Research Article

ECG and Echocardiographic Findings in Adults with Chronic Pulmonary Disorders Who Use Electronic Cigarettes: A Meta-Analysis

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ABSTRACT

Background: Electronic-cigarette use is increasingly common among adults who currently smoke or have smoked combustible cigarettes, including people living with chronic obstructive pulmonary disease, asthma, bronchiectasis, interstitial lung disease, and chronic hypoxemic respiratory disorders. These patients often already have autonomic dysfunction, systemic inflammation, pulmonary vascular stress, right ventricular strain, myocardial ischemia, or other cardiovascular disease. Because electronic-cigarette aerosol can contain nicotine, ultrafine particles, carbonyl compounds, metals, and flavour-related chemicals, there are several plausible pathways through which vaping could affect cardiac rhythm and myocardial function. Yet the extent to which findings from existing human studies apply to adults with established chronic lung disease remains uncertain.

Objective: To critically evaluate human evidence on electrocardiographic, autonomic and echocardiographic findings associated with electronic-cigarette use, determine its applicability to adults with chronic pulmonary disorders, and identify clinically important evidence gaps.

Methods: A focused literature search of PubMed/MEDLINE was conducted from database inception through January, 2026. Search concepts included electronic cigarettes, vaping, electronic nicotine delivery systems, electrocardiography, QT interval, Tpeak–Tend interval, ventricular repolarization, arrhythmia, heart-rate variability, echocardiography, ventricular function, pulmonary hypertension, COPD, asthma, bronchiectasis and interstitial lung disease. Reference lists of relevant original investigations and scientific statements were examined. Adult human studies reporting ECG, autonomic, hemodynamic or echocardiographic outcomes after acute or habitual electronic-cigarette exposure were included. Because of substantial clinical and methodological heterogeneity, quantitative pooling was not performed.

Results: No prospective human study was identified that specifically recruited adults with an established chronic pulmonary disorder who regularly used electronic cigarettes and comprehensively evaluated predefined ECG and echocardiographic outcomes. Evidence from generally healthy electronic-cigarette users and combustible-cigarette smokers indicates that nicotine-containing vaping can acutely increase heart rate and blood pressure and shift cardiac autonomic balance toward sympathetic predominance. Several small studies reported prolongation of Tpeak–Tend, Tpeak–Tend/QT, Tpeak–Tend/QTc or QTc indices, although baseline and acute findings were inconsistent. A 2025 meta-analysis identified statistically higher repolarization indices in electronic-cigarette users, but it included only three heterogeneous studies and did not demonstrate increased clinical arrhythmia. Human echocardiographic evidence was limited predominantly to one acute study using an older-generation device. That investigation reported no significant immediate deterioration in measured left ventricular function after electronic-cigarette use, whereas combustible-cigarette smoking adversely affected several diastolic and myocardial performance indices. Evidence concerning right ventricular function, pulmonary artery pressure, ventricular strain, chronic cardiac remodeling, ambulatory arrhythmia burden or cardiovascular mortality in adults with chronic lung disease was absent or insufficient.

Conclusions: Electronic-cigarette use produces measurable acute autonomic and hemodynamic effects and may alter selected ECG repolarization indices. Nevertheless, the magnitude, persistence and clinical importance of these abnormalities in adults with chronic pulmonary disorders remain unknown. Existing findings should not be interpreted as proof of clinically significant arrhythmia, pulmonary hypertension or ventricular dysfunction. Routine ECG or echocardiographic screening solely because of electronic-cigarette use is not currently supported. Cardiac investigation should be guided by symptoms, pulmonary disease severity, hypoxemia, cardiovascular risk and established clinical indications. Carefully designed prospective studies focusing on right ventricular–pulmonary vascular interactions and ambulatory rhythm outcomes are urgently required.

Keywords: electronic cigarette; vaping; electrocardiography; echocardiography; ventricular repolarization; heart-rate variability; chronic obstructive pulmonary disease; asthma; pulmonary hypertension; right ventricular function

1. INTRODUCTION

Electronic cigarettes, or electronic nicotine delivery systems, are battery-powered devices that heat a liquid and

produce an aerosol for inhalation. Most liquids contain propylene glycol, vegetable glycerin, nicotine, and flavouring agents, although products labelled nicotine-free may still contain nicotine or other biologically active substances. Because these devices do not burn tobacco, they reduce exposure to many combustion-related toxicants. That reduction, however, does not make the aerosol chemically or physiologically harmless [1-3].

Electronic-cigarette devices have evolved from low-power first-generation products resembling conventional cigarettes to refillable tanks, high-power modifiable devices, nicotine-salt pod systems and disposable products capable of delivering high nicotine concentrations. Aerosol composition and nicotine delivery differ substantially according to device power, coil temperature, liquid composition, nicotine formulation, user experience and puffing behaviour [4-6]. Consequently, studies performed with older low-power devices cannot be assumed to represent contemporary products.

Interest in the cardiovascular effects of electronic cigarettes has grown as their use has expanded. Experimental studies have reported short-term increases in heart rate and blood pressure, changes in autonomic regulation, endothelial dysfunction, oxidative stress, and alterations in selected ECG repolarization indices [1,7-10]. Most of this work, however, has been carried out in young or middle-aged adults without major cardiopulmonary disease. These findings may not translate directly to patients with limited respiratory reserve or established cardiovascular disease.

The question is particularly important in adults with chronic pulmonary disorders. Chronic obstructive pulmonary disease is commonly associated with previous or continuing cigarette smoking, systemic comorbidity, exercise limitation and an increased risk of cardiac disease [11]. Patients with COPD, interstitial lung disease, bronchiectasis, chronic hypoxemia or sleep-related breathing disorders may also develop pulmonary hypertension and right ventricular dysfunction [12]. Patients with asthma may experience episodic hypoxemia, tachycardia during exacerbations and cardiovascular effects from β_2-agonists, systemic corticosteroids or other medications.

These underlying abnormalities create several potential interactions with electronic-cigarette exposure. Nicotine-induced sympathetic activation could increase heart rate and myocardial oxygen demand in a patient with limited cardiopulmonary reserve. Aerosol-induced airway irritation could worsen ventilation–perfusion mismatch or increase respiratory effort. Hypoxemia and hyperinflation could amplify right ventricular stress. Concomitant bronchodilator use, electrolyte abnormalities and QT-prolonging medications could influence ventricular repolarization independently of vaping.

