

**Study Final  
Report**

**The Effects of Respiratory Muscle Training  
On Maximal and Submaximal Cardiovascular  
And Pulmonary Measurements**

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## Abstract

This study evaluated the effect of inspiratory and expiratory muscle training on pulmonary function, maximal, and sub-maximal exercise performance. Specifically, how does training using a Powerlung resistive device effect exercise performance and pulmonary function in competitive marathoners and triathletes. The participants in this study (N=12) had a mean weekly aerobic training time of 7.5 hours per week of swimming, cycling, or running. Eight subjects were assigned to a Powerlung treatment group and four control subjects were given a sham device that allowed no greater than 15% resistance on inspiration or expiration. The subjects performed 30 maximal inhalation/exhalation maneuvers on their respective devices two times per day for four weeks. The subjects were tested for forced vital capacity (FVC), forced expiratory volume in one second ( $FEV_1$ ),  $FEV_1/FVC$  ratio, forced inspiratory vital capacity (FIVC), peak inspiratory flow rate (PIFR), and peak expiratory flow rate (PEFR). Each subject was also tested for peak exhalation force test (Pex) as well as a maximal oxygen consumption ( $VO_2$  max), carbon dioxide production ( $VCO_2$ ), tidal volume ( $V_T$ ), ventilation ( $V_E$ ), lactate threshold (LT), and respiration rate (RR). The subjects also completed weekly sub-maximal exercise test at 85% of their  $VO_2$  max with exercise time,  $VO_2$ ,  $VCO_2$ ,  $V_E$ ,  $V_T$ , and RR being measured. The data revealed that training using the Powerlung device produced significant changes in maximal  $V_E$ , maximal  $V_T$ , and sub-maximal  $V_E$ . There were no significant effects on  $VO_2$  max, sub-maximal exercise time, FVC,  $FEV_1$ , FIVC, PEFR, or LT. The study revealed a 1.99 breath/minute decrease in RR coupled with a 4.93 L/min increase in  $V_E$  and a .81L/breath increase in  $V_T$  for the treatment group. Subjects in the treatment group also had a 28.25mm/Hg increase in Pex as compared to only a 2mm/Hg increase for the control group.

## Introduction

The effects of resistance training on skeletal muscle are well documented. When performed with the correct repetition scheme and load assignment, resistance training can produce skeletal muscle hypertrophy, strength, or local muscle endurance. Traditionally, strength and power athletes have used resistance training to augment sport performance while endurance athletes have avoided any type of resistance exercise believing that increased muscle would decrease aerobic performance. However, skeletal muscle controls one of the most important aspects of aerobic conditioning; air movement. The diaphragm, external and internal intercostals, scalene, and abdominal muscles (i.e. respiratory muscles) help to facilitate the increased airflow needed to fuel the increasing needs of oxygen during exercise.<sup>3</sup> If these muscles play such a crucial role in exercise, logically one would think we should train them just as if we would any other skeletal muscle.

Devices have been manufactured that offer resistance on inhalation and exhalation and thus cause an increased strain on the respiratory muscles. This resistance has been shown to facilitate positive pulmonary function changes in people with chronic obstructed pulmonary disease (COPD). Villafranca et al.<sup>17</sup> showed increases in maximal inspiratory pressure ( $PI_{max}$ ) after ten weeks of inspiratory muscle training using a threshold inspiratory trainer allowing 30% resistance. Likewise, Larson and Kim<sup>7</sup> observed increases in  $PI_{max}$  after one month of inspiratory muscle training in people with COPD.

Pulmonary resistance training has also been shown to change enzymatic profiles in sheep. Akabas et al.<sup>1</sup> assigned nine adult sheep to a training group and seven to a control group. The experimental group trained for twenty minutes using inspiratory flow resistance (50-100 cmH<sub>2</sub>O) five to six times per week for three weeks. In the end, biochemical changes were observed between the experimental and control group. There was a 26 percent increase in citrate synthase (CS), a 29 percent increase in hydroxyacyl-CoA dehydrogenase (HAD), and a 36 percent increase in cytochrome oxidase also (COX) in the experimental group as compared to the control group in the diaphragm muscles. It can be concluded from this study that the aerobic enzymatic profiles increased significantly in sheep when put under inspiratory stress. An increase in aerobic enzymes during exercise in humans would equate to more efficient energy utilization of the respiratory muscles and lower fatigueability.

Data involving healthy humans using respiratory muscle training, and specifically with exercise is limited. In two separate studies, Suzuki et al.<sup>12,13</sup> observed changes in the rate of perceived exertion (RPE) after inspiratory and expiratory muscle training in healthy adults. Suzuki concluded that expiratory muscle training did not decrease RPE at a given work load while inspiratory muscle training did decrease RPE.

Cain and McConnell<sup>10</sup> reported increased sub maximal exercise performance after respiratory muscle training. This group found decreases in blood lactate, RPE, and HR along with an increased time to fatigue after four weeks of respiratory training. Boutellier et al.<sup>3</sup> reported similar results also after four weeks of respiratory muscle training. Hanel and Secher<sup>6</sup> observed a decrease in breaths per minute and an increase in tidal volume after exercising twice a day for 28 days in ten physical education students.

The purpose of our research was to investigate the effects of respiratory muscle training using a Powerlung device on pulmonary function and exercise performance in triathletes and marathon runners. We hypothesize that the treatment group will increase pulmonary function during exercise and increase dynamic lung functions. Furthermore, we believe that the exercising group will increase sub-maximal exercise time as compared to the control group. We do not believe that the treatment group or control group will increase  $\text{VO}_2$  max or change lactate threshold.

## **Methods**

### **Subjects:**

A total of twelve subjects (9 male, 3 female) were recruited from area triathlon and running clubs. The subjects had a mean aerobic training time of 7.5 hours per week (range; 5 – 10 hours). Eight subjects (6 male; 2 female) were assigned to a Powerlung treatment group and four subjects (3 male; 1 female) were assigned to a control group. The experimental group had the following mean physical characteristics: age  $36.75 \pm 8.83$  years, height  $174.5 \text{ cm} \pm 9.41$ , weight  $72.94 \text{ kg} \pm 7.22$ . The control group's physical characteristics were age  $34.5 \pm 7.05$  years, height  $172.8 \pm 4.22$  cm, and weight  $71.23 \pm 3.64$  kg. Each subject was given an informed consent form and made aware of their right to drop out of the study at any time.

### **Maximal Exercise Testing:**

Each subject was given a maximal oxygen consumption test ( $\text{VO}_2$  max) before and after Powerlung training. The  $\text{VO}_2$  max protocol consisted of 1 ½ minute stages of work, each made successively more difficult by increasing the grade by 1%. The testing speed was constant, and was selected based on the 5K time of the subjects. The protocol had a 2-stage ramp-up to the target speed to ease the transition to the test speed. In addition to the 2-stage ramp-up built into the protocol, the subject was allowed to warm-up on the treadmill as long as needed before beginning the test. The protocol continued to increase by 1% in incline until the subject reached max exercise (i.e. asked for the treadmill to be stopped or grabbed the handrail). Metabolic data was acquired using a Quinton metabolic cart (QMC). The QMC sampled from a mixing chamber every 15 seconds and provided oxygen consumption ( $\text{VO}_2$ ), ventilation ( $V_E$ ), respiratory rate (RR), volume of carbon dioxide produced ( $V\text{CO}_2$ ), and lactate threshold (LT). Lactate threshold was derived via the v-slope method by the Quinton metabolic software version 3.3.<sup>8</sup> Heart rate (HR) was measured using a five lead electrocardiogram from a Quinton Q-4500 stress-test system.

### **Pulmonary Function Testing:**

Each subject was given a spirometry test using a ChestTest spirometer (Vaccumed, Ventura, CA) before the  $\text{VO}_2$  max test both pre and post training. Each subject stood facing the spirometer and performed a maximal inhalation followed by a forceful exhalation into the tube until all air was expelled. The subject then performed a maximal inhalation to complete the maneuver. The ChestTest spirometer provided

forced vital capacity (FVC), forced expiratory volume in one second (FEV<sub>1</sub>), FEV<sub>1</sub>/FVC ratio, Peak expiratory flow rate (PEFR), and peak inspiratory flow rate (PIFR).

Each subject also performed a peak exhalation force (Pex) test to determine the strength of the expiratory muscles. Force was measured using a standing mercury sphygmomanometer. The subject performed a maximal inhalation then exhaled forcefully against a closed valve. Maximal force in mmHg was recorded over a maximum of three trials. The Pex test was performed pre and post training after the spirometry test but before the VO<sub>2</sub> max test. Test retest reliability was performed on the apparatus using 8 graduate students ( $r = .92$ ).

### **Sub-maximal Exercise Testing:**

One to two days after the pre VO<sub>2</sub> max test and pulmonary testing, each subject returned for a sub-maximal treadmill test. Specifically, each subject ran on the treadmill using the same protocol as performed in their maximal testing. A hold was placed on the protocol in the stage in which the subject's steady-state VO<sub>2</sub> = 85% ± 5%. The subject continued at this workload until failure or their VO<sub>2</sub> exceeded 90% of maximum. The QMC was used to measure the same metabolic data as measured during the maximal test. Each subject performed a sub-maximal treadmill test weekly for a total of four weeks (e.g. five total sub-maximal tests).

### **Training:**

For the duration of the study, the subjects were asked to maintain their present aerobic training and not to increase or decrease their training time in order to eliminate the possibility of adaptations from aerobic exercise.<sup>11</sup> Both groups performed thirty maximal inhalation/exhalation cycles on the Powerlung training device two times per day for four weeks. The device allows for varying resistance on inhalation and exhalation via hand adjusted knobs. After the first sub-maximal test, the treatment group was given the Powerlung device and instructed to perform the above-mentioned protocol as assigned by Powerlung manufacturers. The subjects were instructed to adjust the resistance to allow for the 30<sup>th</sup> breath on both inhalation and exhalation to be close to a maximal effort (i.e. 30-RM). The subjects controlled the resistance via trial and error.

In the same way, the subjects assigned to the control group were given a sham device after their first sub-maximal test. The sham device looked identical to the Powerlung device, but the chambers inside the sham device were constructed to allow a resistance no greater than 15%. The control subjects were given the same instructions as the experimental group. Neither group was informed as to which group they were assigned to until the completion of their last maximal test.

## **Results**

Summaries of the results are provided in Tables 1 and 2, as well as graphical representations of the data in Appendix 1. Table 1 is a summary of all maximal data including metabolic data derived from VO<sub>2</sub> max tests as well as pre and post pulmonary evaluations. Table 2 is a summary of the sub-maximal data across groups.

### Maximal Variables:

As expected, there was no change in  $\text{VO}_2$  max across either the treatment ( $.51 \pm 7.63$  ml/kg/min) or control groups ( $-.92 \pm 3.95$  ml/kg/min). There was also no significant difference seen in LT pre vs. post with the treatment group shifting down 2.63% of  $\text{VO}_2$  max and the control group's lactate threshold shifting up 1.75% of  $\text{VO}_2$  max.

The treatment group saw a statistically significant 4.55L increase (3.29%) in  $V_E$  max while the control group saw a 5.75L decrease (-4.58%) in  $V_E$  max. There was also a significant increase in the treatment group's  $V_T$  (.81 L/breath; 26.7%) compared to the decreased  $V_T$  (-.25 L/breath; -7.58%). Although not statistically significant, the treatment group saw a decrease in RR of 1.88 breaths/min (-3.59%) at maximal exercise. The control group saw only a .75 breath/min (.53%) increase in RR after four weeks of Powerlung sham training.

After four weeks of training neither the treatment nor control group exhibited any changes in FVC (trt=.04 L, cnt= .2 L),  $\text{FEV}_1$  (trt = .01 L, cnt = .03 L),  $\text{FEV}_1/\text{FVC}$  ratio (trt = - .1, cnt = + .1), PEFR (trt = -.3 L/sec, cnt = .8 L/sec), or peak inspiratory flow rate (trt = - .1 L/sec, cnt = - .13 L/sec). However, a large (although not statistically significant) increase was observed in Pex in the treatment group ( 28.25 mm/Hg; 23.35%) while the control group showed only a small increase in Pex (2 mm/Hg; 1.75%).

### Submaximal Variables:

The subject's weight remained constant throughout the training with the treatment group changing on average -.41 kg and the control group -.05 kg. Both groups increased sub-maximal exercise time significantly (trt = 2.72 min, cnt = 2.38 min) from pre to post training. The treatment group saw its largest increase from week three to post training (1.63 min) while the control group saw a large increase from week one to week two (2 min). Because the changes were similar in both the treatment and control group, the increases could possibly be attributed to the subjects becoming more comfortable with the testing procedure and not to any training induced changes by the Powerlung device.

As expected sub-maximal  $\text{VO}_2$  did not change from pre to post. The treatment group increased  $\text{VO}_2$  on average by 1 ml/kg/min while the control group decreased by .67 ml/kg/min.  $\text{CO}_2$  production also remained constant with a .02 L/min increase in the treatment group and a .7 L/min decrease in the control group. A larger decrease in heart rate at 85% of max  $\text{VO}_2$  was seen in the control group (- 3.76 bpm) from pre to post.

The variables that saw the greatest changes from Powerlung training were the respiration values during sub-maximal exercise. The treatment group saw a 4.31 L/min (4.33%) increase in  $V_E$  at the 85% workload while the control group saw a 1.92 L/min (-1.93%) decrease. At the same time, the treatment group decreased its RR by 1.99 breaths per minute (-4.76%) while the control group decreased its RR by only .49 breaths per minute (-1.31%).  $V_T$  was also increased across both the training (.27 L/breath, 9.82%) and the control (.3 L/breath, 1.06%) groups.

