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ORIGINAL ARTICLE

ELECTROCARDIOGRAPHIC CHANGES DURING HYPOBARIC HYPOXIA

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Introduction: Acute hypobaric hypoxia during high-altitude exposure may affect cardiac autonomic regulation and, consequently, ECG intervals and morphology; however, published findings remain heterogeneous due to differences in altitude, exposure profile, and measurement methodology. The aim of this study was to assess short-term ECG changes during routi ne hypobaric hypoxia exposure to 5,000 m above sea level (a.s.l.) in clinically healthy young men.

Materials and methods: The study included 68 healthy men (17–24 years) undergoing a standardized hypobaric-chamber (HC) protocol comprising baseline, ascent, a 30-minute stay at 5,000 m a.s.l. (six consecuti ve 5-minute phases), and descent. Continuous 3-lead Holter ECG monitoring and pulse oximetry were performed throughout the exposure. Heart rate (HR), PQ, QRS, QT, QTc, ST-interval, and R- and T-wave amplitudes were evaluated using repeated-measures analysis with appropriate corrections; associati ons between ECG parameters, HR, and peripherial capillary oxygen saturation (SpO_2) were assessed.

Results: Mean SpO_2 decreased from 96.87±0.97% at baseline to 79.85±3.11% at 5,000 m a.s.l., while HR increased from 85.64±14.22 bpm to 98.20±8.03 bpm. HR showed significant inverse associati ons with SpO_2 within each study stage ($p<0.001$). No rhythm disorders were observed during any stage of chamber exposure. Across successive 5-minute phases at 5,000 m a.s.l., PQ, QRS, and QT intervals showed progressive shortening, whereas QTc demonstrated only a small, non-signifi cant tendency toward prolongati on. No measurable ST-interval deviati on was detected. Changes in R-wave amplitude and T-wave morphology were non-significant.

Conclusions: There are significant negative correlations between oxygen saturation and heart rate. The durations of the PQ interval, QT interval, and the QRS complex shortened significantly during ongoing hypoxia, including the 30-minute stay at the simulated altitude of 5,000 m a.s.l. in the HC (while remaining within the normal range). These changes may reflect integrated chemoreflex-mediated autonomic–chronotropic adaptati on to hypoxemia.

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In contrast, R- and T-wave amplitudes, QTc duration, and the ST interval did not change significantly.

A routine altitude-hypoxia tolerance test performed at a simulated altitude of 5,000 m a.s.l. in the HC in this cohort did not reveal any clinically relevant changes in ECG morphology. Accordingly, continuous rhythm and heart rate monitoring may be sufficient for surveillance in similar low-risk settings. However, extrapolation to other populations, longer exposures, or higher-risk individuals should be approached with caution.

Keywords: Keywords: hypobaric hypoxia, pilots, electrocardiography

INTRODUCTION

Acute altitude hypoxia is considered to be one of the most significant threats to the pilot's body during high-altitude flight in military aviation [5,10,12]. The level of tolerance to altitude hypoxia is an individual matter and, up to an altitude of 4,000 m a.s.l., may vary considerably [2,6,15]. The manifestation and severity of symptoms resulting from hypoxia are influenced by numerous factors, including, but not limited to, altitude and the rate of ascent [2,6]. A decrease in the partial pressure of oxygen (pO_2) in the ambient atmosphere results in a reduction in its concentration in arterial and venous blood. This, in turn, leads to a decrease in the partial pressure of oxygen in tissues [2,5,15]. The effects of hypoxic hypoxia on the human body have been the subject of research and publications for many years. A significant number of studies have been conducted on cardiovascular responses, with some also examining the effect of altitude hypoxia on ECG morphology [13,19,23]. However, the effects on the electrocardiogram under hypoxic conditions have been assessed at various altitudes in individuals of different ages and levels of physical fitness [13,23]. The present paper is consistent with this line of research.

AIM OF THE STUDY

The aim of the study was to assess the effect of the hypobaric hypoxia exposure on the morphology of ECG parameters at a simulated altitude of 5,000 m a.s.l. in the HC in clinically healthy young men.

