physiologia

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Cerebrovascular Reactivity to Hypocapnia Following Maximal Sprint Exercise Is Better Maintained in Females than Males

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Abstract

Background/Objectives: Despite increasing interest in high-intensity exercise and cerebrovascular function, the effects of maximal sprint exercise on cerebrovascular reactivity (CVR), a key indicator of vascular health, remain unclear. Methods: This study investigated the acute effects of a 30-s all-out cycling sprint (Wingate Anaerobic Test, WAnT), on CVR to hypocapnia in 24 healthy young adults (12 males). Following familiarisation and a $\dot{V}O_{2max}$ test, participants completed an experimental session where CVR was assessed at rest and 30 min post-WAnT. CVR was evaluated using a 1-min voluntary hyperventilation protocol (25 breaths·min^{-1}), with middle cerebral artery blood velocity (MCAv) measured via transcranial Doppler ultrasound and end-tidal CO_2 ($P_{ET}CO_2$) recorded breath-by-breath. CVR was calculated as the absolute change in MCAv per 1 mmHg change in $P_{ET}CO_2$ from the final 10 s of hyperventilation. Results: Resting MCAv and $P_{ET}CO_2$ were significantly reduced post-WAnT ($p < 0.01$ and $p < 0.001$ respectively). Consequently, the reductions in MCAv and $P_{ET}CO_2$ during hyperventilation were attenuated after exercise in both males and females ($p < 0.01$ and $p < 0.001$ respectively). Despite these changes, CVR remained unaltered in both sexes following WAnT (males: 1.79 ± 0.35 vs. 1.59 ± 0.26 cm·s^{-1}·mmHg^{-1}, $p = 0.09$; females: 2.01 ± 0.44 vs. 2.01 ± 0.46 cm·s^{-1}·mmHg^{-1}, $p = 0.97$). However, post-exercise CVR was significantly lower in males than females, despite no baseline sex differences ($p = 0.01$). Conclusions: Cerebrovascular reactivity to hypocapnia is preserved 30 min after a single bout of maximal sprint exercise in healthy young adults. Notably, females demonstrated a more favorable maintenance of CVR post-exercise compared to males, suggesting potential sex differences in CVR following maximal sprint exercise.

Keywords: cerebral blood flow; cerebrovascular function; middle cerebral artery velocity; supramaximal exercise; Wingate test

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Academic Editors: Michael Koutsilieris and Anastassios Philippou

Received: 29 November 2025
Revised: 3 February 2026
Accepted: 11 February 2026
Published: 14 February 2026

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1. Introduction

Cerebrovascular reactivity (CVR) reflects the ability of cerebral blood vessels to alter their diameter in response to changes in arterial carbon dioxide (P_aCO_2) [1]. This regulation is essential for maintaining cerebral perfusion and metabolic stability, ensuring an adequate supply of oxygen and glucose to support both vascular health and cognitive function. Impaired CVR is linked to various pathologies, including heart failure, stroke, and Alzheimer's disease [1,2]. Because CVR is sensitive to both vascular health and metabolic status, exercise, which profoundly influences these systems, represents a promising intervention for improving cerebrovascular function. Moderate-intensity continuous exercise

(MICE) has been shown to enhance cerebrovascular function, cognitive performance, and cerebral angiogenesis [3]. However, adherence to such programs is often limited by time constraints. Consequently, time-efficient alternatives such as high-intensity interval exercise (HIIE), which alternate brief bouts of vigorous activity with recovery periods, have gained attention for providing comparable or even superior cardiometabolic and vascular benefits. Despite these advantages, the acute and chronic effects of these modalities on CVR remain poorly understood [4], knowledge that is crucial for implementing exercise as a safe and effective strategy to maintain cerebrovascular health.

A widely used form of HIIE is sprint interval exercise (SIE), which typically alternates supramaximal efforts with recovery periods. SIE has been shown to improve cardiorespiratory fitness, metabolic health, and blood pressure regulation [5] and is associated with better adherence due to its shorter duration and lower perceived exertion compared with MICE [6]. Most SIE protocols are derived from the Wingate Anaerobic Test (WAnT), a 30-s all-out cycling sprint primarily dependent on anaerobic glycolysis following rapid phosphocreatine depletion. This leads to lactic acid accumulation, decreased blood pH, and compensatory hyperventilation that increases expired CO_2 [7,8]. The resulting reduction in arterial CO_2 (hypocapnia), induces cerebral vasoconstriction and alters vascular tone. Indeed, middle cerebral artery velocity (MCAv) remains suppressed for at least 20 min post-WAnT [9], while full autonomic recovery can exceed one hour [10,11]. As a conventional SIE session includes multiple WAnT sprints interspersed with rest periods, examining the acute cerebrovascular response to a single WAnT provides a critical foundation for understanding how sprint exercise influences CVR.

