Electrocardiographic aspects of deep dives in elite breath-hold divers

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ABSTRACT

The cardiac diving response, 12-lead electrocardiogram (ECG) and the prevalence, time of onset, and possible associations of cardiac arrhythmias were examined during deep breath-hold (BH) dives. Nine elite BH divers (33.2 ± 3.6 years; mean ± SD) performed one constant-weight dive of at least 75% of their best personal performance (70 ± 7 meters for 141 ± 22 seconds) wearing a 12-lead ECG Holter monitor. Diving parameters (depth and time), oxygen saturation (SaO_2), blood lactate concentration and ventilatory parameters were also recorded. Bradycardia during these dives was pronounced (52.2 ± 12.2%), with heart rates dropping to 46 ± 10 beats/minute. The diving reflex was strong, overriding the stimulus of muscular exercise during the ascent phase of the dive for all divers. Classical arrhythmias occurred, mainly after surfacing, and some conduction alterations were detected at the bottom of the dives. The BH divers did not show any right shift of the QRS electrical axis during their dives.

INTRODUCTION

During constant-weight breath-hold (BH) diving, divers wear a weight belt and use a monofin to swim down to maximum depth and back on a single breath of air under their own power, without pulling on the rope or changing their ballast and without any type of assistance. The dive has four phases:

(i) a preparation phase at the surface, when the BH divers perform glossopharyngeal insufflation via the mouth in a lying position;
(ii) a second phase, when the divers hold their breath and use their monofins to swim downward until they begin sinking (around 3-3.5 atmospheres/atm);
(iii) a third phase once they begin sinking due to thoracic gas compression, when they perform no further muscular work until they reach the bottom (around 8 atm); and
(iv) the last phase, when they rise to the surface as a result of buoyancy due to gas expansion. Near the end of the dive (around 3-3.5 atm), BH divers do not swim but rise as a result of buoyancy. The official world record in constant-weight diving is 125 m (*www.aida-international.org*).

Breath-hold diving induces reflex responses, referred to as the diving reflex (DR), that include a vagally induced bradycardia with reduction in cardiac output, peripheral vasoconstriction due to sympathetic activity with increased peripheral resistance and blood pressure [18], and splenic contraction releasing more oxygen-carrying red blood cells into the bloodstream [39,43]. Blood flow is likely redistributed toward vital organs, whereas peripheral tissues may be underperfused such that lactate and catabolic acids accumulate in them. The ensemble of these changes, generally referred to as the diving response, is interpreted as an O_2-conserving mechanism [16,20,25]. Prolonged deep BH diving may cause cardiac arrhythmias in susceptible subjects [8,18,24,40], and abnormal automaticity may be triggered by hypoxemia and acidosis [20]. Furthermore, atrial stretch by increased thoracic blood pooling may promote mechanical and electrical feedback mechanisms [32], and the vagal stimulation concomitant to bradycardia may enhance re-entry phenomena [30]. Moreover, during deep BH diving, a relative drop in intrathoracic pressure is likely to enhance

redistribution of blood from the periphery to the chest in what is known as the "blood shift" phenomenon, which might overload the heart, contributing to arrhythmias (dysrhythmogenesis) [21]. Cardiac arrhythmias have thus been reported from both shallow [8,24,38,45,48] and simulated deep BH dives [18,19] and occasionally during submerged breath-holding during static (non-moving) BH competitions [29] or experimental BH tests [23]. These arrhythmias were related to BH duration and associated with individual factors such as the tolerable arterial oxygen saturation decrease [23,31].

Constant-weight BH divers, like most recreational BH divers, dive to depth in the sea using their fins. This diving method requires significant muscular exercise, particularly during the first parts of both the descent and ascent. Most of the published ECG data concern only static (non-moving) or assisted (weight sleds, air bags) BH dives with little muscular effort. Moreover, studies of breath-holding competitions in shallow water indicate that the incidence of clinical complications is non-negligible: about 1% and 10% of the static breath-holding performances have been reported to result in loss of consciousness and loss of motor control, respectively [33].

Fatal events have been reported in BH diving [46], but in most cases the cause of death and the possible contribution of heart rhythm disturbances to these events remain unclear. Cardiac arrhythmias might account for a significant portion of unexplained death during water immersion. However, the prevalence and nature of cardiac arrhythmias have not yet been studied systematically in the setting of constant-weight BH dive competitions or training.

