction in cardiac output, brought about predominantly by the so-called "diving bradycardia" (Fig 1a). Thus oxygen within the body is conserved for the heart and CNS while the rest of the animal metabolises anaerobically and accumulates lactic acid. The total, oxygen conserving response to involuntary submersion in aquatic homeotherms has been termed the "classical" diving response (Butler 1982). Bradycardia is the typical element of the cardiovascular, and hence of the metabolic, adjustments to involuntary diving and is often taken as an indicator of the remainder of the response taking place (Pasche & Krog 1980). It is worth investigating the validity of this assumption in a little more detail.

Heart rate and oxygen conservation during breath-hold submersion at rest

There is ample evidence that the rate of oxygen consumption does in fact decrease during such experimental submersion in air breathing aquatic vertebrates and that anaerobic metabolism increases (Scholander 1940; Andersen 1966; Butler & Jones 1982; Elsner & Gooden 1983). It is also apparent that temperature of the water has no effect on the response in diving birds (Andersen 1963; Butler & Jones 1968) or mammals (Dykes 1974). Although both bradycardia and vasoconstriction in the peripheral circulation occur during apnoeic face immersion in man (Wyss 1956; Heistad *et al* 1968; Campbell *et al* 1969) their intensities appear to be greater at lower water temperatures (Kawakami *et al* 1967; Burch & Giles 1970). The 'critical' water temperature in this respect is around 35°C and if the water is colder than this there is a temperature-related bradycardia during non-apnoeic face immersion, whereas in warmer water little change in heart rate occurs (Kobayasi & Ogawa 1973). Despite the existence of these cardiovascular adjustments during submersion, which in aquatic birds and mammals are concerned with conservation of oxygen, there is some doubt as to whether they are sufficiently intense in man to cause a reduction in the

rate of oxygen consumption.

Wolf et al (1965) demonstrated that all the signs of reduced aerobic metabolism and increased anaerobiosis are present in man during head submersion when a clear bradycardia (from 85 to 50 beats per minute) is present. However, in the absence of bradycardia during a similar period (40 s) of breath-holding under water, oxygen saturation of the blood declines more markedly and there is little post-dive accumulation of lactate in the blood. Similarly, Elsner et al (1971) found that there is a good inverse relationship between the decline in partial pressure of oxygen in arterial blood and the fall in forearm blood flow during face immersion after hyperventilation of oxygen. On the other hand, Hong et al (1971) found that the rate of removal of oxygen from the lungs and blood during a 4 min breath-hold in air is slightly greater than pre-apnoeic resting oxygen consumption. However, it must be noted that at 2 min into the breath hold at least, there were no significant reductions in heart rate and cardiac output nor an increase in peripheral resistance (see Table 6 in Hong et al 1971). Even when a bradycardia was reported to have occurred, the lowest mean heart rate was approximately 75 beats per minute (see Fig 4 in Hong et al 1971). To add to the apparent confusion it has recently been demonstrated that breath-hold duration in man is no longer when submersed in water at 32°C than it is in air despite the fact that heart rate is significantly lower during the former (Sterba & Lundgren 1985). This result would tend to support the contention that, unlike the situation in aquatic homeotherms, the period of breath-hold diving in man does not exceed that which would be expected on the basis of usable oxygen stores and resting oxygen consumption (Lin 1982). The situation is complicated, however, by the fact that afferent information from the respiratory muscles themselves may stimulate respiratory activity during apnoea before critical levels of oxygen and/or carbon dioxide are reached (Fowler 1954;Campbell et al 1967).

Heart rate during breath-hold diving while exercising

As long ago as 1960 Eliassen, on the basis of his own observations of natural dive durations in aquatic birds and of his calculations of the amount of usable oxygen that they could store in their body, argued that it is not necessary for these habitual divers to undergo the intense cardiovascular adjustments of the "classical" diving response during natural dives. They should be able to metabolise completely aerobically while swimming under water. This paper and its provocative suggestion were largely ignored for almost 20 years, but when it was possible to record physiological data from freely diving birds and mammals it turned out that reduction of heart rate below the resting level and anaerobiosis are not components of routine dives in these animals.

By using implanted radiotransmitters it has been demonstrated for several species of birds that, unlike the situation during submersion of the head of a resting animal, there is no maintained reduction of heart rate below the resting level during spontaneous dives (Butler & Woakes 1979; 1984). For example, in the tufted duck, Aythya fuligula, there is a large reduction in heart rate to approximately 30% of the pre-dive value within 30 s of involuntary submersion of the head (Fig 1a) and this is

largely the result of the stimulation of trigeminal receptors in the beak and nares (Butler & Woakes 1982a; Furilla & Jones 1986). During voluntary submersion to a depth of 1.9 m however, heart rate is 60% above the resting value after 15 s and this rate is maintained until shortly before surfacing when an anticipatory increase may occur (Fig 1b). In addition there is no

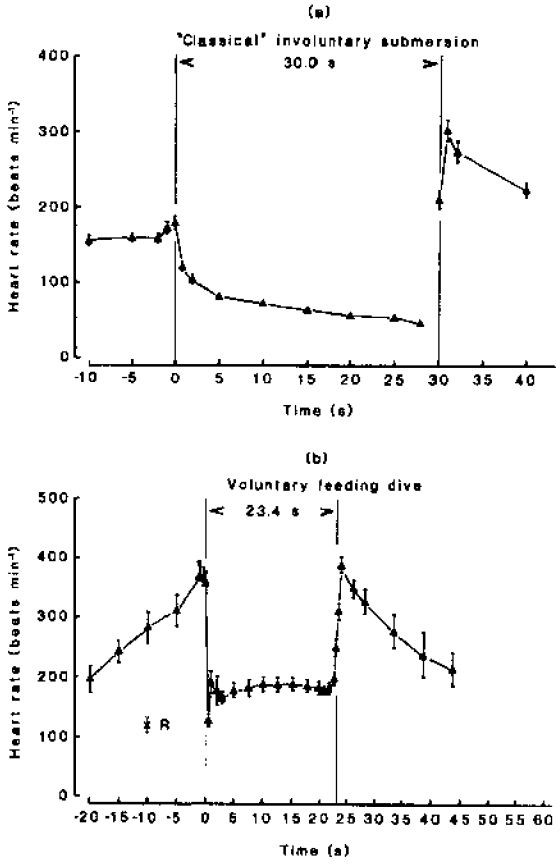

Fig 1 Mean changes in heart rate (± S.E.) in tufted ducks, Aythya fuligula, during (a) involuntary submersion and (b) during voluntary feeding dives to a depth of 1.9 m. R = resting heart rate. Data in (a) are taken from Butler & Woakes (1982b) and are mean values of 38 observations from 10 animals; those in (b) are from Stephenson (1985) and are mean values of 79 observations from 6 animals.

reduction in aerobic metabolism during voluntary dives in either tufted ducks or Humboldt penguins, Spheniscus humboldti (Woakes & Butler 1983; Butler & Woakes 1984). In the ducks, estimated oxygen uptake at mean dive duration is approximately 3.5 times the resting value and in the penguins it is not significantly different from resting. Tufted ducks are positively buoyant and have to work hard to remain submerged (surfacing occurs

passively when the birds cease to paddle their feet). In fact oxygen consumption during diving is not significantly different from that at maximum sustainable (surface) swimming speed (Woakes & Butler 1983). Penguins, on the other hand, are almost neutrally buoyant and more efficient at under water swimming than tufted ducks.

Heart rate in the tufted duck during natural, voluntary, diving is 73% of that during surface swimming when oxygen consumption is the same in both cases (Fig 2). In other words, at the same level of aerobic metabolism there is a slight bradycardia during exercise under water compared with exercise in air. This is a statement worth considering in further detail

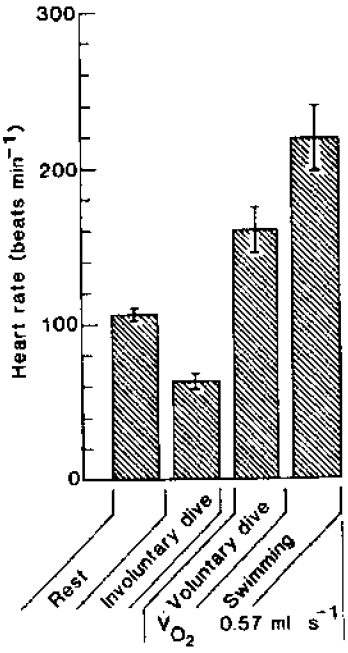

Fig 2 Mean heart rate (± S.E.) for 6 tufted ducks, Aythya fuligula, (except during involuntary dives when 10 ducks were used) at rest on water, 15 s after involuntary submersion of the head, 14 s after voluntary submersion and while swimming. Oxygen consumption ($\dot{V}_{O_2}$) at mean dive duration and while swimming was the same, 0.57 ml STPD per second (Woakes & Butler 1983).

because the use of the word 'bradycardia' in this context is somewhat at variance with its more common usage when it refers to a heart rate below normal resting (i.e. below approximately 60 beats per minute in humans or approximately 110 beats per minute in tufted ducks). It has been suggested that the use of resting heart rate as the baseline for defining bradycardia is inadequate (Stephenson 1985) and this would certainly seem to be a reasonable suggestion as far as breath-hold diving is concerned.

The fact that heart rate in tufted ducks is lower during natural diving than during exercise in air at the same level of aerobic metabolism and yet is substantially above resting (Fig 2) has led Butler (1982) to suggest that the cardiovascular response to natural diving in these birds is a balance between the "classical" diving response and the response to exercise, with the bias towards the latter. This implies that exercise modifies the "classical" diving response (or vice versa) in tufted ducks at least to the extent that the active locomotory muscles, as well as the CNS and heart, receive sufficient blood (and hence oxygen) to metabolise aerobically. The oxygen used from body stores is quickly replaced upon surfacing. Other parts of the body, such as the respiratory muscles and the large flight muscles, may well reduce the rate at which they consume oxygen, as postulated by the "classical" hypothesis, but whether or not they metabolise anaerobically and produce lactic acid remains to be seen.

Data from seals indicate that they also metabolise aerobically, using stored oxygen, during the vast majority of their natural dives. In grey seals (Halichoerus grypus) heart rate is approximately 30 beats per minute during submersion and 110 beats per minute when breathing at the surface, irrespective of whether the animal is resting or swimming. As swimming speed increases, the time spent submerged decreases so that overall mean heart rate increases with oxygen uptake in the typical fashion. There is no increase in blood lactate during such swimming activity. However, if the animal is alarmed in any way then heart rate falls to very low rates of <10 beats per minute whether it is resting or swimming. This is typical of the classical oxygen conserving response (Fedak et al 1984). The majority of dives performed by Weddell seals, Leptonychotes weddelli, in nature are less than 25 min in duration and completely aerobic. Only after those few dives which are longer than 25 min is there an increase in lactic acid in the blood, the level of which is related to the duration of the dive (Kooyman et al 1980).

Man, of course, is not a habitual diver so the difficulty in demonstrating some functional significance of the cardiovascular responses that occur during facial submersion in resting subjects is perhaps not surprising. Breath-hold diving for the "fruits of the sea" is still practised by a (diminishing) number of humans and many of these perform unassisted dives. That is, the diver carries no weights and (like the tufted duck) has to overcome his/her own drag and buoyancy during descent. Unlike the tufted duck, ascent is only partly assisted by the diver's buoyancy. Thus, during these dives the individual is exercising and in view of the data from naturally diving birds and mammals it was decided to look more closely at the cardiac responses during active submersion in humans.

The change in heart rate in man during apnoeic exercise in water has been reported on a number of occasions but there is no consistency in the techniques that were used and there are quantitative as well as qualitative differences in the results that were obtained. A number of authors have found that heart rate falls gradually to below the resting value during submersion in man while exercising. The subjects in the study by Scholander

et al (1962) began from rest in water and there was an initial increase in heart rate followed by the bradycardia. In other studies the subjects were already exercising, either in water (Irving 1963; Stromme et al 1970) or on a cycle ergometer in air (Asmussen & Kristiansson 1968; Bergman et al 1972; Oldridge et al 1978; Bjertnaes et al 1984; Smeland et al 1984), before submerging their head and becoming apnoeic. Heart rate was, therefore, already elevated above the resting level before the "dive", but it often fell to the same level as that recorded during "diving" under resting conditions. On the other hand, Craig (1963) found that when a man resting quietly on the bottom of a pool began to exercise, his heart rate increased above its resting value and declined at the end of the exercise. The tachycardia of exercise, although attenuated, was still present in subjects exercising on a cycle ergometer completely submerged in water (Craig & Medd 1968). In Korean ama, heart rate, 30 s after the start of breath-hold diving or of swimming just below the surface in summer, was slightly higher or similar to resting heart rate (Hong et al 1967).

In the present study heart rate was monitored from 6 male subjects of mean age (± S.E.) of 24 ± 1 years and mean mass 73.5 ± 2.4 kg. A single channel PIM radiotransmitter (Butler & Woakes 1982b) was tucked into the back of the subjects' trunks and leads attached via waterproof connectors to two electrodes; one electrode was attached over the sternum and the other approximately 20 cm lower on the left-hand side of the chest. Measurements were made in a 25 m swimming pool at a water temperature of 28°C. Resting heart rate was obtained from the subjects wrapped in a gown and sitting quietly on a seat and was 67 ± 3.7 beats per minute. When the subjects (with a nose clip) submerged themselves completely in the pool, but remained inactive, there was a prompt but gradual reduction in heart rate which reached 72% of the resting value within 30 s and 60% of the resting rate at mean dive duration of 59 ± 5.6 s (Fig 3). Heart rate was significantly below the resting rate within 15 s of submersion. The subjects then dried, wrapped themselves in a gown and sat on the seat until they had fully recovered. This procedure was repeated between each test. When they propelled themselves under water for 33 s by kicking their legs while breathing through a snorkel tube, there was a progressive increase in heart rate which was 76% above the resting rate at 28 s after commencing the exercise (Fig 3). However, when they performed the same manoeuvre without the snorkel tube (i.e. while holding their breath) there was an initial increase in heart rate to 58% above the resting rate within 10 s. This was followed by a decline in heart rate to 72% of the resting value at mean dive duration of 33 ± 1.8 s (Fig 3). The fact that heart rate reached the same level during active dives as it did during inactive dives means that it declined at a higher rate during the former. Deceleration rates between 10 s after submersion and at either 30 s after submersion or at surfacing were -2.7 beats per minute per second during the active dives and -0.7 beats per minute per second during the inactive dives.

The time under water and the distances travelled in the pool were similar during both types of active dives, so the work performed must have been similar in both cases. Although heart rate was not significantly below the resting level until just before surfacing during the active dives with breath-hold, it was significantly below the corresponding value during

active dives while breathing after 20 s of submersion. So, on the basis of the definition of "bradycardia" given earlier, there is a clear bradycardia after 20 s of active breath-hold diving, and just before surfacing heart rate in the present subjects was 40% of the value it would have been had they been breathing and performing the same level of exercise.

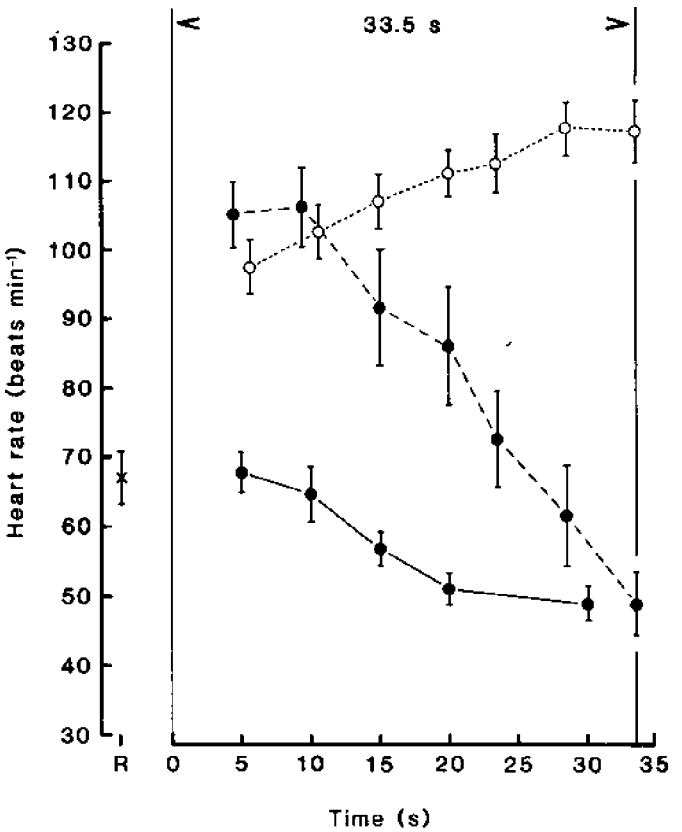

Fig 3 Mean heart rate (± S.E.) from 6 human males during complete submersion in water at 28°C while at rest (●——●), while swimming (by kicking the legs) just below the surface and breathing through a snorkel tube (O- - - -O) and while swimming just below the surface and holding the breath (●— —●). R is the resting heart rate.

There is no doubt, therefore, that exercise modifies the cardiac chronotropic response to breath-hold diving in man. During the first 15 s heart rate is typical of that during normal exercise in water (cf. Scholander et al 1962; Jung & Stolle 1981), but once heart rate begins to fall its rate of decline is greater than that during inactive dives (cf. Bergman et al 1972; Smeland et al 1984). In view of the progressively large difference in heart rate between active dives when breath-holding and active dives while breathing, it is difficult to avoid coming to the

conclusion that during the former there is a progressive involvement of an oxygen conserving response. Certainly some of the subjects in the present study noted that discomfort (pain) in the legs was greater when swimming underwater with breath-hold than when breathing. It seems, therefore, that man may be different from naturally diving homeotherms in that he enlists the oxygen conserving response to a greater extent during active breath-hold dives than during inactive dives. It is worth noting, however, that seals and ducks can habituate to submersion of the head in that the fall in heart rate is reduced or abolished if the animal is taught to "dive" or if it is submerged repeatedly for the same period of time (Elsner 1965; Gabbott & Jones 1986). Also, during unusually long voluntary dives both seals and ducks do exhibit a lower heart rate than during dives of more normal duration (Kooyman & Campbell 1972; Butler et al 1986). Thus, the awareness of the subject (man or animal) of what is happening during passive head submersion and the size of usable oxygen stores relative to the amount of oxygen being used during active breath-hold dives, together with the effect of temperature, may be the major factors involved in the apparent differences in the response to diving (inactive or active) between man and habitually diving homeotherms.

References

Andersen, H. T. 1963. Factors determining the circulatory adjustments to diving. I. Water immersion. Acta physiol. Scand. 58, 173-185.

Andersen, H. T. 1966. Physiological adaptations in diving vertebrates. Physiol. Rev. 46, 212-243.

Asmussen, E. and Kristiansson, N.-G. 1968. The "diving bradycardia" in exercising man. Acta physiol. Scand. 73 527-535.

Bergman, S. A., Campbell, J. K. and Wildenthal, K. 1972. "Diving reflex" in man: its relation to isometric and dynamic exercise. J. appl. Physiol. 33, 27-31.

Bjertnaes, L., Hauge, A., Kjekshus, J. and Soyland, E. 1984. Cardiovascular responses to face immersion and apnea during steady state muscle exercise. Acta physiol. Scand. 120, 605-612.

Burch, G. E. and Giles, T. D. 1970. A digital rheoplethysmographic study of the vasomotor response to 'simulated diving' in man. Cardiology 55, 257-271.

Butler, P. J 1982. Respiratory and cardiovascular control during diving in birds and mammals. J. exp. Biol. 100, 195-221.

Butler, P. J. and Jones, D. R. 1968. Onset of and recovery from diving bradycardia in ducks. J. Physiol. 196, 255-272.

Butler, P. J. and Jones, D. R. 1982. The comparative physiology of diving in vertebrates. In: Advances in Comparative Physiology and Biochemistry, Vol. 8 (ed. O. E. Lowenstein), pp. 179-364. New York, Academic Press.

Butler, P. J. and Woakes, A. J. 1979. Changes in heart rate and respiratory frequency during natural behaviour of ducks, with particular reference to diving. J. exp. Biol. 79, 283-300.

Butler, P. J. and Woakes, A. J. 1982. Telemetry of physiological variables from diving and flying birds. Symp. zool. Soc. Lond. 49, 107-128.

Butler, P. J. and Woakes, A. J. 1982. Control of heart rate by carotid body chemoreceptors during diving in tufted ducks. J. appl. Physiol.: Respirat. Environ. Exercise Physiol. 53, 1405-1410.

Butler, P. J. and Woakes, A. J. 1984. Heart rate and aerobic metabolism in Humboldt penguins, Spheniscus humboldti, during voluntary dives. J. exp. Biol. 108, 419-428.

Butler, P. J., Stephenson, R. and Woakes, A. J. 1986. Variability of the heart rate response during voluntary diving in the tufted duck, Aythya fuligula. J. Physiol. (in press).

Campbell, E. J. M., Freedman, S., Clark, T. J. H., Robson, J. G. and Norman, J. 1967. The effect of muscular paralysis induced by tubocurarine on the duration and sensation of breath-holding. Clin. Sci. 32, 425-432.

Campbell, L. B., Gooden, B. A. and Horowitz, J. D. 1969. Cardiovascular responses to partial and total immersion in man. J. Physiol. 202, 239-250.

Craig, A. B. Jr. 1963. Heart rate responses to apneic underwater diving and to breath holding in man. J. appl. Physiol. 18, 854-862.

Craig, A. B. Jr. and Medd, W. L. 1968. Man's responses to breath-hold exercise in air and in water. J. appl. Physiol. 24, 773-777.

Dykes, R. W. 1974. Factors related to the dive reflex in harbor seals: sensory contributions from the trigeminal region. Can. J. Physiol. Pharmacol. 52, 259-265.

Elsner, R. (1965). Heart rate response in forced versus trained experimental dives in pinnipeds. Hvalradets Skrifter, Norske Videnskaps-Akad., Oslo 48, 24-29.

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

Elsner, R., Gooden, B. A. and Robinson, S. M. 1971. Arterial blood gas changes and the diving response in man. Aust. J. exp. Biol. med. Sci. 49, 435-444.

Eliassen, E. 1960. Cardiovascular responses to submersion asphyxia in avian divers. Arbok Univ. Bergen Mat. Naturv. Ser. 2, 1-100.

Fedak, M. A., Kanwisher, J. and Lapennas, G. 1984. Patterns of breathing and heart rate in relation to metabolic rate of swimming and diving seals. Proc. 1st Congress Comp. Physiol. and Biochem. Section of IUBS, p.A86.

Fowler, W. S. 1954. Breaking point of breath-holding. J. appl. Physiol. 6, 539-545.

Furilla, R. A. and Jones, D. R. 1986. On the initiation of submersion bradycardia in diving compared with dabbling ducks. (In preparation).

Gabbott, G. R. J. and Jones, D. R. 1986. Psychogenic influences on the cardiac response of the duck (Anas platyrhynchos) to forced submersion. J. Physiol. (in press).

Heistad, D. D., Abboud, F. M. and Eckstein, J. W. 1968. Vasoconstrictor response to simulated diving in man. J. appl. Physiol. 25, 542-549.

Hong, S. K., Song, S. H., Kim, P. K. and Suh, C. S. 1967. Seasonal observations on the cardiac rhythm during diving in the Korean ama. J. appl. Physiol. 23, 18-22.

Hong, S. K., Lin, Y. C., Lally, D. A., Yim, B. J. B., Kominami, N., Hong, P. W. and Moore, T. O. 1971. Alveolar gas exchanges and cardiovascular functions during breath holding with air. J. appl. Physiol. 30, 540-547.

Irving, L. 1939. Respiration in diving mammals. Phys. Rev. 19, 112-134.

Irving, L. 1963. Bradycardia in human divers. J. appl. Physiol. 18, 489-491.

Jung, K. and Stolle, W. 1981. Behaviour of heart rate and incidence of arrhythmia in swimming and diving. Biotelemetry Patient Monitg. 8, 228-239.

Kawakami, Y., Natelson, B. H. and DuBois, A. B. 1967. Cardiovascular effects of face immersion and factors affecting diving reflex in man. J. appl. Physiol. 23, 964-970.

Kobayasi, S. and Ogawa, T. 1973. Effect of water temperature on bradycardia during nonapneic facial immersion in man. Jap. J. Physiol. 23, 613-624.

Kooyman, G. L. and Campbell, W. B. 1972. Heart rates in freely diving Weddell seals, Leptonychotes weddelli. Comp. Biochem. Physiol. 43, 31-36.

Kooyman, G. L., Wahrenbrock, E. A., Castellini, M. A., Davis, R. W. and Sinnett, E. E. 1980. Aerobic and anaerobic metabolism during voluntary diving in Weddell seals: evidence of preferred pathways from blood chemistry and behavior. J. comp. Physiol. 138, 335-346.

Lin, Y-C. 1982. Breath-hold diving in terrestrial mammals. In: Exercise and Sport Sciences Reviews, Vol. 10. (ed. R. L. Terjung), pp. 270-307, Philadelphia: Franklin Press.

Oldridge, N. B., Heigenhauser, G. J. F., Sutton, J. R. and Jones, N. L. 1978. Resting and exercise heart rate with apnea and facial immersion in female swimmers. J. appl. Physiol.: Respirat. Environ. Exercise Physiol. 45, 875-879.

Pasche, A. and Krog, J. 1980. Heart rate in resting seals on land and in water. Comp. Biochem. Physiol. 67, 77-83.

Scholander, P. F. 1940. Experimental investigations on the respiratory function in diving mammals and birds. Hvalradets Skrifter, Norske Videnskaps-Akad., Oslo 22, 1-131.

Scholander, P. F., Hammel, H. T., LeMessurier, H., Hemmingsen, E. and Garey, W. 1962. Circulatory adjustment in pearl divers. J. appl. Physiol. 17, 184-190.

Smeland, E. B., Owe, J. O. and Andersen, H. T. 1984. Modification of the 'diving bradycardia' by hypoxia or exercise. Resp. Physiol. 56, 245-251.

Stephenson, R. 1985. The Physiology of Natural Diving Behaviour in the Tufted Duck (Aythya fuligula). M.Sc. (qual.) thesis, University of Birmingham, England.

