Acute Cardiovascular, Metabolic, and Muscular Responses to Blood Flow Restricted Rowing Exercise

Sean J. Mahoney; Nathan D. Dicks; Katie J. Lyman; Bryan K. Christensen; Kyle J. Hackney

INTRODUCTION: Microgravity leads to a progressive loss in muscular strength, endurance, and aerobic capacity ($\dot{V}O_{2peak}$). Blood flow restricted (BFR) exercise has been shown to elicit rapid gains in muscular strength and $\dot{V}O_{2peak}$. Rowing exercise combined with BFR could be a supplemental countermeasure to maintain preflight muscle function and $\dot{V}O_{2peak}$, especially within future space vehicles with restricted physical volume.

METHODS: There were 20 men who completed 19 min of rowing exercise during CON or BFR in a randomized order. Exercise intensity for all sets was 30% of peak work load achieved during a separate incremental maximal exercise test. Kaatsu training cuffs were inflated around each leg during BFR. Muscle oxygen saturation (SmO_2) and heart rate (HR) were measured throughout exercise and rest. Rate of perceived exertion (RPE) and muscle activation, using surface electromyography (sEMG), were measured during the last 30 s of each exercise set. Blood pressure (BP) and whole blood lactate ($[La^-]_b$) were measured at rest and postexercise.

RESULTS: SmO_2 declined significantly in BFR during exercise and rest by 13% and 14%, respectively. HR and RPE showed significant increases during BFR (120.5 ± 5.53 vs. 128.9 ± 9.86 bts $\cdot$ min^{-1}) (9.8 ± 1.85 vs. 11.8 ± 1.88 arbitrary units). No differences were observed for BP, $[La^-]_b$, and sEMG.

DISCUSSION: Findings indicate exercise intensity and cuff pressure elicited acute muscular, cardiovascular, and perceptual responses. BFR rowing exercise could be advantageous as an adjunct for future exercise countermeasures where aerobic and anaerobic exercise may be performed on one device or in limited physical space.

KEYWORDS: long duration spaceflight, in-flight exercise protocols, health and human performance.

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It is well established that long-duration spaceflight leads to a progressive loss in muscular strength, endurance, and aerobic capacity.[2,3,12] This microgravity-induced deconditioning is detrimental to astronaut health, performance, and overall mission success. Exercise countermeasures are in place on board the International Space Station to combat in-flight deconditioning; however, these current methods do not mitigate deconditioning entirely.[19] Postflight strength assessments have demonstrated deficits for the knee (4–9%), ankle (1–4%), and trunk flexor (6%) muscles following 30 d of post-spaceflight exercise recovery.[9,16] In addition, peak oxygen consumption ($\dot{V}O_{2peak}$) and left ventricular mass decrease early in flight by ~17% and ~12%, respectively.[12,15,22] Even a relatively small decrease in $\dot{V}O_{2peak}$ (e.g., 10%) can greatly impact an astronaut's ability to meet the high-energy demands of emergency mission tasks, as these tasks may require ambulatory participants to work at intensities with excessive metabolic demand, which may result in heart rate (HR) responses exceeding 85% of maximum HR.[3] Research has demonstrated high-intensity exercise prescriptions with respect to workload result in positive musculoskeletal and cardiovascular adaptations.[9,26] Current in-flight exercise devices allow for greater exercise intensities but require extensive physical volume in the space vehicle. Performance decrements are still observed despite these highly specialized devices and exercise prescriptions; therefore, more advanced exercise equipment is necessary for future missions beyond low-Earth orbit.[9,19,26]

Earth-based research analogs aiming to counter microgravity-induced deconditioning through exercise primarily use bed

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rest with a 6° head down tilt (6°HDT) to simulate the detrimental physiological effects of microgravity.[12,15,23] Currently, compact rowing devices that allow for both aerobic and resistance training are in development as possible exercise equipment for future missions.[15,23,30] Concurrent rowing and resistance exercise training during 5 wk of bed rest with 6°HDT was capable of preventing the cardiac atrophy and stiffening that occurs with prolonged bed rest.[15] In addition, aerobic and resistance exercise completed on a novel concurrent flywheel exercise device during 70 d of bed rest with 6°HDT was capable of maintaining key myocellular characteristics in the vastus lateralis (VL) muscle; however, researchers indicated further refinement or supplementation of the exercise protocol is necessary to impact the soleus, a primary muscle impacted by microgravity-induced deconditioning.[22,30] These findings demonstrate rowing exercise using isoinertial flywheel devices can maintain cardiovascular function during prolonged bed rest but requires additional supplementation to simultaneously maintain muscle function. It is suggested that new forms of exercise supplementation or forms of "artificial gravity" are necessary, as exercise alone has not been shown to mitigate spaceflight deconditioning.[14,26]

