

## Diving bradycardia and breath-holding time in man

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Sterba JA, Lundgren CEG. Diving bradycardia and breath-holding time in man. *Undersea Biomed Res* 1985; 12(2):139-150.—The hypothesis that the diving response, recorded as diving bradycardia during submersed breath holding in man, would enhance his breath-holding time was tested. Five certified scuba divers served as subjects. They performed breath holds of maximal duration while nonimmersed and during submersion in cool (32°C), cold (20°C), and thermoneutral (35°C) water. The mean breath-holding time and heart rate during the nonimmersed (control) condition were, respectively,  $111.2 \pm 14.1$  (SE) s and  $64.1 \pm 4.7$  (SE) beats/min, the relatively long breath-holding times being due primarily to the so-called short-term training effect. Compared to the control values the breath-holding time in 20°C water was 54.9% shorter and heart rate 25.9% lower, in 32°C water the breath-holding time was not different and heart rate was 28.1% lower, and in 35°C water the breath-holding time was longer by 25.6% while there was no difference in heart rate. In all conditions the breath-hold breaking point alveolar  $PCO_2$  was the same at about 52 mmHg. The shortening of the breath holds in cold water was ascribed to a 256% increase (over nonimmersed control) in metabolic rate as well as a respiratory drive due to stimulation of skin cold receptors. As for the prolongation of breath holds in thermoneutral water, it was hypothesized that immersion caused a delay in the build-up of chemical stimuli at the chemoreceptors.

apnea  
breath holding  
immersion  
diving bradycardia

dive response  
submersion  
alveolar gases  
drowning

The diving response has been studied widely in animals and its presence, especially as diving bradycardia, has been amply demonstrated in man (1-7). The physiology of the diving response has recently been reviewed extensively (8-10). Diving response is induced by apnea and cold water face immersion. The mechanism consists of a vagally induced bradycardia and a sympathetically controlled peripheral vasoconstriction triggered from facial cold receptors and arterial chemoreceptors. Another component of the mechanism may be information from pulmonary mechanoreceptors as lung volume is decreased in connection with a dive. The bradycardia and peripheral vasoconstriction are interpreted commonly as an oxygen-conserving response that allows the diving animal to greatly prolong its breath-holding time. The proposed

mechanism is to reduce blood flow to organs that are relatively insensitive to ischemia, such as the skin, muscles, and gastrointestinal organs. This, according to theory, conserves  $O_2$  and maintains the  $CO_2$  storage capacity of blood and lungs for use primarily by the heart and nervous system, which are particularly sensitive to hypoxia and hypercapnia.

By analogy, it has been suggested that the diving response contributed to the survival of persons who have been resuscitated after accidental submersions of up to 40 min in cold water (11). However, the diving response has not yet been investigated directly for its possible physiological significance in man.<sup>1</sup> In an attempt to do so, this study was directed at determining if the diving response, elicited by breath holding and submersion in water of appropriate temperature, would modify pulmonary gas exchange and influence resting man's breath-holding time. The parameter monitored for the presence of a diving response was heart rate.

## METHODS

Five male nonsmokers between 19 and 26 yr old and with prior experience of underwater breath-hold diving served as volunteer subjects. Before participating in the experiments they underwent a physical examination and a 12-lead EKG recording to exclude any relevant abnormalities.

The subjects wore nose-clip and swim trunks during the experiments. Submersion was performed in the upright posture in a well stirred water bath containing water at either  $35^\circ\text{C} \pm 0.5^\circ\text{C}$ , which may be considered thermoneutral (14), or  $32.0^\circ\text{C} \pm 0.5^\circ\text{C}$  (cool), or  $20.0^\circ\text{C} \pm 0.5^\circ\text{C}$  (cold). The latter temperature was chosen because it is known to induce a significant diving bradycardia in man (4, 5, 15–17). Control experiments were performed with the subject seated upright in air at  $28^\circ\text{C}$  which is thermoneutral (18–20).