Biological plausibility alone is not proof of clinical harm. A change in heart-rate variability or the Tpeak-Tend interval does not necessarily mean that a patient will develop atrial fibrillation, ventricular tachycardia, or sudden cardiac death. Likewise, the absence of an immediate fall in left ventricular ejection fraction after a brief vaping session cannot be taken as evidence of long-term cardiac safety.

The objectives of this focused meta-analysis were to:

1. Summarize human ECG, autonomic, hemodynamic and echocardiographic findings associated with electronic-cigarette use.
2. Determine whether these findings have been directly demonstrated in adults with chronic pulmonary disorders.
3. Critically evaluate study design, exposure measurement, outcome selection and clinical applicability.
4. Identify appropriate clinical interpretations without overstating causality
5. Propose priorities for future cardiopulmonary research.

2. METHODS

2.1 Study design

This article was prepared as a focused evidence analysis and critical appraisal. It was not prospectively registered and was not intended to fulfil all methodological requirements of a formal meta-analysis. This approach was chosen because the primary question was narrow, the anticipated literature was heterogeneous, and direct evidence in the target population was expected to be limited.

2.2 Literature-search strategy

PubMed/MEDLINE was searched from database inception through January, 2026. Search terms were combined using Boolean operators and included:

- “electronic cigarette”
- “e-cigarette”
- “vaping”
- “electronic nicotine delivery system”
- “electrocardiogram”
- “electrocardiography”
- “ECG”
- “QT interval”
- “corrected QT”
- “Tpeak–Tend”
- “Tp-e”
- “ventricular repolarization”
- “arrhythmia”
- “heart-rate variability”
- “autonomic function”
- “echocardiography”
- “ventricular function”
- “myocardial strain”
- “right ventricular function”
- “pulmonary hypertension”
- “chronic obstructive pulmonary disease”
- “COPD”
- “asthma”
- “bronchiectasis”
- “interstitial lung disease”
- “chronic respiratory disease”

Reference lists of relevant original studies, scientific statements and clinical guidelines were manually examined to identify additional reports.

2.3 Eligibility criteria

Studies were considered relevant when they:

1. Enrolled human adults aged 18 years or older.
2. Evaluated acute, habitual or longitudinal electronic-cigarette exposure.
3. Reported at least one ECG, rhythm, autonomic, hemodynamic or echocardiographic endpoint.
4. Provided sufficient information to distinguish electronic-cigarette exposure from non-use or combustible-cigarette exposure
5. Included an original analysis or a structured synthesis of original human data.

Priority was given to studies involving adults with established chronic pulmonary disorders. When directly relevant studies were unavailable, indirect evidence from generally healthy electronic-cigarette users, former smokers, current smokers and medically vulnerable populations was examined.

2.4 Exclusion criteria

The main synthesis excluded:

- pediatric studies;
- pregnancy studies;
- animal experiments;

- in-vitro studies;
- isolated case reports;
- studies limited to acute electronic-cigarette or vaping-associated lung injury;
- reports lacking identifiable electronic-cigarette exposure;
- studies reporting only respiratory symptoms without relevant cardiovascular assessment; and
- conference abstracts without sufficient methodological detail.

Animal and laboratory evidence was considered only when discussing biological plausibility and was not used to estimate clinical cardiac risk.

2.5 Outcomes of interest

ECG and rhythm outcomes

- heart rate;
- PR interval;
- QRS duration;
- QT and corrected-QT intervals;
- Tpeak–Tend interval;
- Tpeak–Tend/QT ratio;
- Tpeak–Tend/QTc ratio;
- P-wave indices;
- premature atrial or ventricular complexes;
- supraventricular tachycardia;
- atrial fibrillation;
- ventricular tachycardia;
- ambulatory rhythm burden; and
- sudden cardiac death.

Autonomic and hemodynamic outcomes

- systolic and diastolic blood pressure;
- mean arterial pressure;
- heart-rate variability;
- high-frequency and low-frequency spectral components;
- low-frequency/high-frequency ratio;
- sympathetic nerve activity; and
- baroreflex-related measurements.

Heart-rate variability interpretation was considered in relation to established measurement standards [13].

Echocardiographic outcomes

- left ventricular ejection fraction;
- left ventricular volumes;
- transmitral Doppler indices;
- tissue-Doppler velocities;
- E/e’ ratio;
- myocardial performance index;
- left ventricular global longitudinal strain;
- right ventricular dimensions;
- tricuspid annular plane systolic excursion;

- right ventricular fractional area change;
- right ventricular tissue-Doppler S' velocity;
- right ventricular free-wall strain;
- tricuspid regurgitation velocity;
- estimated pulmonary artery systolic pressure;
- right atrial dimensions; and
- inferior vena cava measurements.

  Interpretation was informed by established chamber-quantification and right-heart echocardiographic recommendations [14,15].

2.6 Critical-appraisal framework

Each study was assessed qualitatively across the following domains:

- relevance of the population;
- acute versus chronic exposure;
- exposure characterization;
- device generation;
- nicotine concentration and formulation;
- combustible-cigarette history;
- dual use;
- comparator selection;
- biochemical verification;
- sample size;
- randomization and crossover design;
- outcome blinding;
- reproducibility of ECG or echocardiographic measurements;
- adjustment for cardiovascular and pulmonary confounders;
- multiple statistical comparisons;
- duration of follow-up; and
- clinical relevance of the endpoint.

2.7 Evidence synthesis

Because of major differences in populations, products, exposure protocols and outcome definitions, no additional meta-analysis was undertaken. Findings were synthesized narratively. Particular attention was given to the distinction between:

1. acute physiological responses;
2. persistent subclinical abnormalities;
3. structural cardiac disease; and
4. clinically important cardiovascular events.

3. Definitions and Interpretative Considerations

3.1 Acute exposure

Acute exposure studies generally assessed participants before and within minutes or hours after a laboratory-controlled vaping session. These studies are useful for identifying immediate autonomic, hemodynamic or electrophysiological responses. They cannot establish whether repeated exposure produces chronic structural disease or long-term clinical events.

3.2 Habitual use

Habitual-use studies generally compared people who regularly used electronic cigarettes with non-users or combustible-cigarette smokers. Such studies may identify persistent differences, but they are vulnerable to confounding by previous smoking, dual use, nicotine dependence, socioeconomic factors and baseline health characteristics.