Blood flow restriction (BFR) is a novel form of exercise intervention, which involves the application of inflated tourniquet cuffs that safely restrict venous blood flow during exercise.[6] This restriction creates an artificial form of gravity through lower body negative pressure (LBNP), and when coupled with low-intensity exercise, provides a high-intensity workload without requiring increased exercise load or volume.[14,26] BFR exercise has been shown to elicit rapid and progressive gains in muscular strength, endurance, and aerobic capacity.[1,8,25] Low-intensity interval BFR training has been identified as an ideal mode of exercise to simultaneously improve aerobic fitness and muscular strength when compared with low-intensity interval training without BFR, high-intensity interval training, and combined high-intensity interval training and BFR.[8] The restriction of venous blood flow during BFR reduces stroke volume, thus increasing HR to maintain cardiac output and meet energy demands.[12,27] This unique cardiovascular and musculoskeletal response may allow crewmembers to exercise at lower intensities and still elicit a training response capable of maintaining their preflight muscle function and $\dot{V}o_{2peak}$.

The purpose of this study was to identify the acute cardiovascular, musculoskeletal, perceptual, and metabolic responses when combining BFR with low-intensity rowing exercise. This novel exercise intervention fits within the constraints of future spaceflight missions by requiring minimal equipment, physical space, and power, while offering the potential to maintain preflight strength and $\dot{V}o_{2peak}$ using a simulated high-intensity exercise modality during prolonged spaceflight.

METHODS

Subjects

There were 20 healthy and regularly active men (22.1 ± 1.71 yr, 172.89 ± 24.49 cm, 87.26 ± 27.40 kg) who volunteered and provided written informed consent to participate. All procedures and instruments used during this study were approved by the university's Institutional Review Board. Any participant with, or at risk for, hypertension, hypotension, classified as obese (BMI $\geq$ 30 kg $\cdot$ m^{-2}), had recent surgery, muscle disease, or history of cardiovascular disease were excluded. Participants completed both control and intervention sessions in a within-subjects, cross over design.

Instrumentation

$\dot{V}o_{2peak}$ was determined through completion of an incremental exercise test on a Concept2 Model E rowing ergometer (Concept2, Morrisville, VT) and $\dot{V}o_2$ was measured with a TrueOne 2400 metabolic cart (Parvo Medics, Sandy, UT). Telemetry HR monitor values were recorded (Polar Electro Inc, Lake Success, NY, USA) and extracted via streaming software (Golden Cheetah, Version 3.0.2). BP was taken using a manual sphygmomanometer cuff and stethoscope. Muscle activation was collected using surface electromyography (sEMG) self-adhesive Red Dot 2560 monitoring electrodes (3M Healthcare, London, Ontario, Canada) and Biopac Acknowledge 4.0 software (MP150, Biopac Systems Inc., Goleta, CA). Participant rate of perceived exertion (RPE) was ranked using the Borg 6–20 rating scale and whole blood lactate ($[La^-]_b$) was measured with a handheld $[La^-]_b$ analyzer (Lactate Plus, NOVA Biomedical, Waltham, MA). Muscle oxygen saturation (Smo_2) was collected using a MOXY brand near-infrared spectroscopy device (MOXY, Fortiori Design LLC, Hutchinson, MN). During BFR exercise, 5-cm wide Kaatsu Nano training cuffs (Kaatsu Global, Inc., Japan) were applied to the proximal portion of both legs to restrict blood flow.

Procedures

All participants completed a $\dot{V}o_{2peak}$ session followed by the CON and BFR testing session. Each session was separated by at least 48 h and attempts were made to ensure participants completed all exercise sessions at the same time of day.

