

Impact of smoking on left and right ventricular systolic function and left ventricular mass in young healthy males: A two- and three-dimensional echocardiographic study

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Abstract

Objective: Cigarette smoking has been associated with increased mortality, mainly due to cardiovascular diseases (CVDs). The relationship between tobacco use and CVDs is complex, and that between cigarette smoking and echocardiographic measurements of right ventricular (RV) and left ventricular (LV) structure and function has been poorly studied. This study aimed to evaluate the effect of smoking on LV and RV systolic functions and LV mass (LVM) in young healthy males using two (2D) and three-dimensional (3D) transthoracic echocardiography (TTE).

Methods: Data for this observational study were collected by screening of 1013 males (smokers and non-smokers) including relatives of patients, workers, medical students and hospital clerks in Cairo University hospitals for thirty months and after history taking and clinical examination, exclusion was done for any candidate with any comorbidities or history of any drug addiction or cannabis consumption or athletes and/or those who perform forcible sports, any candidate with poor echogenic window or any valvular lesions. Overall, 489 healthy males aged 18–35 years were grouped into smoker (123 participants) and non-smoker (366 participants) groups, then subjected to standard 2D TTE after exclusion of to evaluate dimensions and systolic function of LV and RV, then 3D TTE was used to evaluate LV volumes, systolic function and LVM.

Results: Among the smoker group, the 2D TTE parameters revealed a trend towards increasing most of the values. However, only septal and posterior walls thickness ($p=0.03$ and $p=0.01$, respectively), LV myocardial performance index ($p = 0.029$), LVM and that indexed to body surface are (BSA) ($p=0.004$ and $p=0.008$ respectively), RV basal and mid cavity diameters (both $p<0.0001$), RV end-diastolic area and that indexed to BSA (both $p<0.0001$), and end systolic area and that indexed to BSA ($p=0.016$ and $p=0.041$, respectively) showed statistically significant higher values. The LV 3D TTE parameters also revealed similar trend in the smoker group. However, only LVM and that indexed to BSA (both $p<0.0001$) showed statistically significant higher values.

Conclusion: In young healthy males, smoking was associated with subtle alterations in RV structure and LV structure and function.

Key words: Echocardiography, young healthy males, smoking, left ventricle, left ventricular mass, right ventricle

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Introduction

Cigarettes are the most commonly used tobacco product, and the World Health Organization (WHO) classifies smoking addiction as an epidemic (1). Cigarette smoking significantly increases mortality, primarily due to its role in cardiovascular

diseases (CVDs) and cancer development (2). Regardless of conventional risk factors like hypertension, dyslipidemia, or diabetes, smoking is recognized as an independent risk factor for CVDs, contributing to cardiovascular damage through mechanisms separate from traditional risk factors (3).

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Highlights

- In young healthy males, smoking was associated with increased wall thickness and LV mass together with increased LV myocardial performance index
- Smoking was associated with increased RV basal and mid cavity diameters in addition to increased RV end systolic and diastolic areas
- Our study findings suggest that smoking was associated with subtle alterations in RV structure and LV structure and function

Graphical abstract



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Impact of Smoking on Left and Right Ventricular Systolic Function and Left Ventricular Mass in Young Healthy Males: A Two- and Three-Dimensional Echocardiographic Study

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STUDY DESIGN

Young healthy males



18–35 y

Smokers

n=123

vs

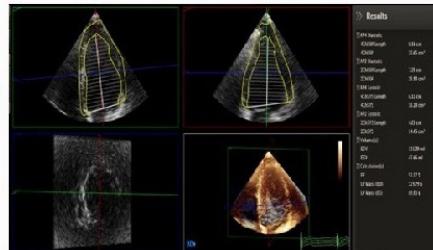
Non-smokers

n=366

KEY RESULT

- ↑ LV wall thickness and LV mass

- ↑ LV myocardial performance index



- ↑ RV basal and mid cavity diameters and RV end systolic and diastolic areas

METHODS



2D and 3D LV and RV
Echocardiography
structure and function

CONCLUSION

In young healthy males,
smoking was associated
with subtle alterations in
RV structure and LV
structure and function

Smoking is one of the most significant preventable risk factors for atherosclerosis (4). Beyond its role in atherosclerosis, smoking has been linked to structural changes in the heart, including increased left ventricular (LV) mass (LVM) and abnormal LV geometry compared with non-smokers, even in the absence of major cardiovascular events (5). Studies examining the relationship between smoking and cardiac structure and function in community-based populations have produced conflicting results. While some research has found an association between active smoking and increased LVM (6-10), others have reported neutral (11) or even inverse relationships (12). In addition, the relationship between smoking and LV and right ventricular (RV) systolic functions remains controversial (7, 9, 10, 13-16). LVM is a known risk factor for cardiovascular morbidity and mortality (17-19). Despite its prognostic importance, its measurement and clinical implications have not been firmly established (20). Additionally, while factors contributing to increased LVM have been extensively studied in older (21, 22) and diseased populations (23), research on young, healthy individuals remains limited. Most studies in this group have involved small sample sizes (24, 25), leading to reduced statistical power and inconclusive findings.

