A Comparison of Respiratory Sinus Arrhythmia at Different Lung Volumes in Athletes, Swimmers, and Nonathletes

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CONCEPTUAL FRAMEWORK

An arrhythmia is a condition in which the heart beats with an irregular or abnormal rhythm. Respiratory sinus arrhythmia (RSA) is in which the heart rate increases with inspiration and decreases with expiration. RSA is not an abnormal rhythm and is most commonly seen in young healthy subjects (Issa et al. 2012). Sinus arrhythmia is present when the P wave morphology is normal and consistent, and the P-P intervals vary by more than 120 milliseconds (Issa et al. 2012). Arrhythmia is not a marker for structural heart disease. Heart rate responses to deep breathing and the Valsalva maneuver are informative autonomic tests (Issa et al. 2012).

RSA is assessed during controlled breathing at a rate of six deep breaths per minute. RSA ratio is calculated by dividing the longest to the shortest R-R interval. This expiratory per inspiratory ratio decreases progressively with age. Subjects younger than 40 years old have a ratio of less than 1.2 (Issa et al. 2012). A Valsalva maneuver is induced by having the subject blow against a 40 mm Hg pressure for 12 seconds. The Valsalva ratio is calculated by dividing the fastest heart rate during phase II by the slowest heart rate in phase IV. As with the expiratory ratio, the Valsalva ratio decreases with age (Issa et al. 2012). The heart rate accelerates during inspiration and slows during expiration (Feher 2012). This is due to the connections to the vagal nuclei from the inspiratory centers in the medulla. These connections hyperpolarize the cardiac motor neurons in the nucleus tractus solitarius (NTS), inhibiting their response to the baroreceptors. This causes the acceleration of the heart rate during inspiration (Feher 2012).

Heart rate variability indices differ in athletes from training status, different types of exercise training, and gender and aging (Aubert et al. 2003). An appropriate response to an acute aerobic exercise stimulus requires vigorous and integrated physiological augmentation from the pulmonary, respiratory, skeletal muscle, and cardiovascular systems (Lavie, C.J., et al. 2015). Age, sex, and genetic predispositions influence the physiological response and performance during aerobic exertion (Lavie, C.J., et al. 2015). Although all systems involved in orchestrating an appropriate response to aerobic exercise, the cardiovascular system, in particular the cardiac systolic and diastolic function, may be thought of as the “central hub” (Lavie, C.J., et al. 2015).

The left ventricle is reduced during aerobic exertion by a synergistic increase in end-diastolic volume and myocardial contractility (Lavie, C.J., et al. 2015). Participation in chronic aerobic exercise training program produces positive morphological and physiological cardiovascular adaptations in healthy individuals, regardless of age and sex (Lavie, C.J., et al. 2015). In a previous study, commonly reported morphological advantages associated with chronic aerobic exercise training is left ventricular dilation and hypertrophy Lavie, C.J., et al. 2015). These advantages are known as exercise training induced cardiac modeling. These morphological left ventricular adaptations parallel enhanced physiological function during exercise through (1) increased early diastolic filling secondary to a combination of increased preload and increased myocardial relaxation, and (2) increased contractile strength (Lavie, C.J., et al. 2015). The morphological adaptations also occur in the right ventricle that appear to mirror the left ventricular adaptations (Lavie, C.J., et al. 2015).

Repetitive amounts of aerobic exercise training results in a number of favorable vascular adaptations as well, such as, significantly reducing deleterious adaptations predicted by the aging process (Lavie, C.J., et al. 2015). When aerobic exercise training involves large muscle groups, systemic vascular benefits are realized (Lavie, C.J., et al. 2015). Aerobic exercise training also improves endothelium-dependent vasodilation in the coronary microcirculation through the increased production of nitric oxide (Lavie, C.J., et al. 2015).

