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Original Article

Investigation of changes in electrocardiography before and after free diving

Serbest dalış öncesi ve sonrası elektrokardiyografik değişikliklerin incelenmesi

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Abstract

Aim: To evaluate the electrocardiographic (ECG) parameters and hemodynamic parameters in predicting the development of arrhythmias after free diving static apnea performance and maximum breath hold.

Material and Methods: Twenty-four volunteer athletes participating in the free diving competition in 2015 (19 males (79.2%) and 5 females (20.8%)) were included in the study. Peripheral O2 saturation (SpO2), heart rate (HR), ECG parameters (PR interval, QRS time, T wave amplitude, corrected QT time, presence of bandle branch block and new bandle branch block development, atrial premature beats, ventricular premature beats) were analyzed.

Results: There was no statistically significant difference between before static apnea measurements (systolic blood pressure (SBP) 124.7 ± 10.8 mmHg, diastolic blood pressure (DBP) 76.5 ± 6.7 mmHg, heart rate (HR) 80.2 ± 13.4 beats / min, SpO2 97.1 ± 0.9%) and after performance (SBP 128.8 ± 13.6 mmHg DBP 78.0 ± 5.9 mmHg, HR 85.8, ± 16.5 beats / min and SpO2 96.7 ± 2.3%)($p = 0.175$; $p = 0.334$; $p = 0.104$; $p = 0.336$, respectively).

Conclusion: No significant changes were observed in ECG parameters, heart rate, saturation and blood pressure values evaluated after static apnea performance. These findings can be used to support that the risk of arrhythmia during static apnea does not persist after apnea has ended.

Keywords: arrythmia; free diving; electrocardiography

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Öz

Amaç: Serbest dalış statik apne performansı ile maksimum nefes tutma sonrası aritmi gelişimini öngörmede elektrokardiyografik (EKG) parametreleri ve hemodinamik göstergeleri değerlendirmek.

Gereç ve Yöntemler: Çalışmaya 2015 yılında düzenlenen serbest dalış yarışmasına katılan 24 gönüllü sporcu dahil edilmiştir (19 erkek (%79,2) and 5 kadın (%20,8)). Performans öncesi ve sonrası (5. dakikada) periferik O2 satürasyonu (pO2), kalp hızı (KH), EKG parametreleri (PR aralığı, QRS süresi, T dalga amplitüdü, düzeltilmiş QT süresi, dal bloğu varlığı ve yeni dal bloğu gelişimi, atriyal erken atım, ventriküler erken atım varlığı) analiz edildi.

Bulgular: Sporcuların statik apne öncesi sistolik kan basınçları (SKB) 124,7±10,8 mmHg, diyastolik kan basınçları (DKB) 76,5±6,7 mmHg, kalp hızı 80,2 ±13,4 atım/dk, pO2 %97,1±0,9 ve performans sonrası SKB 128,8±13,6 mmHg, DKB 78,0 ±5,9 mmHg, KH 85,8±16,5 atım/dk, pO2 %96,7±2,3 arasında istatistiksel olarak anlamlı fark saptanmadı (sırasıyla p=0,175; p=0,334; p=0,104; p=0,336).

Sonuç: Statik apne performansı sonrası değerlendirilen EKG parametrelerinde, kalp hızında, satürasyon ve tansiyon değerlerinde herhangi bir anlamlı değişiklik izlenmedi. Bu bulgular statik apne esnasında gelişebilecek aritmi riskinin apne sonlandıktan sonra devam etmediğini desteklemede kullanılabilir.

Anahtar kelimeler: aritmi; serbest dalış; elektrokardiyografi

Introduction

Free diving is a water sports area that has a long history, a livelihood for people in the past and started with a deep breath. Performance trials are conducted in different branches of free diving sports. Static apnea is one of the free diving sports. It' s a kind of official apnea diving disciplines. In this kind of diving, athletes hold their breath as long as possible. Subjects noses and mounths are submerged and their body floats motionless in a shallow water pool. In speed endurance apnea athlete aims at covering a fixed distance at the least possible time. The usual distance is: for speed 2X50meters (m) in pools of 25m or 50m, and for durability 8X50m and 16X50m. In dynamic apnea the athlete aims at covering the maximum horizontal distance. The athlete keeps his/her body below the surface of the water either with or without fins.

The risk of syncope might be increased by cardiac arrhythmias when the athlete is submerged and this leads to drowning, as a result. The incidence of clinical complications in breath hold competitions in shallow water is well documented: approximately 10% of the static apnea performances resulted in loss of motor control and 1% of loss of consciousness. [1]

This study tested the hypothesis that maximum breath-holding performance after static apnea competition may be associated with cardiac arrhythmias, electrocardiographic changes (bradycardia, prolongation of PR interval, QT prolongation etc.). Thus, we investigated the electrocardiogram (ECG) and peripheral oxygen saturation (SpO2) was assessed using a finger pulse oximeter.

Material and Methods

Twenty-four volunteer athletes who participated in the free diving competition in 2015 (19 (79.2%) males and 5 (20.8%) females) were included in the study (Table 1).