To date, few studies have examined CVR responses to acute exercise. Existing evidence indicates that CVR to hypocapnia, assessed via voluntary hyperventilation, remains unchanged following both MICE and HIIE [12,13], whereas results for hypercapnic responses are inconsistent likely due to differences in experimental methods [12–14]. For instance, Burma et al. reported reduced CVR up to one-hour post-HIIE using a rebreathing method, whereas Weston et al. observed no changes up to three hours post-HIIE using steady-state CO_2 inhalation. Importantly, these HIIE protocols involved intensities below the peak power output (PO_{peak}) achieved during $\dot{V}O_{2max}$ testing. Whether all-out sprint efforts exceeding PO_{peak}, such as the WAnT, acutely influence CVR remains unknown.

Moreover, potential sex differences in CVR responses to sprint exercise have not been examined, as previous studies typically included mixed groups without separate analyses. Studies exploring sex-related differences in resting CVR have yielded mixed findings, with the majority of studies reporting greater responsiveness in females [15–18], others reporting no difference [19,20], and some showing higher responsiveness in males [21]. These discrepancies likely reflect variations in menstrual cycle control, measurement techniques, and analytical approaches. Accordingly, this study aimed to investigate the acute effects of a single 30-s all-out Wingate sprint on CVR in healthy young males and females. CVR was assessed using voluntary hyperventilation-induced hypocapnia immediately before and 30 min after exercise. The 30-min post-exercise time point was chosen to ensure participant safety and measurement reliability. It was hypothesised that CVR would be reduced following the WAnT, with a similar reduction expected in males and females.

2. Results

2.1. Participant Characteristics

Descriptive characteristics and physiological responses from the ramp incremental and Wingate anaerobic tests are shown in Table 1. Males showed higher $\dot{V}O_{2max}$ during the ramp test as well as higher absolute and relative PO_{peak} and PO_{mean} values during the WAnT.

Table 1. Participant characteristics and physiological values from the ramp and Wingate anaerobic test.

	Males	Females	*p* Value
n	**12**	**12**	
Age, yr	23 (6)	21 (3)	0.75
Height, m	1.78 ± 0.06 *	1.63 ± 0.07	0.002
Weight, kg	73.3 ± 9.7 *	59.6 ± 9.8	<0.001
BMI, $kg \cdot m^{-2}$	23.0 ± 2.3	22.3 ± 2.6	0.506
$\dot{V}O_{2max}$, $L \cdot min^{-1}$	3.67 ± 0.81 *	2.31 ± 0.77	<0.001
$\dot{V}O_{2max}$, $mL \cdot kg^{-1} \cdot min^{-1}$	50.1 ± 7.8 *	38.6 ± 8.2	0.002
WAnT PO_{peak}, W	929 ± 260 *	564 ± 158	<0.001
WAnT PO_{peak}, $W \cdot kg^{-1}$	12.8 (2.6) *	9.9 (2.5)	0.003
WAnT PO_{mean}, W	601 ± 122 *	380 ± 89	<0.001
WAnT PO_{mean}, $W \cdot kg^{-1}$	8.2 ± 1.1 *	6.4 ± 1.1	<0.001

Data are means ± SD for variables that were normally distributed and median with interquartile range in parentheses for non-normally distributed variables in one or both groups. *n*, no. of participants. BMI, body mass index; $\dot{V}O_2$, oxygen uptake; PO, power output; WAnT, Wingate Anaerobic Test. * $p < 0.05$ vs. females.

2.2. Cardiovascular and Cerebrovascular Variables

Group mean cardiovascular and cerebrovascular variables at baseline and during the last 10 s of 1 min of hyperventilation before and after the Wingate test are shown in Table 2, while individual MCAv, end-tidal partial pressure of carbon dioxide ($P_{ET}CO_2$) and CVR responses to the hyperventilation stimulus are displayed in Figure 1.

Table 2. Cardiovascular and cerebrovascular variables at baseline and during the last 10 s of 1 min of hyperventilation before and after the Wingate test.