Several studies have shown differences in respiratory measures for swimmers when compared to non-swimmers. One article tested to see whether respiratory muscle strength is related to pulmonary volume differences in swimmers and non-swimmers. The parameters tested included maximal inspiratory pressure and maximal expiratory pressure (Cordain et al. 1990). Swimmers exhibited larger vital capacities, residual volumes, inspiratory capacities, and functional residual capacities than both runners or the controls, but no difference in either maximal inspiratory volumes or inspiratory flow (Cordain et al. 1990). Timed expiratory volumes were significantly lower in the swimmers than in the controls. The data suggests that an adaptational growth may be responsible, in part, for the augmented static lung volumes demonstrated in swimmers (Cordain et al. 1990). Studies of swimmers have demonstrated increases in vital capacities and total lung capacities following training programs suggesting that environmental factors may be responsible for these higher lung volumes (Cordain et al. 1990). It has been suggested that increases in respiratory muscle strength changes in compliance of the chest wall and lung, or perhaps alveolar hyperplasia, account for this adaptive response to water-based training (Cordain et al. 1990).

PURPOSE OF THE STUDY

The purpose of this study is to compare the change in respiratory sinus arrhythmia at different lung volumes, and during apnea following a large inhale, and during apnea following a large exhale among competitive collegiate runners, swimmers, and nonathletic controls. This study seeks to compare RSA changes in swimmers to both other athletes and non-athletes.

RATIONALE AND SIGNIFICANCE

Aim 1: This exercise aims to compare the respiratory sinus arrhythmia during restful breathing.
Question 1: Is RSA during resting tidal volume breaths different in swimmers, athletes, and nonathletes?
Hypothesis: Swimmers will have a higher restful tidal volume breath and a higher RSA than athletes and nonathletes.
Rationale: In a previous study, swimmers exhibited larger vital capacities, residual volumes, inspiratory capacities, and functional residual capacities than both runners and the controls (Cordain et al. 1990). Since the swimmers demonstrated these larger qualities, I predict that swimmers will also have larger tidal volumes. The heart rate accelerates during inspiration and slows during expiration (Feher 2012). Swimmers have to inspire and expire very quickly, and their tidal volumes are significantly larger (Cordain et al. 1990). Since swimmers have to inspire and expire quickly, I predict swimmers will have a higher RSA, which would maximize the efficiency of gas exchange during inspiration.
Methods for Exercise 1: The subject breathed through the spirometer flowhead normally for one minute. I observed the subject's pulse, airflow, lung volumes, and heart rate while the subject was breathing at rest. The exercise was repeated two more times per subject in the same session for a total of three minutes. I recorded the measurements of tidal volume per breath, and

RSA, which is the maximum and minimum heart rate during a breath. Raw data including tidal volume and RSA and the averages were collected and recorded into the lab journal.
Aim 2: This exercise aims to measure the effect of apnea, after maximum inhalation, on the subject's heart rate.
Question 2: Does holding an inhalation over time cause a different RSA in swimmers, athletes, and nonathletes?
Hypothesis: Swimmers will have a higher RSA during a held inhalation than athletes and nonathletes.
Rationale: In a previous study, during breath-holding swimmers demonstrated marked apneic bradycardia expressed as their heart rate change from basal heart rate as opposed to the controls, whose heart rate increased during breath holds (Bjustrom R. L. and Schoene R. B. 1987). Since swimmers can control their breathing easily, I predict that swimmers will have a higher RSA.
Methods for Exercise 2: The subject took 2-3 normal breaths through the flow head before he/she took a deep breath and held their breath for as long as possible. When the subject resumed breathing, they continued to breathe through the flow head until the breathing pattern was back to normal. The procedure was repeated two more times per subject to record the measurements of heart rate per breath before, during, and after apnea. I calculated RSA over the length of the held breath. Raw data includes the RSA and time and length of breath held. The averages were collected and recorded in the lab journal.