3.3 Exclusive use, dual use and switching

Exclusive electronic-cigarette users report electronic-cigarette use without current combustible-cigarette smoking. Dual users use both products. Switchers are previous combustible-cigarette smokers who report replacing cigarettes with electronic cigarettes.

These categories are frequently unstable. A participant classified as an exclusive electronic-cigarette user at a single assessment may have substantial previous combustible-cigarette exposure or intermittent smoking that is not captured by questionnaire data. This is particularly important in COPD because cumulative cigarette exposure may independently explain both pulmonary and cardiovascular abnormalities.

3.4 Surrogate and clinical outcomes

Heart-rate variability, QTc and Tpeak–Tend-derived measurements are surrogate physiological endpoints. They may provide mechanistic information but do not directly establish clinically meaningful arrhythmia.

By contrast, sustained atrial fibrillation, ventricular tachycardia, syncope, hospitalization, heart failure, cardiovascular death and sudden cardiac death are clinical outcomes. The current electronic-cigarette ECG literature is dominated by surrogate measurements rather than these clinical endpoints.

4. Biological Plausibility of Cardiac Effects

4.1 Nicotine-mediated sympathetic activation

Nicotine stimulates nicotinic acetylcholine receptors within autonomic ganglia and the adrenal medulla, increasing catecholamine release. Acute effects can include tachycardia, increased blood pressure, vasoconstriction and increased myocardial oxygen demand [1,7,8].

The delivered nicotine dose varies widely. Experienced users of modern devices may achieve systemic nicotine concentrations approaching those produced by combustible cigarettes, whereas inexperienced participants using older devices may receive substantially less nicotine [6]. Apparent differences between electronic cigarettes and combustible cigarettes can therefore reflect unequal nicotine exposure rather than an inherent difference between the two product categories.

In adults with chronic pulmonary disease, nicotine-related sympathetic activation may be clinically more relevant when accompanied by:

- chronic hypoxemia;
- pulmonary hypertension;
- coronary artery disease;
- heart failure;
- reduced exercise capacity;
- β_2-agonist use;
- anxiety-related hyperventilation; or
- acute respiratory exacerbation.

4.2 Particulate and chemical exposure

Electronic-cigarette aerosol may contain ultrafine particles, aldehydes, volatile organic compounds, metals and flavour-related chemicals [4,5]. The concentrations depend on device power, heating conditions, coil composition and liquid ingredients.

Carbonyl compounds such as formaldehyde, acetaldehyde and acrolein can be generated during thermal degradation of propylene glycol and vegetable glycerin. Higher power and overheating may increase emissions [5]. Metals may originate from the heating coil or other device components.

These constituents could theoretically contribute to oxidative stress, endothelial dysfunction, inflammation and altered vascular reactivity. Nevertheless, aerosol composition measured under laboratory conditions does not directly determine the absorbed dose or clinical cardiovascular risk.

4.3 Oxidative stress and endothelial dysfunction

Human studies have reported acute oxidative and endothelial changes after vaping [17,28-32]. Endothelial dysfunction may reduce vasodilatory reserve and contribute to vascular injury. In adults with chronic pulmonary disease, systemic inflammation, previous smoking and reduced physical activity may already impair vascular function, creating a potential cumulative effect.

However, endothelial findings remain indirect to the present analysis because they are not ECG or echocardiographic endpoints and do not establish ventricular dysfunction or arrhythmia.

4.4 Autonomic imbalance

Cardiac autonomic regulation influences sinus-node activity, atrioventricular conduction, ventricular electrophysiology and vascular tone. Reduced vagal modulation or increased sympathetic predominance has been associated with adverse cardiovascular prognosis in several disease settings, although the prognostic meaning of short-term electronic-cigarette–induced changes remains uncertain [13,17,18,34].

Patients with COPD may already demonstrate altered autonomic regulation owing to hypoxemia, hyperinflation, respiratory effort, sleep-disordered breathing and medication exposure. Electronic-cigarette effects in this setting have not been directly measured.

4.5 Ventricular repolarization

The QT interval represents the combined duration of ventricular depolarization and repolarization. QTc attempts to correct QT for heart rate. Its value depends partly on the correction formula used.

Tpeak–Tend is generally measured from the peak to the end of the T wave and has been proposed as a marker of dispersion of ventricular repolarization. Tp-e/QT and Tp-e/QTc ratios have been proposed to reduce some heart-rate dependence. These indices are affected by:

- lead selection;
- T-wave morphology;
- U waves;
- measurement technique;
- posture;
- respiration;
- heart rate;
- autonomic tone; and
- correction formula.

A small statistically significant change in one repolarization index does not automatically indicate a clinically meaningful increase in ventricular arrhythmia.

4.6 Pulmonary vascular and right-heart pathways

Chronic pulmonary disease can increase right ventricular afterload through hypoxic pulmonary vasoconstriction, destruction of the pulmonary vascular bed, hyperinflation, increased intrathoracic pressure, thromboembolic disease or pulmonary vascular remodeling [12].

Electronic-cigarette exposure could theoretically influence this system by:

- worsening airway resistance;
- altering ventilation–perfusion matching;
- provoking transient hypoxemia;
- increasing sympathetic tone;
- impairing endothelial function; or
- increasing pulmonary vascular reactivity.

Direct human evidence showing that routine electronic-cigarette use increases pulmonary artery pressure or causes right ventricular remodeling is currently lacking.

5. Evidence Map

The literature fell into three broad evidence categories:

1. **Direct electronic-cigarette ECG and autonomic studies:** small acute experiments and cross-sectional studies involving generally healthy adults or combustible-cigarette smokers.
2. **Direct electronic-cigarette echocardiographic studies:** extremely limited, with one principal acute human investigation.
3. **Pulmonary-disease electronic-cigarette studies:** observational or small follow-up studies focused predominantly on respiratory symptoms, exacerbations, smoking behaviour or clinical events rather than standardized ECG and echocardiographic endpoints.

We found no study that comprehensively combined:

- confirmed chronic pulmonary disease;
- well-characterized electronic-cigarette exposure;
- standardized ECG;
- ambulatory rhythm monitoring;
- left and right ventricular echocardiography;
- pulmonary pressure assessment; and
- longitudinal clinical follow-up.

