



Acute Effects of Smoking on Diastolic and Systolic Cardiac Function of Healthy Smokers according to 2D Speckle Tracking Echocardiography

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ABSTRACT

Background: Smoking is a preventable cause of human death. While the chronic effects of smoking on cardiovascular health are well known, its acute effects remain obscure.

Objectives: This study aimed to assess the acute effects of smoking on left ventricle (LV) diastolic and systolic cardiac function and right ventricle (RV) systolic function in healthy smokers.

Methods: In this before-after (quasi-experimental) study conducted at Modarres and Shohada-e-Tajrish hospitals in Tehran, Iran, healthy smokers were asked to discontinue smoking and caffeine for half an hour. Then, they underwent echocardiography by the same echo-cardiologist at baseline and 10 minutes after smoking a single cigarette with 10 mg of nicotine. The 2D speckle tracking mode illustrated the diastolic and systolic ventricular functions.

Results: Among 80 participants were 54 males (67.5%) and 26 females (32.5%). The mean age was 36.8 ± 13.6 years. The mean smoking history was 7.3 ± 7.5 pack years. Significant changes occurred after smoking in the global longitudinal strain (GLS), E/A ratio, and pulmonary artery systolic pressure ($P = 0.001$). No changes occurred in other factors including ejection fraction (EF), S wave velocity of septum, tricuspid annular plane systolic excursion (TAPSE), and right ventricular systolic function (SRV) ($P > 0.05$).

Conclusions: This study was carried out as a pilot for larger clinical studies to demonstrate the acute effects of smoking on heart function and cardiovascular health. Our findings indicate that smoking acutely affects both right and left ventricular parameters on echocardiography.

1. Background

According to the World Health Organization, four million deaths occur annually due to tobacco smoking worldwide (1). This figure is projected to rise to ten million in the upcoming 25 years, 70% of which would be in developing countries (1, 2). Over one billion smokers live worldwide, most of them (80%) residing in developing countries (2). Smoking is a preventable cause of human death, and modification in social habits can help to decrease smoking-related mortality (1, 2). Increased smoking, especially among young female subjects, is alarming (2). Smoking threatens the health of both smokers and secondhand smokers, with an increased rate of cardiovascular disorders and lung cancer (3).

Oxidants like nicotine found in cigarettes cause various changes in the body systems (4). While the chronic effects of smoking on cardiovascular health (e.g., atherosclerosis, coronary artery disease, and heart failure) are well recognized, there is little information about its acute effects. Acutely, smoking may cause QT prolongation, leading to an increased risk of atrial fibrillation and death (5). The more commonly described acute effects of smoking include hypertension, tachycardia, and increased cardiac function (6, 7).

Global longitudinal strain (GLS) is an echocardiography parameter that can predict and detect subclinical cardiac dysfunction (8).

2. Objectives

this study assessed the acute effects of smoking on diastolic and systolic cardiac function using 2D speckle

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tracking echocardiography and RV systolic function with echocardiography.

3. Methods

In this before-after (quasi-experimental) study conducted at the Modarres and Shohada-e-Tajrish hospitals (Tehran, Iran), the demographic data of healthy smokers were collected, and echocardiography with 2D speckle tracking mode was done. The inclusion criteria were healthy smokers who volunteered to participate in the study. The exclusion criteria were a known history of coronary artery disease, pulmonary failure, renal failure, or the use of cardiac drugs. Also, as we aimed to assess the acute effect of ordinary cigarette smoking, all participants smoking other tobacco products, including electronic cigarettes and heated tobacco product devices, were excluded to eliminate possible interactions.

This study was approved by the Ethics Committee of Shahid Beheshti University of Medical Sciences (IR.SBMU.MSP.REC.1397.602). No additional costs were imposed on the subjects. The Helsinki Declaration was respected across the study, and informed consent was obtained. Healthy smokers were asked to discontinue smoking and caffeine use for half an hour. Patients underwent echocardiography at baseline. Then, 10 minutes after smoking a single cigarette containing 10 mg of nicotine, the echocardiography was repeated by the same expert echo-cardiologist using the same device (Philips Affinity 50). The 2D speckle tracking mode illustrated the diastolic and systolic ventricular functions.