MATERIALS AND METHODS

The study group comprised 68 candidates for the Polish Air Force Academy aged 17–24 years (mean age: 19.6 years). All participants had normal results from laboratory and diagnostic evaluations, including hemoglobin concentration, serum sodium and potassium levels, thyroid-stimulating hormone levels, echocardiography, and a standard resting 12-lead ECG.

Participants did not use any pharmacological agents or stimulants. They were clinically healthy and had been cleared by an aeromedical commission to undergo routine hypoxia-tolerance testing in the low-pressure chamber.

Ethics statement: The study protocol was approved by the relevant Military Institute of Aviation Medicine bioethics committee (approval No. 14/2018), and all participants provided written informed consent.

Continuous ambulatory ECG monitoring was performed using a DMS 300-4A digital Holter recorder integrated with the CardioScan 12 system. Recordings were obtained in a three-lead configuration, yielding three bipolar Holter channels with precordial orientation approximating V1-, V5-, and V6-like vectors (hereafter referred to as V1-like, V5-like, and V6-like; these are not equivalent to standard 12-lead precordial leads). The ECG signal sampling rate was 4,096 samples/s. Recordings were time-aligned with the hypobaric chamber protocol and analyzed according to predefined stages and phases. Voltages (in mm) and durations of ECG intervals were evaluated (amplitudes of P, Q, R, S, and T waves with the PQ interval as the isoelectric baseline), as well as PQ interval, QRS duration, QT interval, and heart-rate-corrected QT (QTc) interval calculated using Bazett's formula. ECG intervals and amplitudes were measured manually by a trained physician in each channel using uniform criteria. Values were derived from artifact-free sinus beats within each predefined stage/phase after excluding ectopic beats and intervals with baseline instability; where more than one eligible beat was available, measurements were averaged within the interval and, when appropriate, across channels. Hemoglobin oxygen saturation was monitored continuously using pulse oximetry throughout the hypobaric chamber exposure. Examinations were conducted in the morning, after a normal night's sleep and after a meal (at least 1 hour before testing). The protocol was conducted under standard chamber conditions (temperature 17°C). Participants were instructed to remain in a stable sitting position and minimize

movement and conversation during the analyzed intervals to reduce motion artifacts.

For the assessment of ECG-Holter morphology, the HC examination was divided into four stages: Stage 1—baseline; Stage 2—ascent to a simulated altitude of 5,000 m a.s.l. over 7 minutes; Stage 3—stay at 5,000 m a.s.l. for 30 minutes; and Stage 4—descent/return to sea-level pressure in the chamber (760 mmHg) over 7 minutes. In addition, Stage 3 was subdivided into six consecutive 5-minute phases: Phase 1 (0–5 min), Phase 2 (5–10 min), Phase 3 (10–15 min), Phase 4 (15–20 min), Phase 5 (20–25 min), and Phase 6 (25–30 min). Morphological analysis of Holter ECG recordings was performed for each stage and phase of the test. The time points corresponding to successive simulated "altitudes" were marked by participants by pressing an appropriate button on the recording device. Changes in the rate of altitude increase in the HC corresponded to a pressure decrease of approximately 0.5 mmHg/s. During the hypobaric chamber exposure, peripheral oxygen saturation was continuously monitored using pulse oximetry.

Statistical analyses were performed using STATISTICA. Normality was evaluated using the Kolmogorov–Smirnov test. Within-subject differences across protocol stages/phases were assessed using one-way repeated-measures ANOVA (RM-ANOVA). Sphericity was assessed with Mauchly's test and, when violated, degrees of freedom were adjusted using the Greenhouse–Geisser correction. Post-hoc pairwise comparisons were adjusted for multiple testing using Bonferroni correction. Associations between variables were evaluated using Pearson's correlation coefficient. Results are presented as mean ± SD, and statistical significance was defined as two-sided $p \leq 0.05$.

RESULTS

Mean values and standard deviations of ECG parameters for each stage of the study are presented in Table 1.

Mean values and standard deviations of HR and SpO_2 for the entire period and each stage of the study are presented in Table 2.