**Table 1: Descriptive Statistics for Maximal Test Variables**

Variable	Trt Mean	Trt SdErr	Trt Sd	Cnt Mean	Cnt SdErr	Cnt Sd
N	8			4		
Height (cm)	174.50	3.33	9.41	172.80	2.11	4.22
Age (y)	36.75	3.12	8.83	34.50	3.52	7.05
Gender	6 m; 2 fe			3 m; 1 fe		
PreO <sub>2</sub> max (ml/kg/min)	55.35	2.70	7.63	53.45	3.95	7.89
PstO <sub>2</sub> max (ml/kg/min)	55.86	2.54	7.19	52.53	3.85	7.71
PreV <sub>E</sub> max (L/min)	138.16	7.37	20.85	125.60	8.69	17.38
PstV <sub>E</sub> max (L/min)	142.71	8.29	23.43	119.85	9.69	19.37
PreV <sub>T</sub> max (L/breath)	3.03	.16	.45	3.30	.44	.88
PstV <sub>T</sub> max (L/breath)	3.84	.26	.73	3.05	.45	.70
PreRRmax (breath/min)	52.38	2.40	6.80	47.00	5.48	10.95
PstRRmax (breath/min)	50.50	1.76	4.99	47.25	5.84	11.67
Pre LT (% VO <sub>2</sub> max)	77.13	1.38	3.91	73.25	5.65	11.30
Pst LT (% VO <sub>2</sub> max)	74.50	1.54	4.34	75.00	2.35	4.69
Pre FVC (L)	4.98	.30	.85	4.69	.34	.67
Pst FVC (L)	5.02	.27	.77	4.67	.39	.79
Pre FEV <sub>1</sub> (L)	3.77	.16	.46	3.68	.28	.55
Pst FEV <sub>1</sub> (L)	3.77	.15	.42	3.71	.30	.61
Pre Pex (mmHg)	121.00	7.78	22.01	143.00	29.03	58.07
Pst Pex (mmHg)	149.25	9.97	28.20	145.50	25.33	50.66
Pre Ratio	.77	.003	.008	.79	.002	.004
Pst Ratio	.76	.003	.008	.80	.003	.006
PrePEFR (L/sec)	9.38	.56	1.60	7.95	.48	.95
PstPEFR (L/sec)	9.35	.53	1.51	7.87	.50	1.00
PreFIVC (L)	4.52	.30	.85	4.44	.30	.59
PstFIVC (L)	4.51	.23	.65	4.31	.38	.76

\* Trt = Treatment Group

\* Cnt = Control Group

**Table 2: Descriptive Statistics for the Weekly Sub maximal Tests**

Variable	Trt Mean	Trt SdErr	Trt Sd	Cnt Mean	Cnt SdErr	Cnt Sd
Pre Weight (kg)	72.94	2.55	7.22	71.23	1.82	3.64
Wk1 Weight (kg)	72.48	2.44	6.91	71.18	1.77	3.55
Wk2 Weight (kg)	72.86	2.41	6.82	71.13	1.78	3.57
Wk3 Weight (kg)	73.29	2.47	6.99	71.28	1.74	3.48
Post Weight (kg)	72.53	2.56	7.23	71.18	1.81	3.62
Pre Time (min)	8.44	1.77	5.01	8.31	1.37	2.75
Wk1 Time (min)	9.97	1.82	5.15	7.94	1.07	2.13
Wk2 Time (min)	9.59	1.86	5.25	9.94	1.91	3.82
Wk3 Time (min)	9.53	1.65	4.67	10.06	1.76	3.51
Post Time (min)	11.16	2.71	7.67	10.69	2.20	4.40
Pre VO <sub>2</sub> (ml/kg/min)	49.95	2.09	5.92	47.17	2.21	4.41
Wk1 VO <sub>2</sub> (ml/kg/min)	50.43	1.96	5.54	46.99	2.14	4.27
Wk2 VO <sub>2</sub> (ml/kg/min)	50.17	1.90	5.38	46.49	2.04	4.09
Wk3 VO <sub>2</sub> (ml/kg/min)	50.23	1.92	5.43	46.65	2.16	4.33
Post VO <sub>2</sub> (ml/kg/min)	50.95	1.90	5.37	46.50	2.42	4.84
Pre V <sub>E</sub> (L/min)	113.76	4.21	11.90	99.31	5.64	11.27
Wk1 V <sub>E</sub> (L/min)	116.85	4.33	12.23	101.34	6.05	12.10
Wk2 V <sub>E</sub> (L/min)	115.53	3.98	11.25	102.75	6.75	13.50
Wk3 V <sub>E</sub> (L/min)	118.09	5.09	14.41	97.54	4.30	8.60
Post V <sub>E</sub> (L/min)	118.69	5.47	15.46	97.39	5.09	10.18
Pre RR (breaths/min)	41.82	1.30	3.67	37.38	5.22	10.44
Wk1 RR (breaths/min)	43.39	1.53	4.33	37.98	5.57	11.14
Wk2 RR (breaths/min)	43.66	1.75	4.94	39.26	5.20	10.39
Wk3 RR (breaths/min)	43.16	1.54	4.36	36.16	4.52	9.04
Post RR (breaths/min)	39.83	1.57	4.43	36.89	4.99	9.99
Pre V <sub>T</sub> (L/breath)	2.75	.13	.37	2.82	.39	.79
Wk1 V <sub>T</sub> (L/breath)	2.68	.14	.39	2.87	.48	.95
Wk2 V <sub>T</sub> (L/breath)	2.76	.11	.31	2.74	.37	.74
Wk3 V <sub>T</sub> (L/breath)	2.71	.14	.41	2.84	.39	.78
Post V <sub>T</sub> (L/breath)	3.02	.20	.57	2.85	.39	.78
Pre VCO <sub>2</sub> (L/min)	4.09	.19	.53	3.73	.16	.32
Wk1 VCO <sub>2</sub> (L/min)	4.06	.19	.54	3.78	.16	.32
Wk2 VCO <sub>2</sub> (L/min)	4.08	.19	.53	3.74	.16	.31
Wk3 VCO <sub>2</sub> (L/min)	4.08	.22	.62	3.60	.11	.22
Post VCO <sub>2</sub> (L/min)	4.08	.18	.51	3.66	.08	.18
Pre HR (bpm)	171.56	3.08	8.70	175.57	4.70	9.40
Wk1 HR (bpm)	171.91	3.20	9.04	173.85	4.80	9.59
Wk2 HR (bpm)	170.48	3.08	8.72	172.06	4.49	8.99
Wk3 HR (bpm)	172.78	3.78	10.69	173.03	4.78	9.56
Post HR (bpm)	172.91	4.27	12.06	171.81	4.52	9.05

\* Trt = Treatment Group

\* Cnt = Sham group

## Discussion

### **Maximal Variables:**

The purpose of this study was to determine if pulmonary function or exercise performance could be changed by specifically training the respiratory muscles using a Powerlung resistance device. Respiration is only one component of aerobic capacity. Cardiac output is generally thought to be the major limiting factor to aerobic exercise.<sup>7</sup> At maximal exercise, the ventilation to perfusion ratio reaches 4.0, indicating that we are moving four times the amount of air than is being transferred across the walls of the alveoli<sup>7</sup>. Research shows runners with the lower ventilation responses to high exercise are both hypoxicemic and more acidotic.<sup>4</sup> This could be attributed to the increased percent of lactic acid being produced by the respiratory muscles that are hyperventilating in an attempt to buffer H<sup>+</sup>.

In a study by Boutellier et al.<sup>3</sup> on trained aerobic athletes using a respiratory resistance device different from the Powerlung, they found no significant increases in either VO<sub>2</sub> max or LT during a cycle ergometer test. However, they did see an increase of sub-maximal cycling time. Like Boutellier's group, we did not see an increase in VO<sub>2</sub> max or LT.

We did expect to see changes in pulmonary function during exercise. Because the subjects had trained their pulmonary muscles, they were able to move more air. The increase in V<sub>E</sub> and decrease in RR in the training group indicates that the Powerlung device appears to be increasing the strength of the respiratory muscles. The increased strength of the respiratory muscles allowed the subjects to perform more work (i.e. move more air) while breathing fewer times.

Earlier work by Hanel and Secher<sup>6</sup> shows similar results. The investigators studied inspiratory muscle training on 20 physical education students. The students trained using a device similar to the Powerlung, but only allowed resistance on inspiration. The students trained on the device for 10 minutes twice per day at a progressively increasing resistance for 27.5 days. VO<sub>2</sub> max was measured via treadmill test pre and post training and revealed a 3 breath per minute decrease at max exercise in the training group and no change in the control group. A small increase in VO<sub>2</sub> (~2 L/min) was observed in both the training and the control group from pre to post testing. Likewise, our study revealed changes in RR with a 1.88 breath/minute decrease at max exercise. However, Hanel and Secher saw a 2L/min decrease in the treatment group after 27.5 days of inspiratory muscle training where as we saw a 4.53 L/min increase in V<sub>E</sub> post Powerlung training.

### **Spirometry Pre/Post:**

The spirometry readings did not change significantly with the Powerlung training. O'Kroy and Coast<sup>14</sup> examined nineteen untrained students and randomly assigned them to either a control group, an exercising group, an inspiratory loading group, an expiratory loading group, or a hyperventilating group. The subjects trained four days per week, 20 minutes a day for four weeks with their assigned groups. The subjects training with inspiratory loading showed increases in maximum inspiratory pressure (MIP) and inspiratory force (IF), while the expiratory loading group showed increases in maximum expiratory pressure (MEP) and IF. The only other group showing significant changes was

the hyperventilating group, which exhibited increased MEP, maximum voluntary ventilation (MVV), maximal sustained ventilation for 4 minutes (MSVV), and IF. This research suggests resistance training offers some benefit to fatigue resistance in untrained students. The research also suggests that inspiratory muscles, like all other skeletal muscles, adapt according to the stress placed on them (i.e. SAID principle).<sup>2</sup>

Tzelpis et al.<sup>15, 16</sup> showed similar results in a study with nineteen untrained students using flow specific training. Tzelpis assigned the students to three specific groups: a low, medium, and high pressure groups. The high pressure group performed 30 maximal static inspiratory contractions against a occluded valve. The low pressure group performed 30 sets of three maximal inspiratory contractions against an unoccluded valve, and the medium pressure group performed 30 maximal contractions through a 7mm resistor tube. The results were specific to the type of training. The high pressure training groups had the highest increases in peak esophageal pressure ( $P_{es_{max}}$ ) and maximum inspiratory flow rate ( $VI_{max}$ ). Tzelpis et al. concluded that the groups that trained using high pressure resistance had the greatest increases in  $P_{es_{max}}$  while the groups training with higher flow rates (i.e. low pressure group) had higher increases in  $VI_{max}$  as compared to the other two higher pressure groups.

It is known that pulmonary muscles will adapt to aerobic exercise<sup>11</sup>. So, we would expect to see smaller changes in aerobic athletes as their pulmonary muscles are already developed. Hanel and Sechler<sup>6</sup> showed no change in FEV<sub>1</sub>, FVC, FEV<sub>1</sub>/FVC, and peak expiratory flow rate after 27.5 days of training. In the same way, Boutellier et al.<sup>3</sup> did not find changes in either peak expiratory flow rate or FEV<sub>1</sub>.

Our study produced results similar to those of Hanel and Sechler<sup>6</sup> and Boutellier et al.<sup>3</sup> The changes we saw were specific to the training that is induced by the Powerlung device. When blowing against the pressure gage, as in blowing against the Powerlung, pressure is being exerted back against you. For this reason we saw a significant increase in P<sub>ex</sub> in the training group. When blowing into the spirometer there is no resistance and it is different than the training of Powerlung. For this reason, spirometry readings were not altered pre to post training.

### **Sub maximal Exercise:**

The most important aspect of our research was the effect of the powerlung device on sub maximal exercise performance. The goal of any aerobic athlete is to increase the amount of time they can train or compete at a relatively high workload. It can be deduced that if the respiratory muscles were trained, these muscles would consume less oxygen to do the same amount of work. Fixler et al.<sup>5</sup> reports a significant amount of cardiac output being shared by the diaphragm during high workloads. Because the respiratory muscles have this increased workload, an increased amount of oxygen must be distributed to these muscles. If the respiratory muscles were trained, less oxygen would be distributed via this pathway, allowing for a larger pool of oxygen to be used by other skeletal muscles, and an increased exercise time.

Boutellier et al.<sup>3</sup> showed a 38% increase in exercise time at lactate threshold on a cycle ergometer in aerobically trained athletes after 27.5 days of inspiratory training. This type of increase could create a significant advantage for aerobic athletes. However, Boutellier et al. did not use a control group and it is conceivable that anyone, regardless of inspiratory training, might be able to increase time on a cycle ergometer just by

learning the test. In our study, the subjects were working, on average, 10% above lactate threshold. At this high workload, lactic acid is accumulating exponentially. As lactic acid accumulates, the time to max exercise also decreases proportional to the decrease in pH. At 85% of  $\text{VO}_2$  max there is a very short time until pH levels rise to the point at which the subject cannot continue to exercise. Boutellier et al.<sup>3</sup> trained at lactate threshold, which allowed his subjects to go for a longer period of time. Because the time to max exercise is directly proportional to the buildup of lactic acid, a smaller increase in exercise time at a higher workload could be comparable to a larger increase at a lower workload.

The work of Hanel and Sechler<sup>6</sup> contradicts Boutellier as they found no change in twelve minute walk distance after four weeks of inspiratory muscle training in physical education students. The test that Hanel and Sechler<sup>6</sup> utilized was not as controlled as that of Boutellier et al.<sup>3</sup>, but they did mention that environmental conditions were similar in both the pre and post testing days. Larson and Kim<sup>8</sup> also showed no change in twelve minute walk distance in subjects with COPD.

Because we used athletes who were highly aerobically trained, we did not expect to see many changes in the metabolic variables during sub maximal exercise. We did expect ventilation to change. In order to minimize testing validity errors, our tests were performed in a laboratory on a treadmill at a given speed. Atmospheric conditions within the lab were fairly constant from test to test. Each subject tested at  $85 \pm 5\%$  of their  $\text{VO}_2$  max and ran until exhaustion. The speed that the subjects performed the treadmill test at was the speed at which they run their 5K races. Therefore the speed was a comfortable (but fast) running speed for each subject. The possibility of error still exists due to subject motivation between trials.

Our test did not elicit a difference between the treatment and control group in sub-maximal exercise time. Because the changes we saw were similar in both the treatment and control group (i.e., both increased), the changes could be attributed to the subjects becoming more comfortable with the testing procedure and not to any training induced changes by the Powerlung device. Oxygen consumption did not change significantly across trials. The lack of changes can probably be attributed to the high level of fitness of the athletes.

The areas we saw the most changes from the Powerlung were in ventilation and respiration values during exercise. The decrease in RR with an increase in  $V_T$  and  $V_E$  can be attributed to the effectiveness of the Powerlung device. Because the treatment subjects developed stronger respiratory muscles they were able to move more air with fewer breaths. The respiratory muscles are becoming more efficient at doing their work. Perhaps this would have made more of a difference in sub-maximal exercise performance over a longer period of time. Dempsey et al.<sup>4</sup> cites an increasing hyperventilation pattern with long term exercise at lower workloads and a disproportionate tidal volume as compared to  $\text{H}^+$  build up. In our study, the subjects were working above lactate threshold and the pH is decreasing so rapidly that a change of  $\text{VO}_2$  distribution away from the pulmonary muscles was really not evident. The changes we saw in HR can probably be attributed to subject variation and not to training.

### **Conclusion and Future Research Considerations:**

It can be concluded from our research that the Powerlung device does what the manufactures say it does. The Powerlung device increased the strength of the respiratory muscles as seen by the increase in  $P_{ex}$ ,  $V_E$  and  $V_T$  while decreasing RR at sub maximal workloads. The device did not produce a significant change across groups in sub-maximal exercise time. It could be deduced that a longer training period (i.e., 8-12 weeks) might reveal larger increases in sub-maximal exercise time. Research with a less trained population might also produce larger changes.

It would be good to look at what effect the decreased RR would have at a lower intensity. If the subjects were running at 75% of their  $VO_2$  max, would the decreased workload of the respiratory muscles be more effective in increasing sub-maximal exercise time?

When doing further research with highly trained athletes, one might also consider trying different training protocols. In all forms of resistance training, not only is the intensity increased, but the overall volume and duration is also varied. Perhaps training at a higher volume (i.e. more reps) might produce more favorable metabolic changes in the respiratory muscles. The powerlung device might also produce favorable changes with other types of athletes, such as swimmers. Because it is necessary for swimmers to inhale a large amount of air in a short period of time, it is important for these athletes to have powerful respiratory muscles.