Participants arrived for the first session and were familiarized with the procedures and instruments being utilized by the research team. Informed consent forms were obtained, and inclusion/exclusion screening was implemented. Before exercise, baseline HR and BP were taken. $\dot{V}o_{2peak}$ was determined using an incremental exercise test on a Concept2 Model E rowing ergometer. For all exercise testing, the rower damper setting was set to five. Prior to each test the metabolic cart was calibrated for known gas concentrations and flow via the manufacturer's recommendation. Participants began the test by maintaining an average workload of 100 Watts (W) while rowing for 4 min. After 4 min, the average workload participants were required to maintain was increased by 25 W. This protocol was repeated with an increased workload of 25 W every minute until the participant reached exhaustion. Exhaustion was determined when the participant met two of the following criteria: 1) unable to remain within 10–15 W of average workload for $\geq$ 15 s; 2) no increase in $\dot{V}o_2$ or HR despite increased exercise intensity; 3) respiratory exchange ratio greater than 1.10; or 4) HR exceeded 90% of age-predicted maximum HR for $\geq$ 15 s.

Expired gas was collected and analyzed every 15 s. Peak work rate in Watts (W_{peak}) was determined using the following equation: W_{peak} = [avg. wattage at last stage completed + (time of uncompleted stage/stage duration × stage increment)], and W_{peak} was used to determine exercise intensities during the BFR and CON session. Mean $\dot{V}O_{2peak}$ for all participants was 47.57 ± 6.95 $ml \cdot kg^{-1} \cdot min^{-1}$.

The following exercise session involved two trials: exercise with free blood flow (CON) and exercise with blood flow restriction (BFR). At least 20 min of rest separated each trial and participants completed the trials in a randomized order. Before exercise, baseline HR, BP, $[La^-]_b$, and SmO_2 were measured. In addition to cardiovascular measures, muscle activation was monitored through sEMG by placing electrodes on the medial portion of the right leg VL and biceps femoris (BF) muscles 10–15 cm superior to the proximal border of the patella with a 4-cm interelectrode distance. A reference electrode was placed on the patella to complete the circuit. These placings are similar to those used by Wernbom et al.[31] and allow for the application of the BFR training cuffs without interrupting electrode placement or restricting movement.

After electrode placement, and before exercise began, participants completed five maximal effort rowing strokes on the Concept2 Model E rowing ergometer. These rowing strokes were used for sEMG normalization. Muscle activation during exercise was measured during the final 30 s of each exercise set. For analysis, all sEMG signals were firstly band-pass filtered (20 – 450 Hz) using digital infinite impulse response filtering. Then, the root mean square value using 25-ms intervals of the entire signal was computed using Biopac Acknowledge 4.0 software. Area of the 30-s sEMG signal was divided by the number of rowing strokes completed during the time frame to calculate a sEMG impulse/stroke value for each set. This impulse/stroke value was then normalized to the sEMG impulse/stroke value achieved during the five maximal effort rowing strokes to calculate relative percentage of muscle activation during each rowing stoke.

Once electrodes were secured, a MOXY device was placed below the BFR cuff on the belly of the left VL, midway between the greater trochanter and the lateral epicondyle of the femur and secured with adhesive athletic tape.[21] This device measured muscle oxygen saturation, total hemoglobin (THb), oxygenated hemoglobin (OxyHb) and deoxygenated hemoglobin (DeoxyHb). Crum et al.[7] have established this device to be reliable to measure SmO_2 at low to moderate exercise intensities.

Participants completed five 3-min sets of exercise with a 1-min rest in between sets for a total of 19 min.[25] All exercise was completed on the same Concept2 Model E rowing ergometer used during the incremental max test. Exercise intensity was set at 30% of W_{peak} achieved during the $\dot{V}O_{2peak}$ session.[1,8] Average exercise intensity for all participants was 80.78 ± 14.15 W. Postexercise HR, BP, $[La^-]_b$, and SmO_2 measurements were taken 5 min postexercise. HR and SmO_2 levels were also monitored throughout the entire exercise session including rest intervals, and these data were averaged across all exercise sets and rest intervals. RPE and sEMG were recorded simultaneously during the last 30 s of each exercise set. Participants were verbally encouraged to complete exercise to the best of their ability.

