

Cardiovascular changes during deep breath-hold dives in a pressure chamber

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²Department of Anesthesia, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts 02115; ³Department of Physiology, Centre Medical Universitaire, Universite de Geneve, 1211 Geneva 4, Switzerland; and ⁴Medical Service, Department of Veterans Affairs Medical Center, Buffalo, New York 14214

Ferrigno, Massimo, Guido Ferretti, Avery Ellis, Dan Warkander, Mario Costa, Paolo Cerretelli, and Claes E. G. Lundgren. Cardiovascular changes during deep breath-hold dives in a pressure chamber. *J. Appl. Physiol.* 83(4): 1282–1290, 1997.—Electrocardiogram, cardiac output, and blood lactate accumulation were recorded in three elite breath-hold divers diving to 40–55 m in a pressure chamber in thermoneutral (35°C) or cool (25°C) water. In two of the divers, invasive recordings of arterial blood pressure were also obtained during dives to 50 m in cool water. Bradycardia during the dives was more pronounced and developed more rapidly in the cool water, with heart rates dropping to 20–30 beats/min. Arrhythmias occurred, particularly during the dives in cool water, when they were often more frequent than sinus beats. Because of bradycardia, cardiac output decreased during the dives, especially in cool water (to <3 l/min in 2 of the divers). Arterial blood pressure increased dramatically, reaching values as high as 280/200 and 290/150 mmHg in the two divers, respectively. This hypertension was secondary to peripheral vasoconstriction, which also led to anaerobic metabolism, reflected in increased blood lactate concentration. The diving response of these divers resembles the one described for diving animals, although the presence of arrhythmias and large increases in blood pressure indicate a less perfect adaptation in humans.

apnea; arrhythmias; bradycardia; cardiac output; arterial blood pressure; lactate

THE CLASSIC DIVING RESPONSE in diving animals consists mainly of bradycardia, decreased cardiac output (CO), and peripheral vasoconstriction (for a review, see Ref. 9). Blood flow is redistributed preferentially to vital organs, and lactate accumulates in unperfused muscles. A diving response, although attenuated or modified, has also been described in humans during diving simulated by apneic face immersion at the surface (for a review, see Ref. 31).

Most of the studies in humans during actual breath-hold dives have focused on the diving bradycardia. Electrocardiographic (ECG) recordings have been performed during dives to modest depths (10–20 m), either in a pool or in a pressure chamber (e.g., Ref. 14) or at sea (e.g., Ref. 29). A few suboptimal ECG recordings were also obtained at sea during deep breath-hold dives performed by elite sport divers (8, 13, 28), who have been able to reach depths exceeding 100 m (23). Whereas most of these studies have confirmed the presence of diving bradycardia in human divers, no information on

the hemodynamic aspects of breath-hold dives in humans is available, with the exception of one study in which CO was found to increase (in contrast to the classic diving response) during dives to 20 m (14). These dives were performed by untrained subjects in thermoneutral water in a pressure chamber. On the other hand, high blood lactate concentration ($[La]_b$) values were recently observed in three elite divers at the end of breath-hold dives down to 65 m at sea, even though the rate of energy expenditure during those dives was only slightly higher than at rest (11). This evidence of anaerobic metabolism, in the absence of an elevated metabolism, was attributed to a marked peripheral vasoconstriction, probably resulting in a decreased blood flow and oxygen delivery to muscles. This study was conducted to obtain a more complete picture of the diving response in well-trained breath-hold divers during simulated dives, which closely resembled dives performed at sea.

ECG recordings, measurements of CO, arterial blood pressure (BP), and $[La]_b$ accumulation were obtained during several breath-hold dives to 40–55 m performed in a pressure chamber by three elite divers, from whom our group had earlier obtained ECG recordings during dives to 45–65 m at sea (13). The dives were simulated in the submersed condition, thus closely resembling diving in the ocean, while allowing optimal physiological monitoring. The water temperature was either thermoneutral (35°C) or cool (25°C). The cool water is a stronger stimulus for the dive response (6), and it was used to more closely simulate dives at sea. Deep breath-hold dives are typically performed by using a weight that pulls the diver down during descent and an inflatable flotation device that brings him or her to the surface (23). Thus exertion and oxygen consumption are minimized. To simulate this condition, our subjects were resting during the dives in the chamber.

METHODS

Subjects. The experimental protocol had been approved by the Institutional Review Board for Human Subjects in Research of the university, and the three subjects (*EM*, a man, *PM* and *RM*, women) had given their informed consent. Their ages were 59 (*EM*), 32 (*PM*), and 30 yr (*RM*). Physical examinations including ECG showed the subjects to be in excellent health including absence of signs and symptoms of any heart disease. At the time of this study, their personal depth records established in the sea were 101 m for *EM*, 70 m

for *PM*, and 80 m for *RM*. They typically trained by performing a couple of deep dives a week during the summer, frequent apneic swimming in a pool, and bouts of apneic exercise (stair climbing) several times a week throughout the year.

