Electrocardiogram during deep breath-hold dives by elite divers

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Ferrigno M, Grassi B, Ferretti G, Costa M, Marconi C, Cerretelli P, Lundgren C. Electrocardiogram during deep breath-hold dives by elite divers. Undersea Biomed Res 1991; 18(2):81–91.—A portable ECG recorder was used during breath-hold dives at sea by 3 elite divers to 65 and 45 m. ECG was also recorded during nonimmersed maximal breath holds in the divers and 8 control subjects. Heart rate in the dives decreased rapidly to 20–24 beats · min^{-1}. During the surface experiments in the divers, bradycardia was much slower in onset, reaching 28–36 beats · min^{-1} at the end of the breath holds. The divers showed a more consistent bradycardial response than the controls. The difference in temporal pattern of bradycardia, in the dives and in the breath holds by the divers, may have been due to face immersion in cold water, chest compression, and/or redistribution of blood into the chest with concomitant stimulation of cardiac and other mechanoreceptors. Arrhythmias, mostly supraventricular and ventricular premature complexes, were observed coincidently with the lowest heart rates, presumably reflecting a high vagal tone. In addition, cardiac distention at depth might have made the heart more prone to arrhythmias, while in the surface breath holds hypoxia might have accounted for a similar effect.

ECG
bradycardia
diving response
diving reflex
arrhythmias
apnea
breath-hold diving

Hundreds of breath-hold dives deeper than 50 m (1), and several deeper than 100 m (2), have been carried out by both male and female divers around the world. However, very little information is available about the physiologic mechanisms that allow such exceptional performance by humans. A recent brief account of ECG recordings in breath-hold dives as deep as 72 m lists heart rates (HRs) of 42–45 beats · min^{-1}, first degree A-V blocks, and nodal rhythm (3). Since the so-called diving response (bradycardia, peripheral vasoconstriction, and reduced cardiac output) has been demonstrated in man at the surface and at shallower depths (for reviews, *see* 4, 5),

one might ask if more pronounced changes in circulatory function occur during breath-hold diving to great depths. With increasing depth, stimulation of facial receptors by cold water is likely to increase, as are chest compression and redistribution of blood into the chest. All these factors may lead to circulatory changes (6, 7).

A field study was performed in 3 elite breath-hold divers to investigate physiologic responses to dives down to 65 m. The present report concerns ECG recordings which, for logistical reasons, were obtained 1 yr after collecting respiratory and metabolic data; these data have been reported elsewhere (8). These recordings were also compared to ECG tracings obtained during maximal dry breath holds at the surface by the same subjects.

METHODS

Electrocardiographic recordings were obtained during deep breath-hold dives performed off the coast of Siracusa, Italy, by 3 elite divers: 1 male, Enzo Maiorca (EM, 56 yr old) and his 2 daughters, Patrizia Maiorca (PM, 29 yr old) and Rossana Maiorca (RM, 27 yr old). Their ECGs at rest in the dry condition showed HRs for EM of 60 beats $\cdot$ min^{-1}, RM and PM 65 beats $\cdot$ min^{-1}, without conduction or rhythm abnormalities. These divers have in the past established world records, reaching 101 m (EM), 80 m (RM), and 70 m (PM). The dives described here were part of their training, 1 mo. before world-record attempts. (The authors of this study are aware of the dangers inherent to extreme breath-hold diving, and do not endorse record attempts as being medically safe.) ECGs were also obtained during maximal breath holds at the surface (nonimmersed) in the divers and in 8 sex- and age-matched control subjects, 2 males and 6 females, with no prior diving experience. These breath holds were performed at rest in the supine position and were preceded by variable periods of hyperventilation (1 to 5 min). The subjects gave their informed consent to participate in this study and to disclosure of their identities.