Table 1. Demographic and clinical characteristics of participants

	n=24
Age (years), [mean±SD]	28.1±9.1
Age range (years)	14-44
Gender, [n (%)]	
Female	5 (20.8%)
Male	19 (79.2%)
Duration of water sports (years), [median (min-max)]	5 (0.083-22)
iRBBB, [n (%)]	6 (25.0)
Static apnea (s), [median (min-max)]	168.5 (41-389)

SD: standard deviation, n: participant number, min: minimum, max: maximum, s: second, iRBBB (incomplete right bundle branch block)

The study was conducted in accordance with the principles of the declaration of Helsinki. Written informed consent was obtained from each athletes before enrollment. The data of the athletes were analyzed retrospectively. All athletes performed static apnea performance. Before the competitions, individuals do not eat or drink caffeinated beverages. Additionally, the athletes are told not to do physical activities or apnea related activities for 24 hours before and during the competition day. The investigations were performed at two distinct pools with

water temperatures ranging between 26 and 28°C. In order to perform a maximum static apnea the athletes immersed their face into the water by just flexing the neck and the head is at the surface outside the pool water. Peripheral brachial SBP and DBP measurement, ECG and SpO2 were studied at rest and after performance. A portable ECG and a pulse oximeter was used for measure SpO2 before performance and 5 min after surfacing. Through the whole experiment time was controlled and documented by stop clock manually. Verbal information and tap on the shoulder as a physical signal was given to the athlete during each static apnea performance in every 30 seconds.

The presence of arrhythmia (ventricular - atrial premature beat), PR interval, QRS duration, QT and corrected QT distance, T wave amplitude and variation, bandle branch block presence or development were analyzed on ECG before and after performance.

We didn't include athletes who have heart disease and rhythm disorder in this study.

Statistical Analysis

Whether the distributions of continuous variables were normal or not was determined by Shapiro-Wilk test. Descriptive statistics for continuous variables were expressed mean ± SD or median (min-max), where applicable. Number of cases and percentages were used for categorical data. Whether, the mean differences in clinical measurements (i.e. hemodynamic and ECG components) between before and after the static apnea performance were statistically significant or not was evaluated by Paired t test. Data analysis was performed by using IBM SPSS Statistics version 17.0 software (IBM Corporation, Armonk, NY, USA). A p value <0.05 was considered statistically significant.

Results

Data were collected from 24 athletes aged between 14 and 44 years. The mean age of the athletes was 28.1 ± 9.1 years and 5 of them (20.8%) were female and 19 of them (79.2%) were male. The median water sports duration of the athletes were 5 years and the duration of water sports ranged from 1 month to 22 years. Incomplete right bundle branch block (iRBBB) was observed in 6 (25.0%) athletes on baseline ECGs. Systolic blood pressure 124.7 ± 10.8 mmHg, DBP 76.5 ± 6.7 mmHg, heart rate (HR) 80.2 ± 13.4 beats / min, SpO2 97.1 ± 0.9% was measured before the static apnea of athletes and after the performance SBP 128.8 ± 13.6 mmHg, DBP 78.0 ± 5.9 mmHg, HR 85.8 ±, 16.5 beats/min and SpO2 96.7 ± 2.3%. No statistically significant differences were observed between before and after static apnea performance measurements (p = 0.175; p = 0.334; p = 0.104; p = 0.336, respectively) (Table 2).

Table 2. Hemodynamic measurements of all athletes (n = 24) before and after static apnea performance

	Before static apnea performance	After static apnea performance	p-value †
Systolic blood pressure (mmHg)	124.7±10.8	128.8±13.6	0.175
Diastolic blood pressure (mmHg)	76.5±6.7	78.0±5.9	0.334
Heart rate (beats/minute)	80.2±13.4	85.8±16.5	0.104
Saturation (%)	97.1±0.9	96.7±2.3	0.336

Data; shown as mean ± standard deviation, † Paired t-test.

All subjects completed maximal apnea duration without clinical complications such as lose of motor control or lose of consciousness. The median duration of static apnea was 168.5 seconds and ranged from 41 to 389 seconds. No cardiac arrhythmia was observed before and after static apnea performance.

The mean PR interval (p = 0.744), mean QRS duration (p = 0.197), mean QT distance (p = 0.374), mean heart rate (p = 0.777), and corrected QT distance (p = 0.06) and T wave amplitude (p = 0.782) were evaluated in the ECGs before and after performance. No significant difference was found between measurements (Table 3).

Table 3. ECG measurements of all athletes (n = 24) before performance and after static apnea

	Before static apnea performance	After static apnea performance	p-value †
PR range	150.6±17.9	149.7±21.3	0.744
QRS time (ms)	95.6±8.0	97.6±7.5	0.197
QT distance (ms)	358.5±25.5	362.6±26.1	0.374
Heart rate (beats / min)	82.3±14.0	81.2±14.3	0.777
Corrected QT distance (ms)	411.9±24.3	422.0±27.0	0.060
T amplitude (mV)	3.48±1.53	3.55±1.08	0.782

Data; shown as mean ± standard deviation,† Paired t-test.

