

Maintained critical flicker fusion frequency after acute hyperbaric hyperoxia at 140 kPa

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Abstract

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Introduction: In diving medicine, critical flicker fusion frequency (cFFF) has been employed to assess alertness under varying ambient pressures and gas compositions, yet the effects of elevated oxygen partial pressure on cFFF performance underwater remain inconsistently characterised. This investigation aimed to assess the impact of breathing 140 kPa oxygen on underwater cFFF performance, a pressure level corresponding to established safety limits and common closed-circuit rebreather setpoints during operational diving.

Methods: Nineteen experienced divers (10 females; mean age 23.8 [SD 3.0] years) completed cFFF assessments under three conditions: breathing air at poolside (21 kPa O₂), breathing air at 4 m depth (29 kPa O₂), and breathing 100% oxygen at 4 m depth (140 kPa O₂). Concurrent measurements included heart rate, respiratory rate, and heart rate variability indices representing autonomic function; specifically the LF/HF ratio indicating sympathetic activity and RMSSD reflecting parasympathetic activity.

Results: Mean cFFF breathing air at poolside was 37.9 (SD 2.9) Hz and breathing 100% oxygen at 4 m was 37.2 (SD 2.5) Hz, indicating that mild hyperoxia at 140 kPa does not impair cortical processing speed or visual temporal resolution. However, oxygen breathing produced notable cardiovascular and respiratory effects: heart rate decreased from 70.9 (10) to 67.8 (11.6) beats per minute, respiratory rate declined from 15.2 (2.7) to 13.4 (1.7) breaths per minute, the LF/HF ratio decreased from 5.9 (1.4) to 3.1 (2.0), and RMSSD increased from 73.7 (10.8) to 97.0 (6.6) milliseconds. These physiological changes indicate reduced sympathetic activity and enhanced parasympathetic tone during oxygen breathing.

Conclusions: The findings suggest that breathing 140 kPa oxygen during shallow water immersion maintains neural integrity as measured by cFFF while beneficially modulating autonomic regulation toward parasympathetic dominance. This autonomic shift resembles known effects of both submersion and moderate hyperoxia, supporting the safety profile of current oxygen exposure protocols in recreational and technical diving.

Introduction

Critical flicker fusion frequency (cFFF) is the frequency at which an intermittently illuminated stimulus is perceived as steady and has been discussed as an indicator of cortical arousal and visual-cognitive processing,^{1,2} with a higher cFFF associated with better performance. Conceptually, flicker fusion occurs when temporal modulation exceeds the visual system's temporal resolution, yielding a perception

of continuous light above this threshold.^{2,3} Previous studies reported significant variation in individual cFFF thresholds for healthy adults, i.e., 35–90 Hz, reflecting methodological and stimulus-dependent differences,^{2,4} respectively.

In diving and hyperbaric research, cFFF has been applied to assess the effects of elevated nitrogen and oxygen gas pressures on arousal and cognition,^{5–8} though its suitability without additional markers remains debated.⁹ Furthermore,

findings on the effects of increased inspiratory oxygen pressures, from increased ambient pressure or oxygen fraction in the breathing gas, remain equivocal. In chamber studies, oxygen produced dose-dependent changes in cFFF with no effects at 70 kPaO₂, lower cFFF at 140 kPa (i.e., indicating a negative effect), and a recovery to baseline at 280 kPa. This response is similar to the dose-dependent physiological effects of O₂, from the benefits of increased oxygenation to oxygen toxicity, consistent with overlapping mechanisms of cFFF modulation and O₂ toxicity.¹⁰ When high O₂ and N₂ pressures are combined, like when breathing air at 600 kPa ambient pressure (pO₂ ≈ 120 kPa), reaction times were worse, but cFFF values increased by 2.5 (SD 2.8)%, indicating solely impaired performance.¹¹ In contrast, Lafère et al. reported a transient positive effect with increased cFFF values after breathing 160 kPa O₂ at 400 kPa ambient pressure.¹² Values returned to baseline within 20 min, suggesting an early O₂-linked enhancement of cFFF that dissipates over time. Other work found that 100% O₂ at 100 and 280 kPa decreased cFFF, i.e., had a negative effect on it, with no change at 600 kPa, leading to questions about cFFF's sensitivity to gas narcosis.⁹