EXPERIMENTAL PLAN
Study site: I focused on the three subject groups while carrying out 2 exercises per individual for 3 weeks. This study was completed at McKendree University in the Voigt Building in room 104. I collected the data using the HK-TA Human Physiology Teaching Kit and LabScribe software. The data from each subject in the three groups was anonymous and was recorded as 'subject' followed by a series of numbers.
Subjects: Athletes were defined as marathon runners running at least 25-70 km per week for at least six months (Prakash et al. 2007 and Cordain et al. 1990). Swimmers were composed of both sprinters and long-distance athletes who train between 3,000 to 7,000 meters daily for at least six months (Cordain et al. 1990). Nonathletes were defined as a lifestyle with no leisure-time physical activity or activities done for less than twenty minutes or fewer than three times per week (Prakash et al. 2007). A "smoker" was defined as an individual who has smoked more than 100 cigarettes in their lifetime and currently smokes (Prakash et al. 1990). The subjects consisted of three groups: 5 collegiate Division II runners, 5 collegiate Division II swimmers, and 5 collegiate control subjects that match in height and age. I had a total of 15 subjects. The subjects were male and female of any race, age between 18-25 years of age, non-obese with a BMI of less than 30.0, free of smoking within the previous five years, and free of all known respiratory and cardiovascular disease(s). The female height range was 152-183 cm and the male height range was 165-195 cm. All subjects were to refrain from exercise within 12 hours of reporting to the laboratory for pulmonary function testing since the forced vital capacities are temporarily reduced following acute exercise, whereas residual lung volumes are temporarily increased (Cordain et al. 1990). Participants answered a survey to rule out any respiratory and cardiovascular disease(s). This survey was used to determine if the subjects are eligible to participate. I measured heart rate during normal breathing, heart rate at breaths of different lung volumes, and heart rate during apnea. All measurements were made while the subjects were seated and while breathing through the spirometer flowhead. The subject's vitals were monitored

throughout the experiments. Participants were aware that they may become dizzy and/or nauseated from the experiment and also may because nauseated. Participants may learn how their RSA varies with different breath volumes. Participants may opt-out of the research at any time and the participants are in control of their breathing on their own. The subject will have several minutes of normal breathing between exercises, and I will check on participants between exercises for symptoms of dizziness or nausea. Records identifying participants will be kept confidential. To ensure confidentiality, the subject's identifying information will never be associated with the information or data that is provided.

RESULTS

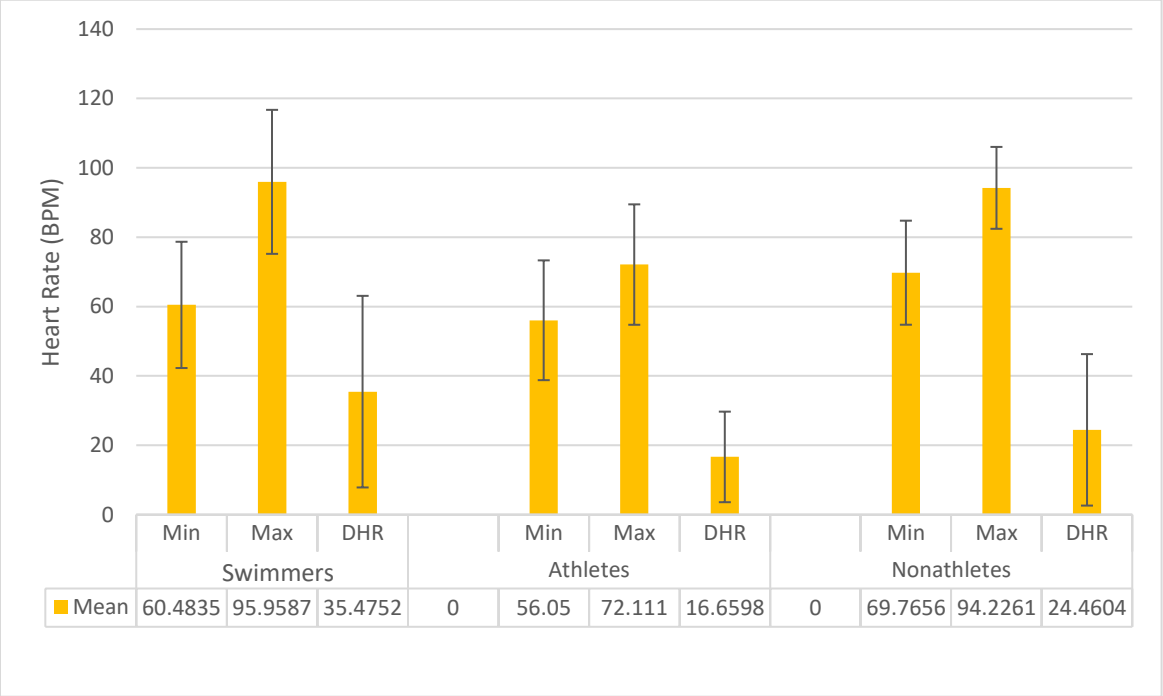


Figure 1: Resting heart rate during rest in swimmers, athletes, and nonathletes. DHR is noted as RSA.