Future research might also look at the Powerlung's effect on sprint recovery time. After short bursts of high intensity exercise (i.e. sprinting) the body hyperventilates in an attempt to recover lost energy stores. A better trained respiratory system might facilitate this recovery.

### **Appendix 1:**

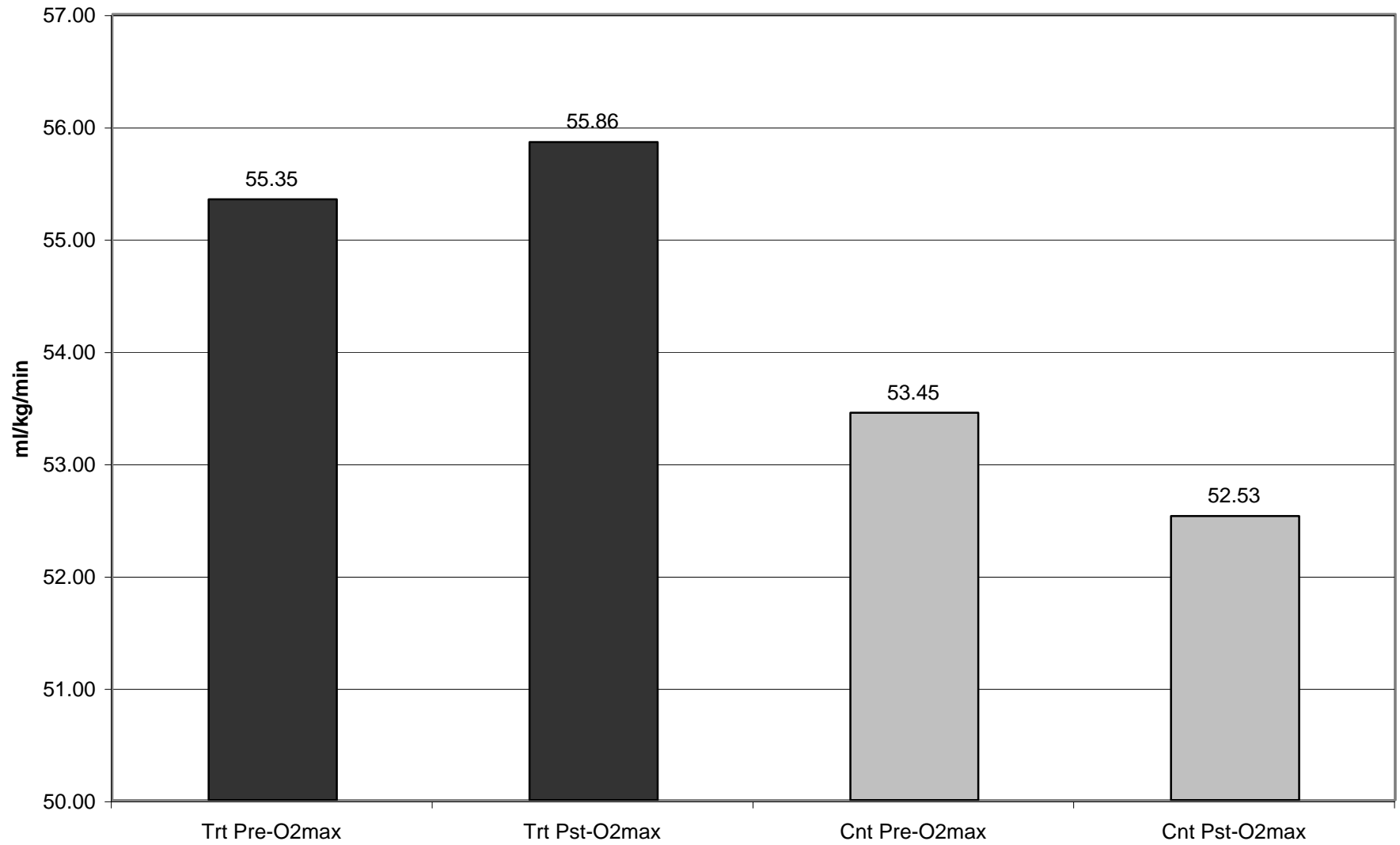
Appendix 1 contains a graphical representation of the changes that occurred across groups in the study.

## **Bibliography**

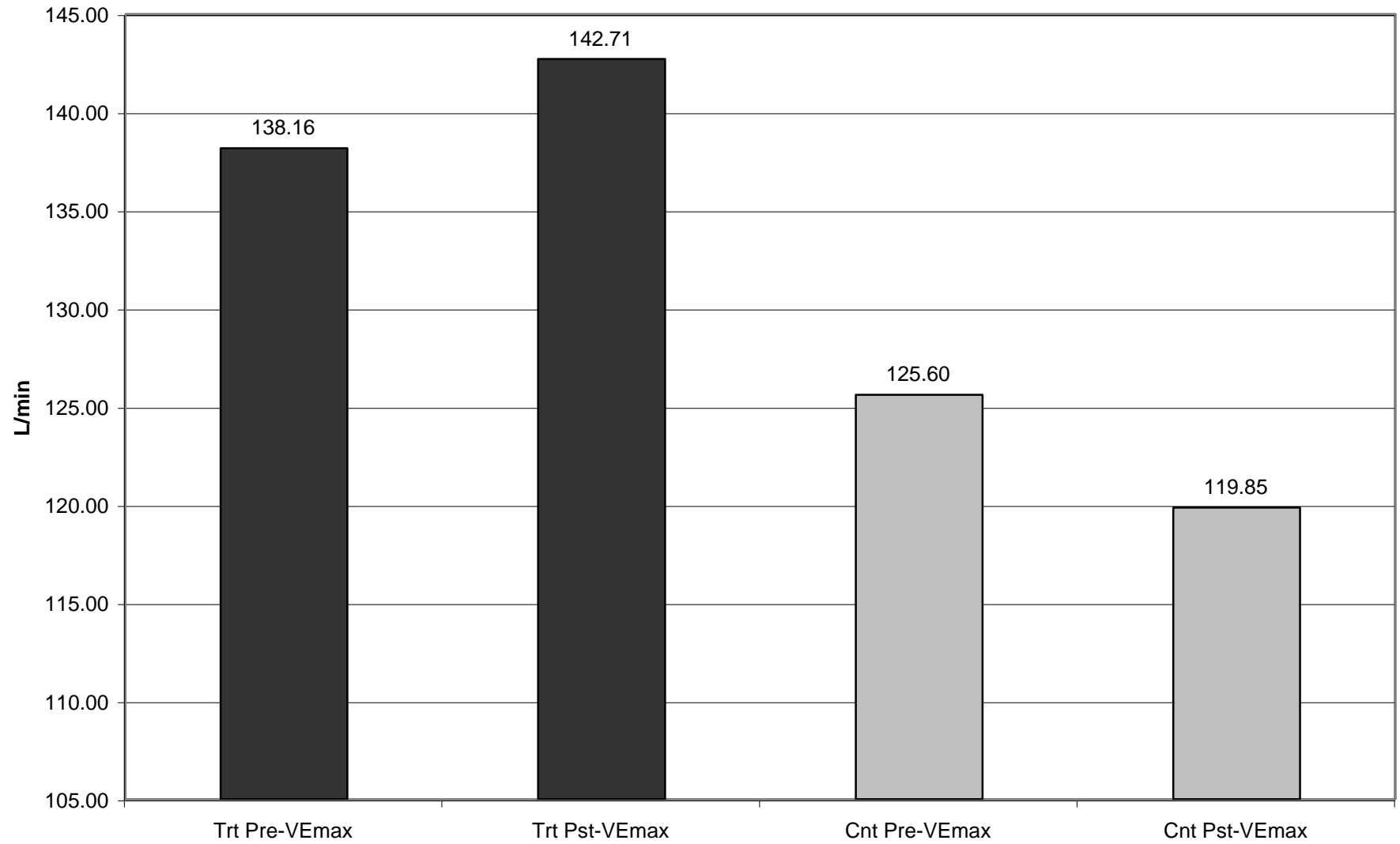
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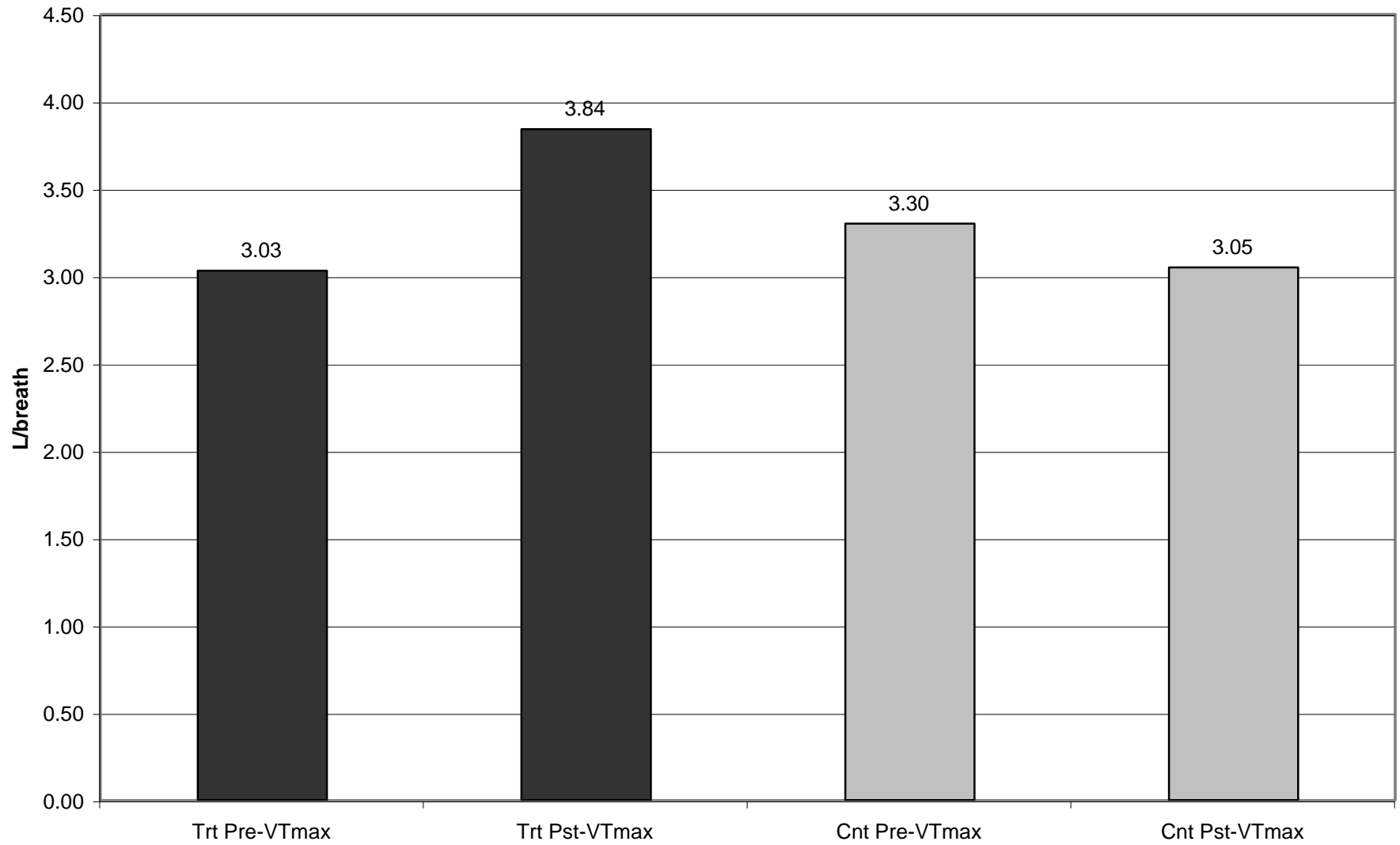
### Maximal Oxygen Consumption



