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REGULAR ARTICLE

Comparison of heart rate variability and cardiac arrhythmias in polluted and clean air episodes in healthy individuals

Gholamreza Davoodi · Ahmad Yamini Sharif · Ali Kazemisaeid · Saeed Sadeghian · Ali Vasheghani Farahani · Mehrdad Sheikhvatan · Mina Pashang

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Abstract
Objective Pathophysiological mechanisms and pathways linking cardiovascular mortality and morbidity with air pollution were recently hypothesized. The present study evaluated association between air pollution and changes in heart rate variability as a marker of cardiac autonomic function in healthy individuals, and also determined the frequency of cardiac arrhythmias and QT interval changes on polluted compared to unpolluted days.
Methods Continuous Holter electrocardiography (ECG) monitoring was conducted on 21 young healthy individuals in the two episodes of clean air and elevated air pollution in Tehran. All subjects underwent a medical history review, a physical examination and echocardiography in order to rule out structural heart diseases. Measured pollutants and parameters included NO_2, CO_2, O_3, SO_2, and PM10, which all showed significantly higher concentrations on polluted days. Holter parameters were measured for 24-h time segments and compared.
Results Maximum heart rate was significantly lower in polluted air conditions in comparison with clean air conditions (115.1 ± 32.2 vs. 128.9 ± 17.7), and the square root of the mean of squared differences between adjacent NN intervals (r-MSSD) was higher in polluted air compared to clean air (99.0 ± 58.2 vs. 58.5 ± 26.4). Also, the occurrence of nonsustained supraventricular tachycardia was reported in 42.9% of participants in air pollution episodes, whereas this arrhythmia was not seen in clear air conditions ($p = 0.001$).
Conclusion Changes in air pollution indices may lead to the occurrence of nonsustained supraventricular tachycardia, a slight reduction in maximum heart rate, and an increase in r-MSSD in healthy individuals. Air quality monitoring in cities associated with a high exposure to air pollutants is recommended in order to prevent such events.

Keywords Air pollution · Heart rate variability · Cardiac arrhythmia

G. Davoodi (✉) · A. Y. Sharif · A. Kazemisaeid · S. Sadeghian · A. V. Farahani · M. Sheikhvatan · M. Pashang
Tehran Heart Center, Tehran University of Medical Sciences, North Kargar Street, Tehran 1411713138, Iran
e-mail: ghdavoodi@yahoo.com; msv_swt@yahoo.com

Introduction

An association between exposure to air particulates and adverse cardiac events due to alterations in cardiac autonomic function such as decreased vagal or increased sympathetic tone has been confirmed in most of the recent studies in this field [1–5]. It seems that cardiopulmonary irritability associated with particulate air pollution may be induced through the sympathetic stress response or via inflammatory processes such as chemokine and cytokine production in respiratory or cardiac circulation [6–8]. It was also shown that the increased risk of cardiovascular mortality and morbidity following exposure to airborne particles can be caused by the progression of heart failure [9].

However, recent studies on the effect of community air pollution on the cardiac systems of healthy individuals have produced conflicting results. Some research has suggested a direct influence of air pollution on health measurements in healthy subjects, such as heart rate elevation and an increase in systolic blood pressure [10]. Some results provide evidence that an increased level of air pollution may lead to detectable adverse effects on the cardiovascular systems of healthy young individuals, such as constriction of blood vessels [11].

The present study evaluated the association between air pollution and changes in heart rate variability as a marker of cardiac autonomic dysfunction in healthy individuals, and also determined the frequency of cardiac arrhythmias and QT interval changes during polluted compared to unpolluted days in Tehran, a city associated with high exposure to air pollutants.

Methods

A single-season prospective study was conducted on 21 young healthy volunteer staff of a university (aged 30.0 ± 3.9 years; male:female ratio 12:9). Their work place and homes were in a predefined area in Tehran, and they all lived and moved in the region where air pollutants were measured during the study period. The individuals all performed the same level of physical activity during the 2 days of study in November and December 2008. They did not change their daily program during those 2 days, and their program was limited to attending their work place and ordinary daily activities. They did not perform vigorous exercise. Moreover, they did not travel outside this region during the days of the study.