The same protocol used in the CON trial was used during the BFR trial. However, during BFR, participants had 5-cm Kaatsu Nano training cuffs applied on the proximal portion of both legs. Immediately before exercise began, participants experienced a traditional Kaatsu Cycle warm-up, which involves eight rounds of acute cuff inflation followed by deflation. In brief, each round had the cuffs inflated for 20 s followed by 5 s of total cuff deflation. The Kaatsu Cycle began at an inflation of 40 Standard Kaatsu Units (SKU) (40 mmHg). After the 5 s of deflation, the cuff pressure increased by ~15–20 SKU (15–20 mmHg) for each subsequent round, ending at ~160 SKU (160 mm Hg). To find optimal SKU for exercise, researchers measured individual participant's capillary refill time, or the time in seconds taken for color to return to an external capillary bed, during the cycle. Pressure was applied to the quadriceps, just above the knee, to cause blanching during cuff inflation. The Kaatsu training manual identifies a capillary refill time of ~3 s as optimal SKU pressure for exercise; pressure was continually increased by ~15-20 SKU (15–20 mmHg) until optimal SKU was reached. However, to ensure participant safety, an individualized training cuff inflation limit was set for each participant at 1.3 × their resting systolic BP, which has been used previously during supine exercise and unloading analogs.[13] During the BFR trial, training cuffs remained inflated throughout all exercise and rest periods. Mean thigh circumference taken at the right leg and cuff inflation pressure for all participants was 57.55 ± 4.04 cm and 157.8 ± 5.27 SKU (157.8 ± 5.27 mmHg), respectively.

Statistical Analysis

All analyses were performed using SPSS version 24.0 (IBM, Armonk, NY). In the event of missing data, pairwise deletion was used in the statistics. Repeated measures ANOVAs (condition × time) were used to analyze HR, BP, $[La^-]_b$, sEMG, and SmO_2 at varying time points. HR data during trials was averaged across all sets and analyzed pre-, during, and postexercise. BP and $[La^-]_b$ were analyzed pre- and postexercise. sEMG was analyzed for each exercise set. SmO_2 exercise data was averaged across all exercise sets and SmO_2 rest data was averaged across all rest periods. Muscle oxygenation was then analyzed comparing SmO_2 during CON exercise and rest to BFR exercise and rest. A paired sample *t*-test was used to compare peak RPE during CON and BFR. Statistical significance was determined by $P < 0.05$. When a significant *F* statistic was found, additional tests with Bonferroni adjustments were performed to reduce type II error rate.

RESULTS

There was a significant condition × time effect for HR [$F(2, 38) = 5.220$, $P = 0.010$, $\eta p^2 = 0.089$]. Follow-up comparisons determined there was a significant elevation of HR during BFR compared to CON [120.5 ± 5.53 vs. 128.9 ± 9.86 bpm; $t(19) = -4.940$, $P < 0.001$] (**Fig. 1**). No statistically significant

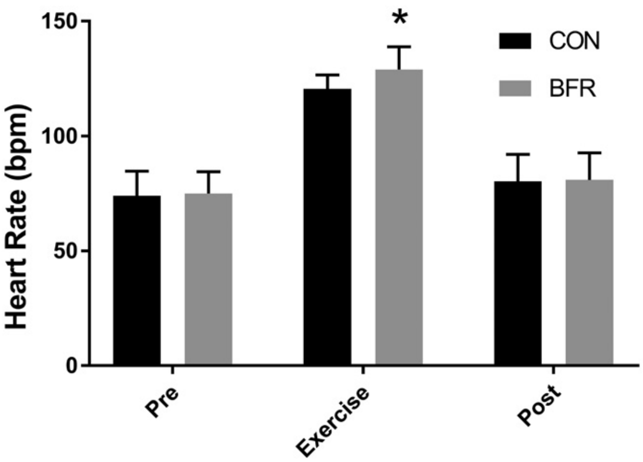

Fig. 1. Peak HR taken pre, during, and postexercise. *Significantly different from pre- and postexercise ($P < 0.05$). BFR = blood flow restricted; bpm = beats per minute. Error bars indicate standard deviation from the mean.

