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CASE REPORT

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Improved ventilatory response during exercise over time after concussion: A case report

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Abstract

This case study reports the exercise ventilatory responses of a 17-year-old female who sustained a traumatic brain injury (TBI) which resulted in loss of consciousness. Subsequently, she suffered from post-concussion syndrome accompanied by orthostatic and exercise intolerance. A cardiopulmonary exercise test (CPET) was performed 2 years post-TBI. The results demonstrated significant hypoventilation with elevated $PetCO_2$ and ventilatory equivalents, progressive desaturation, and pre-syncope symptoms limiting exercise capacity. A repeat CPET 4 years post-TBI demonstrated a similar exercise capacity limited by pre-syncope symptoms. However, there was a marked improvement in the ventilatory response, with appropriate ventilation, $PetCO_2$, and ventilatory equivalents, and only minor desaturation near peak exercise. Hypoventilation during exercise has been reported in subjects with post-concussion syndrome; however, with time the exercise ventilatory response can potentially normalize as observed in this subject.

KEYWORDS
concussion, exercise, hypoventilation, ventilatory response.

INTRODUCTION

Post-concussion syndrome (PCS) may occur following a traumatic brain injury (TBI) and can result in prolonged cognitive, emotional, and physical effects, including exercise intolerance.[1] Increased risk factors of PCS include a history of three or more concussions, female sex, younger age, and affective disorders such as depression and anxiety.[1] Hypoventilation during exercise is common in subjects with PCS and may cause symptom exacerbation during submaximal exercise leading to early exercise cessation.[1] This case study reports the interval difference in the exercise ventilatory responses of a 17-year-old female suffering from PCS at two and 4 years post-TBI.

CASE REPORT

The subject sustained a sport related TBI which resulted in loss of consciousness. In the days following the TBI the subject recalled a further five head knocks, none of which resulted in loss of consciousness. In the following 2 years she suffered from PCS, and experienced symptoms including postural pre-syncope, frequent dizzy spells, tunnel vision, fainting during relatively light-intensity exercise, headaches, and constant neck/shoulder pain. Cardiology investigations diagnosed postural orthostatic tachycardia syndrome. During a physiotherapy session for persistent neck/shoulder pain she desaturated (<90%) during submaximal exercise leading to a referral for a cardiopulmonary exercise test (CPET) at our Respiratory Physiology Laboratory.

CPET 1—Two years post-TBI

The subject completed a CPET using the standardized exponential exercise protocol on a treadmill. Peak oxygen uptake ($\dot{V}_{O2peak}$) was 22.3 mL min^{-1} kg^{-1} (62% predicted), peak workload was 180 W, and the modified Borg dyspnoea score was 3/10 (Table 1). Heart rate (HR) and oxygen pulse response were within normal limits however blood pressure (BP) decreased during exercise. The striking abnormality

was significant hypoventilation throughout exercise as demonstrated by low minute ventilation, breathing frequency, and ventilatory equivalents (O_2 and CO_2), progressive desaturation, and high breathing reserve and end tidal partial pressure for carbon dioxide ($PetCO_2$) (Figure 1). The CPET was stopped by the respiratory physiologist due to presyncope symptoms (light-headedness and tunnel vision), combined with decreased BP and oxygen saturations.

TABLE 1 Peak exercise parameters for CPET 1 and CPET 2

Parameter	CPET 1	CPET 2	Difference
$\dot{V}_{O2peak}$ (mL min^{-1} kg^{-1})	22.3	22.7	↔
Load (W)	180	185	↔
Modified Borg dyspnoea score	3/10	6.5/10	↑
HR (1 min^{-1})	162	157	↔
O_2 pulse (mL)	10.7	11.3	↔
SBP (mmHg) – *baseline / peak*	143 / 119	101 / 94	↔
DBP (mmHg) – *baseline / peak*	80 / 57	75 / 76	↔
$\dot{V}_E$ (L min^{-1})	36	56	↑
BF (1 min^{-1})	22	41	↑
BR (%)	63	45	↓
SpO_2 (%)	93	96	↑
$PetCO_2$ (kPa)	6.78	4.71	↓
$PetO_2$ (kPa)	12.67	14.75	↑
EqO_2	18.9	28.1	↑
$EqCO_2$	19.6	30.0	↑

Note: The arrows in the column labelled 'Difference' indicate: ↔ = no change, ↑ = increase, or ↓ decrease in response between CPET1 and CPET 2. The red arrows highlight the improvements in ventilatory response.
Abbreviations: BF, breathing frequency; BR, breathing reserve; DPB, diastolic blood pressure; $EqCO_2$, ventilatory equivalent for carbon dioxide; EqO_2, ventilatory equivalent for oxygen; HR, heart rate; O_2 pulse, oxygen pulse; $PetCO_2$, end tidal partial pressure for carbon dioxide; $PetO_2$, end tidal partial pressure for oxygen; SBP, systolic blood pressure; SpO_2, oxygen saturation; ṾE, minute ventilation; $\dot{V}O_{2peak}$, peak oxygen uptake.

CPET 2—Four years post-TBI

Compared to CPET 1, the subject achieved a similar $\dot{V}_{O2peak}$ (22.7 mL min^{-1} kg^{-1}, 69% predicted) and peak workload (185 W) but had a greater modified Borg dyspnoea score (6.5/10) (Table 1). Again, HR and oxygen pulse responses were within normal limits, but BP dropped during exercise. There was a marked improvement in the ventilatory response during exercise with an appropriate increase in minute ventilation and breathing frequency, normal $PetCO_2$ and ventilatory equivalents (O_2 and CO_2), and only minor desaturation near peak exercise (Figure 1). The CPET was stopped by the subject due to "dizziness and pressure before the eyes (but not tunnel vision)".