Sterba, J. A. and Lundgren, C. E. G. 1985. Diving bradycardia and breath-holding time in man. Undersea Biomed. Res. 12, 139-150.

Stromme, S. B., Kerem, D. and Elsner, R. 1970. Diving bradycardia during rest and exercise and its relation to physical fitness. J. appl. Physiol. 28, 614-621.

Woakes, A. J. and Butler, P. J. 1983. Swimming and diving in tufted ducks, Aythya fuligula, with particular reference to heart rate and gas exchange. J. exp. Biol. 107, 311-329.

Wolf, S., Schneider, R. A. and Groover, M. E. 1964. Further studies on the circulatory and metabolic alterations of the oxygen-conserving (diving) reflex in man. Trans. Assn. Am. Physns. 78, 242-254.

Wyss, V. 1956. Electrocardiogramma di soggetti in apnea durante immersione in acqua a profondita diverse. Boll. Soc. Ital., Biol. Sper. 32, 503-506.

Discussion following Dr. Butler's presentation

Dr. Hong: I have several points that need clarification. Number one, what is the level of exercise? Have you tried different workloads? Paulev and Hansen (Cardiac response to apnea and water immersion during exercise in man. J. Appl. Physiol. 33:193-198, 1972) found that when the exercise load is heavy, then they are able to override the bradycardial response. The other issue is what Bergman et al. ("Diving reflex in man: its relation to isometric and dynamic exercise. J. Appl. Physiol. 33:27-31, 1972) described about the difference between the responses to static and dynamic exercise at same oxygen consumptions. Why is there this difference?

Dr. Butler: First of all, we did not measure oxygen consumption during swimming, but we got the same individuals to work on a bicycle ergometer, so that their heart rate was at the same level as when they were swimming and breathing. Their oxygen uptake was about one and a half liters per minute, which was about five times their resting value. So when they were swimming, just kicking their legs under water, we can say that oxygen consumption was approximately five times the resting level, so that's the first thing. We had actually done some preliminary work where the individuals used their arms as well as their legs when swimming, so that the workload was increased, and you're absolutely right, the period of tachycardia is much longer, it's almost extended to the end of the period they can stay underwater. Then towards the end, the heart rate starts to decline, but even so it doesn't go down to the resting level. Another interesting thing is that, if the individual is working (swimming) and breathing before breath-hold, the bradycardia that occurs is much faster than when breath-hold swimming starts from rest. So, whether a subject is working before breath-hold submersion, or whether work and breath-hold submersion start together, does seem to have an effect on the cardiac response. Maybe it has something to do with blood flow, muscle contraction.

Dr. Arnold: Bergman et al. (J. Appl. Physiol. 33: 27-31, 1972) compared isometric and dynamic exercise, and found that isometric exercises like hand grip were good vasoconstrictors but do not enhance venous return, whereas dynamic exercise does. There's much more bradycardia with dynamic than isometric exercise. Dr. Elsner's lowest heart rate person (Stromme et al., J. Appl. Physiol. 28: 614-621, 1970) was swimming in 25 degrees C and breath-holding <u>before</u> immersing the face. With swimming, breath-hold, followed by facial immersion, the bradycardia onset was immediate. The last point is that, the heart rate drops from 120 bpm when your exercising swimmers work. Exercise is associated with peripheral vasodilation and an increase in heart rate just as is exposure to heat, but it is due to a different requirement of the cardiovascular system. It's interesting to note that the subjects in my study who were <u>not</u> exposed to an oxygen demanding stress, eliminated their bradycardia, whereas in exercise, the same degree of bradycardia is produced dramatically by facial immersion.

Dr. Butler: From our work on birds we just modified the classical Scholander story by saying that as well as the heart and the central

nervous system, the active muscles also get an adequate blood supply. The heart is an active muscle, and when the birds are swimming underwater, their leg muscles are also active, so they get sufficient blood supply to enable them to maintain aerobic metabolism. You can do the calculations in terms of the amount of usable stored oxygen these birds have, and the duration of their dives and, generally speaking, they keep well within their aerobic limits. The same goes for the Weddell seal as well. It certainly does appear that the cardiovascular response to breath-hold diving in humans depends upon whether there is breath-hold first then exercise, or exercise first then breath-hold, or if the two events begin together.

Dr. Rennie: (I would like to mention some data from) the expedition that Dr. Data invited me to join, off the fantail of the French ship Trident, in the Ustica Harbor, in Sicily, about six years ago during lunch hours. The divers were instructed to swim in a vertical attitude watching a flag on a suspended line and were swimming against an imposed load of 3, 6, and 9 kilograms, while free breathing from SCUBA and after they spat out their mouthpiece. The purpose was to plot the heart rate as a function of time for each of these three loads. This particular little study showed that with exactly the same workloads, when divers breath-held at exactly the same depth, the heart rate dropped down to 40-45 beats per minute compared to 160-170 when free-breathing. We called that the underwater step test.

Dr. Arnold: Dr. Rennie, was the face exposed to the cold water or not?

Dr. Rennie: The water wasn't very cold, this is Ustica in Sicily. It was 22 to 24 degrees centigrade and the mask, from my recollection, was left on. They just spat out the mouthpiece.

CARDIAC ARRHYTHMIAS AS A PRECURSOR TO DROWNING ACCIDENTS

J.R. MCDONOUGH, M.D., J.P. BARUTT, B.S., AND J.C. SAFFRON, R.N.*

Introduction

The principal investigator first became interested in the possibility of water-immersion induced arrhythmia when, as an attending physician in the early 1970's, he participated in the care of a high school girl admitted to hospital because of fresh water near drowning. She was on the school swimming team and was swimming laps in a routine fashion when syncope occurred; and she sank to the bottom of the pool. Recovery was uneventful; subsequent Ecg monitoring, echocardiography and exercise treadmill were negative except for an occasional premature ventricular contraction. Years later, as a medical student, she collapsed while showering, could not be resuscitated by the Seattle Fire Department Medic-One rescue unit, and expired of apparent cardiac arrest.

The investigators propose that cardiac arrhythmias, some of which may predispose to loss of consciousness and accidental drowning, occur during water immersion; that such arrhythmias may be much more common than expected; and that they may comprise a significant proportion, perhaps the majority, of unexplained sudden death during water immersion.

Specific Aims

(1) To define prevalence of arrhythmia during scuba diving and to contrast this with arrhythmia prevalence during pre-dive and post-dive periods in order to find out whether arrhythmia occurs with greater frequency while diving.
(2) To search for potential predictors of diving arrhythmias such as presence of arrhythmia on 12-lead Ecg, treadmill exercise Ecg and during facial immersion in tepid and chilled water; and blood pressure and heart rate responses to exercise and facial immersion.

Significance

A considerable literature exists which deals with cardiocirculatory adaptations to water immersion in animals, particularly diving birds and mammals. The diving reflex results in vasoconstriction and bradycardia,

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altering the circulation to essentially a heart-brain circuit, and sparing oxygen by the use of anaerobic metabolism for performance of muscle work while submerged (1-6). The afferent limb of this reflex is activated by sensory receptors in the distribution of the fifth cranial nerve primarily by cold and wet stimuli of the eyes and nostrils. Efferent components of the reflex involve the sympathetic nervous system for vasoconstriction of most vascular beds, and cardiac vagus nerves to slow the heart (7-9). Facial immersion in cold water with breath-holding will activate this reflex in humans, and has been shown to effectively convert paroxysmal supraventricular tachycardia to normal sinus rhythm (12,13).

Head-out water immersion causes a reduction in vital capacity by displacement of blood volume from peripheral to central areas. Right atrial and pulmonary arterial pressures, and stroke volume subsequently increase. More frequent extrasystoles occur during the first few minutes of head-out immersion, and a suggested mechanism involves right atrial and right ventricular stretch as a result of increased blood volume in these structures (14,15). One study of patients who survived myocardial infarction found an increased frequency of extrasystoles during underwater swimming, and cautioned that water submersion for patients with coronary disease may be hazardous (16).

Accidental drowning while scuba diving is not a rare occurrence. A seven year study conducted by the University of Rhode Island National Underwater Accident Data Center for the years 1970-1976 reported 893 scuba related diving deaths in the United States. The state of Washington had the third highest number of such deaths at 65. A search by the investigators of death certificate records involving scuba related deaths (unexplained by nitrogen bubble disease, trauma, or other recognized causes) for the state of Washington during a four year period 1979-1982 revealed 35 such deaths, with most having occurred in Puget Sound.

A panel discussion entitled "Unexplained Sudden Death During Diving" was presented at the annual meeting of the Undersea Medical Society (San Antonio, Texas, June 2, 1984). Medical examiners from Dade and Palm Beach counties in Florida presented a series of cases with autopsy results on divers who died unexpectedly while diving. Unexplained death was found to occur in otherwise healthy divers irrespective of age, level of training, or duration of diving experience.

If, as suspected, cardiac arrhythmia is a mechanism of unexplained sudden death during water immersion, it becomes important to identify the frequency and types of arrhythmia during water immersion, those having potential hazard, and to look for tests which might predict which individuals are likely to develop such arrhythmias.

Methods

Preliminary observation and measurements on 20 healthy scuba-diving volunteers, carried out in the laboratory, consisted of a self-administered

health questionnaire, height, weight, sitting and standing blood pressure, cardiac and pulmonary auscultation, 12-lead Ecg, and maximal treadmill exercise test using the Bruce protocol. Additional data, obtained before the exercise test, involved continuous Ecg recording and blood pressure measurement before, during, and after maximal duration of full inspiratory breath holding under three conditions: In air, during facial immersion in tepid water (28° C) and during facial immersion in chilled water (5° C). The sequence of the three was randomized so as to minimize serial influences of one upon another.

After laboratory testing, study subjects were instrumented with the Holter Ecg 24-hour recorder to be worn overnight with continuous recording; and the following day taken by boat to a suitable area of Puget Sound for a scuba dive. The instrumented diver was always accompanied by one or more trained divers. Holter Ecg was continuous during pre-dive, dive, and post-dive periods, and recorder removed after approximately 24 hours of recording (table I).

Results

This report comprises the first 20 subjects who have had complete laboratory data collection of whom Holter recording has been obtained from 16, one of whom lost the signal while diving due to "shorting-out" from water contact with electrodes. We hope to eventually collect data on 100 subjects.

Duration of breath-holding is seen to vary significantly with greatest duration, 81 seconds, in air; shortest duration 53 seconds in chilled water and intermediate duration, 65 seconds, in tepid water (table II). Heart rate was obtained from the continuously recorded electrocardiogram during breath-holding (table III). Though heart rate decreased significantly in all forms of breath-holding there were highly significant differences between the three methods. Lowest heart rate 42 beats per minute was seen with chilled water; intermediate, 54, with tepid water; and least decrease, 61, with air. The effect of breath-holding on duration and lowest heart rate is seen graphically in figure 1. Despite a shorter duration of breath-holding in chilled water, there is a more profound reduction of heart rate with observed range of lowest heart rates 29 to 59 beats per minute.

Systolic blood pressure is also influenced by breath-holding in chilled water (table IV), a known reflex vasoconstricting effect of cold water sometimes made use of in clinical medicine as the cold pressor test. Diastolic blood pressure (table V) is also seen to increase significantly with breath-holding in cold water. The effects of breath-holding in air, tepid and chilled water on systolic and diastolic blood pressure, and on heart rate are shown on figure 2.

Next, I would like to turn to the subject of the electrocardiogram and arrhythmias. We began as novices in the techniques of underwater recording

Study Design

Subjects	2 F, 18 M, n = 20
Ages	16-46, mean 26.9
Clinic	PE Ecg Breathhold - Random Air H_2O 28°C H_2O 5°C ETT (Bruce)
Holter 24 hr Ecg	Pre dive Dive Post dive

table I

Breathhold Duration*
(Seconds)

	Mean	SD	t	P
Air	51.1	35.9	2.16	< .05
H_2O 28°C	64.7	27.6		
H_2O 5°C	52.6	28.8	2.01	.05

* Sequence randomized

table II

Minimal Heart Rate
Breathholding*
(beats/min)

	Mean	S.D	t	P
Air	60.8	9.2	3.86	.001
H_2O 28°C	54.2	9.5		
H_2O 5°C	41.9	8.5	5.85	< .001

* Sequence randomized

table III

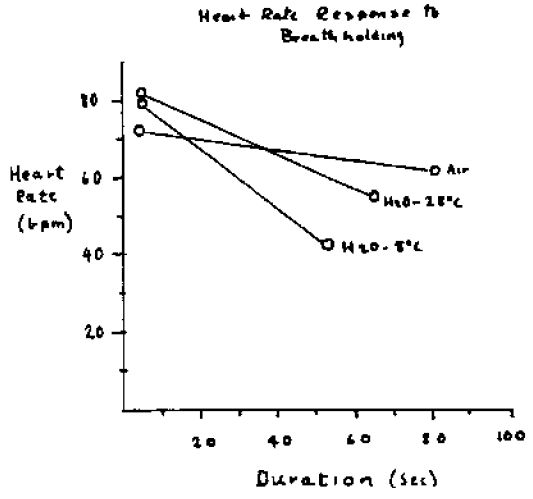

figure 1

Systolic Blood Pressure
Breathholding*
(mm Hg)

	Mean	SD	t	P
Air	127.7	23.8	.03	NS
H_2O 28°C	129.3	17.7		
H_2O 5°C	142.4	20.0	3.70	< .01

* Sequence randomized

table IV

Diastolic Blood Pressure
Breathholding*
(mm Hg)

	Mean	SD	t	P
Air	80.1	11.1	3.48	< .01
H_2O 28°C	83.4	13.1		
H_2O 5°C	89.3	12.7	2.27	< .05

* Sequence randomized

table V

and it took a year of trial and error to obtain diagnostic-quality recordings underwater. I will be showing several examples of these recordings, the first is a normal electrocardiogram using the two-channel recorder (figure 3). This was taken during Holter monitoring prior to a dive.

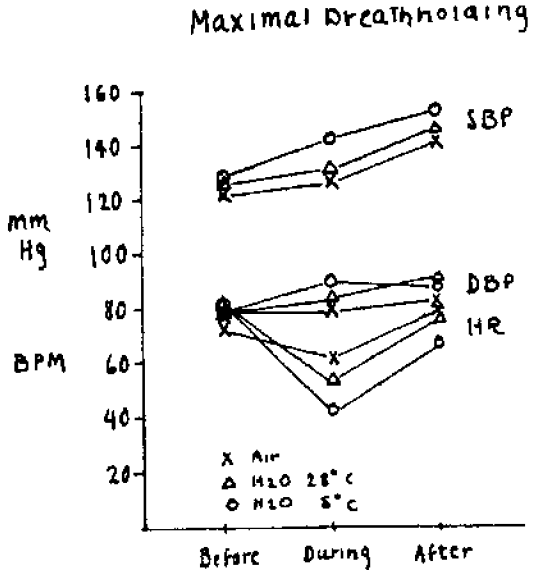

figure 2

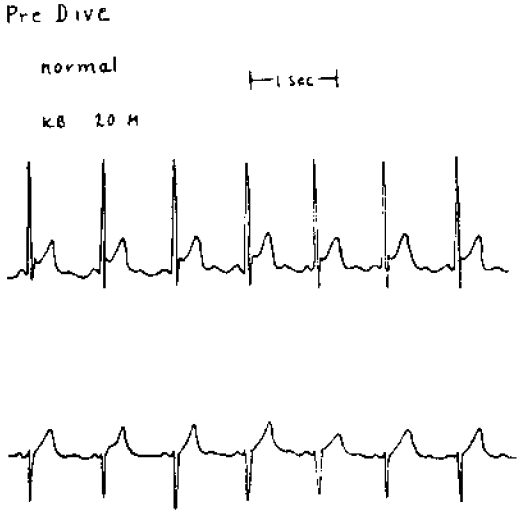

figure 3

Next (figure 4) an electrocardiogram recorded during Stage II of the Bruce multistage maximal exercise test. We see 2:1 sino-atrial exit block with the heart rate of 120 during sub-maximal exercise transiently dropping to 60. Next (figure 5) we see the same 2:1 sino-atrial exit block during

facial immersion in chilled water. This occurred early, prior to the development of bradycardia or heart rate slowing.

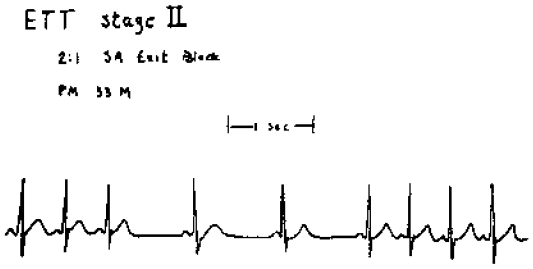

figure 4

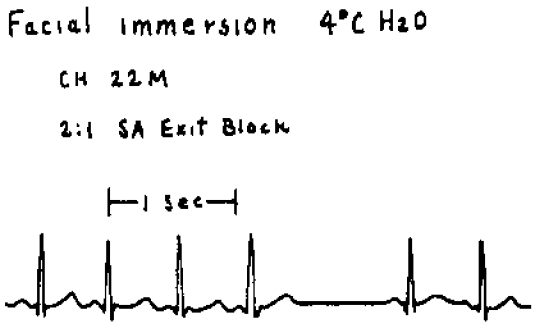

figure 5

The next (figure 6) shows facial immersion in chilled water with a non-conducted junctional contraction, a premature ventricular contraction, and a premature atrial contraction. Another subject (figure 7) during facial immersion in chilled water shows sinus bradycardia or heart rate slowing, but also shows frequent unifocal premature ventricular contractions, and a brief run of ventricular tachycardia defined as 3 or more consecutive ventricular contractions.

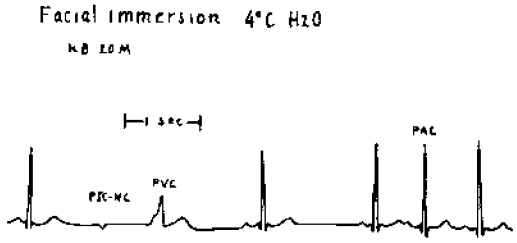

figure 6

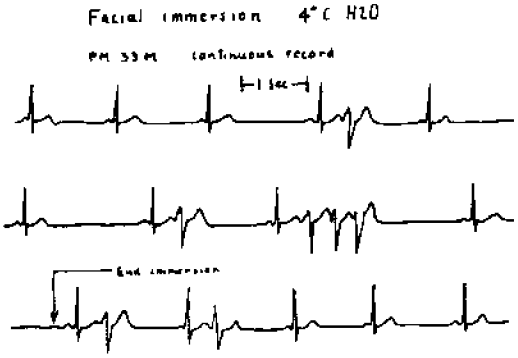

figure 7

Next (figure 8) we see a single premature ventricular contraction during Holter monitoring. This occurred before the dive. Another (figure 9) pre-dive Holter shows two unifocal premature ventricular contractions. There is also a single premature atrial contraction. Next (figure 10) during pre-dive we see multiform premature ventricular contractions. The tracing is not continuous in order to bring in from a later portion of the tape the premature ventricular contraction which originated from a different vnetricular focus than the first. In clinical cardiology practice we regard multiform premature ventricular contractions as more serious than unifocal patterns.

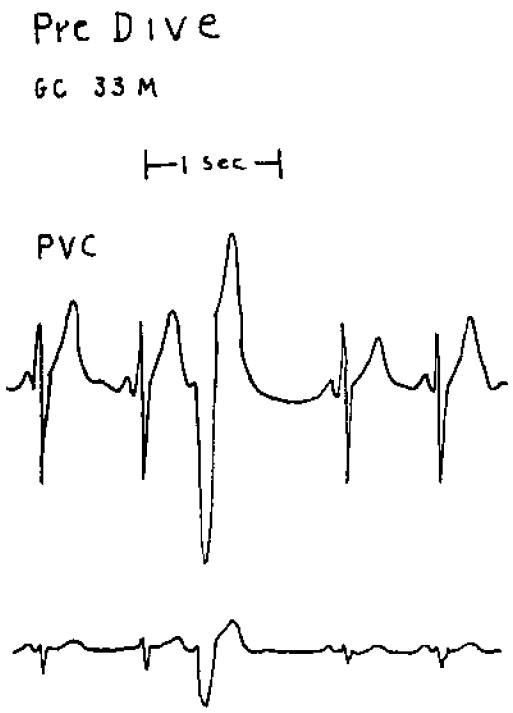

figure 8

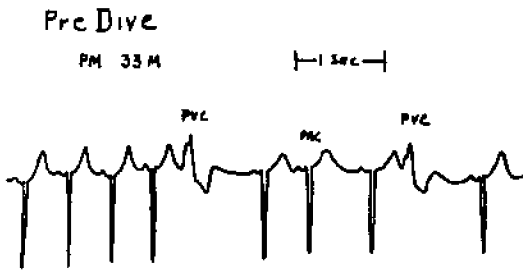

figure 9

The next (figure 11) occurs during a dive, and shows frequent premature atrial contractions and a cluster of paired premature atrial contractions. In clinical practice we regard occasional premature atrial contractions as less serious than unifocal premature ventricular contractions. However, frequent premature atrial contractions, and especially pairs, often precede more serious forms of supraventricular arrhythmia such as atrial tachycardia, atrial flutter, or atrial fibrillation. Next (figure 12) shows a single premature ventricular contraction during a dive. Next (figure 13) is another premature ventricular contraction recorded during a dive..

Next (figure 14) we see multiform premature ventricular contractions during a dive. The next (figure 15) shows paired premature ventricular contractions during a dive. I would like to remind the audience that all of these examples of abnormal cardiac rhythms, or arrhythmias, occurred in healthy young persons.

Let us now turn to a classification of these arrhythmias (table VI). We can classify all observed arrhythmias into 5 categories: Premature supraventricular contractions include atrial and junctional, and we saw examples of both. Complex supraventricular arrhythmias include sino-atrial exit block, which was seen, and several varieties not observed: Including atrial tachycardia, atrial flutter, and atrial fibrillation. Unifocal

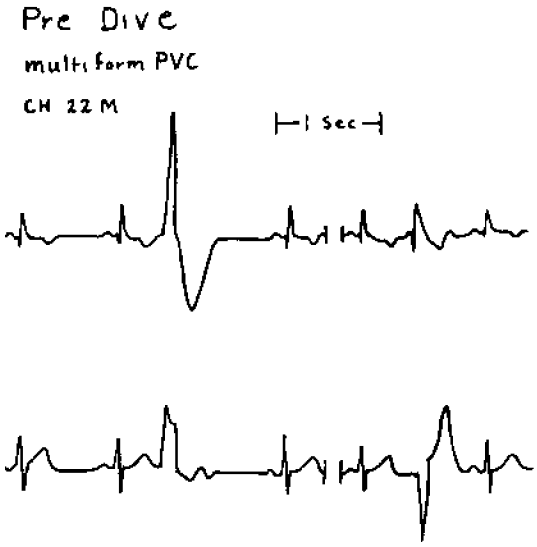

figure 10

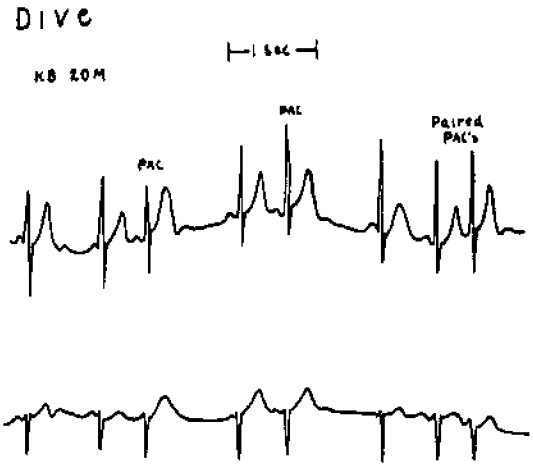

figure 11

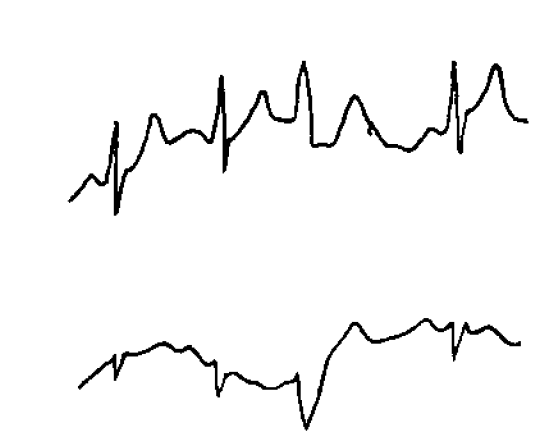

figure 12

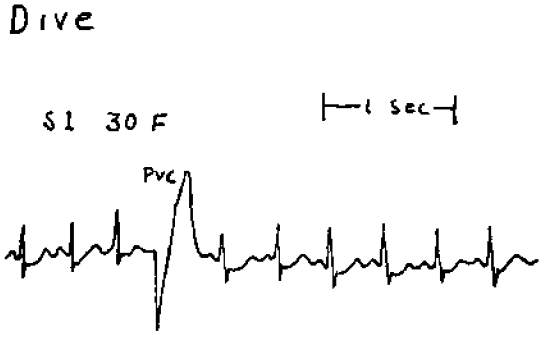

figure 13

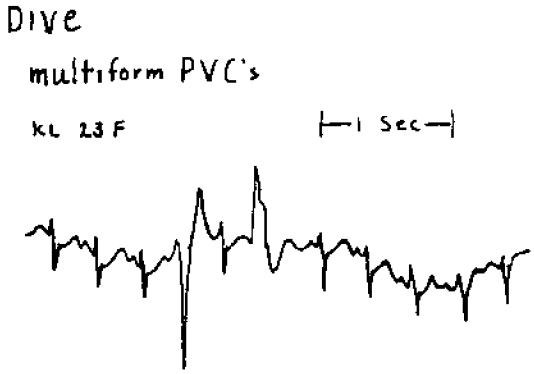

figure 14

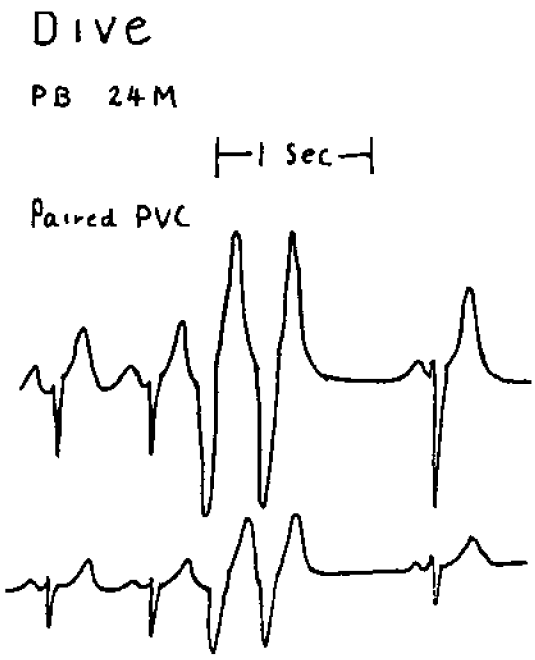

figure 15

premature ventricular contractions have also been presented. Complex ventricular arrhythmias include multiform and paired premature ventricular contractions, and ventricular tachycardia, all seen; and ventricular fibrillation, fortunately not seen. The last category is that of pauses, defined as exceeding 1.6 seconds in length, and exceeding the preceding cycle interval by more than 25 percent. Next (table VII) we will examine the frequency of these categories for all methods of detection. First, let us examine the totals. Of 252 specific arrhythmic events, the most common represented form was premature supraventricular contractions, 147, the next most common unifocal premature ventricular contractions. 45, then complex ventricular arrhythmias 37. Five instances of complex supraventricular arrhythmias were seen and all were 2:1 sino-atrial exit block. Six pauses were seen. When arrhythmia frequency is examined by type of recording we see that only 2 of 252 arrhythmic events were seen in standard 12-lead Ecg, a rather poor showing, while 36 were detected by exercise testing, 60 with breath-holding, and 154 during Holter recording. Next (table VIII) we will examine the distribution of these 5 categories of arrhythmia by breath-holding category. Notice that the majority, 38 of 60, are detected by chilled water immersion whereas air (10) and tepid water (12) detected far fewer. All pauses occurred with breath-holding, that is, none were detected during Holter recording, including dive, and none were seen during standard resting or exercise electrocardiography. Of the more serious categories of arrhythmia, that is the unifocal

Classification of Arrhythmias

PSVC Premature Supraventricular Contractions - atrial and junctial

CSVA Complex Supraventricular Arrhythmias - SA Exit Block (A. Fib. Flutter, A-V Block not seen)

UPVC Unifocal Premature Ventricular Contractions

CVA Complex Ventricular Arrhythmia Paired, Multiform PVC's, VT (VF not seen)

Pauses >1.6 Sec and >1.25x Preceeding R-R Interval

table VI

Arrhythmia Analysis

by Type and Method

	Rest Ecg	ETT	3 Breathhold	Holter	Total
PSVC	1	27	32	87	147
CSVA	0	4	1	0	5
UPVC	1	5	14	37	57
CVA	0	0	7	30	37
Pauses	0	0	6	0	6
Total	2	36	60	184	252

table VII

premature ventricular contractions and the complex ventricular arrhythmias, 18 of 21 occurred during cold water immersion. Next (table IX) we see the distribution of 5 categories of arrhythmia by Holter category, that is pre-dive, dive, and post-dive components. First, note that the categories are now reduced to three, as complex supraventricular arrhythmias and pauses were not seen.