differences were found for systolic [$F(1, 19) = 1.207$, $P = 0.286$, $\eta p^2 = 0.060$] and diastolic [$F(1, 19) = 3.417$, $P = 0.080$, $\eta p^2 = 0.152$] BP from pre- to postexercise for either BFR or CON (**Table I**). Peak reported RPE was significantly greater during BFR compared to CON [9.8 ± 1.85 vs. 11.8 ± 1.88 arbitrary units; $t(19) = -5.878$, $P < 0.001$] (**Fig. 2**). There were no statistically significant differences observed in $[La^-]_b$ from pre- to postexercise [$F(1, 19) = 0.363$, $P = 0.554$, $\eta p^2 = 0.019$] (Table I) for either BFR or CON.

Due to equipment malfunction and data loss, we were unable to report muscle activity and muscle oxygenation on all 20 participants. Three participant's sEMG data were dropped, and eight participant's muscle oxygenation data were dropped, resulting in an *N* of 17 and 12, respectively. There was no significant difference in muscle activation between BFR and CON for the VL [$F(4, 64) = 1.181$, $P = 0.328$, $\eta p^2 = 0.069$] and BF [$F(4, 64) = 0.759$, $P = 0.556$, $\eta p^2 = 0.045$] (**Table II**). Smo_2 and OxyHb were significantly lower during BFR compared to CON [$F(1, 11) = 23.23$, $P < 0.001$, $\eta p^2 = 0.679$; $F(1,11) = 26.39$, $P < 0.001$, $\eta p^2 = 0.706$, respectively] (**Fig. 3** and **Table III**). DeoxyHb was significantly higher during BFR compared to CON [$F(1, 11) = 23.80$, $P < 0.001$, $\eta p^2 = 0.684$] (Table III). There was a significant condition × time effect for THb [$F(1, 11) = 4.912$, $P = 0.049$, $\eta p^2 = 0.309$]. Follow-up comparisons determined significantly greater THb during CON rest [$t(11) = -2.883$, $P = 0.015$] and BFR Rest [$t(11) = -2.250$, $P = 0.046$] compared to CON exercise, and during BFR rest compared to BFR exercise [$t(11) = -3.844$, $P = 0.003$] (Table III).

DISCUSSION

The goal of this study was to establish the acute physiological responses of leg BFR during rowing exercise. The major findings of this study were increased cardiovascular and perceptual responses with BFR without elevating postexercise BP. However, this exercise protocol was insufficient to simultaneously alter metabolic and musculoskeletal responses.

Heart rate response in respect to BFR was greater (6.9%) than during CON. This result is similar to the HR increases observed during previous studies using BFR exercise.[8,27] However, Renzi et al.[27] investigated BFR during walking exercise with similar cuff pressures used in this study and reported an almost 20% increase in HR during walking using BFR compared to free blood flow walking. Interestingly, for the same relative exercise intensity, HR response is lower during ergometer rowing than during treadmill exercise due to its partially supine nature.[32] Rowing exercise promotes venous blood return, thereby increasing stroke volume to a greater extent than other upright exercises (e.g., cycling, walking).[17] Known as the Frank-Starling mechanism, this phenomena may contribute to the ability of rowing exercise to prevent cardiac atrophy and stiffening during prolonged bedrest (a known spaceflight analog) and may be beneficial in preventing left ventricular deconditioning during prolonged spaceflight.[14,15,22]

Consequently, the application of BFR cuffs works to restrict venous blood flow, therefore reducing stroke volume and increasing HR to maintain cardiac output. The interaction of these conflicting mechanics during BFR rowing exercise was beyond the focus of this study, but this interaction could play an important role in prescribing exercise intensity and cuff inflation pressure during BFR rowing exercise during future spaceflight analogs. Further, given this was a 1-G experiment, additional research is required in unloading analogs such as bed rest or parabolic flight to refine exercise prescription.

Loenneke et al.[20] has stated the increased cardiovascular stress traditionally observed during low-intensity BFR exercise is well below the increased cardiovascular stress observed during high-intensity resistance training without BFR. When comparing BP responses during walking with and without BFR, increases in both systolic and diastolic BP are reported.[27,28] The exercise protocol completed in this study showed no changes in either systolic or diastolic BP from pre- to postexercise, demonstrating a beneficial BP response from BFR rowing exercise; however, we cannot rule out the possibility there may have been transient elevations in BP while the cuff was inflated, as cuff inflation pressure and exercise modality are likely factors influencing BP responses. A lack of increased BP response postexercise suggests excess arterial stress was not placed on the vascular system during exercise. The partially supine and seated nature of

Table I. Blood Pressure and Whole Blood Lactate.

