Journal of Science in Sport and Exercise
https://doi.org/10.1007/s42978-024-00314-8

ORIGINAL ARTICLE

Plasma Volume Shifts and Acid–Base Balance After a Single Bout of Resistance Training

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Received: 26 September 2023 / Accepted: 5 September 2024

Abstract

Purpose Changes in plasma volume (PV), acid–base status and ventilation have rarely been investigated in relation to resistance training (RT). This study aimed to investigate the effect of a single set of exhaustive leg press exercise on these basic physiological parameters in an integrated manner.

Methods Twenty-seven male individuals (27.1 ± 4.1 years, 1.82 ± 0.62 m, 84.4 ± 12.5 kg, BMI: 25.4 ± 3.0 k/gm^2) performed a single set leg press exercise during which hemoglobin concentration ([Hb]), hematocrit (Hct), pH, oxygen (pO_2) and carbon dioxide partial pressures (pCO_2), hydrogen carbonate concentration ([HCO_3^-]), standard base excess (SBE) and lactate concentration ([La^-]) were determined. Total buffer capacity was calculated based on pH, [HCO_3^-] and pCO_2.

Results Mean PV decreased by 559 ± 230 mL (13.7%). As a result, arterial oxygen content was significantly increased due to hemoconcentration ($P < 0.001$). At exhaustion, pH (7.30 ± 0.06), [HCO_3^-] (18.6 ± 2.0 mmol/L) and SBE (− 6.6 ± 2.4 mmol/L) were all significantly decreased ($P < 0.0001$). The pCO_2 first remained unchanged (39.4 ± 4.3 mm Hg) but demonstrated a significant decrease one-minute post-exercise (34.4 ± 4.2 mmHg), indicating metabolic acidosis with respiratory compensation, which was maintained until t_{+15}. Non-bicarbonate buffering remained constant during recovery while the respiratory component steadily increased until 15-min post-exercise (50.2 mmol/L per pH).

Conclusion PV shifts following a single set of leg press exercise improve post-exercise arterial oxygen content. The moderate metabolic acidosis was not compensated during exercise because of restricted breathing but partly compensated during the following 15-min recovery period. The respiratory compensation as part of the bicarbonate buffering made up 50% of total buffer capacity in the course of recovery.

Keywords PH · Bicarbonate · Lactate · Acidosis · Blood volume · Hemoglobin concentration

Introduction

Resistance training (RT) is commonly prescribed to increase underlying strength and power qualities in an attempt to improve athletic performance [58]. In recent years, research in the field of RT has dealt in depth with the associated hormonal [32, 37], morphological [25, 31] and neuronal adaptations [26, 56], as well as the molecular determinants of skeletal muscle hypertrophy and force production [33, 41]. However, first and foremost, any exercise stimulus leads to fundamental vegetative-physiological, e.g., cardiovascular, metabolic and respiratory changes that are linked to acute effects and chronic adaptations, ultimately leading to an increase in exercise performance. While many of these physiological mechanisms have been extensively studied in endurance training, much is still unknown with regard to RT.

These mechanisms include, for instance, exercise-induced changes in plasma volume (PV). It is well known that during short duration and high-intensity or prolonged aerobic exercise, PV undergoes intricate alterations, which are influenced by factors such as changes in intravascular pressure, osmotic regulation or sweat loss leading to exercise-induced changes between 5% and 22% [12, 15, 34–36, 45]. In addition, especially during RT, muscle metabolism with regard

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Published online: 10 October 2024

to lactate production and elimination plays an important role. PV changes have only been previously reported as percentage changes which complicates the understanding of the magnitude and temporal dynamics of PV shifts. This is further illustrated by previous research demonstrating that PV shifts are associated with changes in cardiac output and oxygen delivery [49, 50, 57], as well as nutrient transport to exercising muscles [42] and maybe even lactate transport capacity [51]. The latter is of particular importance as it also directly influences acid–base balance [3, 16].

In this context, it is well known that RT can lead to marked alterations in blood pH and lactate concentrations that have been associated with substantial changes in ventilation. At the same time, buffer capacity reflected in bicarbonate and non-bicarbonate buffering, is also substantially altered when exercising to volitional exhaustion. Taking the carbon dioxide partial pressure (pCO_2) into account, the share of the respiratory component in the defence of the pH value can then be determined. To the best of our knowledge, this has not yet been done. To allow for a more holistic understanding of hematological, metabolic and respiratory regulations during RT, this study aimed to determine exercise-induced changes in PV as well as changes in acid–base balance, total buffer capacity and ventilation during and after a single bout of RT in healthy, trained male individuals.

Materials and Methods

Participants and Sample Size Estimation

Twenty-seven healthy, male individuals with a mean age of 27.1 ± 4.1 years, height of 1.82 ± 0.62 m, body mass of 84.4 ± 12.5 kg (BMI: 25.4 ± 3.0 kg/m^2), skeletal muscle mass of 41.1 ± 5.4 kg (48.9% ± 3.3%) and fat mass of 13.1 ± 6.3 kg (15.1% ± 5.5%) participated in this study. Sample size for statistical significance was calculated according to Hopkins using a change in mean in a post-only crossover approach with maximal rates of statistical type I errors at 5% [29]. Calculations were based on a smallest change in BV of 3% with a within-subject standard deviation (typical error) of 2.5%. This led to a sample size of 13 participants. Eligibility criteria included male individuals with no history of cardiac disease who have been following a self-reported resistance exercise training program including a minimum of two sessions per week for a minimum of one year. Inclusion and exclusion criteria were assessed by the same researcher prior to the start of the study. All investigations were conducted in accordance with the Declaration of Helsinki on ethical principles for medical research on humans and guidelines for Good Clinical Practice and the study protocol was approved by the local ethics committee of the University of Bayreuth (Ethics-No: 23-017). Before any trial related activities, all participants provided written informed consent, which included the aims and risks of the study. Participants were allowed to withdraw from the study at any time without further explanations.

