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Electrophysiological Findings on Effects of Smoking on Non-invasive Cardiovascular Parameters among Young Individuals in Etawah District, Uttar Pradesh

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ABSTRACT

BACKGROUND: Short-term cigarette smoking in young individuals exerts a significant impact on cardiovascular physiology, evidenced by acute elevations in heart rate, blood pressure, and sympathetic drive. These hemodynamic alterations are accompanied by a transient increase in arterial stiffness, reflecting early vascular dysfunction. Although such changes may be partially reversible in the absence of chronic exposure, they indicate that even limited smoking episodes can initiate vascular injury and accelerate arterial aging, thereby predisposing young individuals to long-term cardiovascular risk.
AIM: This study aims to evaluate electrophysiological findings among young individuals who are exposed to cigarettes smoking for a short periods and compared with non- smokers. Increase in arterial stiffness, reflecting early vascular dysfunction, almost all intervals and waves of ECG are significantly shortened but electrophysiological implications have not been investigated. The *acute effect of smoking* on cardiovascular and electrophysiological parameters before and after exposure in young smokers and young non-smokers are investigated.
MATERIALS AND METHODS: Descriptive comparative study over total durations of 24 months. A total 150 young healthy smokers and non -smokers aged in between 25-35 yrs. were taken for the study. Non-invasive parameters were recorded with the help of invasive PC based cardiovascular analyser; Blood pressure and ECG were recorded with the help of sphygmomanometer and 12 leads ECG machine.
RESULTS: Statistically significant decrease with almost all waves and intervals of ECG along with total period (R-R interval) were noted in non- smoker group just after smoking, Blood pressure was increased and significant increase with all non- invasive responses such as pulse wave velocity (PWV), arterial stiffness index (ASI) & Ankle brachial index (ABI) in non- smoker group just after smoking.
CONCLUSIONS: Smoking, even for a short time, affects the heart and blood vessels in young people. It raises heart rate by increasing the electrophysiological responses and blood pressure and makes the arteries temporarily stiffer. While these effects may go away if

smoking is stopped early, repeated smoking can damage the arteries and increase the chance of future heart problems. The present findings demonstrate that even acute smoking exposure in non-smokers as compared to smokers elicits significant electrophysiological and hemodynamic alterations.

Keywords: *Arterial Stiffness Index (ASI), Ankle Brachial Index (ABI), Pulse Wave Velocity (PWV), ECG, Smokers & non-smokers*

INTRODUCTION

Electrophysiology is the science of measuring and understanding the body's bioelectric signals, especially in the nervous and cardiovascular systems. It focuses on how excitable cells, such as neurons, muscle cells, and cardiac cells-generate and transmit electrical signals. Acute smoking produces short-term, reversible electrophysiological effects dominated by sympathetic activation: tachycardia, transient HRV suppression, and vasoconstriction. Chronic smoking leads to sustained alterations and structural damage, autonomic imbalance, reduced HRV, prolonged QT interval, arterial stiffness, endothelial dysfunction, and higher arrhythmia risk [1].

Three different types of alterations are observed which includes

A) Physiological alterations: When cigarette smoke is inhaled, nicotine is rapidly absorbed through the lungs into the bloodstream and reaches the brain within seconds. Stimulation of nicotinic acetylcholine receptors (nAChRs) located in the autonomic ganglia and adrenal medulla which stimulates sympathetic postganglionic neurons and enhances catecholamine release (epinephrine & non epinephrine), these later released from sympathetic nerve terminals in the heart and vasculature, resulting↑ Heart rate (via β_1-receptors in the sino-atrial node), ↑ Myocardial contractility (β_1-receptors in cardiac muscle) and Vasoconstriction in peripheral vessels (α_1-receptors), elevating blood pressure. Nicotine also activates central nervous system pathways (locus coeruleus, hypothalamus), further driving sympathetic outflow [2-6].