Participants' ejection fraction (EF), as an index of the systolic function of the left ventricle, was measured by the modified Simpson's method recommended by The American Society of Echocardiography. In this biplane method of disks, the left ventricle is considered the sum of three sections including a cylinder from the base of the heart to the mitral valve, a truncated cone from the level of the mitral valve to the level of the papillary muscles, and another cone attributed to the cardiac apex. Therefore, area tracings of the LV cavity are required. The LV cavity is divided into a predetermined number of disks (usually 20) before the endocardial border in both the apical four-chamber and two-chamber views in end-systole and end-diastole is traced. This modality directly measures the contribution of longitudinal contraction. Since the entire tracing of the LV cavity border is not done, the LVEF is calculated following some geometric assumptions (9).

Global longitudinal strain (GLS) is a myocardial deformation analysis that can demonstrate the abnormal contraction in the setting of apparently normal LVEF because it shows the function of sub-endocardial longitudinally oriented fibers prone to wall stress and ischemic damage. For the measurement of GLS, the three apical long-axis views are considered after dividing the LV into 20 segments covering the entire left ventricular myocardium. From each of the three views, the mean of the global peak systolic strain is used to automatically calculate the GLS (normal value: ~20%). The peak systolic strain was determined using the Bull's eye plot, where normal systolic shortening was illustrated in red color (10).

For differentiating pseudo-normal from normal diastolic

filling patterns, tissue Doppler imaging (TDI) was used, which, in comparison to the standard Doppler methods, it is more sensitive to diastolic function and less load-dependent. This method estimates longitudinal systolic and diastolic ventricular performance by assessing the myocardial tissue velocities. In the apical four-chamber view, after aligning the Doppler beam parallel to the direction of the maximum annular motion at the septal and lateral mitral annulus (LV free wall), the peak systolic (S'), peak early diastolic (E'), and peak late diastolic (A') velocities were measured. Then, the E/A wave ratio was calculated (11).

Tricuspid annular plane systolic excursion (TAPSE) and the tissue Doppler systolic velocity of the tricuspid annulus were measured to assess RV function. TAPSE is a two-dimensional measure of systolic RV function. It is determined in the apical four-chamber view. The M-mode cursor is placed through the lateral portion of the tricuspid valve annulus. Then, the distance traveled between end-diastole and end-systole at the lateral corner of the tricuspid annulus is measured by the excursion of the tricuspid valve from the base of the heart toward the apex. As TAPSE is measured in one dimension and cannot always reflect global RV function (12, 13), we also assessed the tissue Doppler systolic velocity of the tricuspid annulus as an index of longitudinal RV function (13, 14). Furthermore, the pulmonary artery systolic pressure (PASP) was calculated using the tricuspid regurgitation (TR) peak velocity obtained by continuous wave Doppler tracing. According to the simplified Bernoulli equation ($P = 4[TR_{max}]^2$), the PASP was obtained (15).

Demographic and clinical variables were recorded using checklists. Analysis was done by SPSS software version 22.0. The statistical tests included the paired sample t-test and the Kolmogorov-Smirnov test. P-values less than 0.05 were considered statistically significant.

4. Results

Among 80 subjects were 54 males (67.5%) and 26 females (32.5%). The mean age was 36.8 ± 13.6 years. The mean smoking history was 7.3 ± 7.5 pack-years. In the sub-analysis, the mean age was 37.4 ± 14.1 and 36.2 ± 12.9 years in male and female subjects, respectively. The mean smoking history was 9 ± 5 and 6 ± 2 pack years in males and females, respectively. As shown in Figure 1, the GLS was significantly altered after smoking ($P = 0.001$), changing from -20.68 ± 1.31 to -19.24 ± 2.14 .

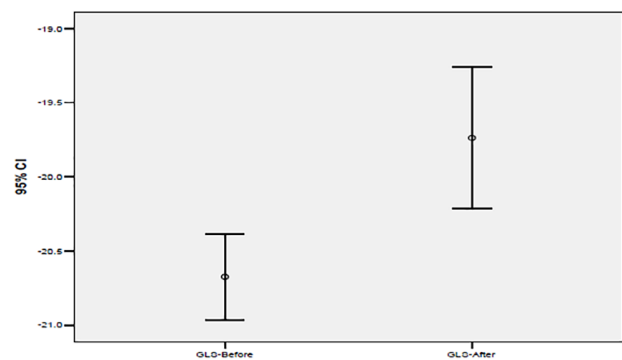


Figure 1. Comparison of Global Longitudinal Strain (GLS) before and after Smoking.