	Males		Females	
	Pre	**Post**	**Pre**	**Post**
n	**12**	**12**	**12**	**12**
Baseline $P_{ET}CO_2$, mmHg	36.8 ± 3.1	32.1 ± 4.0 *	36.5 ± 4.6	32.1 ± 4.7 *
Baseline MCAv, $cm \cdot s^{-1}$	63.5 ± 10.8	57.6 ± 10.1 *	70.1 ± 11.6	66.8 ± 10.6
Baseline MAP, mmHg	81.4 ± 14.6	81.8 ± 9.7	81.4 ± 18.8	86.5 ± 18.5
Baseline CVCi, $cm \cdot s^{-1}/mmHg^{-1}$	0.80 ± 0.19	0.71 ± 0.15 *	0.89 ± 0.18	0.80 ± 0.17 *
Baseline PI	0.93 ± 0.13	0.88 ± 0.14	0.87 ± 0.18	0.83 ± 0.12
Change in MCAv, $cm \cdot s^{-1}$	25.0 ± 6.3	19.3 ± 6.8 *	29.8 ± 9.1	23.8 ± 7.9 *
Change in $P_{ET}CO_2$, mmHg	14.4 ± 4.0	12.2 ± 4.0 *	15.4 ± 5.5	12.5 ± 5.0 *
CVR, $cm \cdot s^{-1}/mmHg$	1.79 ± 0.35	1.59 ± 0.26 †	2.01 ± 0.44	2.01 ± 0.46

Data are means ± SD; *n* = no. of participants. $P_{ET}CO_2$, end-tidal partial pressure of carbon dioxide; MCAv, middle cerebral artery blood velocity; MAP, mean arterial pressure; CVCi, cerebrovascular conductance index; PI, pulsatility index; CVR, cerebrovascular reactivity. * $p < 0.05$ vs. pre within the same group (i.e., within males or within females). † $p < 0.05$ vs. females within same time-point (i.e., within pre- or post-Wingate).