Oxygen uptake ( $\dot{V}O_2$ ) and  $CO_2$  elimination ( $\dot{V}CO_2$ ) were recorded with standard open circuit Douglas bag techniques, using a nose-clip, mouthpiece, and appropriate breathing valves. Gas analyses were made with a mass spectrometer (Model MGA 1100, Perkin Elmer, Pomona, CA), with an accuracy of  $\pm 0.05\%$  of  $O_2$  and  $CO_2$ , respectively. End-tidal gas composition was obtained by sampling from the mouthpiece at a point 3 cm from the mouth. Estimates of alveolar gas composition were obtained before and after breath holdings. For this purpose, gas was sampled during an expiration to residual volume (RV) immediately before inhaling to breath-holding lung volume and during the expiration to RV done at the termination of a breath hold. Readings from strip-chart recordings of the mass spectrometer output were taken on the alveolar plateau at a point 50% through expiration time. These readings were used for calculation of alveolar gas composition, applying a correction which assumed that alveolar air pressure was increased in proportion to water pressure on the chest. The water depth, set at 60–70 cm, was measured to the manubrium of the sternum. This reference point was preferred for its convenience in contrast to the theoretically more appropriate chest pressure centroid (cf. 21) which would have been relatively cumbersome to determine in each subject. The underestimation of pulmonary  $O_2$  and

<sup>1</sup>Since our initial short report on this work (12), another study (13) of breath-hold duration during cold water immersion has been published; cf. "Discussion."

CO<sub>2</sub> tensions by using the lesser depth of the manubrium should not exceed 1.5 mmHg.

Breath holds were initiated by first exhaling rapidly to RV and then inhaling, from a spirometer, a volume of fresh air corresponding to 85% of the subject's vital capacity as measured previously in the nonimmersed condition. This is also the volume used by the professional diving women of Korea and Japan at the beginning of a breath-hold dive (22). In view of the standardized inhalation and the fact that RV does not change substantially during immersion (23, 24), it is reasonable to consider starting lung volumes as having been equal in all experimental runs.

Because maximal breath-holding time may be sensitive to various nonspecific influences, a number of procedures were observed to standardize the experimental conditions. Thus, the subject's were instructed not to consume caffeine- or alcohol-containing beverages within 12 h or to take any medication within a week before an experiment. Certain kinds of muscular activity including those involved in the Mueller and Valsalva maneuvers have been shown to lengthen breath-holding time (25), and attention was therefore given to the need for the subject to assume a standardized relaxed position during breath holds. The so-called short-term training effect may gradually increase the breath-holding time by up to 35% when a series of appropriately spaced breath holds are performed (25-27). Therefore, all experiments started with a series of 3-5 breath holds which were not used in the final data analysis. Between breath holds, 4.0 min rest periods were observed. Such rest periods will not abolish the short-term training effect (26) and yet are sufficient to allow good reproducibility of maximal breath-holding times. The subjects were instructed to do their best to achieve the longest possible breath-holding time in each attempt but they were not coached or allowed any clues as to the duration of their breath holds. They would not let any air escape before reaching the breaking point at which regular rhythmic breathing was resumed. Breath-holding time, defined as the time from cessation of respiratory air flow after the standardized prebreath-hold inhalation to beginning of air flow at the breaking point, was measured by a person closely observing the subject and using a stopwatch.

The heart rate was derived from R-R intervals of four consecutive beats read off a standard 3-lead EKG 30 s before and 30 s into the breath hold.

The experimental procedure consisted of first securing the EKG electrodes covered with water-impermeable surgical tape to the subject who was sitting in 28°C air. After this, the subject rested for 20 min. He then put on a nose-clip and connected himself to the mouthpiece assembly, and expired gas was collected during a 10-min period for measurements of  $\dot{V}O_2$  and  $\dot{V}CO_2$ . The experimenter then initiated the breath hold by telling the subject to expire to RV and, after switching to connect the mouthpiece with the spirometer, to inhale the premeasured air volume corresponding to 85% of his VC (start of stopwatch). Sampling of end expiratory gas for mass spectrometry was done during the exhalation to RV. Upon reaching the breaking point, the subject again expired to RV and the exhaled gas was sampled, after which the subject resumed normal breathing. After a 4-min rest period the procedure was repeated. Enough breath holds were performed so that the short-term training effect would be fully developed, with at least four consecutive breath holds showing a plateau in terms of breath-holding time from which the mean BH<sub>max</sub> would be derived. Within the span of a resting period the subject then transferred to the submersion tank containing water at the appropriate temperature. A diver's weight belt was used to make the