Differences in mean SpO_2 and HR values between each study stage are presented in Figures 1 and 2.

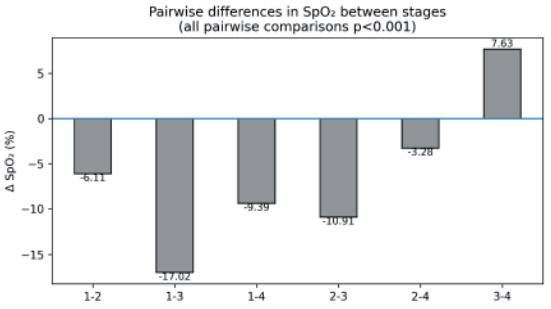


Fig. 1. Differences in mean SpO_2 values between each study stage.

SpO_2 – peripheral capillary oxygen saturation; 1 – first study stage (baseline); 2 – second study stage (ascent to 5,000 m a.s.l.); 3 – third study stage (at an altitude of 5,000 m a.s.l.); 4 – fourth study stage ("descent").

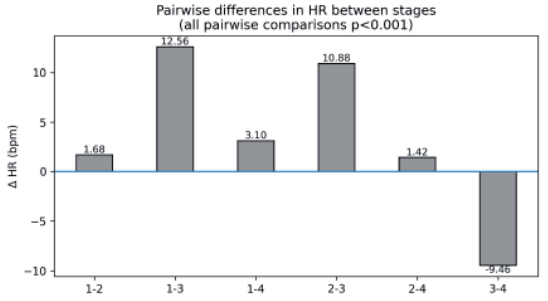


Fig. 2. Differences in mean HR values between each study stage.

Tab. 1. Mean values and standard deviations of ECG parameters for each stage of the study.

Stage	PQ [ms]	QRS [ms]	QT [ms]	QTc [ms]	R [mm]	T [mm]
1	140.33±11.5	94.05±6.2	348.65±10.6	416.54±11.1	16.67±2.8	4.00±0.3
2	131.68±9.1	88.00±5.1	317.67±12.1	418.23±10.9	16.00±1.7	3.70±0.9
3	135.54±6.3	91.09±7.2	334.88±15.3	428.42±8.4	17.68±1.3	3.58±0.2
4	138.72±3.3	99.32±4.9	340.00±16.2	417.49±14.2	16.24±1.5	4.34±0.7

1 – first study stage (baseline); 2 – second study stage (ascent to 5,000 m a.s.l.); 3 – third study stage (at an altitude of 5,000 m a.s.l.); 4 – fourth study stage ("descent").

Tab. 2. Mean values and standard deviations of HR and SpO_2 for the entire period and each stage of the study.

	Entire period	Stage 1 Baseline	Stage 2 Ascent	Stage 3 (5000 m a.s.l.)	Stage 4 Descent
			Mean ± SD		
SpO_2	82.02±5.52	96.87±0.97	90.76±2.04	79.85±3.11	87.48±4.04
HR	96.05±10.15	85.64±14.22	87.32±11.79	98.20±8.03	88.74±12.82

SpO_2 – hemoglobin oxygen saturation; HR – heart rate

During the HC study, SpO_2 ranged from 96% to 76%, with significant differences between individual stages. During hypobaric exposure, heart rate ranged from 57 to 138 beats/min and differed significantly across all study stages. Significant inverse correlations between SpO_2 and HR were observed within each stage (all $p < 0.001$).

No heart rhythm disturbances were observed during any stage of HC exposure.

Mean values and standard deviations of the assessed Holter ECG parameters in successive phases of Stage 3 (30 minutes at 5,000 m a.s.l.) and one-way repeated-measures ANOVA test results are presented in Figure 3.

No measurable ST-interval deviation was detected across stages within the resolution of the three-channel Holter method; therefore, ST-related variability could not be quantified.

Significant phase effects were observed for: HR values, PQ, QRS, and QT duration. No significant phase effects were observed for: R- and T-wave amplitudes or QTc duration.

