Assessment of sensory sensitivity through critical flicker fusion frequency thresholds after a maximum voluntary apnoea

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Key words
Brain; Breath-hold diving; Exercise; Fatigue; Hypoxia

Abstract
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Introduction: The influence of acute exercise on sensory sensitivity (SS) differs according to the type and duration of exercise performed. In the present study, we assessed changes on SS soon after a maximal dynamic apnoea.
Methods: Thirty-nine experienced male breath-hold divers were recruited. Critical flicker fusion frequency (CFFF) thresholds were used to measure SS. Thresholds were determined before and after a maximal dynamic apnoea. Immediately after surfacing, heart rate and oxygen saturation (SpO_2) were recorded for two minutes.
Results: After maximal dynamic apnoea, SpO_2 was significantly decreased (from mean 97.3% pre-dive to mean 63.1% post-dive; $P < 0.0001$; $\eta 2$ $P = 0.86$), but this acute hypoxaemia did not trigger changes in SS (post-dive value 102% of baseline; $P = 0.22$; $\eta 2$ $P = 0.03$). Pearson correlation analysis revealed a moderate association between SS with swimming speed ($r = 0.423$) and apnoea time ($r = -0.404$).
Conclusions: A maximal dynamic apnoea did not produce changes in central nervous system fatigue or cortical arousal. We found no relationship between the hypoxaemia level reached after a maximal apnoea and changes in the CFFF thresholds. This study suggests that the time of exposure to hypoxia during a maximal voluntary apnoea is not enough to produce changes in SS.

Introduction

Critical flicker fusion frequency (CFFF) is considered a quantitative and validated measure of sensory sensitivity (SS), and the CFFF threshold is of interest in sport, as it can be used to determine the degree of CNS fatigue and cortical arousal.[1–4] Tomporowski et al. noted that aerobic and anaerobic exercise at moderate intensities seemed to produce improvements in cognitive performance, while an intense, brief and exhaustive bout of anaerobic exercise did not affect cognitive function.[5] However, sub-maximal exercise taken over a long period, leading to dehydration or/and depletion of energy substrates, seems to decrease both information processing and memory functions.[5] Thus, if we consider breath-hold diving as an intense, brief and exhaustive anaerobic form of exercise, we could hypothesize that apnoea training would not produce changes in the SS, although it must be considered that this type of exercise is considerably different from those examined in the studies above.

Decreased muscular strength, coordination impairment, physical and mental fatigue and somnolence are several functional disorders that can occur due to hypoxia; they develop in proportion to the demand for oxygen and intensify with the duration of hypoxia.[6] Previous studies have analyzed the influence of a stay at altitude ranging from 4000 m to 5500 m, suggesting SS changes that may be attributed to repeated forays to high altitudes.[7–9] CFFF measurements have been made during routine testing of hypoxia tolerance in pilots and skydivers, and these findings suggest that the intermittent hypoxia experienced does not cause changes in SS.[10,11] CFFF has also been used in diving studies. It is thought that post-dive fatigue and an associated reduction in alertness could be caused by one or all of three factors: the effects of nitrogen, oxygen or circulating bubbles on the body after a dive.[12–14]

The exposure to short-term intermittent hypoxia, frequently experienced by trained breath-hold divers, leads to sustained sympathetic activation. However, it is not known if apnoea training can produce changes in SS, either via central nervous system (CNS) fatigue or by increased cortical arousal.[15] The present study aimed to assess changes in SS (mainly cortical arousal or CNS fatigue) after a maximal dynamic apnoea, made by swimming as far underwater as

Figure 1
Breath-hold diver performing CFFF test: undertaken before and after maximal dynamic apnoea

possible during a breath-hold in a swimming pool with fins. The null hypothesis was that soon after a maximal dynamic apnoea there would be no changes in SS.

Methods

The protocol was conducted in accordance with the Declaration of Helsinki and was approved by the institutional Human Research Ethics Committee (UAM-CEI-70-1257).[16]

PARTICIPANTS

Thirty-nine male breath-hold divers were recruited in this multicenter study from different Spanish locations. Their mean (SD) age was 38 (7) years, experience in competitive apnoea was 5 (3) years and their personal best in dynamic apnoea with bi-fins was 102 (19) horizontal metres. Divers were informed of the procedures involved in the trial, including the benefits and risks, prior to signing the informed consent document. Participants with a history of epilepsy were excluded from the study.