B) Biochemical involvement includes: Cigarette smoke contains reactive oxygen species (ROS), including free radicals which deplete antioxidants like vitamin C, glutathione, and superoxide dismutase. This imbalance causes endothelial dysfunction by reducing nitric oxide (NO) bioavailability, impairing vasodilation. Smoking increases C-reactive protein (CRP), interleukins (IL-6, IL-1β), and tumor necrosis factor-α (TNF-α). These inflammatory cytokines damage the endothelium and promote vascular stiffness and early atherosclerotic changes. Nicotine and smoke constituents alter lipid profiles (↑ LDL cholesterol, ↑ Triglycerides, ↓ HDL cholesterol), shifts increase cardiovascular risk, even in young smokers.CO from smoke binds with haemoglobin to form carboxyhaemoglobin, reducing oxygen delivery, causes tissue hypoxia, stimulating further sympathetic activity and endothelial stress. Biochemically, smoking in young individuals leads to nicotine-driven catecholamine release, oxidative stress, inflammation, lipid dys-regulation, and CO-induced hypoxia. Together, these mechanisms impair endothelial function, increase arterial stiffness, and accelerate early vascular injury [2-8].

C) Psychological involvement includes: Activation of Dopaminergic Reward Pathway *f*rom the inhalation of nicotine which rapidly stimulates nicotinic acetylcholine receptors (nAChRs) in the brain, particularly in the ventral tegmental area (VTA). This causes increased dopamine release in the nucleus accumbens, activating the mesolimbic reward pathway, resulting pleasurable sensations, relaxation, and reinforcement of smoking behaviour (Figure-1). Nicotine enhances release of

acetylcholine and norepinephrine, improving alertness, attention, and reaction time [5-9].

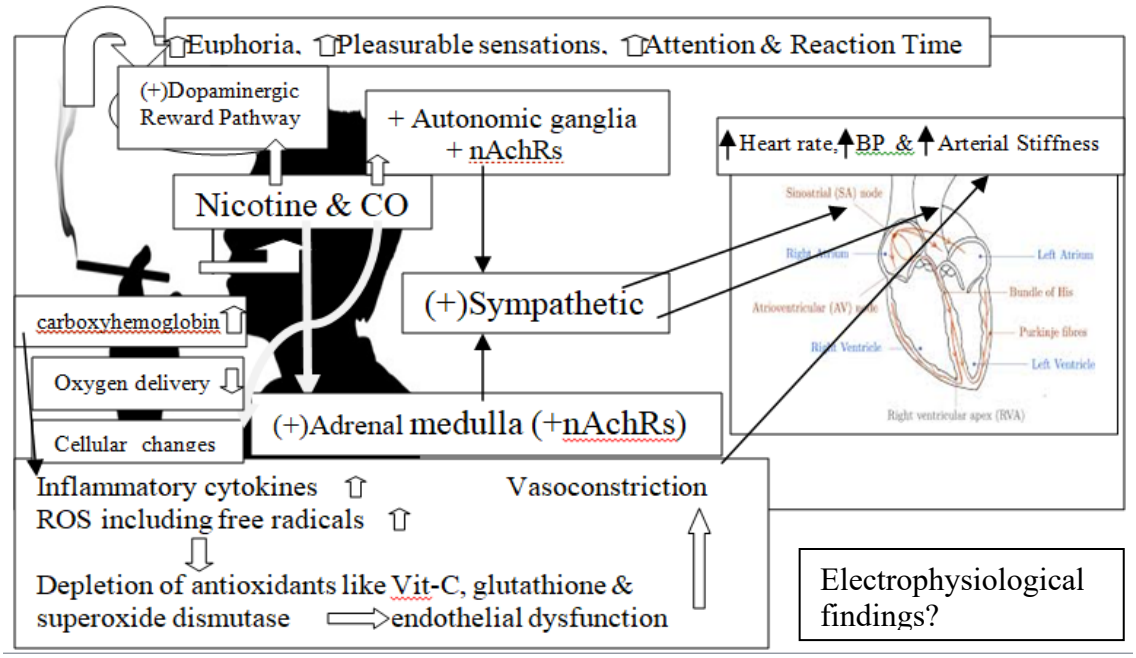


Figure-1: Diagrammatic representation of effect of smoking of physiological aspect on cardiovascular system in healthy young adults.

This study aims to investigate the electrophysiological implications on the effects of smoking on cardiovascular parameters in both smokers and non-smokers and differences if any on non-invasive cardiovascular responses and ECG between before smoking and after smoking in chronic smokers in healthy young subjects. The primary aim is to measure acute changes in ECG parameters (waves and intervals), heart rate/HRV, blood pressure, and arterial stiffness (PWV, ASI, ABI) induced by a single smoking episode in young adults, and compare responses between habitual smokers and non-smokers. The present research will look at the effect of smoking on the ECG, non - invasive responses including blood pressure to improve *public awareness of the dangers of smoking.*

MATERIALS & METHODS

Study design and setting:

The study was carried out at the district hospital, Etawah and department of Physiology, Uttar Pradesh University of Medical Sciences, saifai of Etawah district.

