

# RATIONALE

- Previous investigations have demonstrated a greater bradycardic response to apnea after exposure to acute and chronic hypoxia (versus normoxic responses). (1, 2)
- Hypoxia and hypercapnia elicit similar sympathetic, cardiovascular, and ventilatory responses during free breathing. (3)

### We sought to investigate if:

• Hypercapnia elicits a similarly enhanced bradycardic response as hypoxia during apnea.

We hypothesized that apneas after breathing hypercapnia would cause an augmented bradycardic response compared to apneas conducted during normocapnia.

# METHODS

- Heart rate (3-lead ECG), blood pressure, end tidal partial pressures of O<sub>2</sub> &  $CO_2$ , ventilation, and oxygen saturation were measured continuously.
- There were no differences in responses to apnea in males and females; therefore, data for males and females are pooled.



Figure 1. Experimental protocol. Apnea durations are approximates and participants were asked to hold their breath until volitional failure. Note: participants completed two additional conditions (hypoxia and hypercapnic hypoxia) as part of a larger project; only data from the hypercapnic condition are presented on this poster.



and heart rate (HR) during a hypercaphic apnea.

# **Table 1.** Participant demographics.

	Males (n = 13)	Female
Age (years)	$23\pm2$	23
Height (cm)	$178\pm8$	165
Weight (kg)	$76 \pm 14$	65
BMI (kg/m²)	$24\pm3$	24

Data are presented as mean  $\pm$  SD. \* *P* < 0.05 vs. males.

# Heart Rate Response to End-Expiratory Apnea During Hypercapnia

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10 min Recovery

es (n = 12)  $23\pm3$  $55\pm5^*$  $\pm$  11\*  $4 \pm 4$ 

# RESULTS

<b>Table 2.</b> Baseline values for the minute prior to each apnea (n = 25).				
	Normocapnia	Hypercapnia	Р	
HR (bpm)	$70\pm9$	$73\pm10$	0.006	
MAP (mmHg)	$88\pm5$	$92\pm 6$	<0.001	
SBP (mmHg)	$115\pm7$	$121\pm10$	<0.001	
DBP (mmHg)	$74\pm 6$	$77\pm 6$	<0.001	
P <sub>ET</sub> CO <sub>2</sub> (torr)	$40\pm3$	$46\pm4$	<0.001	
P <sub>ET</sub> O <sub>2</sub> (torr)	$91\pm5$	$90\pm5$	0.004	
SpO <sub>2</sub> (%)	$98 \pm 1$	$98 \pm 1$	0.027	
VE (L/min)	$13\pm4$	$22\pm7$	<0.001	

Data are presented as mean  $\pm$  SD. *HR* heart rate, *MAP* mean arterial pressure, SBP systolic blood pressure, DBP diastolic blood pressure, P<sub>FT</sub>CO<sub>2</sub> end-tidal carbon dioxide, P<sub>ET</sub>O<sub>2</sub> end-tidal oxygen, SpO<sub>2</sub> arterial oxygen saturation, VE minute ventilation. Statistical significance is set at *P* < 0.05 (*bolded*).

<b>Table 3.</b> Apnea duration and cardiovascular values during apneas (n = 25).					
	Normocapnia	Hypercapnia	Р		
Apnea Duration (s)	$23\pm8$	$19\pm7$	<0.001		
ΔHR (bpm)	$-11 \pm 15$	$-14 \pm 14$	0.069		
ΔMAP (mmHg)	$23\pm10$	$21\pm8$	0.395		

Data are presented as mean  $\pm$  SD.  $\Delta HR$  change in heart rate from baseline to nadir during apnea, ΔMAP change in mean arterial pressure from baseline to peak during apnea. Statistical significance is set at *P* < 0.05 (*bolded*).



### Cardiac Cycle

**Figure 3.** Mean (± SD) change in heart rate during the final 10 cardiac cycles of apnea after hypercapnia (diamonds) and normocapnia (circles) relative to the resting heart rate averaged from 1 minute preceding each respective apnea (n = 25). The nadir is the mean response of each participant's single lowest beat during the final 10 cardiac cycles of each apnea. No statistically significant differences between conditions during any beat.

### FUNDING

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### **Figure 4.** Ventilatory response to normocapnia and hypercapnia (n = 25). Hypercapnia elicited a greater ventilatory response (P < 0.001) than normocapnia.



Figure 5. Relationship between the ventilatory response to hypercapnia and the subsequent heart rate response to apnea. Linear regression model suggests there is no relationship (r = -0.324, P = 0.114, n = 25). The y-axis depicts the difference in the change in heart rate from baseline to nadir between hypercapnia and normocapnia (note: a negative value indicates a greater bradycardic response during hypercapnia). The x-axis depicts the difference in ventilation between hypercapnia and normocapnia (note: a positive value indicates a greater ventilatory response during hypercapnia). Shaded area represents the 95% CI of the regression curve.

# INTERPRETATIONS

- during hypoxia. (1)

REFERENCES

- Physiological Reports, 2021.
- altitude natives. Journal of Applied Physiology, 2018.
- function. American Journal of Physiology, 2009.



• Our data demonstrate that hypercapnia does not augment the bradycardic response to apnea; this response differs from that seen

• An absence of statistically significant bradycardia suggests that cardiovagal output is not increased during hypercaphia.

• No individual predictions can be made about the heart rate response to apnea based on the preceding ventilatory response to hypercapnia.

(1) Busch et al., Short-term hypoxia does not promote arrhythmia during voluntary apnea.

(2) Busch et al., Chemoreflex mediated arrhythmia during apnea at 5,050 m in low- but not high-

(3) Steinback et al., Hypercapnic vs. hypoxic control of cardiovascular, cardiovagal, and sympathetic

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