CRITICAL FICKER FUSION FREQUENCY THRESHOLDS

We assessed CFFF thresholds using a Lafayette Instrument Flicker Fusion Control Unit (Lafayette Instrument, Lafayette IN, USA). This device consists of two white light emitting diodes (58 cd·m^2) that are simultaneously displayed in the system, one for the left eye and one for the right. The diodes are separated by 2.75 cm, with a diode-to-eye distance of 15 cm and a viewing angle of 1.9 degrees. The interior of the equipment is painted matte black to minimize interference. The flicker frequency increment (1 Hz·sec^{-1}) changed in two ways: either it increased (from 0 to 100 Hz) until the subject perceived fusion or decreased (from 100 to 0 Hz) until flicker was detected. After a fovea binocular fixation, participants were required to respond by pressing a button upon identification of the visual flicker (descending frequency) and the fusion (ascending frequency) thresholds.

Before the experiment, subjects performed as many practice trials as necessary to familiarize themselves with the CFFF test. During the test, three ascending and three descending trials were performed alternately. The mean of the six values, representing the classical CFFF thresholds, was calculated for each subject.

OXYGEN SATURATION, SWIMMING SPEED AND APNOEA TIME

We used a finger probe pulse-oximeter (NoninPalmSAT® 2500, Nonin Medical, Plymouth MN, USA) to detect changes in oxygen saturation (SpO_2) and heart rate (HR). During the measurement, a green signal indicated good perfusion, while a red light denoted bad perfusion. Only data collected during good perfusion readings were analyzed. Lost motor control in the diver was recorded as a manifestation of symptomatic hypoxia.[17] To assess average swimming speed, we recorded the time and distance reached during dynamic apnoea.

PROCEDURES

The examinations were conducted in a quiet, temperature-regulated and humidity-controlled room, close to the swimming pool (24 ± 1ºC, relative humidity 80–90%). The tests were conducted by a single evaluator who had not participated in the selection process.

Baseline characteristics, SpO_2 and CFFF, were obtained and participants were seated in front of the CFFF viewing chamber to determine CFFF thresholds, using the protocol described previously (Figure 1). The divers were then asked to perform a maximal dynamic apnoea with bi-fins in a 25 m pool. A safety diver accompanied the divers during the attempt, as during competitions. HR and SpO_2 were recorded immediately on surfacing at five second intervals for two minutes. Average swimming speed was calculated as described. Finally, three minutes after the maximal dynamic apnoea, the CFFF thresholds were measured as per the pre-dive protocol (Figure 1).

STATISTICS

G*Power 3.1.7[18] statistical software (Heinrich-Heine-Universität, Düsseldorf, Germany) was used to calculate the sample size needed to complete this study. Based on alpha = 0.05 and an assumed effect size of 0.42 (based on pilot study data; n = 12), for 80% power we calculated that a total sample size of 37 participants would be necessary.

All experimental data analysis was performed using IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA). The statistical analyses were conducted at a 95% confidence level; a P-value of less than 0.05 was

Figure 2
Correlation plot demonstrating the relationship between CFFF (Hz), pre- and post-apnoea measurements and the average swimming speed reached (r = 0.423, $P < 0.05$)

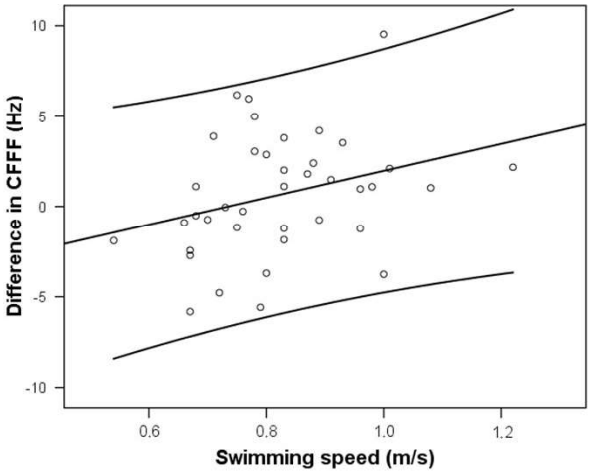
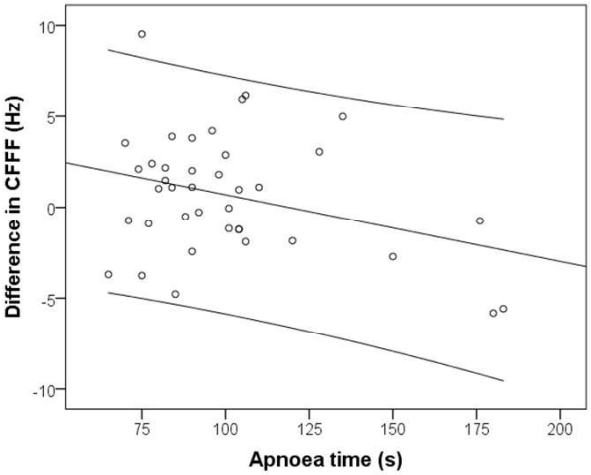


Figure 3
Correlation plot demonstrating the relationship between CFFF (Hz), pre- and post-apnoea measurements and total apnea time (r = -0.404, $P < 0.05$)



considered statistically significant. We expressed our results as means and standard deviations (SD) with 95% confidence intervals. We confirmed the normality of the data using the Shapiro-Wilk test.