Dives. The experimental dives were performed in the summer (after training) in the wet compartment of the hyperbaric chamber in the Center for Research and Education in Special Environments. The chamber compartment in which the dives were performed was filled with water, the level of which was roughly at the waist of the standing subject. To dive, the subject, who was wearing a weight belt, assumed a semiprone head-up position at $\sim 30^\circ$ to the horizontal plane, with the head just below the surface. Simulation of the appropriate depth was achieved by letting compressed air into the dry section of the chamber, the pressure being transmitted through the air-water interface. Decompression was performed by bleeding off gas from the chamber. The rates of compression and decompression were monitored with an electrical pressure transducer and an *X-Y* recorder. The compressions and decompressions were controlled manually so as to reproduce a dive profile drawn in advance on the *X-Y* recorder. This allowed descents and ascents that were essentially linear at 1.0 m/s, except for stops, typically lasting ~ 3 s at 30 and 40 m, to facilitate ear clearing. The time spent at maximal depth was 15 s. After a few initial practice dives to 20 m (lasting ~ 1 min) in thermoneutral water, the depth was increased to 40, 45, 50, or 55 m, depending on the subject's preference. For safety reasons, a decision had been made to limit the dives to depths no greater than 70% of the maximal depth that had been reached by each subject in the sea. A total of 25 dives was performed by the three divers (10 by *EM*, 7 by *RM*, and 8 by *PM*). Nineteen dives were to 40 m or deeper depths. The minimum length of time separating two dives was 30 min.

The subject's face could be observed through a chamber porthole and also on closed-circuit television. As frequently is the case in their sea dives, the subjects did not wear face masks. Hand signals were used by the subject to make the chamber operator stop and resume compression, as needed for ear-clearing purposes. In addition, an acoustic emergency signal outside the chamber could be activated by the subject by pulling a string. The water temperature was either 35°C , i.e., thermoneutral (7), or 25°C , i.e., cool. The subjects wore light swimsuits. During the first 3 days, the dives were performed in thermoneutral water to allow the divers to familiarize themselves with the chamber environment under comfortable thermal conditions. On the following 6 days, water temperature was either thermoneutral or cool, and, on a given day, each subject performed dives in water at only one temperature. Of the 25 dives performed, 17 were in thermoneutral water.

Measurements. All recorded data were stored on magnetic tape as well as displayed in real time on strip-chart recorders. The magnetic tape also had a voice track for timing purposes.

Heart rate (HR) and rhythm. ECG recordings were obtained from lead II with a technique earlier established in this laboratory (13). HRs were calculated from four R-R intervals. Arrhythmias were classified into one of two categories: 1) premature beats thought to arise from a reentry mechanism and consisting of premature atrial and ventricular complexes and 2) inhibitory arrhythmias arising from increased vagal tone and consisting of sinus arrest and/or atrioventricular block with junctional and/or ventricular escape beats.

CO. Stroke volume (SV) was measured with an impedance cardiograph (model 400, Instrumentation for Medicine, Greenwich, CT) by using two Mylar-tape electrodes around the neck

and two around the lower thorax close to the xiphoid process. CO was calculated from the product of HR and SV.

BP. Percutaneous cannulation of the right radial artery with a 20-gauge catheter was performed with aseptic technique in two of the three divers (*EM* and *RM*) in preparation for dives to 50 m in the cool water. The catheter was connected to a sterile, disposable transducer (CDX III, COBE Cardiovascular Laboratories, Arvada, CO). The cannulation site was covered with spray-on surgical transparent dressing tape. The system had been tested in separate (unmanned) compressions for absence of pressure and temperature biases. The transducer was placed at the level of the heart when the subject was standing with the water roughly at his or her waist. BP recordings obtained during submersion were corrected for the height of the catheter fluid column between the transducer and the water level. There were no complications related to arterial cannulations.

$[\text{La}]_b$. $[\text{La}]_b$ was measured by an enzymatic method on 1-ml samples of blood taken from an antecubital vein, with the exception of two samples from an arterial line (see *BP* above). Samples were obtained before the dive and at 1, 4, and 7 min during recovery after the dive. The net $[\text{La}]_b$ accumulation during the dive was calculated as the difference between the peak value during recovery and the resting value before the dive. Data were obtained twice on each subject, for a total of six dives.

Procedures. After being instrumented, the diver entered the pressure chamber and assumed a semiprone head-out position in the wet compartment. After 5 min of rest, control recordings were obtained. Subsequently, the subject stood erect with the water level at the waist and began hyperventilation according to his or her personal habit. Typically, the hyperventilation lasted 5 min and consisted of ~ 4 –5 breaths/min at 80–90% of vital capacity. At the end of the hyperventilation period, the subject made a deep inhalation and promptly assumed the semiprone position underwater, whereupon compression was started. The descent followed the predrawn profile, with occasional brief stops for ear clearing, as mentioned earlier in *Dives*. After 15 s at the predetermined maximal depth, decompression was performed. As the chamber pressure equalized with ambient pressure, the subject lifted his or her head above water and resumed breathing. Postdive recordings from the subject, now resting in the semiprone head-out position, were obtained continuously for 5 min. In the two dives performed with an arterial catheter, the subject continued to hold his or her breath after surfacing (33 s for *EM* and 47 s for *RM*) until an arterial sample had been drawn.

RESULTS

Because only three subjects participated in this study, no attempts at statistical treatment of the results were made. Because the cardiovascular parameters measured during the dives showed a consistent pattern of changes, data from a few representative dives by each of the three divers are shown in Figs. 1–4. HR values, frequencies of arrhythmias, and CO values were averaged for each 10 m of change in depth during descent and ascent, as well as for the entire bottom stay in each dive.