A self-administered questionnaire was used to collect information on medical history, including on their cardiac systems and their use of medications. All subjects underwent physical examination and echocardiography in order to rule out structural heart diseases. Meanwhile, all individuals with a chief complaint of palpitation, chest pain, or a feeling of frequent extra beats were excluded from participation. All subjects with a history of smoking, medications that might affect cardiac rhythm, coronary artery disease, hypertension, diabetes mellitus, and dyslipidemia were also excluded. The Institutional Review Board of the Tehran University of Medical Sciences approved the study, and written informed consent was obtained from all participants.

Subjects were scheduled to undergo a day of ECG Holter monitoring during two episodes of clean air and elevated air pollution by trained technicians in order to assess heart rate variability (on the basis of established standards of the European Society of Cardiology and the American Society of Pacing and Electrophysiology [12], as well as QT intervals and both ventricular and supraventricular arrhythmias. Continuous Holter monitoring was performed after a 15 min rest for each participant in the sitting position using a BioMed Sciences three-channel device. Cardiac electrophysiologists unaware of the setting at which the recordings were taken reviewed the recordings and cleared the data. A complete 24-h time segment of normal-to-normal interval was taken for heart rate variability analysis, including measuring the heart rate, deriving the standard deviation of normal RR intervals (SDNN, ms), the SDNN index (SDNNs, ms), the standard deviation of the means of all corrected RR intervals calculated at 5 min intervals (SDANN, ms), the square root of the mean squared differences between successive RR intervals (r-MSSD), the percentage of the adjacent normal-to-normal intervals that differed by more than 50 ms (pNN_{50}, %), and the HRV triangular index (TRIA, ms). Total numbers of PACs, PVCs, and also the number of episodes of SVT or VT were calculated. QT intervals (QT-max, QT-min, and QT_c) were also measured. Ambient levels of O_3 (ozone), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and carbon dioxide (CO_2) and particulate matter less than 10 μm in size (PM10) were obtained from an automated monitoring station during the two episodes of clean air and elevated air pollution in Tehran. For the episode of elevated air pollution, we selected the day with the highest air pollution based on the predictions of the Iran Meteorological Organization. As shown in Table 1, the means of all measured pollutants and parameters were higher on polluted days.

Table 1 Pollutants measured during the two episodes of clean air and elevated air pollution

Air pollutants	Clean air	Polluted air
Carbon dioxide (μg/m^3)	2.71	3.98
Nitrogen dioxide (μg/m^3)	75.74	149.38
Ozone (μg/m^3)	9.15	21.79
Sulfur dioxide (μg/m^3)	27.38	159.90
PM10 (μg/m^3)	38.82	170.70

PM10 particulate matter less than 10 μm in size

Results were expressed as mean ± SD for quantitative variables and percentages for categorical variables. Categorical variables between the two groups were compared using the chi-square test or Fisher's exact test. Continuous variables were compared using the paired *t* test. The Wilcoxon test was used to check for significant differences between the two groups in variables that did not present normal distributions according to the Kolmogorov–Smirnov test. Multivariable logistic regression analysis was performed to determine odds ratios (corrected for confounding factors: sex and age) for the appearance of supraventricular tachycardia during polluted compared to clean air episodes. Two-tailed *p*-values were obtained, with 0.05 taken to be nominally significant.The statistical software SPSS version 13.0 for Windows (SPSS Inc., Chicago, IL, USA) was used for statistical analysis.

Results

Results of the air pollutant measurements performed during the study are shown in Table 2. Regarding basic

Table 2 Basic rhythms in the two episodes of clean air and elevated air pollution