Echocardiography and cardiac magnetic resonance (cMRI) are the two most commonly used imaging methods for the assessment of LVM. Although cMRI is considered the gold standard for assessing LVM, echocardiography is a well validated, non-invasive method that is more widely used in clinical practice (26). However, traditional two-dimensional (2D) transthoracic echocardiography (TTE) has limitations, as it relies on geometric assumptions of uniform chamber size and shape. Additionally, the unintended use of oblique planes can lead to an overestimation of LVM measurements in M-mode, while foreshortening in apical views can result in underestimation (27). To overcome these limitations, three-dimensional (3D) TTE provides more accurate LVM and volume measurements, with reproducibility comparable to that of cMRI (28).

The primary aim of this study was to evaluate the effect of smoking on LV and RV systolic function and LVM in young healthy males using 2D- and 3D-TTE. The secondary aim was to compare measuring LVM by M-mode echocardiography (linear method) and three-dimensional echocardiography in these apparently healthy males.

Methods

Study design and population

This study is an observational study, which was conducted in Cairo University, Cairo, Egypt hospitals for thirty months. Screening was done for 1013 males (smokers and non-smokers) including relatives of patients, workers, medical students and hospital clerks (Fig. 1) but after exclusion of candidates older than 35 years or less than 18 years (at time of the study), only 758 candidates were subjected to history taking and clinical examination. After exclusion of any candidate with hypertension, diabetes or any other comorbidities or history of any drug addiction or cannabis consumption or athletes and/or those who perform forcible

sports, only 608 candidates were subjected to TTE study. Sixty-nine candidates were found to be technically difficult, had mild valvular lesions or had poor echocardiographic window, so they were excluded. The remaining 539 candidates were grouped into two groups (smoker group and non-smoker group) and after matching the two groups according to age, body surface area (BSA) using the formula of Du Bois (29) and body mass index (BMI), fifty candidates were excluded. Therefore, our study population included 489 candidates: of whom 123 candidates (25.2 %) represented the smoker group (Fig. 1) and rest 366 – non-smoker group.

Informed consent was obtained from all participants in our study. The study was performed according to Helsinki 2024 convention on human studies and the protocol was approved by Ethics Committee of Cairo University, Cairo, Egypt.

Baseline variables

All participants underwent history taking and clinical examination with assessment of age, BSA, BMI, history of drug abuse, participation in competitive sports, and comorbidities, structural heart disease.

Smoking assessment

The history of smoking was taken by a questionnaire to measure the average daily consumption of cigarettes [cigarette per day (CPD)] and time to first cigarette of the day (TTFC). CPD is given codes as zero if CPD equals 1-10; one if CPD equals 11-20; two if CPD equals 21-30; three if CPD greater than or equals 31. TTFC is given codes zero if TTFC greater than or equals 61 min; one if TTFC equals 31-60 min; two if TTFC equals 6-30 min; three if TTFC lesser than or equals 5 min. By summing the two previous scores, a scale with a range of 0-6 will be given which will measure the heaviness of smoking index (HIS) (30).

Echocardiographic study

2D transthoracic echocardiography (TTE)

It was performed using Philips EPIQ 7 unit (Philips Medical Systems, Andover, MA, USA) equipped with 2.5 MHz X5-1 transducer. The patients were studied in the left lateral decubitus position. Image and Doppler acquisitions were obtained at held end-expiration. Standard views for TTE were parasternal long-axis and short-axis views, and apical two-, three-, four-, and five-chamber views.

Quantification of the cardiac chambers was done as per the American Society of Echocardiography and the European Association of cardiovascular imaging for assessment of cardiac chambers (31) including measurement of aortic root diameter at sinus of Valsalva and left atrium anteroposterior diameter by 2D-guided linear measurements. M-Mode guided linear measurements of LV end-diastolic (LVEDD) and end-systolic diameters (LVESD), interventricular septal (IVST) and posterior walls thickness (PWT) at end diastole, and LV ejection fraction (LVEF) were done.

