Comparing the Acute Response of Serum Atrial Natriuretic Peptide to Intensive Anaerobic Exercise in Healthy Athlete and Non-Athlete Men

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Received: 07/17/2016 Revised: 06/20/2016 Accepted: 8/7/2016

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Abstract

Introduction: Atrial natriuretic peptide (ANP) is a type of peptide hormone released into blood in response to atrial distension. ANP has beneficial effects on decreasing hypertension and physical activity may change its secretion. The present study aimed to evaluate the effect of one-session anaerobic exercise on serum ANP in healthy athlete and non-athlete men.

Materials and Methods: Fifteen healthy athlete men and fifteen healthy non-athlete men were selected by convenience sampling. Both groups performed RAST anaerobic test; they ran 35-meter sprints with maximum speed and rested for ten seconds between each sprint. Blood samples were taken from the subjects ten minutes before and immediately after the exercise to measure serum ANP. The data were analyzed by independent t-test, dependent t-test, the analysis of covariance in SPSS-16 and the significance level was considered P ≤ 0.05.

Results: The results showed that one-session anaerobic exercise caused a significant increase in serum ANP in both groups, but the increase was significantly higher in the athlete group than the non-athlete group. The baseline values of serum ANP were also significantly higher in the athlete group than in the non-athlete group.

Conclusion: According to the results, it appears that one-session anaerobic exercise can increase serum ANP in healthy athlete and non-athlete men.

Keywords: Anaerobic Exercise, Serum ANP, Athlete, Non-Athlete

Introduction

Atrial natriuretic peptide (ANP) is a peptide hormone with 28 amino acids, which is released into the blood from atrial myocytes when the atrial wall is stretched (1). Although ANP is known as an agent regulating the blood pressure and volume, its physiological effects are more extensive and encompass the kidney (increasing urine volume and electrolytes, increasing the sodium excretion (natriuretic), increasing the glomerular filtration rate), the heart (coronary artery compliance and reducing the cardiac output), the lungs (bronchodilation and abdominal muscle cells), the central nervous system (preventing the sympathetic activity, elevating lipolysis, thermoregulation, increasing the heart rate as well as reducing the blood volume and pressure), hormones (preventing the secretion of arginine and
vasopressin, inhibiting the activity of plasma renin and catecholamines, lowering the aldosterone and cortisol levels, increasing the testosterone level, preventing the secretion of pancreatic juice and disrupting the secretion of insulin and metabolism) and vascular smooth muscle (reducing the blood pressure) (2-6).

The synthesis and level of this hormone, which is constantly secreted by the heart, are controlled by different stimuli and factors including the stretch of the atrial wall and artery walls, the increase in the body fluids and sodium concentration, sport activities, ventricular failure such as myocardial infarction, increased angiotensin-2 and endothelin (a strong vasoconstrictor) and hypertension (3, 4, 7, 8 & 9). In response to the extensive role of this hormone in regulating the body function and homeostasis, different reports are available on the effects of different levels of physical activities on the secretion of this hormone. Porst et al. found that a 22-week-long treadmill training did not affect the plasma and tissue ANP in two groups of Zucker rats (genetically obese and those with moderate hypertension) (10). Similarly, Bentzen et al. reported that a maximal aerobic exercise test did not affect plasma ANP either in healthy subjects or in those with chronic heart failure (11). Moro et al. found a 16-week aerobic exercise to increase ANP in obese women with polycystic ovary syndrome (12). Suda et al. found prolonged swimming to increase the plasma concentration of ANP in moderately hypertensive rats and to decrease it in hypertensive rats (13). Pan demonstrated that an eight-week exercise with different intensities could increase plasma ANP in rats (14). Ravasi et al. showed that a single session of endurance and speed training is associated with elevated plasma ANP in male athletes (15). Similarly, Naghizadeh and Ravasi found endurance and speed training to be associated with elevated plasma ANP in male non-athletes (16). Gharakhanlou and Habibi also found a 28-session submaximal program to enhance the plasma concentration of ANP and reduce blood pressure in middle-age males (17). Different studies conducted by Souza et al. Lipari et al. and Sajjadi et al. showed that strength training could enhance ANP in Wistar rats (3, 18 & 19). Ahmadizad et al. however found that 12 weeks of strength training did not affect the resting levels of cardiac hormones such as ANP and BNP (20).