To record ECG during the dives, a portable, three-channel magnetic tape cartridge recorder (model 499B, Del Mar Avionics, Irvine, CA) was used. A water and pressure-proof Plexiglas housing for the recorder was designed, constructed, and tested in a hyperbaric chamber at the Center for Research in Special Environments (CRSE). The housing containing the recorder had neutral buoyancy and was strapped to the diver's lower back. Five ECG electrodes were attached to the diver's chest with a technique developed and successfully used in hundreds of wet chamber dives at CRSE. After shaving the skin when needed, alcohol and tincture of benzoin were applied. The electrodes were covered with conductive paste which was checked to be without any visible air bubbles. They then were attached to the skin with double-adhesive rings and covered with adhesive disks (Stomaseal, Personal Care Products/3M, St. Paul, MN), which also secured a 2-in. loop of the electrode wire. The electrodes were placed over bony parts of the chest to minimize motion artifacts. The modified CM lead convention, suggested by the manufacturer of the recorder, was used, providing two separate bipolar leads: a modified V5 left ventricular lead, and a modified atrial and right ventricular lead, V1 (9). Electrode function was also tested at CRSE on 2 subjects at rest, immersed for 15 min in thermoneutral water with salinity similar to the one of the Ionian Sea. No difference in the quality of ECG recordings was noted between the dry and the wet condition.

Recorded tapes were played back on a scanner (Del Mar Avionics, Irvine, CA) which provided ECG strips. Time correlation between ECG recordings and depth-time profiles of the breath-hold dives was obtained by simultaneously starting the timer of the portable recorder and a separate stopwatch. Events during each dive were described and referenced in a diary to stopwatch time. During descent, times of arrival at predetermined ear-clearing stops and at maximal depth were announced by an experienced tender on board, who was holding his hand on the descent line used by the diver. Doing this, he was able to feel when the breath-hold diver stopped his or her descent using a brake, as described below. Descent and ascent rates between known depths were essentially constant. Heart rate at the surface was calculated from four R-R intervals. During the dives, four or fewer R-R intervals were used (as dictated by the presence of motion artifacts).

Diving techniques

The dives were conducted from a boat and the breath-hold divers were assisted by an experienced team comprising four scuba divers, two surface tenders, and a physician. Each dive was preceded by two or three periods of hyperventilation, separated by 10-min intervals. Each period consisted of slow, deep breaths and lasted about 8 min. The last period of hyperventilation before diving was performed with the diver immersed in water up to the mid-thigh level; the diver was wearing a wet suit but no mask or goggles. After taking an almost maximal breath, the diver started the descent, during which he or she was pulled by a heavy device which ran along a vertically suspended rope. The device used for descent included a platform on which the divers could stand (EM and PM). Alternatively, the device could be held onto with one hand while the diver descended head-first (RM). To facilitate ear clearing, the device could be stopped at selected depths, marked on the rope, by applying a brake. During ascent, the diver first pulled himself or herself gently along the rope, then was brought up to the surface by the buoyancy of the expanding wet suit. The average rate of both descent and ascent was about 1 $m \cdot s^{-1}$.

RESULTS

Breath-hold dives

The HR and the depth-time profiles of the dives are shown in Fig 1; EM dived to 65 m, and PM and RM reached 45 m. The duration of the dives was 130 s for EM, 110 s for PM, and 107 s for RM. In EM and RM, descent and ascent lasted about the same time, whereas in PM ascent was 22 s longer than descent. Typically, HR changed according to the following pattern: during the preparatory hyperventilation, tachycardia was observed; it was followed by a precipitous drop in HR with the initiation of the dive. The lowest HR values were reached at or near the maximal depth in RM (20 beats $\cdot min^{-1}$) and PM (20 beats $\cdot min^{-1}$), whereas in EM the minimum HR (24 beats $\cdot min^{-1}$) was observed at 40 m, i.e., 30 s before arriving at 65 m. Afterward, as a rule, HR tended to increase slowly during ascent still showing bradycardia on reaching the surface. As soon as ventilation was resumed, HR showed a rapid increase.