Relative wall thickness (RWT) was calculated using this formula, $RWT = 2 \times PWT / LVEDD$, while LVM was calculated by M-Mode

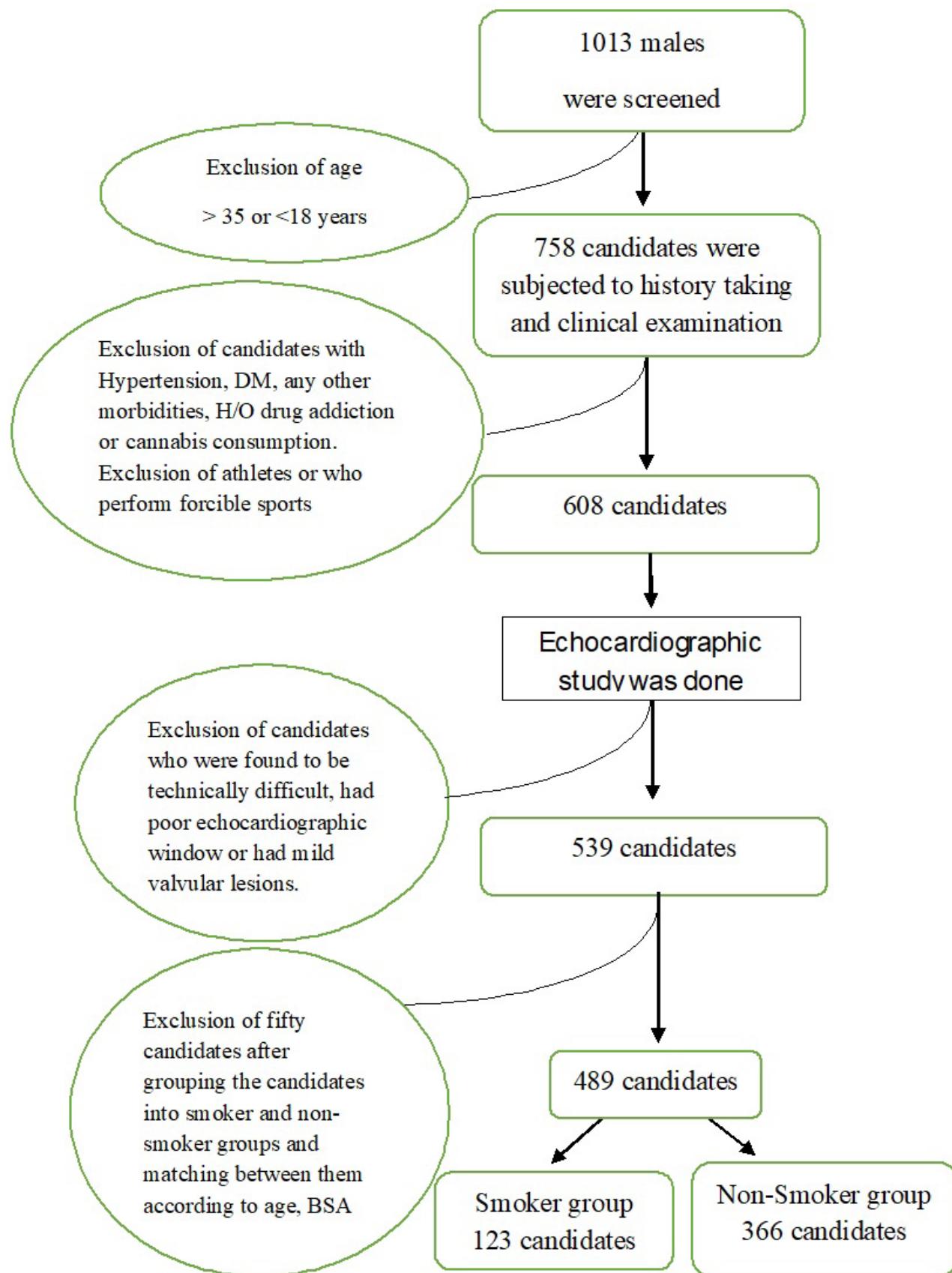


Figure 1. Flowchart showing participants recruitment and number of candidates studied

linear method using the Devereux formula: $LVM = 0.8 \times [1.04 \times (IVST + LVEDD + PWT)3 - LVEDD3] + 0.6$. All measurements were performed at end-diastole (31, 32).

Myocardial performance index (Tei Index, MPI): The pulsed Doppler probe was placed between mitral inflow and LV outflow. Doppler time intervals were measured from mitral inflow and LV outflow Doppler tracings, as described by Tei and co-workers (33). The interval 'a' from end to onset of mitral inflow is equal to the sum of isovolumic contraction time (ICT), ejection time (ET), and isovolumic relaxation time (IRT). ET 'b' is derived from the duration of the LV outflow Doppler velocity profile. The sum of ICT and IRT was obtained by subtracting b from a. The MPI was calculated from the following formula: $(a - b)/b$.

RV linear dimensions (basal, mid and longitudinal RV) were measured at end-diastole, RV end-diastolic area (RV EDA) and end systolic area (RV ESA) were measured by manual tracing of RV endocardial border at end-diastole and at end-systole (31).