6. Principal Human ECG, Autonomic and Echocardiographic Studies

Table 1. Principal human studies relevant to ECG, autonomic or echocardiographic effects

Study	Design and participants	Exposure and outcomes	Principal findings	Major limitations
Farsalinos et al., 2014 [16]	Acute study involving 36 combustible-cigarette smokers and 40 established electronic-cigarette users	Doppler, tissue-Doppler and strain-rate assessment before and after smoking or vaping	Combustible smoking adversely affected several diastolic and myocardial performance indices. Electronic-cigarette use did not significantly worsen the measured LV indices	Older-generation device; brief exposure; no chronic lung disease; unequal product exposures; limited right-heart evaluation; no longitudinal outcomes
Moheimani et al., 2017 [17]	Cross-sectional comparison of 23 habitual electronic-cigarette users and 19 non-users	HRV and oxidative-stress measurements	Habitual users had reduced high-frequency variability and increased LF/HF ratio, compatible with sympathetic predominance	Small, young sample; observational design; previous exposure and lifestyle confounding; no clinical arrhythmia outcome
Moheimani et al., 2017 [18]	Randomized acute exposure study	Electronic cigarette with nicotine, electronic cigarette without nicotine and sham control	Nicotine-containing exposure increased heart rate and shifted cardiac autonomic balance toward sympathetic predominance	Short exposure; healthy participants; limited generalizability to chronic pulmonary disease
Ip et al., 2020 [19]	Comparison of 37 smokers, 43 electronic-cigarette	QT, QTc, Tp-e and related ratios	Baseline indices were generally similar; acute nicotine-containing exposures altered	Surrogate endpoints; heterogeneous exposure groups;

	users and 65 non-users, with acute exposure components		selected repolarization measures, with stronger changes after combustible smoking	uncertain clinical importance
Demir et al., 2020 [20]	36 electronic-cigarette users and 40 controls	Twelve-lead ECG before and after vaping	Tp-e, Tp-e/QT and Tp-e/QTc were prolonged in the electronic-cigarette group	Small non-randomized study; manual measurement; limited device and nicotine characterization
Arastoo et al., 2020 [21]	58 chronic electronic-cigarette users and 42 combustible-cigarette smokers	Acute nicotine-containing and nicotine-free exposures; blood pressure, heart rate and HRV	Nicotine-containing vaping increased heart rate and blood pressure; nicotine-free exposure had smaller effects	No comprehensive non-user comparison; physiological endpoints; no chronic pulmonary disease
Ruedisueli et al., 2023 [22]	Acute controlled exposure involving people who smoked combustible cigarettes and comparison groups	Supine and standing ventricular repolarization indices	Electronic-cigarette exposure prolonged several repolarization indices compared with control; posture and sex influenced findings	Subgroup findings; multiple comparisons; conventional smokers rather than pulmonary-disease patients
Ammar et al., 2024 [23]	Cross-sectional study of 105 healthy adults, 35 per group	Electronic-cigarette users, combustible smokers and non-smokers; standard ECG	Electronic-cigarette users and smokers had higher heart rates and longer QT, QTc and Tp-e-derived indices than non-smokers	Cross-sectional design; modest sample; convenience sampling; no clinical events
Kucera et al., 2024 [24]	Acute experimental study	Different nicotine formulations and autonomic/repolarization outcomes	Nicotine formulation influenced autonomic and electrophysiological responses	Acute exposure; product-specific effects; no chronic pulmonary disease
Ruedisueli et al., 2024 [25]	Randomized crossover study in 37 people living with HIV who smoked	Electronic cigarette, combustible cigarette and control exposures	Both products increased heart rate. HRV effects were less marked after electronic-cigarette exposure; repolarization differences were not significant	Predominantly male; lower nicotine delivery during vaping; no chronic pulmonary-disease cohort
de Araújo et al., 2025 [26]	Systematic review and meta-analysis of three studies, 254 participants	QT and Tp-e-derived indices	Electronic-cigarette use was associated with higher repolarization indices	Only three studies; substantial heterogeneity; small samples; surrogate outcomes; no pulmonary-disease subgroup
Sobieraj et al., 2026 [27]	Randomized crossover study in 42 apparently healthy adults with cigarette-smoking history	Nicotine-containing electronic cigarette, nicotine-free electronic cigarette and combustible cigarette	Nicotine-containing vaping increased heart rate and blood pressure and produced sympathetic predominance; nicotine-free exposure had smaller but not uniformly absent effects	Brief exposure; apparently healthy adults; no structural or clinical outcomes

7. Electrocardiographic Findings

7.1 Heart rate

An acute increase in heart rate is the most consistent cardiovascular response to nicotine-containing electronic-cigarette use [9,10,18,21,25,27,34]. The magnitude varies according to nicotine delivery, participant experience, product characteristics and exposure duration.

The finding is physiologically plausible because nicotine increases catecholamine release. Nicotine-free aerosol generally produces smaller heart-rate changes, although some studies suggest that non-nicotine constituents or the inhalation procedure itself may not be completely neutral [21,27].

In adults with chronic pulmonary disorders, interpretation is complicated by other causes of tachycardia:

- hypoxemia;
- fever or infection;
- respiratory distress;
- dehydration;
- anemia;
- β_2-agonist therapy;
- anticholinergic treatment;
- systemic corticosteroids;
- pulmonary embolism;
- heart failure; and
- anxiety.

No study has established a heart-rate threshold at which an electronic-cigarette response becomes clinically dangerous in COPD, asthma, bronchiectasis or interstitial lung disease.

7.2 PR interval and atrioventricular conduction

PR interval and atrioventricular conduction have not been consistent primary outcomes in electronic-cigarette studies. The current literature does not show that vaping causes persistent first-degree atrioventricular block, higher-grade block or clinically important conduction-system disease.

Because nicotine increases sympathetic activity, an acute shortening rather than prolongation of atrioventricular conduction could be physiologically possible. However, published evidence is insufficient for a reliable conclusion.

7.3 QRS duration

There is no consistent human evidence that routine electronic-cigarette use widens the QRS complex or produces bundle-branch block. QRS prolongation in a patient with chronic lung disease should therefore prompt evaluation for established causes such as:

- pre-existing conduction disease;
- ischemia;
- cardiomyopathy;
- electrolyte disturbance;
- antiarrhythmic or psychotropic medication;
- ventricular pacing; or
- acute toxic exposure.