In figure 1, athletes averaged the lowest RSA value (16.6598 ± 13.06) when compared to swimmers (35.4752 ± 27.62) and nonathletes (24.4604 ± 21.83). Swimmers also had the highest max heart rate (95.9587 BPM ± 20.76) while at rest. Nonathletes had the highest minimum heart rate while at rest (69.7656 ± 14.99). Swimmers had the highest deviation in the minimum heart rate (± 18.20), maximum heart rate (± 20.76), and RSA (± 27.62) categories when compared to nonathletes and athletes. Differences among the four groups were determine using a one-way ANOVA with Duncan's Multiple Range Test for post-hoc analysis. No significant differences ($p > 0.05$) were demonstrated among the three groups.

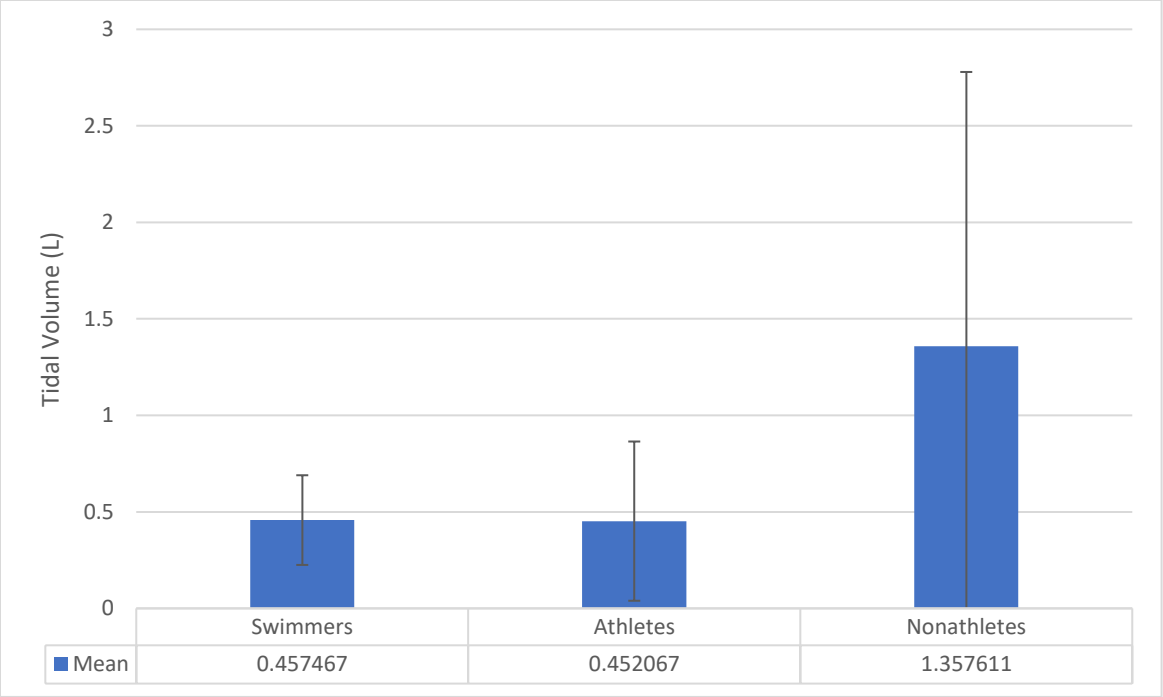


Figure 2: Tidal volumes in swimmers, athletes, and nonathletes during rest. Tidal volume is measured in liters.