Horizontal Leg Press

Upon arrival at the research facility, lean body mass and fat mass were measured twice consecutively using a bioelectrical impedance analysis (InBody 720, InBody Co., Seoul, South Korea). Afterwards, participants were introduced to the horizontal leg press (Cybex Strength Systems, Rosemont, USA) and their individual starting position was determined, which was set so that the knee angle was slightly below 90 degrees ensuring standardized range of motion. Participants were informed that the movement speed was predetermined as 2 s for the eccentric and concentric phase, respectively, equaling 4 s for one full repetition. Movement velocity was acoustically demonstrated using a metronome. Participants then had to remain in a seated position for ten minutes before a warm-up protocol of 20 repetitions with a standardized weight of 40 kg was performed. To control for posture effects on changes in PV in the post-exercise phase, participants were asked to once again remain in a seated position for 10 min. In order to elicit larger metabolic alterations, a time under tension (TuT) > 90 s was targeted but not obligatory and participants were asked to select a weight of their own choice based on their personal experience in resistance training for the following set. One minute before the start of the exercise, participants were asked to enter the leg press and wait for the countdown. Repetitions were initially performed according to the acoustic feedback signal with standardized repetition duration. However, termination of exercise was defined as the moment at which no further voluntary muscular contraction was possible independent of the repetition duration with participants receiving strong verbal encouragement to reach complete physical exhaustion. Upon completion of the last repetition, participants initially remained within the horizontal leg press for 1 min before they transferred in a seated position and remained there until 15 min post exercise to control posture effects on PV shifts.

Blood Gas Analysis, Acid–Base Status and Spirometry

Capillary blood samples were taken from a hyperemized earlobe at rest, immediately after exhaustion as well as 1-, 3-, 5-, 7-, 10- and 15-min post exercise to determine carbon dioxide partial pressure (pCO_2), oxygen partial pressure (pO_2), oxygen partial pressure at which hemoglobin is 50% saturated (p50), pH, hydrogen carbonate ($[HCO_3^-]$), standard bicarbonate (SBC), actual base excess (ABE), standard base excess (SBE), capillary oxygen saturation (ScO_2),

hemoglobin concentration ([Hb]) and hematocrit (Hct) using a portable, fully automated, spectrophotometric blood gas analyzer (ABL80 FLEX CO-OX-RiliBäK, Radiometer, Copenhagen, Denmark). Therefore, both [Hb] and Hct were directly quantified rather than estimated.

At the same time points, capillary blood samples were taken from the other earlobe for the measurement of lactate ($[La^-]$) and glucose concentrations ($[Glu^-]$) (Biosen S-Line, EKF-Diagnostic, Barleben, Germany). To control for posture effects on the PV response, additional capillary blood samples were taken after the warm-up and just before the start of the leg press exercise. Total buffer capacity (β_{tot}), total bicarbonate (β_{bi}) and non-bicarbonate buffering (β_{nbi}) were calculated subsequently according to the following formulas based on the changes in $[La^-]$, pH and $[HCO_3^-]$:

$$\beta_{tot} = \Delta[La^-]_{Pla} \times \Delta pH^{-1}, \quad (1)$$

$$\beta_{bi} = \Delta\left[HCO_3^-\right] \times \Delta pH^{-1}, \quad (2)$$

$$\beta_{nbi} = \left(\Delta[La^-]_{Pla} \times \Delta pH^{-1}\right) - \left(\Delta\left[HCO_3^-\right] \times \Delta pH^{-1}\right). \quad (3)$$

Using formula 2 and the equation $\Delta pCO_2 \times \Delta pH^{-1} = 1.04 + \beta_{nbi} \times [HCO_3^-]$, we also distinguished between the respiratory (β_{bi_r}) and non-respiratory (β_{bi_nr}) component of the bicarbonate buffer [6]. The $[La^-]$ in the plasma was calculated based on blood $[La^-]$ using a ratio between $[La^-]$ in the erythrocyte and in the plasma of 0.78:1 at rest, 0.45:1 at maximum exercise and 0.5:1 during recovery considering the prevailing Hct [8]. Blood acid–base status was defined according to the alignment nomogram by Siggaard Andersen [1, 2]. Arterial oxygen content (CaO_2) was calculated according to the following formula where 1.39 = Huefner number:

$$CaO_2(mL/dL) = [Hb](g/dL) \times SpO_2(\%) \div 100 \times 1.39(mL/g) \quad (4)$$

A portable, high resolution spiroergometry system with breath-by-breath technology (Metamax® 3B, Cortex Biophysik GmbH, Leipzig, Germany) was used for the continuous measurement of ventilation (VE), oxygen uptake and (VO_2) and carbon dioxide output (VCO_2). Additionally, tidal volume (VT), respiratory rate and respiratory exchange ratio (RER) were also quantified. Hereinafter, quantitative changes of the aforementioned variables are given in relation to their respective measurement time points, which are titled as follows: t_{rest} for resting values, t_{Exh} for immediately after exhaustion, as well as t_{+1}, t_{+3}, t_{+5}, t_{+7}, t_{+10} and t_{+15}, for 1-, 3-, 5-, 7-, 10-, and 15-min post exercise, respectively. Additionally, data for spirometric parameters are also presented in 5-s intervals between t_{Exh} and t_{+1}. All other parameters were averaged over a period of 30 s (15 s before and after the respective measurement time point), whereas peak values, e.g., VO_{2peak}, are given as the highest 30-s interval.

