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ECG changes during the experimental human dive HYDRA 10 (71 atm/7,200 kPa)

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Lafay V, Barthelemy P, Comet B, Frances Y, Jammes Y. ECG changes during the experimental human dive HYDRA 10 (71 atm/7,200 kPa). Undersea Hyperbaric Med 1995; 22(1):51-60.—Electrocardiogram (ECG) analysis was performed in three human divers during a 71 atm (7,200 kPa) saturation dive (COMEX HYDRA 10 experiment). The inhaled gas mixture was slightly hyperoxic; its composition was basically helium and oxygen. Hydrogen was introduced during compression and its partial pressure reached 20 atm. ECG changes were the same in the three divers. Marked bradycardia rapidly appeared at the beginning of compression, then this response adapted throughout the dive. P–R, QRS, and Q–T intervals and the S–T segment did not change significantly. The QRS axis remained stable. However, a rightward shift occurred in P and T vector angles. These changes were correlated with time and gas density, respectively. The modifications of ventricular repolarization during compression are similar to those we observed during the HYDRA 9 COMEX dive. They may correspond to changes in duration of myocardial cell repolarization due to increased intrathoracic pressure changes with dense-gas breathing. A marked global diminution of voltage occurred during the decompression period. This suggests that accumulation of micro bubbles in tissues may influence the impedance, causing an artifact in the amplitude of ECG complexes.

electrocardiogram, deep diving, saturation dive, hyperbaria, hydrogen

Few data on electrocardiograph (ECG) changes in humans during experimental deep dives have been published. Most reports focused on the changes in heart rate, and described hyperbaric bradycardia (1–7) with postdive tachycardia (2,8). However, the cardiac response was inconsistent in other works (9,10). Variations in ECG complexes under high ambient pressure are not well known. Wilson et al. (7) first reported ECG changes in 10 divers during four N_2–O_2 saturation dives. These authors observed hyperbaric bradycardia in all subjects, and in some individuals they described transient alterations in myocardial repolarization with decreased Q–T interval and peaked T waves. Decreased P wave amplitude and nodal rhythm combined with reduced right intraventricular conduction

velocity were observed in one subject, and premature ventricular contractions occurred in another diver. Normalization of ECG recordings was observed after inspired O_2 partial pressure was elevated, suggesting the involvement of myocardial hypoxia. Eckenhoff and Knight (2) reviewed data on ECG changes reported in 81 divers during eight air saturation dives (1.06–5.4 atm). Besides bradycardia, they described supraventricular arrhythmias attributed to a high level of parasympathetic tone.

Recently, we reported ECG changes in three divers during the COMEX HYDRA 9 experiment (23.5 atm) (10). We found an increase in amplitude of the T wave, which was correlated with the gas density but independent of the composition of the inspired gas mixture containing helium or a mixture of helium and hydrogen. In one diver we also found a decrease in atrioventricular conduction velocity, correlated with gas density. Heart rate, QRS duration, and Q-T interval did not vary during this dive. In awake dogs, the same alterations in T waves were reported during hyperbaric experiments conducted up to 91 atm (10). In addition, peaked T waves were also present in these animal experiments.

The effects of the hyperbaric environment on ECG were interpreted as the consequences of intrathoracic pressure changes due to breathing dense gas. Indeed, during the ENTEX 11 (46 atm) human dive, our group measured a marked pleural pressure shift toward negative values during eupneic ventilation (11). This condition, similar to an inspiratory threshold load, may induce hemodynamic and ECG changes. Moreover, the hydrostatic pressure and the chemical composition of inspired gas mixture may also influence the electrical cardiac activity, as demonstrated by Örnhagen and coworkers in in vivo and in vitro experiments (12).

The aim of the present study was the description and analysis of cardiac rhythm and the characteristics of ECG complexes in three divers participating in a 71 atm saturation dive using $He–O_2$ and $H_2–He–O_2$ gas mixtures (COMEX HYDRA 10 experiment).

MATERIALS AND METHODS

HYDRA 10 is the deepest experimental human dive carried out so far. It was conducted in the hyperbaric center of COMEX in Marseilles, between 2 November and 14 December 1992. Three professional divers (D1, D2, D3), all 39 yr old, volunteered to participate. They were healthy and free of any cardiovascular and pulmonary abnormalities. The protocol was approved by the ethical committee of the establishment.