Given that hypertension is known as an important health risk in today’s communities, doctors prescribe different pharmacological and non-pharmacological methods to treat it. An effective mechanism on blood pressure changes is associated with the role of ANP (17), and sport activities have been proposed as an elevating factor of this hormone in the body (9). Different exercises however may cause conflicting results owing to using different application methods and subjects. Literature review suggests that few studies are conducted on the effect of anaerobic exercise on this hormone. The present study was therefore conducted to investigate the effect of a single session of anaerobic exercise on serum ANP in healthy male athletes and non-athletes.

**Materials and Methods**

The statistical population of this semi-experimental study comprised all male students in Shiraz University, Iran. Convenience sampling was first used to select 15 athletes and 15 non-athletes who voluntarily and officially announced their willingness to participate in this study. The exercise time was then announced to the subjects in a briefing session. The participants were also briefed on the study objectives and methods and asked to sign an informed consent form before beginning the exercise program. The subjects’ age, height and weight were then recorded. Athletes were those who had regularly exercised over the previous six months (three two-hour sessions a week), while non-athletes were those without a history of...
Comparing the Acute Response of Serum

regular physical exercise in the same period.

**Measurement instruments**
The equipment used in the present research comprised blood sampling tools, a centrifuge for separating serum, a laboratory freezer, a chronometer, Erlenmeyer flasks, standard medical scales and stadiometers. An ELISA kit (Cusabio Company, China) was used to measure ANP levels. The enzymatic activity used in this method is the main difference from radioimmunoassay (RIA), in which radioactive materials are used. In other words, peroxidase labeled hormones are used in ELISA and radiolabeled hormones in RIA. If the reagent is present, peroxidase converts it into a colored product. Colorimetry can be used to determine the amount of the product or the labeled hormone. Standard curves are used in ELISA to determine hormone levels in plasma samples.

**The exercise protocol**
Both groups of athletes and non-athletes underwent the Running-based Anaerobic Sprint Test (RAST) and run six 35-meter distances at maximum speed while resting for 10 s between the consecutive runs.

**Blood sampling**
The blood samples of both groups, taken by a laboratory technician 10 minutes before and immediately after the exercise at 5 pm, were transferred to the specialized laboratory in Shiraz Namazi Hospital for analysis.

**Statistical methods**
The data collected were analyzed in SPSS-16 using descriptive and inferential statistics. Kolmogorov-Smirnov test was used to confirm the normal distribution of the data. The dependent t-test was used for intragroup comparison while the independent t-test and one-way ANCOVA were used for intergroup comparison. P values less than 0.05 were considered significant.

**Results**
Table 1 shows the mean and standard deviation of the subjects’ personal details and Table 2 shows the mean and standard deviation of the pretest and posttest serum ANP.

Table 3 shows the results of the dependent t-test for comparing the mean pretest and posttest serum ANP of the two groups. The results of the independent t-test indicated significant differences between the mean baseline of serum ANP in athletes and non-athletes (P≤0.004, t(28)=−3.14). One-way ANCOVA was therefore used to compare the ANP level in the two groups following a single session of anaerobic exercise and the results are shown in Table 4.

According to Table 4, the mean serum ANP is significantly different in the athletes and non-athletes following a single session of anaerobic exercise for a constant pretest (P≤0.001, F(1,27)=15.62).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Statistic</th>
<th>Athlete</th>
<th>Non-athlete</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>21.60±1.88</td>
<td>21.40±2.16</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>67.68±5.53</td>
<td>69.60±4.61</td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>174.33±5.75</td>
<td>175.66±4.76</td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>22.27</td>
<td>22.56</td>
<td></td>
</tr>
</tbody>
</table>
Table 2: The mean and standard deviation of the pretest and posttest serum ANP

<table>
<thead>
<tr>
<th>Variable</th>
<th>Athlete Before the exercise</th>
<th>Athlete After the exercise</th>
<th>Non-athlete Before the exercise</th>
<th>Non-athlete After the exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANP (pg/ml)</td>
<td>11.59±2.15</td>
<td>14.18±1.74</td>
<td>9.61±1.15</td>
<td>11.33±1.07</td>
</tr>
</tbody>
</table>

Table 3: Comparison of the mean pretest and posttest serum ANP in the group of athletes and non-athletes