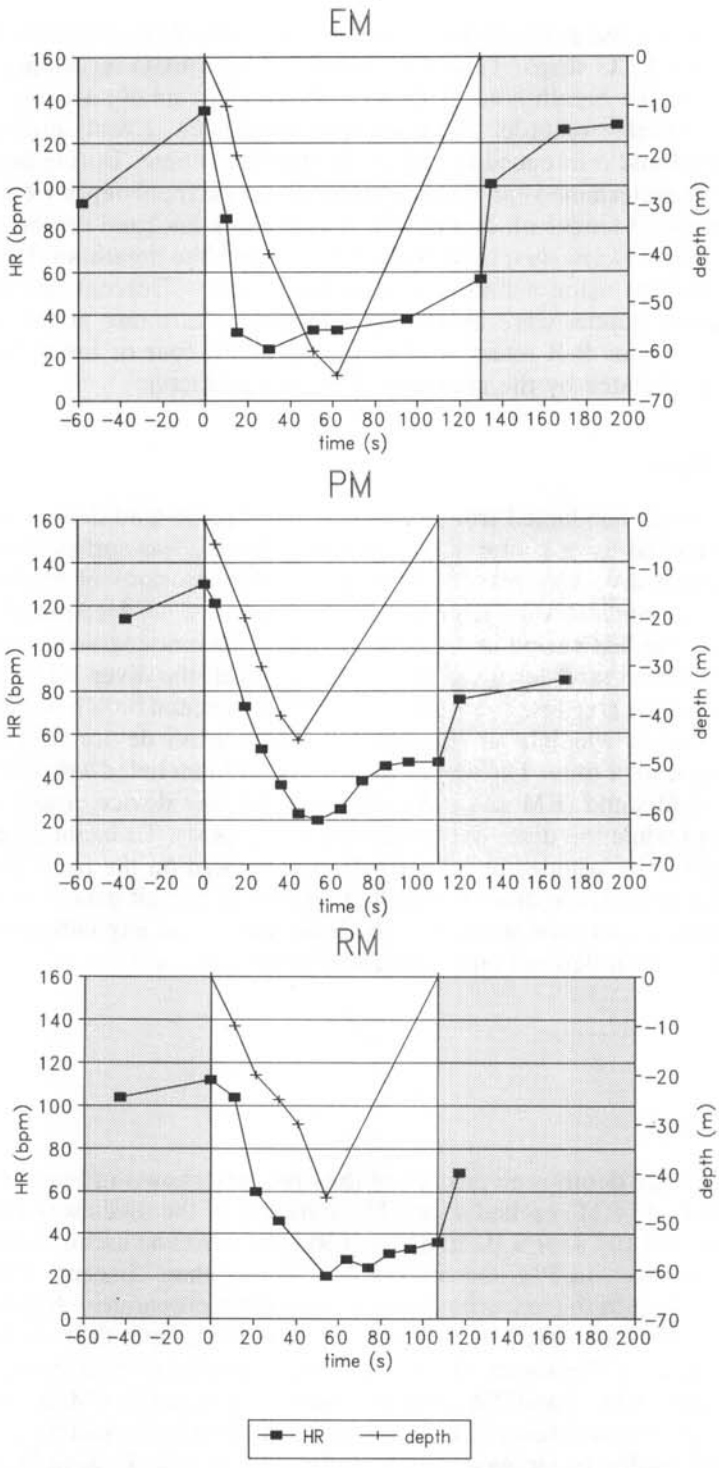

Fig. 1. HR and depth-time profile of the breath-hold dive by EM, PM, and RM. *Shaded areas* represent hyperventilation (before diving) and resumption of breathing (after diving).

With regard to the site of impulse generation in the heart in PM and RM, the pacemaker shifted from the SA node to the AV nodal region during descent, where it remained during the early part or all of the ascent (Fig 2, *top*). In EM, motion artifacts in ECG tracings did not allow recognition of P waves, making impossible the distinction between sinus and junctional rhythm (Fig 2, *bottom*). Frequent, premature complexes, either supraventricular or monomorphic ventricular in nature, were observed at or near the maximal depths when HR was at the lowest values. Ventricular bigeminy, lasting 20 s early in the ascent, was also observed in PM (Fig 2, *center*).