Observed arrhythmia	Clean air ($n = 21$)	Polluted air ($n = 21$)	p-value
Basic rhythm			
Total beats (×1000)	102.6 ± 17.0	100.7 ± 12.7	0.291
Average heart rate (min)	76.2 ± 8.0	75.4 ± 7.5	0.677
Minimum heart rate (min)	48.5 ± 7.0	51.7 ± 8.4	0.231
Maximum heart rate (min)	128.9 ± 17.7	115.1 ± 32.2	0.003
Heart rate variability			
SDNN (ms)	153.1 ± 26.4	190.1 ± 98.6	0.263
SDNNs (ms)	72.5 ± 17.2	110.2 ± 78.7	0.089
pNN_{50} (%)	16.0 ± 10.5	17.1 ± 9.2	0.420
TRIA (ms)	648.0 ± 149.8	624.5 ± 124.4	0.326
SDANN (ms)	136.0 ± 29.5	157.2 ± 97.0	0.940
r-MSSD (ms)	58.5 ± 26.4	99.0 ± 58.2	0.019
NHR (min)	63.0 ± 15.7	68.9 ± 10.6	0.121
QT-min (ms)	250.5 ± 27.8	251.4 ± 22.8	0.920
QT-max (ms)	443.5 ± 29.8	446.6 ± 40.0	0.999
QT_c (ms)	419.1 ± 25.2	426.2 ± 22.4	0.529

SDNN standard deviation of normal RR intervals, *SDNNs* standard deviation of all normal-to-normal intervals, pNN_{50} percentage of the adjacent normal-to-normal intervals that differed by more than 50 ms, *TRIA* heart rate variability triangular index, *SDANN* standard deviation of the means of all corrected RR intervals calculated at 5 min intervals, *r-MSSD* root mean square of successive RR interval differences, *NHR* normalized heart rate, *QT-min* minimum of QT interval, *QT-max* maximum of QT interval, QT_c corrected QT interval

Table 3 Ventricular and supraventricular ectopies during the two episodes of clean air and elevated air pollution

Holter parameters	Clean air ($n = 21$)	Polluted air ($n = 21$)	p-value
Premature atrial complexes			
≤200 beats/24 h	20 (95.2%)	17 (80.9%)	0.343
>200 beats/24 h	1 (4.8%)	4 (9.1%)	
Premature ventricular complexes			
≤200 beats/24 h	19 (90.5%)	20 (95.2%)	0.549
>200 beats/24 h	2 (9.5%)	1 (4.8%)	
Nonsustained supraventricular tachycardia	0	9 (42.9%)	0.001

rhythm, no significant differences were found in terms of mean total beats and mean minimum heart rate between studied individuals during the two episodes of clean air and elevated air pollution; however, the maximum heart rate was reduced in polluted air in comparison with clean air (115.1 ± 32.2 vs. 128.9 ± 17.7, $p = 0.003$). QT intervals (QT-min, QT-max, and QT_c) were not different between the two episodes. With regard to descriptive statistics for heart rate variability measurements, a significant elevation in r-MSSD was observed during the episode of elevated air pollution compared to the episode of clean air (99.0 ± 58.2 vs. 58.5 ± 26.4, $p = 0.019$). However, other parameters were similar between the two conditions.

Regarding ventricular and supraventricular ectopic rhythm (Table 3), there were no significant differences in the number of premature atrial complexes (PAC_s) and premature ventricular complexes (PVC_s) between the two episodes. Participants showed no evidence of nonsustained supraventricular tachycardia (SVT) during the clean air episode, but this arrhythmia was reported in 9 out of 21 individuals under polluted air conditions ($p = 0.001$). Multivariate logistic regression analysis showed that the episode of air pollution was the main determinant of the appearance of supraventricular tachycardia in healthy individuals (OR = 20.370, 95% CI: 1.843–225.166, $p = 0.014$) (Table 4).

Discussion

The current study tried to assess the influence of air pollutants on heart rate variability, cardiac arrhythmia and QT intervals, and had some notable findings. Few studies have

Table 4 Relationship between the appearance of supraventricular tachycardia in healthy individuals and air pollution in the presence of confounders

Variable	Multivariate *p*-value	Odds ratio	95% confidence interval
Male gender	0.087	8.289	0.735–93.503
Advanced age	0.172	0.790	0.564–1.108
Polluted episode	0.014	20.370	1.843–225.166