The equivocal results from chamber experiments are echoed in field studies. After no-decompression, depth-matched dives to 21 m with 32% O₂ in the breathing gas (i.e., Nx32), divers reported less fatigue than after air dives. Furthermore, cFFF increased by 4% with Nx32 and decreased by 6% with air compared to pre-dive, indicating gas-dependent neural modulation.⁵ Breathing gases with increased pO₂ have also been associated with benefits for sustaining alertness and short-term memory when compared to breathing air, but cFFF was not assessed in these studies.¹³ During a 20-min air dive to 33 m (pO₂ ≈ 90 kPa), cFFF acutely increased from the 100% control to 104 (5)% at depth, then decreased to 94 (4)% over ten minutes at depth, with deficits persisting 30 min post-surfacing.¹⁴ Data from closed-circuit rebreather (CCR) dives remain limited, with one study reporting slight increases in alertness/arousal during 50 m dives on air, trimix, or heliox, implicating the roles of nitrogen and oxygen – alone or combined – in modulating neuronal excitability.⁶

Given divergent reports on oxygen's neurophysiological effects under pressure, and the potential for dose-dependent excitatory versus depressive responses, we aimed to isolate the specific impact of oxygen on CFFF by using 100% oxygen during immersion to 140 kPa, replicating an established O₂-exposure limit and rebreather setpoint, while minimising the influence of N₂.

Methods

This study was approved by the Ethics Committee of the German Sport University Cologne (100/2023), following the Declaration of Helsinki and its amendments.¹⁵

PARTICIPANTS

The study included 19 healthy participants (mean age of 23.8 (SD 3.2) years; 10 females), an average height of 175 (22) cm, and an average body mass of 72 (14) kg. Calculated body mass index was 23.4 (1.4) kg·m⁻². All participants were certified divers, medically cleared to dive, and had normal vision. Participants were asked to refrain from alcohol and caffeine intake on the day of testing; however, compliance was self-reported and was not formally controlled.

EQUIPMENT AND MEASUREMENTS

Data collection was conducted in a well-lit indoor pool (20 m × 20 m × 5 m) with water and air temperatures at ~27 to 30°C. The diving gear was provided and consisted of a 3 mm wetsuit, a 10L steel cylinder, a commercially available buoyancy control device (BCD), a breathing regulator, mask, and fins. Oxygen was supplied with a separate system at depth. Some participants used personal masks, and clear vision was ensured for all trials. The participants were equipped with a Polar V800 watch and H7 chest belt (Polar Electro Oy, Kempele, FI).

A manually operated flicker device was employed in this study due to its feasibility for underwater testing (Scaleo flicker, RP-Engineering GmbH, D-Esslingen). The device allowed manual adjustment of flicker frequency. The light source was an LED with a color temperature of 8,000 K (i.e., blueish light). The individual cFFF value was displayed to one decimal place on a readout facing the test conductor only. The frequency display was inactive during testing and activated exclusively by a button press directed toward the conductor, ensuring that participants were never exposed to their frequency scores at any point during the experiment.

PROTOCOL

All participants received a comprehensive explanation of the experimental protocol prior to the binocular testing. Here, they were presented with demonstrations of both flickering and steady light conditions. The protocol consisted of 10 min at the pool bottom, breathing either air or 100% O₂ via an open-circuit underwater breathing apparatus.

All measurements were conducted with participants kneeling on the pool bottom. The stimulus frequency was increased from 20 Hz to the individual cFFF threshold, signaled by the participant. All measures were repeated three times by a single test conductor. In cases where participants were uncertain about their decision, or when a clear outlier was observed, all three measurements were repeated. As the distance to the light source was reported to affect cFFF,¹⁶ all experiments maintained a fixed distance of 1.5 m between the CFFF device and the participants' eyes.

DATA PREPARATION

Cardiac parameters were analysed using Kubios 4.03 (Kubios Oy, HRV Scientific 4.1.0, Kuopio, FI). From the HR signal, the respiratory rate (RespR) and two established measures of heart rate variability (HRV) were derived: low frequency:high frequency ratio (LF/HF),¹⁷ representing sympathetic activity, and root mean square of successive differences (RMSSD), representing parasympathetic activity.

The cFFF values from the baseline session (surface, non-immersed, 21 kPa O₂), at 4 m water depth (29 kPa O₂), and breathing 100% O₂ at 4 m (140 kPa O₂) are presented as means (standard deviation) from three consecutive measurements conducted at each time point.

STATISTICS

Differences between the three experimental conditions were analysed using Wilcoxon signed-rank tests for paired samples (SPSS 29.0.1). Pairwise comparisons of variables were conducted between the 21 kPa O₂ and 29 kPa O₂ conditions, and between the 29 kPa O₂ and 140 kPa O₂ conditions. All tests were two-tailed, with the significance level set at $P < 0.05$. In accordance with the American Statistical Association's guidance on careful interpretation of P -values and statistical significance,¹⁸ effect sizes are presented using Cohen's d ¹⁹. Effect size estimates were reported to aid interpretation of non-significant findings, particularly where physiological relevance may exist despite limited statistical power, and to aid comparisons to the literature.