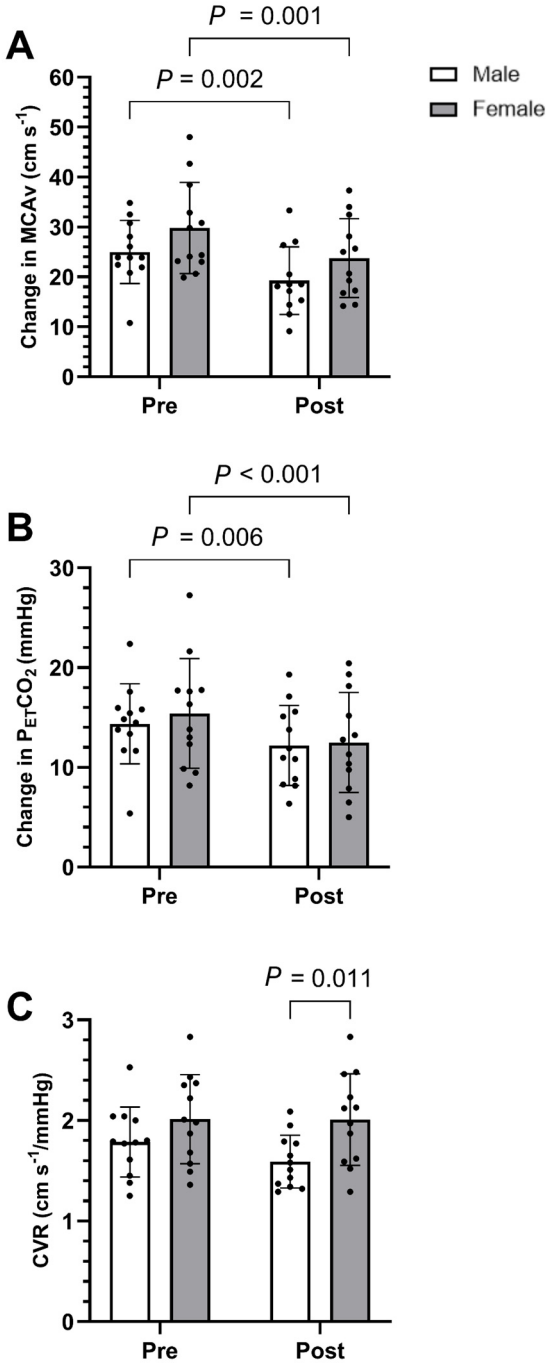


Figure 1. Individual and mean (±SD) physiological responses to 1 min of hyperventilation (i.e., hypocapnia) before and after the Wingate test in males (n = 12; white bars) and females (n = 12, grey bars). The change in MCAv (**A**) and $P_{ET}CO_2$ (**B**) represent the difference between resting baseline and the final 10 s of the hyperventilation. (**C**), cerebrovascular reactivity. MCAv, middle cerebral artery blood velocity; $P_{ET}CO_2$, end-tidal partial pressure of carbon dioxide; CVR, cerebrovascular reactivity.

2.2.1. Baseline Data

At baseline, there was no time × sex interaction for $P_{ET}CO_2$ (p = 0.869, η_p^2 = 0.001); however, there was a main effect of time (p < 0.001, η_p^2 = 0.732), with resting $P_{ET}CO_2$ being lower after the WAnT in both groups (males: p < 0.001, d = 1.31; females: p < 0.001, d = 0.95). Similarly, there was no time × sex interaction for resting MCAv (p = 0.428, η_p^2 = 0.029), but there was a significant main effect of time (p = 0.010, η_p^2 = 0.263). Post hoc analyses showed baseline MCAv decreased after the WAnT in males (p = 0.018, d = 0.57) but not in females (p = 0.172, d = 0.29). A significant main effect of time was also observed for resting

cerebrovascular conductance index (CVCi) ($p = 0.005$, $\eta_p^2 = 0.312$), without a time × sex interaction ($p = 0.989$, $\eta_p^2 < 0.001$), these values being lower following the WAnT in both males and females (males: $p = 0.037$, $d = 1.31$; females: $p = 0.035$, $d = 0.57$). Resting mean arterial pressure (MAP) and the pulsatility index (PI) were unaffected by the sprint exercise in either group.

2.2.2. Cerebrovascular Reactivity

There was no time × sex interaction for the hyperventilation induced change in MCAv ($p = 0.896$, $\eta_p^2 = 0.001$) but a significant main effect of time was present ($p < 0.001$, $\eta_p^2 = 0.535$), with lower MCAv values post-WAnT in both males and females (males: $p = 0.001$, $d = 0.88$; females: $p = 0.002$, $d = 0.71$) (Table 2, Figure 1). Similarly, the hyperventilation-induced reduction in $P_{ET}CO_2$ was significantly smaller after the WAnT (main effect of time, $p < 0.001$, $\eta_p^2 = 0.541$) in both groups (males: $p < 0.001$, $d = 0.54$; females: $p < 0.001$, $d = 0.55$) with no time × sex interaction ($p = 0.465$, $\eta_p^2 = 0.024$). Finally, the WAnT did not significantly alter CVR in either group (main effect of time, $p = 0.207$, $\eta_p^2 = 0.071$; time × sex interaction, $p = 0.226$, $\eta_p^2 = 0.066$). However, there was a significant main effect of sex ($p = 0.028$, $\eta_p^2 = 0.201$) with CVR post-WAnT significantly higher in females than males ($p = 0.012$, $d = 1.14$), whereas pre-WAnT CVR did not differ between groups (Table 2, Figure 1).

3. Discussion

To our knowledge, this is the first study to investigate CVR following a maximal sprint exercise, specifically a Wingate test. The main findings were: (a) despite a reduction in MCAv after the Wingate test, contrary to our hypothesis, CVR was not significantly altered by the sprint exercise in either males or females; and (b) CVR appeared to be better maintained in females than in males, as post-exercise CVR was significantly higher in females, despite no sex differences at baseline.

3.1. Effect of Sprint Exercise on CVR to Hypocapnia

The cerebral vasculature is highly reliant on intrinsic regulatory mechanisms to ensure adequate blood flow, thereby preventing both ischemia and cerebral oedema, conditions to which the brain is particularly susceptible. CO_2 is one of several metabolic factors (alongside potassium and adenosine) that influence cerebrovascular tone. CVR reflects the ability of the cerebral vasculature to adjust its diameter, and thus blood flow, in response to changes in P_aCO_2. This ensures that the brain receives sufficient oxygen and nutrient delivery while facilitating removal of waste products such as CO_2. Due to this relationship, CVR and ventilation are tightly coupled, and the ratio of the change in MCAv (measured via transcranial Doppler ultrasound) to the change in $P_{ET}CO_2$ provides a reliable and reproducible measure of the cerebral vasculature's responsiveness to hypocapnia [22].