Discussion

The development of diving-related arrhythmias has been studied in several studies. The first ECG recording taken during shallow dives in 1963 and the researcher Irving didn't mention presence of any arrhythmias. [2] Abnormal P waves and periods of junctional rhythm were seen in Korean Ama more often especially in winter (incidence was 73% versus 43% during summer) [3]. Also periods of sinus arrest with

junctional escape beats, A-V nodal block, and idioventricular rhythms were noted. [4] These findings resolved immediately upon surfacing. T waves became more peaked, but there was no change in corrected QT interval. In another study, ECG was investigated during the maximal apnea performances with the face submerged but any arrhythmias were not reported. [5] While no arrhythmia was observed in our study, there was no significant change in PR interval, P wave morphology, cQT, T wave, QRS duration and bandle branch block development.

Breath-holding (apnea) triggers a series of known as the diving reflex which collectively lower oxygen utilization and in turn, prolong apneic durations. Breath holding starts many physiological modifications that are known as diving reflex. Many reasons such as an initial parasympathetically induced bradycardial response [6] and decreased cardiac output starts diving reflex. It is followed blood redistribution by a sympathetically induced peripheral vasoconstriction of extremities and non-vital organs [7], As a result of diving reflex the oxygenated blood is preferentially redistributed from non vital organs to the vital organs. [8] Human brain is extremely oxygen-dependent. An interruption in oxygen supply lasting more than a few seconds leads to loss of consciousness, and more than a few minutes leads to irreversible damage. [9] The heart tolerates ischemia better than the brain but there is a risk of conduction block and arrhythmias. Peripheral vasoconstriction activated by diving decreaces blood flow selectively to the more anoxia-tolerant tissues that can maintain substantial anaerobic metabolism.

The diving reflex is triggered by stimulation of the trigeminal nerve on the face, which then causes an increase in vagal tonus, strengthened by cooling of the facial region [10]. Immersion leads to a little to moderate raise in arterial blood pressure. [11] In another study, an important change in blood pressure was not observed despite a decrease in heart rate. [12] In our study, there was no significant change in heart rate, SBP and DBP values after static apnea performance. A considerable amount of human studies were limited to apnea times and it seldom exceeded 2 to 3 minutes. Duration of time was short enough to reach maximum bradycardial response and hypertension induced by sympathetically activated peripheral vasoconstriction. Hansel et al. demonstrated that arrhythmia development was associated with long apnea duration (mean 280 s and over) in static apnea performance. [13] In our study, the duration of static apnea was shorter than the arrhythmia observed studies.

Ferrigno et al. showed that bradycardic response was more pronounced in hypothermic water (25 C) and developed faster than thermoneutral temperature in diving [14]. Diving bradycardia usually occurred in 10 s of submersion, and plateaus was at minimum heart rate approximately 30 to 60s. [11] Heart rates recovered rapidly after cessation of apnea. IN the present study free diving performance was performed in thermoneutral temperature pools and ECG recordings were taken 5 minutes after the end of the competition, we could not be able to determine the development of bradycardic response due to rapid HR recovery after diving. On the other hand several studies shown that arrhythmias are common in deeper breath-hold dives. [12-14]

It was observed that the hydrostatic pressure was also impotant concerning the effects of submersion on diving bradycardia, Immersion is holding the body underwater, and this leads to external hydrostatic pressure. The pooled blood in the lower extremities is directed to the intrathoracic area with the increase of external hydrostatic pressure and decreased gravity, and the central blood volume increases with the contribution of peripheral vasoconstriction. High central blood volume increases cardiac chamber stretch and preload. This increases cardiac force of contraction and stroke volume (SV) by the Frank-Starling law of the heart. More frequently, increase may be explaned by greater stroke volume from increased cardiac contractile forces. During contraction there is no significant change in heart rate. [15]

Immersion, hypoxia, cold stimulus and extreme bradycardia can trigger arrhythmia. Increased vagal tone causes bradycardia, but increased sympathetic tone leads to ectopy. [16] Apart from static apnea, increased metabolic activity and oxygen consumption in muscles related to the use of extremities during dive performances such as dynamic apnea have been found to cause more hypoxemic stress in the body. The degree of hypoxemia is also associated with the development of arrhythmia. Arrhythmias were more frequent underwater than on the surface. [11] Diving in shallow waters, such as the pool where our study is performed, the development of arrhythmia could be less likely than in deep water and no decrease in SpO2 levels after performance may be one of the reasons failure to detect arrhythmias.

Conclusion

As a result, no significant changes were observed in ECG parameters, heart rate, saturation and blood pressure values after static apnea performance. In our study, normal findings after apnea can be used to show that the risk of arrhythmia secondary to hypoxemia associated with increased vagal tonus and bradycardia, which has been shown in various studies during apnea, does not persist after the end of diving.

Further studies are needed to support the current findings.

Declaration of conflict of interest

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