HR. For calculation of HR, both sinus beats and arrhythmic beats were counted. After an initial tachycardia, bradycardia was observed during the dives, and it was more pronounced and developed more rapidly in the cool water, with HR values of 20–30 beats/min at or

near the bottom (Fig. 1). The longest R-R intervals were also recorded during the dives in 25°C water (Fig. 2): 7.2 s for *PM* (at 30 m during descent), 4.6 s for *EM* (at 25 m during descent), and 2.5 s for *RM* (at 25 m during ascent), corresponding to instantaneous HR values of 8, 13, and 24 beats/min, respectively. During the dives in thermoneutral water, the longest R-R intervals were 1.7 s for *PM* (at 10 m during ascent), 3.8 s for *EM* (at 40 m during ascent), and 1.8 s for *RM* (at 10 m during ascent). The corresponding instantaneous HR values were 35, 16, and 33 beats/min, respectively.

Heart rhythm. In both *EM* and *PM*, more arrhythmias, with an earlier onset, were observed in the dives in cool-water than in the thermoneutral-water dives. Also, in *RM*, more arrhythmias were present in the cool-water dives, but they tended to start later, i.e., not until the bottom had been reached. Often, during the dives in cool water, arrhythmias were more frequent than sinus beats. As can be seen in Fig. 3, no predominance of any one type of dysrhythmia was noted. Typically, the arrhythmias continued for 10–20 s during the recovery period. No arrhythmias were noted during control measurements.

CO. Because SV did not change appreciably during the dives, the variations in CO were mostly due to changes in HR. As a consequence, at the beginning of the dive and early during descent, CO was higher than its control value (Fig. 4). Then, it decreased to control levels or, in the cool water, to even lower levels. Thus, as shown for *PM* and *RM* in the course of dives in cool water, their CO fell to <3 l/min compared with control levels of ~6.4 and 8.8 l/min, respectively.

BP. The BP recordings obtained on *subjects EM* and *RM* during a dive to 50 m in 25°C water are shown in Fig. 5, together with the ECG and the depth-time profiles.

The sphygmomanometric BP of *RM* during the pre-dive physical examination was 115/75 mmHg. Her BP (invasive measurement) while standing in the cool water to the waist was 150/75 mmHg. During the hyperventilation, immediately before the dive, it was ~185/120 mmHg (Fig. 5B). About 5 s after the beginning of breath holding and submersion, and coincident with the beginning of compression, her BP rose within 15 s to a maximum of ~280/200 mmHg. A slow decrease then followed to a minimum of ~150/50 mmHg, which

