Response of Cardiovascular Function Determinations to Physical Activity in Polluted Air

Araz Nazari1, Nader Shavandi2, Abbas Saremi2, Fatemeh Javadzade3, Yazgaldi Nazari4* and Abuzar Mohamadimofrad5

1Department of Physical Education and Sport sciences, Higher Educational Complex of Saravan, Iran
2Department of Physical Education and Sport sciences, Arak University, Iran
3Department of Cardiology, Arak’s Medical Sciences University, Iran
4Sama Technical and Vocational Training College, Islamic Azad University, Gorgan Branch, Gorgan, Iran
5Department of Education and Sport Sciences, Shahid Chamran University of Ahvaz, Iran

Email for Correspondence: y.nazari53@gmail.com

ABSTRACT

The aim of this study was to investigate the effect of physical activity on cardiovascular function in polluted air. In this study ten healthy male university students within the age range of 21.12±1.80 were randomly selected. They have taken physical activity tests including (Bruce treadmill test, EKG, blood pressure, heart rate, and percent of oxygen saturation in fresh air (PSI=70) and polluted air (PSI=135). For data analysis, we applied dependent t-test. The findings of this study showed that physical activity in unhealthy polluted air did not affect ST segment. Unhealthy polluted air had significant effect on arterial oxygen saturation in both resting (sig=0.004) and training (sig=0.049) position. Air pollution did not affect heart rate, systolic and diastolic blood pressure in resting position; however, in training position, its effect was significant on heart rate (sig=0.009) and systolic (sig=0.001) and diastolic blood pressure (sig=0.005). It seems that physical activity in polluted air significantly affects cardiovascular function in young healthy men.

Keywords: Unhealthy polluted air, cardiovascular function, heart rate, ST-segment, systolic and diastolic blood pressure.

INTRODUCTION

Epidemiologic studies show that increase in aerosols and toxic inhalable gases in the air increases the number of referrals to hospitals, acute respiratory-cardiac conditions and mortality (Pope, 2000). As various studies showed that there is a direct relationship between respiratory diseases and air pollution. Based on statistics, 800000 people annually die throughout the world because of diseases related to air pollution (Mills et al., 2007).

On one hand, systematic physical exercises have a considerable role in general health (including the functioning of respiratory-cardiac system) (Carlisle and sharp 2001). By increasing physical activity, breathing intensity increases and more air enters lungs. This trend increases the risk of absorbing pollutants through respiration (Tarlo et al., 1990). Exploiting common exercises is suitable when these activities were done considering health conditions including environmental conditions (Carlisle and sharp 2001). Therefore, there is a hypothesis that air pollution limits beneficial effects of exercise on health (Geraint et al., 2004; Lippi et al., 2008). All research concluded that air pollutants lead to hospitalization (Brunekeefand Holgate 2002). Different combinations of air pollutants, rate and time of exposure of people to combinations of pollutants rather than a single type, has different effects on human health. The effects of air pollution include vertigo, respiration problems, skin irritation, and cancer. Other effects of air pollution are birth problems, delay in child growth, weakness of immune system and other diseases. Based on our information it
Table 1. Physical characteristic of the subjects

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Age(year)</th>
<th>Height(cm)</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>mean±standard deviation</td>
<td>21.12±1.80</td>
<td>175.71±3.77</td>
<td>54.75±20.65</td>
</tr>
</tbody>
</table>

Table 2. Comparison of cardiovascular function in polluted and fresh air

<table>
<thead>
<tr>
<th>ST-segment</th>
<th>Oxygen saturation</th>
<th>Hart rate</th>
<th>Systolic blood pressure</th>
<th>diastolic blood pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>Training</td>
<td>Rest</td>
<td>Training</td>
<td>Rest</td>
</tr>
<tr>
<td>0.075</td>
<td>0.085</td>
<td>0.004*</td>
<td>0.049*</td>
<td>0.506</td>
</tr>
<tr>
<td>0.009</td>
<td>0.356</td>
<td>0.001*</td>
<td>0.457</td>
<td>0.005*</td>
</tr>
</tbody>
</table>

*: difference is significant at p≤0.05

seems that research about physical activity in polluted air on cardiac function has not been studied. Therefore, we attempted to investigate the effect of physical activity in polluted environment on cardiovascular system.