Of 154 arrhythmic events noted, 97 occurred pre-dive, 45 during dive, and 12 during post-dive recording. Also note the important differences in time of recording, with pre-dive 267 hours, dive 6.5 hours, and post-dive 89 hours. We shall have more to say about this later when we discuss arrhythmic events per hour.

Arrhythmia Analysis

by Breathholding Category

	Air	H_2O 28°C	H_2O 5°C	Total
PSVC	8	7	17	32
CSVA	0	0	1	1
UPVC	0	1	13	14
CVA	2	0	5	7
Pauses	0	4	2	6
Total	10	12	38	60

table VIII

Arrhythmia Analysis

by Holter Category

	Predive 267 hr	Dive 6.5 hr	Postdive 89 hr	Total 362 hr
PSVC	59	18	10	87
CSVA	0	0	0	0
UPVC	13	22	2	37
CVA	25	5	0	30
Pauses	0	0	0	0
Total	97	45	12	154

table IX

We will next (table X) look at arrhythmias detected for each of the 20 subjects tested. Six had no arrhythmias recorded by any method; however, it needs to be pointed out that subjects 6, 13, 18, and 19 have not had Holter recording as of yet, and subject 1 had Holter recording pre and post-dive, but not during dive because the signal "shorted out" because of a water leak. Be that as it may, 14 subjects had arrhythmias detected by one or more of these methods, and 9 occurred while diving. It is relevant to ask the question how many of these 9 are detected by the other methods? The resting electrocardiogram is a poor detector, as only one of the 9 is concordant by this method. The exercise treadmill test is not much better, able to detect only 2 of the 9. Breath-holding in air only 2 of 9, breath-holding in tepid water 4 of 9, and breath-holding in chilled water 6 of 9. Chilled water breath-holding detected all of these detected by tepid water breath-holding, and one of the two detected by breath-holding in air. The Holter pre-dive detected 6 of the 9, while adding the Holter post-dive did not improve the detection. The total of all methods used could provide detection of 8 of the 9 persons with diving arrhythmias. Next (table XI) we will examine the distribution of the more serious arrhythmias, that is unifocal premature ventricular contractions, and complex ventricular arrhythmias combined, for each of 20 subjects. The form of analysis is the same, though fewer individuals are seen to have arrhythmia. Still, 5 of the 15 subjects recorded during a dive were found to have these ventricular arrhythmias. The resting electrocardiogram was totally insensitive in detection while the exercise treadmill test would identify only one. Chilled water breath-holding would detect 3 of the 5 whereas the other breath-holding methods did not increase the sensitivity of detection. The pre-dive Holter detected 3 of the 5, while the combination of pre-dive Holter and cold water breath-holding detected 4 of 5. The other methods, when added, did not improve detection.

Now (table XII) let us turn to an analysis of arrhythmic events as a function of time, and examine the periods of Holter recording as dive and non-dive categories. The non-dive categories now includes the combination of pre-dive and post-dive categories discussed previously. Note that all three categories of arrhythmia occur with far greater frequency during dive as compared with non-dive periods. Premature supraventricular contractions occur 14 times more often during dive as during non-dive periods. Unifocal premature contractions occur 80 times more often while submerged. Complex ventricular arrhythmias occur 11 times more often during a dive, and total arrhythmias are seen 22 times more often while submerged than when out of water. These differences are all highly significant. A graphic presentation (figure 16) of these differences also highlights the strikingly higher frequency of all categories of arrhythmias while submerged.

Discussion

The mechanism of increased arrhythmia frequency cannot be ascertained by the present study. Professor Lundgren has demonstrated in earlier studies a higher frequency of arrhythmia during head-out water immersion associated with decreased vital capacity, presumably secondary to a more

Arrhythmia Analysis

by Subject and Method

total arrhythmia

Subject	Base Ecg	ETT	Air	28° H2O	5° H2O	Dive	Pre Dive	Post Dive
1								
2								
3					X	X		
4		X		X	X	X	X	
5				X	X	X	X	
6								
7				X	X	X	X	
8	X						X	
9		X			X		X	X
10		X		X	X	X	X	
11								
12								
13			X	X	X			
14								
15	X		X		X	X	X	X
16						X	X	
17						X		
18		X			X			
19								
20			X			X		
Total	2	4	3	5	9	9	8	2

table X

Arrhythmia Analysis

by Subject and Method

ventricular arrhythmia

Subject	Base Ecg	ETT	Air	28° H2O	5° H2O	Dive	Pre Dive	Post Dive
1								
2								
3					X			
4		X			X	X	X	
5					X			
6								
7					X	X	X	
8	X							
9		X			X		X	X
10				X	X	X		
11								
12								
13								
14								
15			X		X		X	X
16						X	X	
17						X		
18		X			X			
19								
20								
Total	1	3	1	1	8	5	5	2

table XI

Arrhythmia Analysis

by Holter Category

Events per Hour

	Dive 6.5 hr	Non-Dive 356 hr	Dive / Non-Dive 362 hr	P
PSVC	2.75	0.19	14.2	< .001
UPVC	3.36	0.04	80.1	< .001
CVA	0.77	0.07	10.9	< .05
Total	6.88	0.31	22.5	< .001

table XII

Holter Ecg

Events per Hour

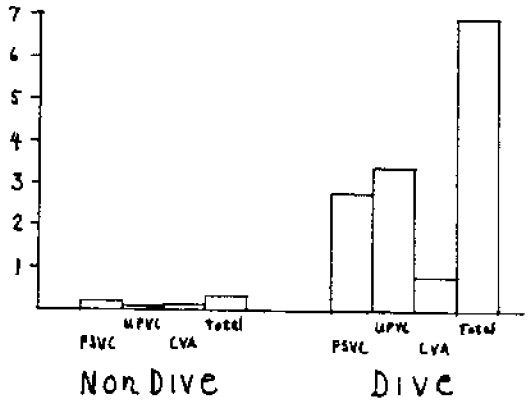

figure 16

central displacement of blood volume. He has postulated that increased filling of cardiac chambers with resultant increased stretch, particularly of right atrium and right ventricle may be the mechanism of increased arrhythmia.

It is of interest in the present study that neither excess bradycardia nor excess tachycardia were noted on any of our dives. We might by inference therefore suggest that neither excess vagal tone (conducive to bradycardia) nor excess sympathetic tone (conducive to sinus tachycardia) provide an adequate explanation of the mechanism of excessive arrhythmia while diving.

We can only speculate as to the connection between arrhythmia excess and risk of sudden death in healthy young subjects. This risk in patients with cardiac disease, especially the syndromes of myocardial ischemia, is well known. I can assure you that many of the arrhythmias seen in these divers when seen in patients in a coronary care unit would initiate a response of immediate treatment with an antiarrhythmic drug such as lidocaine. The onset of the lethal arrhythmia ventricular fibrillation is nearly always thought to be a single premature ventricular contraction. Figure 17 depicts such an event in a patient being monitored in a coronary care unit. Present data are sufficient to speculate that the striking excess of ventricular premature contractions while diving may, in exceptional circumstances, lead to ventricular fibrillation, and that this may be the major cause of unexplained sudden death seen with scuba diving and other forms of water activity.

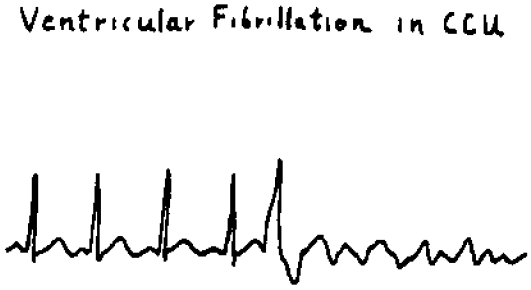

figure 17

Conclusions

1. The primary conclusion is that strikingly higher frequencies of arrhythmia are noted when scuba diving as when out of water.
2. Arrhythmia types include premature supraventricular contractions, unifocal premature ventricular contractions, multiform premature ventricular contractions, and paired premature ventricular contractions while diving; and ventricular tachycardia, sinus bradycardia, pauses, and sino-atrial exit block during breath-holding with facial immersion in water chilled to 5° C.
3. Resting and exercise electrocardiograms are poor predictors of

which individuals will develop diving arrhythmia.

4. Facial immersion in chilled water during held breath, and Holter Ecg recording appear to be the best methods of identifying individuals who later develop arrhythmia while diving. Each separately detected 67 percent, while combined between 80 and 90 percent of those who later developed diving arrhythmia in this small sample.

References

1. Elsner R, Franklin DL, Van Citters RL, and Kenney DV: Cardiovascular Defense Against Asphyxia. Science 153:941-9, 1966.

2. Anderson HT: Physiological Adaptations in Diving Vertebrates. Physiol. Rev. 46:212-243, 1966.

3. DeBurgh Daly M, Elsner R, and Angell-James JE: Cardiorespiratory Control by Carotid Chemoreceptors During Experimental Dives in the Seal. Am. J. Physiol.: Heart Circ. Physiol. 1(5):H508-H516, 1977.

4. Kawakami Y, Najelson BH, and DuBois AB: Cardiovascular Effects of Face Immersion and Factors Affecting Diving Reflex in man. J. Appl. Physiol. 23:964-970, 1967.

5. Whyane Jr. JF, Smith NT, Eger II EI, Stoelting RK, and Whitcher CE: Reflex Cardiovascular Responses to Stimulated Diving. Angiology 23:500-508, 1972.

6. Strauss MB: Physiological Aspects of Mammalian Breath-Hold Diving: A Review. Aerospace Med. 41:1362-1381, 1970.

7. Khurana RK, Watabiki S, Hebel JR, et al: Cold Face Test in the Assessment of Trigeminal-Brainstem-Vagal Function in Humans. Ann. Neurol. 7:144-149, 1980.

8. Gandevia SC, McCloskey DI, and Potter EK: Reflex Bradycardia Occurring in Response to Diving, Nasopharyngeal Stimulation and Ocular Pressure, and its Modification by Respiration and Swallowing. J. Physiology (London) 276:383-394, 1978.

9. Jones DR, Milson WK, and West NH: Cardiac Receptors in Ducks: The Effect of Their Stimulation and Blockage on Diving Bradycardia. Am. J. Physiol. 238 (Regulatory Integrative Comp. Physiol. 7): R50-R56, 1980.

10. Valladares BK, and Lemberg L: Use of the "Diving Reflex" in Paroxysmal Atrial Tachycardia. Heart and Lung 12(2):202-205, March 1983.

11. Wildenthal K, and Atkins JM: Use of the "Diving Reflex" for the Treatment of Paroxysmal Supraventricular Tachycardia. Am. Heart J. 98(4):536-537, October 1979.

12. Condry P, Jain A, Marshall R, and Bowyer A: Ventricular Tachycardia Caused by the Diving Reflex. Lancet 2(7947):1263, December 20, 1975.

13. Hughes T, Carter J, and Wolf S: Disorders of Cardiac Conduction Accompanying the Dive Reflex in Man. Pav. J. Biol. Sci. 16(1):25-33, January-March 1981.

14. Arborelius Jr. M, Balldin UI, Lilja B, and Lundgren CEG: Hemodynamic Changes in Man During Immersion with Head Above Water. Aerospace Med. 43(6):592-598, 1972.

15. Kurss DI, Lundgren CEG, and Påsche AJ: Effect of Water Temperature on Vital Capacity in Head-Out Immersion. Proceedings of the Seventh Symposium on Underwater Physiology, Eds. Bachrach and Matzen, Undersea Med. Soc, Inc., Bethesda, MD, 1981.

16. Giesler H, Jungmann H, and Stein G: Sind Koronarkranke beim Tauchen durch Herzrhythmusstörungen gefährdet? Med Klin 75:518-523, 1980.

Discussion following Dr. McDonough's presentation

Dr. Lanphier: Well I wasn't quite clear on what proportion of SCUBA diving vs breath-hold diving was involved here. Can you clarify that as the first question?

Dr. McDonough: They were separate, and at different times. The breath-hold testing was conducted in the laboratory as part of a busy cardiology practice on a day off and and three types of breath-holding were done. These were done in sequence with intervals of probably 5 to 10 minutes in between, and because it was suspected that perhaps there might be an influence of one upon the other when done serially, the sequencing was randomized. I'm pleased that we did this, because of what I heard yesterday from Dr. Arnold's comments about a second breath-hold bradycardia being more profound than a first. The SCUBA dive itself was done on a day when an individual was connected to a continuously recording electrocardiogram obtained for 24 hours. The SCUBA dives were usually down to somewhere between 50 and 110 feet, and they lasted 25 to 35 minutes.

Dr. Lanphier: Well, I wanted to see if we could figure out, from what Dr. McDonough has done, what role hypoxia might have had in promoting these arrhythmias, but obviously not during scuba diving. Furthermore, face immersion might have had a role, but it's hard to say. Now one final thing, one of the big puzzles in loss of consciousness in divers is what happens to these individuals who made a significant dive and are now heading for shore on the surface, perhaps snorkeling, and all of a sudden they just go dead in the water and are supposed to have had some kind of fatal cardiac event. At least, that seems the best explanation. And, one of my thoughts about this, is that the change in position might have something to do with this: that they've been immersed where position doesn't make all that much difference, they come up in a vertical position, and then when they reach the surface, they go to the horizontal position to swim. If they have trouble, they may go vertical (head-out) again, which changes quite a few things. I just wonder if you have any thoughts about that.

Dr. McDonough: None really beyond what I've expressed, except that we know the posture changes have important influences on circulatory dynamics and you know, it's conceivable that that posture change may have an important role.

Dr. Lanphier: I should add that most of these recorded cases have been in relatively cold water.

Dr. Lundgren: As a SCUBA diver comes up the surface to return to shore, he/she is often low on air and likely to take out the mouthpiece and change to snorkel; this will cause a rather hefty immersion effect on their circulation, with atmospheric pressure in the lungs and more or less the full immersion effect on the circulatory system.

Dr. Lanphier: I would assume very few people would try to swim to shore using their tank.

Dr. Lundgren: Right.

Dr. Shiraki: I have noted many cases of arrhythmias during head-out immersion.

Dr. Lin: Your high frequency of arrhythmias during scuba diving seems to agree well with Dr. Hong's studies, especially in cold water, but, I am quite surprised to see in your study that face immersion had a high incidence of arrhythmias. We, in 10 to 15 years of laboratory studies, rarely observed arrhythmias during face immersion even during cold water immersion. In the field, however, arrhythmia seems to be a common occurrence.

Dr. McDonough: We see a lot of arrhythmias during facial immersion.

Dr. Lin: That's surprising.

Dr. Landsberg: A similar study was done in 1970 by Sem-Jacobsen in Norway. At that stage he suggested a Panic Button for Aircraft Pilots to sound an alarm when they developed Cardiac Arrhythmias. The Panic Button was wired to a lead which monitored the heart beat and would sound an alarm when the Arrhythmia occurred. He recorded several potentially fatal Arrhythmias in operational divers and actually described Ventricular Fibrillation in one diver who was particularly apprehensive during a dive. To what extent are we justified in subjecting prospective divers to this type of selection testing, which could be fatal, in order to exclude them from operational diving?

Dr. McDonough: It's a very interesting question. One of the things that we would be interested in doing as a continuing study is to repeat the dive using Holter electrocardiogram recording in the same subjects, to see whether the same individuals develop arrhythmia with repeat testing.

Dr. Boysen: Just a couple of quick questions. First of all, have these subjects discontinued nicotine, caffeine, and that type of thing before they were tested?

Dr. McDonough: They were leisure time divers in the young age groups that were tested. Generally they were very careful about not using beverages or eating before they went in the water. Smoking was virtually unknown for most of them, alcohol was in light use by some of them. In general, there was no use of coffee, other beverage or food within probably about 2 or 3 hours of diving.

Dr. Boysen: Was there any correlation between time of the dive and the occurrence of these arrhythmias?

Dr. McDonough: I did not see a pattern.

Dr. Boysen: The other issue was, as you said, the importance of repetitive dives. Can you reliably reproduce arrhythmias with a facial immersion in subjects?

Dr. McDonough: We haven't done this yet.

Dr. Arnold: I have done repetitive studies and though I wasn't looking for it, I have found arrhythmias in several healthy subjects. Arrhythmias frequently occur at a heart rate which one would expect to be the ventricular inherent automaticity, around 40 beats per minute. When the heart rate is slowly aiming towards 40 and going beyond, there are many subjects who will start throwing ventricular escape rhythm at about that point, so that they're going to bottom out at 40 bpm even though it looked like they were aiming for 30 bpm. And I've seen atrial premature contractions also. It appears that a given individual may have a tendency towards a stereotyped arrhythmia, my brother for instance. He, on repeated dives, 3 minutes apart would show ventricular escape at 40 beats per minute.

Dr. McDonough: Let me comment upon that, because I think that's an important distinction. We did see escape rhythms, junctional or ventricular, during breath-hold facial immersions on some occasions. During the SCUBA dives I thought we would see a lot of escape rhythms, but we didn't. The SCUBA diving arrhythmias were all ectopic. Furthermore, we did not see bradycardia during SCUBA diving. These were free swimming persons whose ambient heart rates generally ranged from 80 to 140 beats per minute.

Dr. Arnold: And they had a face mask.

Dr. McDonough: Yes, a face mask. It is also of interest to me that our divers, who had these recordings, were unaware of the occurrence of arrhythmic heart beats while they were diving, they were totally unaware and there was no apparent effect of these arrhythmias upon their underwater overall performance. I would tend to agree then that the arrhythmic heart beats did not importantly interfere with cardiac or circulatory performance in the water. Conversely, they may be a precursor to something more severe, and potentially lethal, such as sustained ventricular tachycardia or ventricular fibrillation.

GENERAL DISCUSSION

Dr. Elsner: Dr. Hong, I quite agree with what you have shown in regard to the effects of temperature in face immersion, there's clearly no doubt about that. But I think there might still be room for some specific effect of some magic quality of water. We performed an experiment on subjects lying quietly totally immersed in water, 25 degree water approximately, and the subject is holding his breath for a total of 100 seconds. First he puts his face in the water and, 20 seconds later, a signal tells him to lift his face from the water still holding his breath, his face is dripping wet, not dry, and he has an increase in rate of about 10 beats a minute for the rest of the period. When the reverse experiment is performed, in which he holds his breath in the air, and this time puts his face into the water, and responds with further slowing of heart rate. How would this response be simply a temperature effect?

Dr. Arnold: I have two figures to show. The first (Fig. A) shows the effect of varied recovery times between two apneic 30-second facial immersions on the degree of bradycardia induced by those dives at room temperature and in ice water. (Arnold, R.W., S.M. Krejci, and R. Elsner, 1979 - Unpublished results). Note that short recovery times

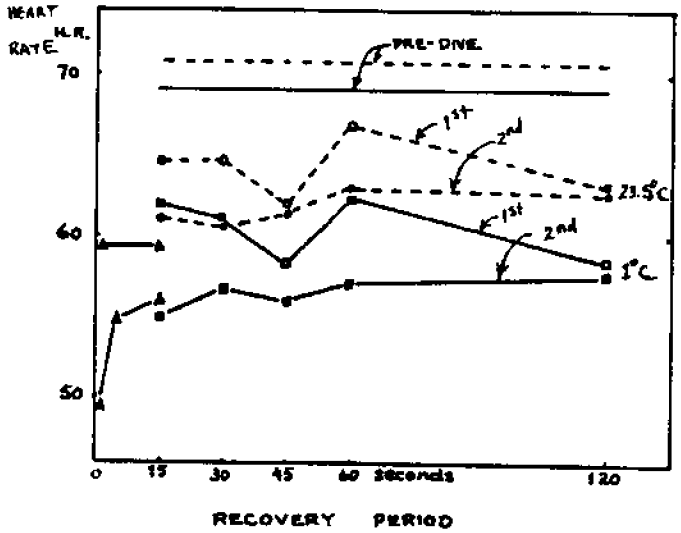

Fig. A. The Recovery Time for Human Diving Bradycardia. The effect of varying the recovery period between two, identical, consecutive, 30-second breath hold facial immersion dives. Open figures indicate first dive, solid figures indicate subsequent dive. Circles indicate water temperature of 23.5°C, n=10. Squares indicate water temperature of 1°C, n=10 and triangles indicate water temperature of 1°C, n=4.

were associated with an enhanced second dive bradycardia especially in ice water. The subject's face was cold and dripping wet just before the subsequent immersion. The second figure (Fig. B) shows the effect on facial temperature (measured by thermocouple) of a series of ice water facial immersions before and during exposure to intense ambient

heat. The bradycardia induced by the first six dives was equivalent. This demonstrates that the facial contact with cold medium is more important than the pre-immersion facial temperature. These figures support Dr. Elsner's comment that additional factors, other than just facial cooling, are involved when facial immersion in cold water induces diving bradycardia. However, immersion medium temperature or heat transfer are probably the most important factors.

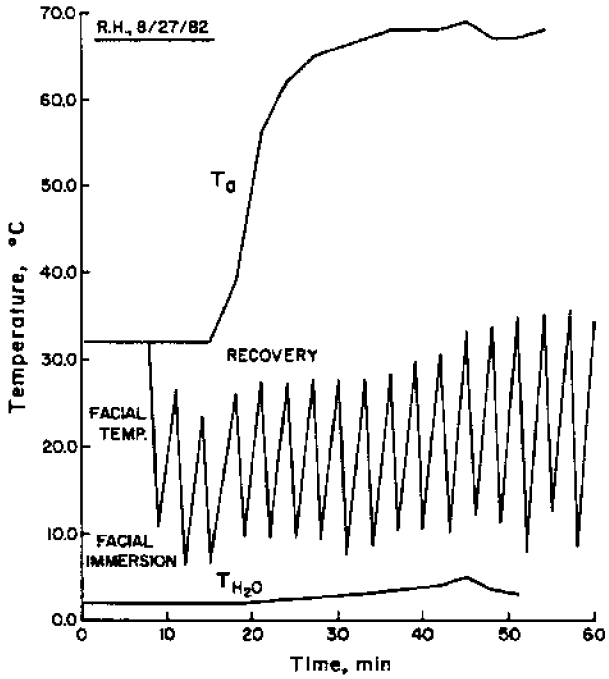

Fig. B. The effect of repeated facial immersions on facial temperature before and during exposure to intense ambient heat.

Dr. Van Liew: I'd like to ask Dr. Hong to sum up what we have learned.

Dr. Hong: Yes, I'm going to do that. I think the most important thing is that there are differences between human divers and diving mammals, and these two should not be confused. There are qualitative similarities to be sure, but there are some differences that are major. Now in what respect? Number one, there's no temperature sensitivity in animals, while this is one of the most important elements in human subjects. The blood pressure does not go up in diving mammals, it goes up significantly in the human subjects in whom we also observe cardiac arrhythmias. To say a few. These are at least three major differences. I think the bottom line is that overall there is the same conservation of oxygen even in human divers, and that's the result of my survey of the literature. However, now any oxygen conserving attempt displayed by the human diver is offset by

the increase in blood pressure, which offsets the advantage one gains by bradycardia, so it is fair to say that, yes, we can describe some oxygen conservation in human divers, but not to the extent that we find in animals. Even diving mammals, sometimes, don't have oxygen conservation. So maybe the difference is no big deal.