subject slightly negatively buoyant. During the resting periods in the water, the subject, standing on a step on the bottom, assumed an erect body posture with the water level at the neck. After inhalation for a breath hold, he stepped off the step so as to allow the water to completely cover his head. After a sufficient number of breath holds had been performed,  $\dot{V}O_2$  and  $\dot{V}CO_2$  were recorded while the subject remained immersed in the water to his neck. In a few experiments these measurements of gas exchange were omitted and the subject was instead, within the span of a resting period, transferred back to the nonimmersed situation in which, after being thoroughly dried off, he performed a final control series of breath holds. In these cases, breath-holding times after immersion in 35°C water were the same as before immersion, whereas after cold water immersion breath holds varied more in duration probably due to fatigue and lasting cooling effects.

The subjects served as their own controls. Paired comparisons were subjected to Student's *t*-test for statistical evaluation.

## RESULTS

Maximal breath-holding times ( $BH_{max}$  time) recorded in one subject on 6 different days are shown in Fig. 1. Each day a series of breath holds was first made sitting nonimmersed in air of neutral temperature (28°C). The subject then transferred to the submersion tank where a second series of breath holds were performed. The water temperature was different on each day, viz, 15°C, 20°C, 28°C, 32°C, or 35°C (35°C was used in two different series in this subject and water at 15°C and 28°C was only

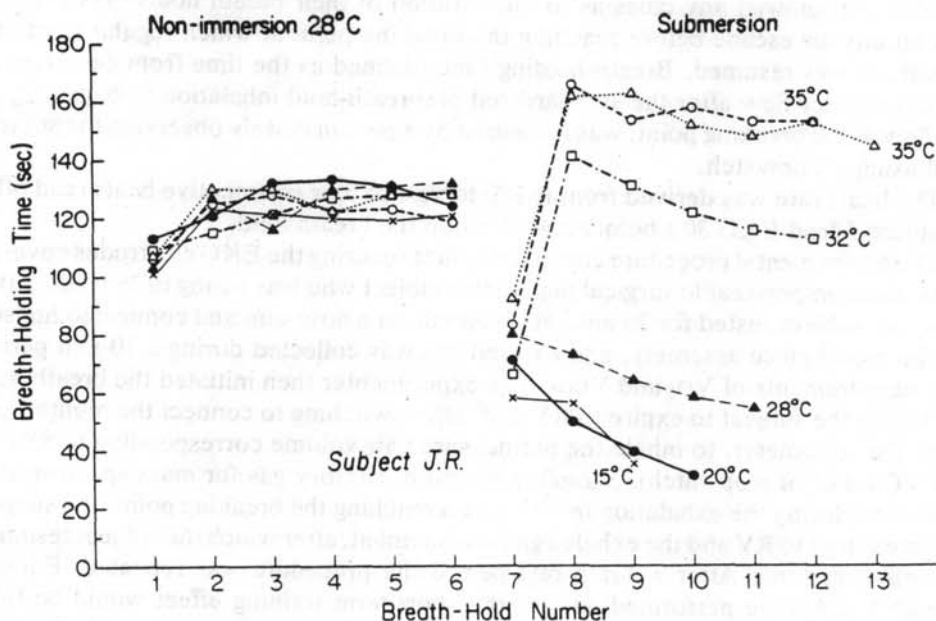


Fig. 1. Maximal breath-holding time in series of up to 13 breath holds recorded on 6 different days in a resting subject during nonimmersion in an ambient air temperature of 28°C followed by submersion in water at temperatures between 15°C and 35°C (two series of breath holds). Resting intervals (immersion to the neck) between breath holds lasted 4.0 min.

used in a few experiments in other subjects). The results shown in Fig. 1 are representative for all the subjects studied. The salient observations are as follows: A short-term training effect occurred with the first 2 to 3 breath holds and the  $BH_{max}$  time reached a plateau that was preserved throughout the nonimmersed control breath holds.