In more recent studies, underwater Doppler-echocardiography has shown that chest volume reduction, associated with a simultaneous increase in pulmonary blood content, exerts an overload on the pulmonary circulation, even in shallow water [35,36]. Thus, BH dives may induce a right shift of the electrocardiogram (ECG) electrical axis, since this blood shift is known to dramatically increase pulmonary vascular pressures. However, ECG axis changes have never been described during BH dives. To date, the pathophysiology of deep BH diving is still not completely understood and research toward understanding the electrophysiology of deep BH diving could turn out to be useful in preventing future diving-related adverse events.

This study therefore tested the hypothesis that voluntary deep constant-weight BH dives may be associated with the occurrence of cardiac arrhythmias and a right shift of the ventricular ECG axis. We monitored the constant 12-lead ECG of expert BH divers during deep BH dives in the sea and also arterial blood oxygen saturation (SaO_2) before and after this dive. The aims were to evaluate alterations in the electrical axis and the prevalence and time of onset of cardiac arrhythmias. Possible association of arrhythmias with dive parameters and physiological parameters were investigated. In addition, the influence of glossopharyngeal insufflation on cardiac parameters and ventilation are described in the results.

METHODS

Subjects

Nine competitive BH divers volunteered for this study. Table 1 presents the baseline morphological characteristics and sports activities per week (as assessed by questionnaire two days before the diving tests), the percentage of fat mass (%FM) (assessed by the skinfold method according to Durnin and Womersley [13] using a calibrated skinfold caliper), cumulative BH exposure, documented years of BH training (YBH), maximal static BH performance (MSBH) and maximal constant-weight BH performance (CWTBH). These elite divers were international competitors, and this study was conducted during a training session before a world championship at Dean's Blue Hole in the Bahamas. The subjects were non-smokers and did not have a previous history of cardiovascular, pulmonary or neurological diseases. None of them was taking medications at the time of the study. The experimental procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki and were approved by the local ethics committee. Methods were explained in detail and informed written consent was obtained from all subjects.

Experimental protocol

The divers were evaluated in the Bahamas sea at a constant water temperature until the bottom at 8 atm (26-27°C), in the morning. Upon arrival at the site near Dean's Blue Hole, Bahamas, the divers were fitted with a 12-lead ECG Holter system (Spiderview, Ela Medical; Sorin Group, Milan, Italy). Then, they assumed the supine position not speaking and breathing normally; five minutes were allowed to achieve steady-state conditions. At that point, 10 minutes of measurements were obtained with the subject in the same position. The divers were asked to perform one constant-weight dive to at least 75% of their best personal performance but

TABLE 1. Characteristics of breath-hold divers

	Elite BHDs (*n*= 9)
Age (years)	33.2 ± 3.6
Height (cm)	180.0 ± 12.1
Body mass (kg)	74.5 ± 12.6
Fat mass (%)	18.5 ± 3.8
BMI (kg.m^{-2})	22.8 ± 1.4
Sports activities (hours.week^{-1})	7.1 ± 2.8
Breath-hold training (hours.week^{-1})	4.7 ± 2.5
YBH (years)	6.7 ± 2.5
MSBH (s)	432 ± 62
CWTBH (m)	93 ± 16

Characteristics of breath-hold divers (BHDs) and training parameters expressed as years of breath-hold training (YBH), maximal static breath-holding (MSBH), and maximal constant-weight breath-hold diving (CWTBH).
Body mass index (BMI = weight.height^{-2}) was calculated.

not to their maximum, to avoid disturbing them before the competition. This depth (75% of their best personal performance) was chosen because it was not unduly stressful for the BH divers, while still providing the significant compression routinely reached by these elite divers. They were allowed to undertake their usual predive warm-up protocol and breathing routine before BH diving. Before diving, most divers performed a protocol without any immersion. All briefly warmed up with stretching exercises for the chest, then performed glossopharyngeal insufflation via the mouth in a lying position at the water surface until they felt "full enough" [34]. The number of gulps needed to reach each participant's maximum level of glossopharyngeal insufflation was counted and compared with the number obtained during the pulmonary function tests. The divers also wore a nose-clip to avoid air leakage [4,12], glasses filled with water, and a depth watch (Suunto, D4, Vantaa, Finland) to record the diving parameters. Immediately after the end of the dive, the SaO_2 was recorded, and then the subject remained supine at rest for 10 minutes.