Dr. Lin: Well, I couldn't agree with that; the reason is this: we can describe reduced oxygen consumption experimentally. However, this doesn't necessarily mean oxygen conservation. For example, the body consumes less oxygen under many physiological conditions such as hibernation, torpor, or in accident in ice cold water. Such levels of O_2 consumption are appropriate for these conditions. Oxygen consumption is reduced because demand of oxygen is reduced, but, are they really conserving oxygen? At a body temperature of 37°C, O_2 consumption of 250 ml/min is normal, at 10°C, 50 ml/min is normal. Contrary to these, diving mammals obviously use less oxygen during diving where physical activity increased. In order to describe O_2 conservation, an equivalent physiological demand should exist and use a reduced amount of oxygen.

Dr. Arnold: I think another point is that the human diving response is quite variable. When trained seals are forced to dive, their responses are quite similar whereas when humans are asked to dive, their cardiovascular response can be reasonably variable from subject to subject.

Dr. Hong: Another important issue is that there is a myth in diving physiology introduced by several physiologists: in case of drowning in cold water, the diving reflex saves one's life. I don't know if this is true. This is a big issue, actually. I was hoping to get into that discussion, but we ran out of time. But maybe sometime we can spend 10 to 15 minutes and really talk to each other. This is a dangerous myth and we've got to do something about it.

PATHOPHYSIOLOGY OF NEAR-DROWNING: ASPIRATION VS. NON-ASPIRATION

P. G. Boysen, M.D.

Various Responses to Submersion

The same sequence of events does not occur in all victims of submersion. In fact, what ultimately happens depends on the patient's degree of laryngospasm, type and length of immersion, and underlying physiological condition.

It appears that most victims of submersion accidents sustain laryngospasm initially. In about 80% of cases, the subsequent carbon dioxide build-up results in laryngeal relaxation before the onset of respiratory paralysis. This sequence allows water to enter the lungs, resulting in "wet drowning"--a process that usually occurs in patients who struggle in the water, hold their breath, and make violent, terminal inspiratory efforts as they asphyxiate (6). Alternatively, some 10-20% of victims develop sustained laryngospasm until the onset of respiratory paralysis (3). This effectively bars the entrance of water into the lungs. As a result, early resuscitative efforts in these latter patients often succeed, and most of them recover without complications, unless aspiration of water entering the stomach occurs during resuscitation. This type of episode, which is sometimes termed "dry drowning," typically presents in patients who accidentally

fall in the water, as opposed to those who suffer from exhaustion following prolonged swimming or struggling (1,2).

Also of importance in submersion accidents are the type and length of immersion. For example, a sudden immersion in cold water may result in cardiac arrest mediated through vagal mechanisms. This happens most commonly in patients depressed by alcohol (1,6). On the other hand, some patients become profoundly hypothermic due to prolonged exposure in water. The water in which the submersion accident occurs need not be particularly cold in order to cause hypothermia. In contrast to the drowning deaths when the Titanic sank in icy seas, the Laconia disaster proves that hypothermia can incapacitate victims even in 15°C (60°F) water in the summertime (7). Moreover, actively struggling or swimming a long distance can cause the more rapid onset of hypothermia, since such physical activity leads to peripheral vasodilation and increased body heat loss.

Another type of submersion accident can occur in swimmers who purposely alter their physical condition before swimming. As a means of increasing their endurance, these swimmers hyperventilate, hoping that by becoming hyperoxic they will be able to further their underwater swimming distance during a breath-hold. But they also are obviously hypocapnic (more so than hyperoxic) and may lapse into unconsciousness before the

rising arterial carbon dioxide tension initiates inspiration or prompts them to surface and terminate the breath-hold.

Multisystem Pathophysiology

It has been difficult to understand pathophysiological changes that occur in near-drowning victims. Indeed, extrapolation from early animal experiments helps little and, more often than not, leads only to confusion and misinterpretation. Early dog experiments dealt with aspiration volumes exceeding 22 ml/kg of fluid. Clinical studies, however, now suggest that only about 15% of humans involved in drowning accidents suffer physiologic changes consistent with this degree of aspiration (8,9). Because of this disparity and the many myths generated by reliance on such experimental studies, we must take a fresh look, on a system-by-system basis, at pathophysiologic changes occurring in most near-drowning patients.

PULMONARY. Although fresh and salt water aspiration cause somewhat differing effects on the lungs, some changes occur despite the type of fluid. Among those common lesions are factors that cause severe hypoxia, including alveolar inflammation, right-to-left shunting, and injured alveolar membranes due to foreign materials. When patients aspirate fresh water, however, two characteristic events occur: a) surfactant abnormalities arise, which result in a lowered

functional residual capacity, unstable alveoli, and stiff, noncompliant lungs, and b) the lung absorbs much of the fluid rapidly (10,11). But since sea water is hypertonic, this fluid remains in the lung for a period of time and exerts an osmotic gradient, which causes a further influx of physiologic fluid into affected alveoli.

NEUROLOGIC. Near-drowning victims frequently present with signs of cerebral hypoxia, especially coma, and the metabolic acidosis produced by tissue hypoxia further complicates this problem by compromising cerebral circulation. The cumulative effects of these insults can be devastating and include increased intracranial pressure due to brain swelling and, possibly, brain stem herniation and sudden death (12,14). However, the degree to which neurological changes occur varies considerably, depending on: a) the extent and time period of anoxia, b) the previous condition of the patient, and c) the temperature of the water. Obviously, the shorter the period of anoxia and the better the patient's underlying condition, the more favorable is the outlook. Likewise, the colder the water, the better is the chance of neuron survival. The evidence for this latter effect comes from the numerous reports of successful resuscitation and the complete return of normal neurological function following prolonged (10-40 minutes) submersion in cold water (8,12,13,15). The importance of such reports is that the

absolute anoxic time limit for neurological survival is unknown in such cases (8,14).

HEMATOLOGIC. Two myths surrounding near-drowning are: a) that aspiration of fresh water decreases the patient's hemoglobin and hematocrit by hemodilution and hemolysis and b) that sea water aspiration increases these same values due to a positive fluid shift into the lungs. Actually, clinical observations now make it clear that neither of these two effects occur in practice. The reason is that most near-drowning patients aspirate far smaller quantities than required to cause such changes, and equilibration of any fluid shifts occurs rapidly. In fact, a decreased hemoglobin level should initiate a search for a source of blood loss (1).

CARDIOVASCULAR. While dogs that aspirate huge quantities (44 ml/kg) of water develop ventricular fibrillation, one rarely encounters this phenomenon in normothermic near-drowning victims. As with hemodilutional changes, ventricular fibrillation occurs rarely because most survivors of submersion accidents aspirate a lesser quantity than this. Moreover, patients who aspirate larger quantities of fluid probably die before receiving medical therapy (16, 17). The cardiovascular change that can occur, however, is arterial hypotension. In general, the degree to which this manifests primarily depends on the patient's vascular tone, which is affected by the degree of blood loss, the patient's peripheral body temperature, and

levels of circulating catecholamines and carbon dioxide. We have assessed the hemodynamic changes that occur in a laboratory model during asphyxia, fresh water aspiration and salt water aspiration. No matter which of the above occurs, there is arterial oxygen desaturation and mixed venous oxygen desaturation. During asphyxia there is no change or an increase in arterial pressure, pulmonary artery hypertension and a fluctuating central venous pressure with continued inspiratory efforts. Fresh water aspiration is followed by hypotension, increased PA pressure and a rising CVP; cardiac output falls by 20-30%. After salt water aspiration hypotension is severe but PA pressure and CVP are elevated; cardiac output is severely reduced by 50%.

In each case, the most notable changes are 1) arterial and mixed venous oxygen desaturation; 2) elevated PA pressure (secondary to #1) and 3) fall in cardiac output, also in part due to hypoxemia. Clearly the most important aspect of early treatment of the near-drowned is management of the airway and maintenance of oxygenation.

METABOLIC. Metabolic acidosis is one of the most significant abnormalities occurring in near-drowning victims, and in most cases, it results almost totally from hypoxia rather than poor tissue perfusion. This problem can be so severe that some patients present with a pH in the range of 6.8-6.9. The severity of the metabolic acidosis, however does not reliably

predict survival, since some patients with a pH in this low range do survive if they receive appropriate treatment.

As mentioned above, hypothermia acts as a double-edged sword with victims of submersion accidents. On the one hand, it may rapidly claim the patient's life. On the other, it may provide a protective effect by preserving both brain and cardiac tissue function for short periods of time, despite severe hypoxia (5).

Finally, the potential for electrolyte abberations with near-drowning victims is minimal. As is the case with other unfounded fears concerning near-drowning, reports of severe electrolyte imbalance with drowning arise frm early animal experiments using large volumes of water. In practice, only slight changes in the serum sodium and chloride occur following salt water aspiration, and electrolyte changes following fresh water are usually negligible. In fact, when compared to the profound hypoxemia and metabolic acidosis that most victims experience, these electolyte changes pose no significant management problems (8).

Restoring Vital Physiological Function

The goals of therapy for near-drowning victims basically encompass the restoration of vital physiological function. In particular, this means reversing the hypoxemia and metabolic acidosis, protecting the brain from further insult, and treating

both the pulmonary complications associated with aspiration and hypothermia, when present. But because of the controversies surrounding which patients to resuscitate and the need for lung drainage, one first must be clear on the relative value of these procedures.

RESUSCITATION. The controversy surrounding resuscitation stems from the conflicting conclusions of several series of drowning and near-drowning victims (12,13,18,19). The fear is that resuscitation of near-drowning victims may result in a living but permanently brain-damaged patient (20). In fact, this concern has merit, since many near-drowning victims do survive in a vegetative state. The problem, however, is that there exists no simple way to predict any given patient's outcome during the initial resuscitative period and, in some cases, even with specialized monitoring during the first 24-48 hours (18,19,21). This fact leaves little choice but to employ aggressive resuscitation with all victims.

Unfortunately, the question of draining fluid from the lungs is not so easily answered. On one side of the debate are those who advocate an abdominal thrust (Heimlich maneuver) as the initial resuscitative procedure (22). Theoretically, this clears the lungs of excess fluid and, thus, allows for better gas exchange. On the other side are those who find this maneuver unnecessary and problematic (23,24). In the first place, the lung rapidly absorbs the fluid in patients sustaining

fresh water aspiration, and in those with salt water aspiration,the excess fluid rarely appears to cause significant problems with ventilation. In the second place, an abdominal thrust may cause the forceful expulsion of the gastric contents from the patient's stomach, risking pulmonary acid aspiration and all the terrible consequences that it engenders. For these reasons, most authorities feel that the abdominal thrust maneuver may waste time, and they reserve it solely for patients in whom solid material obstructs the larynx and prevents the establishment of a patent airway.

OXYGENATION is the primary treatment consideration in near-drowning patients.

When recovering a submersion victim who has no spontaneous respirations, the need for immediate ventilation is clear. Anyone who is present should clear the airway of food or foreign objects, support the airway by elevating the jaw and soft tissues, and begin mouth-to-mouth or mouth-to-nose ventilation. In some cases, an oral or nasopharyngeal airway proves necessary to maintain an adequate airway, but it is better to avoid such devices when possible because of the chance that their insertion may precipitate laryngospasm or vomiting (25). As soon as possible, all patients should also receive supplemental oxygen, since even conscious patients may suffer significant pulmonary damage with severe hypoxemia and still be conversant.

METABOLIC ACIDOSIS is partially resolved by treating the patient's hypoxia, but sometimes this does not correct the problem rapidly enough. To correct this problem, use intravenous sodium bicarbonate in patients with a pH of less than 7.2, calculating the amount needed with the following formula: [HCO_3 deficit] = body weight (kg) x 0.2 x [(desired HCO_3) (measured HCO_3)].

CEREBRAL RESUSCITATION. After initiating the above resuscitative procedures and stabilizing patients from a cardiopulmonary and metabolic standpoint, evaluation of neurological status is essential. Patients who are awake and alert and have minimal cardiopulmonary changes may only require observation for 24 hours as indicated above. Alternatively, patients who remain unconscious, but maintain normal pupillary and pain withdrawal responses, need hourly neurological monitoring to detect any further changes or deterioration in function. Those with signs of increased intracranial pressure or severe neurological dysfunction, however, require more definitive therapeutic intervention. Intravenous mannitol (1 g/kg), dexamethasone (80 mg/day) and hyperventilation are all effective in lowering intracranial pressure (to maintain patient's $PaCO_2$ in the range of 30 torr). After instituting these measures, most authorities recommend the placement of intracranial pressure (ICP) and cerebral perfusion pressure (CPP) monitors to gauge the need and effect of further intervention (28,29). Such intervention may include

hypothermia, barbiturate coma, and muscle relaxants. However, the precise benefits of this kind of therapy for near-drowning victims remains unclear. Unfortunately, those patients who develop an ICP greater than 20 mm Hg and a CPP less than 50 mm Hg generally fare poorly despite therapy, and most either die or persist in a vegetative state (29).

PULMONARY COMPLICATIONS. Early oxygenation and ventilation are important in terms of outcome. Once transported to the hospital, manipulation of positive end-expiratory pressure (PEEP), which may require invasive hemodynamic monitoring, can be initiated to improve gas exchange.

HYPOTHERMIA. The treatment for hypothermia is somewhat controversial, but proves necessary in all near-drowning patients with a core temperature below 35°C. However, one should employ different techniques depending on the degree of hypothermia, and they also should be aware of special considerations when managing dysrhythmias in these victims. For patients with mild hypothermia (32°-35°C), passive external warming techniques usually suffice, as long as these victims are able to produce endogenous heat. This method generally involves placing victims at room temperature and covering them with dry, warm insulating blankets. For patients with a core temperature below 32°C, one can institute active external or core rewarming techniques. Active external rewarming techniques include the use of electric blankets, heated objects, such as

hot-water bottles, and, possibly, immersion in warm (40°C) water using a Hubbard tank. When utilizing active external rewarming techniques other than immersion, however, heat the trunk only, since extremity rewarming may cause hypotension due to peripheral vasodilation and also "afterdrop," which may result from the sudden return of cold, hypokalemic, acidotic blood to the core organs. Immersion in warm water proves controversial because of both these latter problems with extremity rewarming and the need for arterial blood gas, blood pressure, pulse, and CVP monitoring, which are cumbersome in this setting. In addition to nonimmersion external techniques, active core rewarming methods can be used. The most practical of these methods encompasses the inhalation of heated (45°C), humidified oxygen via a mask or endotracheal tube and irrigation of the stomach, bladder, and/or peritoneum, using an appropriate solution at a temperature near 42°C.

The treatment of dysrhythmias in hypothermic victims differs considerably from that used in normothermic patients. First, atrial arrhythmias need no therapy, since they generally resolve spontaneously as the patient's core temperature rises. Ventricular fibrillation, on the other hand, presents greater therapeutic problems. In patients with this problem, defibrillation usually proves unsuccessful unless their core temperature is above 30°C. However, resuscitative techniques should be continued until patients have a core temperature in

excess of 35°C. Likewise, the use of some antiarrhythmic agents, including lidocane and procainamide, helps little. In hypothermic patients, lidocaine works poorly and procainamide may actually increase ventricular irritability. As a result, one should consider the use of bretylium (Bretylol) in fibrillating, hypothermic patients at an initial intravenous dose of 5 mg/kg in children or 350-500 mg in adults, given over 8-10 minutes.

PREVENTION. The magnitude of submersion accidents in numbers, as well as in terms of morbidity and mortality, makes prevention a critical social issue. Though improved medical management certainly saves lives, the fact that 91% of pediatric drowning victims are deceased at the scene speaks for the need for a solid program of water and pool safety.

Education is of primary importance in order to implement safety principles whether it involves monitoring and fencing backyard pools, breath-hold diving, SCUBA diving, or any other water sport. Reaction to urgent or emergent situations in the event of an accident is important to preserve life and function.

References

1. Donaldson JC, Royall JD: Drowning and near-drowning. Postgrad Med 64:71, 1968.
2. Clark EB, Niggemann EH: Near-drowning. Heart Lung 4:946, 1975.
3. Rowe MI, Arongo A, Allington G: Profile of pediatric drowning victims in water-oriented society. J Trauma 17:587, 1977.
4. Pleuckhahn VD: Drowning: Community aspects. Med J Aust 2:226, 1979.
5. Hunter TB, Whitehouse WM: Fresh water near-drowning: Radiological aspects. Radiology 112:51, 1974.
6. Miles S: Drowning. Br Med J 3:597, 1968.
7. Conn AW: The near-drowned child. Emerg Med 12:16, 1980.
8. Modell JH, Graves SA, Ketover A: A clinical course of 91 consecutive near-drowning victims. Chest 70:231, 1971.
9. Modell JH, Boysen P: Respiratory crises. Critical Care: State of the Art. Shoemaker WC, Thompson WL (eds), Fullerton, California, Society of Critical Care Medicine, 1980.
10. Giammona ST, Modell JH: Drowning by total immersion: Effects on pulmonary surfactant of distilled water, isotonic saline and sea water. Am J Dis Child 114:612, 1967.
11. Modell JH, Calderwood HW, Ruiz BC, et al: Effects of ventilatory patterns on arterial oxygenation after near-drowning in sea water. Anesthesiology 40:376, 1974.
12. Peorn JH, Bort RD, Yamaok AR: Neurologic sequelae after childhood near-drowning: A total population study from Hawaii. Pediatrics 64:187, 1979.
13. Peterson B: Morbidity of childhood near-drowning. Pediatrics 59:364, 1977.
14. Conn AW, Edmonds JF, Barker GA: Cerebral resuscitation in near-drowning. Ped Clin North Am 26:691, 1979.
15. Siebke H, Breivik H: Survival after 40 minutes' submersion without cerebral sequelae. Lancet 1:1275, 1975.
16. Modell JH, Moya F: Effects of volume of aspirated fluid during chlorinated fresh water drowning. Anesthesiology 27:662, 1966.
17. Modell JH: The Pathophysiology and Treatment of Drowning and Near-drowning. Springfield, Illinois, Charles C Thomas, 1971.

18. Oakes DD, Sherck JP, Maloney JR, Charters AC III: Prognosis and management of victims of near-drowning. J Trauma 22:544, 1982.
19. Dean JM, McComb JG: Intracranial pressure monitoring in severe pediatric near-drowning. Neurosurgery 9:627, 1981.
20. Frates RC Jr: Analysis of predictive factors in the assessment of warm-water near-drowning in children. Am J Dis Child 135:1006, 1981.
21. Modell JH, Graves SA, Kuck EJ: Near-drowning correlation of level of consciousness and survival. Can Anaesth Soc J 27:211, 1980.
22. Heimlich JH: Subdiaphragmatic pressure to expel water from the lungs of drowning persons. Ann Emerg Med 10:476, 1980.23. Wilder RJ, Wedro BC: "Heimlich Maneuver" for near-drowning questioned (letter). Ann Emerg Med 11:111, 1982.
24. Orlowski JP: "Heimlich Maneuver" for near-drowning questioned (letter). Ann Emerg Med 11:111, 1982.
25. Modell JH: Drowning and near-drowning. Disorders of the Respiratory Tract in Children. Kendig EL, Churnick D (eds), Philadelphia, WB Saunders, 1983.
26. Gong H Jr: Toward a better understnading of ARDS. Fam Med Reports 1:79, 1983.
27. Boysen PG: Individualizing mechanical ventilation to patient requirements. ER Reports 2:63, 1981.
28. Pfenninger J, Sutter M: Intensive care after fresh water immersion accidents in children. Anaesthesia 37:1157, 1982.
29. Nussbaum E, Galant SP: Intracranial pressure monitoring as a guide to prognosis in the nearly drowned, severely comatose child. J Pediatrics 102:215, 1983.
30. Petty TL, Fowler AA: Another look at ARDS. Chest 82:101, 1981.
31. Nicholson DP: Corticosteroids in the treatment of septic shock and the adult respiratory distress syndrome. Med Clin North Am 67:717, 1983.
32. Sibbald WJ, Anderson RR, Reid B, et al: Alveolar-capillary permeability in human septic ARDS. Chest 79:138, 1981.
33. Wolfe JE, Bone RC, Ruth WE: Effects of corticosteroids in the treatment of patients with gastric aspiration. Am J Med 63:719, 1977.
34. Calderwood HW, Modell JH, Ruiz BC: The ineffectiveness of steroid therapy for treatment of fresh water near-drowning. Anesthesiology 43:642, 1975.

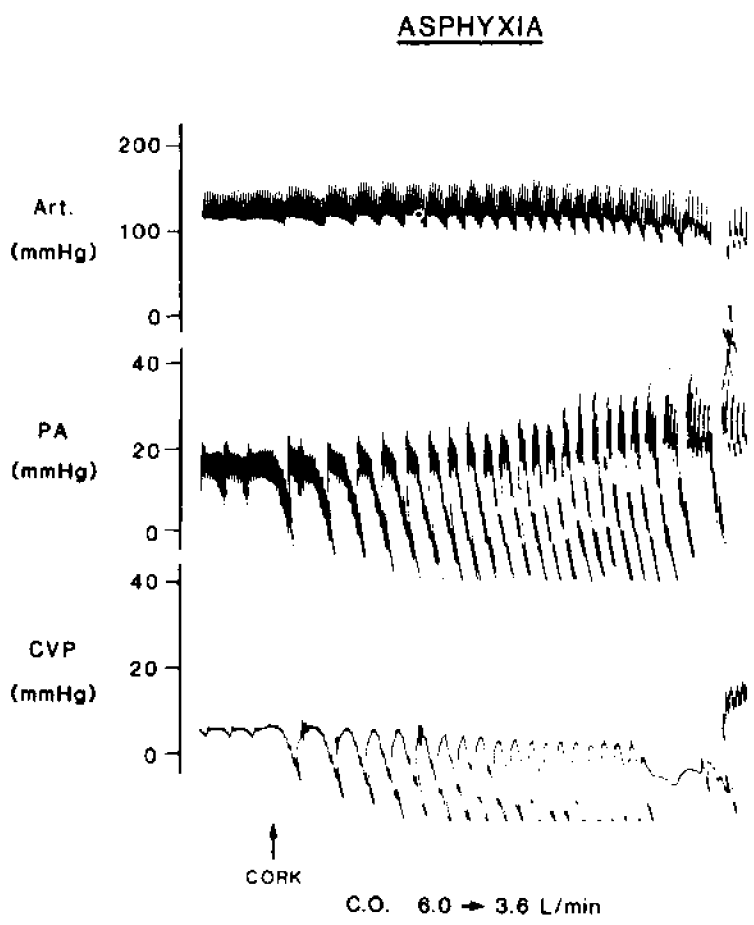

Fig. 1a

ASPHYXIA: HEMODYNAMICS

Arterial (Art) pulmonary artery (PA) and central venous pressure (CVP) tracings during a brief period of upper airway occlusion and asphyxia. Calculated systemic and pulmonary vascular resistance increases. Note negative pressure swings in the PA and CVP tracing with continued inspiratory effort. The hemodynamic changes, including the fall in cardiac output are reversed with relief of hypoxemia.

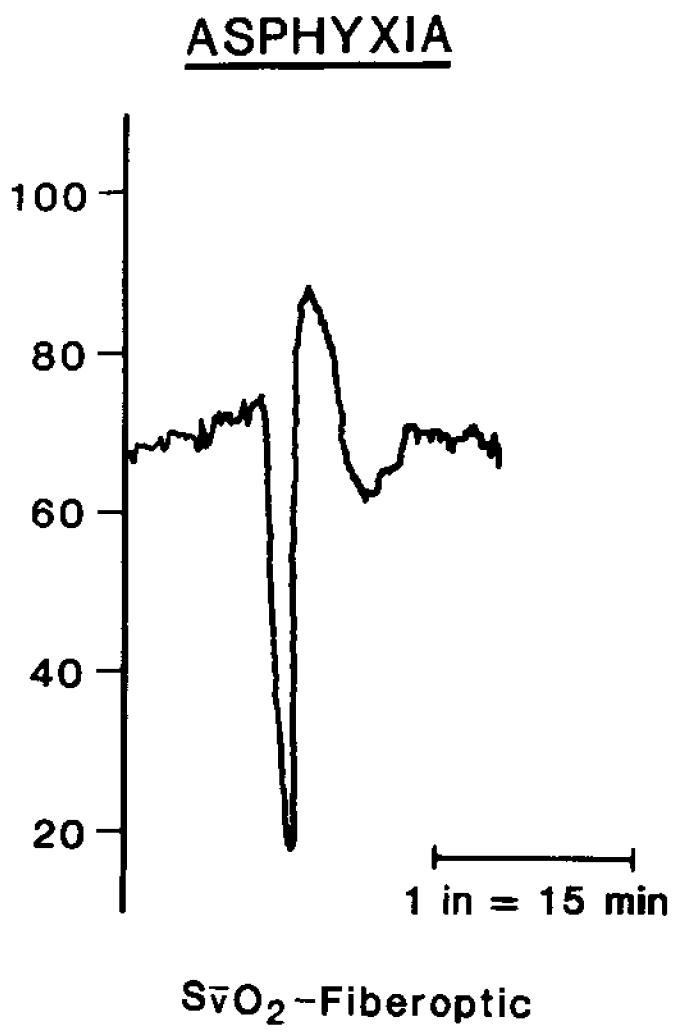

Fig. 1b

ASPHYXIA

An indwelling pulmonary artery catheter equipped with a fiberoptic system to measure SvO_2 continuously shows marked mixed venous oxygen desaturation during upper airway obstruction and continued inspiratory effort. With removal of the obstruction the SvO_2 immediately improves. The overshoot implies not only improved gas exchange but also a temporary augmentation of cardiac output.