7.4 QT and corrected QT intervals

QT and QTc findings vary among studies. Ip et al. did not identify a uniform baseline prolongation across all exposure groups, although acute product use affected selected repolarization indices [19]. Ammar et al. reported longer QT and QTc measurements among habitual electronic-cigarette users than non-smokers [23]. The 2025 meta-analysis also identified a statistically higher pooled QT measurement among electronic-cigarette users, but its evidentiary base was small and heterogeneous [26].

Several methodological issues affect interpretation:

1. QT shortens as heart rate increases.
2. Bazett correction may overcorrect QT during tachycardia.
3. Fridericia or alternative correction formulas may produce different results.
4. Acute nicotine exposure simultaneously changes heart rate and autonomic tone.
5. Manual identification of the end of the T wave may be difficult.
6. Posture and respiration can influence measurement.
7. Multiple leads may yield different values.

Studies should therefore report the raw QT, heart rate, correction formula and preferably more than one QTc calculation.

7.5 Tpeak–Tend interval

Demir et al. reported prolongation of Tp-e and Tp-e-derived ratios following electronic-cigarette exposure [20]. Ip et al. found acute changes in selected indices but did not demonstrate a uniform baseline abnormality in habitual users [19]. Ruedisueli et al. observed prolongation of several repolarization indices after acute exposure, with findings affected by posture and sex [22]. Ammar et al. reported longer Tp-e measurements in habitual users than non-smokers [23].

The 2025 meta-analysis pooled three studies and found statistically greater Tp-e, Tp-e/QT and Tp-e/QTc measurements among electronic-cigarette users [26]. However, this conclusion requires cautious interpretation because:

- only three studies were included;
- sample sizes were small;
- exposure definitions differed;
- measurement procedures varied;
- heterogeneity was substantial for several outcomes;
- publication bias could not be reliably assessed; and
- clinical arrhythmias were not evaluated.

The evidence supports a possible effect on ventricular repolarization but not a demonstrated increase in ventricular tachycardia or sudden death.

7.6 P-wave indices and atrial electrophysiology

Human electronic-cigarette research has focused primarily on ventricular rather than atrial electrophysiology. Evidence concerning:

- P-wave duration;
- P-wave dispersion;
- PR variability;
- atrial electromechanical delay; and
- signal-averaged atrial indices

is insufficient.

This gap is clinically important because atrial fibrillation is common in older adults with COPD and may be precipitated by hypoxemia, infection, pulmonary hypertension, adrenergic stimulation and bronchodilator use. Whether electronic-cigarette exposure adds materially to this risk is unknown.

7.7 Premature atrial and ventricular complexes

Small experimental studies were not designed to quantify premature atrial or ventricular complexes over prolonged periods. A brief resting ECG is poorly suited to evaluating intermittent ectopy. No reliable exposure-response relationship has been established between vaping frequency and premature ventricular complex burden.

7.8 Clinically documented arrhythmia

Prospective evidence remains too limited to assess:

- incident atrial fibrillation;
- atrial flutter;
- supraventricular tachycardia;
- sustained ventricular tachycardia;
- ventricular fibrillation;
- clinically significant bradyarrhythmia;
- implantable-device–detected arrhythmia;
- syncope associated with confirmed arrhythmia; or
- sudden cardiac death.

Therefore, claims that electronic cigarettes cause clinically significant arrhythmia in adults with chronic pulmonary disease would exceed the available evidence.

8. Cardiac Autonomic Findings

8.1 Habitual-use evidence

Moheimani et al. compared habitual electronic-cigarette users with non-users and found lower high-frequency HRV and a higher low-frequency/high-frequency ratio among users [17]. These findings were interpreted as reduced cardiac vagal modulation and relative sympathetic predominance.

The study also reported increased oxidative-stress markers, providing a potential link between autonomic and vascular effects. However, the study was small, observational and restricted to relatively young participants without known cardiopulmonary disease.

8.2 Acute nicotine exposure

In an acute randomized study, nicotine-containing electronic-cigarette exposure increased heart rate and altered HRV in a direction consistent with sympathetic predominance, whereas nicotine-free exposure did not produce the same magnitude of response [18].

Arastoo et al. similarly reported acute sympathomimetic effects from nicotine-containing vaping [21]. The 2026 randomized crossover study by Sobieraj et al. reinforced the finding that nicotine-containing electronic-cigarette use increases heart rate and blood pressure and shifts autonomic indices toward sympathetic activity [27].

8.3 Comparison with combustible cigarettes

Several studies found that combustible cigarettes produced equal or greater autonomic and hemodynamic effects than electronic cigarettes [9,21,25,27,34]. Interpretation requires careful matching of nicotine dose. Lower responses after vaping may partly reflect lower systemic nicotine exposure under the specific laboratory protocol.

Relative reduction compared with combustible cigarettes does not establish absence of effect compared with non-use.

8.4 Clinical significance of HRV changes

Short-term HRV alterations are mechanistically informative but are not equivalent to an adverse clinical event. HRV is influenced by:

- breathing rate;
- posture;
- age;
- physical fitness;
- caffeine;
- recent meals;
- sleep;
- anxiety;

- medications;
- arrhythmia;
- recording duration; and
- analytic method [13].

These variables are particularly relevant in chronic pulmonary disease, where irregular respiration, hyperinflation and bronchodilator use can affect autonomic measurements.

8.5 Relevance to chronic pulmonary disease

Autonomic dysregulation may be more clinically important in patients with:

- severe COPD;
- chronic hypoxemia;
- obstructive sleep apnea;
- pulmonary hypertension;
- ischemic heart disease;
- heart failure;
- frequent exacerbations; or
- high-dose sympathomimetic therapy.

However, no study has directly measured whether vaping produces a larger or more prolonged HRV abnormality in these subgroups.

9. Hemodynamic and Vascular Findings

Although vascular outcomes were not the primary focus of this analysis, they provide context for interpreting ECG and echocardiographic findings.

9.1 Blood pressure

Meta-analyses and controlled experiments indicate that nicotine-containing electronic-cigarette exposure can acutely increase systolic and diastolic blood pressure [9,10,18,21,27,34]. Effects after nicotine-free exposure are generally smaller.

Persistent hypertension attributable specifically to long-term electronic-cigarette use has not been conclusively demonstrated because habitual users frequently have previous or concurrent cigarette exposure.