Table 1. Indices Measured before and after Smoking

Marker	Meaning/Method	Before	After	Interpretation
GLS	Subclinical LV systolic function	-20.68 ± 1.31	-19.24 ± 2.14	Decreased significantly (P = 0.001)
LVEF	Clinical LV systolic function by Simpson's method	58.50 ± 5.01	58.01 ± 3.42	Decreased insignificantly (P > 0.05)
SV	LV systolic function by TDI method	8.14 ± 0.76	8.01 ± 0.77	Decreased insignificantly (P > 0.05)
E/A ratio	LV diastolic function by TDI method	1.08 ± 0.30	0.92 ± 0.27	Decreased significantly (P = 0.001)
TAPSE	RV systolic function by M Mode	2.06 ± 0.36	1.92 ± 0.32	Decreased insignificantly (P > 0.05)
SRV	RV systolic function by TDI method	12.61 ± 1.50	12.26 ± 1.59	Decreased insignificantly (P > 0.05)
PASP	Systolic pressure of pulmonary artery	23.29 ± 5.34	27.2 ± 6.44	Increased significantly (P = 0.001)

Abbreviations: GLS; global longitudinal strain, LVEF; left ventricle ejection fraction, SV; peak systolic velocity of Septum, TAPSE; tricuspid annular plane systolic excursion, SRV; RV systolic function, PASP; pulmonary artery systolic pressure

As shown in Table 1, the ejection fraction (EF), tricuspid annular plane systolic excursion (TAPSE), the peak systolic velocity of the septum (SV), and RV systolic function (SRV) did not show any significant alterations after smoking (P > 0.05). However, the PASP and E/A ratio changes significantly (P = 0.001).

As shown in Figure 2, in the subanalysis for sex, the alterations in E/A ratio, PASP, and GLS were significant in both men and women, but differences in women were more significant in PASP and GLS (P = 0.001).

As shown in Figure 3, in the subanalysis for smoking intensity, while changes were significant in both groups, alterations in PASP and E/A ratio were more prominent in patients with under 10 pack-years of smoking history (P = 0.001).

5. Discussion

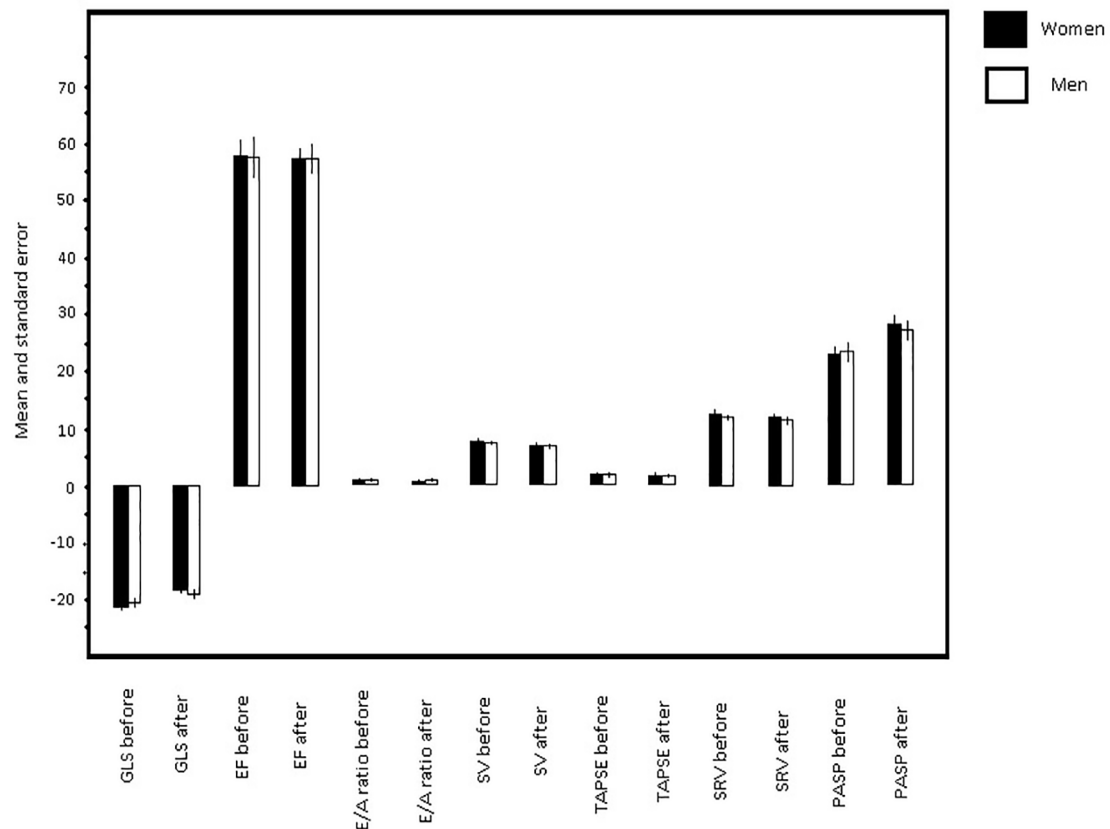
This before-after study demonstrated that smoking has an