PQ interval duration recorded over the entire study remained within the normal range (120 to <200 ms); however, across successive 5-minute phases of Stage 3, it gradually shortened. Significant differences in atrioventricular conduction time were found, depending on the duration of exposure to altitude hypoxia (Phase 1 vs Phases 2–6; Phases 2, 3, and 4 vs Phases 5 and 6).

QRS complex duration recorded over the entire HC study remained within the normal range (<120 ms); however, across successive 5-minute phases of Stage 3, it gradually shortened. Significant differences in intraventricular conduction time were found, depending on the duration of exposure to altitude hypoxia (Phase 1 vs Phase 5; Phase 6 vs Phases 1–5) of Stage 3 of the study.

QT interval duration recorded over the entire HC study remained within physiological limits for healthy young adults; however, across successive 5-minute phases of Stage 3, it gradually shortened. Significant differences in QT interval duration were found, depending on the duration of exposure to altitude hypoxia (Phase 1 vs Phases 4–6; Phases 2 and 3 vs Phases 5 and 6). QTc interval duration showed a small increase across phases relative to Phase 1, but differences were not significant in any phase.

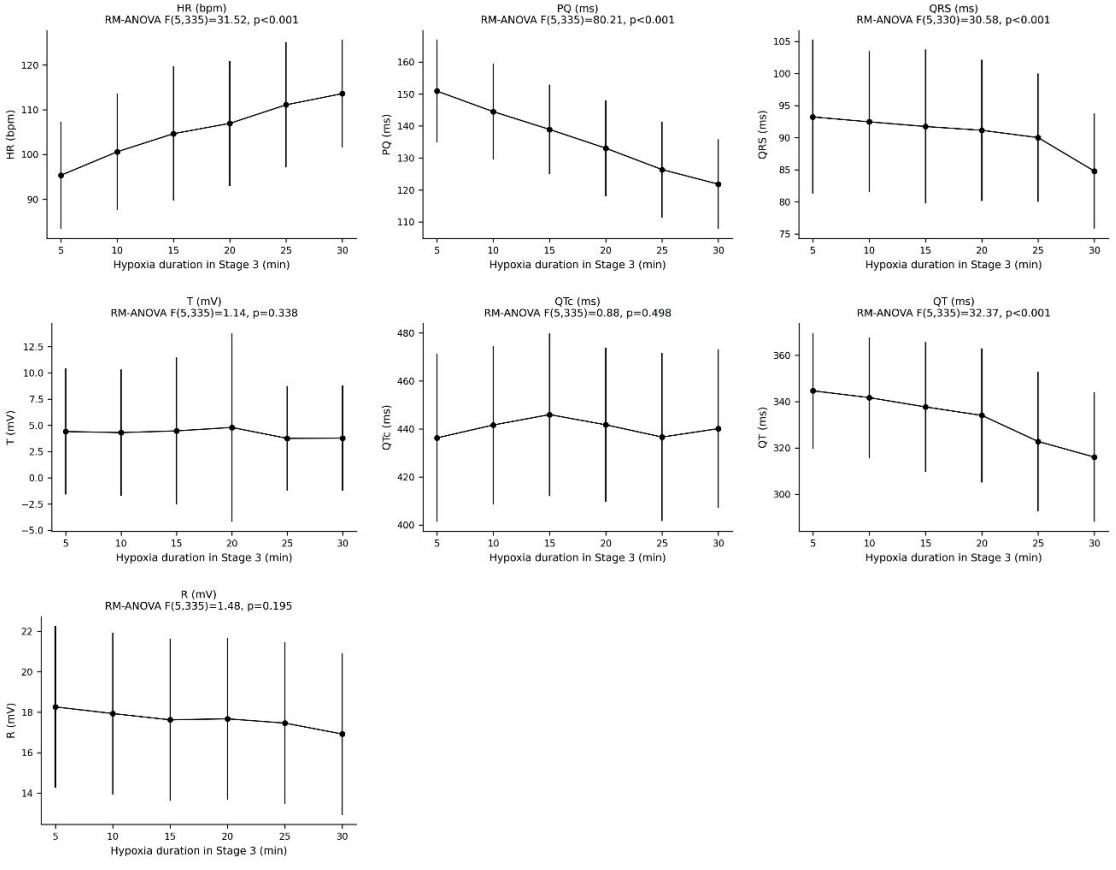


Fig. 3. Mean values and standard deviations of the assessed Holter ECG parameters in successive phases of Stage 3.