The dive lasted 42 days: 15 days of compression, a 3-day stay at about 68 atm with one excursion to 71 atm for one diver (D2), and 24 days of decompression (Fig 1). Table 1 shows the experimental conditions: the gas density varied from 1.18 g/liter ATPS (sea level) to 11 g/liter ATPS (71 atm). The chamber temperature, depending on the gas density, was maintained at an optimum level of 31°C. PI_{O_2} value was 400 mbar throughout the compression, 500 mbar during most of the decompression period, and 600 mbar for the last 5 days of the hyperbaric experiment. The inhaled gas mixture was basically $He–O_2$, and H_2 was introduced at 21 atm. Maximal PI_{H_2} value was 20 atm (Fig 1).

Fifteen ECG recordings were performed in each diver. Control data at sea level (1 atm) were obtained 2 days before the dive, and sea level measurements were repeated 24 days after the end of decompression (Table 1). ECG was recorded in resting supine subjects with a digital ECG (Marquette MAC PC 12 SL). Twelve ECG leads (DI, DII, DIII, aVR, aVL, aVF, and V1–V6) were recorded with silver electrodes plus saline gel. A large thoracic rubber strap supported silver electrodes for V1–V6 ECG leads.

Electrocardiograph activities were automatically analyzed by a computer-assisted method for consecutive 10-s periods and systematically controlled by a qualified physician. Total

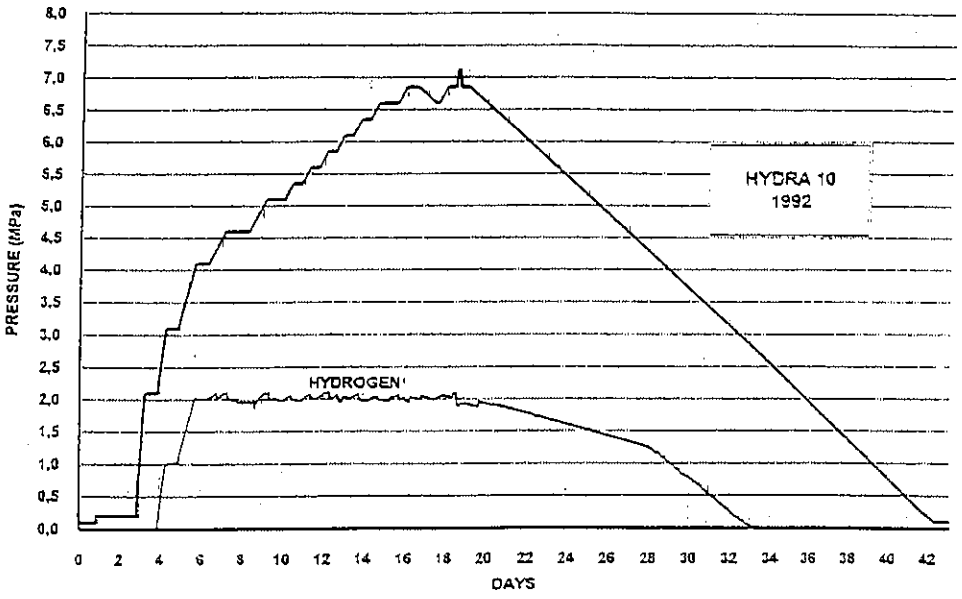

FIG. 1—Compression profile and hydrogen partial pressure during HYDRA 10.

Table 1: Experimental Conditions Associated with the Periods of ECG Recordings During the HYDRA 10 COMEX Experiment

Days of Dive	Total Pressure, atm abs	Density, g/liter ATPS	Gas Mixture	Pi_{H_2}	Pi_{He}	Pi_{O_2}
Day −2	1.0	1.18	Air			0.21
Day 2	2.0	1.54	$He–O_2$	10.0	20.6	0.4
Day 4	31.0	5.39	$H_2–He–O_2$	20.0	23.1	0.4
Day 6	43.5	6.60	$H_2–He–O_2$	20.0	25.6	0.4
Day 7	46.0	7.00	$H_2–He–O_2$	20.0	30.6	0.4
Day 9	51.0	7.80	$H_2–He–O_2$	20.0	35.6	0.4
Day 11	56.0	8.60	$H_2–He–O_2$	20.0	42.4	0.4
Day 13	62.8	9.70	$H_2–He–O_2$	20.0	48.1	0.4
Day 15	68.5	10.61	$H_2–He–O_2$	20.0	50.6	0.4
Day 16	71.0	10.99	$H_2–He–O_2$	19.3	46.2	0.4
Day 17	66.0	10.21	$H_2–He–O_2$	19.0	45.5	0.5
Day 20	65.0	10.10	$H_2–He–O_2$	16.0	38.5	0.5
Day 23	55.0	8.62	$H_2–He–O_2$	13.5	32.9	0.5
Day 26	46.4	7.35	$H_2–He–O_2$		20.5	0.5
Day 35	21.0	4.40	$He–O_2$		5.4	0.5
Day 40	6.0	2.21	$He–O_2$			0.6
Day 66	1.0	1.18	Air			0.21

duration of ECG recording in each individual and each saturation was 60 s. Analysis was conducted in terms of:

1. QRS, P–R, S–T, and Q–T segments durations (in ms). Q–T was normalized with respect to R–R value, and called Q–Tc.
2. P, QRS, and T wave amplitudes and S–T segment position (in μV). Wave amplitudes were corrected considering the variations of P, T, and QRS vector frontal angles. Trigonometric computation of the true value of the amplitude of waves was performed from two to three ECG leads. Reported data corresponded to the maximal amplitudes of P, T, and QRS waves.
3. P, QRS, and T vector angles (in degrees).

Statistical analysis

Linear regression analysis was used. ECG changes were plotted against the ambient pressure, or gas density, or time (duration of diving from the beginning of compression to the end of decompression). In each individual, one-way analysis of variance was performed to assess the linearity of the population regression. The value of r was tested against zero. P values less than 0.05 were considered significant.

RESULTS

Heart rate

Bradycardia occurred at the beginning of compression. Heart rate (HR) increased progressively throughout the experiment and these changes were significantly correlated with time in each subject: D1: $r = 0.672$, $P < 0.01$; D2: $r = 0.844$, $P < 0.001$; D3: $r = 0.739$, $P < 0.001$ (Fig. 2). Control values of HR were returning toward normal during the last days of the dive. No correlation was found between HR and ambient pressure or gas density.

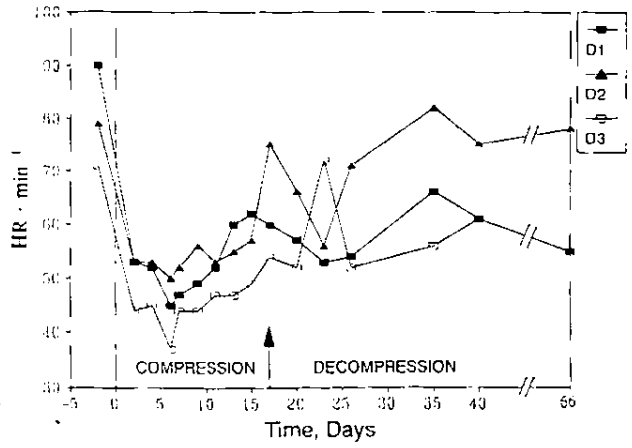

FIG. 2—Individual HR values against time. *Arrow* indicates maximal pressure level.

P-R, Q-Tc intervals, and QRS duration

The QRS duration and Q-Tc interval did not change significantly during the dive. Only the P-R interval was positively correlated with time in one diver (D1): P-R (ms) = 0.971.time (day) + 153 ($r = 0.834$, $P < 0.001$). No correlation was found between P-R interval and ambient pressure or gas density.

P, QRS, and T vector angles

The QRS vector angle did not change significantly. A rightward shift of the T vector was measured during compression in the three subjects, and these changes are significantly correlated with gas density and not with time: D1: $r = 0.603$, $P < 0.05$; D2: $r = 0.739$, $P < 0.001$; D3: $r = 0.660$, $P < 0.01$ (Fig. 3). A rightward shift of the P vector was also measured, but these changes were highly correlated with time (D1: $r = 0.814$; D2: r = 0.971; D3: $r = 0.844$; $P < 0.001$) and not with gas density (Fig 4).

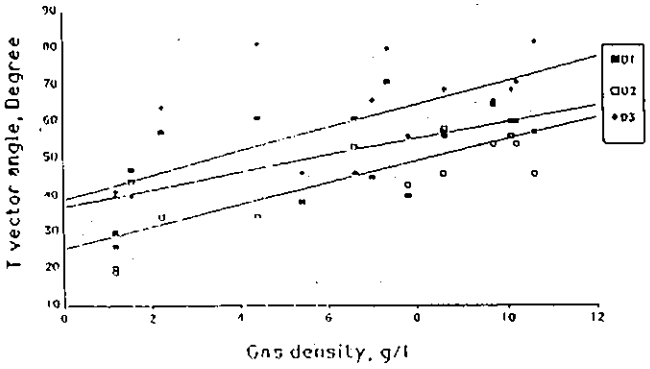

FIG. 3—Individual values of T vector angle plotted against the gas density.