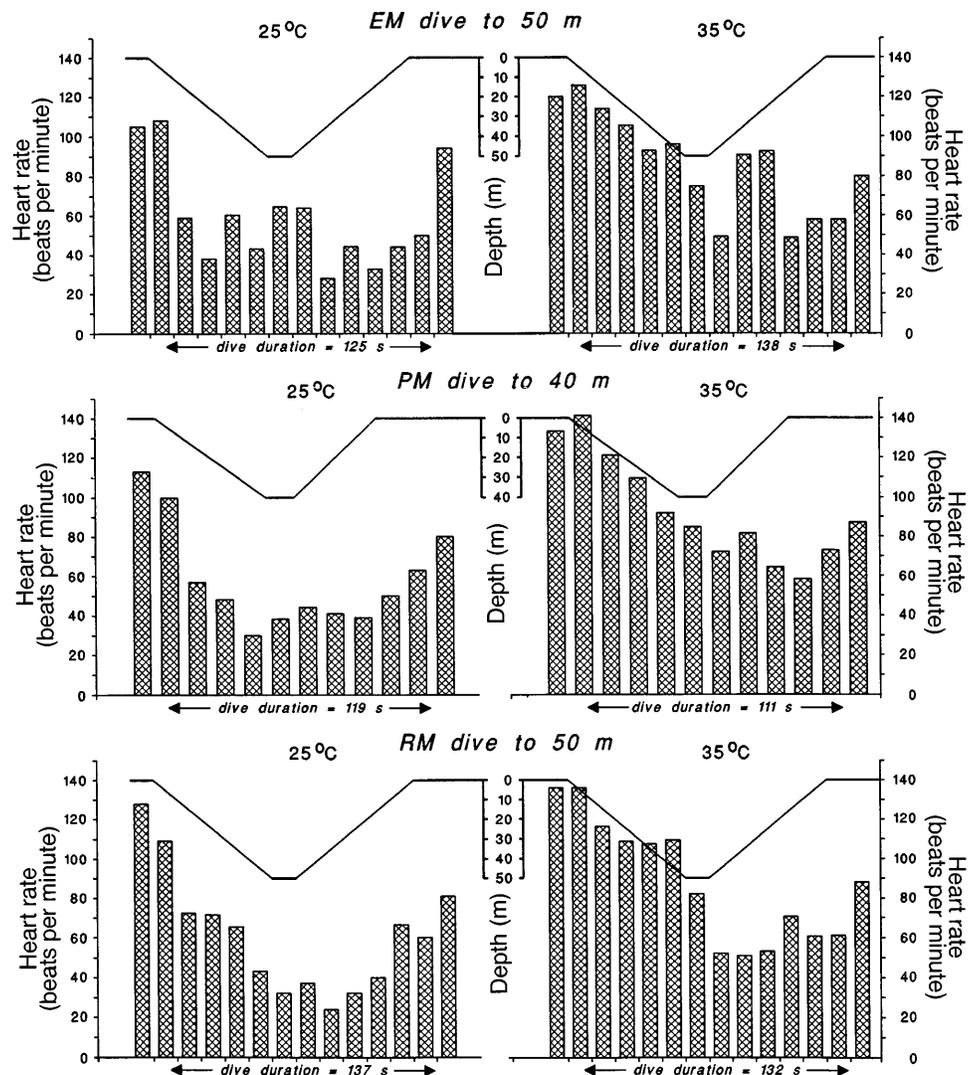


Fig. 1. Heart rate and depth vs. time of breath-hold dives to 50 m by *EM* (top) and *RM* (bottom) and to 40 m by *PM* (middle). Measurements before and after dives were averaged over 10-s intervals. Water temperature was either thermoneutral (35°C) or cool (25°C).

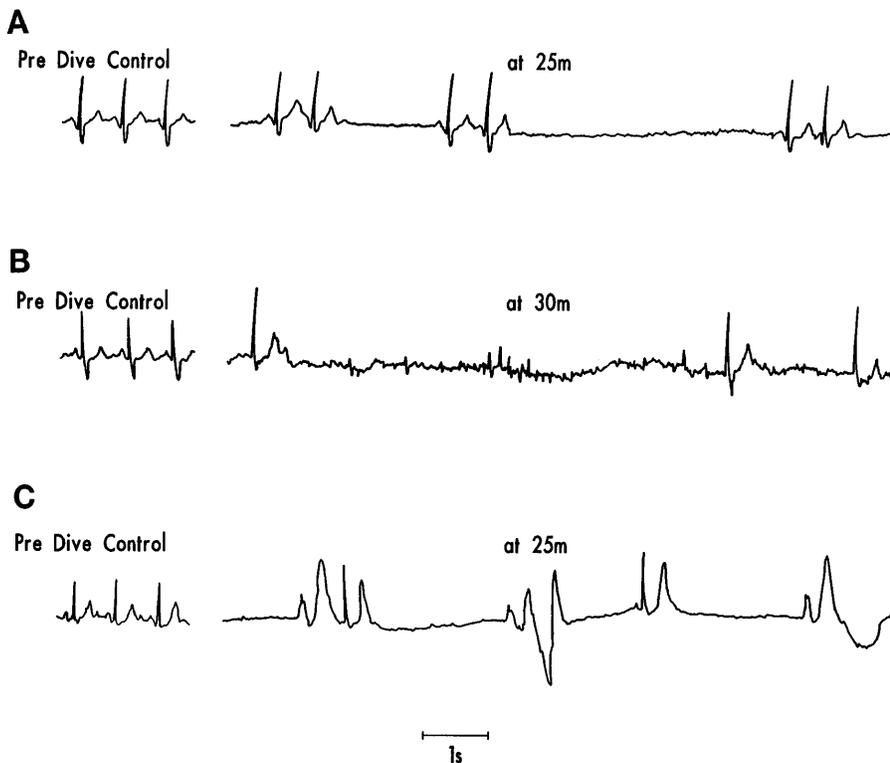


Fig. 2. Electrocardiographic tracings showing longest R-R intervals during breath-hold dives to 50 m by *EM* (A) and *RM* (C) and to 40 m by *PM* (B). Water temperature in these dives was cool (25°C).

occurred early during ascent. Then, her BP started to rise gradually, and, during the 47-s-long continued breath hold at the surface, her BP reached 240/115 mmHg. Thereafter, it decreased slowly to 165/95 mmHg in ~2 min.

A similar pattern was observed in *EM* (Fig. 5A), whose BP during physical examination was 145/90 mmHg. After he entered the cool water, it was 160/80 mmHg, and it did not change appreciably during pre-dive hyperventilation. During compression, his BP showed variations due to an irregular heart beat but reached a maximum, sustained over many beats, of 290/150 mmHg, with occasional systolic peaks reaching 345 mmHg. The variability in his BP continued during the bottom and ascent phases of the dive, with values ranging from 225/110 to 265/155 mmHg. After he reached the surface, during the 33-s-long continued breath hold, his BP was ~250/105 mmHg. Thereafter, it gradually decreased to 150/75 mmHg in ~3 min.

A comparison of the simultaneous ECG and BP tracings showed that all the arrhythmias generated pulse pressures of at least 50 mmHg, and, therefore, the arrhythmias were arbitrarily considered hemodynamically effective. By application of this criterion, the HR obtained from the BP tracing was identical to the HR from the ECG.

$[La]_b$. The $[La]_b$ was always higher in the recovery period compared with the resting control values, indicating net lactate accumulation during the dives. In particular, during two dives to 50 m by *EM*, $[La]_b$ increased from 1.07 and 0.93 mM pre-dive to 2.26 and 3.97 mM post-dive, respectively. In *PM*, during a dive to 40 m and one to 45 m, $[La]_b$ increased from 1.92 and 1.67 mM pre-dive to 3.05 and 2.49 mM post-dive, respec-

tively. Finally, during two dives to 50 m by *RM*, $[La]_b$ increased from 1.29 and 0.87 mM pre-dive to 5.30 and 1.41 mM post-dive, respectively.