Estimation of RV contractile function by tricuspid annular plane systolic excursion (TAPSE) measuring tricuspid annular longitudinal excursion by M-mode between end-diastole and peak systole. Fractional area change (FAC) of RV measured in RV-focused apical four-chamber view: $RV\ FAC\ (%) = (RV\ EDA - RV\ ESA)/RV\ EDA \times 100$ (31).

3D transthoracic echocardiography (TTE)

It was performed using Philips EPIQ 7 unit (Philips Medical Systems, Andover, MA, USA) equipped with a dedicated broadband, wide angle, matrix array transducer. Images were acquired from the apical window, with the patient in the left lateral decubitus position. Care was taken to include the entire LV cavity within the pyramidal scan volume. 3D echo datasets were acquired and obtained over 4 different electrocardiogram

gated cardiac cycles during a short breath hold to avoid stitching artifacts.

Measurements of LV end-diastolic (LV EDV) and the end-systolic (LV ESV) volumes, LVEF and LVM were performed off-line. The 3D echo datasets were analyzed with a prototype version of 3D QLab 10 software (cardiac motion quantification (CMQ); Philips Medical Systems) that allowed for semi-automatic detection of endocardial and epicardial borders. The 3D volume datasets were displayed in 3 different cross-sections that can be modified interactively. Markers were then placed onto the mitral annulus and the apex, both in end-diastole and end-systole. Using these markers, the software program created truncated ellipsoid end-diastolic and end-systolic 3D meshes of the LV. The end-diastolic and the end-systolic 3D meshes deformed until equilibrium were reached between closeness to the borders and minimal curvature. If needed, manual adjustments were applied. The volumes of the obtained 3D-end-diastolic and end-systolic LV meshes included the papillary muscles into the LV cavity and corresponded to the LV EDV and LV ESV and so LV EF was calculated (31, 34, 35).

Measuring of LVM was done by detecting the epicardial contours, manual corrections were then applied, if needed. These epicardial contours were used to calculate the myocardial volume by subtracting the end-diastolic endocardial volume from the end-diastolic epicardial volume. The difference between these two volumes was then multiplied by the specific mass of myocardial tissue (1.05 g/ml) to obtain LVM (36, 37) as shown in Figure 2.

Statistical analysis

Data was statistically analyzed using the Statistical Package of Social Science Software program (SPSS), version 20 (IBM, New York, USA).

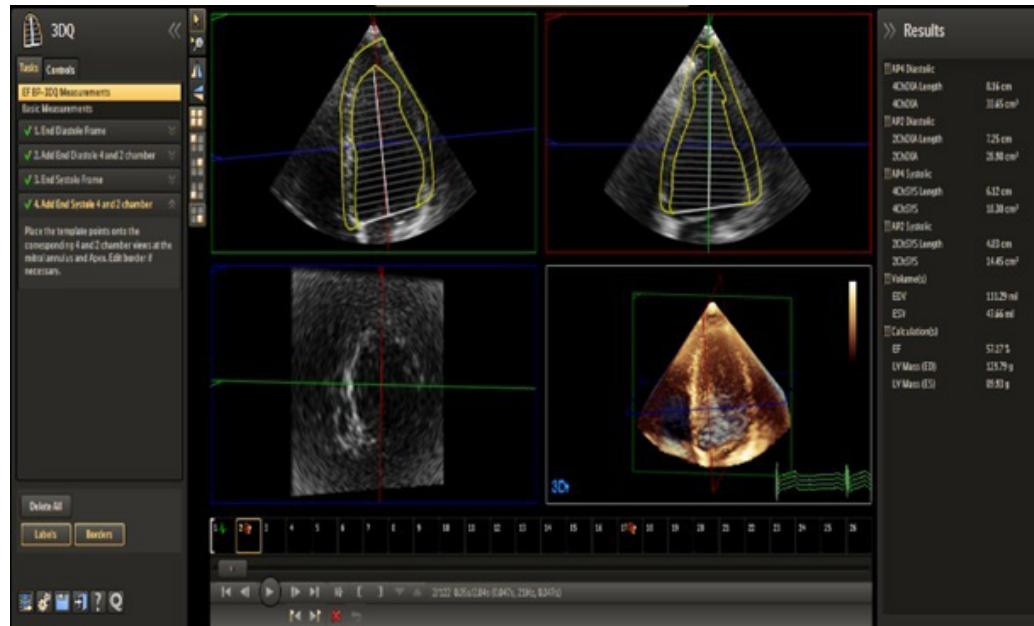


Figure 2. Three-D measurement of LV mass of one of cases

D-dimensional, LV - left ventricular

Continuous variables were described in terms of mean (standard deviation, SD) while categorical variables were described in terms of frequencies (number of cases) and percentages (%) when appropriate.