	CON		BFR	
VARIABLE	**PRE**	**POST**	**PRE**	**POST**
Systolic BP (mmHg)	120.6 ± 6.59	122.05 ± 7.23	121.5 ± 5.65	117.6 ± 22.63
Diastolic BP (mmHg)	77.8 ± 5.73	79.9 ± 3.14	79 ± 4.47	78.6 ± 4.45
$[La^-]_b$ (mmol/L)	1.21 ± 0.66	1.19 ± 0.56	1.12 ± 0.50	1.20 ± 0.43

Data are mean ± SD. No significant changes for any variable ($P > 0.05$). BP = blood pressure. $[La^-]_b$ = whole blood lactate.

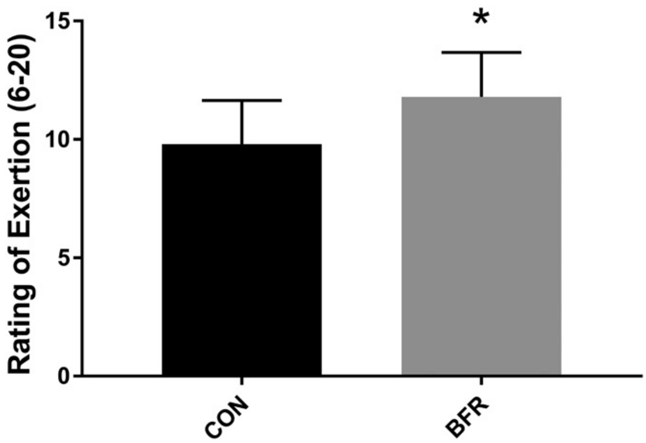

Fig. 2. Peak RPE recorded during the last 30 s of each exercise set for CON and BFR. *Significantly different from CON exercise ($P < 0.05$). CON = exercise with free blood flow. BFR = exercise with blood flow restriction. Error bars indicate standard deviation from the mean.

rowing could be a critical factor influencing this beneficial hemodynamic response.

Perceptual responses of exertion have previously been reported to be significantly higher during high-intensity [80% of one rep max (1RM)] resistance exercise compared with low-intensity (20% 1RM) BFR exercise.[24] Additionally, reported RPE in young and older adults was shown to be lower during BFR treadmill exercise compared to traditional high-intensity leg press exercise.[28] However, Hackney et al.[13] also showed that low-intensity BFR resistance exercise at 20% of 1RM with 140 mmHg of cuff inflation using a 5-cm wide cuff was insufficient to elicit differences in RPE compared with low-intensity resistance exercise without BFR. These findings demonstrate traditional high-intensity (e.g., 80% of 1RM) resistance exercise elicits greater perceptual responses of exertion compared to either aerobic or resistance BFR exercise at low-intensities and a threshold of increased perceptual responses may exist with BFR exercise. This threshold appears to be influenced by the complex interplay between relative exercise intensity, cuff width, and pressure. We believe our exercise protocol was above this threshold.[13] Despite reported RPE being greater during BFR than CON, it is important to note perceptual exertion was not near maximal, indicating the exercise prescription used could be increased in future research to elicit increased physiological responses.

Similar to our results, Wernbom et al.[31] reported BFR during low-intensity dynamic knee extension at 30% of 1RM did not increase muscle activity. Additionally, increased relative integrated sEMG of the VL during low-intensity BFR knee extension was shown to be related to the production and accumulation of $[La^-]_b$ during a hypoxic intramuscular environment. The relationship between muscle activation and $[La^-]_b$ accumulation appears to be linear as more glycolytic muscle fibers are recruited with decreases in Smo_2 during hypoxic exercise.[29]