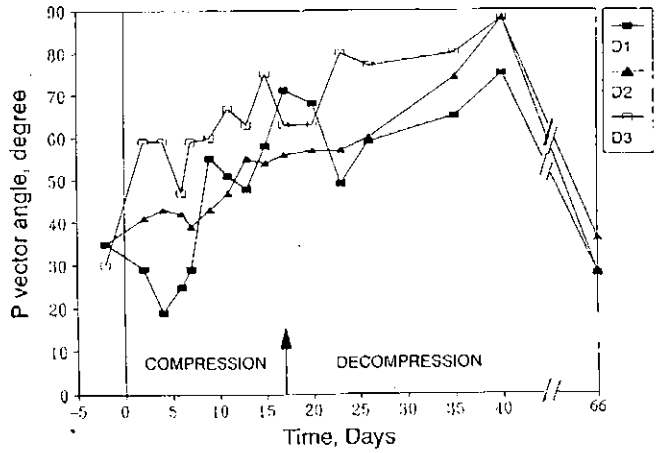

FIG. 4—Changes in individual values of P vector angle during the dive. *Arrow* indicates maximal pressure level.

QRS, P, and T wave amplitudes

Amplitude of the QRS complex did not change significantly during compression (Fig. 5). However, the QRS amplitude decreased at the beginning of decompression, reaching a plateau value (−40%) when the gas density ranged within 7–2 g/liter. Profiles of changes in P and T wave amplitudes were similar during this dive. No significant change occurred during compression despite the changes in composition of the breathing mixture. During decompression, P and T wave amplitudes decreased until the gas density reached 2 g/liter (last day of measurement during the dive). Thus, the end of decompression was characterized by a marked diminution of voltage of QRS (−40%), P (−30%), and T waves (−50%).

S–T segment analysis

No significant modification of the S–T segment was found in any subject during the experiment. Only weak changes, without any negative voltage, were measured in V1 to V4 leads. These alterations were not the same in the three divers.

Electrocardiograph changes obtained in one diver (D2) at 71 atm are not presented because they did not differ from those observed in the same individual at 68.5 atm.

DISCUSSION

The main results of the present study are:

1. A rightward shift of the T vector axis, correlated with the density of inspired gas.
2. A progressive rightward shift of the P vector axis, correlated with the duration of the dive, including the decompression period.
3. A diminution of voltage of all ECG complexes during decompression.

These ECG alterations seemed not to be significantly correlated with hydrogen partial pressure and were no longer observed 24 days after the end of decompression. Except for the diminution of voltage, all ECG changes were found after automatic analysis and so could not be assessed by clinical interpretation.

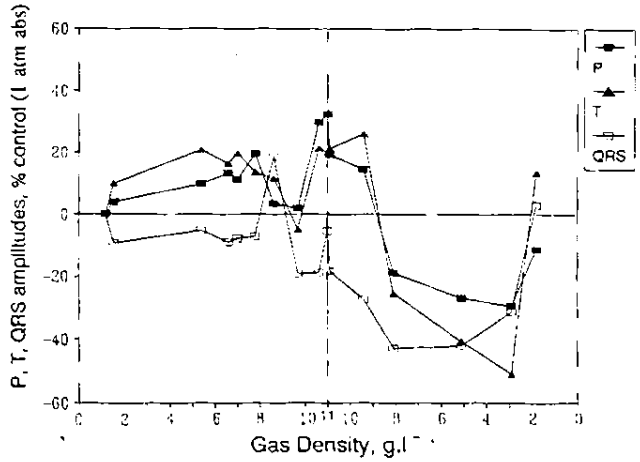

FIG. 5—Relative change in amplitude of P, T, and QRS waves, expressed in percent of control values (1 atm abs) during the dive. *Arrow* indicates maximal pressure level.

We found no correlation between ECG changes and eventual high pressure nervous syndrome (which will be described in a subsequent publication).

A marked bradycardia was only present at the beginning of the dive, corroborating other human observations (13–15). Meanwhile, control values may be considered high for healthy professional divers and were probably due to some anxiety before the dive. As we have reported, during the HYDRA 9 COMEX experiment, hyperbaric bradycardia was not related to the ambient pressure nor to the composition of inhaled gas mixture. Flynn et al. (14) also reported that heart rate depression was independent of pressure and density for deep dives. In fact, bradycardia was not detected when in vivo and in vitro hyperbaric studies were performed under normoxic or slightly hyperoxic conditions (16–18). Adaptation of HR changes throughout the dive including decompression may correspond to an adaptive cardiac response to chronic hyperoxia, the sole parameter that did not vary under these circumstances.