The Wingate Anaerobic Test primarily relies on anaerobic glycolysis for ATP resynthesis following the depletion of phosphocreatine stores within the first ~5 s of activity [7]. This reliance on glycolysis leads to a rapid accumulation of lactic acid, causing a significant drop in blood pH. As a compensatory mechanism, hyperventilation increases CO_2 exhalation, functioning as a respiratory buffer [8]. The net result is a state of hypocapnia, often reflected by $PaCO_2$ below 35 mmHg, as observed herein. Hypocapnia leads to cerebral vasoconstriction, likely through reduced proton concentrations as described by Le Chatelier's principle [1]. Changes in pH may also modulate enzyme activity associated with vascular tone regulation, such as nitric oxide synthase and cyclooxygenase [1]. In the present study, despite the observed post-exercise reductions in MCAv and $P_{ET}CO_2$, CVR to hypocapnia was not significantly affected. These findings are consistent with

previous studies reporting no significant changes in CVR to hypocapnia following acute bouts of HIIE and/or MICE [12,13]. Therefore, the current study extends prior findings demonstrating that, even after a maximal sprint effort, the vasoconstrictive capacity of the middle cerebral artery is preserved in young healthy individuals.

Although CVR to hypercapnia was not assessed in the present study, previous work by Burma et al. reported a significant reduction (37%) in hypercapnic CVR immediately and one hour after a HIIE session consisting of 10 × 1 min at 85% heart rate reserve [13]. This impairment was attributed to the repeated intense exercise bouts which likely induced prolonged periods of hyperventilation. The resulting hypocapnia may have caused cerebral vasoconstriction, altering vessel tone and limiting the capacity for maximal cerebrovascular vasodilation. In contrast, Weston et al. did not observe any change in CVR to hypercapnia at one and three hours following two different HIIE protocols (5 × 2–3 min at either 75% or 90% $\dot{V}O_{2max}$), suggesting that the vasodilatory capacity of the cerebral vessels is preserved following both protocols [12]. The divergent findings between these studies likely reflect methodological differences in the assessment of CVR. While Burma et al. employed a rebreathing method that elicited large increases in $P_{ET}CO_2$ (~30 mmHg), Weston et al. used steady-state increases in $P_{ET}CO_2$ through fixed concentration CO_2 breathing, which resulted in more moderate increases in $P_{ET}CO_2$ (~10 mmHg). Whether a single Wingate test can induce similar alterations in cerebrovascular tone remains unknown. Future research should therefore examine CVR to hypercapnia following sprint exercise to clarify the broader cerebrovascular consequences of maximal effort activity.

3.2. Sex-Related Effects on CVR to Hypocapnia Following Sprint Exercise

This study is the first to examine sex-related differences in CVR following an acute bout of sprint exercise in young, healthy adults. Although CVR was not affected by sprint exercise within either group, females exhibited significantly greater CVR than males after the Wingate test, suggesting that cerebrovascular function was better preserved in females post-exercise.

Previous research on sex differences in resting CVR remains inconclusive. While findings vary, the majority of studies have reported greater responsiveness in females [15–18], with some showing no difference [19,20], and others indicating greater responsiveness in males [21]. These inconsistencies are probably due to methodological differences, including how menstrual cycle phases were controlled, the nature of the stimuli employed, and the measurement or data analysis techniques used. Among the studies reporting higher responsiveness in females, Skinner et al. found that MCAv–CO_2 responsiveness to hypo-to-hypercapnia was greater in females than in males irrespective of menstrual phase, suggesting a hormonal basis for these sex-related differences [18]. Oestrogen promotes vasodilation by enhancing nitric oxide production and reducing vascular tone, whereas progesterone exerts vasoconstrictive effects. Consequently, authors proposed that the higher oestrogen-to-progesterone ratio in females (and higher testosterone levels in males) likely contributed to lower vascular tone and greater cerebrovascular CO_2 responsiveness in females, despite hormone levels and cerebrovascular tone not being measured in that study [18]. Differences in ventilatory sensitivity to CO_2 may also influence these responses, although evidence for sex-related effects remains inconclusive [1]. Although no significant sex-related differences in resting CVR were observed in the present study, despite values being ~12% higher in females, it is possible that these hormone-dependent differences in cerebrovascular tone may have contributed to the better preservation of CVR in females following exercise. It is also important to note that both absolute (W) and relative ($W \cdot kg^{-1}$) power outputs during the 30-s all-out effort were significantly greater in males compared with females. This difference likely elicited a larger hyperventilatory stimulus during and

after the sprint exercise in males, potentially contributing to the more favorable maintenance of CVR observed in females post-exercise. Future studies employing matched relative exercise intensities between sexes are warranted to more accurately delineate sex-specific cerebrovascular responses to sprint exercise.