Reproducibility of control  $BH_{max}$  times from day to day was excellent—in this subject the range was about  $\pm 6\%$ . The duration of the first breath hold during submersion was considerably less in all water temperatures than that of the control breath holds, the reduction being more pronounced the colder the water. With the second breath holds in the water (breath hold No. 8 in each series), marked separation in the breath-hold duration between waters of different temperatures became evident. Cool water ( $28^{\circ}\text{C}$ ), and even more so cold water ( $20^{\circ}\text{C}$  and  $15^{\circ}\text{C}$ ), led to further gradual shortening of subsequent breath holds. Water at  $32^{\circ}\text{C}$  allowed breath-holding times which were comparable with the nonimmersed control breath holds. Remarkably, the breath-hold durations in thermoneutral water ( $35^{\circ}\text{C}$ ) were substantially prolonged.

The results on breath-holding time obtained in all five subjects are summarized in Fig. 2. Two complete experimental series at each water temperature were run (on two separate days) in each subject. Typically, only 3 or 4 breath holds were performed

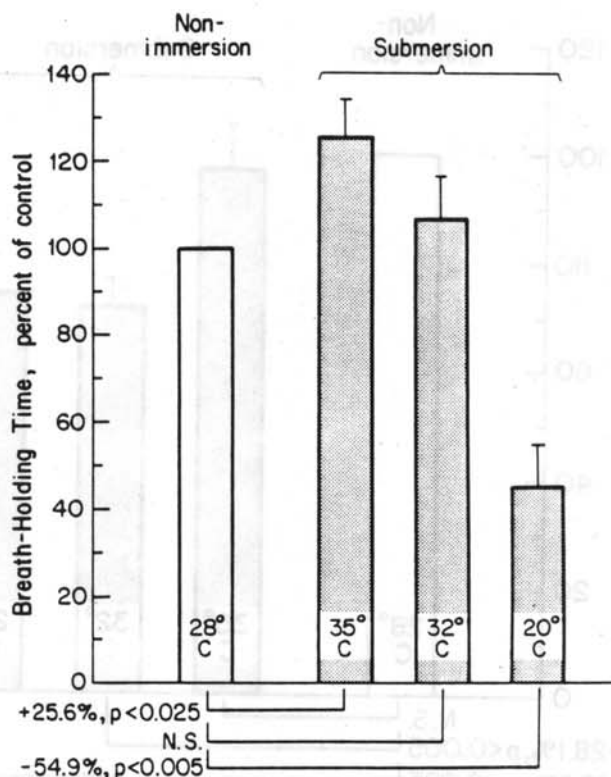


Fig. 2. Maximal breath-holding times during submersion in water at  $35^{\circ}\text{C}$ ,  $32^{\circ}\text{C}$ , and  $20^{\circ}\text{C}$  expressed as percent of breath-holding time under control conditions, i.e., nonimmersion, ambient air temperatures  $28^{\circ}\text{C}$ . Values are means  $\pm$  SE of two series of breath holds in each of 5 subjects; statistics were made by paired comparisons and *t*-test.



in water at 20°C due to cold discomfort, whereas 5 to 7 breath holds were produced in water at 32°C and 35°C. The mean breath-holding time during nonimmersion was  $111.2 \pm 14.1$  (SE) s. The average breath-holding time in each water temperature was computed from the 3–5 breath holds showing either a relative plateau or, in the cold water, a steady slow rate of change in duration (cf. Fig. 1). These values were normalized to the mean duration of the nonimmersed breath holds for each subject. The results of such paired comparisons were then pooled for the presentation in Fig. 2.

Overall, the  $BH_{max}$  times in the thermoneutral water (35°C) were 25.6% longer than nonimmersed breath holds. The duration of the breath holds in cool water (32°C) was not different from the control breath holds whereas those performed in cold water (20°C) were shorter by 54.9%.