The 12-lead ECG was continuously recorded throughout the experiments. Blood O_2 saturation was monitored every five seconds by infrared spectroscopy (Palmsat 2500, Nonin Medical, Plymouth, Calif., USA) in the supine position for five minutes at rest, and up to five minutes after the dive. In addition, at rest and at three minutes after the dive, a 5-µL capillary blood sample was drawn from a finger and analyzed immediately (Lactate proTM, LT-1710, Arkray Inc., Kyoto, Japan).

Pulmonary function tests

Several parameters were measured two days before the diving tests: forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), FEV_1/FVC, peak expiratory flow (PEF), maximal expiratory flow rates at 75%, 50% and 25% of FVC ($MEF_{75\%}$, $MEF_{50\%}$, $MEF_{25\%}$), and forced mid-expiratory flow rate ($FEF_{25-75\%}$). Spirometry was collected according to the ATS/ERS published recommendations NHANES III [41]. Ventilatory function variations (Δ) were calculated as the index of change (magnitude and direction) induced by glossopharyngeal insufflation [for example, ΔFVC= (FVCGI-FVC)/FVC)*100)].

Δ values were negative or positive, depending on the parameter kinetics (increase or decrease). This method minimized the differences between the values of the ventilatory parameters before and after glossopharyngeal insufflation. All of the parameters were measured using a Microquark spirometer (Cosmed, Rome, Italy) in a room with the same conditions, with air temperature and humidity monitored by the same technician. The pulmonary function tests (before and after glossopharyngeal insufflation) were performed in a sitting position, with the subject breathing through the mouthpiece with a nose-clip. The spirometer volume was calibrated twice daily with a 3-L calibrated syringe. The results were corrected to BTPS conditions and compared with predicted values [41].

Electrocardiogram

After shaving and preparing the skin, 10 electrodes were attached to the skin and connected to the Holter-ECG, which was placed in a small, cylindrical, waterproof hyperbaric container (Comex SA, Marseille, France) and fixed onto the back of the diver *(Photo 1)*. The container was made from a plastic tube with a welded bottom and a returned airtight top with the aid of a joint including an electrical connector cable with multibite quenches; it had been tested up to 20 atm one month earlier. The electrodes were specially prepared to avoid contact with water and covered with conductive paste which was checked to be without any visible air bubbles. They were attached to the skin with adhesive rings and covered with transparent adhesive (Tegaderm, 3M, St. Paul, Minn., USA). A transmission cable built specially for this experiment was fixed at the neck enabling the online 12-lead ECG monitoring. An SD-card (128 Mo) was placed in the Holter. The device and the methods were previously (one month before) tested in the sea with a BH diver to 8atm.

10 electrodes were attached to the skin and connected to the Holter-ECG, which was placed in a small cylindrical waterproof hyperbaric container and fixed onto the back of the breath-hold diver.

TABLE 2. Lung function parameters of breath-hold divers before and after glossopharyngeal insufflation

	Before GI	*p*	After GI	(Δ%)
FVC	119±12^{++}	***	149±27^{+++}	25±11
FEV1	111±12^{++}	**	127±18^{+++}	14±15
FEV1/FVC	93±8	ns	85±9	-8±11
PEF	102±12	ns	105±18	3±17
MEF75%	97±16	ns	101±20	4±15
MEF50%	88±19	ns	97±18	10±18
MEF25%	89±24	ns	99±17	11±20
FEF25-75%	95±18	ns	111±18	17±29

Lung function parameters of breath-hold divers before and after glossopharyngeal insufflation (GI) expressed as percentage of predicted values and percentage of changes [Δ%=(after GI - before GI)/before GI)*100], ns: non-significant. **: $p<0.01$; ***: $p<0.001$ between before GI and after GI; ++: $p<0.01$; +++: $p<0.001$ between measured and predicted values.

Data processing

Time correlation between ECG recordings and depth-time profiles of the BH dives was obtained by simultaneously starting the timer of the Holter and the depth watches and analyzed offline. Except for the diving depth, each parameter was averaged at fixed timepoints: at rest on the surface before the dive, during glossopharyngeal insufflation, at the bottom of the dive, and just after surfacing. ECG data were examined with Synescope software (Ela Medical; Sorin Group, Milan, Italy) and concerned several parameters. First, heart rate (HR) was documented in beats/minute and averaged every five seconds during the entire dive. Second, the QRS axis, atrioventricular conduction time (PR), ventricular depolarization duration (QRS) and total time of ventricular electrical activity (QT) were recorded and averaged from five consecutive complexes. The QT duration was adjusted to the HR using the Bazett formula [6]. All arrhythmias or conduction events were examined by two experienced investigators and blinded with respect to the subject. The diving profile was analyzed using Suunto Dive Manager 3© software. For SaO_2, an average value was calculated at rest for the period 15 seconds to 0 seconds before the dive, and just after surfacing.