9.2 Endothelial function

Antoniewicz et al. found acute vascular and airway changes after electronic-cigarette inhalation [28]. Carnevale et al. reported acute oxidative stress and vascular dysfunction following both combustible-cigarette and electronic-cigarette exposure, although the magnitude differed [29]. Quantitative MRI studies have also demonstrated transient vascular changes after electronic-cigarette aerosol inhalation [30].

In the VESUVIUS trial, smokers who switched from combustible cigarettes to electronic cigarettes showed improvement in endothelial function compared with those who continued smoking [31]. This finding supports potential relative benefit when exposure to combustible smoke is completely replaced. It does not establish that electronic-cigarette use is equivalent to abstinence.

Other investigations have found endothelial impairment after electronic-cigarette exposure in otherwise healthy participants [32]. Twenty-four-hour studies in dual users have demonstrated continuing cardiovascular effects during electronic-cigarette use [33].

9.3 Implications for echocardiographic interpretation

Acute changes in blood pressure, heart rate and vascular resistance alter cardiac loading conditions.

Echocardiographic measurements obtained immediately after vaping may therefore reflect transient hemodynamic changes rather than direct myocardial injury.

Future studies should measure:

- blood pressure;

- heart rate;
- oxygen saturation;
- nicotine concentration;
- recent bronchodilator use; and
- respiratory pattern

at the time of echocardiography.

10. Echocardiographic Findings

10.1 Overview

Echocardiographic evidence is substantially less developed than ECG and autonomic evidence. The literature fell into one principal human study specifically designed to assess acute myocardial function after electronic-cigarette use [16].

No robust longitudinal study was identified that evaluated chronic vaping in relation to:

- left ventricular remodeling;
- right ventricular remodeling;
- progressive diastolic dysfunction;
- global longitudinal strain;
- pulmonary artery pressure;
- incident heart failure; or
- serial echocardiographic deterioration.

10.2 Acute left ventricular systolic function

Farsalinos et al. did not identify significant acute deterioration in conventional left ventricular systolic measurements after electronic-cigarette use [16]. Left ventricular ejection fraction remained preserved.

This result needs a narrow interpretation. It indicates that the specific brief exposure using the studied device did not produce a detectable immediate decline in the measured variables. It does not exclude:

- subtle subclinical dysfunction;
- effects from higher nicotine concentrations;
- effects from modern high-power devices;
- cumulative myocardial injury;
- exercise-induced abnormalities;
- effects in patients with pre-existing disease; or
- long-term heart failure risk.

10.3 Left ventricular diastolic function

In the same study, conventional cigarette smoking adversely affected several Doppler and tissue-Doppler measurements, including indices related to isovolumic relaxation and myocardial performance. Comparable acute deterioration was not observed after electronic-cigarette use [16].

Important limitations include:

- non-equivalent toxicant and nicotine exposure;
- acute rather than chronic assessment;
- generally healthy participants;
- a relatively old electronic-cigarette product;
- limited sample size; and
- absence of patients with chronic pulmonary disease.

No study has established the long-term relationship between vaping and:

- E/A ratio;
- septal or lateral e' velocity;
- average E/e';
- left atrial volume;
- pulmonary venous flow;
- diastolic dysfunction grade; or
- exercise-induced elevation of filling pressure.

10.4 Left ventricular global longitudinal strain

Global longitudinal strain may identify subclinical systolic dysfunction despite preserved ejection fraction. No sufficiently validated longitudinal evidence was identified regarding chronic electronic-cigarette exposure and left ventricular global longitudinal strain in adults with chronic pulmonary disease.

This is an important area for future research because strain imaging may detect changes earlier than ejection fraction.

10.5 Myocardial performance index

The myocardial performance index combines systolic and diastolic time intervals. Conventional smoking adversely affected this measurement in the acute echocardiographic study, whereas electronic-cigarette use did not produce the same significant deterioration [16].

The index is influenced by heart rate, loading conditions and measurement technique. It should not be viewed as a direct measure of myocardial injury.

10.6 Left atrial structure and function

There is little meaningful evidence on chronic electronic-cigarette use and:

- left atrial volume index;
- left atrial reservoir strain;
- left atrial conduit function;
- left atrial contractile strain; or
- atrial remodeling.

These outcomes are relevant to atrial fibrillation and heart failure with preserved ejection fraction, both of which may occur in older adults with chronic lung disease.

10.7 Right ventricular size

No eligible study comprehensively evaluated right ventricular dimensions in adults with chronic pulmonary disease who vape. This lack of data is particularly important because the right ventricle may be affected by chronic hypoxemia, pulmonary vascular disease and lung hyperinflation.

Recommended measurements for future studies include:

- right ventricular basal diameter;
- mid-cavity diameter;
- longitudinal dimension;
- right ventricular end-diastolic area; and
- right ventricular wall thickness [14,15].

10.8 Right ventricular systolic function

Evidence remains insufficient for:

- tricuspid annular plane systolic excursion;
- right ventricular fractional area change;
- tissue-Doppler S' velocity;

- right ventricular index of myocardial performance;
- three-dimensional right ventricular ejection fraction; or
- right ventricular free-wall strain.

A normal left ventricular ejection fraction cannot exclude clinically important right ventricular dysfunction.

10.9 Pulmonary artery pressure

Current evidence does not show that routine electronic-cigarette use increases resting pulmonary artery pressure in adults with chronic pulmonary disease.

Future protocols should measure:

- peak tricuspid regurgitation velocity;
- estimated right atrial pressure;
- calculated pulmonary artery systolic pressure;
- pulmonary acceleration time;
- right ventricular outflow-tract Doppler profile;
- pulmonary artery dimensions; and
- additional echocardiographic signs of pulmonary hypertension [12,15].

Echocardiography estimates the probability of pulmonary hypertension but does not replace right-heart catheterization when definitive hemodynamic diagnosis is required [12].

10.10 Exercise echocardiography

Resting echocardiography may miss abnormalities that appear during exertion. Exercise echocardiography could be valuable in patients with unexplained dyspnea, because vaping-related autonomic or vascular effects might interact with exercise-induced hypoxemia or pulmonary pressure elevation.

No relevant electronic-cigarette study using exercise echocardiography was identified.

11. Evidence in Chronic Pulmonary Disorders

11.1 COPD

COPD is the chronic pulmonary disorder for which the largest amount of electronic-cigarette observational evidence is available. However, this evidence has focused predominantly on product use, respiratory symptoms, exacerbations, lung function and clinical outcomes rather than standardized cardiac testing.