Surface measurements

Heart rates in the divers during maximal breath holds at the surface are shown in Fig. 3, together with the HRs recorded during the dives. They held their breath for 309 s (RM), 304 s (EM), and 251 s (PM). The HR values shown are averages of four surface breath holds, during which HR consistently showed a more gradual decline, compared to the bradycardia during the dives. In fact, as evident in Fig 3, in the first 50 s of the surface breath holds, HR, although decreasing, was consistently 4 times higher than the corresponding values at depth. In the course of these breath holds, HR decreased from 104 to 28 beats · min^{-1} in EM, from 103 to 34 beats · min^{-1} in RM, and from 94 to 36 beats · min^{-1} in PM. In the latter 2 divers, a small increase in HR occurred in the last 30 s of the longest breath hold, probably due to increasing respiratory muscle activity. The initial HR was consistently higher in the case of the dives than in the surface breath holds.

Heart rates during maximal breath holds at the surface by the controls and the divers are shown in Fig. 4. The 2 male controls showed a progressive decrease in

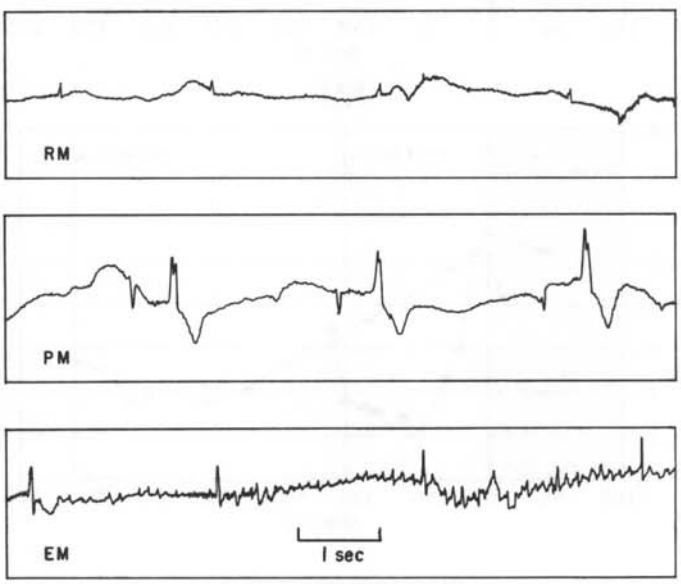

Fig. 2. ECG tracings during the dives: at 36 m during ascent in RM (*top*), at 31 m during ascent in PM (*center*), and at 40 m during descent in EM (*bottom*). HR was 28 beats · min^{-1} in RM, 25 in PM, and 24 in EM. Junctional rhythm in RM and ventricular bigeminy in PM are shown.