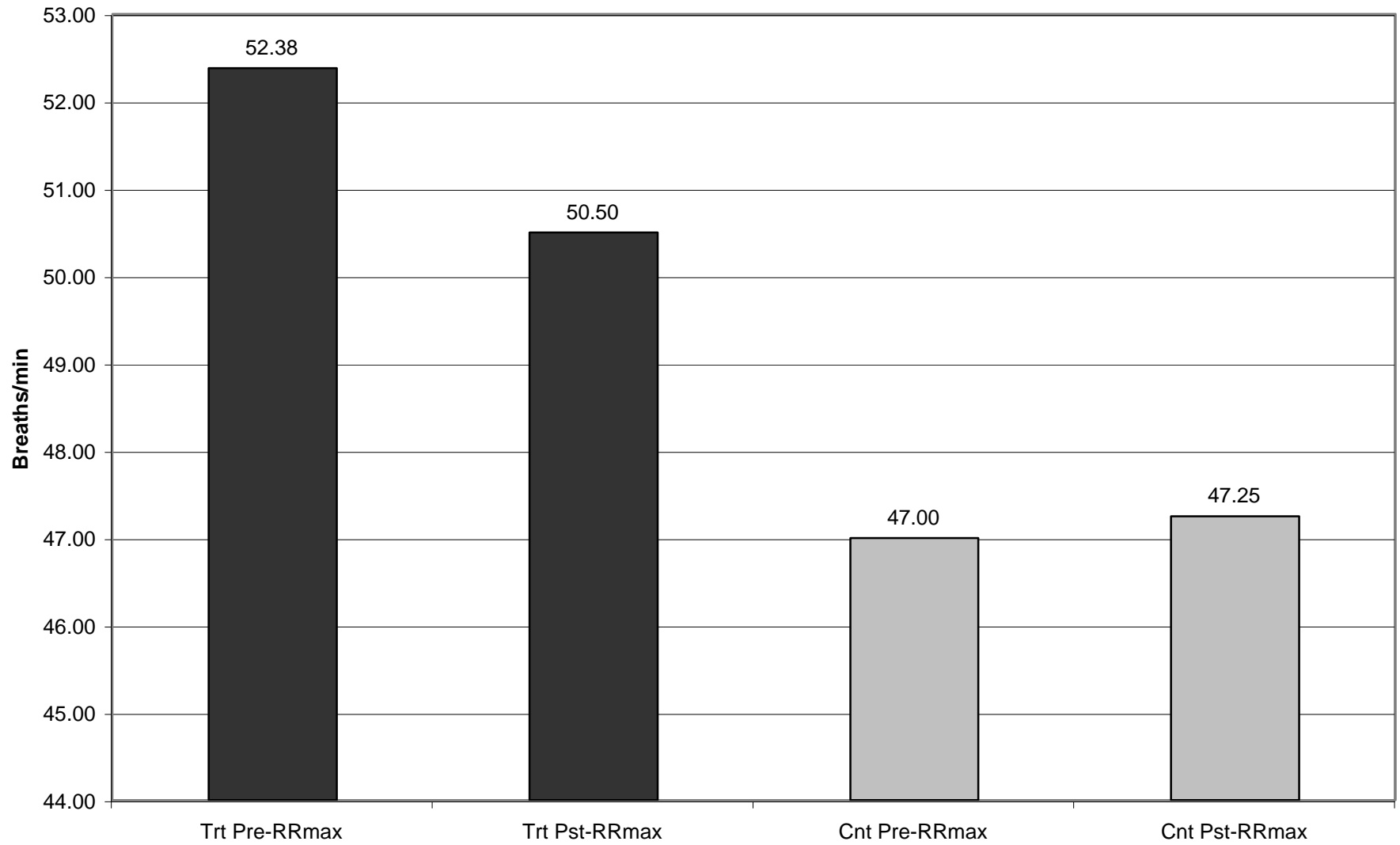
### Maximal Minute Ventilation



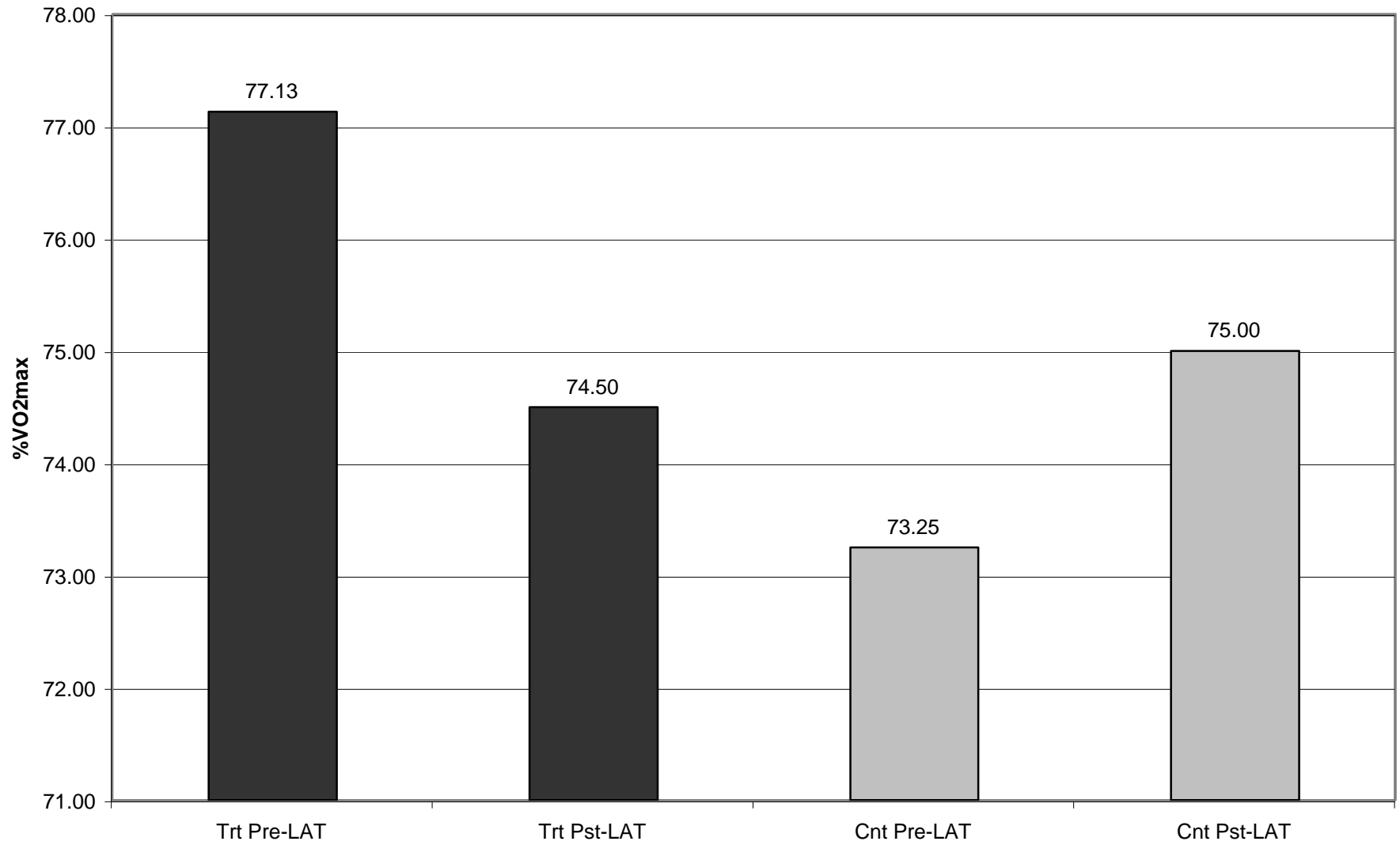
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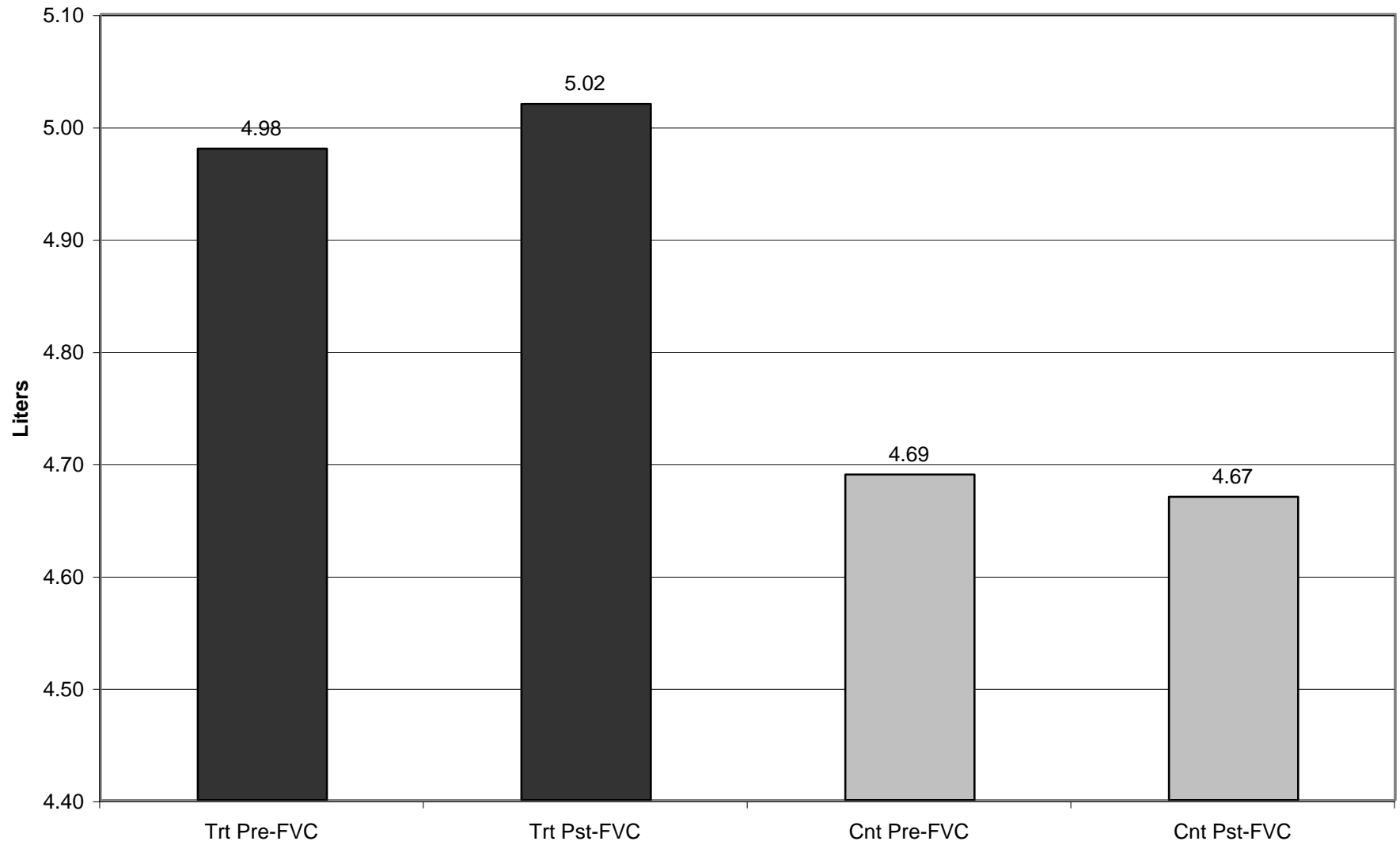
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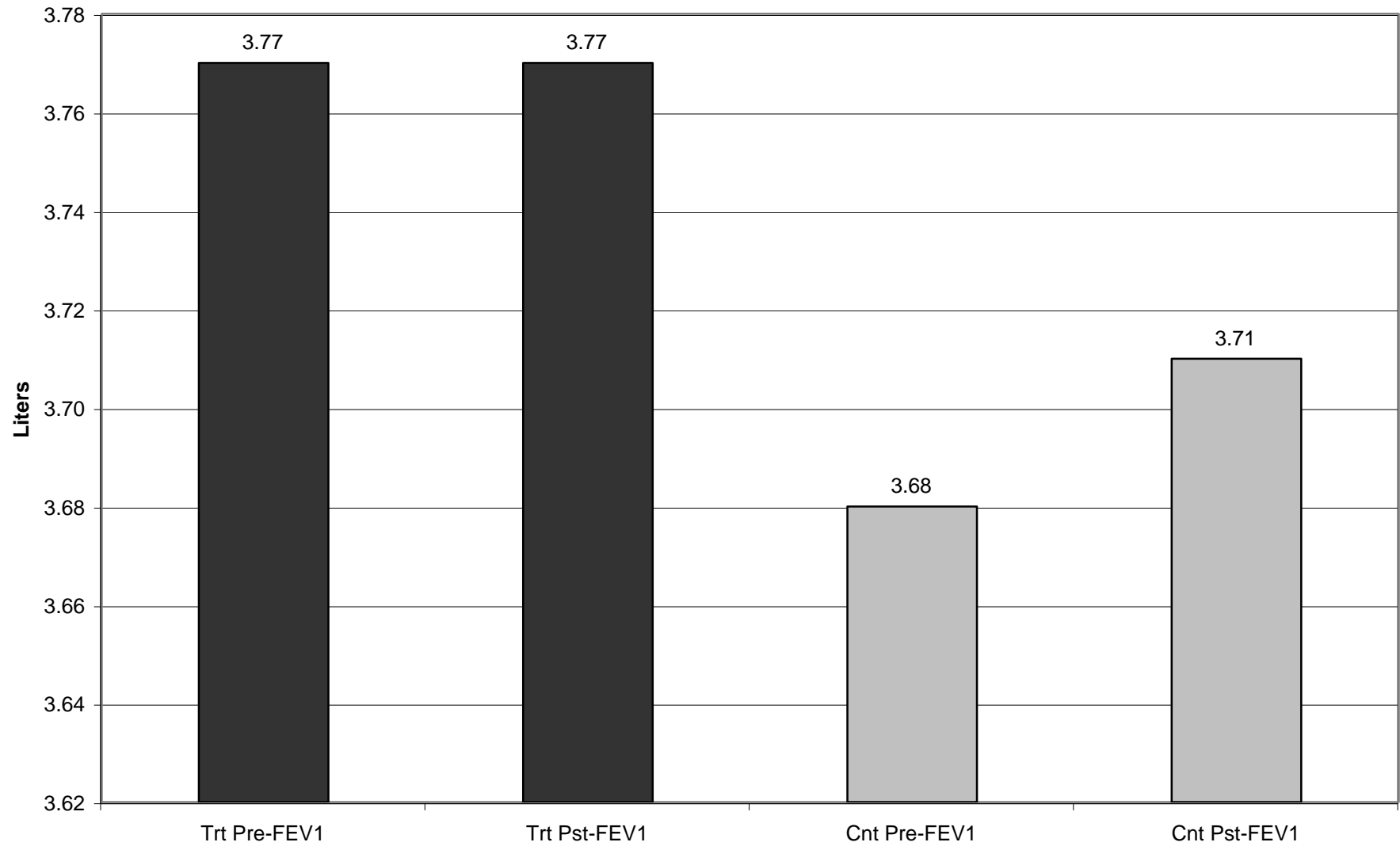
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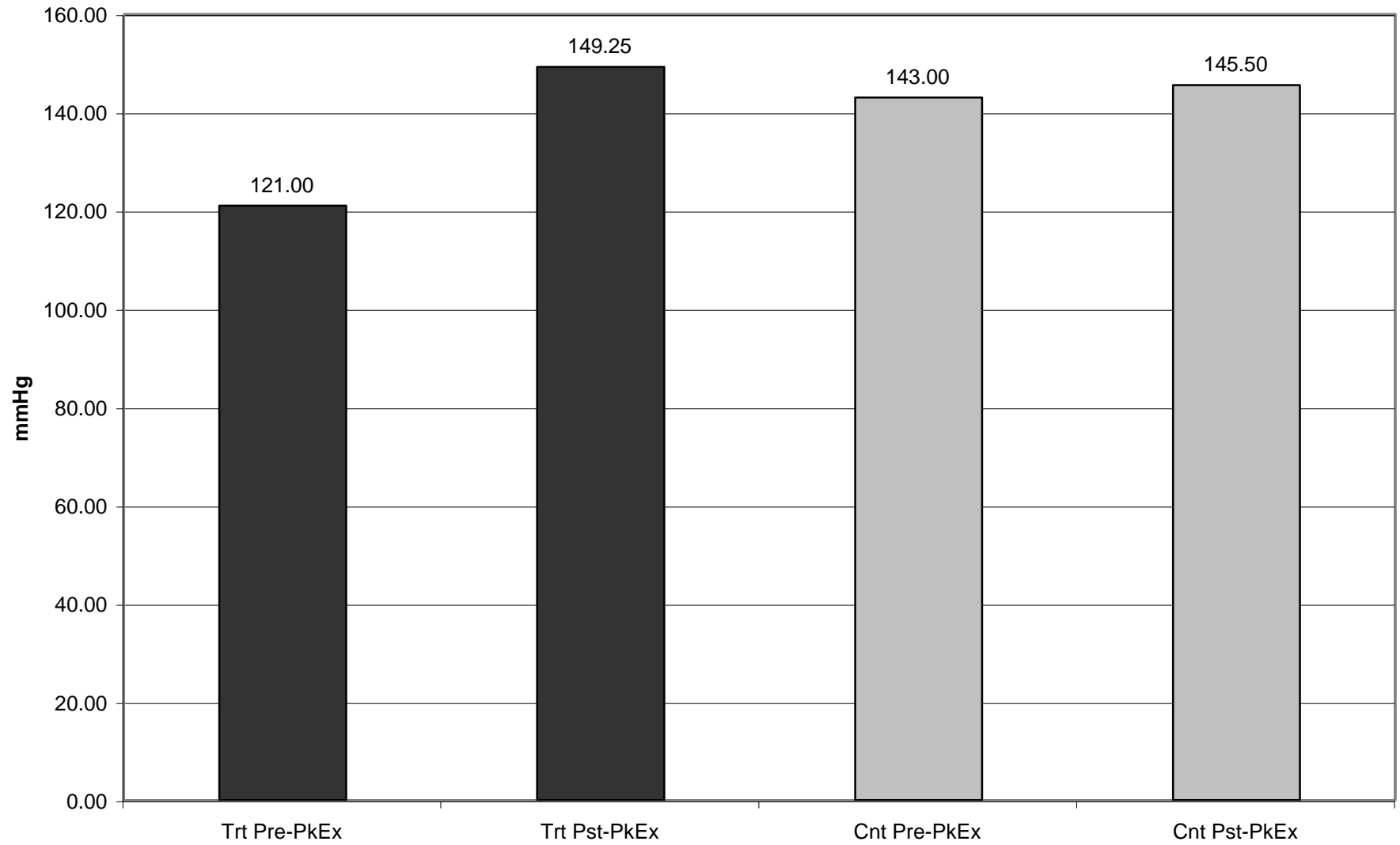
### Forced Vital Capacity