Mann Whitney U test was used for independent samples for comparison between smoker and non-smoker groups with continuous variables, which were not normally distributed. The confidence interval was set to 95% and the margin of error was set to 5%. The p value was considered significant if <0.05 and highly significant if <0.01 . Wilcoxon Signed Ranks test was used to compare between two different modalities: 3D TTE and

M-mode linear method, which were used to assess LVM. Bland Altman analysis was used to evaluate the differences between the M-mode linear method and 3D TTE as regard to assessment of LVM.

Results

The study population included 489 candidates: of whom 123 candidates (25.2%) represented the smoker group. Baseline clinical characteristics of the whole study population showed that the mean age was 26.3 (4.8) years, BSA was 1.97 (0.15) m² and BMI was 25.34 (3.91) kg/m² as in Table 1.

Table 1. Study population baseline characteristics

Variables	mean (SD)
Age, years	26.3 (4.8)
Body surface area, m ²	1.97 (0.15)
Body mass index, kg/m ²	25.34 (3.91)

Smoking parameters of the smoking group showed that the average years of smoking were 5.0 (3.4) years, the mean amount of cigarette smoked per day were 16 (9) cigarettes/day, the mean TTFC was 32.0 (21.6) min and the mean HIS score was 2.4

(1.3) as shown in Table 2. There were no significant correlations between these smoking parameters and the 2D or 3D TTE parameters.

Table 2. Smoking parameters of the smoking group

Variables	mean (SD)
Average years of smoking, years	5.0 (3.4)
Amount of cigarette smoked per day, cigarettes/day	16 (9)
Time to first cigarette of the day (TTFC), min	32.0 (21.6)
Heaviness of smoking index (HIS) score	2.4 (1.3)

Effect of smoking on 2D TTE variables

The comparison of LV 2D TTE parameters between smoker and non-smoker groups demonstrated that there was a trend towards increased values of most of the LV parameters in the smoker group. However, septal wall thickness IVST ($p=0.03$), PWT ($p=0.01$), LV MPI ($p=0.029$), LVM ($p=0.004$) and LVM indexed to BSA (LVMi) ($p=0.008$) showed statistically significant higher values in smokers as compared to non-smokers.

Regarding RV 2D TTE parameters, the comparison displayed also a trend towards increased values of most of the parameters in the smoker group. However, RV basal diameter ($p<0.0001$), RV mid diameter ($p<0.0001$), RVEDA ($p<0.0001$) and RVEDA indexed to BSA ($p<0.0001$), RVESA ($p=0.016$) and RVESA indexed to BSA ($p=0.041$) showed statistically significant higher values in smokers group as compared to non-smokers group, as shown in Table 3.

Table 3. Comparison of 2D echocardiographic parameters of the left ventricle, left atrium and aorta and RV ventricle between smoker and non-smoker groups

Variables	Smoker (n=123)	Non -smoker (n=366)	p*
LVEDD, cm	4.91 (0.47)	4.85 (0.45)	0.198
LVEDD indexed to BSA, cm/m ²	2.5 (0.29)	2.48 (0.28)	0.585
LVEDV, ml	114.67(24.67)	111.66 (23.87)	0.211
LVEDV indexed to BSA, ml/m ²	58.21 (12.53)	57.05 (12.48)	0.341
LVESD, cm	3.22 (0.32)	3.16 (0.35)	0.182
LVESD indexed to BSA, cm/m ²	1.64 (0.21)	1.62 (0.22)	0.436
LVESV, ml	41.95 (9.98)	40.68 (10.91)	0.231
LVESV indexed to BSA, ml/m ²	21.34 (5.23)	20.82 (5.81)	0.277
IVST, cm	0.85 (0.1)	0.82 (0.11)	0.003
PWT, cm	0.81 (0.1)	0.78 (0.1)	0.01
LV EF, %	63.16 (5.24)	63.58 (5.55)	0.56
LV MPI	0.39 (0.04)	0.38 (0.04)	0.029
RWT	0.33 (0.05)	0.33 (0.05)	0.202
LVM, gr	139.58 (30.95)	129.99 (26.4)	0.004
LVMI, gr/m ²	70.71 (15.06)	66.37 (13.65)	0.008
LA diameter, cm	3.35 (0.38)	3.36 (0.4)	0.904
LA diameter indexed to BSA, cm/ m ²	1.71 (0.23)	1.72 (0.23)	0.881
Aortic root diameter at sinus of Valsalva, cm	2.92 (0.31)	2.91 (0.29)	0.967
Aortic root diameter indexed to BSA, cm/ m ²	1.49 (0.21)	1.49 (0.18)	0.534
RV basal, cm	3.62 (0.44)	3.39 (0.44)	<0.0001
RV mid, cm	3.04 (0.44)	2.79 (0.51)	<0.0001
RV longitudinal, cm	7.2 (0.76)	7.08 (0.73)	0.15
RVEDA, cm ²	18.41 (2.68)	17.12 (2.45)	<0.0001
RVEDA indexed to BSA, cm ²	9.36 (1.48)	8.76 (1.39)	<0.0001
RVESA, cm ²	9.68 (1.98)	9.18 (1.82)	0.016
RVESA indexed to BSA, cm ²	4.92 (1.04)	4.7 (1.0)	0.041
FAC, %	47.53 (6.64)	46.45 (6.77)	0.091
TAPSE, cm	2.37 (0.34)	2.35 (0.33)	0.587