DISCUSSION

HR and heart rhythm. A picture very different from the classic, smoothly developing and subsiding diving bradycardia described in diving animals emerges from this study of human elite breath-hold divers. Our subjects showed a large number and variety of arrhythmic beats in their ECG recordings, particularly during dives in cool water. As an example, no sinus beats were observed for ~45 s in a dive in 25°C water by *RM* (Fig. 3). This confirms earlier observations of arrhythmias in human subjects during breath-hold diving in cold water to depths of 15 m or shallower (4, 19, 20, 26, 30). It also agrees with our previous observations in the same subjects during their dives in the sea to 45 and 65 m (13). Those ambulatory ECG recordings, done during dives, were at times distorted by motion artifacts; therefore, several arrhythmic beats may have been missed.

Apneic face immersion in cold water is known to strengthen the diving bradycardia by enhancing vagal tone (21), but it also causes arrhythmias (24). Furthermore, immersion in cold water causes a larger intrathoracic blood pooling than does immersion in thermoneutral water (22), and this may distend the heart, making it more susceptible to arrhythmias, as suggested by Arborelius et al. (1). Additionally, during breath-hold diving, a pressure gradient develops across the chest wall during descent (15). This caused the intrathoracic pressure to fall in our subjects (to -40 cmH₂O in *EM*

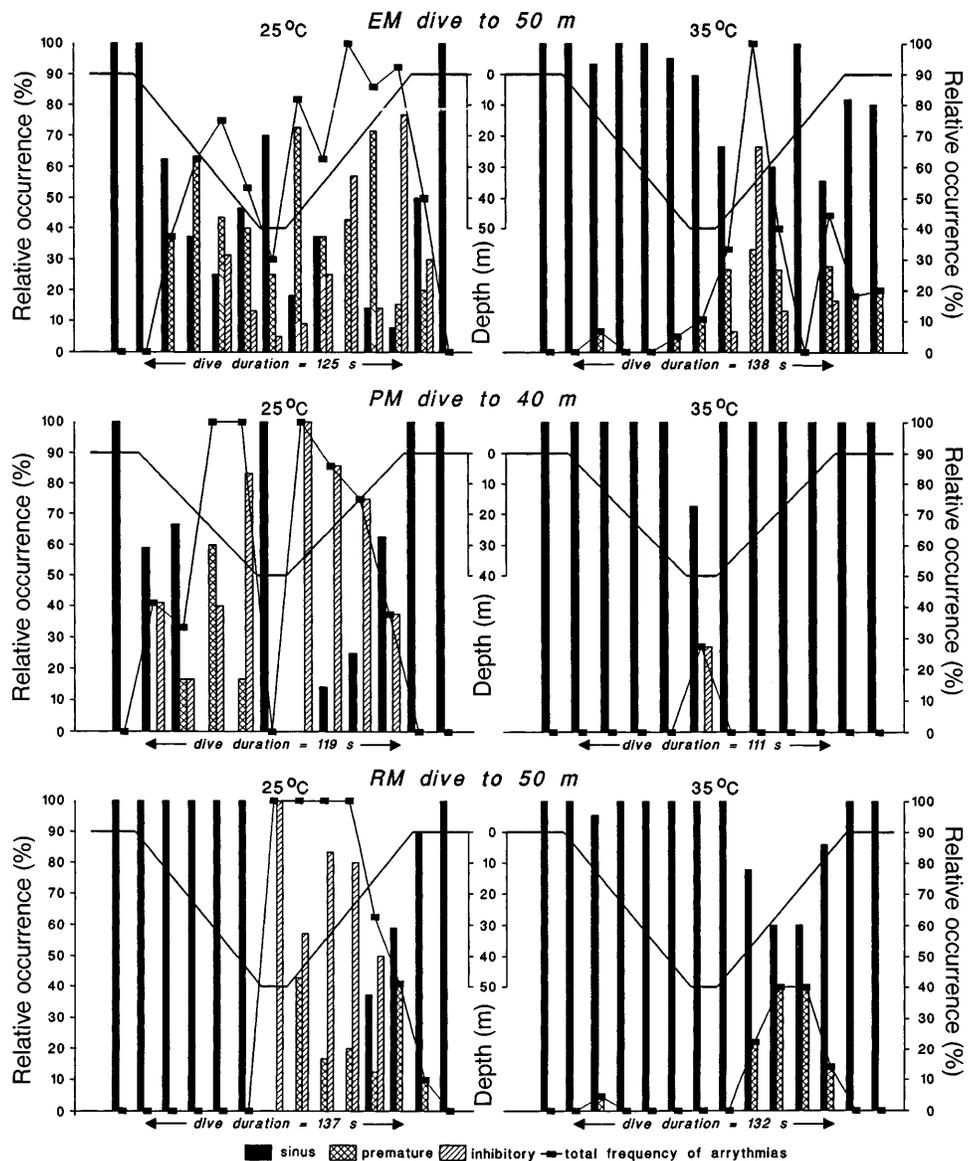


Fig. 3. Relative occurrence of arrhythmias and depth vs. time during breath-hold dives to 50 m by *EM* (top) and *RM* (bottom) and to 40 m by *PM* (middle). Measurements before and after dives were averaged over 10-s intervals. Water temperature was either thermoneutral (35°C) or cool (25°C).

and -20 cmH₂O in *RM*) (33), which probably augmented redistribution of blood from the periphery into the chest. As a result, the heart may have been distended even more, contributing to dysrhythmogenesis. In agreement with these concepts is our observation that arrhythmias were more frequent in cool water, at depth, and early in the ascent.