A total of 150 smokers and 150 non-smokers were included in this study, each between the ages of 25-35, were recruited from the student population of this University and people of Etawah district. Subjects who agreed to take part in the study after hearing about its scope and goals were enrolled with their informed permission. The individuals were interviewed extensively before undergoing a thorough physical examination. Both height and weight were recorded, the physical examination was also carried out which involved palpating the radial artery to determine resting pulse rate. Blood pressure was measured using a mercury sphygmomanometer and stethoscope. All these measurements were recorded in the patient's medical record. A thorough evaluation of the cardiovascular and respiratory systems was carried out by medical personnel. Inclusion and exclusion criteria were used to choose participants for the research.

Prospective, controlled, observational before-and-after study with two parallel groups: Group A: Young male habitual

smokers (smoke daily; short abstinence pre-test—see below), daily cigarette smokers for >6 months; typical consumption e.g., ≥5 cigarettes/day. Group B: Young male non-smokers, never smokers or <5 lifetime cigarettes; no regular second-hand exposure
Smoking exposure: We used a commercially available standard cigarette (standardized nicotine cigarette) in research. Instructed participant to take one cigarette and smoke in a standardized fashion (e.g., one puff every 30 seconds until cigarette finished). We measured systolic pressures at ankle and brachial; computed ratio. Each participant acted as their own control (pre- vs post-smoking). Non-smokers were exposed to the same standardized smoking challenge (supervised single cigarette) to compare acute effects.

Inclusion criteria:

- Male, 25–35 years.
- Written informed consent.
- BMI 18.5–29.9 kg/m².
- Resting SBP <140 and DBP <90 mmHg.
- For smokers: self-reported smoking ≥1 cigarette/day for ≥12 months and cotinine > cut-off.
- For non-smokers: never or <100 lifetime cigarettes and cotinine below cut-off.
- Not taking medications that affect autonomic function.

Exclusion criteria:

- Known cardiovascular disease, diabetes, hypertension, arrhythmia
- Use of cardiovascular, psychotropic, or vasoactive medications
- Substance abuse (excluding nicotine for smokers)
- Recent vigorous exercise, caffeine (>200 mg) or alcohol within 12 hours
- Allergy or sensitivity to nicotine products
- Any contraindication to temporary smoking exposure (for non-smokers)

Standardization & pre-test preparation: Subjects were abstained from food for 2 hours and caffeine for 12 hours and abstained from vigorous exercise for 24 hours. Smokers were asked to follow standardize pre-test abstinence (e.g., 8 hours nicotine abstinence). We measured baseline room temperature, humidity, performed testing in a quiet, temperature-controlled room (20–24 °C).
Timing / measurement schedule: Baseline (pre-smoking, after 10–15 min rest): ECG (5 min), BP (triplicate), PWV, ASI, ABI, SpO_2, symptoms, sample for catecholamines if used.
Post-smoking measurements at these time points (typical acute pattern):

- Immediately (0 min) - within 1–2 min after finishing cigarette: ECG (2–5 min), BP, SpO_2, symptoms
- 5 minutes post
- 15 minutes post
- 30 minutes post
- 60 minutes post

At each time point: ECG (short recording), BP, SpO_2, symptoms were recorded. Repeated PWV/ABI at baseline and at 15 and 60 min (PWV often measured at fewer time points due to logistics).

Data collection workflow (step by step):

1. Verified inclusion/exclusion; obtain consent; complete screening checklist.
2. Participant was asked to go for rests either supine /seated 10–15 minutes before baseline recordings.
3. Smoking exposure was done under supervision (in ventilated room) and with safety staff present.
4. Followed timing schedule and, collected data per instrument SOP.
5. Monitored and recorded any adverse event. Provided medical care if needed.

Safety & ethical considerations:

- Institutional ethics approval was taken (IEC).
- Special ethical attention was considered and maintained if exposing non-smokers — considered local rules which include justification and minimize harm (single cigarette only).