PQ – PQ interval; QRS – QRS complex duration; QT – QT interval; QTc – QT corrected interval; R – R-wave amplitude; T – T-wave amplitude; HR – heart rate; time of exposure at 5,000 m a.s.l. presented in minutes (5, 10, 15, 20, 25, 30).

During Stage 3, R-wave amplitude showed a downward trend across successive 5-minute phases; however, these changes were not significant in any phase.

T-wave amplitude recorded over the entire HC study remained within the normal range (<10 mm). As the study progressed through the successive five-minute phases of Stage 3, the T wave became flatter; however, these differences were not significant. Assessment of the relationships between individual components of the ECG recording and SpO_2 during successive 5-minute phases of Stage 3 showed a significant negative association only between HR and SpO_2.

DISCUSSION

The cardiovascular response to high-altitude hypoxia is mediated by a reflex from peripheral arterial chemoreceptors, which are the most sensitive "sensors" of reductions in blood oxygen content. Historically, changes in blood oxygen concentration were attributed exclusively to chemoreceptors in the carotid and aortic bodies. There is now broad consensus that other cells in the human organism can detect acute and chronic alterations in oxygenation. These cells have been shown to respond to such changes through changes in protein activity and the expression of specific genes [21]. In response to hypoxia, changes also occur in the activity of various autonomic nervous system (ANS) components. Many investigators report an increase in sympathetic drive during acute hypoxia; however, biochemical markers of sympathetic activation (plasma/urine catecholamines) do not invariably rise under laboratory conditions, despite increases in direct indices of sympathetic activity (e.g., muscle sympathetic nerve activity), which may result, among other factors, from enhanced catecholamine clearance and differences in exposure protocols [19]. Sevre et al. demonstrated a transient reduction in both sympathetic and parasympathetic activity during a stepwise, 3-day simulated "ascent" in a hypobaric chamber to an altitude of 4,500 m a.s.l.; this could, however, reflect physiological adaptation to the specific exposure profile [22]. Thus, the literature indicates heterogeneity of autonomic responses depending on the mode of exposure (abrupt vs. gradual) and the duration of hypoxia [5,6,15,16,18,22].

Although activation of the sympathetic–circulatory reflex in hypoxia is associated with a range of non-uniform changes in systemic vascular resistance and pulmonary vascular resistance, the net effect of these complex circulatory responses is an acceleration of heart rate [6,11,14,15,18].

In our study, we demonstrated that, as the partial pressure of oxygen decreased across successive stages and phases of the hypobaric chamber protocol, there was a gradual acceleration of heart rate. We also identified significant inverse associations between SpO_2 and HR at each stage of the study. The results obtained are consistent with those reported by other investigators. In numerous studies, an increase in HR has been observed in response to high-altitude hypoxia [7,8,23].

The PQ interval recorded throughout the entire HC protocol remained within the normal range (120 to <200 ms). Nevertheless, during the individual 5-minute phases of Stage 3, gradual shortening was observed. No significant associations were observed between PQ interval duration and SpO_2 at any stage of the HC study.

The PQ interval reflects conduction time from the onset of atrial depolarization to the onset of ventricular depolarization and thus primarily captures atrial conduction and atrioventricular nodal delay, with subsequent propagation through the His–Purkinje system to the initiation of ventricular activation. Its duration shows a physiological dependence on heart rate and autonomic modulation; it typically shortens with increasing heart rate and sympathetic activation and/or vagal withdrawal [4]. At the same time, it should be emphasized that under hypoxic conditions, the net direction of PQ changes may depend on the balance between autonomic influences and potential direct effects of hypoxia on atrioventricular conduction. In our study, the observed shortening of the PQ interval co-occurred with an increase in HR across successive phases of hypobaric exposure, which is consistent with a potential autonomic influence and with results obtained by others [9].