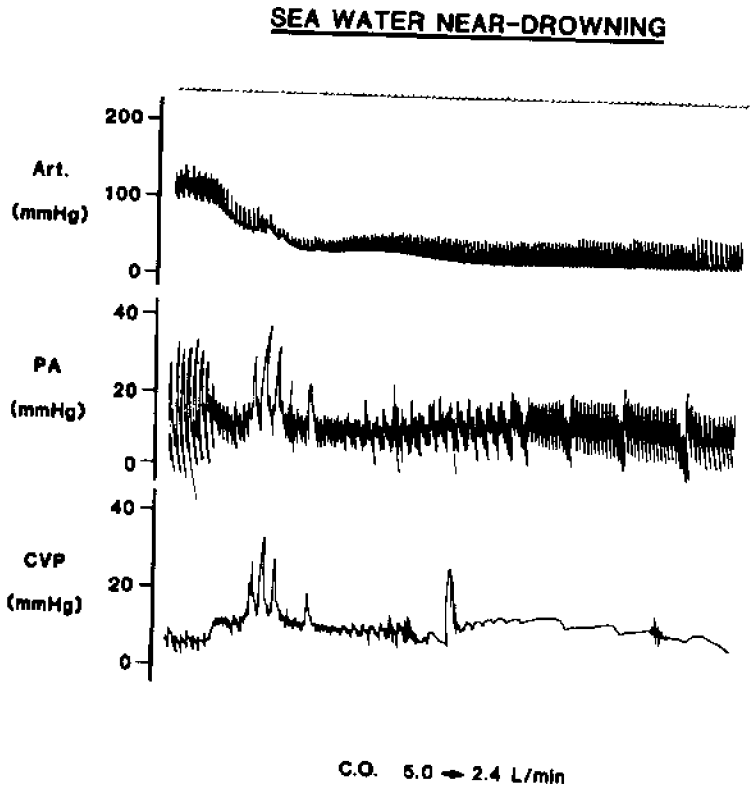

Fig. 2a

SEA WATER NEAR-DROWNING: HEMODYNAMICS

Arterial (Art) pulmonary artery (PA), and central venous pressure (CVP) tracings after aspiration of salt water. There is immediate and sustained arterial hypotension, and an eventual rise in PAP and CVP. Changes are due to lung damage and movement of intravascular fluid into lung parenchyma in response to aspiration of hyperosmotic fluid.

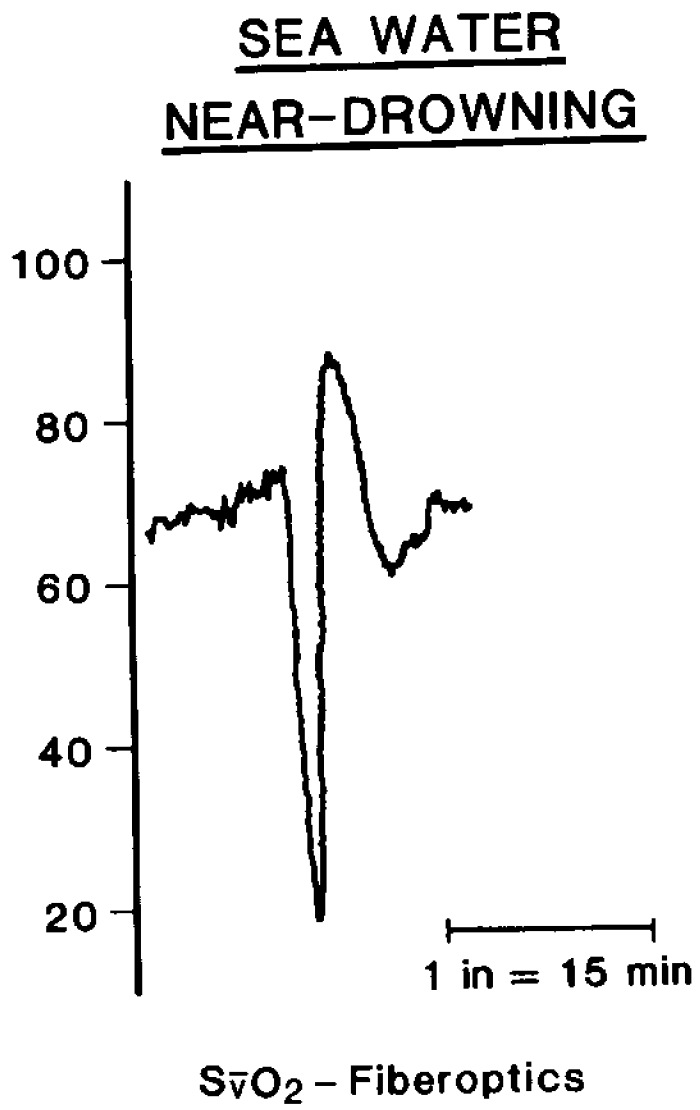

Fig. 2b

SEA WATER NEAR-DROWNING

Continuous recording of SvO_2 following sea water aspiration (22 cc/kg) in a canine model. Following a fall in SvO_2 to less than 20% ventilation was begun with a bag valve system and 100% O_2. Note the similarity in the shape of the tracing to asphyxia.

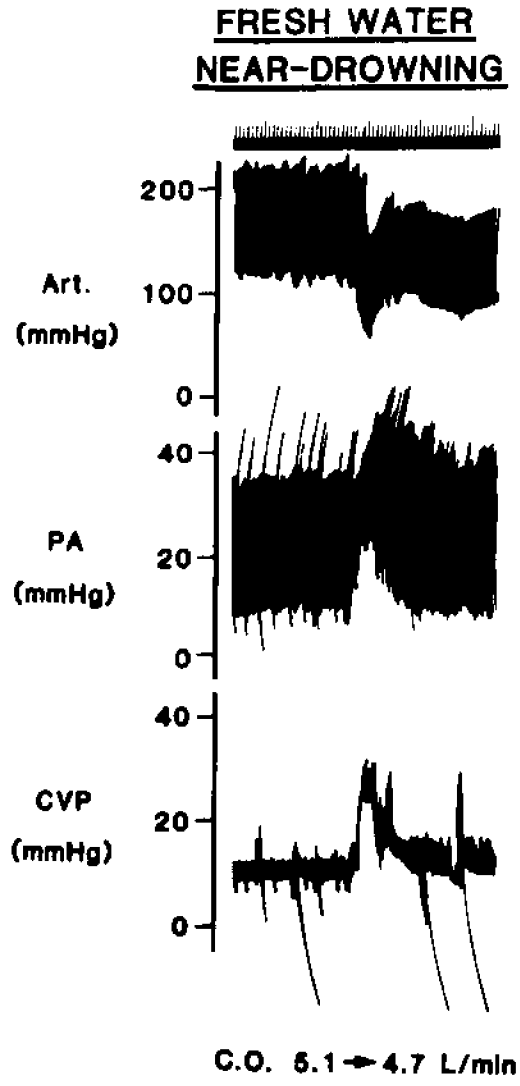

Fig. 3a

FRESH WATER NEAR-DROWNING: HEMODYNAMICS

Arterial (Art), pulmonary artery (PA), and central venous pressure (CVP) tracings after aspiration of fresh water. Arterial hypotension and pulmonary artery and central venous hypertension. After a brief episode of apnea all three variables stabilize. There is a minimal fall in cardiac output because of movement of hypotonic fluid from the lung into the intravascular space.

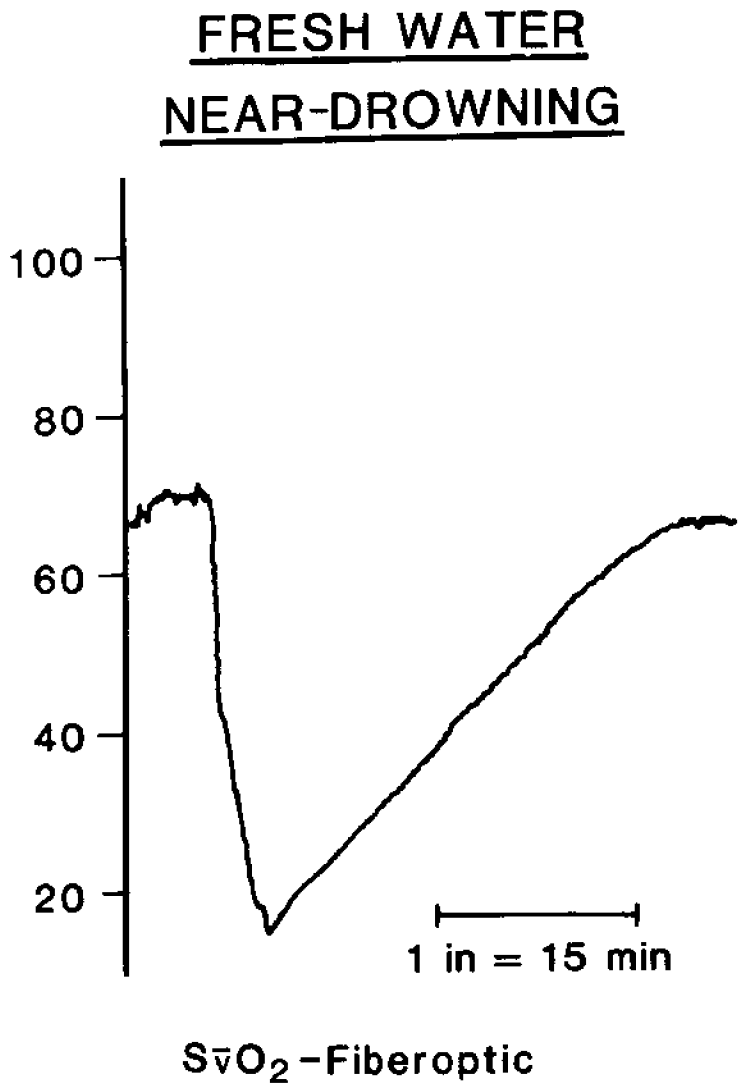

Fig. 3b

FRESH WATER NEAR-DROWNING

Continuous SvO_2 recording following aspiration of fresh water (22 cc/kg) in a canine model. At the nadir of the tracing (SvO_2 < 20%) ventilation with a bag-valve system and 100% O_2 was begun. Despite a minimal fall in cardiac output (see Fig. 3a) recovery takes approximately 30 minutes. Thus disruption of gas exchange mechanisms is more severe.

Discussion following Dr. Boysen's presentation

Dr. Norfleet: Did you observe any seizure activity in your experimental model, and, if so, was it related to the type of insult?

Dr. Boysen: We didn't observe any seizure activity. That question has come up before, we may have masked it; these dogs were anesthesized.

Dr. Norfleet: What was the anesthesia?

Dr. Boysen: Pentobarbital, intravenous. So that may have influenced that.

Dr. Siesjo: I was wondering about the blood pressure at the end of the asphyxia.

Dr. Boysen: The blood pressure depends on the length of the asphyxia.

Dr. Siesjo: Yes. If you have a long period of asphyxia, yet short enough not to interfere with adequate resuscitation, what would the blood pressure be?

Dr. Boysen: The blood pressure is still normal or elevated. Once the blood pressure starts to go down in the asphyxiant, usually you don't get much in the way of recovery. It just starts to go down before ten minutes. The asphyxia time, and I'm sure it's the same for patients, tends to be about four to six minutes. Now the issue is when we intervene, it's typical when patients are pulled from the water, the paramedic will find that they have a laryngospasm. You can break a laryngospasm with continuous positive airway pressure, but in general air cannot be insufflated with a bag-mask valve system. And so the point is that, once they relax, it's important to insufflate the airway and intubate the patient almost immediately.

Dr. Siesjo: I was just wondering if you can clarify whether or not the cardiac reaction to the asphyxia is related to what type of anesthesia you use.

Dr. Boysen: I haven't done it with a different anesthetic, I can't answer that.

Dr. Elsner: Could I bring up a question which may have critical bearing here? Some years ago, when we were doing studies of the neural control mechanisms in the diving response of the seal, we were very much impressed by the power of the reflex of lung inflation and stimulation of pulmonary stretch receptors in producing a tachycardia. So it might be that a by-product of CPR and lung inflation is to restart or to stimulate the heart as well.

Dr. Boysen: Well, I think that is absolutely true. Although if you look at the patient histories, of most of the patients that are pulled from the water and survive, there is not a great incidence, or at least recorded incidence, of arrhythmias which I might expect if there is some stimulant effect.

Dr. Elsner: Yes, but they do likely respond to the lung inflation of CPR with reversing of cardiac arrest.

Dr. Boysen: That would be hard to know because I think the major thing is reversal of hypoxia, and cardiac function in our experience tends to improve immediately once you relieve the hypoxia, so I don't know if we can separate the two.

HYPERVENTILATION : AN UNPREDICTABLE DANGER TO THE SPORTS DIVER

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INTRODUCTION

Under conditions of normal atmospheric pressure, breath-holding results in important changes in the mechanism whereby CO_2 is transported and exchanged in the lungs, but a reversal of the CO_2 gradient from the lungs to the blood does not occur unless the inspired air contains a high concentration of CO_2 (Lanphier & Rahn 1963).

Under conditions of raised atmospheric pressure, as occurs in breath-hold diving, the whole aspect of CO_2 transport and exchange is markedly altered and cannot be equated with apnoea at normal atmospheric pressure (Lanphier & Rahn 1963). Prolonged apnoea under raised atmospheric pressure, results in a reversal of CO_2 exchange from the lungs to the blood (Lanphier & Rahn 1963). During apnoea a shrinkage of lung volume occurs, suggesting that CO_2 output decreases while O_2 uptake is continuous (Craig 1961). Arterial PCO_2 will increase and exceed venous PCO_2 during apnoea, as a result of the oxygenation of Hb in the presence of falling CO_2 output to the lungs (Haldane effect). Schaefer (1975) showed the flow of CO_2 into the lungs during ascent from a dive is regulated in part by the speed of ascent. Mithoefer (1959) offered an explanation for the "aberrations in CO_2 transport and exchange" which occur during apnoea with O_2.

Craig (1961) showed the unconsciousness following hyperventilation and breath-hold diving, was probably caused by hypoxia.

In 1963, Lanphier, working with four divers under simulated breath-hold diving conditions in a compression chamber, established the course of alveolar gas exchange under pressure and also determined from the nomogramme of Dill the time course of mixed venous CO_2 content during simulated dives. He worked in a pressure chamber in order to determine accurately the pressure-time relationships and to facilitate the taking of alveolar gas samples. He concluded that hypercapnia (CO_2 narcosis) may cause unconsciousness in prolonged, deep breath-hold dives. Hong (1963) in a field study of the Korean diving women (Ama), noted that they did not hyperventilate to any great extent and followed the relatively safe pattern of the 60 second dive to 10 meters in which neither hypoxia or CO_2 accumulaton is a serious hazard.

Smit (1967, 74, 81) determined the alveolar CO_2 % and $=O_2$ % after breath-hold dives in a swimming bath and diving tank, but no blood CO_2 levels were determined. Black-outs occurred when the P_AO_2 dropped to below 4,5 kPa, (33 - 34 mmHg). In divers whose exhaled values came close to this figure but were not below 4,5 kPa, hypoxic black-outs did not occur (e.g. at 4,8 and 5,3 kPa). The probable limit of hypoxic stress during apnoea may be at a P_AO_2 of 4,0 - 4,5 kPa, which, when transformed, will be close to the critical arterial oxygen pressure (P_aO_2) of 4 kPa (30 mmHg) associated with

loss of consciousness.

An early preliminary experiment on 5 experienced breath-hold divers indicated the possibility that the effects of hyperventilation on the CO_2 content of peripheral venous blood may be unpredictable in different divers under the same diving conditions and also in the same diver from day to day (Landsberg 1971).

More recently it was shown that the partial pressure of CO_2 in arterialised peripheral venous blood, during the hyperventilation breath-hold-dive cycle under a steady state condition, is unpredictable and that hypoxia (P_AO_2 = 4 - 6 kPa) superimposed on hypocarbia ($PvCO_2$ = 4 - 5 kPa) may enhance loss of consciousness underwater (Landsberg 1982,1984).

Unpredictable, wide differences in the alveolar PCO_2 of SCUBA divers in the sea at 10 m and 30 m have also been found (Dwyer & Pilmanis 1975). Unpredictably the highest alveolar PCO_2 of 8,9 kPa (68 mmHg) was found at the shallowest depth in the most experienced diver. CO_2 retention was not consistent at any workload or depth. Dwyer found 3 groups of SCUBA divers; those that eliminate CO_2 normally, those that retain CO_2 and those that hyperventilate and eliminate excessive amounts CO_2. He also found that divers may shift from one group to another in a manner not related to workload or depth, as well as a wide individual variation.

Morrison et al., (1978), investigating loss of consciousness in 2 SCUBA divers, noted a low respiratory response to inspired CO_2 in these divers: i.e. insensitivity to CO_2 must have been inherent in the divers. They inferred from their experimental results that loss of consciousness was associated with hypercapnia, although hypercapnia alone could not have caused unconsciousness. In their experiments loss of consciousness was not the direct result of hypoxia. They quote Case & Haldane (1941) who observed that divers lost consciousness at very different partial pressures of CO_2, which indicates a large range of individual susceptibility.

Several physiological mechanisms can potentially cause unconsciousness underwater during breath-hold and SCUBA diving:- hypoxia (Craig 1961), hypercapnia - CO_2 narcosis - (Lanphier & Rahn 1963; Paulev 1969), various combinations of hypoxia with hyper- or hypocapnia or even a normal C_a blood tension with hypocapnia (Rahn, Otis & Hodge, 1946; Dhenin et al., 1978), cardiac arrhythmias, ventricular fibrillation (Sem Jacobson & Styri 1970, Brown 1974), bradycardia (Landsberg 1972, Bove et al., 1973).

Brown (1974) maintains that "shallow water blackout" has been confusing the diving community long enough and that underwater blackout is an entity with a number of possible causes, which he classifies.

Wolf (1970), has postulated the mechanism of sudden death during submergence as follows: the stimulus is anticipation or fear of diving or forced immersion which can cause extrasystoles; the response during submergence will be the oxygen conserving "diving reflex" consisting of bradycardia initiated by parasympathetic vagal activity; blood pressure will decrease, but peripheral vasoconstriction brought about by sympathetic

activity, will maintain blood pressure; skin, muscle and peripheral blood flow will decrease with lactic acid and CO_2 accumulation causing metabolic acidosis with low pH, high K levels and hypoxia; all these factors combined will cause extreme bradycardia, extrasystoles, arrhythmias, ventricular fibrillation and cardiac arrest. Anderson and Blix (1974) have confirmed this mechanism in ducks with alpha and neuronal adrenergic blockade experiments. They have demonstrated the pharmacological components in the autonomic control of the diving reflex.

UNPREDICTABLE EFFECTS OF HYPERVENTILATION (LANDSBERG, 1984)

This study was initiated by the drowning of an expert and world class South African spearfisherman on the 9th March, 1968. What factors contributed to the tragic and unnecessary death of a superbly fit and very experienced diver? Spearfishermen admit they hyperventilate, otherwise their deep diving in search of fish becomes physiologically impossible. Hyperventilation gives them 10 - 20 seconds extra "bottom time", as shown by Lanphier & Rahn (1963), but increases the danger of hypoxic black-out (Craig 1961). They appear to understand the dangers of hypoxic "black-out" on ascent. They control their hyperventilation prior to breath-hold diving and appear to heed the physiological alarms set up in their bodies urging them to surface to breathe. In spite of this, several have arrived on the surface unconscious, automatically breathing a mixture of water and air through their snorkels until they overcome their hypoxia and regain consciousness, coughing, cyanosed and bewildered, yet continuing to dive after a period of rest. Some "dive on their watches" taking 75 seconds to dive $\pm$ 20 m (surface to surface). They have worked out their individual dive times by experience with previous hyperventilation and near black-outs. The effects of hyperventilation in divers are unpredicable as we cannot predict a high or low level of CO_2 at any point in the hyperventilation-breath-hold-dive cycle. It is therefore very difficult for an experienced diver who has been hyperventilating prior to breath-hold diving for many years, to be dogmatic about his ability to heed the physiological alarms that he may experience during a dive. One day he may have a perfect CO_2 accumulation and stimulation of his respiratory centre with rapid diffusion of CO_2 from blood to alveolar gas taking place long before hypoxia causes unconsciousness. The next day CO_2 accumulation may be slow due to excessive hyperventilation, or CO_2 diffusion into the alveolar gas may be slow due to rapid ascent causing excessive CO_2 accumulation in the blood, producing CO_2 narcosis - hypercapnia (Paulev 1969). Hypoxia, which occurs due to the breath-holding and underwater exercise combined with CO_2 narcosis will cause unconsciousness and drowning. Another factor involved is the over-riding competitive spirit that prevails at spearfishing competitions, which causes the diver to wilfully shut off his physiologically protective alarm systems (Craig 1961, Deppe 1971).

THE PROBABLE CAUSES OF UNCONSCIOUSNESS DURING THE HYPERVENTILATION-BREATH-HOLD-DIVE CYCLE (H-B-H-D-CYCLE)

1. During descent

On the surface a diver may have a high level of CO_2 in his lungs

after a strenuous swim. If he does not hyperventilate to blow off the excess CO_2 and decided to dive to 10 m the blood CO_2 tension may rise above 6 kPa (46 mmHg) reaching levels of 6,5 - 9,2 kPa (50 -70 mmHg) the range of "CO_2 narcosis" (Lanphier & Rahn 1963, Paulev 1969). This high level of CO_2 may cause unconsciousness on descent. This may also occur if the diver decides to dive deeper than 10 m after exercising at that level. It may also occur if the diver does not allow an adequate rest period during repeated hyperventilation-breath-hold-dive cycles (Schaefer 1975).

2. On the bottom

Unconsciousness is due to prolonged submergence (more than 80 seconds) which causes hypoxia and CO_2 narcosis. This occurs due to the diver wilfully ignoring his desire to breathe (Craig 1961, Deppe 1971).

3. On ascent

Unconsciousness is caused by the drop in the arterial O_2 tension producing hypoxia. A very rapid ascent may also cause CO_2 accumulation in the blood, and possibly CO_2 narcosis, by not allowing sufficient time for the CO_2 to enter the alveolar gas (Schaefer 1975) However, the P_ACO_2 decreases because of air expansion in lungs on ascent. Therefore this cannot block CO_2 diffusion into the alveoli (Guyton 1982).

DISCUSSION

An analysis of the dive patterns found in 30 apnoeic divers in the compression chamber (Landsberg 1984) show various unpredictable blood and alveolar gas tensions all potentially capable of causing near loss of consciousness (near black-out) at different breaking points. Some blood and alveolar gas tension combinations will be more dangerous than others.

1. Because of the relationship between P_ACO_2 and the $PvCO_2$ in the arterialised peripheral venous blood of divers, it may be concluded that in general the arterial bood CO_2 levels in trained divers are reduced by hyperventilation and increased by repeated breath-hold-diving.

2. This is not true in all cases as divers show a variation in the arterialised $PvCO_2$ (Landsberg 1984). This may possibly explain the unpredictable nature of the effects of hyperventilation in divers.

3. The alveolar O_2 partial pressure, P_AO_2, increases after hyperventilation. This is due to the loss of CO_2 from the alveolar gas volume during hyperventilation and the partial pressure space left by the exhaled CO_2 is taken up rapidly by O_2 (Green 1978). In one diver, at the end of 100 s breath-hold-dive, the P_AO_2 is 4,5 kPa, the accepted lower limit of normal maintenance of consciousness (Smit & Giesel 1981).

4. When considering the mean values of $PvCO_2$ kPa±SD:- The resting $PvCO_2$ = 4,09 kPa in this group of 30 divers. This is a low figure because of apprehension in the divers with hyperventilation and acclimatization to an altitude of 4 000 - 5 000 ft in Pretoria, South Africa.

ACCLIMATIZATION TO ALTITUDE. When, after an acute exposure to altitude, the steady state has been reached (½ - 1 hour), then the alveolar gas values must come to rest somewhere along the R =,8 diagonal of that particular altitude. In figure 1, the upper curve represents the alveolar values found at various altitudes after the steady state is reached. If man now remains longer at these altitudes, he very slowly increases his ventilation as he becomes acclimatized. Thus the PCO_2 is lowered over several days, and the alveolar point progresses down along the R =,8 diagonal with proportional increase in alveolar PO_2 until the acclimatization curve of Figure 1 is reached (Rahn & Fenn 1960).

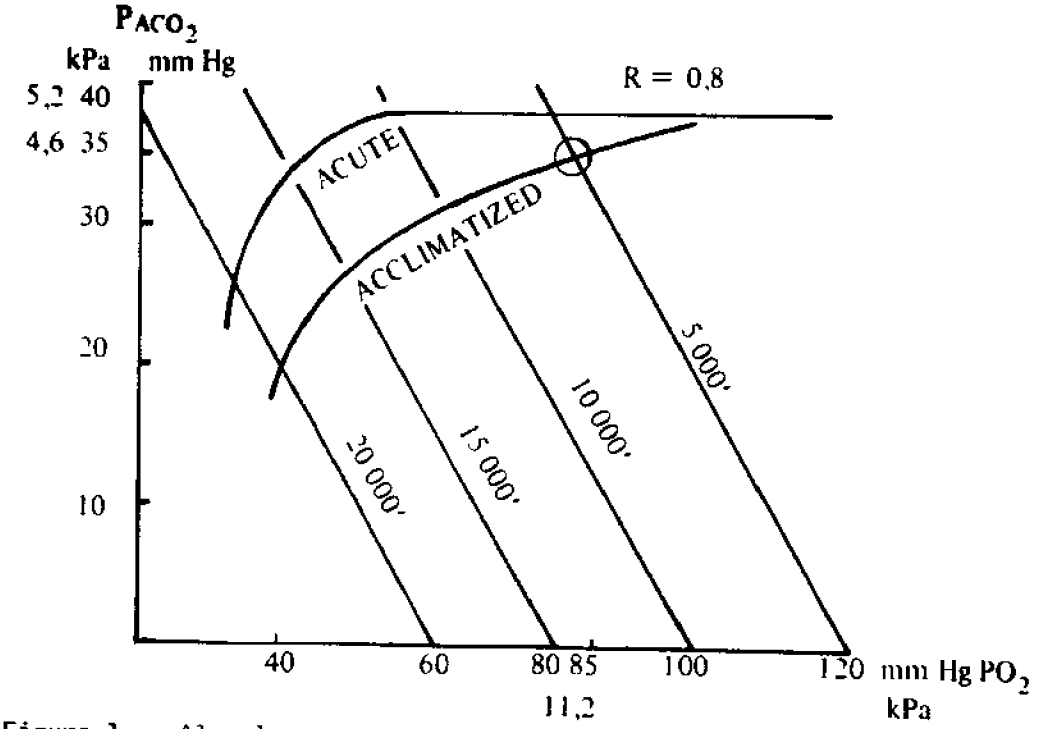


Figure 1 Alveolar gas concentrations at various altitudes during acute exposure and after acclimatization. The altitude diagonals are drawn for a R =,8. O = alveolar point at Pretoria: PO_2 = 11,2 kPa (85 mmHg) PCO_2 = 4,6 kPa (35 mmHg).