*p value is significant < 0.05 and is highly significant < 0.01, Data are presented as mean (SD), Mann Whitney U test

BSA - body surface area, FAC - Fractional area change, LA - Left atrium, LV - Left ventricle, LV EF - Left ventricle ejection fraction, LV MPI - Left ventricle myocardial performance index, LVEDD - left ventricular end-diastolic dimension, LVEDV - left ventricular end-diastolic volume, LVESD - left ventricular end-systolic dimension, LVESV - left ventricular end-systolic volume, PWT - posterior wall thickness, RV - right ventricle, RVEDA - right ventricle end-diastolic area, RVESA - right ventricle end-systolic area, RWT - relative wall thickness, IVST - interventricular septum thickness, TAPSE - tricuspid annular plane systolic excursion

Table 4. Comparison between smoker and non-smoker groups regarding 3D echocardiographic parameters of the left ventricle

Variables	Smoker (n=123)	Non -smoker (n=366)	P*
LVEDV, ml	123.76 (22.91)	120.23 (22.98)	0.158
LVEDV indexed to BSA, ml/ m2	62.9 (11.99)	61.44 (12.15)	0.268
LVESV, ml	47.4 (9.03)	46.12 (10.37)	0.304
LVESV indexed to BSA, ml/ m2	24.09 (4.68)	23.59 (5.53)	0.354
LV EF, %	61.48 (4.17)	61.58 (4.73)	0.879
LVM, gr	154.23 (25.89)	142.59 (23.2)	<0.0001
LVMI, gr/m2	78.19 (12.5)	72.8 (12.13)	<0.0001

*p value is highly significant < 0.01, Data are presented as mean (SD), Mann Whitney U test

BSA - body surface area, LV - Left ventricle, LV EF - left ventricular ejection fraction, LVEDV - left ventricular end-diastolic volume, LVESV - left ventricular end-systolic volume, LVM – left ventricular mass, LVMI – LVM indexed to BSA

Comparison of LVM measured by M-mode echocardiography (linear method) and 3DTTE

In the whole study population (489 candidates), LVM measured by 3DTTE was significantly higher (p<0.0001) when compared to that measured by the linear method [145.52 (24.41) gr vs. 132.4 (27.89) gr, respectively] and [LVMI 74.15 (12.44) gr/ m2 and 67.46 (14.13) gr/m2, respectively].

Wilcoxon Signed Ranks test revealed that LVM and LVMI values measured by 3DTTE were higher than that measured by the

linear method in 455 candidates (positive ranks) and were higher in the linear method compared with 3DTTE in only 34 candidates (negative ranks). Bland-Altman analysis in the whole study population showed a trend toward lower values of LVM measured by M-mode compared with that measured by 3DTTE by demonstrating a bias of -13.12 gr with 95% limits of agreement at \pm 10.86 gr, the upper limit of agreement = 8.16 gr and the lower limit of agreement = -34.39 gr (Fig. 3).



Figure 3. Bland-Altman analysis of agreement between LVM measured by M-mode and that measured by 3D TTE

3D TTE – 3-dimensional transthoracic echocardiography, LV – left ventricular, LVM – left ventricular mass

Also, Bland-Altman analysis showed a trend toward lower values of LVMI measured by M-mode compared with that measured by 3D TTE by demonstrating a bias of -6.7 gr/m² with 95% limits of

agreement at ± 5.58 gr/m², the upper limit of agreement = 4.25 gr/m² and the lower limit of agreement = -17.64 gr/m² (Fig. 4).