In figure 2, nonathletes averaged the highest tidal volumes (1.357611 L ± 1.42) when compared to swimmers (0.457467 L ± 0.23) and athletes (0.452067 ± 0.41). Swimmers had the lowest standard deviation (± 0.23) whereas nonathletes had the highest standard deviation (± 1.42).

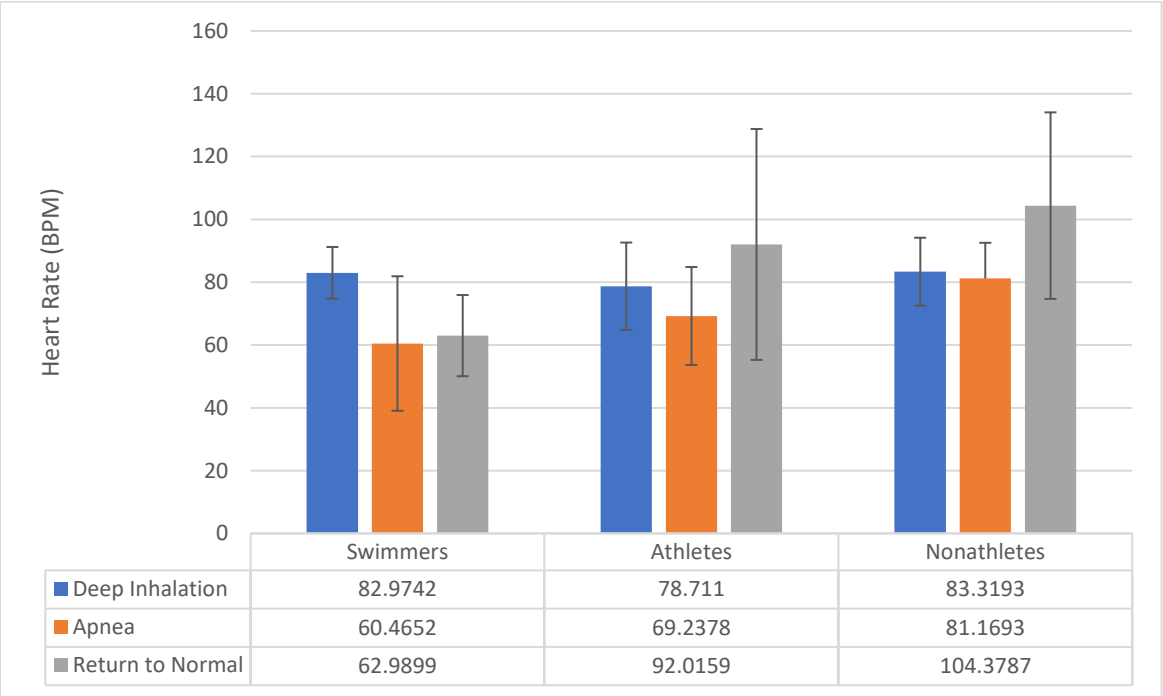
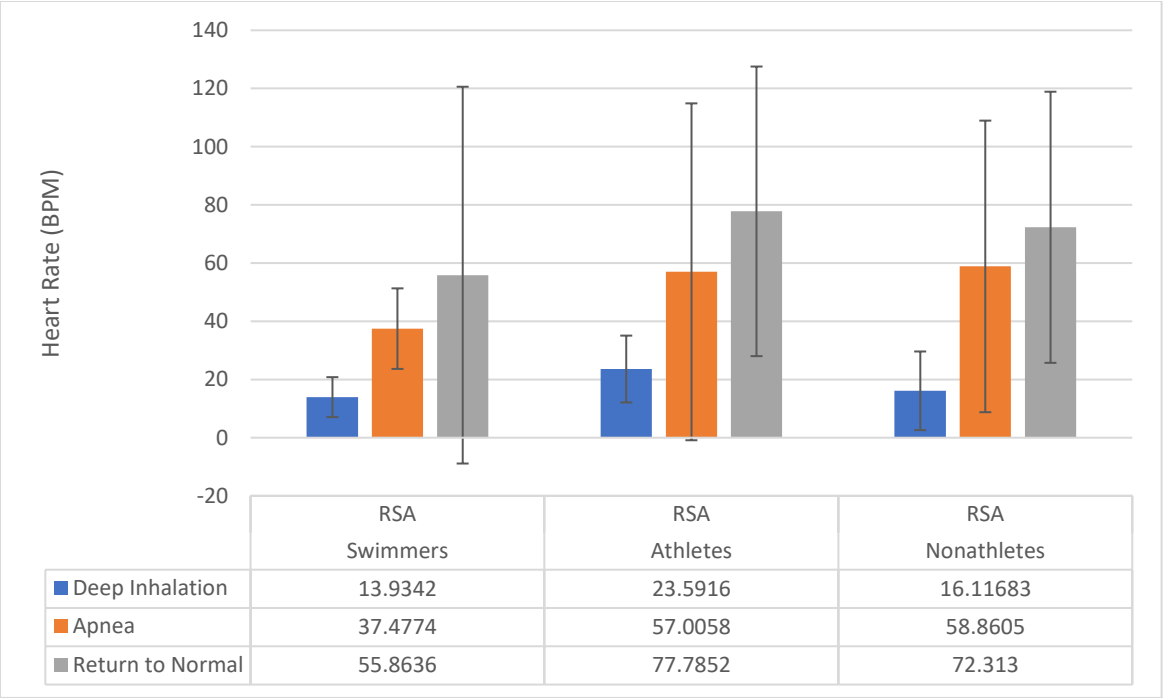