Student's *t*-test (one sample, two-tailed) was used to analyze changes in CFFF thresholds and SpO_2 between baseline and post-apnoea; we also assumed partial η^2 as a measure of the effect sizes of exercise on the CFFF. Effect sizes (Cohen's d) were calculated for the outcome variables. According to Cohen's method, the magnitude of the effect is classified as small (0.20–0.49), medium (0.50–0.79), or large (≥ 0.8).[19] Taking the baseline value as 100%, percentage changes were calculated, allowing an appreciation of the magnitude of CFFF change between each measurement rather than the absolute values.

The relationships between sensory sensitivity, swimming speed and apnoea time were examined using Pearson correlation coefficients. A Pearson correlation coefficient > 0.60, between 0.30 and 0.60, and < 0.30 indicated high, medium, and low correlations, respectively.[20]

Results

SENSORY SENSITIVITY AND OXYGEN SATURATION

CFFF and SpO_2 reached after the maximal dynamic apnoea are illustrated in Table 1a. Compared to the baseline, there was a significant decrease of SpO_2 during apnoea, from 97.1 (0.7) to 63.1% (13.7) ($P < 0.0001$; η^2 $P = 0.86$). However, CFFF measurements made three minutes after the maximal dynamic apnoea were not statistically different from the baseline values (102.0%; $P = 0.22$; η^2 $P = 0.03$).

Upon subgrouping subjects (Table 1b), adoption of the lost motor control criteria used to determine hypoxia effects revealed that SaO_2 decreased from 97.3 (0.7) to 66.1% (13.1) in divers with no hypoxic effect ($n = 31$) and from 96.8 (0.9) to 49.2% (7.5) in divers with lost motor control ($n = 7$). In both groups, CFFF results were not statistically different from the baseline value: 35.3 (4.4) to 35.9 Hz (4.4) in divers with no hypoxic effect (101.7%; $P = 0.22$; η^2 $P = 0.04$) and 30.5 (7.7) to 30.9 Hz (4.3) in divers with lost motor control (101.3 %, $P = 0.77$; η^2 $P = 0.01$).

CORRELATION ANALYSES

Pearson correlation showed only a small relationship between the SpO_2values reached during the maximal dynamic apnoea and changes in SS (r = 0.268, $P < 0.05$). There was a moderate association between SS with swimming speed (r = 0.423, $P < 0.05$; Figure 2) and apnoea time (r = -0.404, $P < 0.05$; Figure 3).

Discussion

This study analyzed the effect of maximal dynamic apnoea on SS using CFFF. If maximal apnoea leading to exhaustion does induce a transitory CNS fatigue, a decrease in SS should be observed.[21] Alternatively, if apnoea induces an increase in cortical arousal, an increase in SS should be observed. Our findings showed that no changes were observed in CNS fatigue or cortical arousal after a maximal dynamic apnoea. Thus, the induced fatigue during a maximal apnoea, if any, is most likely to be linked to peripheral fatigue (in the muscle) or perceived fatigue (exertion tolerance) and not due to CNS fatigue. In addition, there is no evidence that repeated hypoxic syncope, caused by apnoea training, has long-term effects on cognitive functions or neurological effects.[22]

Table 1

(a) Percentage variation of CFFF and oxygen saturation (SpO_2) between pre- and post-apnoea measurements. (b) Participants are subgrouped into no hypoxic effects (n = 32) and with loss motor control (LMC) (n = 7). *** indicates $P < 0.0001$

Table 1 (a)		Pre-apnoea	Post-apnoea		Sig	η^2P
		Mean (SD)	Mean (SD)	% from baseline		
Participants (n = 39)	CFFF threshold (Hz)	34.4 (5.3)	35.1 (5.2)	102.0	0.221	0.039
	Oxygen saturation (%)	97.1 (0.7)	63.1 (13.7)		***	0.862
Table 1 (b)		**Pre-apnoea**	**Post-apnoea**		**Sig**	**η^2P**
		Mean (SD)	**Mean (SD)**	**% from baseline**		
No hypoxic effects (n = 32)	CFFF threshold (Hz)	35.3 (4.4)	35.9 (4.4)	101.7	0.223	0.048
	Oxygen saturation (%)	97.3 (0.7)	66.1 (13.1)		***	0.994
Loss motor control (n = 7)	CFFF threshold (Hz)	30.5 (7.7)	30.9 (4.3)	101.3	0.770	0.015
	Oxygen saturation (%)	96.8 (0.9)	49.2 (7.5)		***	0.998