Generally, elevations in $[La^-]_b$ concentrations occur following BFR exercise due to increased rates of fast glycolysis in the ischemic muscle.[13,29] Therefore, there are two possible explanations for this event. First, the lack of elevated $[La^-]_b$ during BFR indicates a possibility the "setting related" stress (exercise intensity, cuff width, and inflation pressure) was not sufficient to significantly challenge metabolic stress. Similar results when using the same cuff size and lower cuff pressure during resistance exercise have been previously reported.[13] Second, it is worth noting that during prolonged aerobic exercise, $[La^-]_b$ can be used to fuel oxidative metabolism.[4] Due to the exercise protocol length, it is possible elevated $[La^-]_b$ was not observed due to increased aerobic metabolism, specifically given the full body muscle activity during rowing exercise. Staunton et al.[28] also demonstrated $[La^-]_b$ measures remained unchanged from baseline after treadmill walking in both young and old adults. It has been previously suggested cuff inflation pressures required to elicit resistance training adaptations are between 160 and 230 mmHg.[5,25,29] The inflation pressures used in this study were between 150 and 160 SKU (i.e., 150–160 mmHg). Therefore, it is also possible the cuff inflation pressures used in the current study were not high enough to elicit additional motor unit recruitment and a lack of $[La^-]_b$ production was observed.

Despite no increase in muscle activation or $[La^-]_b$ measures, the cuff inflation pressures used were sufficient to cause a pooling of blood in the lower extremities and restrict muscle oxygen reperfusion. This cardiovascular response increases metabolic stress as the working muscle loses access to OxyHb. Increasing metabolic stress has been shown to decrease calcium sensitivity of muscle fibers, reducing contractile velocity and force leading to additional recruitment of type II muscle fibers to maintain force output.[18] We hypothesized that sEMG would increase throughout BFR exercise as access to OxyHb decreased. Interestingly, our study did not observe increases in muscle

Table II. Muscle Activation of the Vastus Lateralis and Biceps Femoris.

CONTROL SESSION	**SET 1**	**SET 2**	**SET 3**	**SET 4**	**SET 5**
Vastus Lateralis (%)	31.77 ± 32.81	29.92 ± 20.85	26.49 ± 12.33	26.58 ± 10.13	25.28 ± 9.58
Biceps Femoris (%)	18.50 ± 10.35	26.44 ± 35.18	24.61 ± 29.40	24.91 ± 14.64	18.77 ± 13.54
BFR SESSION	**SET 1**	**SET 2**	**SET 3**	**SET 4**	**SET 5**
Vastus Lateralis (%)	21.75 ± 9.91	24.19 ± 9.07	26.21 ± 12.52	27.94 ± 17.03	29.43 ± 12.51
Biceps Femoris (%)	19.83 ± 14.28	22.09 ± 16.39	18.26 ± 11.61	17.15 ± 13.23	20.05 ± 13.57

Data are mean ± SD, $N = 17$. Percentage of muscle activation (impulse/stroke) during final 30 s of exercise sets after normalization to maximal voluntary muscle activation (impulse/stroke) during maximal rowing attempts. No significant changes for any variable ($P > 0.05$). BFR = blood flow restricted exercise.

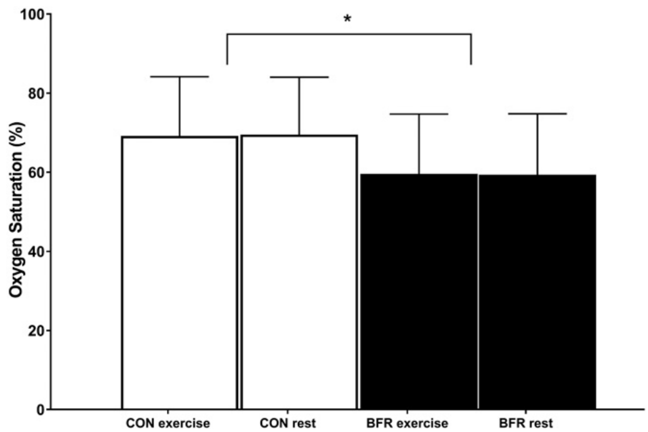

Fig. 3. Averaged Smo_2 during exercise and rest for CON and BFR. *Significantly different from one another ($P < 0.05$). Smo_2 = muscle oxygen saturation. CON = exercise with free blood flow. BFR = exercise with blood flow restriction. Error bars indicate standard deviation from the mean.

activation during BFR, despite lower Smo_2 during exercise and rest. Therefore, it is possible a threshold of muscle oxygen loss is required before a hypoxic intramuscular environment is achieved and additional type II muscle fibers are recruited, enhancing muscle activation. We believe this threshold was not reached during BFR, and the prescribed workloads and cuff pressures used primarily acted as a buffer to OxyHb delivery, evidenced by the still relatively normal Smo_2 during BFR exercise.