It is noteworthy that arrhythmias have only rarely been observed in diving animals (9). From a description by Elsner and colleagues (10) of the inferior vena cava sphincter in seals, it may be inferred that this sphincter reduces venous return during dives and protects against overdistension of the heart and consequent arrhythmias. Furthermore, the aortic bulb in the seal not only helps to maintain arterial pressure during diastole at very low HR but also reduces the increase in left ventricular afterload, which would be expected secondary to peripheral vasoconstriction during diving (9). This protective mechanism would tend to decrease

myocardial oxygen consumption. By contrast, the large increases in BP that we recorded during the present dives (see *BP* below) may have caused some subendocardial ischemia. This could also have played a role in the occurrence of arrhythmias. Ischemic ECG changes were described by Oliveira and Gomez Patiño (25) in subjects emerging from breath-hold dives down to 16 m. Finally, a high vagal tone in our divers may have contributed to dysrhythmogenesis, as we have previously suggested (12).

In a recent study, Tipton and colleagues (32) found frequent arrhythmias including supraventricular and ventricular premature beats that were similar to the ones described in the present study and confirmed our preliminary, previously published results (12). However, in their study, arrhythmias were observed predominantly just before the termination of breath holding and within the following 10 s. In the present study, most of the arrhythmias occurred during the dives,

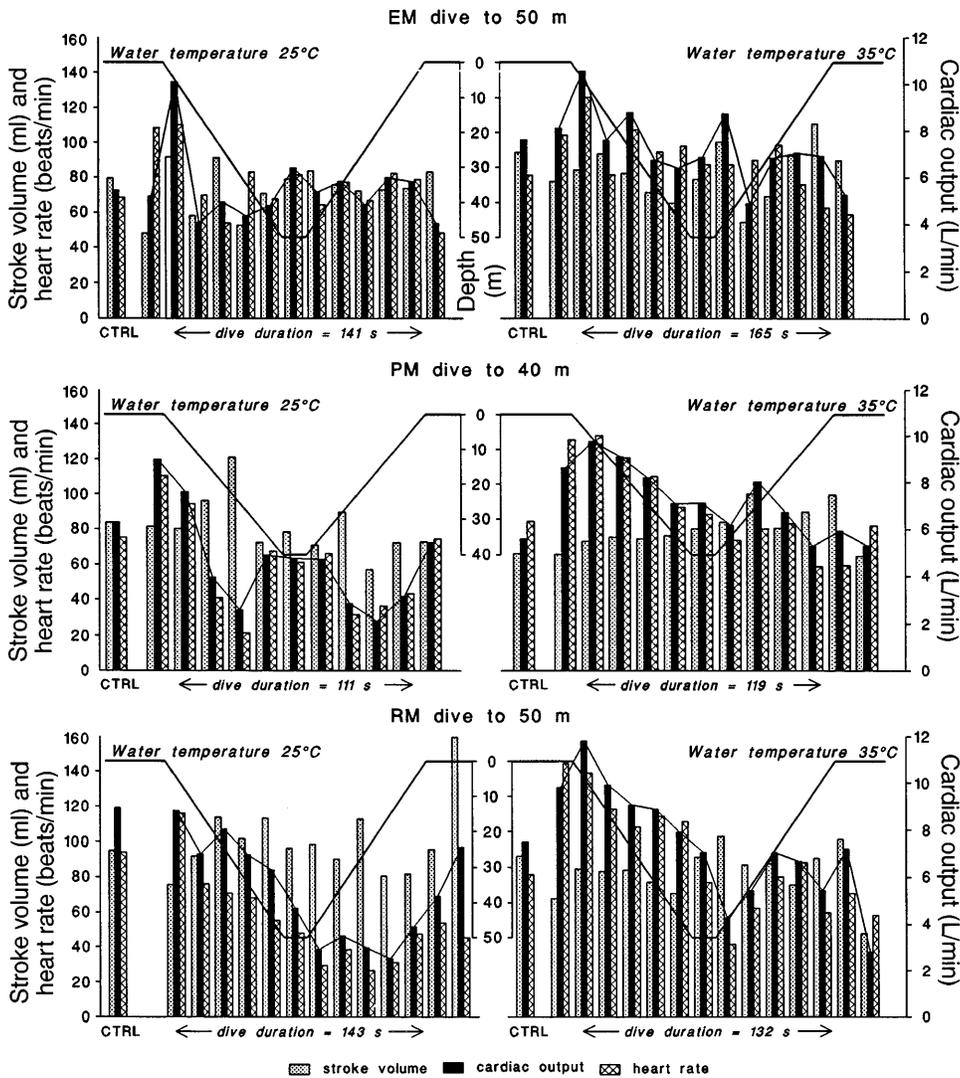


Fig. 4. Stroke volume, cardiac output, and depth vs. time during breath-hold dives to 50 m by EM (top) and RM (bottom) and to 40 m by PM (middle). Measurements before and after dives were averaged over 10-s intervals. Water temperature was either thermoneutral (35°C) or cool (25°C). CTRL, control.

possibly due to the larger intrathoracic blood redistribution caused by the much greater depths reached by our elite divers.