MATERIALS AND METHODS

A total sample of 10 male students in Arak University within the age range of 18-27 years old whose characteristics are shown in Table 1, participated in the present study. They were randomly selected from the students who volunteered for the study and all procedures were in accordance with the Declaration of Helsinki. Selected students completed a medical history questionnaire and written consent form. Exercise test was conducted in Amir Kabir hospital in a clean day in Arak (Pollutant standards index (PSI=70)) and it was repeated in a dirty day (PSI=135). Pollution rate of healthy and unhealthy environments were calculated by HORIBA (made in Japan) in 24-hour mean. Also exercise tests were done according to the Bruce test on treadmill (model TMX 425, made in America). Electrocardiogram (ECG) tests were done by a cardiologist using Stress PC ECG-Version 5.0.3, made in America. To measure the percent of oxygen saturation, pulse oximetry set model OXY 500 (Type FB) made in Korea was used. To analyze the data, descriptive study (mean and standard deviation) was used for classifying data and inferential statistics including variance analysis with repeated measurement and dependent t-test were used to determine the difference between two tests and Kolomogrov-Smirnov test was also used for checking the normality of data. All statistical operations were conducted by SPSS version 16 and the significance level was defined as P<0.05.

RESULTS

Findings of the study, as shown in Table 2, indicate that physical exercises in polluted air did not have a significant effect on ST (sig=0.085) but it had a significant effect on oxygen saturation in rest (sig=0.004) and training (sig=0.049). Polluted air did not have a significant effect on heart rate, systolic and diastolic blood pressure but this difference was significant during training (sig=0.009, sig=0.001 and sig=0.005, respectively).

DISCUSSION AND CONCLUSION

Clinical exercise test is an effective method for evaluating cardiovascular system response to controlled physiologic exercise activity stress (Robergs and Roberts 2000) which is a non-invasive method (Zipes et al., 2005). There are rated exercise test protocols, which Bruce protocol is a well-known one in clinical settings (Ellestad, 1996) and it is common in studies on healthy people (Lippi et al., 2008; Flectcher and Balady, 2001; Froelicher and Myers, 2000; Gibbons et al., 2002). Bruce, Kiosomi and Hammer exercise test consists of multistep protocol which is done on rotation band. Intensity of the exercise increases by changing the velocity and slope in this protocol.

Pekkanen et al had studied 45 subjects running 72 tests for 342 times in 6 months and reported that ST reduced more than 0.1 millivolt; however, this result is not consistent with our findings (Pekkanen et al., 2002). Contrarily, in a study whose protocol was 5 minutes rest in seated condition and 5 minutes rest in standing condition and 5 minutes walking in
mild slope and 5 minutes recovery, concluded that air pollution had no effect on going from rest to exercise and from exercise to recovery. Results of this research are consistent with our study (Diane et al., 2005). Polluted air had significant effect on arterial blood oxygen saturation in rest and exercise in this study. Similarly, in consistence with our results, Pope et al observed no change in oxygen saturation using Pulseoximetry in old people (Pope et al., 1999).

Effects of 12 weeks of aerobic activity in polluted environment on oxygen saturation on 28 subjects were studied. Exercise protocol was 5 minutes rest in seated position and 5 minutes rest in standing position, 5 minutes walking in mild slope, 5 minutes recovery and 5 minutes slow breathing. They concluded that concentration of pollutants was related to the reduction of oxygen saturation but it had no effect during exercises (Demeo et al., 2004). Results of this research are consistent with our research but not consistent with physical activity research. This inconsistency can be due to difference in the environments or intensity of exercise protocol or physical fitness of participants.

In this study polluted air had no significant effect on heart rate during rest but its effect during exercise was significant. Study on old people, observed that heart rate was significantly increased (Pope et al., 1999). An increase of 100ug/m³ in aerosols in previous day increases heart rate by 10 beats per minute with 95% confidence. Results of this study were consistent with our findings.

Exposure to SO₂ increases heart rate 1.75 beats per minute, and by increasing the concentration of aerosols to about 75ug/m³, heart rate increases 1.12 beats per minute(Peters et al., 1999). Findings of this study are consistent with our results. However, another study concluded that aerosols cause a reduction in heart rate (Ibald-Mulli et al., 2004). This finding is not consistent with our study. Likewise, another study showed that exposure to diesel exhaust gases does not affect heart rate (Magnus et al., 2009). Result of this study is not consistent with our study as according to our findings, polluted air did not have a significant effect on systolic and diastolic blood pressure in rest but its effect was significant in activity. However, this inconsistency was also because of difference in type of pollutants or intensity of exercise protocol or difference in age and fitness of the participants. In the same vein, Linn et al showed that systemic blood pressure in healthy subjects decreased in confronting NO₃ (Linn et al., 1985). Results of this study were not consistent with our study. Also using non-invasive cardio-graphic impedance for cardiac output, (Drechsler- parks, 1995) has tested healthy subjects after 2 hours of exposure to NO₃ environments with 0.60ppm concentration and in O₃ environment with 0.4ppm concentration and combination of NO₂ and O₃. In comparing gases, after encountering NO₂ and O₃, cardiac output has decreased but there was no change in blood pressure (Drechsler- parks, 1995). Results of this study were not consistent with our study. Gong et.al investigated the effects of N₃ on cardiac output in which men with and without hypertension exercise 3 hours, with cardiovascular catheter in an environment in which concentration of O₃ was 0.3ppm. They observed that there is no difference in the index of cardiac functions but heart rate, rate pressure product and Alveolar oxygen pressure gradient was higher in exposure to O₃. These effects are more pronounced in those with coronary diseases (Gong et al., 1998). Results of this study were consistent with our findings. Similarity, with 1ug/m³ increase in particulate matter (PM), systolic and diastolic blood pressures increased 0.172 and 0.095mmHg, respectively. PM₉₀ was related to blood pressure but this relation was significant in PM₂₅ (Linn et al., 1999). Results of this study were consistent with our study but this increase was not significant.