Results

BASELINE MEASUREMENTS (21 kPa O₂, dry)

During this step (no masks worn), critical flicker fusion frequency (cFFF) was 37.1 (2.2) Hz and was thus within the range described for a similar age group⁴. The participants had a mean heart rate (HR) of 76 (11), a respiratory rate (RespR) of 14 (2), LF/HF equalled 11.0 (7.1), and RMSSD was 53.5 (39.7) ms.

MAIN SUBMERSION SESSION (29 vs. 140 kPa O₂)

No significant differences for cFFF thresholds were detected after 10 min breathing either air or oxygen at 4 m water depth (Δ -0.7 Hz). However, heart rate was lower after breathing oxygen compared to air (Δ -3 bpm). For respiration rate, no statistically significant difference was detected based on P -value analysis; however, effect size estimates suggested a substantial reduction during oxygen breathing (Δ -2 breaths·min⁻¹), indicating a potentially meaningful physiological change that may not have reached statistical significance. The LF/HF ratio was markedly decreased under oxygen, reflecting a decreased sympathetic dominance.

RMSSD did not differ significantly according to P -value criteria, but effect size estimates indicated a moderate increase by O₂-breathing (Δ -23 ms), suggesting enhanced parasympathetic activity (see Table 1 and Figure 1).

Discussion

The critical flicker fusion frequency (cFFF), originally described by Braunstein (1903),²⁰ defines the temporal resolution of the visual system, with higher cFFF thresholds reflecting greater cortical processing speed and alertness. Under conditions of elevated breathing gas pressures, such as during diving or hyperbaric exposure, cFFF has been widely applied as a functional index of cortical activity and vigilance.²¹ However, results regarding the influence of hyperbaric oxygen on cFFF remain inconsistent.^{9,10,12,22,23}

Similarly, studies investigating oxygen's neural effects have produced divergent findings. Hyperbaric oxygen can act either depressively or excitatorily, producing narcotic^{24,25} or stimulating²⁶ effects. Reports vary from cognitive impairment^{27,28} to increased neuronal excitability.^{8,29,30} Some authors have suggested that these opposite outcomes may represent different phases of oxygen toxicity, from an initial hyperexcitability to depression,¹⁰ while others observed no measurable changes.^{31,32}

In our previous normobaric study using the same cFFF measurement system, 5 min of oxygen breathing at the poolside (i.e., 100 kPa pO₂) had no impact on cFFF compared with air.²¹ The present investigation extends this finding to underwater mild hyperbaric conditions and the frequently cited limit and rebreather setpoint for oxygen of 140 kPa. However, no effects for breathing 140 kPa O₂ on cFFF were observed relative to air breathing at identical pressure and time.

The absence of a cFFF change at 140 kPa O₂ agrees with Vrijdag et al. (2020), who reported similarly unchanged cFFF in a hyperbaric chamber breathing oxygen at 142 kPa.⁹ Yet, other research demonstrated dose-dependent alterations in neuronal excitability, where cFFF was reduced at 140 kPa.¹⁰ The reasons for these discrepancies remain unclear. Psychological stress and sleep deprivation, which are known to decrease cFFF,^{2,33,34} are unlikely, as the study participants were well-rested and tested under conditions with minimal stress. Likewise, temperature differences between chamber and aquatic environments (~27 to 30°C) are unlikely confounders, as an increase in ambient temperature has been shown to increase, not lower, cFFF.^{35,36} Responses to elevated pO₂ and their potential effects on cFFF appear to be highly individual, as reflected by the pronounced inter-individual variability in cFFF observed in the present dataset (33.2–43.4 Hz). Comparisons across studies are further complicated by differences in exposure duration, which ranges from as little as five minutes to 30 minutes in the existing literature, and by the heterogeneity of study populations, spanning recreational divers, technical