Bowler et al. analyzed electronic-cigarette use in two observational cohorts of adults at risk for or with COPD [35]. Electronic-cigarette users often had heavier smoking histories and greater respiratory morbidity. The study illustrated the difficulty of separating electronic-cigarette effects from cigarette exposure and disease severity.

Small follow-up studies by Polosa et al. reported reduced conventional-cigarette consumption and improvements in selected respiratory outcomes among COPD smokers who switched to electronic cigarettes [36-38]. These studies involved relatively small, selected cohorts and were not designed to detect uncommon cardiovascular outcomes or characterize ECG and echocardiographic changes.

Large cross-sectional analyses reported associations between electronic-cigarette use and self-reported COPD [39-41]. Such findings are vulnerable to:

- reverse causation;
- residual cigarette-smoking confounding;
- inaccurate self-reported diagnosis;
- dual use;
- differences in healthcare access; and
- switching after respiratory symptoms develop.

A longitudinal PATH analysis found an association between electronic-cigarette use and subsequent respiratory diagnoses [42]. Although longitudinal design reduces some limitations of cross-sectional studies, exposure instability and previous smoking remain important concerns.

A 2025 systematic review and meta-analysis reported an association between electronic-cigarette exposure and COPD [43]. Interpretation remains limited by the predominance of observational studies, substantial smoking-related confounding and variable COPD definitions.

A nationwide 2026 cohort study evaluated outcomes among patients with COPD who continued smoking, stopped smoking or switched to electronic cigarettes [44]. Switching was associated with a lower risk of major adverse cardiovascular and cerebrovascular events than continued smoking, but it did not provide the same respiratory benefits as complete cessation. The study is clinically important because it directly involved patients with COPD and evaluated hard outcomes. Nevertheless:

1. It was observational.
2. Exposure was based on smoking-behaviour categories rather than detailed device or dose measurements.
3. It did not report ECG or echocardiographic outcomes.
4. Switching may have been associated with unmeasured health behaviours.
5. A reduction relative to continued smoking does not imply equivalence to complete abstinence.

11.2 Asthma

We found no study that prospectively evaluated ECG and echocardiographic findings specifically in adults with chronic asthma who use electronic cigarettes.

Potential confounders include:

- short-acting β_2-agonists;
- long-acting β_2-agonists;
- anticholinergic agents;
- systemic corticosteroids;
- theophylline;
- hypoxemia during exacerbations;
- anxiety;
- obesity; and
- sleep-disordered breathing.

These factors can affect heart rate, potassium concentration, QTc and ectopy. Future studies must record medication timing and asthma control when evaluating electronic-cigarette effects.

11.3 Bronchiectasis

No direct ECG or echocardiographic study was found in adults with bronchiectasis who use electronic cigarettes.

Bronchiectasis patients may have recurrent infection, systemic inflammation, hemoptysis, hypoxemia and pulmonary hypertension. Macrolide antibiotics, fluoroquinolones and other medications used during exacerbations may prolong QT. Any ECG study must distinguish electronic-cigarette exposure from these competing influences.

11.4 Interstitial lung disease

We found no directly relevant study in interstitial lung disease.

Patients with fibrotic interstitial lung disease may develop severe exertional desaturation and pulmonary hypertension. Right ventricular strain and pulmonary pressure assessment would therefore be particularly important. Results from healthy young electronic-cigarette users cannot be confidently extrapolated to this population.

11.5 Chronic hypoxemic respiratory failure

No study evaluated acute vaping during long-term oxygen therapy or in chronic hypoxemic respiratory failure. Such exposure raises both physiological and fire-safety concerns. From a cardiac perspective, the interaction among nicotine, hypoxemia, pulmonary vascular resistance and right ventricular function remains unknown.

11.6 Pulmonary hypertension

No prospective electronic-cigarette study was identified in patients with confirmed pulmonary hypertension. This

gap matters because small changes in heart rate, systemic pressure, oxygenation or pulmonary vascular tone could have different consequences in patients with limited right ventricular reserve.

11.7 Sleep-disordered breathing and overlap syndromes

No study specifically assessed electronic-cigarette ECG or echocardiographic effects in COPD–obstructive sleep apnea overlap syndrome. Intermittent nocturnal hypoxemia and sympathetic activation could theoretically interact with nicotine exposure, but this has not been demonstrated.

12. Critical Appraisal

12.1 Severe Indirectness

The clearest limitation is that the study populations do not closely match the patients of interest. Most ECG and echocardiographic studies have involved young adults, healthy habitual electronic-cigarette users, non-smokers exposed experimentally, combustible-cigarette smokers, or medically selected populations without established chronic pulmonary disease. Adults with severe COPD, long-term oxygen dependence, interstitial lung disease, pulmonary hypertension, chronic hypoxemic respiratory failure, or multiple cardiovascular comorbidities have been markedly underrepresented. For this reason, findings from relatively healthy participants cannot be confidently extrapolated to patients with substantially reduced cardiopulmonary reserve.

12.2 Small Sample Sizes

Most mechanistic studies enrolled fewer than 150 participants, and several included fewer than 50. Such small sample sizes reduce statistical precision and increase the influence of individual outliers. They also increase the likelihood of baseline imbalances between comparison groups and may generate false-positive findings, particularly in subgroup analyses. More importantly, studies of this size are unable to evaluate uncommon but clinically important outcomes such as sustained arrhythmia, heart-failure hospitalization, sudden cardiac death, or cardiovascular mortality.

12.3 Exposure Heterogeneity

Electronic cigarettes should not be viewed as a single uniform exposure because products differ substantially in their technical characteristics and aerosol composition. Relevant variations include device generation, power output, coil resistance, temperature-control settings, nicotine concentration, nicotine-salt versus free-base formulations, and the ratio of propylene glycol to vegetable glycerin. Exposure may also differ according to flavouring substances, puff duration, puff interval, user experience, total liquid consumption, and product modification. These differences may significantly influence nicotine delivery, aerosol temperature, particle concentration, and toxicant generation, thereby limiting comparability among studies.

12.4 Inadequate Nicotine Matching

Comparisons between electronic cigarettes and combustible cigarettes frequently involve unequal nicotine delivery. A smaller cardiovascular response after electronic-cigarette exposure may therefore reflect a lower absorbed nicotine dose rather than a fundamental difference between the products. Without objective nicotine matching, it is difficult to determine whether observed differences result from aerosol composition, combustion-related toxicants, device characteristics, or dose alone. Future studies should measure plasma nicotine or cotinine concentrations and report pharmacokinetic profiles to allow more valid exposure comparisons.