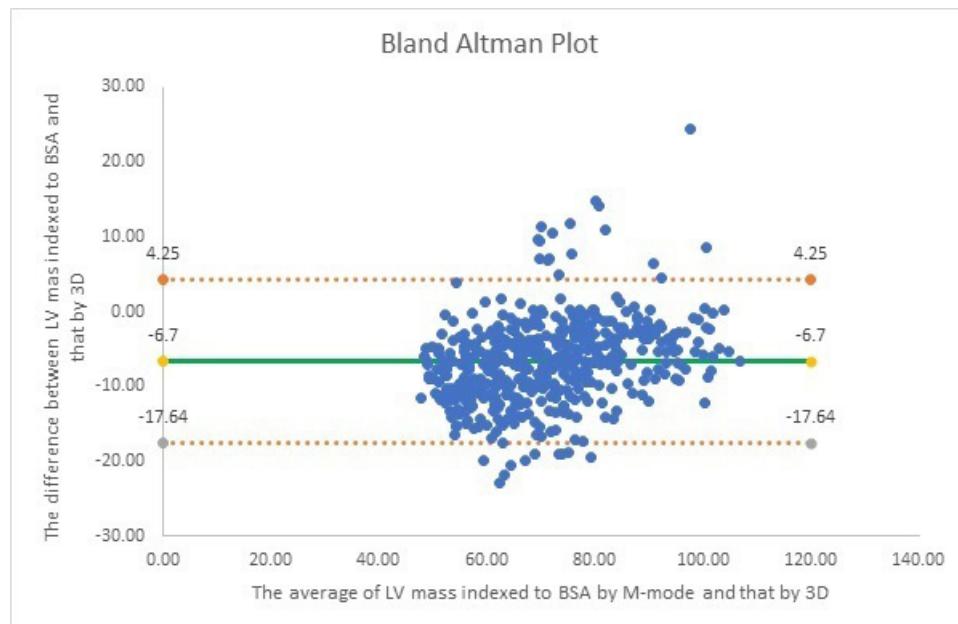


Figure 4. Bland-Altman analysis of agreement between LVMI measured by M-mode and that measured by 3D TTE

3D TTE – 3-dimensional transthoracic echocardiography, LV – left ventricular, LVMI – left ventricular mass index

Discussion

Smoking has been associated with structural changes in the heart including LVM and LV geometry, 5 these changes in myocardial structure and function can be evaluated by echocardiography.

TTE is a widely used imaging technique known for its safety, absence of ionizing radiation, affordability, portability, and quick to perform. It remains the most commonly employed method worldwide for assessing cardiac chamber dimensions and LV wall thickness (38). This study was conducted on young healthy males aiming to evaluate the effect of smoking on LV and RV structure and systolic function and LVM using TTE and it was found that the 2D TTE parameters revealed a trend towards increasing most of the values. However, only IVST and PWT, LV MPI, LVM and LVMI, RV basal and mid cavity diameters, RV EDA and that indexed to BSA, and RV ESA and that indexed to BSA showed statistically significant higher values. The LV 3D TTE parameters also revealed that only LVM and LVMI showed statistically significant higher values in smokers as compared with non-smokers.

Previous 2D TTE studies have reported conflicting findings regarding the impact of smoking on structure and function of LV and RV compared with our data (7, 9, 10, 12).

The ECHO-SOL study (10) (Echocardiographic Study of Hispanics/ Latinos) examined the effect of smoking in 1818 of Hispanic/Latino population of the United States with mean

age 56.4 years. In agreement to the current study, comparing the current smokers with never smokers population showed significant increase in LVEDV and LVESV, and increased RVEDA and LVM, which tend to be statistically significant ($p=0.06$ and $p=0.09$, respectively). But in contrast to our data, the ECHO-SOL showed significant decrease of LVEF and RV FAC. Also, the current study showed a significant increase of LV MPI in smoker group which may be considered as a subtle LV systolic and diastolic dysfunction. The decreased LVEF and RV FAC in smoker population of ECHO-SOL can be explained by the differences of mean age of both studies. The ECHO-SOL study population was significantly older than the current population by an average of 30 years, so ECHO-SOL study population was exposed to more years of smoking and more cigarettes smoked and altered RV function was associated with more cigarettes smoked.

Cigarette smoking is also the most common risk factor for development of chronic obstructive pulmonary disease (COPD) (39) which was not an exclusion criterion in ECHO-SOL. COPD is associated with pulmonary hypertension (40) and RV enlargement, LV enlargement, as well as LV systolic and diastolic dysfunction (41). Even subclinical impairment of lung function has been shown to be associated with LV systolic and diastolic dysfunction (42), which could potentially contribute to RV dysfunction as well.

The ARIC Study (9) (Atherosclerosis Risk in Communities) examined the effect of smoking in 4580 participants of the United States free of overt coronary artery disease, heart

failure, and significant valvular disease with mean age 74 years. Comparing their current smokers with never smokers population showed statistically significant increase of IVST, LVM and LVMI, aligning with our findings. In contrast to our data, there is a marginally higher LVEF in current smokers. Also, ARIC Study showed no significant association between RV structural and functional features and smoking status. This increase in LVEF may be influenced by cardiac geometry (43) and was known to increase in parallel with LVM/volume ratio (44). In addition ARIC study researchers found that the difference in LVEF between current and never smokers became no longer apparent after adjusting for LVM/volume ratio in multivariable analysis, suggesting that the higher LVEF seen in current smokers could be more a consequence of variation in LV geometry rather than resulting from enhanced LV systolic function. Furthermore, in the ARIC Study, the global longitudinal LV strain, which has been reported as a more sensitive measure of systolic function, (45) did not differ among their study groups.