Hemoglobin Mass and Total Blood Volume

The total hemoglobin mass (Hbmass) as well as blood (BV), plasma (PV) and erythrocyte volumes (RCV) were determined twice within 5 days from the leg press exercise using the optimized CO-rebreathing method according to the methods reported previously [22, 46, 52]. In brief, an individual dose of carbon-monoxide (CO, 0.8–1.0 mL/kg, CO 3.7, Linde AG, Unterschleißheim, Germany) was administered and rebreathed along with 3 L of pure medical oxygen (Med. O_2 UN 1072, Rießner-Gase GmbH, Lichtenfels, Germany) for 2 min. Capillary blood samples were taken before and 6- and 8-min post administration of the CO dose and analyzed for the percentage of carboxyhemoglobin (%HbCO) using a blood gas analyzer (ABL80 FLEX CO-OX RiliBäK, Radiometer, Copenhagen, Denmark). The Hbmass was calculated based on the mean change in %HbCO before and after the rebreathing procedure. Subsequently, the total BV was calculated based on the Hbmass and the [Hb] and the PV was calculated based on the total BV and erythrocyte volume (ECV) according to the following formulas:

$$BV\ (mL) = Hbmass\ (g) \times 100 \div [Hb](g/dL) \div 0.91 \quad (5)$$

$$ECV\ (mL) = Hbmass\ (g) \div ([Hb](g/dL) \div Hct \times 100) \times 100 \quad (6)$$

$$PV\ (mL) = BV\ (mL) - ECV\ (mL) \quad (7)$$

where [Hb] = venous hemoglobin concentration and 0.91 = cell factor at sea level representing the ratio between central and peripheral hematocrit [19]. Hematological variables, e.g., [Hb], Hct and PV are also referred to in relation to their measurement times as described above. Since the Hbmass does not change over short periods of time, the temporally offset determination of the [Hb] for the calculation of the BV is possible without compromising accuracy [17]. The typical errors for Hbmass and BV in our laboratory are 1.5% and 2.5%, respectively, which is comparable to previous investigations [30, 47].

Statistical Analysis

The data are presented as means and standard deviations. Statistical analysis was conducted using GraphPad Prism Version 8.0.2 (GraphPad Software, Inc., San Diego, USA). Testing for normality was performed using the Shapiro–Wilk test. On the basis of these results, a repeated measures ANOVA or mixed-effects analysis followed by Tukey's multiple comparisons tests was performed to

prove any significant differences between time points. A repeated measures two-way ANOVA or mixed-effects analysis followed by Tukey's multiple comparisons tests was performed to prove any significant differences between measured values and values corrected for PV changes. Pearson's product moment correlations were performed to prove any relationship between two variables. The level of significance was set to $P < 0.05$ and P-values are presented as * ($P < 0.05$), ** ($P < 0.01$) and *** ($P < 0.001$).

Results

Leg Press Exercise

The mean selected weight in the horizontal leg press was 127.0 ± 22.8 kg equalling 151% ± 23% of body mass. TuT ranged between 75 and 250 s (129 ± 43 s) and maximum heart rate was 157 ± 11.2 beats/min. At t_{Exh}, TuT was significantly correlated with RER (1.26 ± 0.13, $r = 0.58$, $P = 0.001$), [La^-] (7.20.32 ± 1.73 mmol/L, $r = 0.58$, $P = 0.001$), pH (7.30 ± 0.06, $r = -0.3$, $P = 0.17$) and HCO_3^- (18.6 ± 2.0 mmol/L, $r = -0.41$, $P = 0.04$) but not pCO_2 (39.4 ± 4.3 mm Hg, $r = 0.03$, $P = 0.88$).

PV, [Hb], Hct

Total BV and Hbmass were 6938 ± 773 mL (83.0 ± 8.5 mL/kg) and 949 ± 129 g (11.3 ± 1.2 g/kg), respectively. Compared to t_{rest}, PV was significantly decreased until t_{+15} (4046 ± 405 vs. 3753 ± 380 mL, $P < 0.01$) with a maximum decrease observed at t_{+3} (−559 ± 229 mL,−13.7%, see Fig. 1). Individual, maximum percentage changes in PV (ΔPV) ranged between −2.9% and −23.9% (−123 and −1091 mL) and were not correlated with the lowest pH values ($r = 0.25$, $P = 0.22$), $[La^-]_{max}$ ($r = -0.23$, $P = 0.24$) or TuT ($r = 0.02$, $P = 0.89$). We also found no correlation between ΔPV and resting PV ($r = 0.07$, $P = 0.36$) or BV ($r = 0.05$, $P = 0.40$). As a result of the PV decrease, both capillary [Hb] (15.6 ± 0.7 vs. 16.9 ± 1.1 g/dL, $P < 0.0001$) and Hct (48.0 ± 2.0 vs. 51.8% ± 3.3%, $P < 0.0001$) significantly increased between t_{rest} and t_{Exh} and remained significantly increased until t_{+15} ($P < 0.05$). For both [Hb] (17.2 ± 1.0 g/dL) and Hct (52.4% ± 3.2%), maximum values were obtained at t_{+3}.

Acid–Base Balance

pH significantly decreased between t_{rest} and t_{Exh} (7.42 ± 0.02 vs. 7.30 ± 0.06, $P < 0.0001$) and remained decreased until t_{+15} (7.31 ± 0.06, $P < 0.0001$, see Fig. 2). Lowest mean values were observed at t_5 (7.24 ± 0.05). [La^-] were significantly increased until t_{+15} (1.0 ± 0.3 mmol/L vs. 8.9 ± 2.2 mmol/L, $P < 0.0001$, Fig. 2) when compared to t_{rest} with peak values observed at t_{+5} (11.3 ± 1.8 mmol/L). pH and [La^-] were significantly negatively correlated at all post-exercise measurement points (e.g., $r = -0.72$, $P < 0.0001$ at t_{+5}). HCO_3^- (24.6 ± 1.3 vs. 18.6 ± 2.0 mmol/L, $P < 0.0001$) and ABE significantly decreased between t_{rest} and t_{Exh} and