acute effect on cardiac function, reflected by alterations in the GLS (the most sensitive index) and other indices such as TAPSE, EF, and PASP.

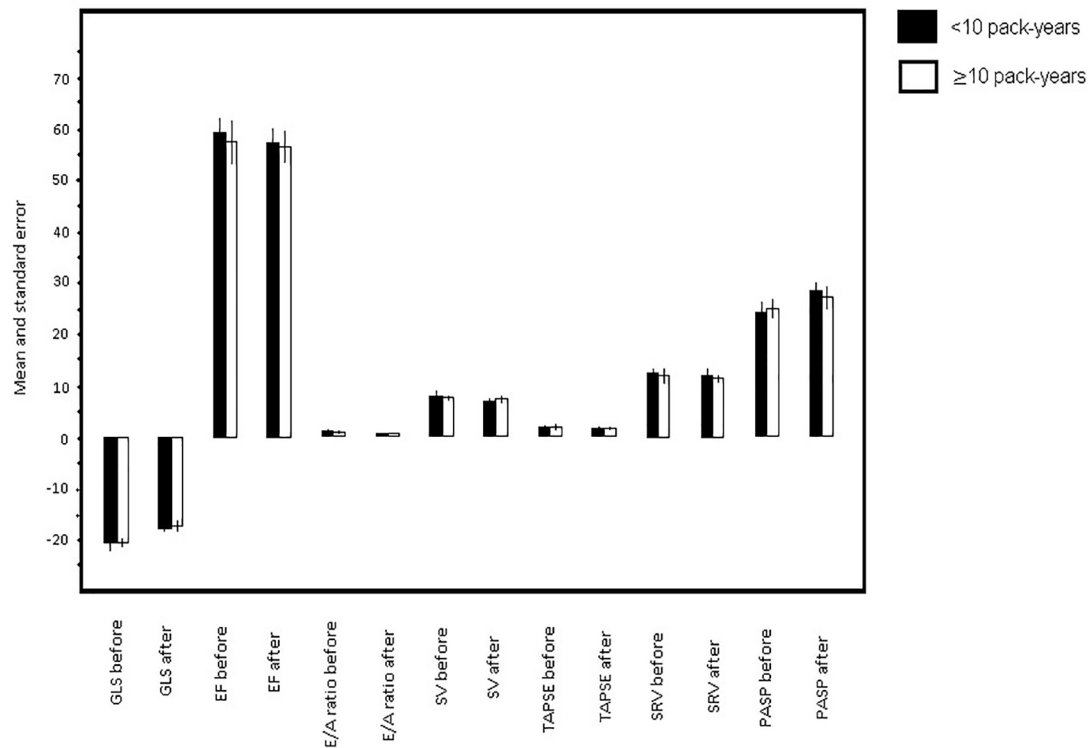
The clinical trial by Karakaya et al. (16) among 15 smokers showed that smoking led to increased heart rate variability, especially in the first 5 to 10 minutes after smoking, accompanied by a decreased R-R interval. Alshehri et al. (17) reported diastolic ventricular dysfunction with 0.4 mg of nicotine during smoking. Ramakrishnan et al. (18) reported increased heart rate from 84 to 91 beats after smoking, and ectopic beats were raised from 5 to 10. This may explain the cause of echocardiographic changes in our study. Lichodziejewska and colleagues (19) reported ventricular dysfunction after smoking, aligning with our findings.

Ilgenli et al. (20) reported right and left ventricular dysfunction and increased pulmonary artery pressure, which normalized after thirty minutes. However, we had no final assessment to see when the tests normalized. Barutcu

Figure 2. Comparison of Changes in Echocardiographic Parameters after Smoking in Males and Females.