Statistical analysis

The results are presented as means and standard deviations (± SD) and as percentages of predicted values according to Pellegrino *et al.* [41] for ventilatory parameters. Morphological characteristics, lung parameters (percentages of predicted, and before and after glossopharyngeal insufflation), lung function changes, HR and ECG changes were compared by a Wilcoxon Rank Sum test. Repeated measures analysis was also performed for all ECG data (HR, QRS axis and duration). Multiple linear regression analysis, performed in a stepwise backward fashion, was used to assess relevant correlations of age and BH exposure with the lung function parameters. Pearson correlations were also performed. ANCOVA analysis was performed to test differences on regression slopes. A p-value < 0.05 was considered significant. Analyses were performed with Statview software (Abacus Concepts, Inc., Berkeley, Calif., USA; 1992).

The subjects had a mean age of 33.2 ± 3.6 years and a mean body mass index (BMI) of 22.8 ± 1.4 kg.m^{-2} and had been training in breath-hold sports for 6.7 ± 2.5 years *(Table 1)*. All subjects performed a BH dive without any clinical complications such as loss of motor control or loss of consciousness. The duration of the dives was 141 ± 22 seconds, and the depth was 70 ± 6 meters, which corresponded to 75% of the personal best dives.

FIGURE 1a. QRS duration at the surface before BH dive

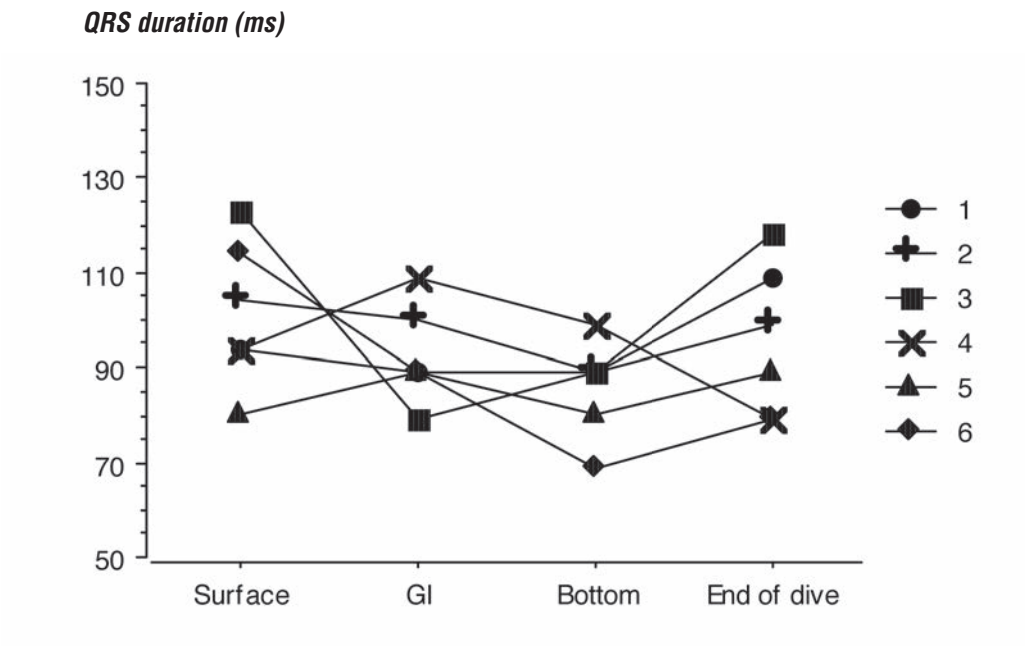
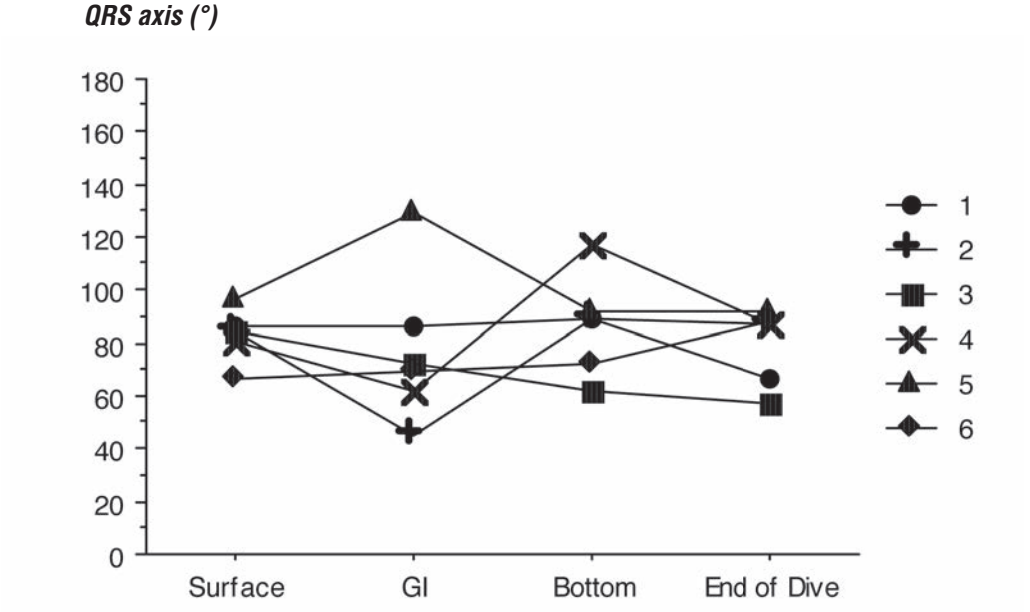