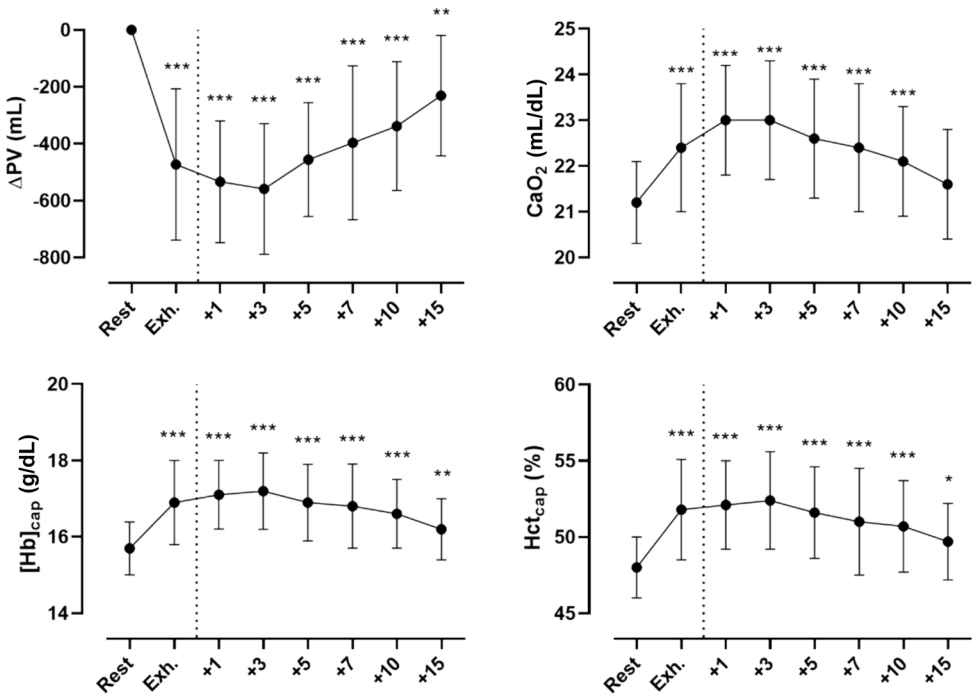

Fig. 1 Exercise-induced changes in plasma volume (ΔPV), arterial oxygen content (CaO_2), hemoglobin concentration ([Hb]) and hematocrit (Hct) during and after single set leg press exercise ($^{*}P < 0.05$, $^{**}P < 0.01$ and $^{***}P < 0.001$ indicate significant compared to resting value)

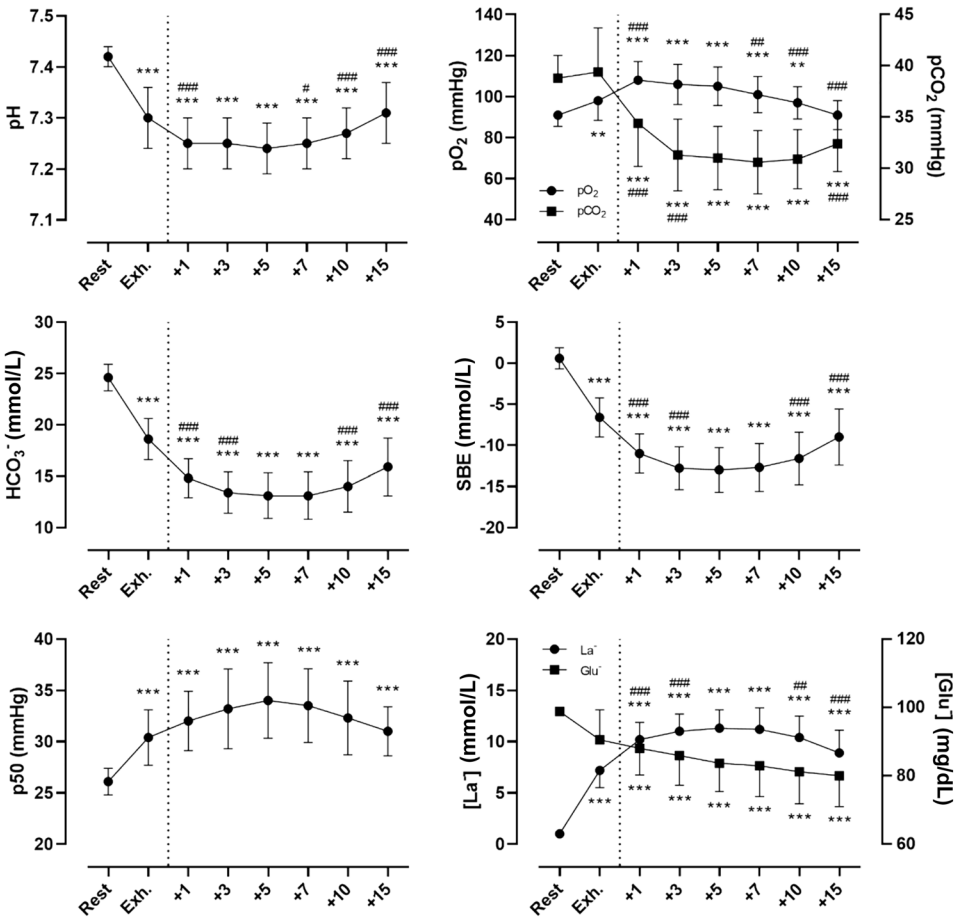

Fig. 2 Measured blood gases and acid–base parameters during and after single set leg press exercise ($^{*}P < 0.05$, $^{**}P < 0.01$ and $^{***}P < 0.001$ indicate significant compared to resting value; $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ and $^{\#\#\#}P < 0.001$ indicate significant compared to previous value)

remained decreased until t_{+15} (all $P < 0.0001$). Lowest values were found at t_{+5}, respectively (see Table 1).

The pO_2 significantly increased between t_{rest} and t_{+10} (91.0 ± 5.5 vs. 97.0 ± 7.9 mmHg) and the highest value was found at t_{+1} (108 ± 9.1 mmHg, see Table 1). The pCO_2 showed no change between t_{rest} and t_{Exh} but a significant decrease between t_{Exh} and t_{+1} (39.4 ± 4.3 vs. 34.4 ± 4.2 mm Hg, $P < 0.0001$) and t_{+3} (31.3 ± 3.5 mm Hg, $P < 0.0001$), respectively. Lowest pCO_2 values were obtained at t_{+7} (30.6 ± 3.1 mm Hg). At t_{+15}, pCO_2 was still significantly decreased compared to t_{rest} (32.4 ± 2.7 mm Hg, $P < 0.0001$). ScO_2 slightly but significantly decreased between t_{rest} and t_{Exh} (97.3% ± 0.7% vs. 96.1% ± 0.9%, $P < 0.0001$) and remained decreased until t_{+15} (95.7 ± 1.2, $P < 0.01$, see Table 1). CaO_2 was significantly increased between t_{rest} and t_{Exh} (21.2 ± 0.9 vs. 22.4 ± 1.4 mL/dL, $P < 0.001$). Highest values were obtained at t_{+3} and t_{+5} and CaO_2 remained significantly increased until t_{+10} ($P < 0.0001$, see Fig. 1). The p50 was significantly increased between t_{rest} and t_{Exh}. Highest values were obtained at t_{+5} (see Table 1) and p50 remained increased until t_{+15} ($P < 0.0001$, see Fig. 2).