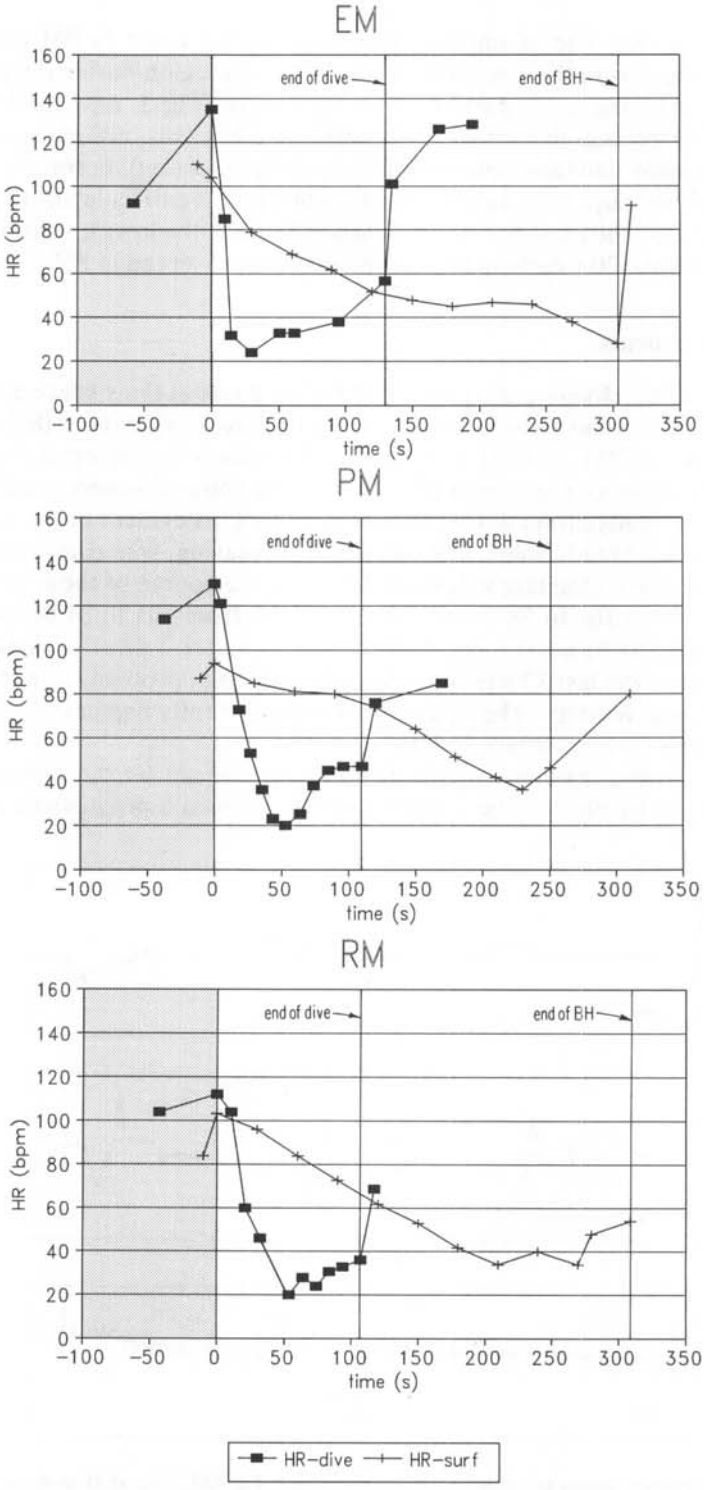

Fig. 3. HR during maximal breath holds at the surface (*BH*) and during breath-hold dives by the 3 divers. *Shaded areas* represent preparatory hyperventilation.

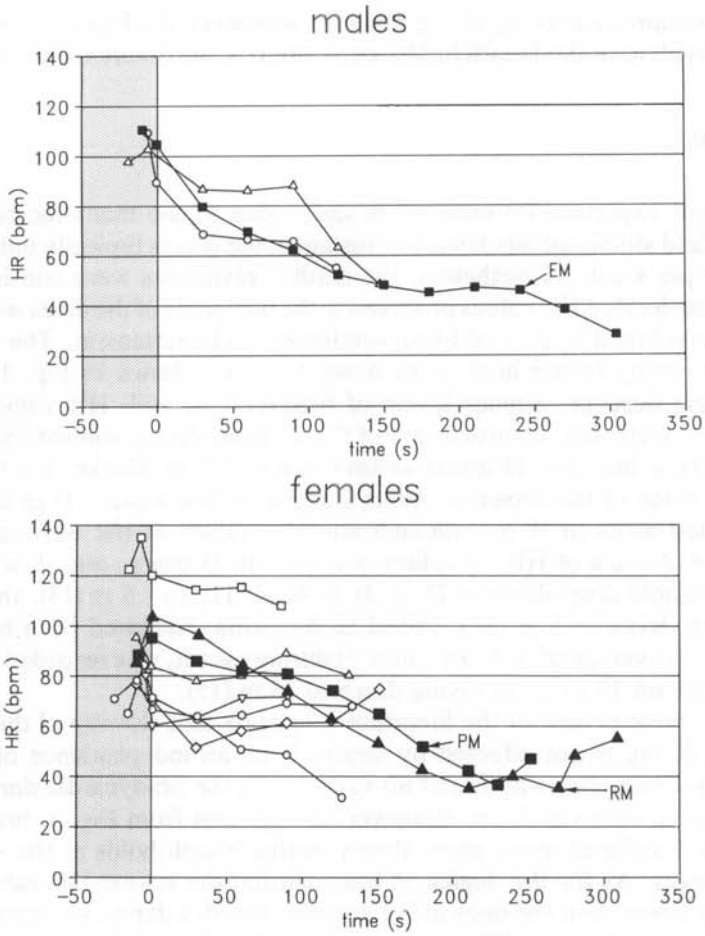

Fig. 4. HR during maximal breath holds at the surface by the divers and 6 sex- and age-matched control subjects. *Shaded areas* represent preparatory hyperventilation.