5. This decreases to $PvCO_2$ = 2,67 kPa (20 mmHg) after hyperventilation.

6. This increases to $PvCO_2$ =6,28 kPa after hyperventilation and breath-hold-diving.

FLUCTUATIONS IN $PvCO_2$ OF ARTERIALISED PERIPHERAL VENOUS BLOOD DURING HYPERVENTILATION AND BREATH-HOLD-DIVING IN THE SAME DIVERS ON DIFFERENT OCCASIONS (LANDSBERG 1984).

A comparison of 30 apnoeic divers shows that these divers had different $PvCO_2$ values during the hyperventilation breath-hold-dives, showing that it is impossible to predict the PCO_2 changes that take place during a breath-hold-dive. The factors responsible for this are:-

1. The different tidal volumes in divers (Miles 1971).
2. The variable rates of CO_2 diffusion in and out of the lungs (Donald 1971).
3. The physical exertion of hyperventilation (Hong et al., 1963, Schaefer & Carey 1962).
4. The amount of CO_2 produced by the exercise of hyperventialtion which is added to the CO_2 produced during the dive (Schaefer 1975).
5. The activity of the diver while submerged; this may be considerable in a spearfisherman and an underwater hockey player.
6. The time that a diver is submerged; this may be up to 90 seconds (Lanphier & Rahn 1963, Craig 1961), and 158 seconds (Landsberg 1984).
7. The rate of ascent (Schaefer 1975).
8. The length of recovery period on the surface before the next dive; adequate time must be allowed to blow off the accumulated CO_2 from a large reservoir (Schaefer 1975).
9. The diver may wilfully "shut-off" or ignore the physiological alarm set up by the high blood CO_2 stimulating the respiratory centre and urging him to breathe (Craig 1961, Deppe 1971).

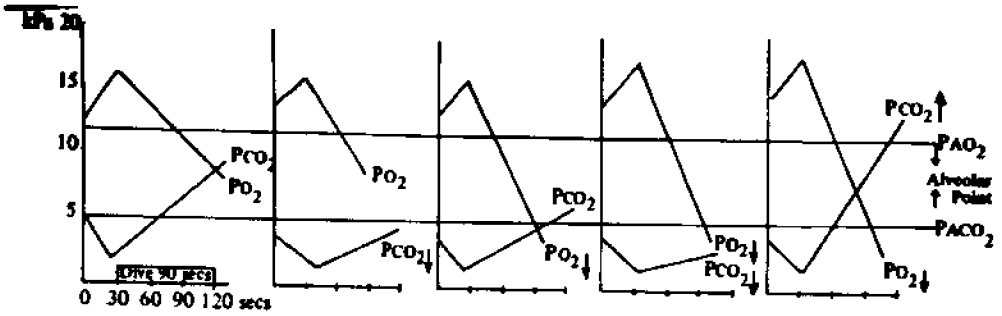
Loss of consciousness. There is a close relationship in hypoxia between the oxygen tension of the cerebral venous blood and the level of consciousness. Consciousness is lost when the jugular venous oxygen tension is reduced to 2,23 - 2,50 kPa (17 - 19 mmHg). The corresponding arterial oxygen tension varies with the cerebral blood flow which depends in turn on the arterial tensions of oxygen and carbon dioxide.

The arterial oxygen tension which produces a cerebral venous tension of 2,32 - 2,50 kPa (17 - 19 mmHg) and unconsciousness, can lie between 2,63 - 4,60 kPa (20 and 35 mmHg) depending on the degree of hypocapnia. Whilst, on average, a man becomes unconscious when the alveolar oxygen tension is reduced to 4 kPa (30 mmHg) or below for a significant period of time, he may lose consciousness at an alveolar oxygen tension as high as 5,26 kPa (40 mmHg) if there is marked hyperventilation, and retain consciousness at an alveolar oxygen tension as low as 3,28 kPa (25 mmHg) if there is no hypo-capnia (Dhenin et al., 1978).

Using these physiological facts as a basis and integrating the object of this study - to show that during the hyperventilation-breath-hold-dive cycle, the human diver's brain is exposed to wilfully imposed blood gas changes which, depending on their particular, unpredictable summation and combination, will cause loss of consciousness (black-out), near drowning and death if immediate rescue precautions are not instituted - an analysis of the dive patterns found in 30 apnoeic divers show various unpredictable blood and alveolar gas tensions all potentially capable of causing near loss of consciousness (near black-out) at different apnoeic breaking points. Some blood and alveolar gas tension combinations will be more dangerous than others.

These points illustrate why hyperventilation, prior to breath-hold diving, to increase the period of apnoea underwater, is a physiologically dangerous practice. They also explain why some divers and not others are adversely affected by hyperventilation, and that the effects of hyperventilation are unpredictable at any time during the hyperventilation-breath-hold dive cycle. Some divers show a marked rise in PCO_2 after apnoea. This is probably due to the accumulation of metabolic CO_2 and the reverse CO_2 gradient that occurs during underwater breath-hold diving (Lanphier & Rahn 1963). Other divers show a drop in PCO_2. This paradoxical result may be explained by variable alveolar CO_2 accumulation rates found in different divers on ascent after a breath-hold dive (Schaefer 1975), depending on the rate of ascent, e.g. fast ascent - low alveolar CO_2 high blood CO_2. The last group may have a rapid diffusion of CO_2 from blood to alveolar gas, e.g. slow ascent - high alveolar CO_2, low blood CO_2, and may also not have produced large amounts of metabolic CO_2 during submergence.

Five groups of O_2: CO_2 combinations are possible during hyperventilation breath-hold dives. These five groups are shown on the O_2: CO_2 diagram in fig. 2 (Rahn & Fenn 1960) on page 8. A diver may develop any one of the five combinations during a dive. The most dangerous combinations are **HYPOXIA + HYPOCAPNIA** and **HYPOXIA + HYPERCAPNIA** as shown below. (1 to 30 refer to 30 apnoeic divers : LANDSBERG 1984).



SAFE	HYPOCAPNIA	HYPOXIA	HYPOXIA HYPOCAPNIA	HYPOXIA HYPERCAPNIA
8 14 16 23 24 26 27	13 15 18 20 22	17 21 28 29	Near Black-out 5 —— 1 6 —— 1 10 11 —— 1	1 2 BLACK-OUT 3 —— 1 4 —— 1 7 9 Near black-out 12 —— 1 19 25 30
TOTALS 7	5	4	4	10

PARTIAL PRESSURE OF O_2 AT AN ALTITUDE OF 86 kPa. The barometric pressure at Pretoria, altitude 4000 - 5000 ft. is P_B = 86 kPa. (656 - 653 mmHg). Water vapour pressure at BTPS = 6,2 kPa (47 mmHg). The inspired O_2 pressure (PIO_2) is calculated by multiplying $P_B - H_2O$ with the fractional concentration of O_2 (FO_2) in the atmospheric air 20,9%.

$$PIO_2 = FO_2(P-H_2O)kPa = \frac{20,9}{100}(86 - 6,2) = 0,209\ (79,8) = 16,6\ kPa\ (126\ mmHg)$$

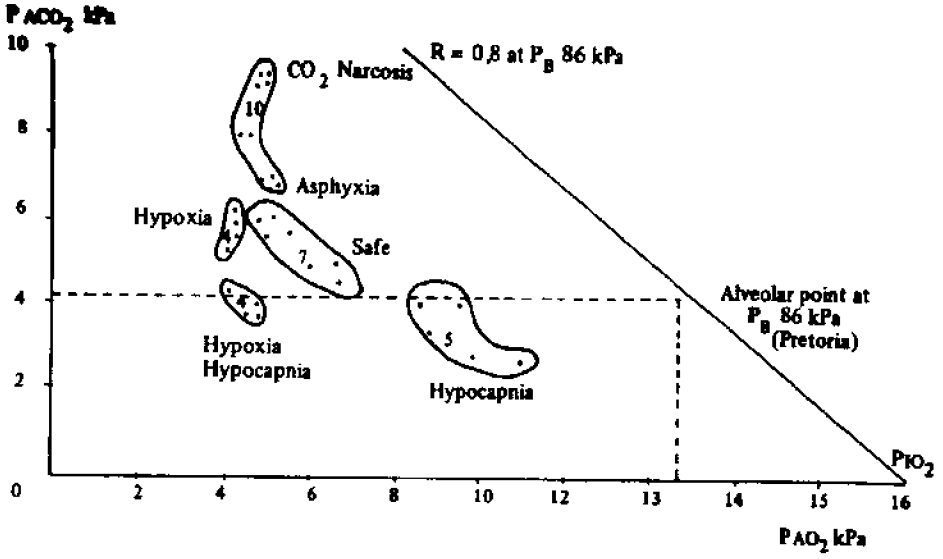


Figure 2 The post dive P_AO_2 and P_ACO_2 values for 30 divers, plotted on the O_2 : CO_2 diagram (Rahn & Fenn, 1960).
Data for plotting the diagram taken from (Landsberg 1984).
P_B = 86 kPa (altitude 4 000 - 5 000 ft)
PIO_2 = 16 kPa, PAO_2 = 13,7 kPa, $PACO_2$ = 4,09 kPa, R = 0,8

POST DIVE PO_2 : PCO_2 COMBINATIONS ON THE O_2 : CO_2 DIAGRAM

GROUP	N	NEAR-BLACKOUT	BLACKOUT
1 SAFE	7	0	0
2 HYPOCAPNIA	5	0	0
3 HYPOXIA	4	0	0
4 HYPOXIA + HYPO-CAPNIA	4	3	0
5 HYPOXIA + HYPER-CAPNIA (Asphyxia, CO_2 narcosis)	10	3	2

CONCLUSIONS

To prohibit hyperventilation during breath-hold diving, is not a successful safety measure as Lanphier & Rahn (1963) have shown that this practice gives a diver an extra 10 to 20 seconds diving time. A strict buddy system and ensuring that the divers are not negatively buoyant will be more effective safety measures. However, the unpredictable nature of the effects of hyperventilation should be explained to the divers who should be encouraged to work out their own individual safe dive profiles using a depth gauge and watch. SCUBA divers should be made aware of the adverse, unpredictable effects of hyperventilation and breath-holding following exhaustion and anxiety while swimming underwater (Dwyer & Pilmanis 1975).

The CO_2 changes in the blood of breath-hold divers during the hyperventilation-breath-hold-dive cycle are unpredictable, show individual variations and vary from day to day (Landsberg 1971, 1982, 1984).

Similar unpredictable wide differences in the alveolar PCO_2 of SCUBA divers in the sea at 10,20 and 30 m have also been found (Dwyer & Pilmanis 1975).

Morrison et al., (1978), investigating loss of consciousness in 2 SCUBA divers, noted a low repiratory response to inspired CO_2 in these divers, i.e. insensitivity to CO_2 must have been inherent in the divers.

In the selection and training of divers (both breath-hold and SCUBA) the following should be added to the medical screening process:-

1. Alveolar CO_2 and O_2 responses during hyperventilation breath-hold-dive cycles (Lanphier & Rahn 1963, Craig 1961, Hong 1963 Landsberg 1982, 1984.)
2. Respiratory response to inspired CO_2 (Morrison et al., 1981).
3. Assessment of psychological motivation (Deppe 1971).

REFERENCES

ANDERSON, H T. & BLIX, A.S. (1974): Pharmacological exposure of the components in the autonomic control of the diving reflex. Acta. Physiol. Scand. 90,381.

BOVE, A.A., PIERCE, A.L., BARRERA, F., et al (1973): Diving bradycardia as a factor in underwater blackout. Aerospace med. 44 : 3,245 - 248.

BROWN, C.V. (1974) : Shallow water blackout. Pressure, 3 : 1,5,IN: Undersea Biomed. Research, 1 : 3.

CRAIG, A.B. Jr., (1961) : Causes of loss of consciousness during underwater swimming. J. Appl. Physiol., 16 : 4,583 - 586.

DEPPE, A.H.(1971) : Psychological factors relevant to the selection of SCUBA divers. D. Litt. et. Phil., thesis, University of South Africa, 44.

DHENIN, G.,SHARP, G.R. and **ERNSTINE, J. (1978)** : Aviation Medicine I : Physiology and Human Factors. P.77 - 82. Trimed Books Ltd., London.

DONALD, K.W. (June, 1971) : Personal communication. Professor of medicine, The Royal Infirmary, Edinburgh, Scotland.

DWYER, H.J. & PILMANIS, A.A. (1975) : Physiological studies of divers working at depths to 99 ft in the open sea. IN : Proceedings of the Sixth symposium on underwater physiology, Federation of American societies for experimental biology, 9659 Rockville Pike, Bethesda, Maryland 20014, U.S.A.

GREEN, J.H. (1978) : An Introduction to Human Physiology, 4th (S.I.) Edition, Oxford University Press. 78 - 80.

GUYTON, A.C. (1982) : Professor, Department of Physiology University of Mississippi. Personal Communication.
HONG, S.K., RAHN, H., & KANG, D.H. et al., (1963) : Diving pattern, lung volumes and alveolar gas of the Korean diving women (Ama). J. Appl.Physiol. 18 : 457-465.
LANDSBERG, P.G. (1971) : CO_2 changes in hyperventilation and breath-hold diving. IN: Abstracts of the first scientific congress of the Physiological and Biochemical Society of Southern Africa, University of the Witwatersrand.
LANDSBERG, P.G. (1972) : Bradycardia during human diving, IN : Abstracts, Science week '72, University of the Witwatersrand. 43.
LANDSBERG, P.G.(1982) : Alveolar Gas and Peripheral Venous Blood Changes During Breath-Hold-Diving. S.Afr. Med.J.61.
LANDSBERG,P.G.(1984): Hyperventilation: An unpredictable danger to the sports diver. Thesis: Dept. of Physiology University of Pretoria,South Africa.
LANPHIER,E.H. & RAHN,H.(1963): Alveolar gas exchange in breath-hold diving. J. Appl. Physiol 18 : 3, 471 - 477.
LANPHIER,E.H. & RAHN,H.(1963): Alveolar gas exchange during breath-holding with air. J. Appl. Physiol. 18 : 3, 478 - 482.
MITHOEFER, J.C. (1959) : Mechanism of pulmonary gas exchange and CO_2 transport during breath-holding. J. Appl. Physiol, 14 : 5, 706 - 710.
MORRISON, J.B. et al., (1978): Observations after loss of consciousness under water. Undersea Biomed. Res. 5(2) : 179 - 187.
MORRISON, J.B. et al.,(1981) : Effects of CO_2 Intensitivity and Respiratory Pattern on Respiration in Divers. Undersea Biomed Res. 8(4):209-217.
MILES, S (July 1971) : Personal communication. Dean of post graduate studies, University of Manchester, England.
PAULEV, P.E. (1969): Respiratory and cardiovascular effects of breath-holding. Acta. Physiol Scand. Suppl. 324: 1 - 116.
RAHN, H., OTIS, A.B. HODGE, M., et al (1946): The effect of hypocapnia on performance. J. Aviation Med. 17:164.
RAHN,H. & FENN, W.O.(1960): A Graphical Analysis of the Respiratory Gas Exchange. The O_2 - CO_2 Diagram. The American Physiological Society, Washington, D.C.
SCHAEFER, K.E. & CAREY, C.R. (1962): Alveolar Pathways during 90 ft breath-hold dives. Science, N.Y. 137 : 1051 - 1052.
SCHAEFER,K.E. (1975) : CO_2 effects under conditions of raised environmental pressure. IN: The physiology and medicine of diving and compressed air work, (2nd ed) Eds. Bennett, P.B. & Elliott, D.H., Baillers Tindall, London.
SEM-JACOBSEN. C.W. & STYRI. O.B. (1970): E.K.G. arrhythmias monitored from free swimming SCUBA divers. IN: Proceedings, Journees internationales d'hyperbarie et de physiologie subaquatique. Marseilles. 8-11 Juin, 1970.
SMIT,P.J. (1967) : Die invloed van apnoise duike op sekere fisiologiese prosesse. Voeding, 28:8, 380- 409.
SMIT,P.J.(1974): Alveolar O_2 content of apnoeic divers, IN: Proceedings of the 3rd South African symposium for underwater sciences (Ed.Smit,P.J.), Baralogia, University of Pretoria,9.
SMIT,P.J. & Giesel,J (1981) :Apnoeic diving Safety - Experimental approaches to O_2 augmentation. S. Afr.Med. J. 60.959.
WOLF, S. (1970): The O_2 - conserving dive reflex. Abbotempo,8:2, 14-17.

Discussion following Dr. Landsberg's presentation

Dr. Rahn: You've opened up a whole new window for me in terms of high CO_2 at the start of a dive. I was not prepared for that. Are these the divers who cannot wait long enough to go down for their next fish, is that a way of putting it?

Dr. Landsberg: We actually lost one of our champion spearfishermen during an individual spearfishing competition. He was lost on the first breath-hold dive - went down and never came up. His body was recovered on the sea bed.

Dr. Rahn: On the first dive?

Dr. Landsberg: On the first, yes.

Dr. Rahn: That wouldn't be hypercarbia

Dr. Landsberg: But he was swimming towards the diving site, I don't think he gave himself enough time to recover.

Dr. Boysen: When you talked about dive profiles, can a diver change his profiles depending on his dive history?

Dr. Landsberg: No, it's completely unpredictable. In fact I've found that the same diver, under different diving conditions, produced different breath-hold dive profiles. There is no standard profile, it changes from one group to the other. When you interview these spearfishermen, when you <u>warn</u> them about this unpredictability, they say:- "Look Doctor, I've been diving for 10-15 years and I've never come near to blacking out" ... and then in the very next competition, that same diver is drowned. This is what had happened in South Africa, we have lost most of our champion spearfishemen, so we came up with this warning. We now also have a buddy system and surface cover during competitions.

Dr. McDonough: Wouldn't duration of dive be an important determinant of whether or not risk was higher, and if that is so, shouldn't the types of divers that you're talking about time their dives?

Dr. Landsberg: Yes, I didn't have time to go into this; it will appear in the paper that I have submitted, but in effect, this is what we are doing now. We are giving them a "safe" dive profile. The spearfishermen and underwater hockey players are completely different animals to the Scuba diver in South Africa. They, the spearfishermen and hockey players, are the supreme athletes in underwater sport. The spearfishermen do use a watch and work out a safe dive profile; they try and stick to their profile and do not digress from it, even if they shoot some magnificent fish at the bottom - when the dive profile says to come up, they just forget about the fish and surface. This has been the saving grace in preventing more fatalities.

Dr. Hong: In your schemes diving bradycardia is detrimental, right? Because it reduces cardiac output and cerebral blood flow ...

Dr. Landsberg: But, it's the quality of blood which is being pumped to the brain - very low O_2 partial pressure, high CO_2 partial pressure, which causes unconsciousness. This is the extreme case, I think that is already beyond the dive profile on the way to asphyxia and death.

BREATH HOLD DIVING ACCIDENTS IN THE MEDITERRANEAN AREA

Damiano Zannini, M.D.

Breath hold diving became a mass sport in 1950 in the Mediterranean area and the main countries involved were naturally Italy, France, Spain and Yugoslavia. This led to the organization of national and international spear-fishing contests, which soon became part of the World-wide Championship.

Of course, both doctors interested in sports medicine and doctors who practiced this sport themselves got involved in problems of prevention and safety in the field of breath hold diving, as well as Scuba diving (their interest was almost inevitable, considering that the technique of diving is tightly tied to physiology).

Dating back to the second half of the 50's, medical groups were organized in Sports Associations with the participation of doctors interested in Sports Medicine. Studies and public messages about diving safety problems were developed at national and international meetings, using all means on hand, i.e., articles on newspapers, diving magazines, T.V. shows, etc., especially at the beginning of summer. Technical manuals were also published, as well as scientific literature and proceedings of the meetings held.

For some time physicians of countries on the south-eastern side of the Mediterranean sea (such as Greece, Turkey, Egypt, Israel, Tunisia) kept in contact with their colleagues in the European countries.

In effect, what we have accomplished is a better understanding of opinions among doctors of the Mediterranean area, with help from CMAS (World Confederation of Underwater Activities), FFESSM (Federation Francaise Etudes et Sports Sousmarines) and SIMSI (Italian Society of Underwater and Hyperbaric Medicine), and also thanks to EUBS (European Undersea Biomedical Society).

Through these societies new research contributions about the physiology of breath hold diving became available to several universities, such as Marseille and Genoa. Starting from 1960 on, many up-to-date training classes for physicians were organized, first in Genoa, then in Naples. Hundreds of doctors attended. At the same time, schools for sport diving developed and doctors were called to collaborate in teaching divers how to avoid accidents.

In 1976 with the constitution of the SIMSI and then the Cocchi Association for Safety in Water, meetings among doctors or between doctors and technicians multiplied.

Posters were prepared for primary and high schools. Legal

procedures relating to death by accidents in pools and responsibility of the life-guards were studied intensively, especially with regard to rescue procedures.

During meetings we have always been astonished by the great number of fatal accidents that occur in breath hold diving. This risk is generally undervalued by the practicing sportsmen and overestimated by the propagandists of prevention.

In the Mediterranean area, as well as in other parts of the world, statistic data that are exact, or indicative of the number of deaths, are not yet available, because of the fact that breath hold diving accidents are usually listed under "death by drowning or by submersion".

To know these data would be extremely useful, both to evaluate the exact seriousness of the risk, and likewise, to improve our knowledge on causes of syncope other than prolonged apnea. A better propaganda for public awareness of this problem would be possible.

All the European countries that touch the Mediterranean sea are now making efforts to organize a systematic gathering of data on these accidents, so most probably there should be more reliable data available next year.

These data could probably be used in preparing laws so as to achieve safer breath-hold diving, but this is a juridical and technical problem (for example, even if accident rate in mountain activities is much higher than accident rate in the sea, yet nothing has been done by law).

The safest rules, such as spear-fishing in couple, watching from above, presence of a scuba diver, connection with the surface by a line and buoy (all means which doctors agree should be applied) are too often considered non-applicable, because fishermen state they act as antifishing implements, especially during the competitions. As a matter of fact, this summer a young Italian diver died during a competition.

At present, the only numerical data that can be obtained are from a few insurance companies that take care of divers, members of a sport federation (in France and Spain); but these regard only the members, who generally represent a minority of those practising immersion in apnea; e.g., in Italy 30,000 divers are members of different Sport Federations, while those practising are certainly over 200,000. In France there are about 260,000 members of the Sport Federation, in Spain 6,500.

I got these data from colleagues of different Mediterranean Countries (fig. 1) and received most of the information from Italy.

Fig. 1. The Mediterranean area

The Central Institute of Statistics in Italy lists as number of deaths by "submersion" about 1100 persons a year, comprising seawater, inland lakes, rivers, pools or well, etc. About 80% of the victims are male. Females are not listed as victims of breath hold diving accidents. Thus, we can only list as possible victims of fatal breath hold diving accidents the males between 15 and 40 years of age, who are the most exposed during sports or recreational activity (see Table 1).

The years in Table I refer to 1975 through 1978. Unfortunately, since then the years are not any more divided in the same way and national statistics have not been published since 1980.

In the five years that followed I was able to obtain data from the undersea forces of the police and firemen, who were recovering corpses of divers who had died during breath hold diving.

TABLE 1

NUMBER OF DEATHS BY SUBMERSION OR DROWNING (ITALY)

YEAR	DURING RECREATIVE ACTIVITY - IN MEN (AGE BETWEEN 15-40)	TOTAL NUMBER M + F
1975	61	1093
1976	11 (a)	929
1977	22	1102
1978	33	1040

(a) skin divers, fishermen and swimmers are included in these numbers

On these reliable grounds, one can say that the victims in Italy cannot be less than five and not more than 20 to 30 per year. In Table II, I listed the estimates and the reliable data obtained from the five countries.

TABLE II

NUMBER OF DEATHS DURING BREATH-HOLD DIVING IN FIVE COUNTRIES OF THE MEDITERRANEAN AREA

COUNTRY	MINIMUM DOCUMENTED NUMBER PER FIVE YEARS	ROUGH ESTIMATE FROM NEWSPAPERS AND PERSONAL INFORMATION MEAN PER YEAR
Spain	7 (a)	5
France		20 (c)
Italy	25 (b)	10
Yugoslavia		10
Greece		10
TOTAL		55

(a) among 6.000 skin and scuba divers
(b) corpse recovery
(c) among 250.000 skin and scuba divers

In effect, it involves a minimum of about 55 deaths per year in all the controlled European area of the Mediterranean sea. This excludes the south and eastern coasts, which are much less frequented by tourists.

In addition to these data I can mention that about 70% of Italian divers who participate in national and international competitions have reported one or more presyncopal symptoms in their career.

Inasmuch as regards Italy and Greece, I can say that the victims of breath hold diving are mostly competition divers or persons with much experience.

Now, do old divers present more risks than young people? Can we hypothesize some factors as: CO_2 retention and lower sensitivity to CO_2, too deep or too long dives, heedlessness of the danger, age-related causes of syncope?

To conclude, I would like to express a certain amount of perplexity which I feel for two opposite facts. First, divers will not follow proper rules of safety to avoid fatal consequences; secondly, researchers in the physiological field are developing an always deeper knowledge. Let us hope that this knowledge will help the divers to consider more seriously the risks they face.

Since the number of accidents seems to be decreasing rather than increasing (even though the practice of breath hold diving has been increasing, especially after the prohibition to use SCUBA for fishing), I would like to believe that all the studies and propaganda has at least contributed to save a certain number of persons. Reliable individual observations already confirm this.