No alteration in intraventricular conduction velocity and ventricular repolarization time were found. These results suggest an absence of marked changes in electrolyte balance (namely, calcium and potassium concentrations). The progressive increase in atrioventricular conduction time with time, reported in one diver, was not correlated with the density or the gas mixture composition. This observation differs from that reported previously in humans (7,10) or animals (10). This suggests the possible interaction of hyperbaric and hyperoxic conditions on atrioventricular conduction velocity, which is partially regulated by parasympathetic tone and reveals marked interindividual variations in the cardiac response to these extreme environments.

The absence of changes in QRS vector angle during hyperbaric exposure confirms previous data (2,10). This demonstrates that elevation of ambient pressure or the changes in intrathoracic pressure, or both, did not affect the intraventricular conduction velocity or induce marked variation in the anatomical position of the heart in the thorax. This negative result also allows one to exclude the existence of severe ventricular ischemia or marked ventricular overload.

Isolated rightward motion of the T vector, correlated to gas density, is similar to the desynchronization of changes in T and QRS vectors already observed during the HYDRA 9 experiment (10). This could be related to mechanical factors, namely, the modifications of intrathoracic pressure which could involve changes in duration of myocardial cell repolarization.

The progressive changes in P vector angle during the stay under high pressure, including compression and decompression periods, have never been reported before. Anatomic variations, atrial ischemia, intra-atrial alteration of conduction velocity, or a wandering pacemaker do not seem to be involved. We speculate the existence of a right atrial overload. Two explanations are proposed which may combine:

1. *Pressure-dependent overload.* This was suspected by Hebden et al.(19) and Tao et al. (20). These authors reported an increase in urinary excretion of atrial natriuretic factor (ANF) during hyperbaric human studies. This may partly explain the hyperdiuresis often observed under hyperbaric conditions. Pressure-dependent atrial overload could result from a rise in right preload as a consequence of increased negative intrathoracic pressure associated with breathing high density gas (16). This explanation is supported by the variations in gas density with elevated ambient pressure, but it cannot explain the persistence of a rightward shift in P vector during decompression.
2. *Modifications of body-fluid volumes.* Previous investigators (19–22) have suspected the existence of hypovolemia as a result of increased ANF secretion and decreased blood Pitressin concentration during prolonged hyperbaric stays. Furthermore, human obser-

vations under clinical practice commonly attribute right atrial electrical overload as "pulmonary P wave" changes to acute hypovolemia (23). Chronic hypovolemia could induce similar changes that are generally classified as "electrical overload" and may combine with pressure-dependant overload.

The most striking finding of the present study is the observation of marked diminution of voltage during decompression. This phenomenon began early in decompression in the QRS complex. Decreases in P and T wave amplitude occurred later. Decrease P wave amplitude (−30%) was less than the changes in T and QRS waves (−50 and −45%, respectively). This discrepancy may be related to the persistence of atrial overload throughout the decompression. Diminished voltage could result from depressed cardiac electrogenesis or from altered ECG signal transmission. The first hypothesis may be rejected because of the absence of clinical manifestations associated with such marked depression of electrogenesis. Altered intrathoracic ECG transmission is generally observed under two clinical circumstances: pericardial effusion and pulmonary emphysema. However, the divers never showed symptomatic manifestations of pericardial effusion, and if increased intrathoracic volume can be suspected during compression (24), it probably did not occur during decompression. Thus, these hypotheses are not satisfying and need further study during decompression in the next human saturation dives to determine whether lung volume changes under these circumstances.

Another way to explain ECG signal transmission must be discussed. Helium or hydrogen oversaturated tissues may not conduct an ECG signal in the same way as they do at sea level. Accumulation of microbubbles increases tissue resistivity for electric signal transmission. This has been described in the works of Hills (25) and Searle and Hills (26), and described in chronic preparations in rats and dogs by Jossinet et al.(27). Thus, the pattern of change in ECG wave amplitude during decompression could be compared with the profile of tissue desaturation, which started at the beginning of decompression and lasted until ambient gas pressure reached atmospheric values. If this hypothesis is correct, the measure of bio-impedance should be a non-invasive method to evaluate the supersaturation and gas formation level of biological tissues during decompression.

This study showed modifications of the ECG during compression and decompression. Right atrial overload, changes of myocardial cells repolarization durations may be suspected. Alteration of ECG transmission due to oversaturated tissues may explain the marked diminution of voltage observed during decompression. These observations need to be confirmed during future saturation dives.

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