Even when the many arrhythmic beats are counted, bradycardia was present, particularly in the cool water, during the breath-hold dives in this study. Tachycardia just before and at the beginning of the dives was similar in the cool and the thermoneutral water and may have been due to anticipatory excitement and hyperventilation. However, the more rapid onset of bradycardia and the lower HR values during the dives in cool compared with thermoneutral water point to water temperature as the most important factor in eliciting bradycardia in our experiments. Depth by itself did not appear to affect HR because diving bradycardia developed slowly and to a similar extent in both the chamber dives in thermoneutral water (independent of maximal depth) and the breath holds at the surface performed in a dry environment by the same subjects during an earlier study (13).

CO. Although there are several studies of CO during breath holding at the surface with and without face immersion (for a review, see Ref. 15), to our knowledge,

only one study has looked at CO during actual breath-hold diving in both the dry and submersed condition in thermoneutral water (14). In that study, breath holding by the subjects with large lung volume at the surface, just before they dove, decreased cardiac index by 20.8% in the dry condition and 24.4% in the submersed condition. This was thought to be due to a high intrathoracic pressure impeding venous return. When the subjects dove to 20 m, cardiac index returned to the control values, probably secondary to a fall in intrathoracic pressure improving venous return. Those changes in cardiac index reflected similar changes in SV because no changes in HR were observed in those untrained subjects. A different picture was observed in the present study involving elite divers. Both bradycardia and many hemodynamically effective arrhythmias influenced CO, which overall showed a tendency to decrease in the dives in the cool water. Changes in CO were caused by concomitant changes in HR because SV showed no significant variations. The presence of a diving response, that is, bradycardia with reduction of CO, in the present study may have been due to the colder water, the deeper depths, and the higher level of

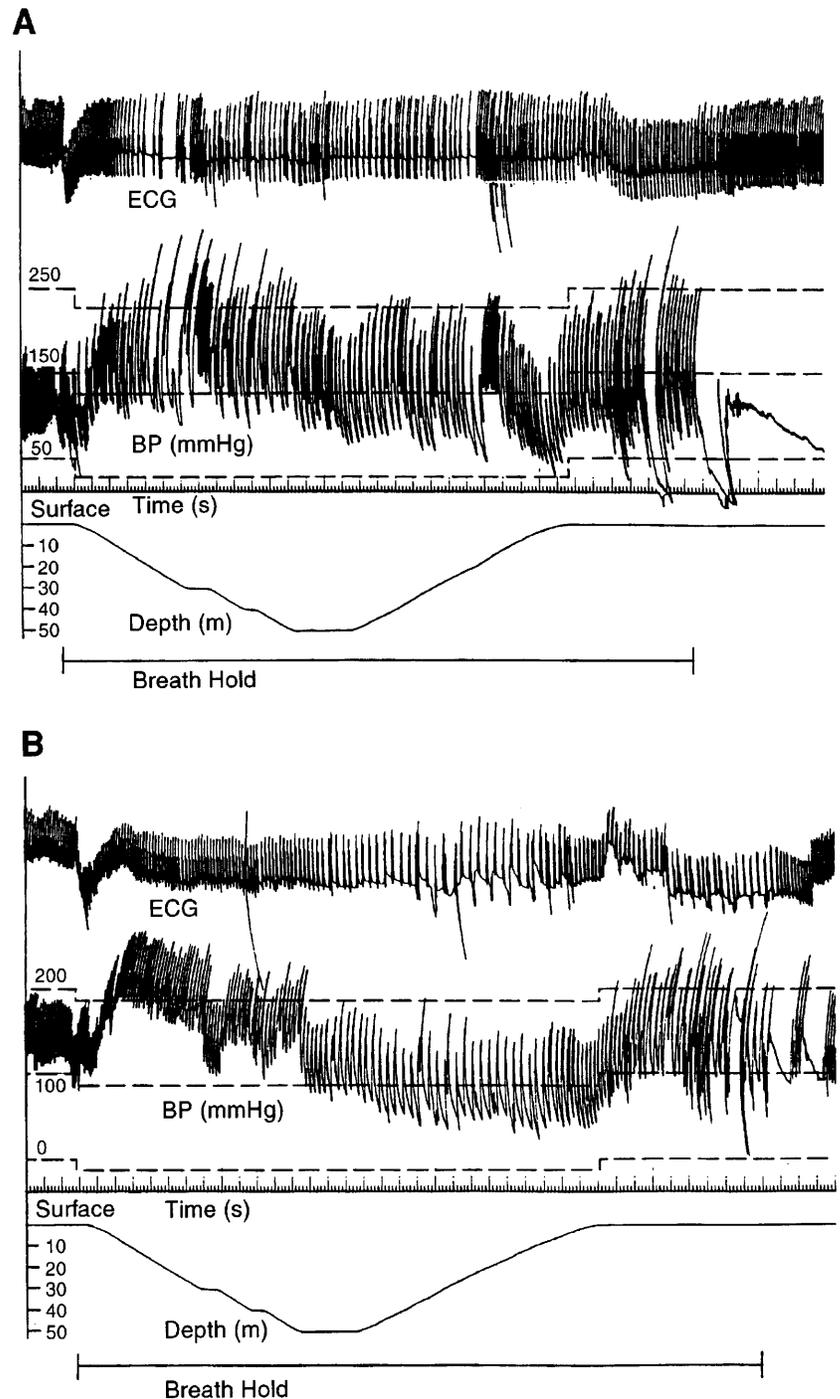


Fig. 5. Arterial blood pressure (BP), ECG tracings, and depth vs. time in *EM* (A) and *RM* (B) during a breath-hold dive to 50 m in cool water (25°C).

training in the elite divers compared with the subjects of the study mentioned above (14).