Abbreviations: GLS; global longitudinal strain, LVEF; left ventricle ejection fraction, SV; peak systolic velocity of Septum, TAPSE; tricuspid annular plane systolic excursion, SRV; RV systolic function, PASP; pulmonary artery systolic pressure.

Figure 3. Comparison of Changes in Echocardiographic Parameters after Smoking according to the Pack Years of Smoking.

Abbreviations: GLS; global longitudinal strain, LVEF; left ventricle ejection fraction, SV; peak systolic velocity of Septum, TAPSE; tricuspid annular plane systolic excursion, SRV; RV systolic function, PASP; pulmonary artery systolic pressure.

et al. (21) found systolic dysfunction after 30 minutes of smoking, reflected by changes in mitral and tricuspid lateral annulus diastolic velocities. This matter is co with our findings. Alam et al. (22) reported left ventricular diastolic dysfunction within 30 minutes of smoking, reflected by a decrease in the trans-mitral E/A ratio. Similarly, our results showed a significant decrease in the E/A ratio.

Giacomin et al. (23) reported decreased TAPSE and increased mitral Aa and tricuspid Sa, while our results failed to show any significant decrease in TAPSE. Mrkaić et al. (24) showed decreased systolic filling and decreased diameters of blood vessels, which may partially explain the cause of our findings. Also, Khosropanah et al. (25) showed that smoking even a single cigarette in both smokers and non-smokers can lead to a rise in the QT interval. Electrocardiogram (ECG) findings may be interesting for future studies. Karakaya et al. (26) showed that acute cigarette smoking might impair smokers' right and left ventricular diastolic function, similar to our research. In another study, Belma Yaman et al. assessed the systolic and diastolic ventricular function by two-dimensional speckle tracking echocardiography following both cigarette and heated tobacco product smoking in the same participants, but each one on different days; they finally reported a significant decrease in GLS (27). In our study, as we aimed to assess the acute effect of ordinary cigarette smoking, all participants smoking other tobacco products, including electronic cigarettes and heated tobacco product devices, were excluded to eliminate their possible interactions. Nevertheless, a significant LV GLS reduction was also observed in our study.

Regarding sex interaction, a significant difference in diastolic dysfunction between men and women was previously reported (28). In addition, increased sympathetic system activity following smoking has been proposed (29) and may be a potential mechanism of its acute effect on the systolic and diastolic cardiac function. Also, a significant difference had been observed between men and women regarding autonomic nervous system activity (30). Therefore, unlike previous studies that were either conducted only on the male population (17) or did not compare the changes between women and men (27), a sex-specific sub-analysis was considered in our study, which showed that the alterations in PASP and GLS were more prominent in women following cigarette smoking.

Smoking intensity affects the incidence of cardiac and non-cardiac complications and has been categorized as < 10, 10 to < 25, 25 to 40, and ≥ 40 pack-years in prior investigations (31). As the acute effects of smoking intensity on systolic and diastolic cardiac function were unknown, we used the lowest degree, i.e., consumption of 10 pack-years, as the cutoff in this study. The results showed a more prominent PASP and E/A ratio alteration in participants with less than ten pack-years of smoking.

This study's major strength was being the first to assess the sex-specific acute effects of conventional cigarette smoking on systolic and diastolic cardiac function using 2D speckle tracking echocardiography. Considering the smoking intensity (measured by pack-years) in the sub-analysis was another point of strength compared to previous studies. As a limitation, ECG findings were not simultaneously assessed and may be considered in future studies.

5.1. Conclusion

This study showed significant differences in global longitudinal strain (GLS), E/A ratio, and pulmonary artery pressure ten minutes after smoking. It was carried out as a pilot for larger clinical studies to demonstrate the acute adverse effects of smoking on cardiac function and cardiovascular health. Larger studies across numerous centers would yield more generalizable results.

5.2. Ethical Approval

This study was approved under the ethical approval code of IR.SBMU.MSP.REC.1397.602.

5.3. Informed Consent

Written informed consent was obtained from all participants.

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Authors' Contribution

M.P. and K.K.T designed the evaluation and drafted the manuscript. F.M. participated in designing the evaluation, performed parts of the statistical analysis and helped to draft the manuscript. All authors read and approved the final manuscript.

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Financial Disclosure

The authors have no conflicts of interest to declare.

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