Heart rate was obtained from recordings 30 s into breath holds when typically a stable heart rate had established itself. In Fig. 3 results are expressed as means of individual results normalized to observations in breath holds during nonimmersion. During breath holding in thermoneutral water the heart rate was unchanged compared to the control situation whereas in cool (32°C) and cold (20°C) water the heart rate

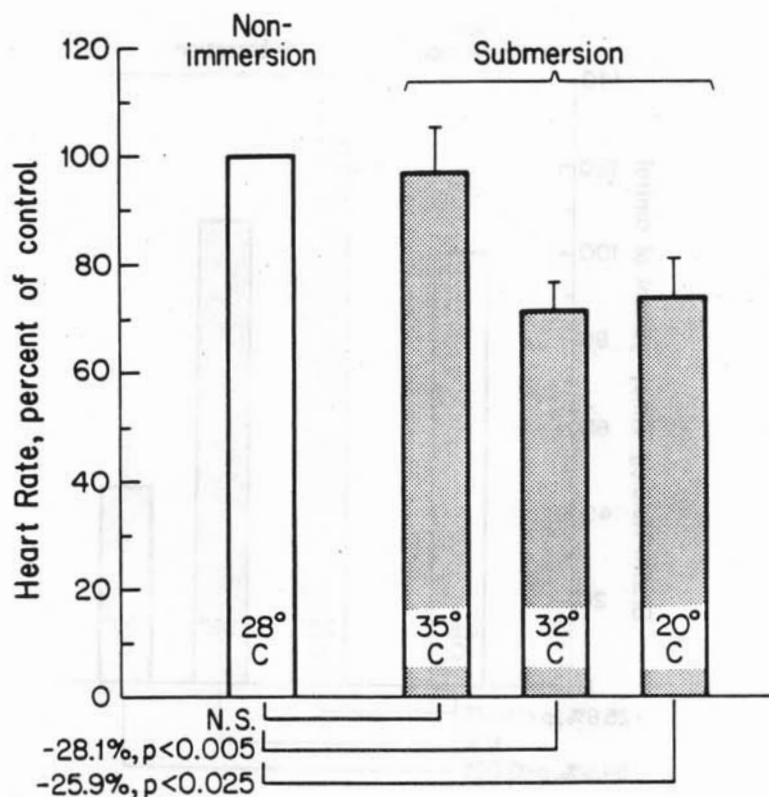


Fig. 3. Heart rates 30 s into breath holds during submersion in water at 35°C, 32°C, and 20°C expressed as percent of heart rate during breath holding under control conditions, i.e., nonimmersion, ambient air temperatures 28°C. Values are means  $\pm$  SE of two series of breath holds in each of 5 subjects;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

was, respectively, 28.1% and 25.9% lower than the average of  $64.1 \pm 4.7$  (SE) beats/min recorded in the control situation.

The mean values of alveolar carbon dioxide tensions ( $P_{ACO_2}$ ) immediately before breath holds and at the breath-hold breaking point are shown in Fig. 4. The prebreath-hold  $P_{ACO_2}$  levels were not significantly different from the nonimmersed control level of about 40 mmHg when the subjects were immersed in thermoneutral (35°C) water but there was a depression by 3.1 mmHg of  $P_{ACO_2}$  in the cool water (32°C) and by 3.8 mmHg in the cold (20°C) water.

Regardless of water temperature, all submersed breath holds terminated with the same  $P_{ACO_2}$  levels as did the nonimmersed breath holds, the average value being about 52 mmHg.

Figure 5 illustrates the results of the measurements of  $\dot{V}O_2$  and  $\dot{V}CO_2$  during resting breathing in the nonimmersed situation and after completing each series of submersed breath holds in water at, respectively, 35°C, 32°C, and 20°C. Given the carefully controlled resting conditions of the subjects, the mean  $\dot{V}O_2$  of  $0.26 \pm 0.02$  l (STPD)  $\cdot \text{min}^{-1}$  and  $\dot{V}CO_2$  of  $0.22 \pm 0.2$  l(STPD)  $\cdot \text{min}^{-1}$  in the nonsubmersed as well as the resulting respiratory exchange ratio (R) of 0.84 are reasonable. These gas exchange levels were exceeded by 256% ( $\dot{V}O_2$ ) and 277% ( $\dot{V}CO_2$ ), respectively, during immersion in water at 20°C and R was 0.89 on the average. Compared to nonimmersion there was a small increase (16%) in  $\dot{V}O_2$  in 35°C water but no difference in gas exchange in water at 32°C.