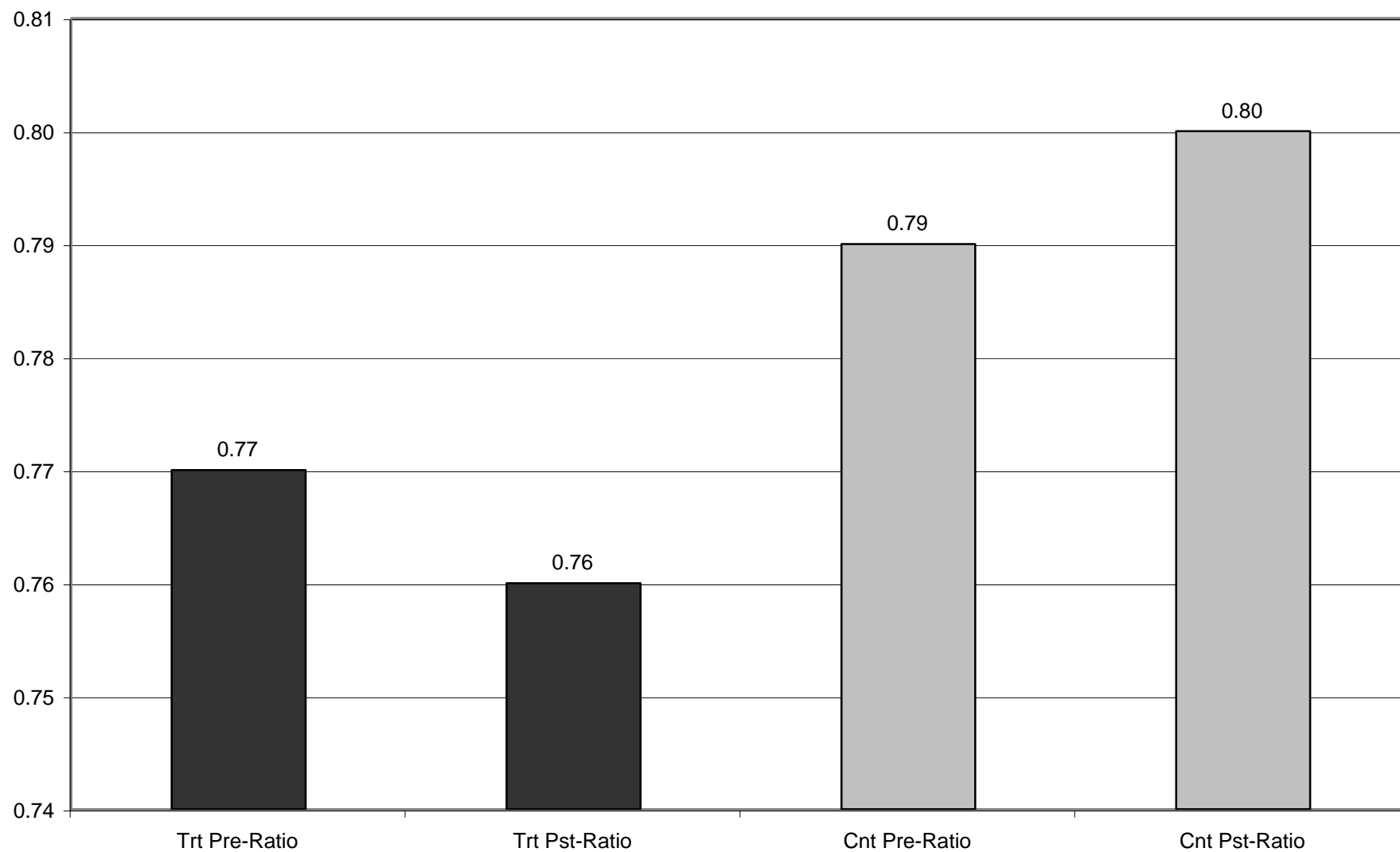
### Forced Expiratory Volume 1 Second



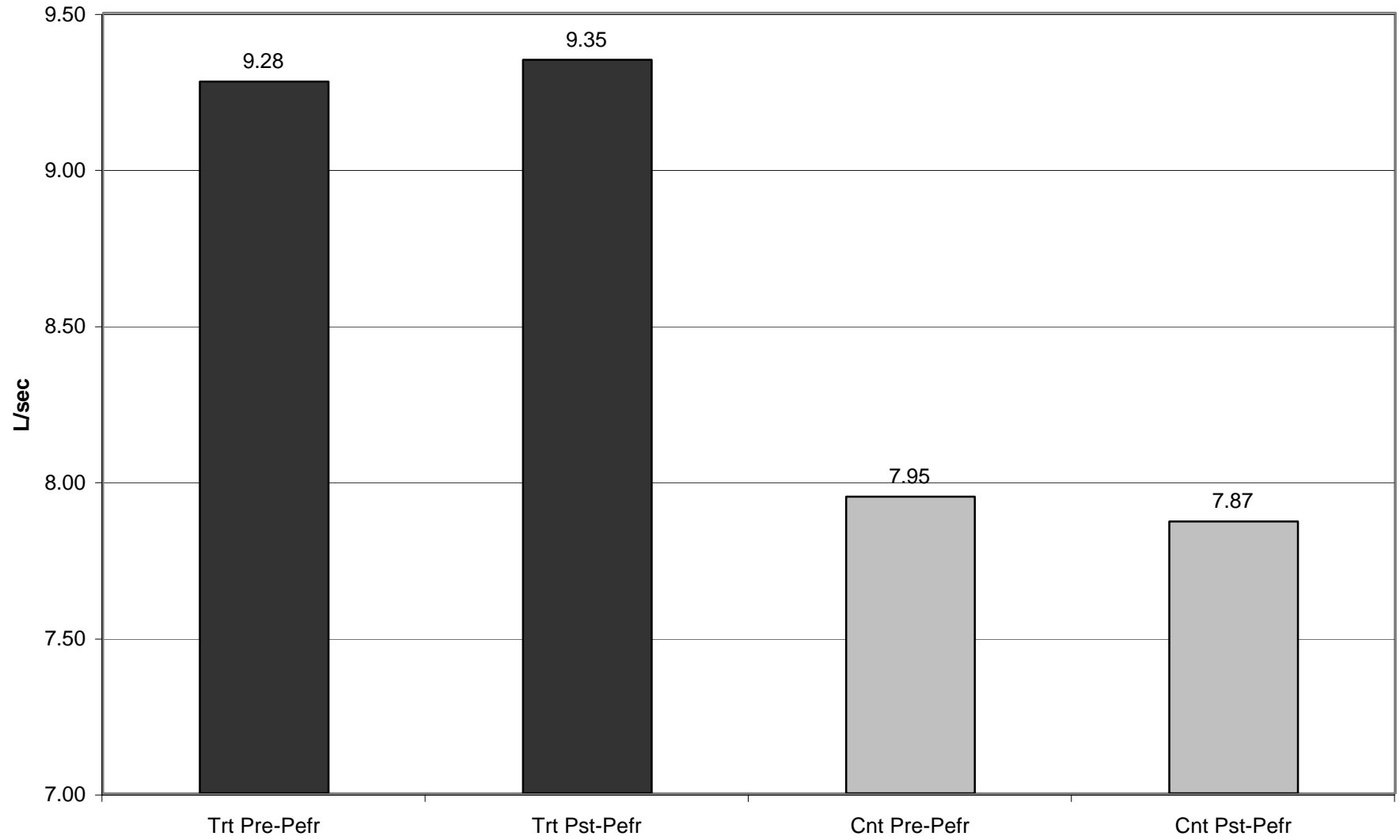
### Peak Exhalation Force



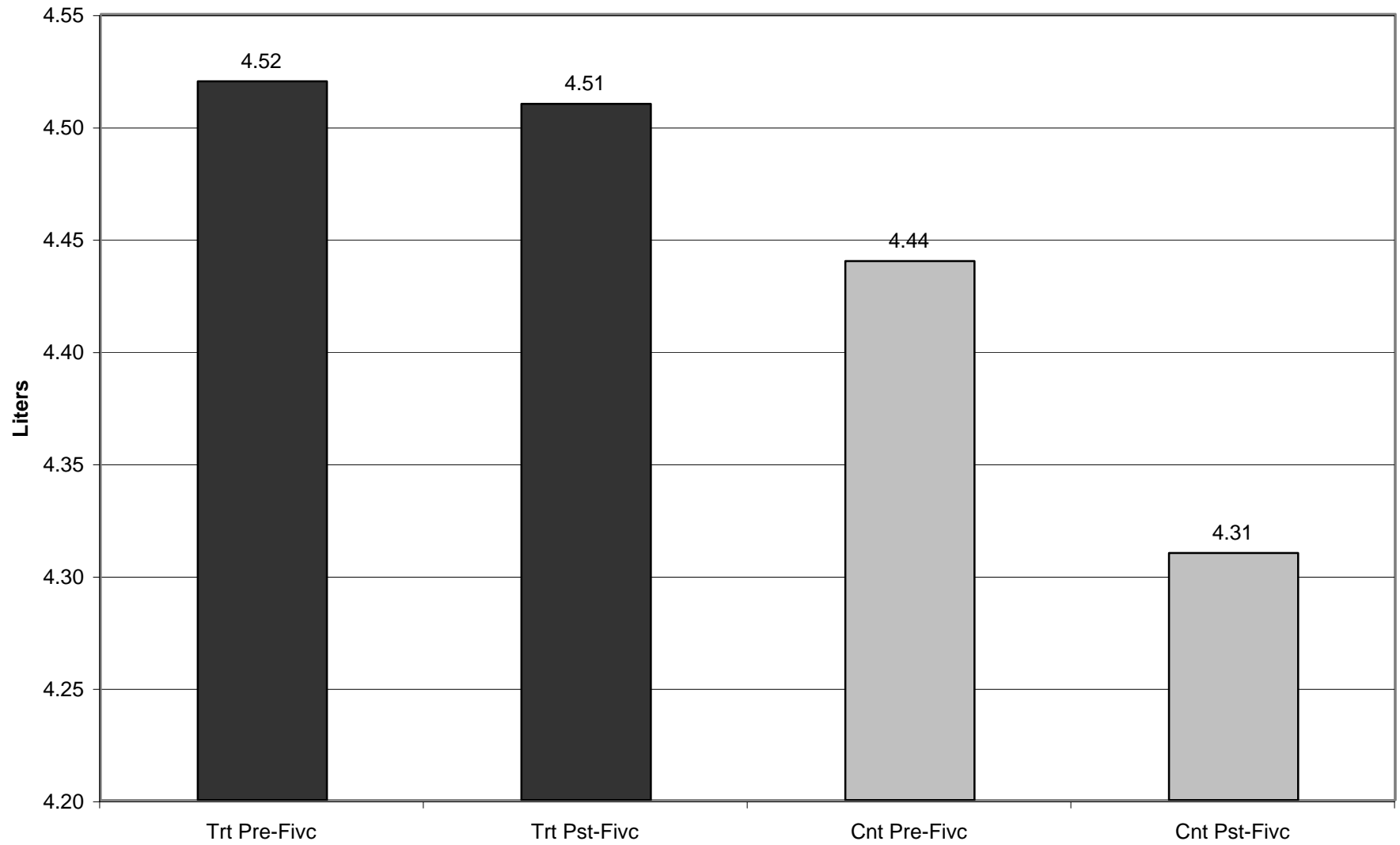
### FEV1/FVC Ratio



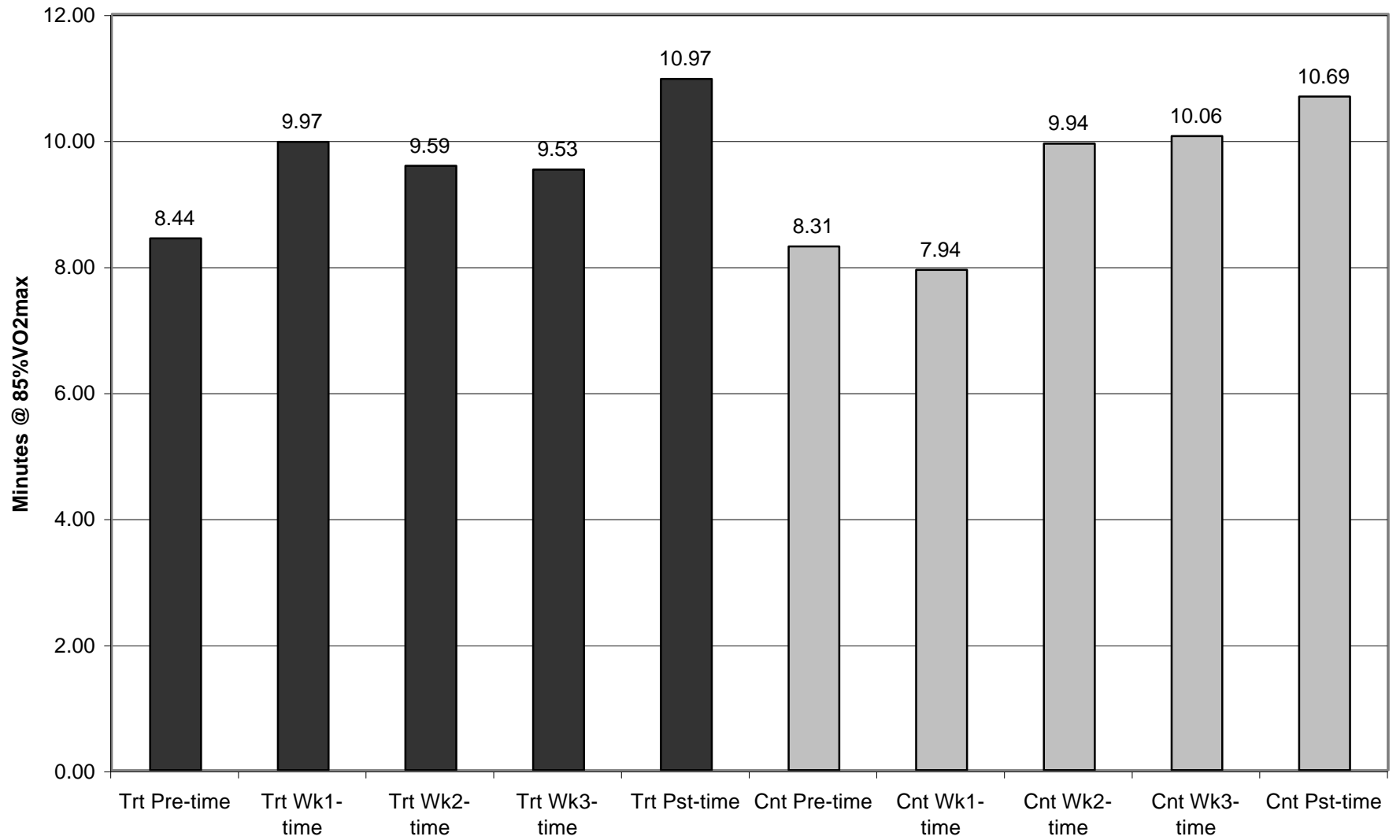
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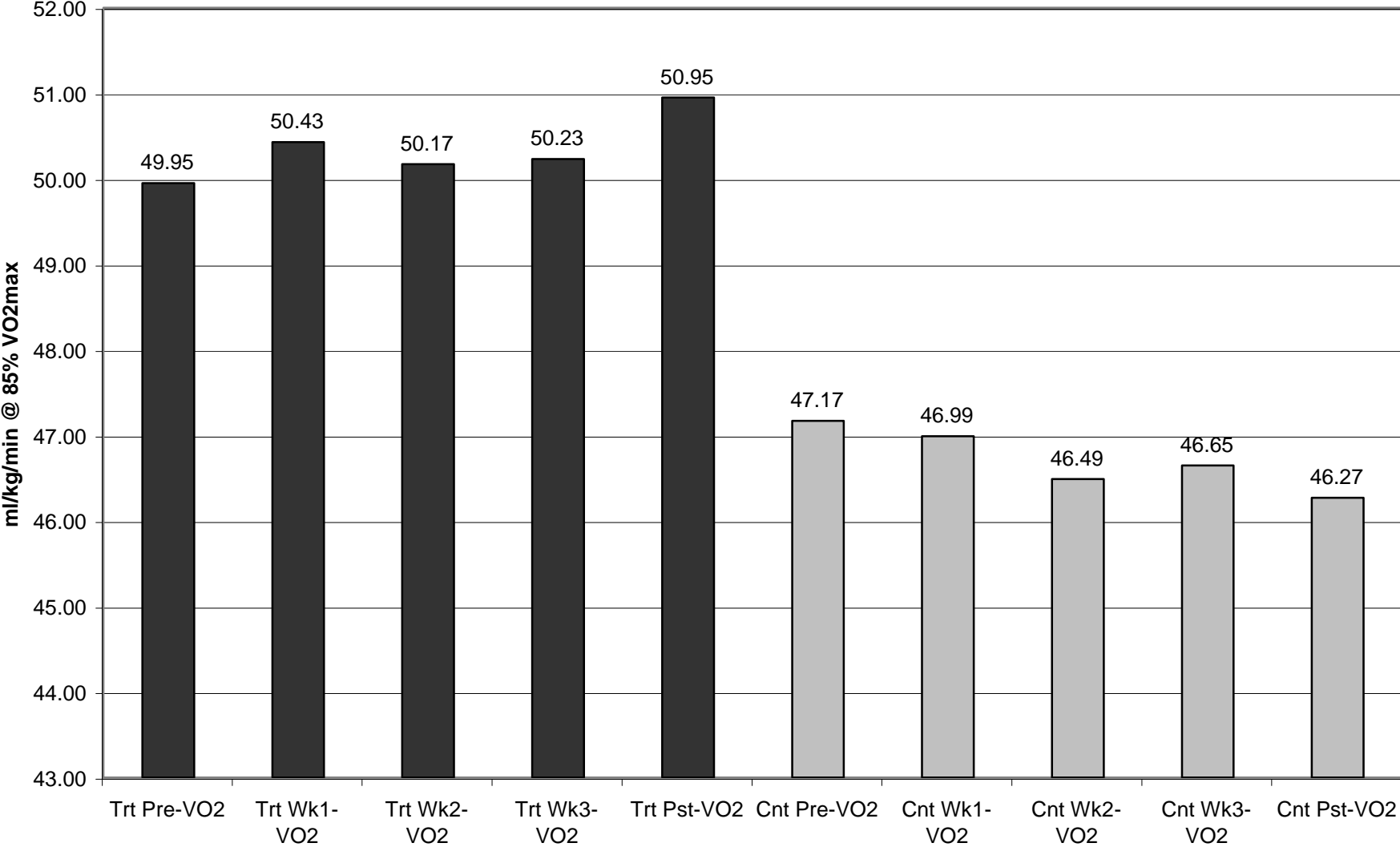
### Forced Inspiratory Volume