- Resuscitation equipment was kept on site; trained personnel also were present.
- Provided post-test counselling and smoking-cessation resources, especially for smokers and non-smokers who needed help.
- Infection control and ventilation were done.

Data processing & signal analysis:

- ECG: filter, detect R-peaks algorithmically, and then manually verify. Compute averages for intervals and amplitudes. Use QTc correction with at least two formulae and report both.
- HRV: pre-process for ectopic beats; used established software (e.g., Kubios scripts). Reported time & frequency domain metrics.
- PWV/ABI: PC based non- invasive cardiovascular analyser
- Documented all pre- processing steps and criteria for exclusion of segments.

Calculation of BMI: Weight in kilograms and height in meters were taken from each participant for BMI and was calculated in a following way.

BMI = Weight (in kilograms/Height (in meters)

Recording of ECG: Electrocardiogram of each subject was recorded by using BPL ECG Recorder – Cardiart 108T (1 Channel). ECG was recorded in supine position after several minutes' rest using digital surface ECG recorder, and was printed at a paper speed of 25 mm/sec and amplitude of 5 mm/mV. Absolute QT interval (m sec) was measured from the beginning of the QRS complex to the end of the T wave. The end of the T wave was defined as the intercept between the isoelectric line and the regression line derived from the descending slope of the T wave. U wave was carefully distinguished from T wave. Strictly, absolute QT interval is different in different leads. In the present study, QT interval in limb leads II was adopted, because this interval is longest in lead II, and T wave is easily distinguished from U wave in this lead. ECG data were diagnosed based on Minnesota code, transferred using A/D converter, and stored automatically to a personal computer. Thereafter, ECG recordings were reviewed by experienced cardiologists in a blind manner

Measurement of noninvasive parameters: Periscope (Figure-1) is a PC based low-cost instrument hence used with a computer (Figure-1). It used ECG as a marker. Periscope thus facilitates use in epidemiological studies which has been validated and has good intraday and inner observer reproducibility for various estimated central and peripheral arterial velocities. In brief, PWV was determined by a non-invasive pulse wave analyzing device (Periscope). All participants, Group A & B, were asked to have vegetarian diet, asked to refrain from drinking caffeine-containing beverages 12 hours before the test for non-smokers. Procedure was performed always by the sale operator in the morning hours between 7 and 10 a.m. with subject resting in supine position at least 10 min before the recording. Electrodes for electrocardiogram were placed in ventral surface of both wrists and medial side of ankles and BP cuffs were wrapped on both upper arm brachial artery and tibial artery above ankles. The cuff was connected to a plethysmography sensor which determines volume pulse form and an oscillometer pressure sensor, which measures blood pressure volume waveform from the brachial and tibial arteries. (Figure -2).

Draft screening questionnaire to take basic information, smoking history, medical history, pre-test checklist along with consent paragraph for smoking exposure are also mentioned in details just after reference section. Data collected after 15, 30 and 60 min were not included for analysis, kept to know the overall health status.

STATISTICAL ANALYSIS

The data was compiled in Microsoft Excel and analysed using SPSS (Statistical

Package for Social Sciences, version 20), Level of significance was kept at $p<0.05$. Students' 'T' test was performed. Accepted time point before and after (5 min) was fixed and concluded using paired t-test.

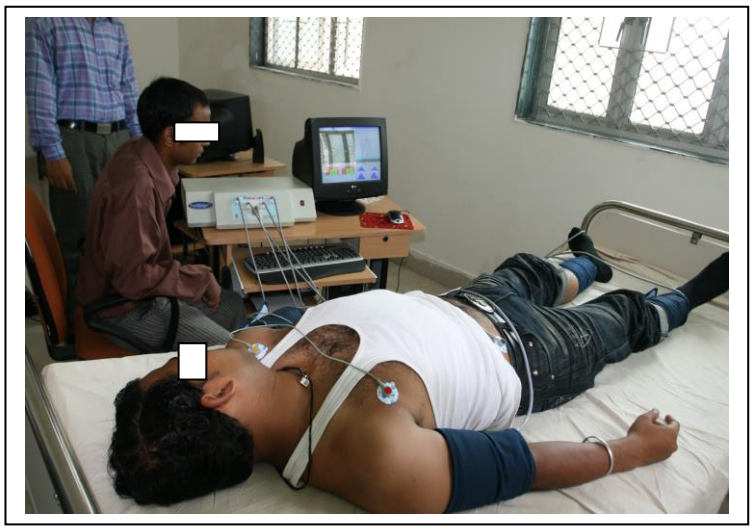
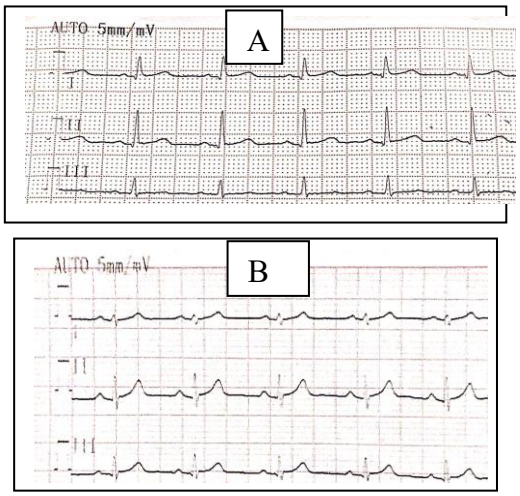