Figure 3: Heart rate during apnea in swimmers, athletes, and nonathletes.

In figure 3, nonathletes had the highest heart rate during all three categories when compared to swimmers and athletes. Athletes, swimmers, and nonathletes had very similar heart rates during deep inhalation. During apnea, swimmers had the lowest heart rate (60.4652 BPM ± 13.84) than athletes (69.2378 BPM ± 57.89) and nonathletes (81.1693 BPM ± 49.77).



	RSA Swimmers	RSA Athletes	RSA Nonathletes
Deep Inhalation	13.9342	23.5916	16.11683
Apnea	37.4774	57.0058	58.8605
Return to Normal	55.8636	77.7852	72.313

Figure 4: RSA during deep inhalation, apnea, and returning to normal breath in swimmers, athletes, and nonathletes.

In figure 4, swimmers had the lowest change in heart rate in all three categories whereas nonathletes had the highest change in heart rate in all three categories.

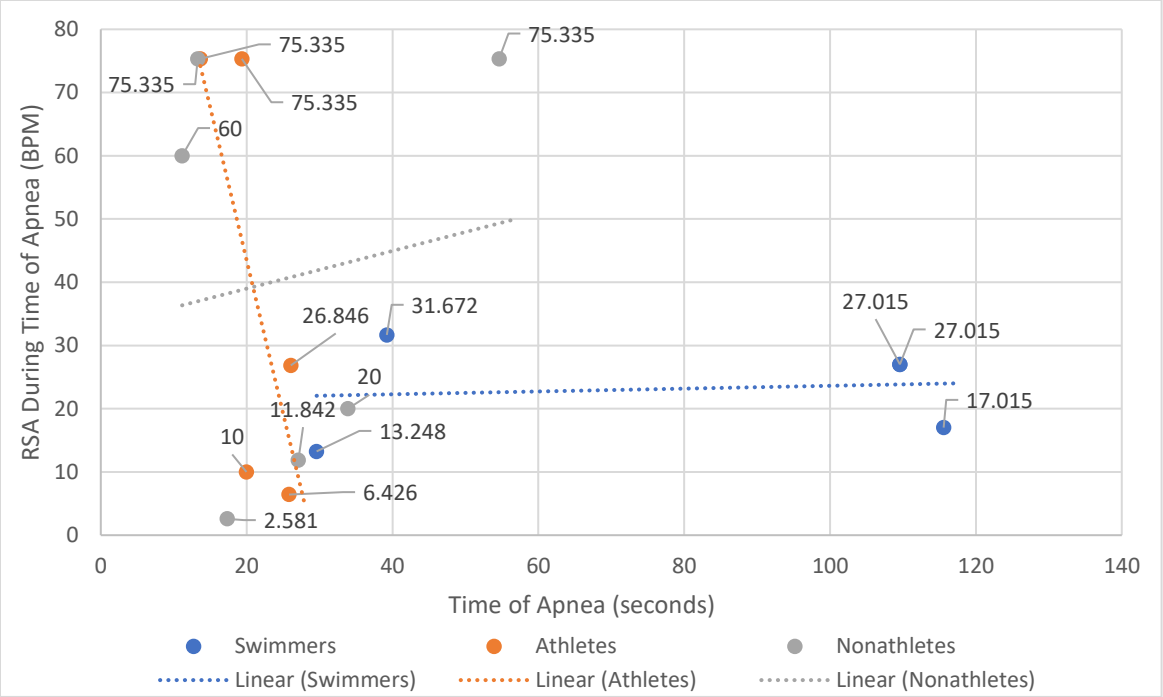


Figure 5: Time of apnea versus RSA during apnea in swimmers, athletes, and nonathletes.

In figure 5, swimmers had the longest time of apnea and were under 120 seconds. The nonathlete's time of apnea was under 60 seconds and the athlete's time of apnea was under 30 seconds. Differences among the four groups were determine using a one-way ANOVA with Duncan's Multiple Range Test for post-hoc analysis. No significant differences ($p > 0.05$) were demonstrated among the three groups. Since the data is not significant, I reject the null hypothesis.

DISCUSSION

The aim of experiment 1 was to determine the effect of breathing while at rest on the subject's heart rate and change in heart rate during respiratory sinus arrhythmia (RSA). The question I looked at was, does tidal volume cause a different RSA in swimmers, athletes, and nonathletes? I tested the hypothesis that swimmers will have a higher tidal volume and a higher RSA than athletes and nonathletes. The data in Figure 1 exhibits nonathletes had a higher tidal volume which is inconsistent with my hypothesis. Swimmers exhibited a higher RSA which is consistent with my hypothesis. Differences among the four groups were determine using a one-way ANOVA with Duncan's Multiple Range Test for post-hoc analysis. No significant differences ($p > 0.05$) were demonstrated among the three groups. Since the data is not significant, I reject the null hypothesis.