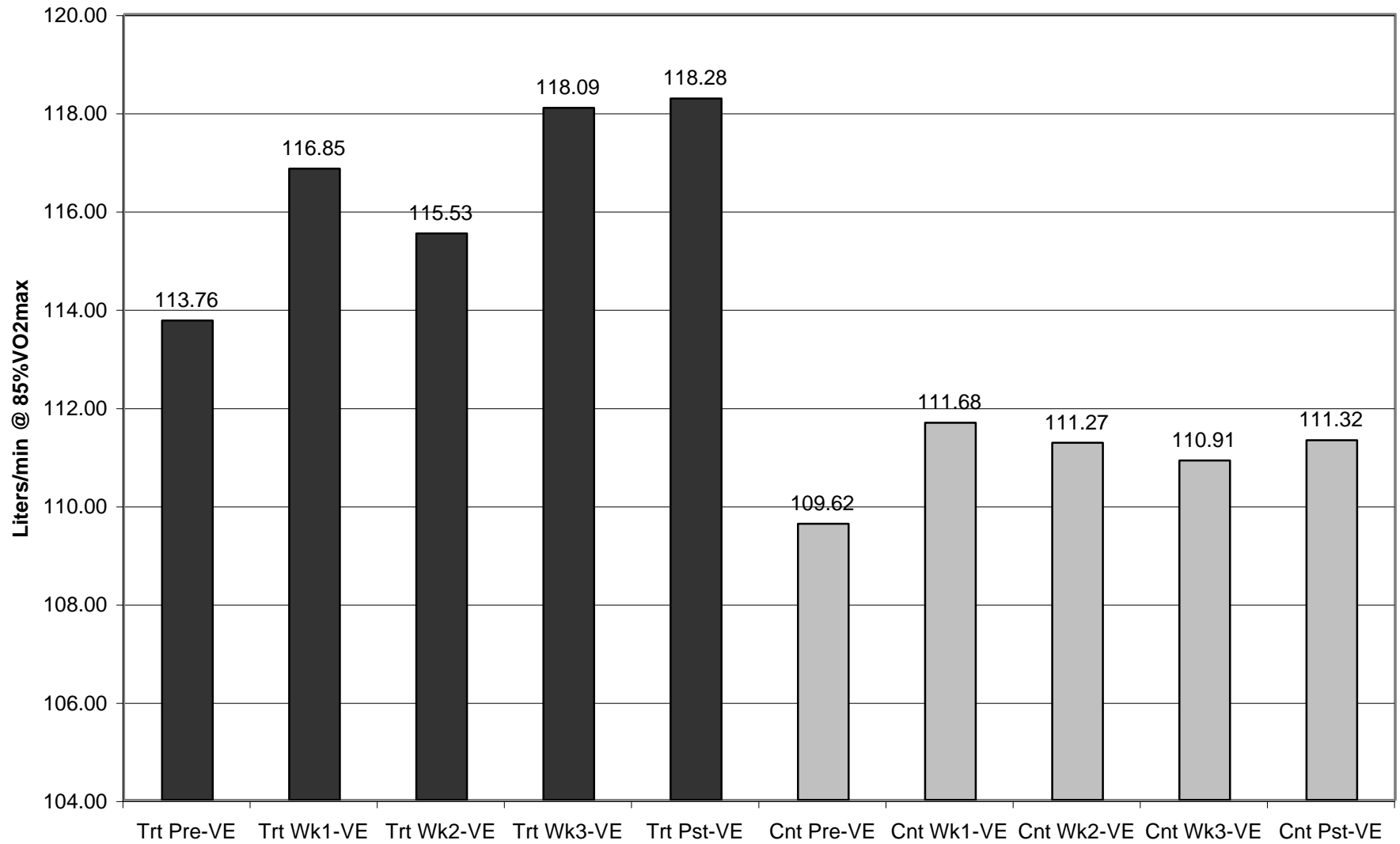
### Submaximal Exercise Time



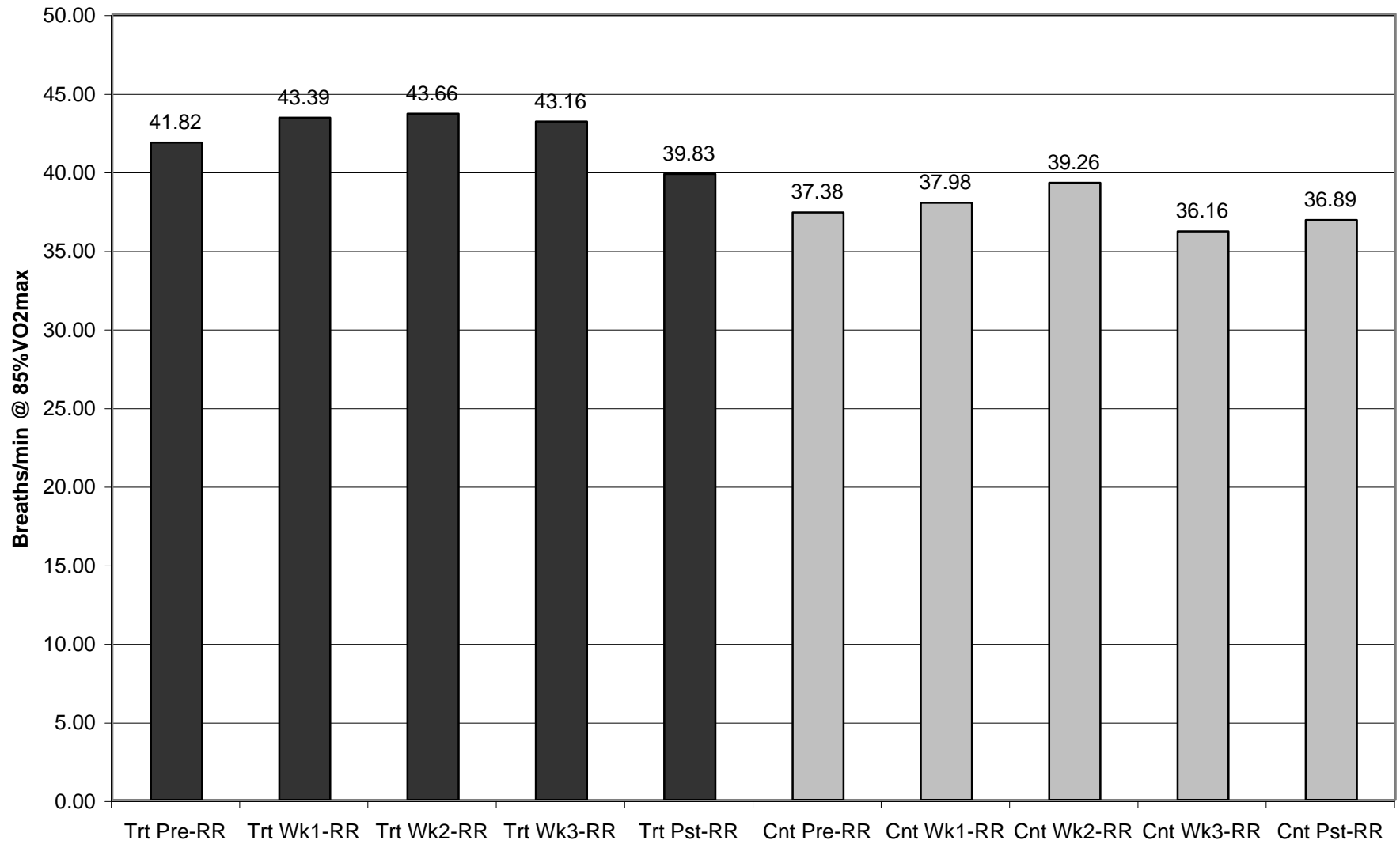
### Submaximal Oxygen Consumption



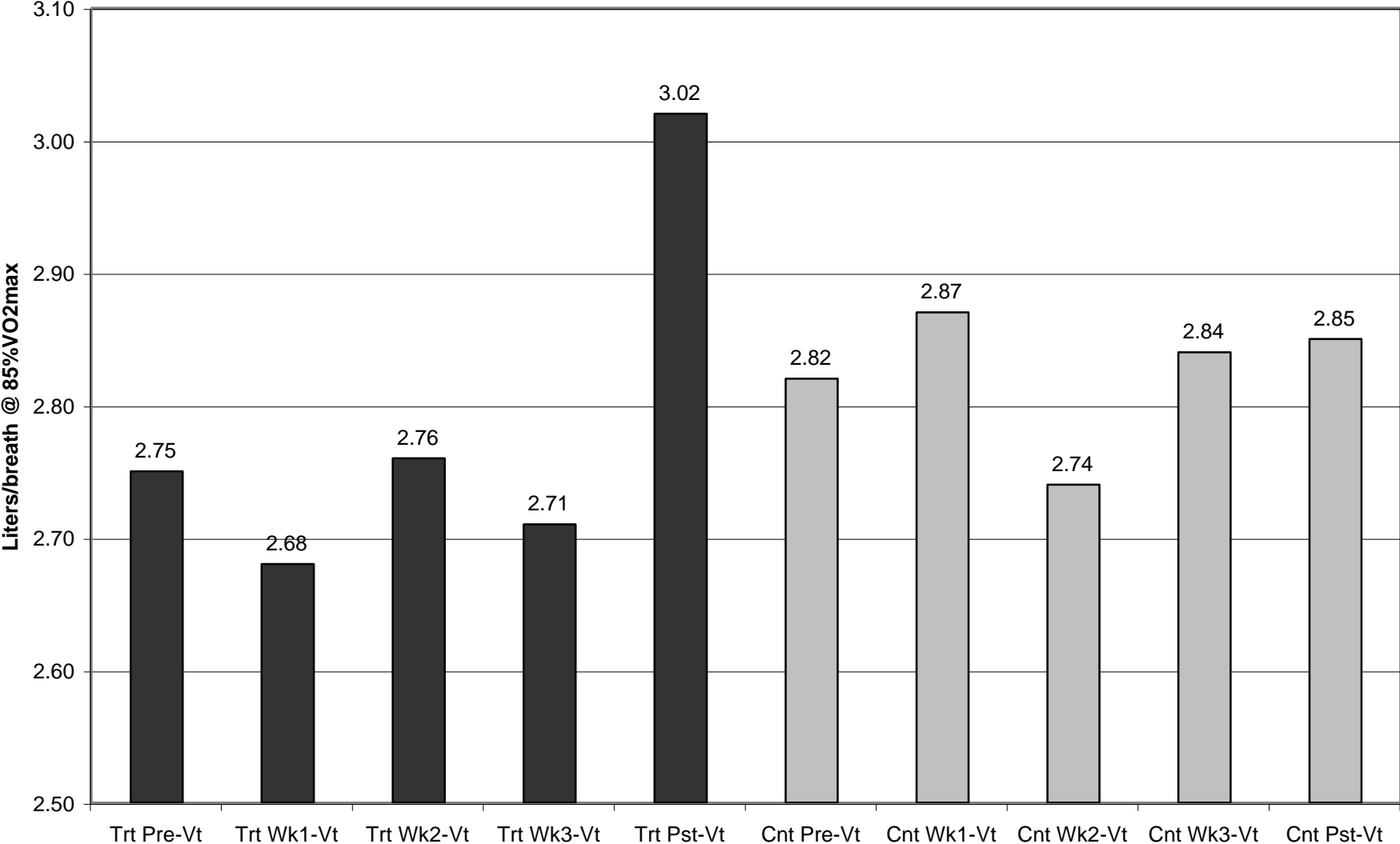
### Submaximal Minute Ventilation



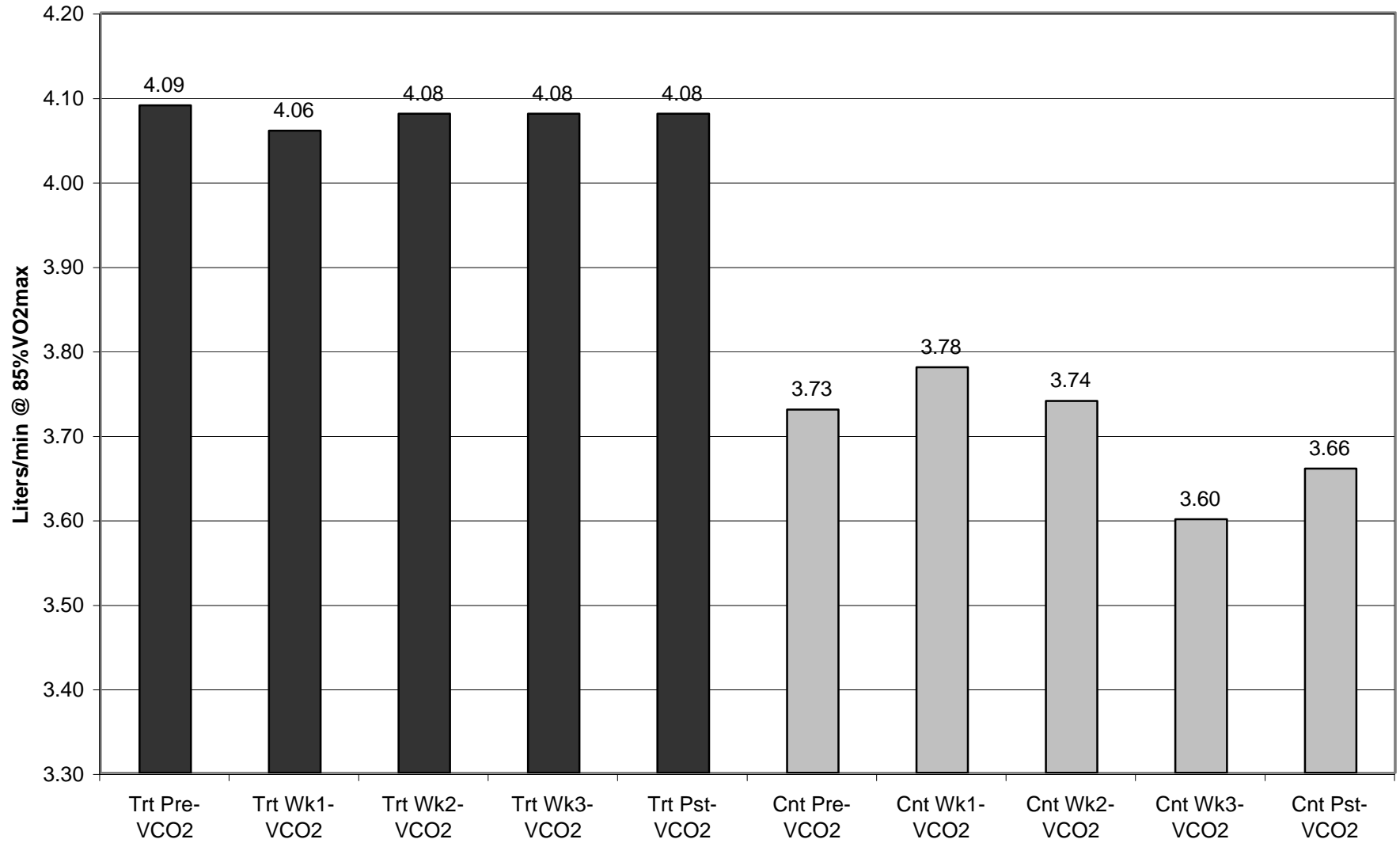