Discussion following Dr. Zannini's presentation

Dr. Hong: Do you have any breakdown of accidents by seasons, for example, winter versus summer?

Dr. Zannini: No, I don't; but the accidents are more frequent in summer when most people are on vacation. However, also in winter a lot of people dive regularly along the coasts, as the water temperature does not drop below 10-15^{o}C.

LOSS OF CONSCIOUSNESS DURING BREATH HOLD DIVING

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The best known, most frequent and most dangerous type of accident connected with apnea diving is the sudden loss of consciousness under water. It is most common at depths less than 2 m and hence during distance underwater swimming in pools when it may occur at distances from 45 m or more (1,2).

These accidents are due to hypoxia and more common after hyperventilation which decreases CO_2 tension and prolongs breath-holding time without increasing O_2 supply significantly. Very well-trained divers may however have hypoxic syncope without significant hyperventilation (2). The same type of "black outs" may occur also after deep apnea dives if the diver stops ascent at depths between 5 m and surface.

We have however recorded abnormal heart rhythms with ECG during a series of deep apnea dives conducted over several years which most probably were the main cause of loss of consciousness (3).

Material and Methods

ECG recordings during apnea diving were obtained in about 150 healthy (age 20-40 years) sport divers with repeated dives to different depths in each. As several dives were followed in each a total of close to 500 have been recorded.

In 18 subjects measurements were obtained with a percutaneous right heart catheter during 70 dives from 3 to 30 m of depth. None of the subjects had a history of cardiac arrhythmias or other heart disease, neither has such occurred later. All subjects gave informed consent to the study.

The divers hyperventilated for one minute at surface before the dive. They were pulled head first to the desired depth by a lead weight and thereafter turned head up and held on to a rope during the apnea. They were followed under water by experienced Scuba-divers in case of accidents. All used wet suits. The depths varied between 3 and 72 meters.

Two lead precordial ECG and pulmonary artery pressures were recorded with a waterproof tape recorder tied to the back of the subject. Pulmonary artery pressure (PAP) and surrounding hydrostatic pressure were recorded with piezo electric transducers inside the shielding, with subtraction of hydrostatic pressure from the PAP .

Results

During diving various degrees of bradycardia were seen in all experiments. The bradycardia increased with time at depths down to 20 m. At greater depths the decrease in heart rate was more rapid and related to the increase in depth. However, after 60 s the same degree of bradycardia was finally seen at all depths (f=26-46 min.). Down to 20 m sinus bradycardia was most common while at greater depths disappearance of the P-wave often indicated a nodal rhythm or a His'bundle pacemaker. Single or series of 3 to 5 ventricular extrasystole were often recorded.

Four subjects lost consciousness during ascent. They had dived to 20, 20, 23 and 30 m of depth, respectively. One of those ascending from 20 m had a normal heart rythm but ascended slowly and got unconscious at 4 m of depth after 92 s. He probably had hypoxic syncope. Another subject swimming up from 20 m had several extrasystole before ascent and ventricular tachycardia when he lost consciousness at a depth of 6 m. He was brought to surface after 52 s and rapidly regained consciousness. Two of those with heart catheterization had "black outs" during ascent. One had dived to 30 m of depth and got unconscious after 95 s also with ventricular arrhythmia with a high frequency. One subject diving to 23 m with a total time of submersion of 118 s is shown in the following illustrations. Sinus tachycardia was seen after hyperventilation with a decrease in heart rate during descent (fig. 1). Immediately after arriving to 23 m of depth the heart rate was down to 60/min. There was probably a nodal rhythm and series of extra-

systole occurred (fig. 2), while PAP had increased to 30-40 mm Hg. After 42 seconds a series of ventricular extrasystole occurred which turned into a ventricular fibrillation on ECG with a maximum frequency of 584/min. and an average frequency of 230/min. (fig. 3). However, there was still a dynamic frequency on the PAP registration of 90 and the pulmonary artery pressure showed a small decrease. After 50 s to 60 s ECG showed ventricular fibrillation while small pulmonary artery systole were seen with a frequency around 60/min. The subject still felt well as related later on. He started climbing up the rope but lost consciousness when reaching 9.5 m after 90 s (Fig. 4). Electrocardiographically he still had ventricular fibrillation but now with very irregular PAP systole with a rate of 110. When he was brought to the surface he had an electrocardiographic sinus rhythm but with no dynamic right heart action and a pulmonary artery pressure of close to 80 mm Hg. He was resuscitated with oxygen on mask and cardiac compressions at 142 s (Fig. 5). After half a minute he had regained electrocardiographic sinus tachycardia with synchronous pulmonary artery pressure fluctuations. The pulmonary artery pressure was still high and he had single extrasystole but was mentally alert and no further complications were noted (fig. 6).

Discussion

It cannot be excluded that irritation from the right heart catheter has contributed to the ventricular tachycardia or fibrillation seen in some of the subjects.

However, ventricular extrasystole occurred in most subjects also without heart catheterization and it seems probable that the extensive acute cardiac dilation, at depths below 20 m (fig. 7), contributes to the increased irritability. Hypoxia during extended apnea may also increase irritability and echocardiographic registrations at our laboratory during apnea at RV in air have actually shown VES excited by each diaphragmal contraction occurring near breaking point. It seems contradictional with sustained right ventricular function during tachycardia/fibrillation. We have recorded the same phenomenon in dogs with ventricular fibrillation caused by local applica-

tion of aconitin (fig. 8). Likewise, everybody working with resuscitation will have noticed patients with electrocardiographically normal sinus rhythm but with no measurable pulse or blod pressure.

Conclusions

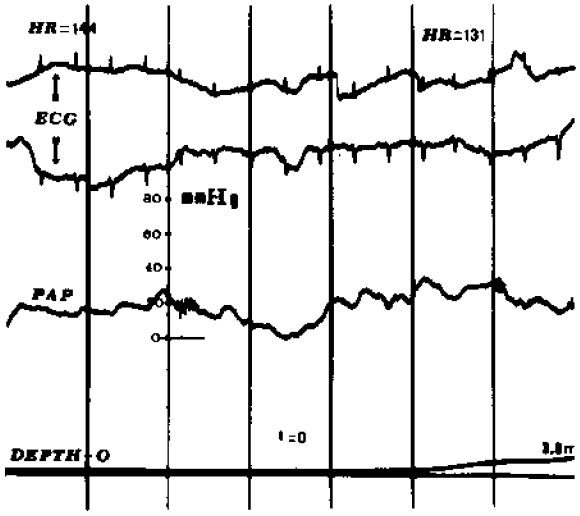
Heart distension, hypoxia or other factors connected to deep apnea diving seem to increase myocardial sensitivity and may cause extrasystole, ventricular tachycardia and ventricular fibrillation with loss of consciousness. However, prognosis seems good as no complications were noted in four such incidents and only one of four subjects needed resuscitation.

References

1. Craig AB. Causes of loss of consciousness during underwater swimming. J Appl Physiol 1961;16(4):583-586.

2. Arborelius M Jr. Om medvetslöshet vid undervattenssimning. (About unconsciousness during underwater swimming). Sv Läkartidningen 1962:59:1-7.

3. Data PG. La sincope anossica. Aspetti fisiopatologici. In: Sicurezza in acqua. Atti del 1° convegno nazionale sulla sicurezza in acqua. Prato 5-5-1979. Nistri-Lischi Editori, Pisa, 1981. Page 83-98.



Data PG Fig. 1

The figure shows sinus tachycardia on ECG registered at surface after hyperventilation. Pulmonary artery pressure (PAP) was normal. Apnea dive starts at time 0 (t=0) and heart rate (HR) decreases from 144 to 131 beats/min. during the first m.

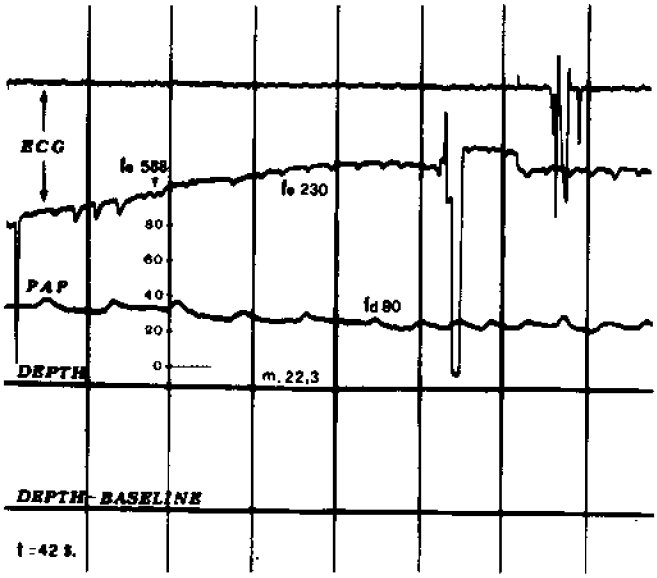
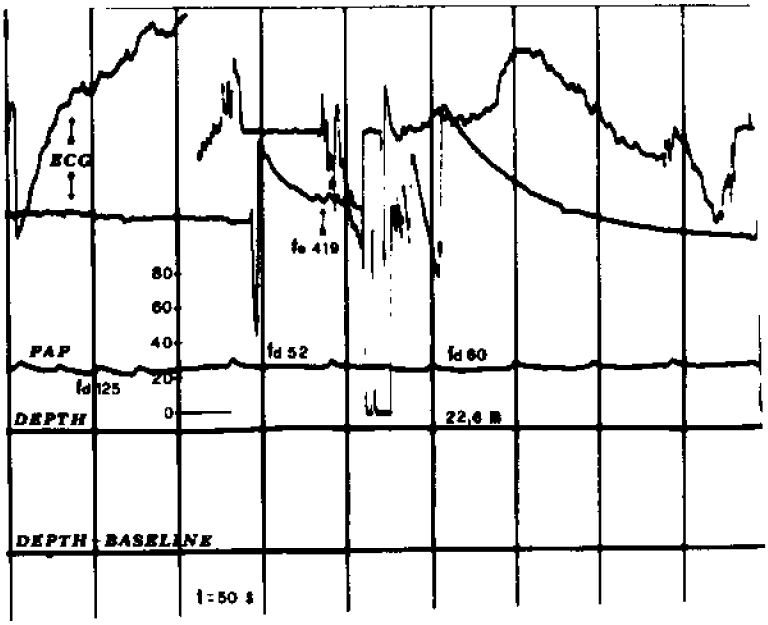


Fig. 2

The subject has arrived to 22 m of depth after 42 seconds. The ECG shows ventricular tachyarrhythmia with an electrical frequency (fe) of 230/min. to 588/min. while dynamic frequency (fd) in the pulmonary artery was 90/min.



Data PG Fig. 3

At time 50 s the dynamic frequency decreases (60 min.) while ventricular fibrillation continuous. Pulmonary artery pressure has decreased from 40-50 to 25-30 mm Hg. Subjectively the diver felt well.

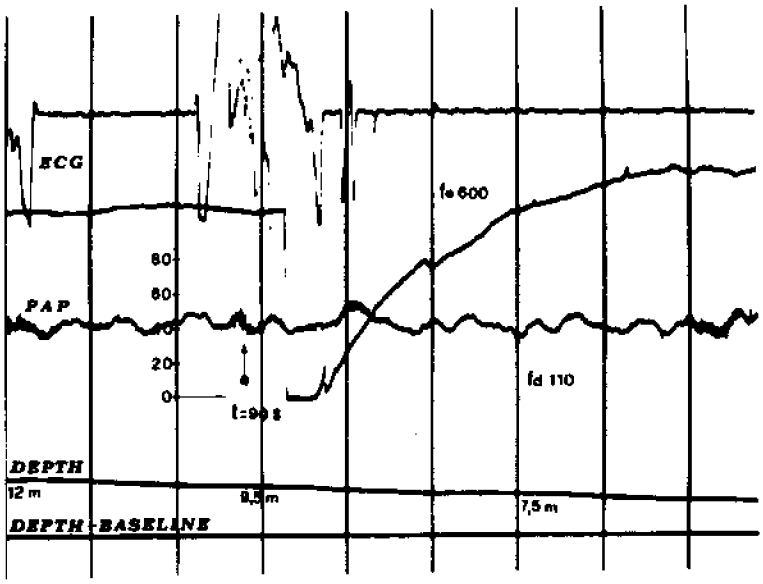
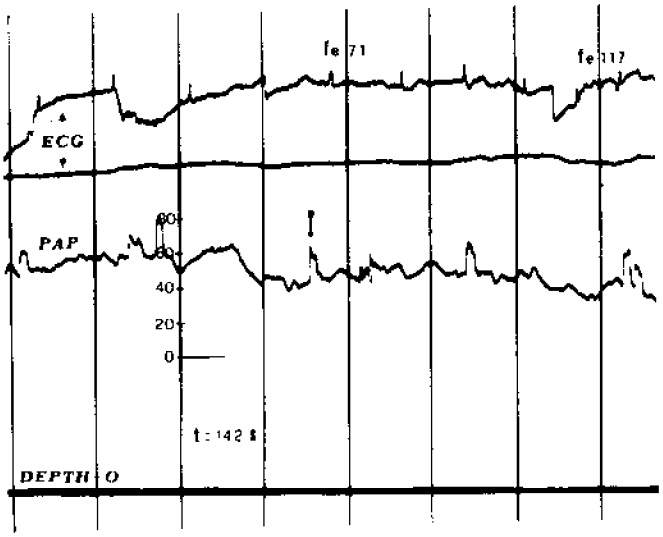


Fig. 4

During ascent by climbing the rope the subject gets unconscious at time 90 s (arrow). The increase in pulmonary artery pressure to 40-50 may indicate decreased left ventricular output. The depth is 9.5 m.



Data PC Fig. 5

The subject was brought to the surface at time 118 s and was still unconscious after 142 s. Cardiac resuscitation by external heart compression (arrow) and oxygen by mask had begun. Electrical heart rate was 71–117/min. but no coordinated pump function was seen.

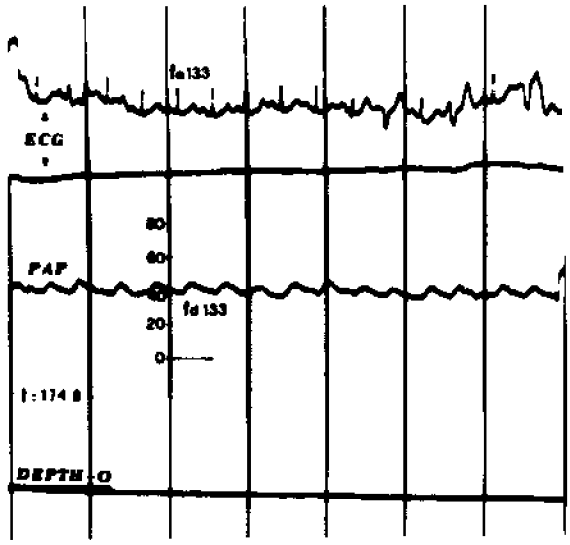
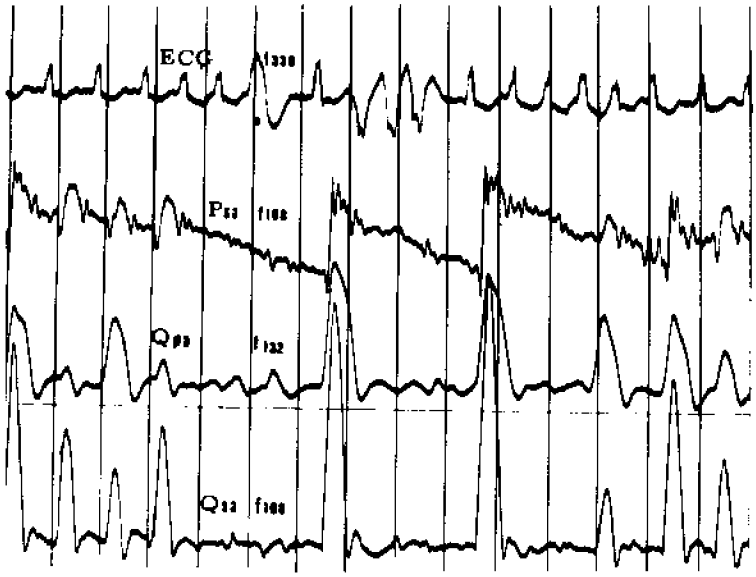


Fig. 6

The subject is conscious at time 174 s. He had coordinated sinus tachycardia and synchronous pulmonary artery pressure swings but still an elevated PAP. He normalizes all variables within some minutes.

Fig. 7 Thoracic X-ray obtained during open sea breath hold dive at 20 meters. Note the very distended heart and elevated diaphragm. The contours of the heart and diaphragm at 1.0 ATA, obtained from a separate X-ray picture, have been indicated by dashed lines.



Data PG Fig. 8

Recording of tachyarrhythmia induced by local application of aconitin in the dog. The mean electrical frequency is 388/min. with periods of fibrillation with frequency 500/min. Frequencies of systemic arterial pressure (Psa) and pulmonary artery flow (QPa) and aortic flow (Qa) are not synchronous with electrical events and the rate is about 50% of electrical frequency.

Discussion following Dr. Data's presentation

Dr. Lundgren: It is certainly food for thought and quite impressive that you would be able to get these recordings under those conditions; there's really only one measurement that I'm missing and that's the heart rate of the experimenter as this was happening.

Dr. McDonough: Were these Swan-ganz catheters, that is, balloon tipped catheters floating in the pulmonary artery, following insertion through a peripheral vein?

Dr. Data: Yes.

Dr. McDonough: What was your access route, was it antecubital, neck, or groin?

Dr. Data: Antecubital.

Dr. Boysen: Did you get fairly good tracings when your subjects were trying to swim with that catheter in the arm? When we tried to do experiments like that with arm movement, the catheter tends to move back and forth.

Dr. Data: We had no problems in this regard, as the arm was splinted, so that they could not bend it.

GENERAL DISCUSSION

Dr. Hong: I proposed this morning that we should discuss more about the issue dealing with cold water drowning. Now we heard a number of papers showing the development of bradycardia and cardiac arrhythmias, which are potentiated by cold water. That's one phenomenon. But the other story involves cold water drowning of little babies, who were successfully resuscitated after 40 minutes. There is a general myth, a statement made by some people that this is due to the diving reflex. The media tell their story that, as soon as a baby drowns in cold water, he behaves like a diving seal, reducing his oxygen consumption. Does this view have a scientific basis? Since most of us who are interested in this question are in this room, maybe we should get some feedback from you about this issue.

Dr. Elsner: Yes, I quite agree. There's a preamble, though, that I'd like to suggest. You know, I think we ought to finally discard, stamp out, and throw away the term diving reflex.

Dr. Hong: Right.

Dr. Elsner: There's no way that that makes physiological sense. But on the other hand, is it possible that all of the events that you see in the child who falls into cold water, and is revived, could be explained simply on the basis of the rapid cooling of the central nervous system and having nothing whatever to do with bradycardial response?

Dr. Arnold: I think there are probably a lot of components. Brain cooling is important. There are two similar situations: the case of asphyxia as opposed to the case of water inhalation. The asphyxial case has a much better prognosis. For the same length of submergence time and the same temperature of water, one individual will survive well and another will either be severely compromised or will not survive. So I suspect that there's a variability in our ability, one person to another, to survive. In the period of submergence, before achieving brain cooling (which may take longer in an adult than in a child), the diving response may maintain cerebral integrity.

Dr. Hong: The reason why this diving reflex or response is invoked by some people is the notion that, because of this response, only brain and coronary blood flow is maintained at the expense of other regions. However, are we justified to say that the blood redistribution developed in human subjects during cold water face immersion, as it seemed to develop in diving mammals, is responsible for the conservation of oxygen?

Dr. Elsner: There's not one scrap of data for that. All I'm saying is that there is not a single measurement to support this statement. However, the diving response may be involved, for example, in the initiation of apnea and the early distribution of blood flow.

Dr. Hong: Number two, what we need is an hypothetical experiment with human subjects, in which we let the subject fall into cold water, without his knowledge. This procedure may induce a severe

bradycardia and the distribution of blood flow similar to that in diving seals, whereby conserving oxygen.

Dr. Lundgren: Forced diving.

Dr. Hong: That's right.

Dr. Siesjo: I have a question to Dr. Boysen and a comment which may be relevant to the variability we are discussing. I was a bit intrigued by the fact that you can have asphyxia with maintained blood pressure for, say, eight minutes and brain damage does not result. The reason of course is that during all that time you must have some oxygen delivery to the brain. I'm wondering what your actual PO_2's were. When working with rats we have always been rather impressed with the resistance of the brain to hypoxia, as long as the perfusion pressure is maintained. However, there is one factor which gives a lot of variation to the outcome in hypoxia and also in ischemia. That's the blood glucose concentration. For example, if you induce incomplete ischemia in a fasted animal which would maintain a blood glucose concentration which is normal or below normal, then the brain can sustain perhaps 10 or 12 minutes of virtual anoxia without any real brain damage. But if you perform the same experiments on a fed animal, so that hyperglycemia develops, then perhaps four or five minutes may be sufficient to give irreversible brain damage. Of course the difference is that in the last case you get exaggerated lactic acidosis in the brain. So it means that in your experiments, where the blood pressure is maintained, the reaction of the brain would depend very much on the blood glucose concentration. Since you have maintained circulation to the hypoxic brain, conditions are at hand for enhanced lactic acidosis. I wonder, first, what was the P_{O2} in your experiment and, second, what was the blood glucose concentration.

Dr. Boysen: The PO_2 at the end of by four or five minutes of asphyxia would be less than twenty torr.

Dr. Siesjo: And at the end of, say, seven or eight minutes?

Dr. Boysen: Usually, at the end of seven or eight minutes, if the measurement were correct, it was nine or ten torr, something like that. I don't know the answer to the rest of your question because I didn't measure blood glucose, but having known about what you said, these were fasted animals that had water but no food overnight, and we did not use glucose infusion when we administered the anesthetic.

Dr. Siesjo: So they were fasted.

Dr. Boysen: Yes, they were.

Dr. Hong: However, blood glucose could considerably go up because possible increase in catecholamine release. I can see that glycogenolysis might be stimulated because of the catecholamine release due to frightening.

Dr. Siesjo: Yes, that's absolutely true, but if you fast a rat for

24 hours, there would be virtually no liver glycogen left. So it means that your blood glucose concentration cannot rise much.

Dr. Hong: But here's another thing we've been talking about, well-fed children walking around a lake and suddenly drowning in cold ice water, so in that case blood glucose level could considerably go up.

Dr. Siesjo: That could be potentially dangerous.

Dr. Rahn: Could some very simple experiments in man be done on the induction of laryngospasm with cold and warm water?

Dr. Boysen: We did that with cats, not with dogs, and you're absolutely right. Almost any way to manipulate the airway can induce laryngospasm, and even if I haven't any clinical experience with cold water drowning, when you put cold water versus warm water into a cat's air way, the cold water has a much higher incidence of instituting or initiating a significant degree of laryngospasm. So in the cold water that may be another factor, irrespective of whatever the reflexes we try to name.

Dr. Rahn: But don't you think these rather simple experiments could be done in man.

Dr. Boysen: I don't think we could get any volunteers. The subjective feeling is not a very nice one.

Dr. Lundgren: Is anything known about the possible influence of hypocapnia or hypercapnia, for that matter, on the susceptibility to laryngospasm?

Dr. Boysen: You mean as it starts?

Dr. Lundgren: Yes, realizing that hypocapnia is related with increased tendency for spasm in other skeletal muscle, I was just wondering.

Dr. Boysen: Yes, it's the same in the upper airway.

Alveolar Gas Composition and Performance

Hermann Rahn

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During breath-hold diving alveolar gases become greatly altered and their composition shortly before the surface is reached reveals not only the degree of hypoxia and hypercarbia and hemoglobin de-saturation that has occurred, but also to what degree motor and mental performances have been impaired. Divers are aware that excessive hyperventilation before a dive leads to typical signs of hypocapnia, namely, tingling sensations in the extremities, dizziness, and carpopedal spasms. They are also aware of mental confusion when CO_2 concentration becomes very high. The gradually developing hypoxia during a breath-hold is not easily recognized, and on occasion a brief state of euphoria may precede blackout and loss of consciousness because of lack of oxygen. Furthermore, following excessive hyperventilation hypoxic states can be reached at the end of the dive while the diver has not recovered from his hypocapnia and where performance impairment of each of these two conditions is additive (Otis et al, 1946).

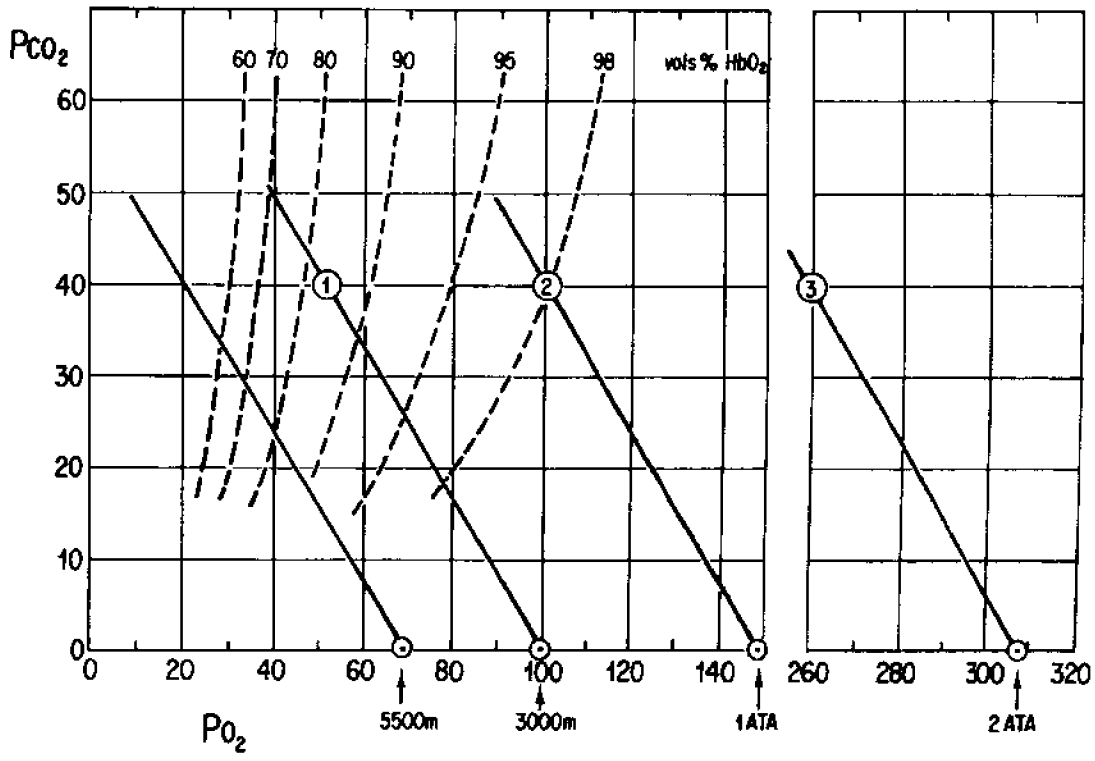

Fig. 1. The O_2–CO_2 Diagram (Rahn and Fenn, 1955).