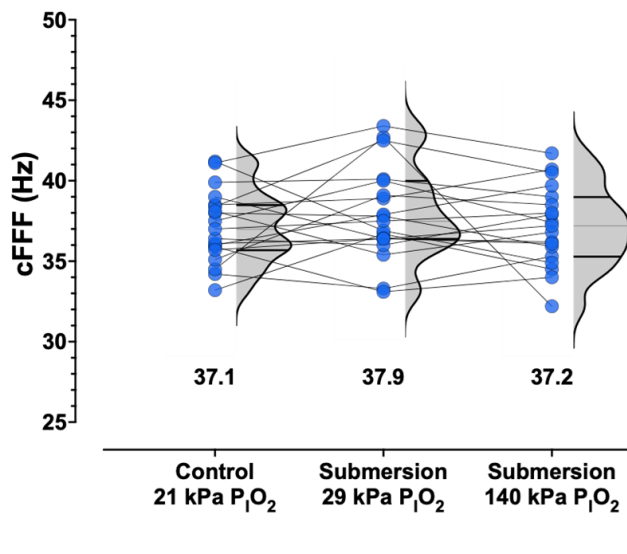
Table 1

Critical flicker fusion frequencies (cFFF), heart rate (HR), respiratory rate (RespR), root mean square of successive differences (RMSSD), and low frequency:high frequency ratio (LF/HF) during breathing air at the pool side (21 kPa O₂), at 4 m (29 kPa O₂) or breathing 100% O₂ (140 kPa O₂); data are presented as mean (SD). Cohen's effects are: small (0.2), medium (0.5), and large (0.8 or greater)

Parameter	cFFF (Hz)	HR (min ⁻¹)	RespR (min ⁻¹)	LF/HF	RMSSD (ms)
21 kPa O ₂	37.1 (2.2)	75 (11)	15 (2)	11.0 (7.1)	53.3 (39.7)
29 kPa O ₂	37.9 (2.9)	71 (10)	15 (3)	5.9 (1.4)	73.7 (10.8)
140 kPa O ₂	37.2 (2.5)	68 (12)	13 (2)	3.1 (2.0)	97.0 (6.6)
<i>P</i>	0.59	0.03	0.13	0.03	0.95
Cohen's d	0.13	0.77	0.89	1.40	0.28

Figure 1

Critical flicker fusion frequencies (cFFF) during air breathing at the pool side (21 kPa O₂) and at 4 m water depth breathing air (29 kPa O₂) or 100% O₂ (140 kPa O₂); data are depicted as individual mean values with connecting lines and violin plots with median and interquartile ranges to show data distribution. Group means are indicated in bold within the figure



divers, and military or hyperbaric personnel with regular oxygen exposure. Both factors likely contribute to the variability in reported cFFF responses and should be considered when interpreting divergent findings.

Apart from cFFF, oxygen breathing at 4 m depth produced notable physiological effects, with a decreased heart rate and respiratory rate compared with air. This likely reflects enhanced autonomic balance, as parasympathetic activity (RMSSD) increased and sympathetic modulation (LF/HF ratio) decreased, consistent with the diving response. These findings align with data from professional divers breathing normobaric oxygen, where similar shifts in autonomic tone were observed.³⁷ It is therefore worth emphasising that both submersion^{38,39} and moderate hyperoxia at 140 kPa pO₂ appear to promote parasympathetic dominance.

Our chosen protocol mirrors setpoints commonly used in closed-circuit rebreather diving during the bottom phase^{40,41} and in decompression procedures, where 100% oxygen at ~140 kPa improves nitrogen offgassing driving pressure.⁴² This pO₂ also corresponds to the widely accepted safety threshold for operational diving.^{43,44}

Taken together, our findings show no change in cFFF from mild hyperbaric hyperoxia at 140 kPa over 10 min duration, while parasympathetic activity is increased. Future research should aim to disentangle potential interactions between oxygen and nitrogen under hyperbaric conditions and clarify the thresholds at which oxygen-induced effects transition from neurofunctional neutrality to excitatory or narcotic effects.

LIMITATIONS

The sample comprised young, well-rested certified divers, limiting generalisability to broader diving populations, including older, less experienced, or fatigued individuals.

Only a single oxygen partial pressure (140 kPa) and exposure duration (10 min) were tested, precluding assessment of dose-response relationships or effects during longer exposures. However, these parameters closely mimic the real-world diving conditions of decompression stops at the tested ambient and inspiratory pressure. The controlled pool environment with stationary participants does differ from actual diving scenarios involving navigation, task performance, and variable conditions, though this approach enabled precise physiological measurements under standardised conditions.

Finally, we cannot fully explain the discrepancy between our maintained cFFF thresholds at 4 m depth breathing air or O₂ and the decreased cFFF thresholds reported by others at the same ambient pressure.¹⁰ Environmental differences between the aquatic and dry hyperbaric chamber settings – particularly concerning posture and temperature – may have contributed to these divergent findings.

Conclusions

Breathing 140 kPa O₂ at 4 m depth maintains cFFF stability, suggesting no cortical depression, while a lower heart rate suggests parasympathetic upregulation. A trend toward reduced respiratory rate was also observed, though this did not reach statistical significance and should be interpreted with caution: reduced ventilation under hyperoxia may promote hypercapnia, which is a recognised potentiator of CNS oxygen toxicity. This validates standard setpoints for bottom time and decompression without neurofunctional risk, but calls for more pooled data on O₂ / N₂ interactions at depth to refine protocols.

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