During breath-hold diving, progressive hypoxaemia stimulates the respiratory centers in the brain and in our study, severe hypoxaemia was noted in all divers after the maximal apnoea swim.[23] One of our main findings is that there was no relationship between the levels of hypoxaemia reached after a maximal apnoea (even in divers who lost motor control) with changes in the CFFF thresholds measured three minutes later. However, it should be noted that although severe, the hypoxaemia experienced during an apnoea swim is of very short duration in comparison to the exposure that mountaineers or pilots undergo.[9,10] If we consider apnoea as an intense, brief and exhaustive anaerobic exercise, the findings of the current study support the hypothesis that the influence of acute exercise on cognitive function differs according to the type and duration of exercise performed.[5]

We also noted a moderate, inverse relationship between apnoea time and SS. This finding could support the hypothesis that it is the time and not the intensity of exposure to hypoxia that is the factor crucial to the production of SS changes. A similar conclusion was reached by Cavalade, who used CFFF to measure the effects of hypoxia on skydivers, and suggested that repeated jumps above 4,000 m were not long enough and therefore of great enough hypoxic stimulus to alter SS.[11] It is possible that the time of exposure to hypoxia is critical to produce CNS changes, but neither our data nor that in the literature have definitively established this.

CFFF thresholds are affected by both non-sensory and sensory factors.[1] During maximal apnoea, divers can be exposed to factors other than hypoxia, including hypercapnia, muscular acidosis, mental effort, stress or fear. Due to the correlations determined in our study, we could suggest that the divers who reached a higher underwater swimming speed also produced a higher increase in SS. As such, it could be speculated that faster divers had a greater stimulation of the sympathetic nervous system due to a requested higher inter- and intramuscular coordination and processing speed. This would be in accordance with a previous study, which found that short submaximal and repeated apnoeas in trained divers are powerful enough to intensify sympathetic nervous system activity.[24]

CFFF could be a practical substitute for various psychological measures in assessing SS in a diving/hyperbaric environment.[25] As mentioned previously, changes in nitrogen or oxygen concentration could be considered to explain the variations in post-dive SS. Lafère stated that the changes in perceived fatigue level after a single dive were significantly lower when divers breathed enriched air nitrox compared to air dives.[14] Balestra found that inert gas narcosis produced a cerebral impairment that then persisted for at least 30 minutes after surfacing from a 20 min dive to 33 m.[12] However, increasing the oxygen fraction improved cognitive competence during a dive to 24 m due to the reduced narcotic effects of the nitrogen fraction.[13]

Scuba diving involves complex phenomena in the functional modifications of the nervous system according to the type of gas used for the dive; although freedivers who perform deep apnoeas may be affected by episodes of narcosis, the participants involved in our study (in a swimming pool) were not.[14,26] On this basis, we must suggest that the results from

the present study should not be applied to breath hold divers who perform deep dives.

Changes in blood pressure or blood flow in the freedivers during the maximum dynamic apnoea were not evaluated. An increase in blood pressure is associated with disruptions in neurovascular coupling, which leads to a decrease in vascular reserve capacity and can cause cognitive changes.[27] Factors other than perfusion or autonomic failure have also been proposed to contribute to cognitive decline.[28] In view of the above, in future studies, analysis of blood pressure and brain blood flow should be included in order to clarify the changes produced in the SS after a maximal apnea.

Clearly there are limitations in the techniques we used when trying to measure hypoxaemia. It has been suggested that a finger probe is inferior to an ear probe for measuring SpO_2 during apnoea, but the use of the former is recommended in the patient with poor peripheral perfusion, as, for example, in a diver affected by limited peripheral blood flow triggered by the diving response.[29,30] Another methodological limitation was that the average speed was calculated by total distance and apnoea time during the maximal dynamic apnoea. No consideration could made for a change in speed, for example during a brief sprint.

In conclusion, maximal dynamic apnoea did not produce changes in CNS fatigue or cortical arousal. In addition, no relationship between hypoxaemia levels reached after maximal apnoea and changes in the CFFF thresholds was found. This study suggests that the time of exposure to hypoxia, during a maximal voluntary apnoea, is not enough to produce sensory sensitivity changes.

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