Total buffer capacity (β_{tot}), which is composed of bicarbonate (β_{bi}) and non-bicarbonate buffering (β_{nbi}), is shown in Table 2. The β_{nbi} was largest at t_{Exh} (25.2 ± 7.9 mmol/L per pH unit) while no respiratory compensation was detected ($\beta_{bi\_r} = 1.1 \pm 0.13$ mmol/L per pH unit). During post-exercise recovery, β_{nbi} remained on a constant lower level while the respiratory component (β_{bi_r}) steadily increased from t_{+1} (23.8 ± 13.1 mmol/L per pH unit) until t_{+15} (50.2 ± 18.3 mmol/L per pH unit).

Spirometry

VE significantly increased from t_{rest} to t_{Exh} (18.9 ± 3.7 vs. 84.6 ± 18.5 L/min, $P < 0.0001$). Compared to t_{Exh}, VE showed a further significant increase after the first 5 s after t_{Exh} ($P < 0.001$, Fig. 3). VE remained significantly increased until t_{10} (25.2 ± 6.7, $P < 0.01$, see Fig. 3). VO_2 was significantly increased between t_{rest} and t_{Exh} (0.62 ± 0.11 vs. 2.32 ± 0.44 L/min, $P < 0.0001$) and remained significantly increased until t_{+3} (0.96 ± 0.31 L/min, $P < 0.0001$). VCO_2 was significantly increased until t_{+5} when compared to t_{rest} (0.54 ± 0.10 vs. 0.84 ± 0.18 L/min, $P < 0.0001$, see Fig. 3). Peak values for VO_2 and VCO_2 were 2848 mL/min (34.1 ± 7.0 mL/min/kg) and 3640 mL/min (43.6 ± 8.0 mL/min/kg), respectively (see Table 1). Compared to t_{rest},

Table 1 Resting and peak mean values of acid–base, blood gas, spirometric and hematological parameters

Variable	Rest			Peak		
	Mean ± SD	Min	Max	Mean ± SD (t_x)	Min	Max
[La^-] (mmol/L)	0.99 ± 0.25	0.64	1.57	11.32 ± 1.79 (t_{+5})	7.28	14.72
[Glu^-] (mg/dL)	98.8 ± 12.8	80.2	120.4	80.1 ± 9.1 (t_{+15})	56.0	94.2
pH	7.42 ± 0.02	7.38	7.49	7.24 ± 0.05 (t_{+5})	7.14	7.32
HCO_3^- (mmol/L)	24.6 ± 1.3	21.6	27.5	13.1 ± 2.2 (t_{+5})	8.7	19.0
SBC (mmol/L)	25.1 ± 1.1	22.7	27.2	14.8 ± 2.0 (t_{+5})	11.1	19.2
ABE (mmol/L)	0.7 ± 1.3	−2.0	3.2	−13.1 ± 3.1 (t_{+5})	−19.6	−6.5
pO_2 (mmHg)	91.0 ± 5.5	78	105	108 ± 9.1 (t_{+1})	90	126
pCO_2 (mmHg)	38.8 ± 2.2	32.5	42.8	30.6 ± 3.1 (t_{+7})	22.6	38.8
p50 (mmHg)	26.1 ± 1.3	24	29	34.0 ± 3.7 (t_{+5})	28	41
VO_2(L/min)	0.62 ± 0.11	0.38	0.82	2.32 ± 0.44 (t_{Exh})	1.62	3.18
VCO_2 (L/min)	0.54 ± 0.10	0.37	0.85	2.92 ± 0.61 (t_{Exh})	1.83	3.97
RER	0.85 ± 0.07	0.73	0.96	1.68 ± 0.12 (t_{+1})	1.44	1.88
[Hb]cap (g/dL)	15.7 ± 0.7	14.5	16.9	17.2 ± 1.0 (t_{+3})	15.1	19.5
[Hct]cap (%)	48.0 ± 2.0	44.5	51.8	52.4 ± 3.2 (t_{+3})	46.2	59.5
ScO_2 (%)	97.3 ± 0.7	95.5	98.8	96.0 ± 1.0 (t_{+10})	93.9	98.7
CaO_2 (mL/dL)	21.2 ± 0.9	19.8	23.0	23.0 ± 1.2 (t_{+1})	20.8	25.5
PV (mL)	4046 ± 405	2831	4631	3463 ± 397 (t_{+3})	2366	4167
BV (mL)	6938 ± 773	4733	8294	6355 ± 779 (t_{+3})	4391	7510

Table 2 Calculated post-exercise total buffer capacity (β_{tot}), non-bicarbonate (β_{nbi}) and bicarbonate buffering including respiratory (β_{bi_r}) and non-respiratory (β_{bi_nr}) component (numbers in brackets represent the percentage of β_{tot})

Time point	Total buffer capacity	Non-bicarbonate buffering	Bicarbonate buffering	
	(β_{tot})	(β_{nbi})	(β_{bi_nr})	(β_{bi_r})
t_{Exh}	75.8 ± 29.1	25.2 ± 7.9 (33.3%)	49.5 ± 14.8 (65.3%)	1.1 ± 0.13 (1.4%)
t_{+1}	77.5 ± 15.3	18.0 ± 6.7 (23.3%)	35.6 ± 7.8 (46.0%)	23.8 ± 13.1 (30.7%)
t_{+3}	80.8 ± 15.3	15.3 ± 4.1 (18.9%)	30.5 ± 6.5 (37.8%)	35.0 ± 18.5 (43.3%)
t_{+5}	79.2 ± 11.1	14.7 ± 2.8 (18.6%)	30.5 ± 5.0 (38.5%)	34.0 ± 14.5 (42.9%)
t_{+7}	81.2 ± 13.3	14.1 ± 5.6 (17.3%)	29.6 ± 4.6 (36.5%)	37.5 ± 16.2(46.2%)
t_{+10}	87.9 ± 13.5	15.1 ± 3.8 (17.2%)	28.9 ± 5.3 (32.9%)	43.9 ± 18.5 (49.9%)
t_{+15}	98.2 ± 21.6	18.7 ± 5.3 (19.0%)	29.3 ± 6.2 (29.8%)	50.2 ± 18.3 (51.2%)