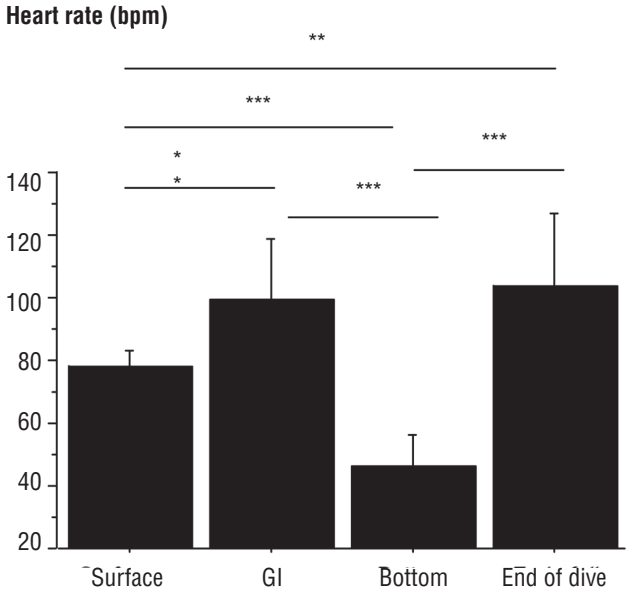
FIGURE 1b. QRS axis at the surface before BH dive



1a. QRS duration obtained at the surface before the BH dive, during the glossopharyngeal maneuver (GI), at the bottom, and at the end of the dive.

1b. QRS axis obtained at the surface before the BH dive, during the glossopharyngeal maneuver (GI), at the bottom, and at the end of the dive.

FIGURE 2. Heart values



Heart rate values obtained at the surface before the BH dive, during the glossopharyngeal maneuver (GI), at the bottom, and at the end of the dive.

Table 2 shows the results of lung function testing before and after glossopharyngeal insufflation. Before glossopharyngeal insufflation, the BH divers had higher FVC and FEV_1 values than predicted (p<0.01). After glossopharyngeal insufflation, they had higher FVC and FEV_1 values than before insufflation (p<0.01 and p<0.001, respectively). The number of gulps needed to reach maximal glossopharyngeal insufflation was not different from that obtained during the pulmonary function tests and that just before the dive (22 ± 8 *vs.* 23 ± 9).