The QRS complex duration recorded throughout the entire HC protocol remained within normal limits (<120 ms); however, across the individual 5-minute phases of Stage 3, it exhibited gradual shortening, indicating significant differences in ventricular depolarization duration depending on exposure time. No significant associations were observed between QRS duration and SpO_2 at any stage of the HC study. The findings reported by Laciga [9] indicated that with increasing altitude, QRS duration in bipolar leads did not differ significantly, whereas in unipolar leads a significant shortening of QRS was observed only in lead V2; the authors considered these changes to be non-characteristic of hypoxia.

Discrepancies among studies may be explained by the fact that QRS duration reflects the global time of ventricular depolarization and is sensitive both to intrinsic conduction properties and to the method of recording and measurement (lead dependence). Changes in heart rate may co-occur with changes in QRS duration; however, the direction of this relationship is not uniform (in some individuals, QRS shortens, whereas in others it widens as HR increases). Conversely, under conditions of severe tissue hypoxia/ischemia, slowed conduction has been described (among other mechanisms related to altered excitability and impaired intercellular coupling), which may promote QRS prolongation; however, this mechanism may not necessarily manifest during high-altitude hypoxia in healthy individuals or when recordings are limited to selected Holter channels. Moreover, in our study, the Holter recording was analyzed in channels corresponding to V1/V5/V6-like orientation, whereas ventricular depolarization generates different vectors depending on the lead, and the identification of QRS onset/offset may differ between channels [4,9,16].

The QT interval duration recorded throughout the entire HC protocol remained within the normal range; however, across the individual 5-minute phases of the study, it exhibited gradual shortening. Significant differences in QT interval duration were observed between phases, indicating an effect related to the duration of high-altitude hypoxic exposure. No significant associations were found between QT interval duration and SpO_2 at any stage of the HC study. Roche et al [17], in a study performed at a simulated altitude of 4,800 m a.s.l., also reported a significant shortening of the QT interval, which is consistent with our findings. Analysis of QTc using Bazett's correction demonstrated prolongation during phases of hypoxic exposure at 5,000 m a.s.l. relative to the first phase; however, these changes were small and not statistically significant. Other studies have reported that the uncorrected QT interval shortens with increasing altitude, whereas the heart-rate–corrected value may prolong [11]. Overall, findings regarding QT/QTc under hypoxic conditions are inconsistent in the literature and depend, among other factors, on altitude level, exposure duration, and the correction method used [16].

During the HC protocol, R-wave amplitude decreased, and T-wave flattening was also observed. These differences did not reach statistical significance, possibly due to the limited number of analyzed leads. These findings are consistent with those of previous studies that have evaluated the effect of hypoxia on electrocardiographic morphology.

Except for the well-characterized effect of hypoxia on peripheral chemoreceptors, the direct consequences of oxygen deficiency on the cardiovascular system—including the ECG pattern in a healthy individual—are difficult to investigate, primarily because of the large number of indirect influences, such as hypocapnia resulting from respiratory alkalosis and activation of the sympathoadrenal system. Despite extensive efforts to evaluate these alterations, the outcomes have remained ambiguous. One study demonstrated that administration of propranolol, a β-adrenergic receptor blocker, resulted in a significant reduction in ST-interval depression and T-wave flattening, when compared to the control group. Conversely, alterations in the depolarization phase—exemplified by a reduction in R-wave amplitude and an augmentation of the QRS complex, which attained statistical significance only in lead V6—occurred in both the propranolol group and the control group, suggesting a possible direct effect of hypoxia [1]. Another study [20], also conducted at a simulated altitude of 6,000 m a.s.l., in which ECG changes at altitude were evaluated without and with β-receptor blockade as well as after inhalation of isoprenaline, demonstrated that β-receptor stimulation also accounts for most ECG changes during altitude exposure, such as shortening of RR intervals, QT prolongation, and particularly T-wave flattening. After "switching off" catecholamine effects at altitude, ECG changes were less pronounced but still significant. The authors attributed these changes to other, not yet fully defined factors, such as myocardial hypoxia, inhibition of vagal activity, or an increase in pulmonary vascular resistance. Other investigators, during high-altitude hypoxia, reported an increase in P-wave amplitude in leads II and III, shortening of the PQ interval, rightward deviation of the cardiac axis, and features of right ventricular overload and hypertrophy. A transient right bundle branch block was also described.