DISCUSSION

A review at 4 years post-TBI showed the subject had experienced an improved, yet still reduced, exercise capacity due to pre-syncope, presumably due to hypotension. During the CPET she achieved the same exercise capacity but demonstrated a markedly improved ventilatory response which may be explained by improved CO_2 sensitivity or

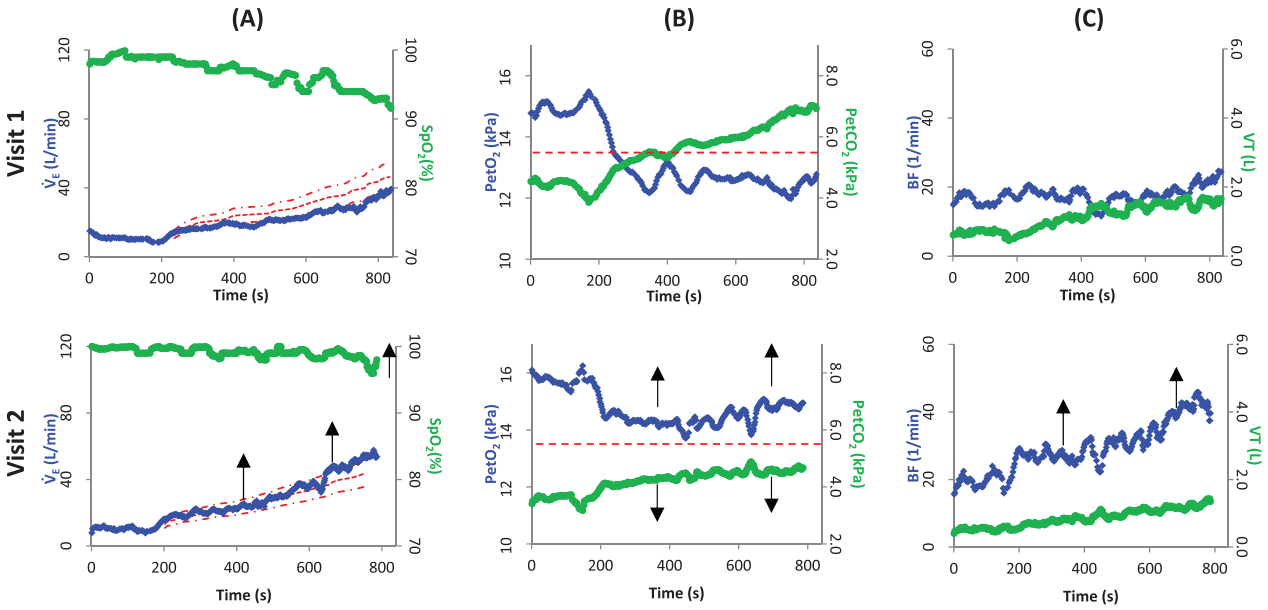

FIGURE 1 Shows CPET data from visits 1 and 2. The important changes at visit 2 are indicated by the black arrows. Column A: plots minute ventilation ($\dot{V}_E$) on primary *y*-axis and oxygen saturations (SpO_2) on secondary *y*-axis versus time (s). The red dashed lines represent expected $\dot{V}_E$ and the upper and lower limits of normal. Column B: presents end tidal partial pressure of oxygen ($PetO_2$) on primary *y*-axis and end tidal partial pressure of carbon monoxide ($PetCO_2$) on secondary *y*-axis versus time (s). The red dotted lines indicate the expected peak exercise $PetCO_2$. Column C: shows breathing frequency (BF) on primary *y*-axis and tidal volume (VT) on secondary y-axis versus time (s)

neuroplasticity that remodelled the ventilatory response during exercise.

In a controlled study, Clausen et al. (2016) showed that young adult females with PCS had abnormally low CO_2 sensitivity.[2] This caused hypoventilation which caused arterial carbon dioxide levels ($PaCO_2$) to rise disproportionately with exercise intensity. As cerebral blood flow is correlated with $PaCO_2$, they observed an increase in cerebral blood flow velocity which was associated with pre-syncope symptoms.[2] Despite normal $PetCO_2$ being observed during CPET 2, our subject still experienced exercise-induced pre-syncope symptoms which were likely attributed to hypotension during exercise. The exact mechanism of this cardiovascular response remains unclear if not driven by hypercapnia. Additionally, submaximal exercise training (e.g., 20 mins of aerobic exercise at 80% of the max HR achieved during a CPET performed on 5–6 days per week) has been shown to improve CO_2 sensitivity and may normalize the ventilatory response during exercise.[1,2] In the subsequent years post-TBI, the subject continued to perform informal submaximal exercise which may have improved her CO_2 sensitivity and increased her ventilatory drive during exercise.

Neuroplasticity occurs in the central nervous system (CNS) and is defined as the ability of the brain to make adaptive changes to neuronal circuits on a structural and functional level.[3] The CNS may recover or adopt secondary compensatory mechanisms after injury. The literature suggests that subjects who sustain severe head injuries during adolescence or young adulthood tend to have better recovery than older adults.[4,5] It is plausible that the subject's ventilatory response during exercise improved because of neuroplasticity.

Assessment of the respiratory and cardiovascular responses during exercise by a CPET is useful in subjects struggling with PCS and may identify the physiological mechanism of exercise intolerance. Hypoventilation during exercise can occur in subjects with PCS; however, with time the exercise ventilatory response can potentially normalize as observed in this subject.

AUTHOR CONTRIBUTIONS

Data collection/analysis/interpretation: All authors. Article drafting/critical revision: RW and BK. Final review: All authors.

CONFLICT OF INTEREST STATEMENT

None declared.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

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