Regarding 3D TTE parameters, the comparison between the smokers and non-smokers groups in the current study showed statistically significant differences only for LVM and LVMI. 3D TTE was chosen in the current study to detect the effect of smoking on LV systolic function and structure especially the LVM.

However, previous studies as Multi-Ethnic Study of Atherosclerosis (MESA) (7) and LARGE Heart study (12) used cMRI as a method of assessment to detect the effect of smoking. The MESA study (7) examined 4869 participants from 4 racial/ethnic groups: African, American, Hispanic, and Asian enrolled from six U.S. communities with mean age 62 years to assess the effect of traditional cardiovascular risk factors including smoking on LV mass, volume, and systolic function by cMRI. Current smokers of MESA study were associated with higher LVM in agreement to our data but lower LVEF in contrast to our data, this may be explained by the larger sample size in MESA study. Also the use of cMRI, as a method of assessment in MESA study, permitted researchers to identify relationships of smoking with subclinical alterations in LV size and systolic function that were difficult to be detected using previously available noninvasive methods as echocardiography, so MESA study allowed the detection of very small differences in LV mass, volumes, and systolic function in relation to smoking.

The LARGE Heart study (12) also used cMRI as a method of assessment of LVM and its association with lifestyle including cigarette smoking. But in comparison to MESA, they examined healthy Caucasian male army recruits from United Kingdom which were younger study population (with mean age 19.8 years) and a smaller sample size (n = 541). Unexpectedly in LARGE Heart study, there was an inverse relationship, with LVM being less in current smokers. Researchers tried to suggest that such an association was mediated through blood pressure that the systolic blood pressure (SBP) was significantly lower in current smokers. However, such an observation was misleading. Although cross-sectional studies demonstrate that blood pressure is often lower amongst smokers (46), brachial artery SBP is affected by pulse pressure amplification (47) — a

phenomenon that exaggerates aortic pulse pressure when measured peripherally. This effect is particularly pronounced in young individuals and is known to be diminished by smoking. As a result, reduced pulse pressure amplification in smokers could lead to lower brachial artery SBP, especially in studies involving young populations. Moreover, aortic SBP, a key determinant of LV workload, has been found to be higher in young smokers when measured using applanation tonometry (47). This suggests that the observed lower LVM in smokers within the LARGE Heart study cannot be solely explained by reduced SBP, and an alternative explanation should be explored.

Comparison between measuring LVM by M-mode linear method and 3D echocardiography

Our data showed that the LVM and LVMI measured by 3D TTE was higher than that measured by the M-mode linear method. Data from previous studies showed that M-mode linear method measuring LVM (probably due to oblique cuts) usually overestimated LVM in comparison to cMRI, which is the gold standard for assessment of LVM..

The LVM measurement by 3D TTE showed improved accuracy compared to conventional echo techniques. In the study done by Yap et al. (48) which evaluated the accuracy of real-time 3D TTE in determining LVM compared to cMRI, their data showed good agreement between the two modalities.

Meta-analysis done by Shimada and Shiota (49) detected the accuracy of LVM measurement by 3D TTE and evaluated the controversial data on the systematic bias in LVM assessment by 3D TTE, they revealed the existence of significant underestimation in the past, improvement of accuracy over the past decade, and excellent accuracy with most recent 3D TTE technologies. Inaccuracy of LVM measurement can result mainly from 2 sources: tracking of the endocardium and the epicardium. Tracing the inner side of the endocardium leads to underestimation of cardiac volumes and overestimation of LVM, which has been repeatedly shown in previous series of studies (50). In contrast, tracking the inner side of the epicardium leads to the underestimation of LVM. They found that the underestimation arising from epicardial tracking had outweighed the overestimation from endocardial tracking, resulting in a systematic underestimation of LVM in the earlier studies.

Study limitations

Firstly, we described results of a single-center study with a relatively small number of the studied population and included males only so other studies including larger number with females are needed. Secondly, estimation of tissue Doppler measurements and diastolic function assessment were not performed to detect the effect of smoking on diastolic function. Thirdly, our study did not include speckle-tracking echocardiography to detect LV and RV global longitudinal strain which has been reported as a more sensitive measure of the effect of smoking on systolic function and can detect even any subtle changes in systolic function.

Conclusion

In young healthy males, smoking was associated with increased wall thickness and LVM together with increased LV MPI. Also, smoking was associated with increased RV basal and mid-cavity diameters in addition to increased RV end systolic and diastolic areas. These findings suggest that smoking was associated with subtle alterations in RV structure and LV structure and function.

Ethics: Informed consent was obtained from all participants in our study. The study was performed according to Helsinki 2024 convention on human studies. The study protocol was approved by Ethics Committee of Faculty of Medicine, Cairo University, Cairo, Egypt.

Peer-review: External and internal

Conflict of interest: None to declare

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