The question is what are the limits of alveolar gas composition during which normal motor and mental functions prevail and what are the compositions where hypocapnia, hypoxia, hypercarbia or a combination of hypoxia and hypocapnia or hypoxia and hypercarbia interfere with normal performance. These have been described in detail using multiplication, choice-reaction times, and visual discriminmation tests during various degrees of hypocapnia breathing air at ground level (Rahn et al., 1946) and hand-steadiness and visual discrimination tests during various degrees of hypocapnia, hypercarbia, and anoxia performed in an altitude chamber breathing air (Otis et al., 1946). The results are shown in figure 2.

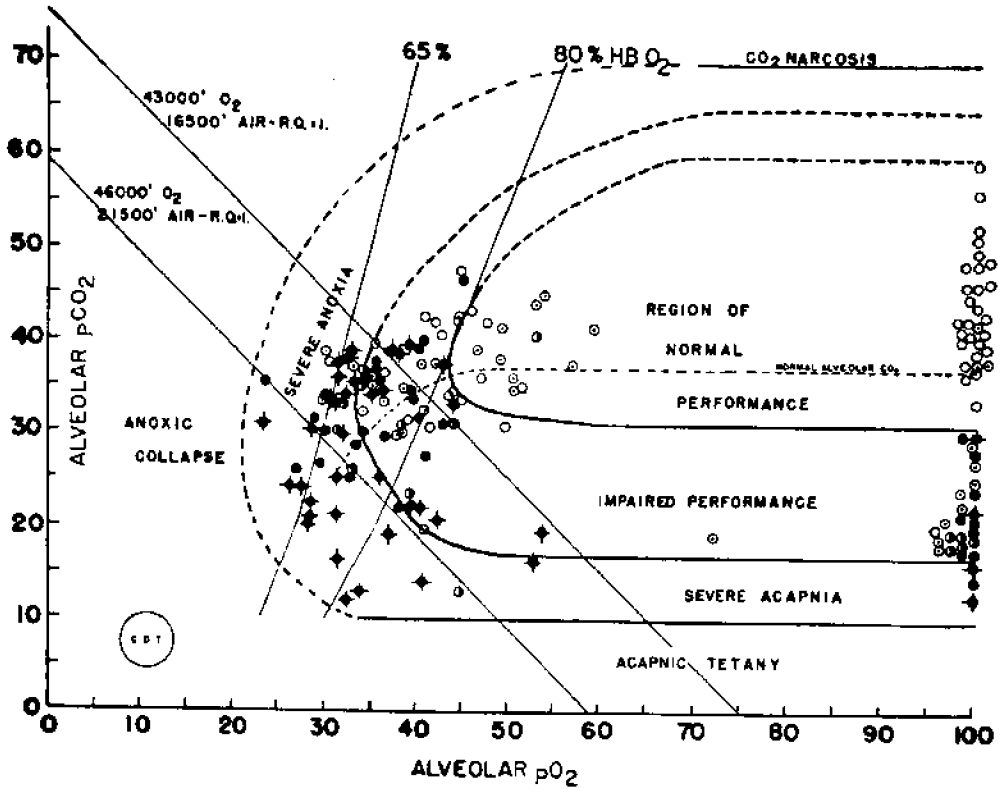

Fig. 2. Contrast discrimination test. For details see text. From Otis et al., 1946.

As an introduction to this plot figure 1 provides a basic interpretation of the O_2 -CO_2 diagram where PCO_2 (torr) is plotted against PO_2 (torr) (Rahn and Fenn, 1955). The inspired oxygen tensions, 0.2095 (PB -47), at 5500 m and 3000 m altitude, at 1 and 2 ATA are shown by circles on the abscissa. The diagonal originating at these points describes all simultaneous O_2 and CO_2 tensions whenever the respiratory quotient is 0.8. For example, at 1 ATA PIO_2 = 149 torr, and circle 2 indicates the alveolar composition when $PACO_2$ = 40 torr and R.Q. = 0.8. The dotted lines are isopleths of hemoglobin saturations (vols. % HbO_2),

which is 98% at circle 2. Circle 1 is the alveolar composition at 3000 m where the hemoglobin saturation is 85%, and circle 3 represents the alveolar composition at 2 ATA when breathing air. It can be seen that by going to various altitudes for performance tests, various degrees of hypoxia can be achieved during normal ventilation. In addition, by adding a large dead space to the mask, higher than normal alveolar CO_2 tensions were obtained at various altitudes, and by breathing with a resuscitation device (pneumolator) various degrees of hypocapnia were achieved at altitude.

Figure 2 shows the contrast-discrimination test on the O_2-CO_2 diagram. Each type of symbol represents the probability (P) that a subject's score was not different from his control score. The blacker the symbol the greater the certainty of abnormal function. Solid circle with cross $P < 0.01$; solid circle $P > 0.01 < 0.05$; half solid circle $P > 0.05 < 0.1$; open circle with dot $P > 0.1 < 0.5$; open circle $P > 0.5$. A very similar plot was obtained for motor performance impairment using a hand-steadiness test (Otis et al., 1946). Of particular interest is the impaired function during various degrees of hypocapnia when O_2 tensions were normal.

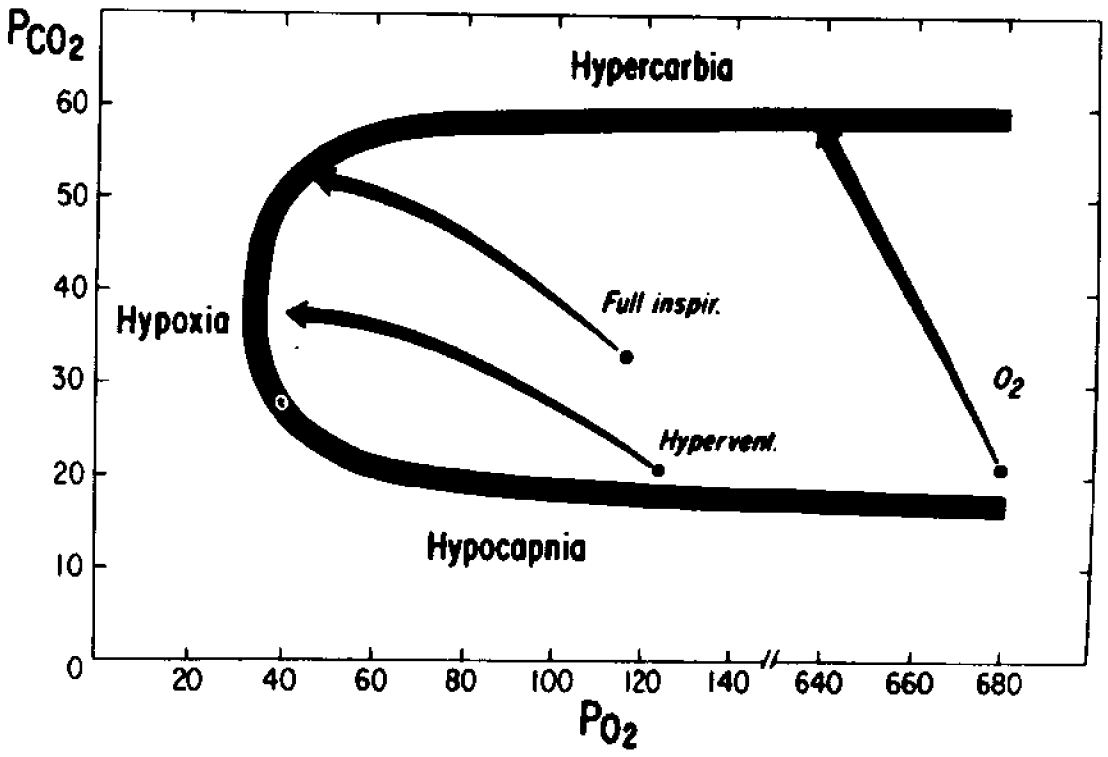

Fig. 3. Useful area of performance during breath-holding bordered by regions of hypocapnia, hypoxia, and hypercarbia. From Rahn et al., 1986.

Assuming that these performance criteria are applicable to the breath-hold diver, then figure 3 charts the approximate boundaries for safe performance on the O_2-CO_2 diagram (Rahn et al., 1986). The pathways for three breath-holds are shown, all originating in the useful performance area. For example, after a full inspiration breathing air at sea level, the alveolar P_{CO2} = 35 torr and the breaking point at the borderline of safe performance is determined by a combination of hypoxic and hypercarbic stimulus. With hyperventilation, in this example the alveolar P_{CO2} is reduced to 20 torr. If this is done with pure oxygen, the apneic pathway is shown at the right. It is a straight line ending at the breaking point of 60 torr and hypercarbia is the only stimulus. On the other hand, if the same degree of hyperventilation is done breathing air at sea level, the only stimulus to breaking is hypoxia. However, this stimulus alone is frequently rather weak, and if apnea is prolonged, alveolar O_2 will continue to fall to levels of 30 - 35 torr, where black-out and unconsciousness set in.

References

Otis, A. B., H. Rahn, M. A. Epstein and W.O. Fenn. 1946. Performance as related to composition of alveolar air. Am. J. Physiol. 146: 207-221.

Rahn, H., and W. O. Fenn. 1955. A graphical analysis of the respiratory gas exchange. The O_2-CO_2 diagram. Am. Physiol. Soc., Washington, D.C.

Rahn, H., A. Olszowka and C. Lundgren. 1986. Shallow water and ascent blackout during breath-hold diving. In: Adaptive Physiology to Stressful Environments, Edited by J. S. Samueloff and M. K. Yousef. CRC Press (in press).

Rahn, H., A.B. Otis, M. Hodge, M. A. Epstein, S. W. Hunter, and W.O. Fenn. 1946. The effects of hypocapnia on performance. J. Aviation Med. 17: 164-172.

Depth records: practical considerations

Enzo Maiorca, Siracusa, Italy

In 1913 Georghios Kaggi', a Greek sponge diver, made several breath-hold dives down to 80 m of depth (more than 260 feet) , to recover the lost anchor of the Italian battleship "Regina Margherita" (1). However, it was not until 1949 that deep breath-hold diving started as a sport. In that year Raimondo Bucher, an Italian diver, made and won a wager: holding his breath and pulled underwater by a weight, he reached 30 m of depth (about 100 feet). At the bottom, an astonished hard-hat diver gave him a waterproof envelope containing the prize: twenty thousand Italian lira. Since that time many deep breath-hold divers, both men and women, have descended to increasing depths recently diving down to more than 100 m (330 feet). In this brief review of breath-hold dives to record depths I would like to mention, besides Raimondo Bucher, Ennio Falco, Alberto Novelli, Americo Santarelli, Bob Croft, Jacques Mayol, Maria and Giuliana Treleani, Angela Bandini, my two daughters Patrizia and Rossana and myself (2). Rossana has recently established the new world record dive for women, descending to 68 m.

Unluckily, in 1960 the World Underwater Federation and in 1981 the Italian Underwater Federation ceased to acknowledge these records, because their Medical Commissions judged these attempts dangerous. I heartily disagree with this opinion that was based on the threat of thoracic squeeze. The depth at which thoracic squeeze was predicted kept moving deeper and deeper with every new successful record dive.

In Table I you can see the three principal techniques for deep breath-hold diving: free diving, assisted-descent diving and assisted diving. In free diving, both descent to the bottom and ascent to the surface are performed by the diver by his own effort, that is swimming down during descent, and pulling himself up along a line during ascent. This type of diving is performed not only by record divers, but also by the much more numerous spear-fishermen and by the Ama of Japan, in particular by the Cachido. In the case of assisted-descent diving, the diver descends with the assistance of a weight, but he or she goes back to the surface again using his or her muscles. This technique is used by record and pearl divers. These are the two types that I have been performing for many years and I still hold the last official records of both types of diving, with 87 m (285 feet) reached in 1974 during an assisted-descent breath-hold dive, and with 60 m (196 feet) during a free breath-hold dive, again in 1974. There is a third type of breath-hold diving, and this has been used by Mayol in his last record dive to 105 m of depth (more than 340 feet) (3): this is the assisted diving. In this

form of diving the diver is helped in both the descent and ascent. He is pulled by a weight on his way down, and he is lifted by a self-inflatable balloon or by a rope coming up. This type of diving is performed by record divers and by the Ama (Funado). In the past it was performed also by the Greek Sponge Divers.

TABLE I

Types of Breath-hold Dives with regard to means of Descent and Ascent

	Type	Descent	Ascent	Performed by
1.	Free Diving	swimming	hand-over-hand & swimming	record divers spear fishermen Ama-Cachido
2.	Assisted-descent diving	pulled by a weight	hand-over-hand & swimming	record divers pearl divers
3.	Assisted Diving	pulled by a weight	lifted by a rope or air balloon	record divers Ama-Funado

Figure 1 depicts the depth-time profile of my deepest breath-hold dive, in which I reached 94 m during training. This was an assisted-descent dive. After an hyperventilation lasting 8 min, I took a full inspiration and I started my head-down descent, pulled by a weight of 26 Kg that ran along a wire and that I could stop by means of a brake. Despite the fact that I started clearing my ears from the surface, I had to stop at 30, 40 and 50 m during my descent. At these depths I turned head-up for a few seconds for additional ear-clearing. From 50 to 94 m I did not need to clear my ears anymore. Once I reached the maximal depth, I left the weight and I started the ascent, pulling myself up along the wire with my hands and swimming with my legs. Closer to the surface, as the wet suit expanded and regained buoyancy, it helped my ascent. The total duration of this dive was 3 min and 40 sec, with approximately an equal amount of time spent on descent and ascent, at a rate of about 1 m/sec .

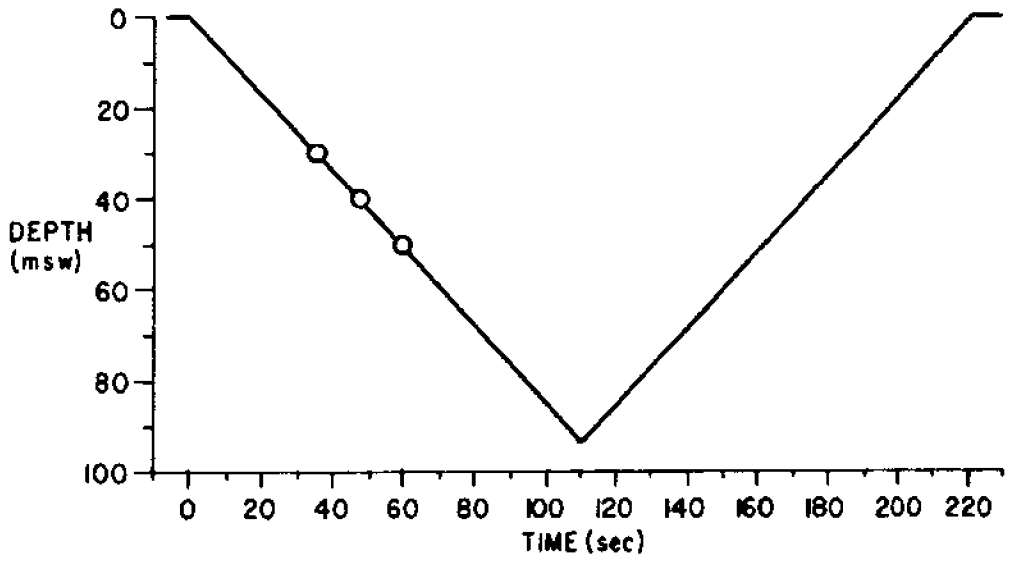

Fig. 1 Depth-time profile of my breath-hold dive to 94 m. Also shown are stops at 30, 40 and 50 m for additional ear clearing.

In my career I have performed more than one hundred breath-hold dives to depths greater than 50 m (165 feet) and many more to shallower depths. I have suffered seven episodes of syncope, three of which happened during training in a swimming pool. The training consisted in a horizontal underwater swim of 130 m (more than 420 feet). The other four episodes of syncope occurred at sea, during attempts to break records. No fatalities have ever occured throughout the years among dozens of deep breath-hold divers during their numerous record attempts, thanks to the assistance of skilled SCUBA divers. These safety divers are positioned at different depths along the line from the surface to the bottom, in such a way that the breath-hold diver could be easily assisted and recovered, if anything went wrong.

Besides horizontal underwater swims for more than 100 m (330 feet), training in the winter includes "dry" breath-holds during heavy exercise (that is, climbing 55 stairs with a 10 Kg weight belt) and it also includes calesthenics (2). Whenever the sea conditions are good, an important part of training is of course breath-hold diving. I perform training dives to increasing depths and with decreasing bottom times (that is the time spent at the maximal depth), starting with dives to 50 m (about 165 feet) at which depth I spend at least 1 min.

Finally, I wish to thank all of you Scientists, who work hard everyday for us divers, and I want to give you a picture from my experience. Many years ago I was doing a deep breath-hold dive, and the weather was bad even if the sea was calm, the sky was gray and so there was almost no visibility underwater. I was at depth and suddenly saw a fish, a very, very, very big fish and I was afraid because I did not know what to do. Suddenly, a ray of sun fell down from the sky into the sea and hit the big fish, which exploded in a myriad of small silver fish. It was not a big fish, it was a school of sardines which disappeared in all directions. This is the picture that I give to you.

References

1. Musenga, G. Un pescatore di spugne (sommozzatore) che senza apparecchio si tuffa fino a 80 metri. Annali di Medicina Navale e Coloniale, 1915: 163-167.

2. Maiorca, E. Scuola di apnea. Editrice la Cuba, Roma; 1982.

3. Missiroli, F.M. and B. Rizzato. Mayol a 105 metri. In: Mondo Sommerso, no. 272 (Dec. 1983, Jan. 1984): 32-37.

Discussion following Mr. Maiorca's presentation

Dr. Lundgren: I think we should begin by thanking Mr. Maiorca for allowing us to edit his fascinating films in this way. Let me acknowledge Mr. Marky and Dr. Ferrigno for doing this. Perhaps a point worth making is that a deep breath-hold dive like this is no small feat from the logistic point of view. The assistance of the SCUBA divers can be extremely critical for your well being and we heard and saw that they are positioned along the line, all the way down to the deepest point where the breath-hold diver turns. In contrast to the breath-hold diver, they, of course, are faced with the problems of accumulating inert gas and if they had to ascend rapidly in assisting the breath-hold diver, they might run the risk of decompression sickness. So it's a very sophisticated operation logistically.

Dr. Ferrigno: The other thing is that the SCUBA divers also run the risk of nitrogen narcosis because they are breathing air even below 50 meters. These are very experienced divers but still you are talking about 90 meters in air for SCUBA divers. We should also appreciate the technical difficulties of taking films at these depths. I would also like to add that Mr. Maiorca doesn't follow the suggestion of Dr. Craig to start from outside the surface and then jump in the water. Instead, he does his hyperventilation in the water and then he starts his descent.

Dr. Rahn: Mr. Maiorca, do you have to turn head-up for ear-clearing?

Mr. Maiorca: Yes, I have to turn head up for a few seconds at 30, 40 and 50 m, and then I resume my head-down descent, despite the fact that I start clearing my ears from the surface. For some reason I need some additional ear-clearing at these depths.

Dr. Rahn: I'd like to have Mr. Maiorca answer a few questions now. Can you tell us how you hyperventilate before the dive?

Mr. Maiorca: There are two types of hyperventilation, one that we called hyperoxygenation, with fast breaths, but then we were told that hyperventilation was not useful for hyperoxygenation, so we changed to a different type of hyperventilation, that is, long and slow breaths. Four breaths per minute.

Dr. Rahn: For how many minutes.

Mr. Maiorca: Eight minutes. At the beginning of training, I hyperventilate only for two minutes, but after about one month, I feel that these two minutes are not enough, so I am forced to add more time for hyperventilation until the day of the record, when I arrive to eight minutes of hyperventilation. So there is a gradual increase in the time of hyperventilation. And the same is felt also by my daughters and other people in the School for Deep Breath-hold Diving in Siracusa.

Dr. Rahn: Another question. How careful are you, to completely relax just before the dive?

Mr. Maiorca: I am not totally relaxed because I know I am doing something dangerous. I am a man with all my feelings and my preoccupations, and not a superman.

Dr. Rahn: I was thinking about the CO_2 production.

Dr. Lundgren: Speaking of hyperventilation. With your method of hyperventilation you seem to create a high pressure in the thorax during expiration. Is that correct?

Mr. Maiorca: Yes.

Dr. Lundgren: I would like to suggest that it ties in with Dr. Craig's suggestion that the best way to start a dive would be to be out of the water, in order to have as little blood in the chest for starters as possible, so as to make maximum use of the return of the blood into the thorax during the dive, for compression of pulmonary gas. Now, perhaps this type of breathing, if in the water, would be an alternative to being out of the water in the preparatory phase, because the breathing seems to generate repetitive Valsalva maneuvers; that is, high pressure manuevers in the chest that would drive the blood out of the chest and that would provide room for a larger starting air volume.

Dr. Rahn: The Ama have been doing that for 2,000 years by whistling just before they dive.

Dr. Lundgren: Is it not true that all the Amas, when asked why they do it, answer that they always did it that way.

Dr. Rahn: Yes.

Dr. Lundgren: So, is there a philosophy, a special philosophy, behind this particular breathing pattern?

Mr. Maiorca: I just feel the need to do it, and we have seen that with this hyperventilation, we can obtain the best results.

Dr. Lundgren: In terms of depth or duration?

Mr. Maiorca: For both depth and duration.

Dr. Norfleet: In your record breaking dive, when you came to the surface and lost consciousness, did any of your companions notice what color your skin was?

Mr. Maiorca: I don't know, no one told me, I don't know.

Dr. McDonough: Were you breathing spontaneously, or did you have assisted ventilation?

Mr. Maiorca: They told me that I was unconscious for about one minute and thirty seconds, and that I was not breathing at all.

Dr. Rennie: What was the duration of the 130 meters horizontal

underwater swim?

Mr. Maiorca: Two minutes and five seconds.

Dr. Whitelaw: Mr. Maiorca, I would like to know what tricks you use to postpone or reduce the urge to breathe, and also whether you think that your breathing muscles are working toward the end of the dive.

Mr. Maiorca: Coming from a very deep dive, I don't feel diaphragmatic contractions, but I feel them during the horizontal underwater swims, even after I have swum only twenty meters. So, I feel the urge to breathe especially when I swim underwater horizontally for training, either at sea or in a swimming pool. And I think it's mainly a psychological problem. I fight the diaphragmatic contractions, and I train myself in doing that throughout the year, for example, climbing stairs (55 steps) while holding my breath and carrying a 10 Kg weight belt. And, it's much more difficult to hold your breath while climbing stairs because you have air around you, so you develop will power, which is essential for these very deep dives. I don't perform any special maneuver, like swallowing or anything else, to fight diaphragmatic contractions, which cease after awhile.

Dr. McDonough: Could I ask the question whether you were aware of any impending loss of consciousness during the dive we have seen or any of the six other dives during which you lost consciousness?

Mr. Maiorca: Not at all. No warning signs. I went from the very exciting feeling of having reached the record depth to the loss of consciousness.

Dr. Hong: When Jacques Mayol came to Japan last year, he told us he uses Yoga for his deep dives. What do you think of this?

Mr. Maiorca: As I told you before, I like to dive as a human being, with all my feelings, and I think that Yoga allows man to become part of the Cosmos and to achieve great results, because it helps from a psychological point of view, but I prefer to go down as a man, and I love the sea as an air-breathing human.

Dr. Siesjo: Can I come back to the symptoms of ascent, when Mr. Maiorca is approaching the surface. I wonder if you can notice any difference in the way you see things, in your vision.

Mr. Maiorca: Nothing at all. At the beginning of my career I used to dive without a mask, and my vision improved at depth. Then I started using a mask. During the last dives, I have not used masks anymore, but instead, I now use perforated goggles with 180 dioptric lenses, so that now I don't have to waste air in clearing the mask.

Dr. Siesjo: I wondered because one of the earlier symptoms in experimental hypoxia is blurred vision and also narrowed visual fields.

Mr. Maiorca: Absolutely not. And I know that for sure because I have to watch the divers, the SCUBA divers around me who give me signals,

for example, they tell me to take off my goggles close to the surface, because they bother me.

Dr. Lin: I'd like to know, when you are at depth, under such very high pressures, do you feel anything physically?

Mr. Maiorca: I don't have any feelings of pressure at all, but I start feeling discomfort in my ears, but you have to remember that I do not clear my ears after 50 meters.

Dr. Lanphier: Do you notice anything like nitrogen narcosis?

Mr. Maiorca: I am perfectly conscious. In fact, I am excited when I reach the record depth where I have to pick a mark showing the depth, but then I am very scared that I will not be able to make it back to the surface. May I have two minutes? Because I wish to say something. Scientists work hard everyday for us divers, as I am a diver today and I wish to thank you, and I want to give you a picture from my experience. Many years ago, I was doing a deep breath-hold dive, and the weather was bad even if the sea was calm, the sky was gray and so there was not visibility underwater. I was at depth and suddenly saw a fish, a very, very, very big fish and I was afraid and I did not know what to do. Suddenly, a ray of sun fell down from the sky into the sea and hit the big fish which exploded into a thousand small silver fish. It was not a big fish, it was a school of sardines which disappeared in all directions. This is the picture that I give to you.