HR, similar to the one in EM, with the lowest value of 48 beats · min^{-1} reached by GF at the end of his maximal breath hold, which lasted 148 s. Of the 6 female controls, only 1 (VC) showed a clear bradycardial response with her HR going from 66 to 31 beats · min^{-1}. On average, the divers could hold their breath almost twice as long as the controls.

Sinus rhythm was often replaced by junctional rhythm toward the end of the surface breath holds in the divers but not in the controls. Prompt return to sinus rhythm was the rule with resumption of breathing. Several premature complexes, either supraventricular or monomorphic ventricular, and also ventricular bigeminy in PM, were observed in the last third of the breath holds by the divers. A short episode of ventricular bigeminy was also observed in RM at the time she resumed ventilation after a long breath hold. In EM, occasional premature atrial complexes were present throughout breath holding. Premature supraventricular and ventricular complexes were also present in 1 female and in 1 male control subject at the end of their breath

hold. Furthermore, flattening of the P wave, whenever the latter was present, was sometimes evident in the breath holds, more often in the divers than in the controls.

DISCUSSION

The present experimental material is small, due to the many-faceted problems inherent to field studies of this kind. For instance, the divers typically only performed 1 deep dive per week. Nonetheless, the cardiac responses were consistent among them. The accelerated HR values observed at the beginning of the dives were probably due to the combined effects of hyperventilation and excitement. The pronounced bradycardia during breath-hold dives down to 65 m, shown in Fig. 1, developed rapidly during descent. Similar levels of bradycardia, with HR values below 30 beats · min^{-1}, were also recorded in 4 of the 17 pearl divers studied by Scholander et al. (10). Those breath-hold divers swam down to 5–7 m. Marked bradycardia also occurred in some of the Japanese Amas studied by Sasamoto (11) in both assisted and unassisted dives to 10 m, although absolute values of HR were not reported. More modest slowing of HR, to values of about 40–45 beats · min^{-1}, was observed during breath-hold dives down to 72 m (3), to 40 m (12), to 2.5 m (13), and in Korean Amas during dives to 5 m (14). Initial tachycardia, followed by a bradycardial response (to an average of 60 beats · min^{-1}) during ascent, was recorded by Bonneau and colleagues on 10 subjects diving down to 15 m (15).

From the above review of the literature it appears that slowing of the HR during breath-hold diving is not affected by depth. Such an independence of depth was suggested by Craig (16), who found no variation in the bradycardia during dives to different depths, down to 27 m. However, as apparent from Fig. 3, bradycardia in our 3 divers developed much more slowly during breath holds at the surface than during the dives. As for the degree of bradycardia, the lowest HR values at depth were slightly lower than the ones at the surface. Besides depth, an important difference between the two conditions was water immersion in the case of the dives. Apneic face immersion in cold water has been proven to play an important role in eliciting diving bradycardia (17); some individuals may exhibit extreme levels of bradycardia, i.e., to less than 20 beats · min^{-1} (18). It is noteworthy that all 3 divers showed marked bradycardia (20–25 beats · min^{-1}). They were partially protected by wet suits but their faces were exposed to sea water, the temperature of which typically is about 22°C at the surface and 15°C at 40 m at that time of the year (12). Therefore, face immersion in cold water during the dives probably caused the faster onset of bradycardia, compared to the surface breath holds.