3.3. Limitations

This study has limitations that should be acknowledged. Firstly, menstrual cycle phase was not controlled among female participants due to time constraints. Although the influence of menstrual phase on CVR remains debated [23,24], evidence suggests that sex-related differences in CVR seem largely independent of menstrual phase [18]. Nonetheless, menstrual phase can affect CVR in females [18]; therefore, future studies should account for this variable. Secondly, transcranial Doppler ultrasound was used to measure cerebral blood velocity, which serves as a valid surrogate for cerebral blood flow only if the middle cerebral artery diameter remains constant [25]. However, MCA diameter can vary with changes in CO_2, potentially leading to over- or underestimation of CBF when velocity is used as a proxy [26,27]. Despite this limitation, transcranial Doppler ultrasound continues to be a well-established and extensively utilised technique for evaluating cerebrovascular responsiveness, offering meaningful information about cerebrovascular function and overall vascular health when applied and interpreted carefully [22]. Thirdly, CVR was assessed only under hypocapnic conditions. As hypocapnia primarily induces cerebral vasoconstriction, the present findings reflect a single aspect of cerebrovascular control. Future studies incorporating hypercapnic stimuli would enable a more comprehensive evaluation of cerebrovascular reactivity and determine whether the preserved responsiveness observed herein extends across both vasoconstrictive and vasodilatory ranges. Finally, as participants were recreationally active university students, the findings may not be generalisable to highly trained or sedentary populations, older individuals or clinical populations.

4. Materials and Methods

4.1. Participants

Twenty-four recreationally active participants (12 males) were recruited from the student community at Trinity College Dublin. The study protocol was approved (protocol code: 230906) by the Faculty of Health Sciences Research Ethics Committee, Trinity College Dublin, and written informed consent was obtained from all participants before enrolment. Participants were screened for exclusion criteria, including contraindications to maximal exercise, smoking, and history of metabolic, cardiovascular, or cerebrovascular disease.

4.2. Experimental Protocol

4.2.1. Overview

In this study participants completed one preliminary and one experimental visit. During the preliminary visit, participants completed a ramp incremental and verification test to exhaustion to determine their maximal oxygen uptake ($\dot{V}O_{2max}$), and were familiarised with the WAnT protocol and the procedures involved in the cerebrovascular data acquisition. The experimental visit involved completing a WAnT, with CVR measurements to hypocapnia as well as dynamic cerebral autoregulation (dCA) measurements (both, following a single sit-stand and 5 min repeated sit-stands maneuvers) taken immediately before and 20 min after the sprint exercise bout (dCA first, followed by CVR). dCA responses to single sit-stand were also recorded immediately after the WAnT. Data presented in the current manuscript are based on the CVR measurements to hypocapnia. Data on dCA following 5 min of repeated sit-stands have been reported previously [28], whereas data on dCA following single sit to stand are not presented herein. The experimental visit was

completed at least 48 h, but no more than 2 weeks, after the preliminary visit. All exercise sessions were conducted on the same electronically-braked cycle ergometer (Lode Excalibur, Lode, Groningen, The Netherlands). For each visit, participants confirmed compliance with the pre-test instructions, which included being at least 2 h postprandial, abstaining from vigorous exercise and alcohol for 24 h, and avoiding caffeine on the day of testing.

4.2.2. Preliminary Visit: Familiarization and Ramp Incremental Exercise

Participants were first familiarised with the cycle ergometer, during which the saddle and handlebars were adjusted for comfort. They were then equipped with a nose clip and mouthpiece (Hans Rudolph, Shawnee, OK, USA) attached to a metabolic measurement system (Innocor, Innovision A/S, Odense, Denmark) for the continuous breath-by-breath assessment of oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), and minute ventilation ($\dot{V}E$). The system was calibrated before each testing session in accordance with the manufacturer's guidelines.