Figure-2: PC based Cardiovascular Analyser for recording ASI, ABI and PWV

Figure-3: ECG records of both smokers (A) and nonsmokers (B).



RESULT

1. Duration of various intervals (PR, TP & QRS) are less in smokers as compared to non- smokers, were significantly ($p<0.05$) before smoking. But no alteration with duration of various waves between these two groups (Figure-3&Table-1).
2. Duration of various intervals and waves (PR, TP & QRS) are altered less ($P>0.05$) in smokers as compared to non- smokers, alteration after smoking in smokers is not significant ($P>0.05$) but the same was significant ($p<0.05$) after smoking in non-smokers (Figure-3&Table-1).
3. Heart Rate (HR), Pulse wave Velocity (PWV) and Arterial Stiffness Index (ASI) in all four regions were found significantly high in smokers than non-smokers before smoking, but no significant alteration found in smokers after smoking, significant alteration or were increased in non-smokers just after smoking (Figure-2).
4. Systolic (SBP), Diastolic (DBP), pulse pressure (PP) and mean arterial blood pressure were at higher side in smoker group as compared to non-smokers,

Significant increase (P<0.05) was found in non-smokers just after smoking as compared to smokers' group (Figure-2&Table-2)

5. No significant alteration (P>0.05) was noted with ABI in both smokers and non-smokers when compared before smoking as well as after smoking in these groups (A&B). No significant alteration was found with age, body weight and BMI when compared between smokers and non-smokers (Table-3).

Table-1: Comparison of various waves & intervals of ECG between smokers and non-smokers and between before and after smoking in both smokers and non-smokers.

Measurements	Smokers (Group-A) (N=150) Mean ± SD Smoking			Non-smokers (Group-B) (N=150) Mean ± SD Smoking			P value* (base line value between smoker & non-smoker)
	before	After	P value	before	After	P value*	
PR interval (sec)	0.142	0.141	± 0.08	0.155	0.141	±0.010*	± 0.020*
QRS interval (sec)	0.121	0.120	± 0.07	0.191	0.090	±0.051	± 0.020*
QTc interval (sec)	0.451	0.410	± 0.07	0.499	0.322	±0.021*	± 0.031
TR interval (sec)	0.702	0.700	± 0.09	0.82	0.61	±0.01*	± 0.030*
P wave (sec)	0.091	0.090	± 0.07	0.12	0.10	±0.01*	± 0.010
QRS complex (sec)	0.094	0.090	± 0.08	0.11	0.09	±0.031	± 0.011
T wave (sec)	0.201	0.182	± 0.07	0.22	0.11	±0.021*	± 0.010

***Student 's 't' test, <0.05 was considered significant.**

Table-2: Non- invasive responses using PC based cardiovascular analyser in both smokers and non-smokers before and after smoking.