Data are presented as mean ± standard deviation. Buffer capacity is measured as mmol/L per pH unit [48]

RER was significantly increased at t_{Exh} (0.85 ± 0.07 vs. 1.26 ± 0.13, $P < 0.0001$) and remained significantly increased until t_{+10} (0.99 ± 0.12, $P < 0.05$) with maximum mean values observed at t_{+1} (1.65 ± 0.25, $P < 0.0001$). Both respiratory rate and VT significantly increased between t_{rest} and t_{Exh} (21.3 ± 4.3 vs. 39.1 ± 8.9 and 0.83 ± 0.15 vs. 2.28 ± 0.65 mL, both $P < 0.0001$). Thereafter, respiratory rate and VT demonstrated an opposing trend between t_{Exh} and t_{+1} (see. Figure 3) with respiratory rate showing a significant decrease while VT was significantly increased. Respiratory rate remained significantly increased until t_{+3} ($P = 0.03$) while VT was significantly increased until t_{+7} ($P < 0.01$).

Discussion

The aims of this study were to quantify the effect of RT on exercise-induced PV changes and the associated effects on oxygen transport, and to examine the acute changes in acid–base balance with special consideration of respiratory adaptation and buffer capacity. Our results demonstrate that mean PV was significantly reduced by − 559 mL (~ 14%) and remained decreased until 15-min post-exercise. Due to the hemoconcentration, CaO_2 was significantly increased throughout the post-exercise period. Immediately at exhaustion, acid–base balance showed mild acidosis without respiratory compensation while from t_{+1} until t_{+15}, moderate acidosis with incomplete respiratory compensation was detected. The respiratory compensation

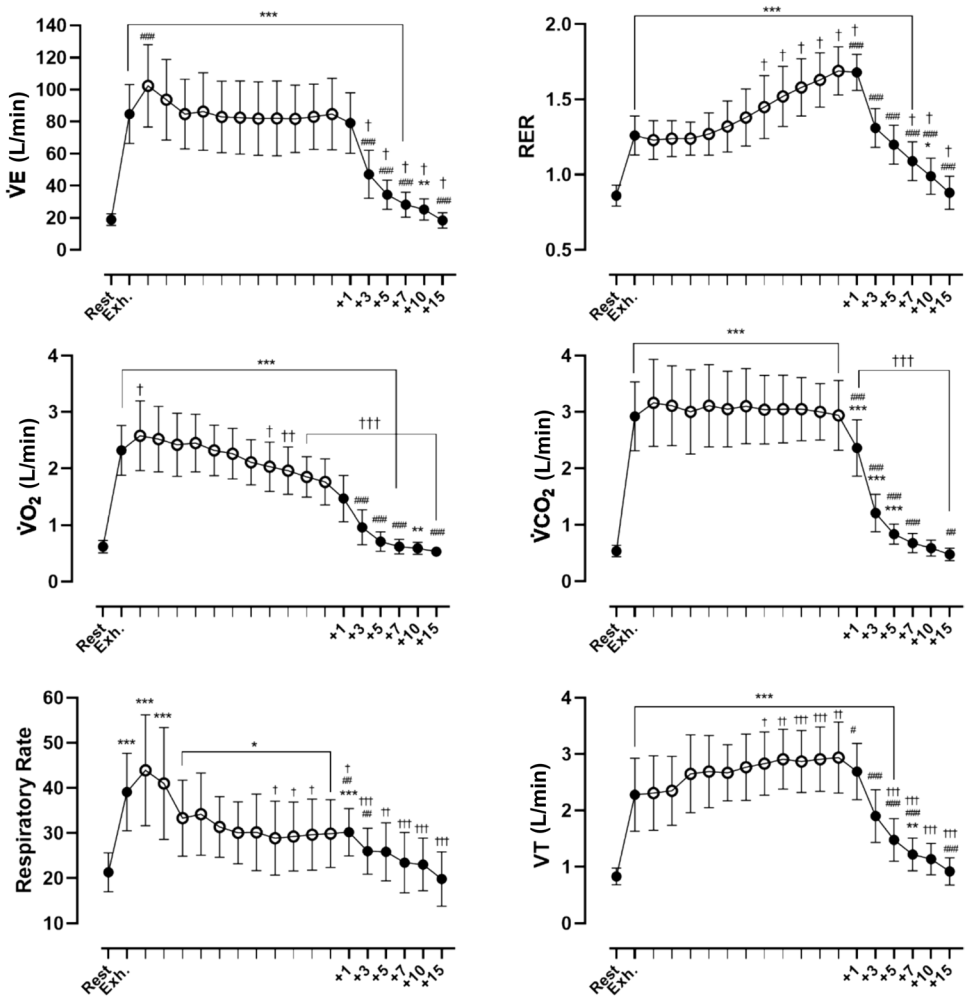

Fig. 3 Ventilation (VE), respiratory exchange ratio (RER), oxygen uptake (VO_2), carbon dioxide output (VCO_2), respiratory rate and tidal volume (VT) during and after single set leg press exercise (open circles indicate 5-s intervals between t_{Exh} and t_{+1}, $^{*}P < 0.05$, $^{**}P < 0.01$ and $^{***}P$ 0.001 indicate significant compared to resting value; $^{\dagger}P < 0.05$, $^{\dagger\dagger}P < 0.01$ and $^{\dagger\dagger\dagger}P < 0.001$ indicate significant compared to t_{Exh}; $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ and $^{\#\#\#}P < 0.001$ indicate significant compared to previous value)

as part of the buffering was increased by up to 50% in the course of recovery.