The aim of experiment 2 was to measure the effect of apnea, after maximum inhalation, on the subject's heart rate. The question I looked at was, does holding an inhalation over time cause different RSA in swimmers, athletes, and nonathletes? I tested the hypothesis that swimmers will have a higher RSA during held inhalation than athletes and nonathletes. The data in Figure 5 exhibits swimmers exhibited a higher RSA during held inhalation than athletes and nonathletes. Athletes exhibited a higher RSA than nonathletes, but a shorter time of apnea, whereas nonathletes had a long time of apnea and a smaller RSA than athletes. The data was consistent with my hypothesis. The results were expected. Again, differences among the four

groups were determine using a one-way ANOVA with Duncan's Multiple Range Test for post-hoc analysis. No significant differences ($p > 0.05$) were demonstrated among the three groups. Since the data is not significant, I reject the null hypothesis. The data provides a good starting point for discussion and further research. Further studies should investigate why athletes and swimmers are more efficient at gas exchange because of RSA.

CONCLUSION

In conclusion, respiratory sinus arrhythmia (RSA) is in which the sinus rate increases with inspiration and decreases with expiration. RSA is not an abnormal rhythm and is most commonly seen in young healthy subjects (Issa et al. 2012). RSA is a normal phenomenon wherein heart rate accelerates slightly during inhalation and decelerates during exhalation. Other studies have shown the RSA of athletes is more prominent compared to RSA in non-athletes. This study seeks to compare RSA changes in swimmers to both other athletes and non-athletes. My first hypothesis was inconsistent with my data and my second hypothesis was consistent with my data. Differences among the four groups were determine using a one-way ANOVA with Duncan's Multiple Range Test for post-hoc analysis. No significant differences ($p > 0.05$) were demonstrated among the three groups in both of my hypotheses. Since the data is not significant, I reject the null hypothesis 1 and I reject the null hypothesis 2.

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