## DISCUSSION

The present study was designed to determine whether the diving response induced by breath holding and submersion in man would enhance his breath-holding time

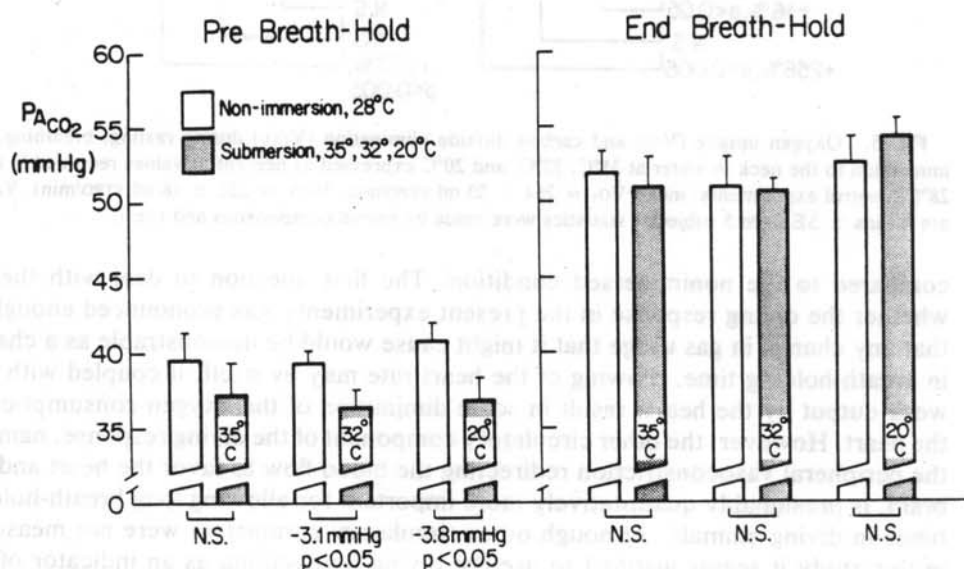


Fig. 4. Alveolar carbon dioxide tensions ( $P_{ACO_2}$ ) prebreath hold and at end of breath holds of maximal duration during nonimmersion in air at 28°C and during submersion in water at 35°C, 32°C, and 20°C. Values are means  $\pm$  SE of two series of breath holds in each of 5 subjects; statistics were made by paired comparisons and *t*-test.