ST segment depression in EKG in exercise test showed myocardial ischemia (Gibbons et al., 1997). It was main abnormality during exercise. When enough blood did not reach the cardiac muscle, subendocardium is involved. At the beginning of ischemia, the reduction in oxygen changes the natural functioning of cells due to ST depression. ST segment depression because of exercise is usually expressed as ischemia in subendocardium (Robergs and Roberts 2000). Air pollutants reduce oxygen availability for myocardial muscle or increase its need to oxygen or both (Pekkanen et al., 2002). Blood flow in coronary system is regulated by the vasodilation of coronary vessels. When strength of myocardial muscle increases, coronary blood flow increases too. In contrast, reducing activity is consistent with lower coronary blood flow. Blood flow in coronary arteries is regulated by myocardial muscle’s need for energy. In rest position 70% of oxygen in coronary artery is taken from the blood passing heart. Because there is no oxygen, there is no possibility to increase oxygen unless through increasing the coronary blood flow. This coronary blood flow increases according to metabolic oxygen use in heart (Guyton and hall 2006).

It is not clear yet that how an increase in oxygen consumption dilates coronary arteries. Many researchers believe that reducing oxygen in heart releases dilation materials from heart cells. Adenosine is a material with high dilation activity. If oxygen concentration reduces, adenosine transforms to Adenosine mono phosphate. Then it changes to Adenosine which releases into cell fluids in cardiac muscles. High amount of adenosine is absorbed in heart cells after dilation for reuse. Adenosine is not the only vasodilator and materials like adenosine phosphate, K ions, hydrogen ions, carbon dioxide, Bradykinin and prostaglandins have this property. However, dilation hypothesis has problems. First, materials that neutralize the effects of adenosine cannot prevent coronary dilation in response to increasing myocardial activity. Second, studies on skeleton muscles showed that continuous adenosine injection maintains dilation for 1 to 3 hours, while after adenosine effect cardiac muscle can dilate blood arteries (Guyton and hall 2006). Therefore, we can conclude that this pollution range on ST has no effect during activity and heart meets its need for oxygen by increasing coronary blood flow. Reduction in oxygen saturation indicates oxidative stress which leads to neural feedback or
changes cardiovascular physiology through lungs and artery inflammation. Inflammatory factors and cytokines produced facing pollutants can influence autonomic nerves activities through lung vagus nerve stimulation (Demeo et al., 2004).

Reduction in oxygen saturation may decrease oxygen supply for active tissues and influence them. Results showed that polluted air may reduce artery oxygen saturation. Research shows that air pollution is related to an increase in C-reactive protein (Peters et al., 2001), fibrinogen (Schwartz, 2001) and plasma viscosity (Peters et al., 1997). Reduction in oxygen saturation indicates oxidative stress which leads to neural feedback in heart or changes cardiovascular physiology through lungs inflammation. Inflammatory factors and cytokines produced in exposure to pollutants can influence autonomic nerves activities through lung vagus nerve stimulation (Peters et al., 1998; Pope et al., 1999) that increases mortality (Liao et al., 1999).

Potential mechanism caused in encountering PM is inflammation by producing reactive oxygen species (ROS) and oxidative stress (Finkelstein et al., 2002; Whitekus et al., 2002). Evidence shows that ROS has an important role in damage mechanism. It is proved that PM and exhaust gases produce ROS in target cells like macrophages, epithelial and endothelial cells (Li et al., 2002; Edgar et al., 2004). These studies consider effects of physical activity in polluted air and showed that physical activity in polluted air reduces cardiovascular function. Therefore, regarding the importance and role of physical activities in maintaining health, reducing fatness, and show increases in cardiovascular function, it is necessary to use the benefits of physical exercises.

REFERENCES


Magnus L., Nicholas LM., Andrew L., Stefan B., Ken D and David EN (2009). Experimental exposure to diesel exhaust increases arterial stiffness in man; Particle and FibreToxicology. 6:7.