### Submaximal Respiration Rate



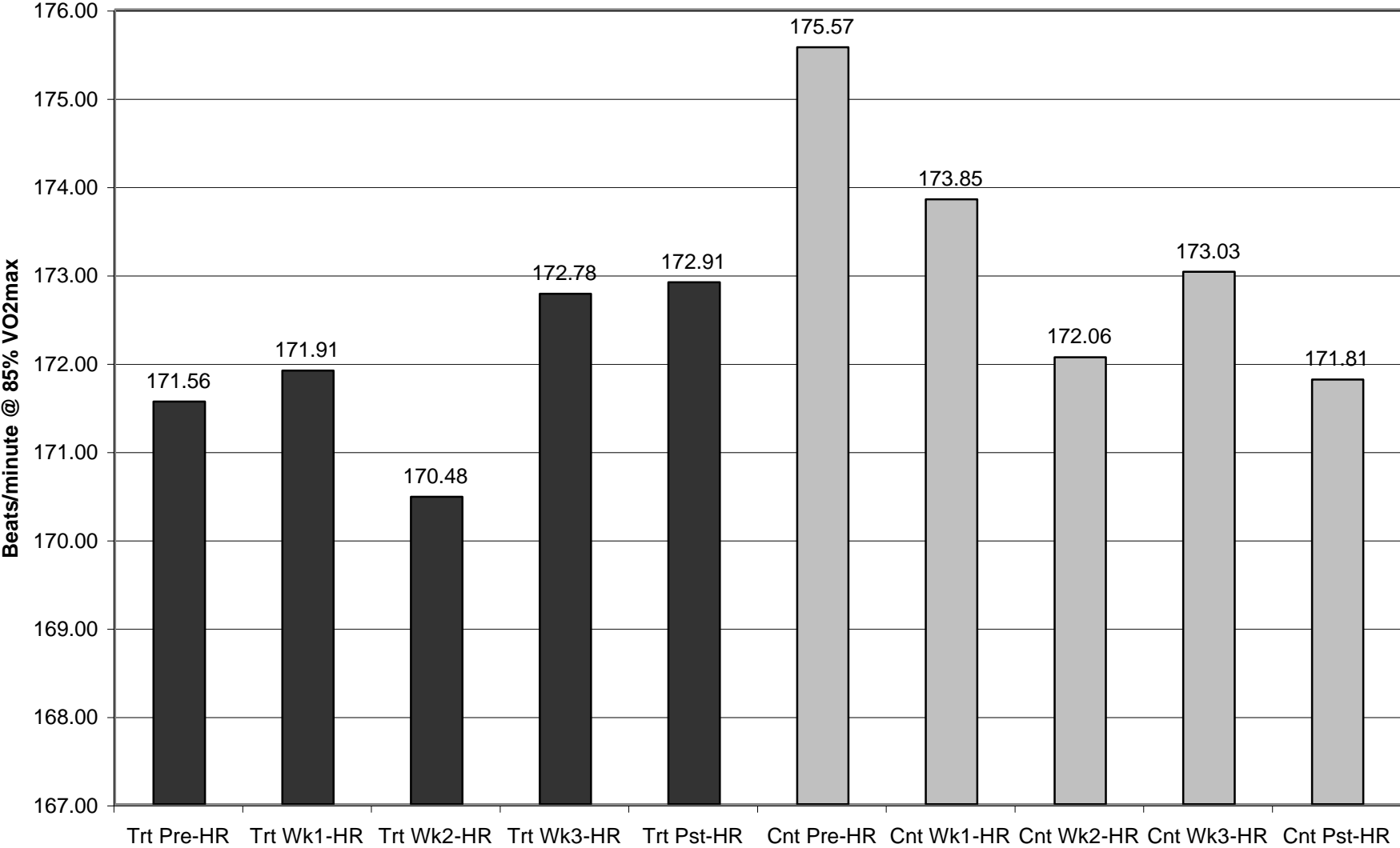
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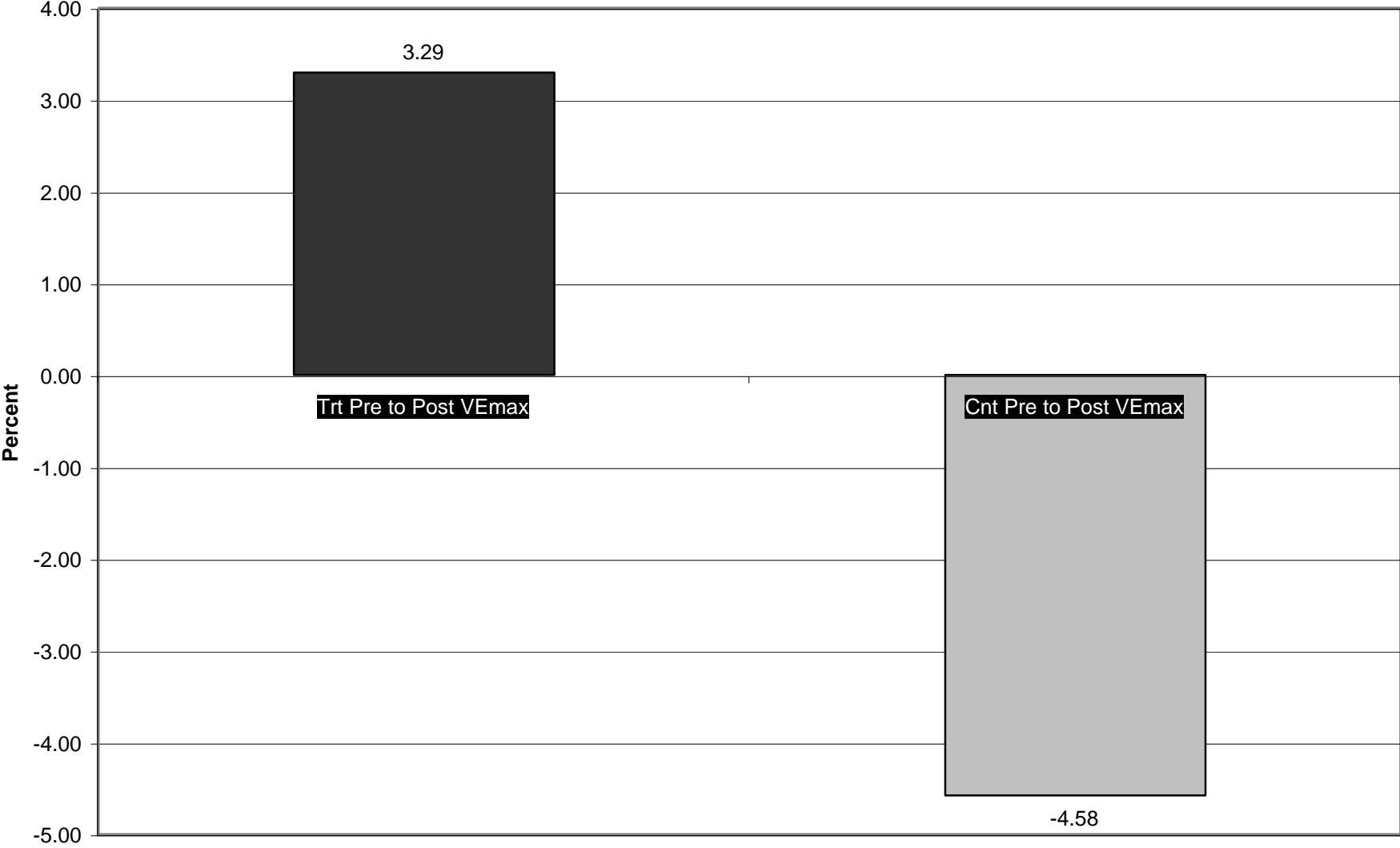
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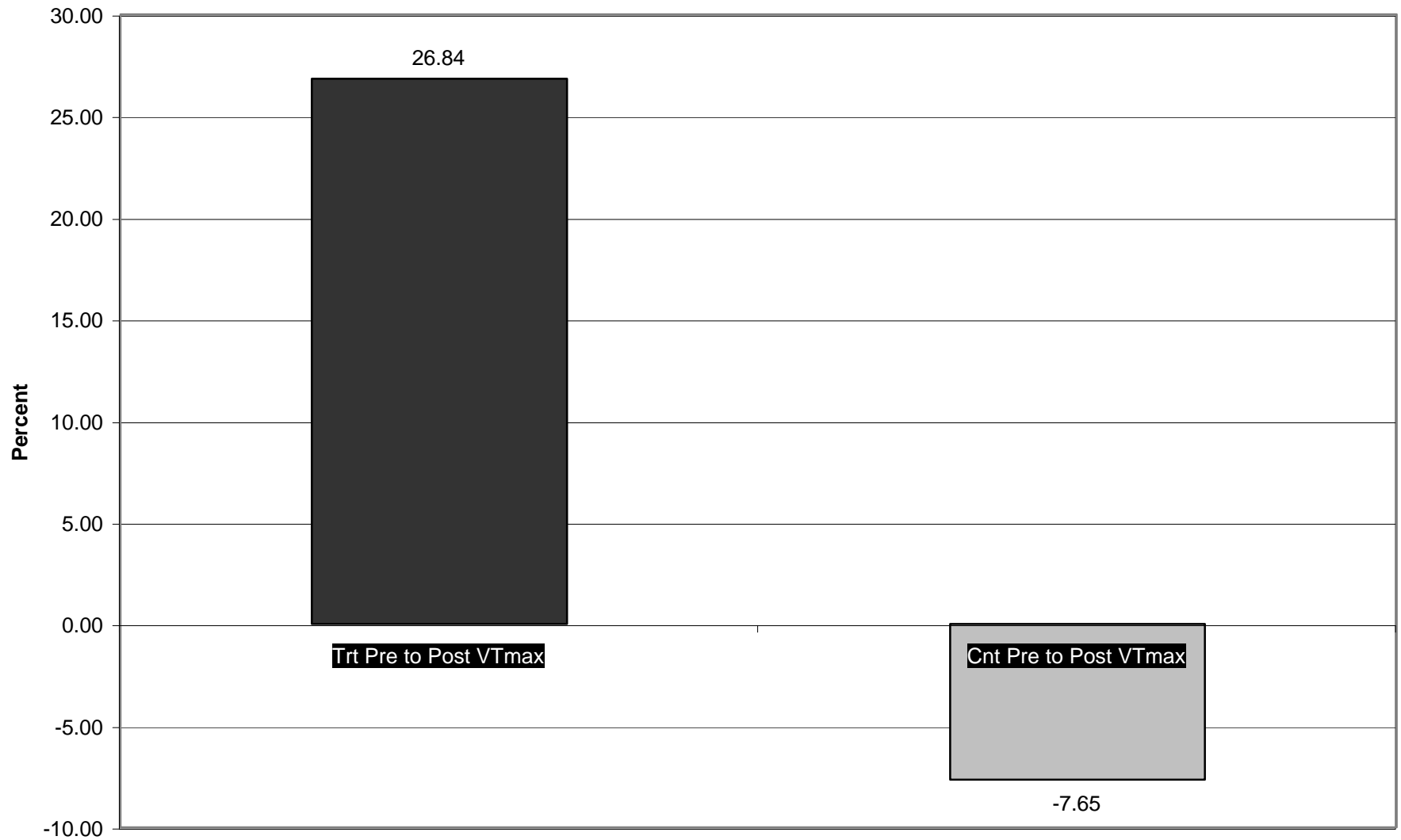
### Submaximal Heart Rate