The divers showed a modest increase in HR during ascent. Although they performed light exercise, pulling themselves along the line in the first part of their ascent (until the buoyancy of their wet suit started bringing them toward the surface), the HR increase was probably not exercise related inasmuch as physical activity has been shown to potentiate the bradycardial response (19, 20). A possible explanation for the increase in HR during ascent could be the changing water temperature during the dives, with the lowest values, i.e., the most powerful stimulation of face cold receptors, at depth. However, other factors might have affected HR during the dives reported here. An increase in intrathoracic blood volume during deep breath-hold dives was suggested by Craig (21) and measured at 850 and 1047 ml by Schaefer et

al. (22) during dives to 130 and 90 ft, respectively. A similar accumulation of blood in the thorax during descent in our divers could have contributed to their bradycardial response, whereas during ascent a decrease in venous return would have had the opposite effect on HR. This hypothesis is in agreement with Craig's observation that increasing venous return, by immersion or changes in posture, caused bradycardia (16). Receptors in the heart, stimulated by mechanical distension, probably play a role in this response, although their effects are variable (23). The picture is further complicated by the possible stimulation of "J" receptors in the pulmonary vascular bed, which should be engorged at depth. This might also contribute to the bradycardial response (24).

Chest and lung compression during breath-hold diving might also have influenced HR. Elsner et al. (25) reported that the bradycardial response to apneic face immersion was accentuated when it was performed in the expiratory position. Thus, during descent, chest compression might contribute to HR slowing, whereas thoracic expansion during ascent might accelerate HR. Associated with chest compression and expansion in breath-hold diving are changes in intrathoracic pressure, which has been shown to decrease during descent to 20 m (26). Changes in intrathoracic pressure have been associated with parallel changes in HR (27), and this could help to explain the profile of bradycardia in our dives. However, the effects of superimposed Valsalva maneuvers during descent (for ear-clearing purposes) make the picture more complicated.

The female divers showed a more consistent bradycardial response than the controls. This might be related either to physical conditioning (28) or to the stress and anxiety of performing an unfamiliar task in our female controls. During apnea, bradycardia was observed in female synchronized swimmers, whereas control subjects typically showed tachycardia (29). Like the present divers, the synchronized swimmers could also hold their breath almost twice as long as their controls. We have discussed elsewhere (8) possible adaptational mechanisms in the divers of the present study.

Cardiac arrhythmias have been described during both actual breath-hold diving (10, 11, 13, 14, 30, 31) and breath holding at the surface (32), especially when associated with face immersion in cold water (14, 31, 33). However, a difference in the timing of these cardiac phenomena in the two conditions was observed in our divers: premature complexes, either supraventricular or ventricular in nature, and ventricular bigeminy occurred at or near the maximal depths during the dives, whereas they were present toward the end of the surface breath holds. It is possible that two distinct mechanisms are responsible for arrhythmias at surface and at depth, respectively. At the end of their maximal surface breath holds, the 3 divers were in an asphyxic condition, typically showing $P_{A_{O_2}}$ levels of 30–40 mmHg and $P_{A_{CO_2}}$ levels of 45–55 mmHg (8). This condition would be conducive to arrhythmias because hypoxia is known to make the myocardium irritable (34). It is also interesting to note that heart rhythm disturbances were less frequent in the controls, who could only hold their breath for much shorter periods of time and who had higher alveolar P_{O_2} than the divers at the end of the breath holds (8). However, hypoxia was not a factor at depth, where chest compression due to hydrostatic pressure greatly augments alveolar oxygen pressure (35). Acute distension of the heart, secondary to intrathoracic blood redistribution, is a possible cause of the cardiac arrhythmias in the divers at depth. Ventricular premature complexes have been recorded by Arborelius et al.

(36) during the first minutes of immersion of breathing subjects, at a time when signs of right heart engorgement were also recorded. In addition, arrhythmias may have been induced by high vagal tone since they occurred coincidentally with the lowest HRs; stimulation of the vagus has been described as a factor in the genesis of various arrhythmias (37). In support of this is also the fact that, at the end of the surface breath holds, the divers typically had both lower HR values than the controls and more frequent arrhythmias.

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At the time when this study was carried out, Mario Costa was on leave from the Department of Anesthesiology, Regional Hospital, Siracusa, Italy.

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