Heart rate

HR increased from 78 ± 5 beats.minute^{-1} at rest on the surface to 99 ± 19 beats.minute^{-1} just before the dive during glossopharyngeal insufflation (p<0.01) *(Figure 2)*. HR decreased and stabilized at a level of 46 ± 10 beats.minute^{-1} (p<0.001), which corresponded to a 52.2 ± 12.2% change (range 28.4-68.4%). HR decreased at the time of diving for all divers, and the lowest HR was reached in less than 20 seconds after the dive onset. Afterward, and for all divers, HR tended to increase very slowly during ascent, still showing bradycardia until 20-25 meters, at which point HR increased until surfacing and then increased further upon surfacing (p<0.001), becoming higher than before the dive (p<0.01) *(Figure 3)*. The relative decrease in HR (%) was associated with the constant-weight performance (m) (r=0.733, p<0.05) and the %FM (r=0.936, p<0.01), but it was not associated with age, BMI, BH duration or years of BH training.

ECG data

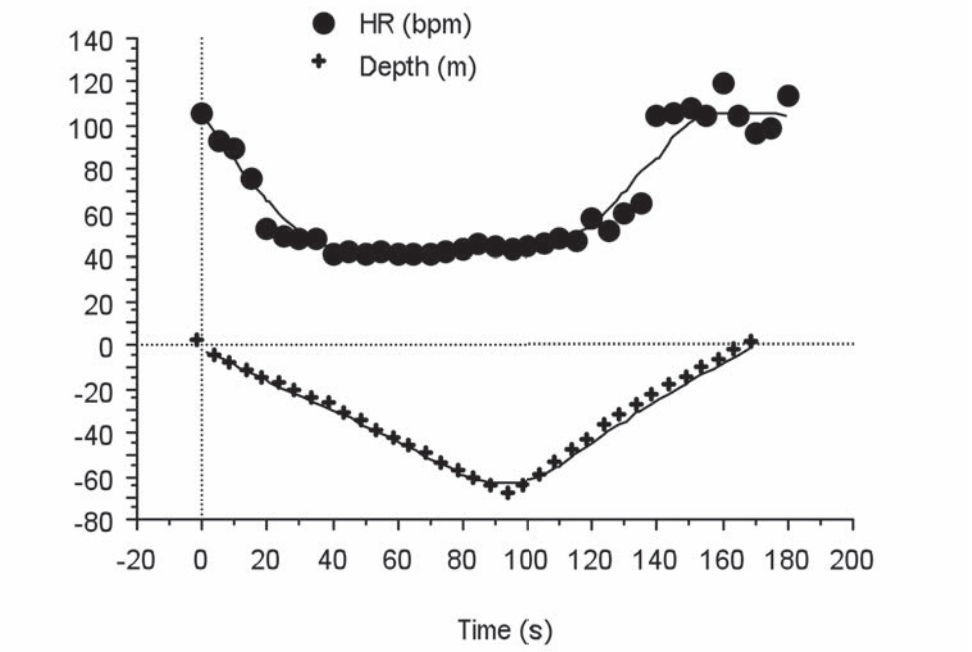
The ECGs before diving in the dry condition showed HR of 78 ± 5 beats.min^{-1}, without conduction or rhythm abnormalities. No difference was observed in either QRS axis deviation or QRS duration during these dives obtained only in seven BH divers *(Figures 1a and 1b)*. PR increased from 135 ± 20 at rest to 171 ± 27 at the bottom of the dives, consistent with the HR decrease (p<0.01). The QT slightly increased at the bottom of dive, but these changes vanished when considered as QTc.

Cardiac arrhythmias

Cardiac arrhythmias or conduction events were documented in 4/9 divers. No episodes of any symptomatology were reported by the divers before, during or after the dives. We observed one short episode of atrial fibrillation in one diver. This episode occurred 15 seconds after the onset of the dive, lasted eight seconds, and rose to a rate of 165 beats.min^{-1}. In the same diver, frequent premature atrial complexes (PACs) were observed just after surfacing. PACs were observed in one other diver after surfacing. A transitory junctional rhythm with normal QRS was observed in two divers at the bottom of the dive. For one, the HR decreased to 23 beats.min^{-1} for a 10-second period, for the second to 41 beats.min^{-1} for a 70-second period.

Oxygen saturation and lactate concentration SaO_2 decreased from 98.3 ± 0.7% one minute before the dive to 87.3 ± 5.5% at surfacing (p<0.01) and remain reduced when compared with before the dive until one minute after (94.4 ± 2.7 %, p<0.05). The SaO_2 at surfacing was not associated with age, years of experience in BH sports, diving performance (depth and duration) or %HR change, but it was negatively correlated with BMI (r=-0.77, p<0.05). The lactate concentration was increased after surfacing (6.9 *vs.* 1.6 mmol.L^{-1}, p<0.05).