Measurements	Smokers (Group-A) (N=150) Mean ± SD Smoking			Non-smokers (Group-B) (N=150) Mean ± SD Smoking			P value (Base line value between smokers and non-smokers
	before	After	P value	before	After	P value	
PWV C-F (cm/sec)	976.3	978.5	0.08	765.8	834.2	0.03*	0.01*
Right baPWV(cm/sec)	1455.3	1483.8	0.06	1201.2	1287.2	0.04*	0.04*
Left baPWV(cm/sec)	1344.5	1347.2	0.07	1123.1	1232.7	0.01*	0.04*
R Bra ASI (mm Hg)	75.4	79.1	0.09	43.5	67.3	0.03*	0.03*
L Bra ASI (mm Hg)	82.3	87.1	0.07	65.2	76.2	0.03*	0.02*
R AnkASI (mm Hg)	79.1	86.2	0.09	64.3	76.5	0.02*	0.01*
L Ank ASI (mm Hg)	86.1	92.1	0.06	61.3	78.2	0.01*	0.01*
HR (beats/min)	85.3	89.4	0.08	78.4	89.2	0.05*	0.04*
ABP (mm of Hg)							
Systolic (SBP) mmHg	123.4	133.4	0.08	121.4	132.1	0.04*	0.07
Diastolic (DBP) mmHg	81.3	85.3	0.06	80.3	85.1	0.03*	0.07
PP (Pulse) mmHg	45.6	46.6	0.07	41.6	43.2	0.05	0.08
Mean ABP mmHg	95.8	98.8	0.08	93.8	96.5	0.03*	0.06
ABI (Ankle Brachial Index)	1.08	1.09	0.06	1.01	1.18 0.	0.06	0.08

***Student 's 't' test**

Table-3: Physical parameters of both the groups (A & B) in both smokers and non –smokers, found insignificant when compared.

Measurements	Smokers (Group-A) N=150	Non-smokers (Group-B) N=150	P value
Age (Years)	26.5 ± 2.54	27.5 ± 2.64	0.08
Body weight (kg)	76.4 ± 6.54	74.7 ± 3.54	0.93
BMI (kg/m2)	23.88 ± 2.04	24.65 ± 2.01	0.98

Student 's 't' test

DISCUSSION

The electrophysiological findings indicate a statistically significant reduction in almost all ECG wave amplitudes and intervals, including the R–R interval, following acute smoking exposure in the previously non-smoker group. A shortened R–R interval reflects enhanced heart rate due to sympathetic overdrive and vagal withdrawal, consistent with the acute autonomic imbalance induced by nicotine and carbon monoxide. Decreased P-wave and PR interval durations may suggest accelerated atrial and atrioventricular nodal conduction, while reduced QRS and T-wave amplitudes imply altered ventricular depolarization–repolarization dynamics, potentially reflecting transient myocardial ischemia or electrophysiological instability [6-15].

Concurrently, the observed increase in systemic blood pressure highlights the hemodynamic consequences of sympathetic stimulation and peripheral vasoconstriction mediated via nicotine-induced catecholamine release. The significant elevation in non-invasive vascular indices - pulse wave velocity (PWV), arterial stiffness index (ASI), and ankle–brachial index (ABI) - further demonstrates an acute rise in arterial stiffness and vascular resistance. These changes collectively suggest that even a short-term smoking challenge in non-smokers can precipitate early vascular dysfunction, reduce arterial compliance, and elevate cardiovascular load [6-16].

When interpreted together, the electrophysiological disturbances (reduced wave amplitudes and shortened intervals) and vascular alterations (increased PWV, ASI, and ABI) indicate an acute but adverse cardiophysiological remodeling. This not only compromises the electrical stability of the myocardium but also accelerates vascular aging processes, thereby providing mechanistic insight into how early smoking exposures predispose to long-term cardiovascular morbidity in smokers [6-18].

Potential limitations & mitigations: Short-term exposure does not equal chronic effects - explicitly state this limitation. Exposing non-smokers carries ethical concerns - mitigate or avoid if local IRB disallows.

CONCLUSION

The present findings demonstrate that even acute smoking exposure in non-smokers elicits significant electrophysiological and hemodynamic alterations. The observed reduction in ECG wave amplitudes and intervals, particularly the shortened R–R interval, indicates autonomic imbalance with enhanced sympathetic activity and altered myocardial conduction. Simultaneously, the elevation in blood pressure and vascular indices (PWV, ASI, ABI) reflects acute arterial stiffening and increased vascular resistance. Taken together, these results suggest that smoking, even in short-term exposure, disrupts cardiac electrophysiology and vascular compliance in otherwise healthy individuals, thereby providing mechanistic evidence for the early onset of cardiovascular risk.

Declaration by Authors

Ethical Approval: Approved

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Conflict of Interest: The authors declare no conflict of interest.

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