<table>
<thead>
<tr>
<th>Group</th>
<th>Step</th>
<th>Number of samples</th>
<th>t value</th>
<th>P value</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Athlete</td>
<td>Pretest</td>
<td>15</td>
<td>-5.27</td>
<td>&lt;0.001</td>
<td>Significant</td>
</tr>
<tr>
<td>Non-athlete</td>
<td>Pretest</td>
<td>15</td>
<td>-9.43</td>
<td>&lt;0.001</td>
<td>Significant</td>
</tr>
</tbody>
</table>

Table 4: Comparing the results of ANCOVA for the serum ANP following a single session of anaerobic exercise in the athletes and non-athletes

<table>
<thead>
<tr>
<th>Variable</th>
<th>Statistic</th>
<th>Mean Square</th>
<th>Degree of freedom</th>
<th>F value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>The study groups</td>
<td>18.78</td>
<td>1</td>
<td>13.61</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Pretest</td>
<td>21.55</td>
<td>1</td>
<td>15.62</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>1.37</td>
<td>27</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Discussion

A single session of anaerobic exercise significantly increased serum ANP in healthy male athletes and non-athletes. These results are consistent with those obtained by Ravasi et al., Naghizadeh and Ravasi, Gharakhanlou and Habibi, Sajjadi et al., Pan and Suda et al. (13-17 & 19). The increase in the atrial pressure and secondary atrial stretch as well as in venous return and catecholamines are the mechanisms proposed to explain the increase in ANP (17). Many studies reported the sympathetic nerve stimulation and the intensity of physical activity as the stimulating factors associated with the ANP release during physical exercises (21). The increase in the intensity of activity increases plasma ANP (14), which is consistent with the present study suggesting increased ANP following the increased activity based on RAST. The increase in plasma ANP is linearly associated with the increase in the heart rate and intensity of work (15). The heart rate increase approximately explains 40% of the changes in plasma ANP (22). The mechanism of these effects might be associated with the role of ANP in the regulation of the cardiovascular system and fluids during and after the strength and speed activities. Beta-adrenergic receptors and ANP interact with the cardiovascular system regulation and metabolism during physical activities. The cardiovascular effects of ANP on vascular tone, control of renal sodium and cardiac hypertrophy can be mediated by beta-adrenergic receptors (10). ANP is synthesized in response to the stretching of the heart muscle while monitoring and lowering the blood pressure (17). Given the mechanism of respiratory and muscle pumps, physical exercise increases venous return and exerts tension on heart muscles, which in turn increases the secretion of ANP. Anaerobic exercises cause vasodilatation and increase the heart power by affecting blood vessels and the heart muscle. The conformity in the results of this type of exercises seems therefore reasonable. Vasodilatation may cause the stretch of the artery walls in speed athletes, which can elevate the release of ANP (16).
ANP also affects the regulation and transfer of fluids from the intravascular space to the extravascular space (23). The receptors of urinary peptides in sweat glands may account for the significant role of these peptides in thermoregulation (5). Higher ANP baselines were notably found to be associated with athletes compared to non-athletes. This can be explained by the presence of cardiac hypertrophy, higher heart volume, higher venous return and therefore higher pressure on myocytes and vessels in athletes (13). Based on medical physiological principles, this phenomenon is normal and rational as the purpose of physiological changes is to maintain hemostasis in normal state (24). The stretch of heart muscles caused by the increased atrial dimensions is the main stimulus for the increase in the ANP release at rest. The central blood volume is the potential cause of the atrial pressure and expansion during short term physical activities, which increase the ANP level (5). The gradual increase in the intensity of activity increases plasma ANP while moderate and high intensity activities cause the synthesis and storage of ANP in cardiomyocytes and create adaptive changes in their structure (14). Furthermore, the relationship between ANP and the troponin release can explain the transient pathological process associated with the simultaneous release of heart muscle cells caused by physical activities (21). The change in the cellular permeability caused by oxidative stress followed by the lack of proteins may explain this phenomenon although ANP and troponin originate from different parts of the heart (5).

Conclusion
Even a single session of anaerobic exercise seems to significantly increase the ANP level and it can therefore be used as a positive mechanism to maintain the state of homeostasis and control hypertension problems.

Acknowledgment
The authors would like to express their gratitude to Dr. Farhad Daryanouch, Associate Professor of Shiraz University, and all students and friends who provided selfless services and helped conclude this study.

Conflict of interest
The Authors declare that there is no conflict of interest in this paper.

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Comparing the Acute Response of Serum
Seyed Ali Naseri et al.