FIGURE 3. Heart rate time response



Example of heart rate (HR) time response relative to the depth change in one breath-hold diver during a constant-weight dive.

DISCUSSION

The principal findings of this study were that:

(i) bradycardia slowly resolved during the ascent but changed abruptly after 2/3 of the ascent and after most of the muscular effort of ascent;
(ii) breath-hold divers did not show a right shift of the QRS axis;
(iii) cardiac arrhythmias occurred mainly after surfacing and conduction alterations occurred mainly at the bottom of the dives; and
(iv) the only correlations between cardiac and physiological parameters were between %HR change and constant-weight dive performance or %FM.

Heart rate

In trained divers, the initial fall in HR during a BH dive is to approximately 60% to 70% of the pre-dive level. Heart rates as low as 10 to 15 beats.min^{-1}, however, are reported in elite BH divers diving to depths of 65 meters [3,19,47]. This is lower than what we observed in our divers, who showed a mean heart rate of 46 beats.min^{-1} with a 54% fall during these dives. However, the diving reflex is strongly related to water temperature [22,44], being more significant at lower temperatures. Thus, the difference with previous studies could be due to the constant and relatively high water temperatures in Dean's Blue Hole, even at 60- to 70-meter depths (27°C). This phenomenon might have reduced the diving reflex and slightly altered the divers' performances in this study. Muscular effort is intense at the beginning of the ascent and slows as the diver approaches the surface. Despite this, we noted that the HR in the first stage of the ascent did not rise much. It was only in the last phase of the ascent, when the BH divers considerably step down their muscular effort (20 to 25 seconds before surfacing), that the HR increased. The lack of HR increase in spite of muscular exercise has been described during BH dives [8] but this is the first observation of a delayed increase in HR during deep constant-weight dives. Diving reflex bradycardia and exercise under water exert opposite influences on HR. Most evidence shows that, during the ascent phase of the constant-weight dive, the diving response is powerful enough to override the exercise tachycardia for the period of breath-holding [7,11]. The diving reflex diminishes exercise HR by 11-36% [20].

ECG data

This is the first study utilizing the 12-lead ECG in real-time elite breath-hold dives. Although there is much published data on HR variations or disturbances in conduction and cardiac rhythm during breath-hold dives, we have no data concerning the changes in the ECG itself. The major and quite surprising result of this study was the lack of change in the QRS axis. The hypothesis was an expected right ventricular overload [35] with a right shift of the QRS axis. Yet no QRS axis changes were found in any of the divers of this study. The explanation may be that left afterload is also very high during BH diving, since peripheral resistance is increased due to vasoconstriction during diving [9,18]. This is not the case during clinical right heart overload, such as in acute pulmonary embolism. It may be that the left heart overload during a dive, which is due to peripheral vasoconstriction, balances the expected QRS axis deviation. This therefore suggests that, with respect to the ECG findings, the workload increase was equivalent for the right and the left ventricles.

Stability of the QRS axis and echographic left-right ventricular balance were previously observed in experimental human saturation dives down to 701 m [27,28]. During these dives, the increase in pressure of the ventilated compressed gas was responsible for a substantial increase (x 4) in intrathoracic pressure, exerting an external load on the heart. On the other hand, during this experiment, we also observed stability of the QRS axis during glossopharyngeal insufflation, which induces a mean flow decrease in the pulmonary artery [15] and hypotension [42], with no associated BH diving blood shift. Thus, although the lack of QRS axis variation is a concept widely acknowledged with regard to loaded compressed-air ventilation, this phenomenon was also observed in these BH divers in whom the increase in both right and left afterloads was highly significant. However, there may be a potential to mask QRS axis changes from immersion by changes also seen in body position.

Cardiac arrhythmias and conduction disturbances

The DR is the combination of bradycardia with cardiac arrhythmias, due to vagal inhibition of sinus pacing combined with sympathetically induced enhancement of automaticity in other latent pacemakers enhancing ectopic beats. Other arrhythmogenic factors likely to operate during breath-hold diving are the distension of the heart due to high intrathoracic blood volume and an increased afterload. Hypoxia due to prolonged breath-holding and acid blood venous return, due to the reversal of peripheral vasoconstriction upon surfacing, are also arrythmogenic factors. This might explain why PACs were observed in two divers after surfacing. One study found ECG changes indicative of subendocardial ischemia immediately post-dive [37]. These changes, which were absent in non-submersed breath-holds, included (but not limited to) ST depression, heightened T wave, and slowed repolarization with the addition of a positive U wave to the QRS complex.