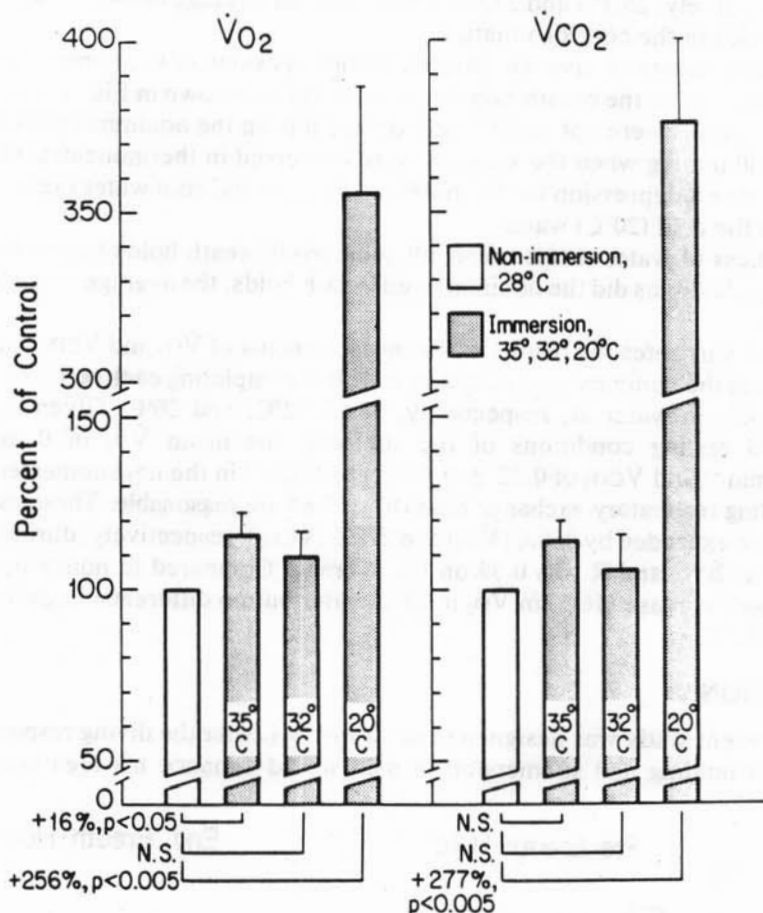


Fig. 5. Oxygen uptake ( $\dot{V}O_2$ ) and carbon dioxide elimination ( $\dot{V}CO_2$ ) during resting, breathing, and immersion to the neck in water at 35°C, 32°C, and 20°C expressed as percent of values recorded in air at 28°C (control experiments: mean  $\dot{V}O_2 = 264 \pm 20$  ml STPD/min,  $\dot{V}CO_2 = 222 \pm 18$  ml STPD/min). Values are means  $\pm$  SE from 5 subjects; statistics were made by paired comparisons and *t*-test.

compared to the nonimmersed condition. The first question to deal with then is whether the diving response in the present experiments was pronounced enough so that any change in gas usage that it might cause would be demonstrable as a change in breath-holding time. Slowing of the heart rate may by itself, if coupled with less work output by the heart, result in some diminution of the oxygen consumption of the heart. However, the other circulatory component of the diving response, namely, the peripheral vasoconstriction redirecting the blood flow to favor the heart and the brain, is presumably quantitatively more important for allowing long breath-holding times in diving animals. Although other circulatory parameters were not measured in this study it seems justified to use the diving bradycardia as an indicator of the presence of a more generalized circulatory diving response. It has been shown repeatedly in man that diving bradycardia initiated by breath holding and face immersion or whole body submersion is accompanied by peripheral vasoconstriction in the limbs (28–31). Indeed, the relative reduction in limb blood flow in several of these



studies was considerably more pronounced than the relative reduction in heart rate, viz -51% (forearm) vs. -15% (HR) (31), -28% (forearm) and -47% (calf) vs. -10% [HR; deduced from absolute values in (30)] and -50% (forearm) vs. -23% (HR) (29). A marked increase in total peripheral resistance coincident with the diving bradycardia in man has also been reported (32). In view of the consistency of the breath-holding times recorded in our subjects it would seem that even a modest diving response, if of consequence for the subject's breath-holding capacity, would have been noticeable as a prolongation of the breath-holding time.

The present experiments showed no prolongation of breath-holding time as diving bradycardia was elicited. The breath-holding time was unaffected in the presence of a heart rate reduction of about 28% during breath holding in cool water (32°C) and it was shortened by as much as 55% during breath holding in cold water (20°C) which reduced heart rate by about 26%. These observations which have been briefly reported earlier (12) have been corroborated by Hayward et al. (13) who extended the observations to water temperatures down to 0°C. They found that breath-holding time in a single first attempt (in each of 160 subjects) decreased with lowering of the water temperature according to the linear regression: breath-holding time =  $15.01 + 0.92 \times \text{water temp } (^{\circ}\text{C})$ . This occurred in combination with a suppression of the heart rate to an average of between 50–60 beats/min from a presubmersion rate which, due to excitement, averaged about 102 beats/min.

Shortening of the breath-holding time in the cold water experiments was probably partly due to stimulation of skin cold receptors, a mechanism known to exert a powerful respiratory drive (33, 34). This respiratory stimulation expressed itself as a slight prebreath-hold hypocapnea in water at 32°C and 20°C. Cold, acting to shorten the breath hold, is the explanation favored also by Hayward et al. (13). A likely additional factor reducing the breath-holding time in water at 20°C in the present study was a substantial increase (about 270% at the end of the immersion period) in metabolism causing a more rapid build-up of respiratory stimuli. In fact, Lin et al. (35) have shown an inverse relationship between breath-hold duration and  $\dot{V}\text{O}_2$ . In harmony with this relationship is the present observation that the breaking point  $\text{P}_{\text{ACO}_2}$  levels were the same irrespective of breath-holding times (and water temperatures) and also closely agreed with those reached in the nonimmersed control experiments. Put differently, it appears that the differences in breath-holding times that we recorded in water of different temperatures were not due to differences in  $\text{CO}_2$  sensitivity of the chemoreceptors.