It is also worthwhile to relate our findings to the large study by Coustet et al. [3], which analyzed ECG recordings obtained during moderate exercise in normoxia and hypoxia at a simulated altitude of 4,800 m. The authors reported a systematic reduction in P/QRS/T-wave amplitudes in hypoxia (remaining within physiological limits). The amplitude reduction was associated with exercise-induced desaturation and individual characteristics, suggesting interindividual variability in cardiorespiratory responses to

hypoxia and a possible link to intracellular oxygen availability and ATP-dependent ion-channel activity. In our material, we did not observe a statistically significant analogous "amplitude phenotype" (R and T), plausibly reflecting a different stimulus profile (no exercise), shorter exposure, and the inherent limitations of three-channel Holter monitoring. Both approaches collectively support the idea that, in low-risk populations, ECG changes typically remain within normal limits during short-term hypoxic exposure. Accordingly, in our hypobaric chamber cohort, the ECG pattern did not indicate any clinically significant morphological abnormalities during the analyzed exposure period.

In summary, these data contribute to the expansion of current knowledge by providing a detailed temporal characterization of PQ/QRS/QT/QTc changes in 5-minute intervals during hypobaric hypoxia at 5,000 m a.s.l.. This approach also enabled the assessment of trends that evolved over the course of high-altitude hypoxic exposure, which represents a key strength of the study. In the studied group of young, clinically healthy individuals, longer exposures under such conditions would likely not lead to progression toward the changes described by others [22]. Rather, earlier cerebral symptoms of intolerance to high-altitude hypoxia would be expected. Under rest-and-monitoring conditions, the predominant changes are those consistent with chronotropic loading and conduction adaptation within physiological limits. The direction of axis shifts, ST–T changes, and QTc alterations in altitude studies vary between reports; however, they are most often transient and reversible after return to sea level [16]. Overall, our findings are consistent with broader evidence that ECG changes during temporary high-altitude exposure are often modest, protocol-dependent, and typically reversible after return to normoxia [16].

Limitations. Several limitations in our study should be acknowledged. First, an ECG was recorded using a **3-lead Holter configuration**, which may be less sensitive than a standard 12-lead ECG for subtle repolarization changes; therefore, the absence of measurable ST changes should be interpreted in the context of the recording method. Second, ventilatory parameters and CO_2 indices were not measured, precluding direct assessment of hypocapnia-related effects on ECG intervals. Finally, the protocol involved short-term responses to staged exposure.

CONCLUSIONS

There are significant negative correlations between oxygen saturation and heart rate. The durations of the PQ interval, QT interval, and the QRS complex shorten significantly with ongoing hypoxia, including during a 30-minute stay at the simulated altitude of 5,000 m a.s.l. in the HC (while remaining within the normal range), which may reflect integrated chemoreflex-mediated autonomic–chronotropic adaptation to hypoxemia; in contrast, R- and T-wave amplitudes, QTc duration, and the ST interval course do not change significantly.

A routine altitude-hypoxia tolerance test performed at a simulated altitude of 5,000 m a.s.l. in the HC in this cohort did not reveal any clinically relevant changes in ECG morphology. Accordingly, continuous rhythm and heart rate monitoring may be sufficient in similar low-risk settings. However, extrapolation to other populations, longer exposures, or higher-risk individuals should be approached with caution.

AUTHORS' DECLARATION

Study Design: Ewelina Zawadzka-Bartczak, Lech Kopka. **Statistical analysis:** Ewelina Zawadzka-Bartczak, Lech Kopka, Katarzyna Barwińska. **Data Collection:** Ewelina Zawadzka-Bartczak, Lech Kopka, Katarzyna Barwińska. **Manuscript Preparation:** Ewelina Zawadzka-Bartczak, Lech Kopka, Katarzyna Barwińska. The authors declare that there is no conflict of interest.

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