Plasma Volume Changes

There are several factors influencing the PV response during and after exercise, e.g., posture [4, 23], heat stress [43, 59], hydration [54] or the type of exercise itself [15, 49]. Most studies have investigated the PV response during or after endurance-type exercise and only a handful of studies are available for RT. Moreover, these studies have only reported percentage changes in PV based on [Hb] and Hct. There, PV demonstrated a mean decrease between 0 and 22% during and after RT, while most of the differences can be attributed to different protocols [11]. For instance, PV remained unaltered during constant load RT performed at lactate threshold intensity [16], while Collins et al. demonstrated that PV was shown to decrease linearly in relation to exercise intensity, i.e., the percentage of the one-repetition maximum, i.e. 1-RM [10]. In contrast, Craig et al. found that a 10-RM protocol yielded a greater overall change in PV compared to a 5-RM protocol [12], indicating that volume rather than intensity may influence the extent of PV changes during RT.

In this study our participants explicitly performed a single set of horizontal leg press exercise in order to eradicate the influence of inter-set rest or changes in posture on PV changes. Moreover, to the best of our knowledge, this is the first study to report absolute changes in PV during RT. In our study, mean maximum changes in PV were -559 ± 229 mL (-13.7%), which is similar to what we previously observed in men after an exhaustive cardio-pulmonary exercise test (CPX) [50]. However, these values were observed at maximum exercise and do not reflect the total extent of PV shifts in the post-exercise phase. Based on the results in this study, it is reasonable to assume that PV shifts during and after a CPX are higher compared to a single set of RT. The PV shifts are generally the result of a greater filtration rate caused by an increase in blood pressure, sweat loss and especially lactate accumulation and the breakdown of creatine phosphate within the muscle cell [12]. The latter of which causes an increased osmotic gradient that, in turn, leads to

an influx of water into the intracellular and interstitial space [7, 40]. At this point it must be stated that sweat and respiratory water losses are likely to be negligible in this study, as previous studies including several sets of RT showed no to minor changes in total body mass measured pre- and post-exercise [21, 45]. Although posture may have an effect on PV changes during RT [12], we found no changes in PV at rest, after warm-up or before the start of the single set exercise. Considering that our participants transferred from a seated into a supine position for the warm-up, we would argue that PV changes after t_{Exh} are not markedly influenced by posture effects.

Nonetheless, we observed a wide range of inter-individual changes in PV between −123 and −1091 mL (−2.9% and −23.9%). This might in part be due to the large range of TuT, which, when repetition duration is controlled, serves as a correlate for repetition number and can be used as a marker for estimating exercise intensity, i.e., the percentage of an individual's 1-RM [13]. In this study, TuT ranged between 75 and 250 s indicating a vast range of an individual's 1-RM. However, we found no correlation between the maximum percentage changes in PV and TuT ($r = 0.06$, $P = 0.78$), lowest pH ($r = 0.22$, $P = 0.27$) or highest [La^-] ($r = -0.25$, $P = 0.21$). It is also possible that the systemic values of [Hb] that we measured could also be influenced by volume changes between non-exercising tissues as a result of the change in osmotic gradient attributing to the larges differences in PV shifts [53].

As the data in Table 1 and Fig. 1 suggest, the participants in this study exercised to volitional muscular fatigue ensuring that they received a similar metabolic stimulus which in turn allows for inter-individual comparisons [14]. We would therefore conclude that the observed PV changes after a single set of leg press exercise are mainly the result of exercising to volitional muscular fatigue rather than exercise intensity.

PV Changes and Oxygen Transport

The PV shifts induced a significant increase in [Hb] by 1.2 g/dL at t_{Exh}, which was highest at t_{+3} (+1.5 g/dL, $P < 0.0001$). At the same time, ScO_2 was significantly decreased by 1.2% ($P < 0.0001$). In turn, both factors led to a significant increase in CaO_2 by 1.2 mL/dL at t_{Exh} ($P < 0.001$). This number is higher than what was previously reported during incremental cycling exercise in men [50], which is explained by the smaller decrease in ScO_2 for similar [Hb] in this study. These findings confirm our previous assumption that the exercise-induced hemoconcentration more than compensates for the drop in ScO_2 serving as a physiological adjustment to improve arterial oxygen content and possibly performance [49].

While VO_2 demonstrated a significant increase from baseline compared to t_{Exh}, evidencing partially aerobic metabolism, it seems that VO_2 during RT is impeded by frequent vasoconstriction [60]. The elevated VO_2 between t_{Exh} and t_{+1} can be explained by replenishment of O_2 stores, i.e., myoglobin, and by increased aerobic metabolism, particularly by increased glycogen replenishment [61]. In the blood, these processes are facilitated by an increase in available O_2, as mentioned earlier. Moreover, as a result of metabolic acidosis, the O_2 dissociation curve (ODC), reflected in p50, is shifted to the right, which is known as the Bohr effect [5]. However, this right shift seems to have only a small negative effect on arterial O_2 saturation due to the increased pO_2 at t_{+1} and in the further course of exercise as a result of increased ventilation. On the other hand, the rightward shift of the ODC also improves O_2 delivery within the muscle cells. This process is further optimized by the fact that due to the restricted blood flow during RT, the pCO_2 within the muscle compartment increases massively as a result of the released lactate in the temporarily closed system [5], thereby further decreasing pH and almost de-saturating capillary blood. In this way, the exercise-induced reduction in PV and the concomitant metabolic acidosis allow for a more effective muscle oxygenation during and after RT, which, in theory, should also contribute significantly to muscular recovery between multiple sets of RT.

Acid–Base Balance and Ventilation

The effect of moderate intensity RT on acid–base balance has been reported previously. For, instance, De Sousa et al. have demonstrated that pH remained unchanged during constant load resistance exercise at the lactate threshold [16]. Other studies demonstrated a progressive decline in pH during multiple sets of RT, i.e., leg press exercise [63] or following intermittent hand grip exercise [27]. In this study, we found a significant decrease in pH and a significant increase in [La^-] until 15-min post-exercise (Fig. 2). Despite exercising to exhaustion, the changes in pH were substantially less than what was previously reported during maximal effort competitive rowing [44]. Compared to incremental treadmill running in men [64], highest mean [La^-] were lower in his study, which is most likely due to a larger metabolically active muscle mass during running exercise.