The ramp incremental test began with a 2-min warm-up at 10 W, after which workload increased continuously at 20–30 $W \cdot min^{-1}$ until volitional exhaustion. Participants were instructed to maintain a pedalling cadence of 70–90 revolutions per minute (rpm) throughout the protocol. Exhaustion was defined as the inability to sustain a cadence > 70 rpm for five consecutive seconds despite strong verbal encouragement. $\dot{V}O_{2max}$ was subsequently confirmed using a supramaximal verification bout performed at 105% of peak power achieved during the ramp test, following a 10-min recovery period [29]. Following an additional 15–20 min of rest, participants were familiarised with the Wingate WAnT.

4.2.3. Experimental Visit

Participants arrived at the laboratory and completed at least 10 min of seated rest whilst they were fitted with the experimental measurements. The assessment of CVR was conducted before and 30 min after completing the WAnT.

4.3. Experimental Measures

At baseline, participants remained seated for 60 s and were asked to complete deep hyperventilation at a rate of 25 breaths/min for 60 s following a 50 bpm rhythm-based online metronome, beginning with inhalation on the first beat and exhalation on the second beat.

MCAv was assessed bilaterally using transcranial Doppler ultrasonography. The left and right MCAs were insonated at depths of approximately 45–50 mm with 2-MHz probes secured using a robotic headset (Multigon Industries, Inc., Yonkers, NY, USA). In instances where the MCAv signal could only be located or maintained on one side (n = 18 herein), unilateral MCAv was used, with the same side used for pre- and post-exercise measurements within each participant [30]. To account for the potential influence of arterial pressure on CVR, beat-to-beat blood pressure was recorded via the arterial volume clamp method (Finometer-M2, Finapres Medical Systems, The Netherlands), with the handheld at heart level throughout the protocols. $P_{ET}CO_2$ was continuously measured using a calibrated gas analyser (AD Instruments, Colorado Springs, CO, USA). All data were simultaneously collected at 1000 Hz through an analogue-to-digital converter (PowerLab 8/30, AD Instruments, Colorado Springs, CO, USA) and stored for off-line analysis using dedicated software (LabChart 7, AD Instruments, Colorado Springs, CO, USA).

4.4. Wingate Anaerobic Test

Participants completed a maximal WAnT on the cycle ergometer against a resistance equivalent to 7.5% of their body mass. Following a 2 min warm-up at 20 W, participants were given a countdown and instructed to accelerate to maximal cadence and sustain an

all-out effort for 30 s while remaining seated, with strong verbal encouragement throughout the protocol. Immediately afterward, participants completed 2 min of unloaded cycling, before a 30 min seated recovery under supervision prior to repeating the CVR assessments.

4.5. Data Analyses

Cerebrovascular reactivity was quantified as the absolute change in MCAv relative to baseline per 1 mmHg change in $P_{ET}CO_2$ calculated using the final 10 s of hyperventilation. To examine changes in the relationship between mean arterial pressure (MAP) and MCAv, the cerebrovascular conductance index (CVCi) was calculated as the baseline MCAv/baseline MAP. MCA pulsatility index (PI) was determined using the Gosling flow pulsatility index, as the difference between systolic and diastolic MCAv divided by MCAv mean [31]. Pilot work conducted within our laboratory (n = 24) demonstrates that the within-day coefficient of variation for the CVR statistic (based on two measurements separated by 50 min of seated rest) is 9.9%, while the between-day coefficient of variation (based on measurements obtained on four separate days), is 16.2%.

4.6. Statistical Analysis

This study was powered to detect a large effect size for a sex*time interaction using a mixed model (within-between) ANOVA design. This was based on previous research which has reported a large effect size (d = 0.82) for baseline sex differences in CVR to hypocapnia using hyperventilation in healthy young adults (N = 9 to 11 per group) [18], and large-very large (d > 1.4) effect sizes for post-exercise reductions in CVR following HIIE in a sample of 9 young adults [13]. At an α of 0.05, a power of 0.8 and a Cohen's f = 0.42 (large), 10 participants were needed per group. In order to account for a ~20% drop-out or difficulty acquiring or maintaining robust MCAv traces, 12 males and 12 females were recruited per group.