Hosmer–Lemeshow goodness of fit test, $\chi^2 = 3.323$, $df = 8$, $p = 0.912$

been published about the influence of exposure to ambient air particles on heart rate variability in young healthy individuals, although some similar studies have considered the influence of fine airborne particulate matter on heart rate variability in special subgroups of healthy workers [1, 13]. We were able to show a reduction in the recorded maximum heart rate during days with air pollution in healthy individuals. This finding has also been confirmed in some other recent studies on human subjects and animals. In a study by Mar et al. [10], a direct association between health measurements and air pollution was found in the elderly: healthy subjects taking no medications exhibited decreases in heart rate, while those on cardiovascular medications (antihypertensives, beta-blockers, calcium channel blockers, and cardiac glycosides) experienced increased systolic blood pressure. However, contrary to our study, some other studies have shown a negative relationship between heart rate and pollution of the air with particulate matter [14]. Animal studies have also shown different results. Although Chang et al. [15] reported an increase in heart rate in rats exposed to concentrated particulate matter, Tankersley [16] and Campen [17] found that air pollutant exposure could be associated with a reduced heart rate in rats compared with the heart rate during clean air exposure. It seems that traffic-related particles can affect autonomic function in healthy subjects, especially those traveling a great deal in the city. This autonomic alteration may be mediated by an enhanced decline in parasympathetic control of the heart, as indicated by the changes observed in heart rate. It has been demonstrated that alterations in parasympathetic and sympathetic balance can result in electrical instability, cardiac arrhythmias, as well as increased arrhythmic death. Therefore, these effects of air pollutants on heart rate variability (such as maximum heart rate reduction) should be considered.

Among all of the parameters of heart rate variability considered in our study, only an increase in r-MSSD was found to be significant during the episode of elevated air pollution compared to clean air conditions. Similarly, Whitsel et al. [18] found a strong ambient particulate matter–heart rate variability association for r-MSSD. Overall, other previously published findings suggested a change in heart rate variability measured as r-MSSD that was associated with particulate air pollution in healthy elderly subjects [19, 20]. Although heart rate variability analysis is a powerful and sensitive method for early detection of autonomic dysfunction, among all of the measurable indices of heart rate variability, r-MSSD was reported to have the lowest positive predictive accuracy for risk stratification and prediction of arrhythmic events in post-acute MI patients [21]. It was also indicated that, among these indicators, SDNN was the most efficient index for risk stratification, and that it allowed practical identification of patients with the highest likelihood of arrhythmic events [22]. Others suggested that only SDNN and SDANN could predict ventricular tachycardia on Holter monitoring of patients with heart failure [23, 24]. Therefore, r-MSSD may not be a sufficiently strong index to show the effects of air pollution on heart rate variability in healthy subjects. Thus, one can conclude that, since the main indicators and most sensitive indices of heart rate variability gave comparable results during the episode of air pollution and during the episode of clear air, it seems that short-term exposure of healthy individuals to air-based pollutants does not affect these indicators.

Our study, to the best of our knowledge, was the first report to show a notable increase in the prevalence of nonsustained supraventricular tachycardia triggered by air pollution in healthy individuals. Similar results have previously been shown in patients with underlying coronary artery disease [25]. Dockery et al. [26] found increased risks of supraventricular arrhythmia associated with two-day exposure to all of the air pollutants considered. It was also suggested that increased levels of ambient sulfate and ozone might increase the risk of supraventricular arrhythmia, especially in the elderly [27]. It seems that the associations of supraventricular tachycardia triggered by air pollution with fine particle mass, carbon monoxide, nitrogen dioxide, and black carbon suggest a link with motor vehicle pollutants, and the association with sulfate are linked to stationary fossil fuel combustion sources. This can induce autonomic nervous system dysfunction or inflammation, which can affect cardiac rate and rhythm and increase the risk of cardiovascular morbidity and even mortality.

The current study has some limitations. Because only a limited number of Holter monitoring recorders were available during the study, and since we had to perform the study on the same date for all of the participants, the number of participants was limited.

In summary, regarding the impact of air pollutants on heart rate variability in healthy individuals, air pollution does not affect cardiac autonomic function indicators except for r-MSSD, which increases, and maximum heart

rate, which reduces following air pollutant exposure. Changes in air pollution indices may also lead to the occurrence of nonsustained supraventricular tachycardia. Considering the potential effects of air pollution on arrhythmic events, appropriate air quality monitoring in cities associated with high exposure to air pollutants is recommended in order to prevent these events.

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