Although our data highlight the acute cardiovascular, muscular, perceptual, and metabolic responses to BFR rowing, there are some limitations for this study. Participants recruited were not elite-trained rowing athletes and inexperience with the rowing ergometer could have impacted exercise efficiency during rowing. Additionally, many participants were highly aerobically trained which may have affected cardiovascular and metabolic responses during the exercise protocol. Recruited participants were young, healthy men, making it difficult to generalize the findings to astronaut-aged populations. Lastly, the study was not completed in a microgravity environment and the exercise device for long-duration missions has yet to be decided. The Concept2 rower used here does not benefit from the isoinertial flywheel design seen in previous deconditioning research.[15,23,30] Astronauts are placed at risk for developing behavioral and psychiatric disorders.[11] While exercise provides psychological benefits, one form of exercise may become monotonous, and motivation and adherence to exercise prescriptions may become compromised in the socially isolated environment of space.[10] Therefore, the methods of exercise intervention presented here may prove to be a supplemental prescription, rather than a primary regimen. Nevertheless, our study presents preliminary acute exercise findings that could be important for developing future exercise countermeasures and physiological stimuli to mitigate microgravity-induced deconditioning, warranting further investigation.

The findings of this study were novel in that they indicate low-intensity BFR during rowing exercise was sufficient in elevating HR, decreasing Smo_2, and enhancing subjective exertional responses during exercise without negatively impacting normal postexercise BP responses. However, this protocol was insufficient to elevate muscle activation and carbohydrate utilization. Due to the partially supine nature of rowing, increased relative exercise intensity, cuff width, or inflation pressure may be required to simultaneously elicit cardiovascular and musculoskeletal responses during future training, bed rest, or spaceflight models. Additionally, while decreases in muscle oxygen were observed, the threshold to generate a hypoxic intramuscular environment was not reached; evidenced by the lack of increases in both muscle activation and $[La^-]_b$ production.

Maintenance of preflight muscular and aerobic fitness has yet to be observed with exercise alone. Research suggests that LBNP or other forms of "artificial gravity" will be necessary to maintain fitness during long duration spaceflight missions.[25] Low-intensity BFR during rowing exercise could prove to be a useful exercise countermeasure for microgravity deconditioning as it allows for high-intensity exercise without placing increased physical stress on astronauts or the mission spacecraft. Further studies should focus on evaluating different ranges of exercise intensity with respect to W_{peak}, cuff width, and inflation pressure to elicit elevated muscular and metabolic stress and mimic increased aerobic and anaerobic responses during one method of exercise. One exercise device with multiple capabilities or adjuncts performing at minimal external power within a small amount of volumetric space would be efficacious for future long duration space missions beyond low Earth orbit.

Table III. Hemoglobin Values During Exercise and Rest for CON and BFR.

	CON EXERCISE	CON REST	BFR EXERCISE	BFR REST
THb ($g \cdot dL^{-1}$)	12.33 ± 3.45	12.37 ± 3.46*	12.34 ± 3.46	12.42 ± 3.47*†
OxyHb ($g \cdot dL^{-1}$)	8.48 ± 2.82	8.56 ± 2.82	7.31 ± 2.57‡	7.33 ± 2.63‡
DeoxyHb ($g \cdot dL^{-1}$)	3.85 ± 2.17	3.81 ± 2.11	5.04 ± 2.37‡	5.09 ± 2.40‡

Data are mean ± SD, *N* = 12. Thb = total hemoglobin; OxyHb = oxygenated hemoglobin; DeoxyHb = deoxygenated hemoglobin. *Significantly different from CON Exercise. †Significantly different from BFR exercise. ‡Significantly different from Con Exercise and Con Rest (main effect of exercise type).

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