Concerning the atrial fibrillation episode observed in one diver, it occurred just after glossopharyngeal insufflation. Glossopharyngeal insufflation compresses intrathoracic vessels, increases intrathoracic pressure, and decreases venous return, thus compromising cardiac output [5]. This maneuver can lead to symptomatic arterial hypotension and syncope [14].

Asystole was recorded in one diver during glossopharyngeal insufflation, which indicates that susceptible individuals may be at risk of a serious cardiac incident if the lungs are excessively overinflated by glossopharyngeal insufflation [1]. In addition, we observed short periods of junctional rhythm in two divers at the bottom of their dives. This is a common observation due to the intense vagal stimulation, which is maximal during this phase of the dive.

In a later study, submersed wet dives to 55 meters in a pressure chamber were performed by three experienced breath-hold divers (all from the same family); after an initial tachycardia, their heart rates fell to 20-30 beats.min^{-1} near the "bottom." The longest R-wave to R-wave intervals corresponded to instantaneous heart rates of 8, 13, and 24 beats.min^{-1} [18]. Furthermore, a very high frequency of premature and inhibitory arrhythmias was recorded during their dives in cool (25°C) water, whereas such disturbances were much less prevalent in dives in thermoneutral (35°C) water [18]. Marked but asymptomatic bradycardia (temporarily 5-6 beats.min^{-1}) induced by apnea with cold-water face immersion has also been reported [3]. The diving reflex is strongly related to water temperature and may lead to cardiac dysrhythmias with increasing incidence of supraventricular extrasystoles, particularly at the end of the dives [8,19,38]. This phenomenon is believed to be caused by cardiac dilatation resulting from the dive-related increase in intrathoracic blood volume [16,18,19]. Furthermore, the incidence of such arrhythmias markedly increases at low water temperatures [22].

Oxygen saturation and lactate concentration

Our results are in agreement with Andersson *et al.* [2], but we found higher SaO_2 values at surfacing than previously reported by Ferretti *et al.* [17] at rest. With similar BH depth and dive durations in one elite diver after an assisted dive: Ferretti *et al.* [17] found an SaO_2 of 40%, very far from our results (87.3 ± 5.5% at surfacing), which may indicate a higher training status and perhaps a stronger diving response in our BH divers. This latter point seems to be supported by the similar lactate results.

Joulia *et al.* [26] found that BH training may reduce lactate accumulation, which would explain the similar lactate values found in our study but for longer BH duration. The high buffering capacity resulting from BH training will contribute to this effect [16]. However, we were unable to determine whether this lactate increase was due to vasoconstriction during diving, or just hypoxia or exercise. In addition, we found an association between the change in SaO_2 and the BMI and between the percentage of HR change and %FM. In fact, the more BMI and %FM increased, the more SaO_2 and %HR change respectively diminished. This finding may indicate that constitutional factors can enhance the susceptibility for arrhythmias. This result was found by other authors [23] in a group of 16 recreational BH divers with comparatively different BMI (23.9 ± 2.2 *vs.* 22.8 ± 1.4 $kg.m^{-2}$, respectively). It is noteworthy that arrhythmias in marine mammals, which have high body fat mass for thermal insulation, are rarely seen [10].

These results observed in elite BH divers cannot be generalized to less trained BH divers because:

(i) elite BH divers probably experience fewer arrhythmias than less well- trained divers, and

(ii) the unexpected lack of change in the QRS axis may be explained by their diving training status and their ability to sustain a high left afterload during BH diving to equilibrate their right afterload.

CONCLUSIONS

No ECG axis alterations, not even a right shift of the QRS axis despite the sizable blood shift at these depths, were observed. Yet, some of the BH divers showed rare cardiac arrhythmias occurring mainly after surfacing and conduction alterations at the bottom of the dives but with no signs or symptoms. It appears that elite BH divers exhibit an intense DR which is powerful enough to override the exercise tachycardia during the ascent phase of the BH dive. In additional, the correlation between SaO_2 and BMI and the correlation between %HR change and %FM deserve further investigation.

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