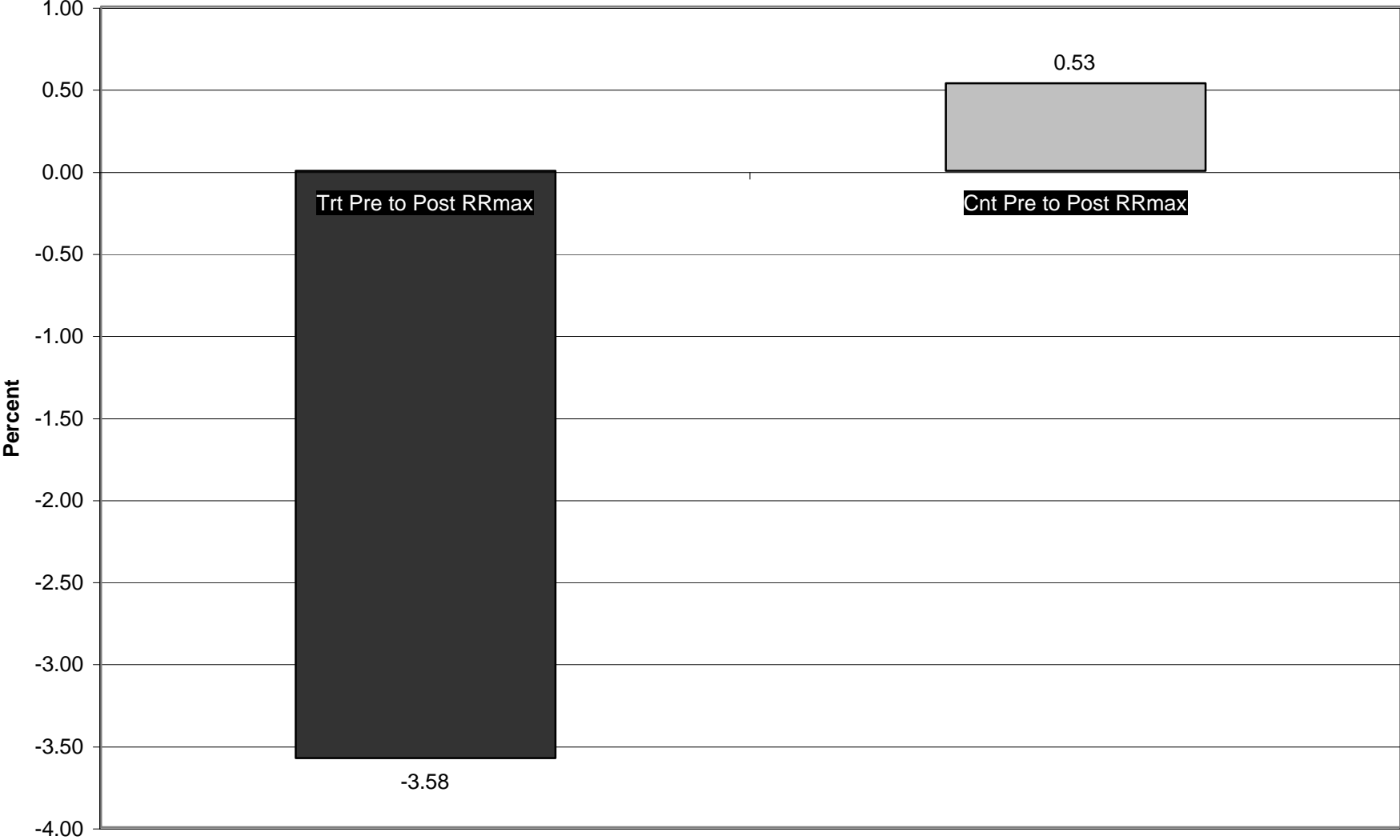
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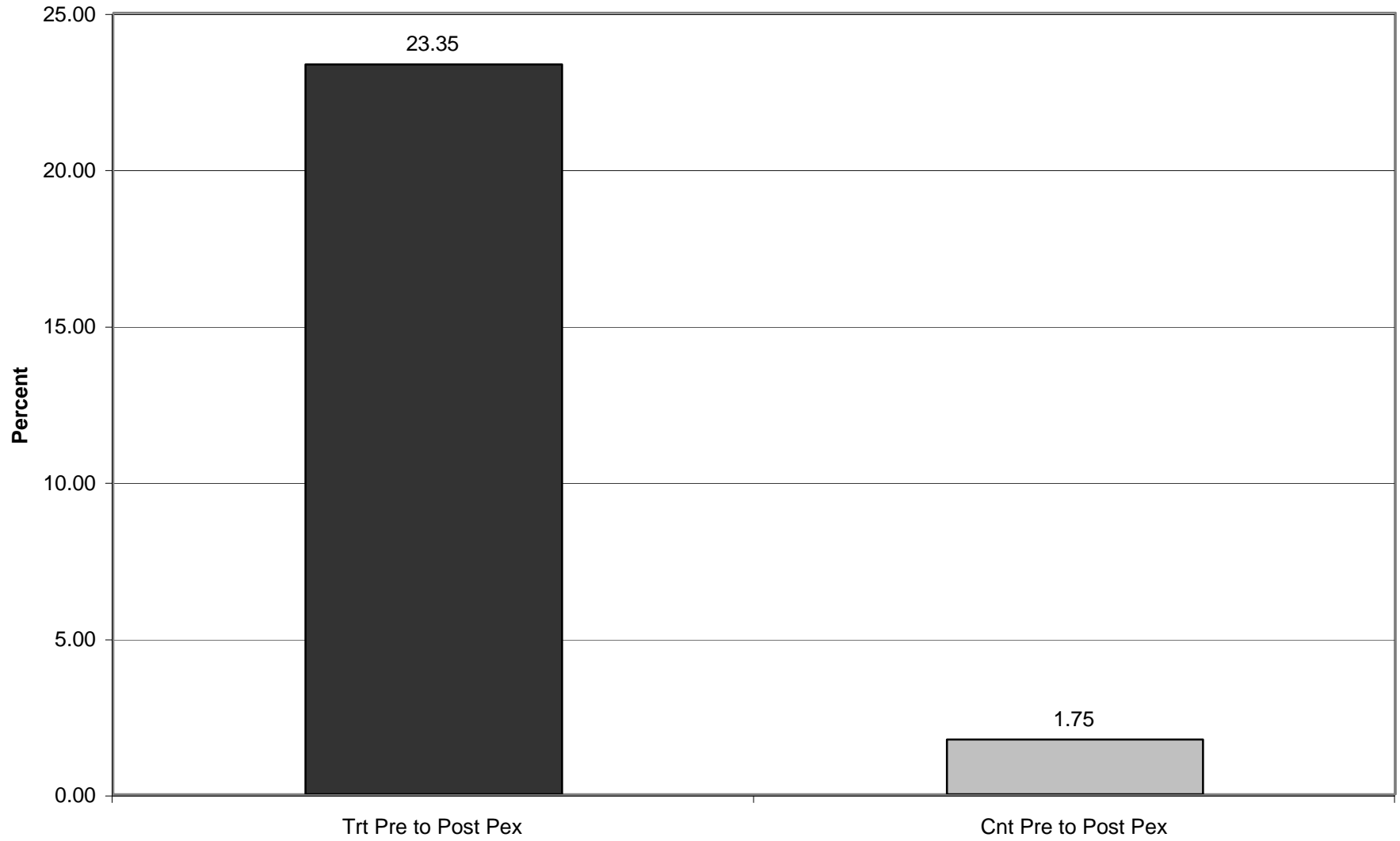
### Percent Change in VTmax



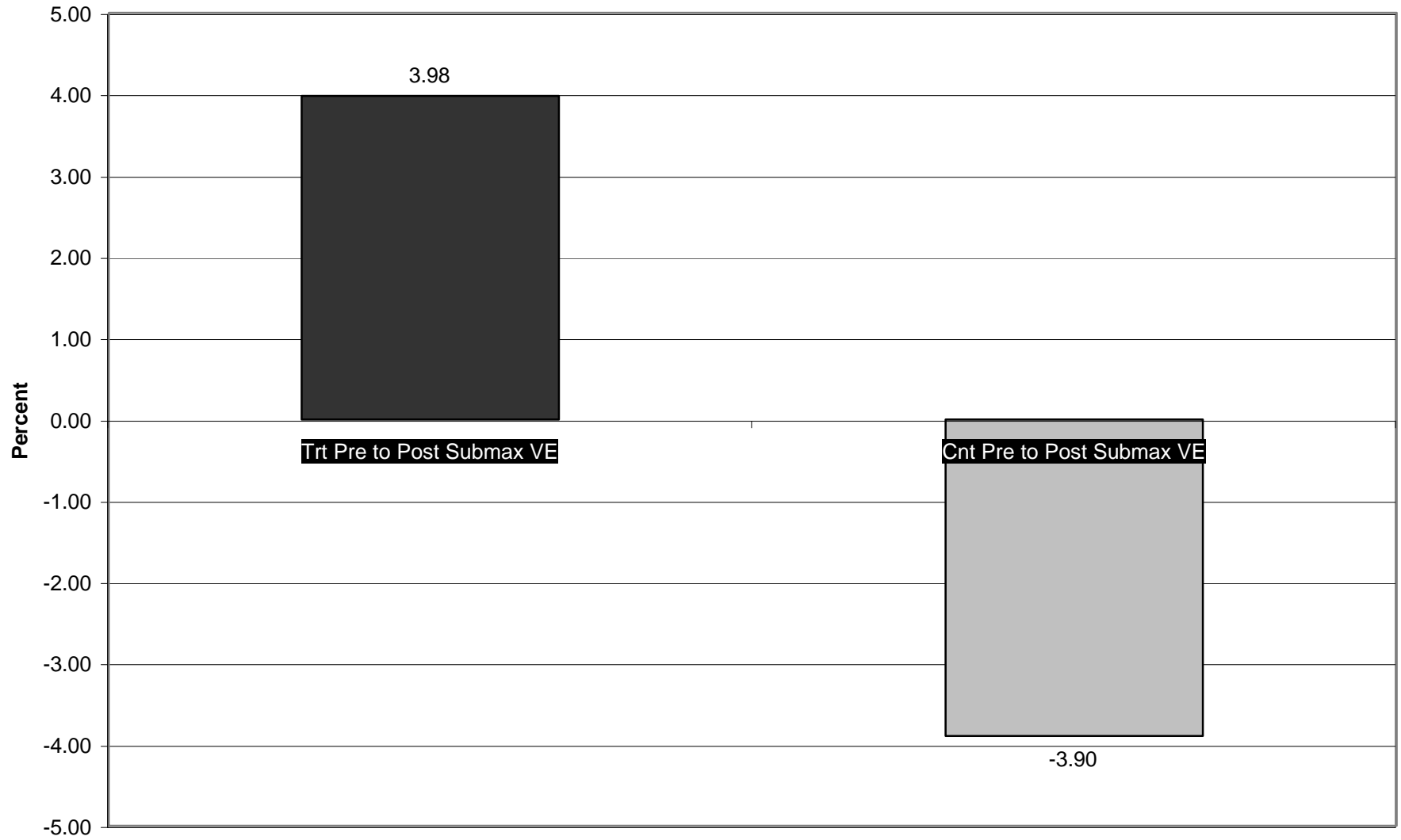
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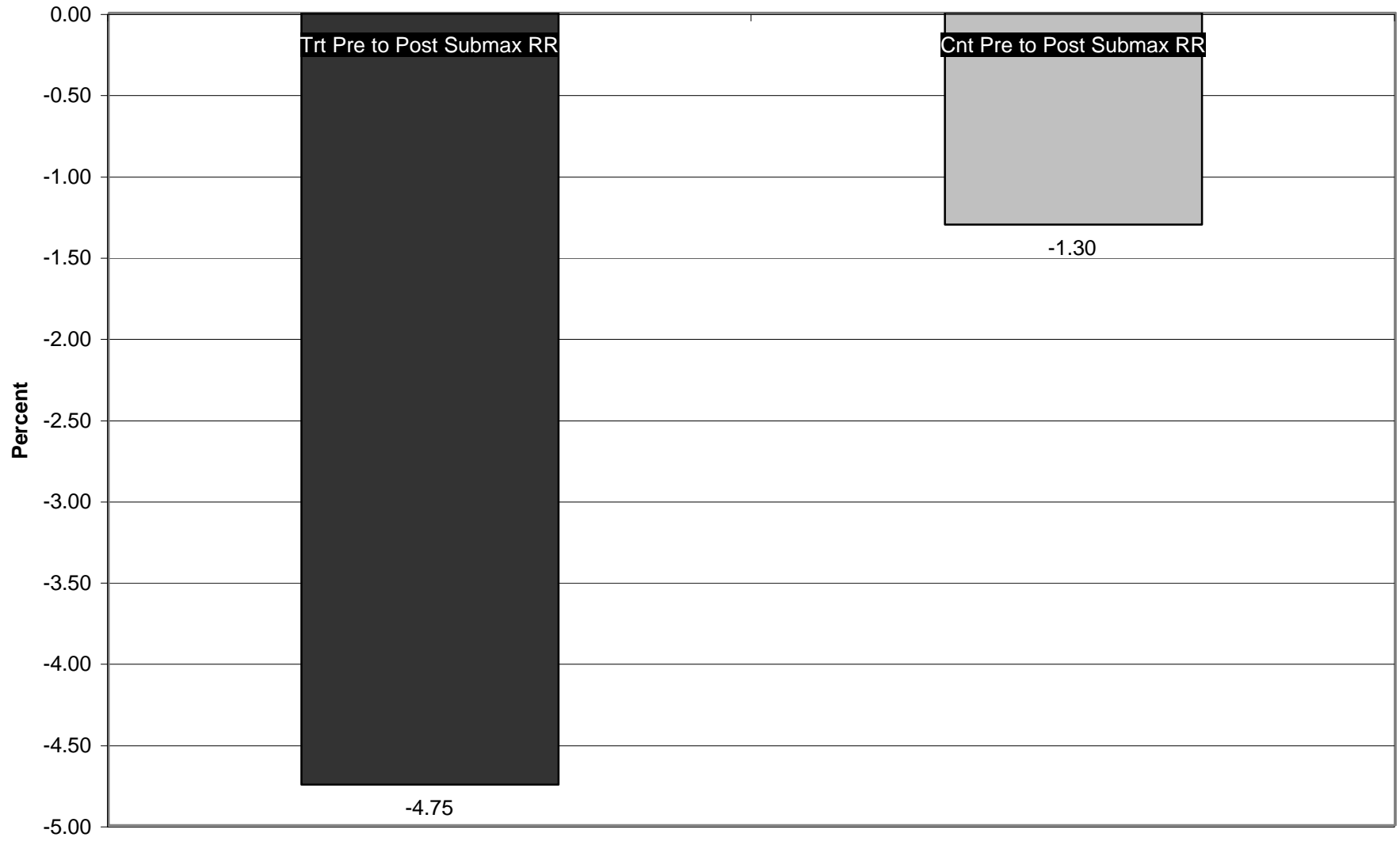
### Percent Change in Peak Exhalation



### Percent Change in Submax VE



### Percent Change in Submax RR



### Percent Change in Submax VT