BP. To our knowledge, no information was available in the literature about BP in humans during breath-hold diving. Before this study, measurements of BP had been done either during apneic or nonapneic face immersion at the surface (3, 17, 21, 31) or during breath holding just below the surface (5, 18, 27, 30). Most of these studies showed no or only a modest and gradual increase in BP. An exception is the study by Bjertnæs et al. in 1984 (3), in which mean arterial pressures as high

as 25.33 kPa (~190 mmHg) were observed at the end of the experiments involving apnea, face immersion in ice water, and exercise.

The very large and sudden increases in BP at the beginning of the dives, up to 280/200 mmHg in *RM* and 290/150 mmHg (with occasional systolic peaks reaching 345 mmHg) in *EM*, are by far the highest values reported in the diving literature. These increases may have been due to a combination of factors, besides apnea and submersion in cool water. A markedly accentuated vasoconstrictor response may have developed

secondary to the subjects' training, which involves frequent deep dives. Similarly, in the study by Campbell et al. (5), 1 of the 18 subjects, who was a competitive swimmer, showed consistently higher BP responses than did the other subjects when performing breath holds in and out of the water. Excitement, similar to what probably happens during our divers' record attempts, may have also played a role, particularly in the case of *RM*, who exhibited high BP already during the last part of the pre-dive hyperventilation.

The high BP recorded during the early part of the two dives was caused primarily by peripheral vasoconstriction because CO, after a short-lasting initial increase, tended to be at or below the pre-dive level. The drop in BP that developed as the dives continued appears to have been due, at least in part, to lowered CO secondary to bradycardia (presumably elicited by baroreceptor stimulation). The many arrhythmic beats, which proved to be hemodynamically effective, contributed to variations in BP during the dives. After the subjects surfaced, the rise in BP during the continued breath holding may have been due to an accentuation of peripheral vasoconstriction reflex caused by hypoxia, which commonly occurs at the end of deep breath-hold dives (11).

The slow drop in pulse pressure, particularly evident at low HR, is suggestive of intense peripheral vasoconstriction, similar to what happens in the seal. A vasoconstrictor response has been described in humans performing dives simulated by breath holding with face immersion (18). However, in the seal, large increases in BP are prevented by a concomitant reduction in CO (9), much greater than the one recorded in our divers. Furthermore, humans lack the "aortic bulb," which not only reduces systolic pressures during peripheral vasoconstriction (as mentioned in *HR and heart rhythm* above) but also helps to maintain BP during prolonged diastolic intervals (9).

Peripheral vasoconstriction implies a reliance of peripheral tissues on anaerobic metabolism, conducive to accumulation of lactate. In fact, an increase in $[La]_b$ has been described after breath-hold diving in both diving animals (e.g., Ref. 9) and humans (e.g., Refs. 27 and 30). Furthermore, an increase in $[La]_b$ was found after both the present chamber dives and some dives at sea performed by the same subjects (11). This accumulation of lactate occurred despite the low metabolic cost of diving, which was calculated for the dives at sea. That level of metabolism should not have been accompanied by any lactate accumulation during ordinary dynamic exercise. On the other hand, whereas prolonged dry breath holds by the same subjects led to a large fall in arterial oxygen pressure, they still did not cause accumulation of blood lactate, probably because there was no intense peripheral vasoconstriction (11). In the present study, there must have been a marked reduction of limb blood flow preventing oxygen delivery to the muscles, thus inducing anaerobic metabolism. Support for such a mechanism is offered by earlier observations in our laboratory of a >50% reduction in forearm blood

flow when subjects held their breath at one atmosphere while their faces were flushed with water at 20°C (31).

Despite the frequent prolonged R-R intervals, no clinical signs of a failing circulation were observed and no symptoms were reported by the divers. This is in sharp contrast to the light-headedness or even syncope occurring in patients after only 5–10 s of absent or ineffective cardiac contraction (16). Similarly, Arnold (2) described cases of extreme, still asymptomatic, diving bradycardia elicited by apneic facial immersion in ice water, with the longest R-R interval being 10.8 s. An important factor in protecting cerebral perfusion during the accentuated diving bradycardia is the intense peripheral vasoconstriction, which helps to maintain cerebral perfusion pressure.

In conclusion, it appears that human elite breath-hold divers exhibit an intense diving response. The cardiovascular changes of the elite divers in this study, that is, bradycardia, a reduction in CO, and peripheral vasoconstriction, resemble the ones described for diving animals, in which they supposedly have an adaptive value. Yet, the divers also showed other reactions, including many arrhythmias and very high BP during their dives. These phenomena are possibly due to anatomic differences from the diving animals, and, if encountered in a clinical setting, they would be cause for considerable alarm. However, in our subjects, these cardiovascular aberrations were not related to any apparent cardiovascular disease and did not cause any signs or symptoms; still, they point to some imperfections in the human response to deep breath-hold diving. Finally, because our three subjects belonged to the same family, these observations may be the expression of genetic factors rather than representing a physiological adaptation, and they may not apply to other human breath-hold divers.

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