According to the alignment nomogram by Siggaard Andersen [1, 2], blood acid–base status at t_{Exh} can be described as moderate metabolic acidosis (pH = 7.30) without respiratory compensation ($pCO_2 = 39.4$ mmHg). At t_{+1}, however, blood status can be described as metabolic acidosis (pH = 7.26) with incomplete respiratory compensation ($pCO_2 = 34.4$ mm Hg) and this blood status is present until t_{+15}.

In theory, acidosis can be buffered in different ways. First, via non-bicarbonate buffers, which mainly happens through Hb and is of special importance in this study since PV changes induced an increase in [Hb]. Second, pH is also regulated via respiratory mechanisms, which have not yet been fully elucidated for both endurance and RT. However, lack of data is especially evident for RT. Considering the latter, pCO_2 was first unchanged with termination of exercise but then demonstrated a significant decrease until t_{+15}. The fact that the pCO_2 was unchanged at t_{Exh} can be explained by the partially impaired ventilation during RT as the rhythm of breathing is determined by the repetitions. This would also explain why VE was highest upon termination of exercise, mainly due to an increase in VT, while respiratory rate quickly decreased (see Fig. 3). The observed maximum values of VE were similar to what was reported during a 30-s Wingate tests [20], however, they are still substantially lower than what was reported during incremental cycle exercise [50]. The hyperventilation during and after strenuous exercise is, at least in part, due to the simultaneous metabolic acidosis which results in the stimulation of peripheral chemoreceptors and so provides the extra drive to breathe [9], therefore strongly contributing to the regulation of acid–base balance. This mechanism is also seen in patients with McArdle's disease, albeit their hyperventilation is associated with an increase in pH because of no underlying metabolic acidosis [24].

For the first time, our data show the share of non-bicarbonate and bicarbonate buffering in total buffer capacity during a single bout of RT. Generally, our results are comparable to those presented by Böning et al., who calculated the total buffer capacity during incremental cycle exercise. This also applies to the temporal course of the different buffer components in the post-exercise phase [6]. However, in contrast to cycling exercise in the Böning study, we detected no respiratory compensation at t_{Exh}, which can possibly be explained by the aforementioned restriction of breathing during RT. In the further course of recovery, however, the respiratory component substantially increases (Table 2) and corresponds to the regulatory mechanisms known after endurance exercise. This culminates in a share of up to 50% of β_{tot} at the end of the recovery period. Since the non-bicarbonate buffer is also mainly based on Hb, the increase in [Hb] due to the PV shifts contributes to the metabolic buffering capacity by approx. 10%.

Limitations

We did not perform isometric or isotonic maximum strength testing to determine a standardized exercise stimulus, i.e., in terms of percentages of 1-RM. The weight was chosen by the participants themselves and solely based on their RT experience. Although it was previously demonstrated that a given number of repetitions is not always associated with the same percentages of the 1-RM [28], our protocol led to a vast range of TuT. However, the metabolic and respiratory findings we observed in this study as well as PV changes were all independent of TuT. In contrast, the significant correlations that we found between TuT and RER or $[La^-]$ at t_{Exh}, respectively, are most likely explained by a higher repetition number with higher TuT. This was also demonstrated previously, where performing a training protocol with higher repetition numbers led to increased $[La^-]$ compared to a protocol with lower repetition numbers [38]. From a practical perspective, it seems that exercising until muscular exhaustion rather than TuT itself is more important in order to induce changes in acid–base balance.

Practical Applications

Our data illustrate an insufficient respiratory compensation during a horizonal leg press exercise, but a major effect of respiration on acid–base balance during recovery. From a practical point of view, these findings may be particularly relevant for multi-set resistance training. It is assumed that metabolic acidosis accumulates during the course of multi-set training, which is linked to progressive fatigue and a performance decrease in the following sets [16, 39]. In RT, the inter-set rest usually between two and five minutes, depending on intensity and volume [18]. However, in this study, metabolic acidosis was not only present after the set, but also 15 min thereafter, which would favour such an accumulation effect. Future studies should therefore investigate the effect of exercising smaller muscle groups, where breathing is not restricted, on the metabolic state in the post-exercise phase. Lastly, controversy still exists over the general relevance of PV shifts with plasma constituent levels. It has been previously suggested that conclusions based on changes in levels of plasma constituents without evaluation of associated changes in PV might lead to erroneous interpretations [55, 62]. For this reason, it was generally recommended to consider the effect of PV changes on blood constituent levels to allow for accurate and informed intra- and inter-individual comparisons [34]. However, most studies still neglect this effect presenting uncorrected values, due to the frequently lacking clinical relevance. However, as even small volume changes and their effect on hemoconcentrations may be important under exercise [51], PV changes should always be included in the overall consideration of exercise-dependent regulatory processes. This is already known for dynamic exercise, but must also be considered for strength training.

Conclusions

In summary, following a single set of horizontal leg press exercise in healthy, trained individuals, PV is reduced by ~ 560 mL (~ 14%) hereby improving post-exercise arterial oxygen content due to hemoconcentration. RT leads to moderate metabolic acidosis, which, however, was not compensated during exercise because of restricted breathing but partly compensated during the following 15-min recovery period. The respiratory compensation as part of the buffering was increased by up to 50% of total buffering capacity in the course of recovery.

Funding Open Access funding enabled and organized by Projekt DEAL.

Data Availability The data that support the findings of this study are available from the University of Bayreuth, Germany, but restrictions apply to the availability of these data, which were used under licence for the current study and so are not publicly available. The data are, however, available from the authors upon reasonable request and with the permission of University of Bayreuth, Germany.

Declarations

Conflict of Interest W.S. is a managing partner of the company Blood tec GmbH, but he is unaware of any direct or indirect conflicts of interest with the contents of this paper. All other authors declare they have no conflicts of interest.

Ethical approval The study protocol was approved by the local ethics committee of the University of Bayreuth (Ethics-No: 23-017). All investigations were conducted in accordance with the Declaration of Helsinki on ethical principles for medical research on humans and guidelines for Good Clinical Practice.

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