Data are expressed as mean ± standard deviation for variables meeting normality assumptions and as median (interquartile range, IQR) for variables that were not normally distributed. Statistical analyses were conducted using SPSS (Version 29; IBM Corp., Armonk, NY, USA). Normality was evaluated with the Shapiro–Wilk test alongside visual inspection of histograms and Q–Q plots. Sex-based differences in participant characteristics and physiological responses to the ramp incremental test and WAnT were examined using independent samples t-tests for normally distributed variables, whereas the Mann–Whitney U test was applied when parametric assumptions were not satisfied. Acute responses to the WAnT were assessed using a two-way mixed-model ANOVA, with time (pre vs. post) specified as the within-subject factor and sex as the between-subject factor. Where significant main effects or interactions were identified, post hoc pairwise comparisons were performed using the Least Significant Difference (LSD) procedure with effect sizes reported as partial eta squared (ηp^2) and categorized as small ($\leq$0.06), medium (0.06–0.14), or large ($\geq$0.14) [32]. Additionally, standardized effect sizes for pairwise comparisons are provided as Cohen's d, classified as small (<0.5), moderate (0.5–0.8), or large ($\geq$0.8). Statistical significance was defined as $p < 0.05$. Figures were prepared using GraphPad Prism (GraphPad Software, San Diego, CA, USA, version 10).

5. Conclusions

These findings suggest that both resting MCAv and $P_{ET}CO_2$ remain reduced 30 min after a single bout of maximal sprint exercise in healthy young adults. Nevertheless, cerebrovascular reactivity to hypocapnia appears preserved. Notably, females demonstrated a more favorable maintenance of CVR post-exercise compared to males. These results provide new insights into sex-specific cerebrovascular responses to sprint exercise and

support the resilience of the cerebral vasculature to short-term perturbations in arterial CO_2 following maximal sprint exercise in healthy young individuals.

From a practical standpoint, a single bout of maximal sprint exercise, such as the Wingate anaerobic test, appears to be well tolerated from a cerebrovascular perspective in young recreationally active individuals when conducted under controlled conditions. The preservation of CVR further indicates that sprint-based exercise does not acutely impair cerebrovascular regulatory capacity. These observations provide cautious support for sprint interval training as a time-efficient exercise strategy in this population. However, extension of these findings to clinical or older populations should be approached with caution and requires further investigation. The more favourable maintenance of CVR observed in females underscores the importance of considering sex as a biological variable in cerebrovascular research and may have implications for individualized exercise prescription. Finally, training status may influence cerebrovascular responses to high-intensity exercise, as physiological adaptations associated with regular training could modify cerebrovascular regulation and recovery [33]. Therefore, future research should examine the moderating role of training status, while longitudinal studies are needed to determine whether repeated exposure to sprint exercise elicits beneficial cerebrovascular adaptations.

Author Contributions: Conceptualization, P.B., M.E.W., E.L.C., N.G. and M.E.; methodology, P.B., M.E.W., E.L.C., N.G. and M.E.; software, P.B., M.E.W. and E.L.C.; validation, P.B., M.E.W. and E.L.C.; formal analysis, P.B.; investigation, P.B., M.E.W. and E.L.C.; resources, N.G. and M.E.; data curation, P.B., M.E.W. and E.L.C.; writing—original draft preparation, M.E.; writing—review and editing, P.B., M.E.W., E.L.C., N.G. and M.E.; visualization, P.B. and M.E.W.; supervision, M.E.W., N.G. and M.E.; project administration, M.E.W.; funding acquisition, N.G. and M.E. All authors have read and agreed to the published version of the manuscript.

Funding: This research was funded by Research Ireland, Grant Number: 21/FFP-P/10097.

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the by the Faculty of Health Sciences Research Ethics Committee, Trinity College Dublin (protocol code 230906, 13 May 2024).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: Data related to the current study outcomes will be provided upon reasonable request by emailing the corresponding author (megana@tcd.ie).

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

CVCi	Cerebrovascular conductance index
CVR	Cerebrovascular reactivity
dCA	Dynamic cerebral autoregulation
HIIE	High-intensity interval exercise
MAP	Mean arterial pressure
MCAv	Middle cerebral artery velocity
MICE	Moderate-intensity continuous exercise
min	minute
P_aCO_2	Partial pressure of arterial carbon dioxide
$P_{ET}CO_2$	Partial pressure of end-tidal carbon dioxide
PI	Pulsatility index
PO	Power output
$\dot{V}O_2$	Oxygen uptake

$\dot{V}O_{2max}$	Maximum oxygen uptake
WAnT	Wingate Anaerobic Test

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