The remarkable prolongation of the breath-holding time in water at 35°C by about 26% occurred in the absence of any prebreath-hold hyperventilation or diving bradycardia. The second-attempt breath holds performed by the subjects in the study of Hayward et al. (13) after a brief habituation to the various water temperatures and hyperventilation also appear to have shown a longer breath-hold duration in water at 35°C than during presubmersion. However, this extension could depend on several mechanisms such as the hyperventilation, the short-term training effect (25–27), and possible variation in breath-hold starting lung volume. In the present experiments these factors were controlled. Furthermore, the short-term training effect in particular, as well as some earlier experience of breath-hold diving in our subjects, who all were certified scuba divers, may explain the relatively long duration of their control breath holds (111 s) compared to that of the subjects in the study of Hayward et al.

(13) who were not selected in this regard and who performed only one control breath hold.

As in the cold water breath holds, the breaking point  $P_{ACO_2}$  in water at 35°C was the same as in the nonimmersed control breath holds, and the postbreath-hold measurements of  $\dot{V}O_2$  indicated a small (16%) increase in metabolism during submersion. This probably justifies the notion that the prolongation of breath holds in 35°C was not due to a reduction in metabolism. In view of these considerations, one may hypothesize that, to the extent that metabolically generated respiratory stimuli brought about the breaking point of breath holds, these stimuli were somehow delayed in reaching the chemoreceptors when our subjects performed breath holding in water at 35°C. Alternatively, a slowing of metabolism during the breath hold could delay the build-up of respiratory stimulation, or metabolites could be withheld from entering the circulation. As reviewed by Elsner and Gooden (8) there are experimental observations to indicate that the aerobic metabolism in certain parts of the animal body may be subject to control by the regional circulation. Furthermore, the release of lactic acid from the skeletal muscles into the circulation of the seal is delayed until after the dive (8, 36). However, such mechanisms are unlikely in the present breath-hold experiments in which submersion in water at 35°C did not cause any diving bradycardia.

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Sterba JA, Lundgren CEG. Bradycardie de plongée et durée de l'apnée volontaire chez l'homme. *Undersea Biomed Res* 1985; 12(2):139-150.—L'hypothèse que la réponse à la plongée, enregistrée comme une bradycardie de plongée durant une apnée volontaire en immersion, prolongerait le temps de l'apnée volontaire fut vérifiée chez l'homme. Cinq plongeurs SCUBA certifiés servirent de sujets. Ils exécutèrent des apnées volontaires de durée maximale sans être immergés ainsi que pendant l'immersion en eau tiède (32°C), froide (20°C) et thermoneutre (35°C). Le temps d'apnée volontaire et la fréquence cardiaque durant la condition non-immergée (témoin) furent en moyenne de  $111.2 \pm 14.1$  (SE) s et  $64.1 \pm 4.7$  (SE) battements/min, respectivement; les durées relativement longues de l'apnée volontaire étant dues principalement à l'effet dit d'entraînement à cours terme. Comparé aux valeurs témoins, le temps d'apnée volontaire dans l'eau à 20°C fut 54.9% plus court et la fréquence cardiaque 25.9% plus lente, dans l'eau à 32°C le temps d'apnée volontaire ne fut pas différent et la fréquence cardiaque fut 25.9% plus lente, et dans l'eau à 35°C le temps d'apnée volontaire fut prolongé de 25.6% tandis qu'il n'y eut pas de différence dans la fréquence cardiaque. Dans toutes les conditions, la valeur critique de la  $PCO_2$  alvéolaire en apnée volontaire était la même et d'environ 52 mmHg. Le raccourcissement de la durée des apnées volontaires en eau froide fut attribué à une augmentation de 256% (comparé aux témoins nonimmergés) dans le taux métabolique ainsi qu'à l'envie de respirer due à la stimulation des récepteurs cutanés au froid. Quant à la prolongation des apnées volontaires en eau thermoneutre, il fut proposé comme hypothèse que l'immersion produisit un délai dans l'accumulation de stimulus chimiques au niveau des chemorécepteurs.

apnée  
apnée volontaire

réponse à la plongée  
submersion

immersion  
bradycardie de plongée

gaz alvéolaires  
noyade

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