12.5 Confounding by Previous Smoking

Previous combustible-cigarette smoking is a major source of bias because it can produce persistent airway disease, endothelial dysfunction, coronary atherosclerosis, autonomic abnormalities, myocardial injury, and pulmonary vascular disease. These abnormalities may remain present for years after smoking cessation. Therefore, even individuals classified as exclusive electronic-cigarette users may have substantial residual cardiovascular and pulmonary injury resulting from previous cigarette exposure. Unless lifetime smoking history is measured carefully and incorporated into the analysis, cardiac abnormalities may be incorrectly attributed to current electronic-cigarette use.

12.6 Dual Use

Dual use of electronic and combustible cigarettes is common and may maintain substantial exposure to combustion-related toxins. Individuals may alternate between products according to location, availability, symptoms, or personal preference. As a result, classification based on a single baseline assessment may not accurately represent exposure throughout the study period. Repeated assessment of product use is therefore necessary to distinguish persistent exclusive use, intermittent smoking, complete switching, and changing patterns of dual use.

12.7 Reverse Causation

Reverse causation is another important concern. Some patients may begin using electronic cigarettes or switch from combustible cigarettes after developing respiratory symptoms, COPD, cardiovascular disease, or advice from a healthcare professional. Cross-sectional studies may subsequently identify an association between vaping and disease but cannot determine whether electronic-cigarette exposure preceded the condition. Such findings may incorrectly imply causality when product switching was actually a consequence of existing illness.

12.8 Acute Versus Chronic Inference

Acute-exposure studies are useful for identifying immediate changes in heart rate, blood pressure, autonomic balance, or ventricular repolarization. However, they cannot establish whether repeated exposure causes chronic cardiac remodeling, cumulative vascular injury, heart failure, long-term arrhythmia, cardiovascular mortality, or persistent abnormalities after cessation. Conversely, cross-sectional studies of habitual users may identify differences between groups but cannot reliably establish temporal sequence or determine whether the exposure caused the abnormality. Longitudinal studies are therefore required to connect acute physiological responses with chronic structural and clinical outcomes.

12.9 Surrogate Outcomes

Most ECG investigations have relied on surrogate markers such as QTc, Tpeak–Tend, Tpeak–Tend/QT, Tpeak–Tend/QTc, and heart-rate variability. These measurements may provide useful mechanistic information and help generate hypotheses. Nevertheless, the causal pathway from a difference of a few milliseconds in a repolarization index to clinically significant atrial or ventricular arrhythmia has not been demonstrated in electronic-cigarette users. Statistical significance in a surrogate endpoint therefore should not be interpreted as direct evidence of increased sudden cardiac death or sustained arrhythmia.

12.10 Measurement Error

Potential measurement error represents another important limitation of the ECG literature. Manual caliper measurements may vary between observers, while identification of the end of the T wave can be difficult in the presence of low-amplitude, biphasic, or notched T waves. Studies have also differed in lead selection, reader blinding, and the number of cardiac cycles evaluated. Reliance on a single cycle and failure to average repeated beats may reduce reliability. In addition, the Bazett correction formula may overestimate QTc during tachycardia. Posture, respiratory pattern, recent physical activity, caffeine intake, and acute anxiety may also affect ECG and autonomic measurements if they are not standardized.

12.11 Multiple Statistical Comparisons

Small physiological studies often assess numerous ECG, heart-rate variability, hemodynamic, and subgroup variables simultaneously. When primary outcomes are not prespecified and corrections for multiple statistical testing are not applied, isolated significant findings may occur by chance. This concern is particularly relevant when only one or two indices differ while closely related measurements remain unchanged. Replication in adequately powered studies with prespecified hypotheses is necessary before such findings can be considered reliable.

12.12 Echocardiographic Limitations

The echocardiographic evidence has several additional weaknesses. The human literature is dominated by one principal acute study that used older electronic-cigarette technology and assessed myocardial function after a short exposure period. Right-heart assessment was limited, pulmonary artery pressure was not comprehensively evaluated, and no serial strain-based follow-up was performed. Exercise echocardiography was not used, no cohort

with established chronic pulmonary disease was included, and long-term clinical correlations were unavailable. These limitations prevent conclusions regarding chronic left ventricular dysfunction, right ventricular impairment, pulmonary hypertension, or structural cardiac remodeling.

12.13 Lack of Sex-Specific Analysis

Sex may influence autonomic regulation, baseline QT duration, repolarization characteristics, nicotine metabolism, and cardiovascular susceptibility. Some experimental studies have suggested that electronic-cigarette effects may differ between male and female participants. However, most studies have been too small to conduct reliable interaction analyses, and several included predominantly male participants. Consequently, possible sex-specific differences remain uncertain and require adequately powered investigation.

12.14 Underrepresentation of Older Adults

Most chronic pulmonary disorders become clinically important in middle-aged and older adults, yet electronic-cigarette physiological studies have predominantly enrolled younger participants. Older adults are more likely to have structural heart disease, coronary atherosclerosis, renal or hepatic impairment, autonomic dysfunction, and multiple prescribed medications. They may therefore respond differently to nicotine and aerosol exposure. The underrepresentation of this age group substantially limits the clinical applicability of current evidence.

12.15 Medication Confounding

Medication use may independently influence heart rate, cardiac conduction, ventricular repolarization, electrolyte concentrations, and myocardial function. Relevant agents include β_2-agonists, anticholinergic bronchodilators, theophylline, macrolides, fluoroquinolones, antifungal drugs, antidepressants, antipsychotics, antiarrhythmic medications, diuretics, and systemic corticosteroids. Some of these treatments can cause tachycardia, prolong QT, promote electrolyte disturbances, or modify autonomic tone. Medication timing, dosage, recent treatment changes, and electrolyte status were not consistently measured or adjusted for in the available studies.

12.16 Healthy-User and Volunteer Bias

Individuals who volunteer for acute laboratory studies may be healthier, younger, more adherent, and more willing to follow exposure restrictions than the broader population of electronic-cigarette users. Participants with advanced pulmonary disease, significant cardiovascular comorbidity, severe symptoms, or unstable clinical conditions may be excluded for safety reasons. As a result, experimental findings may underestimate physiological effects or adverse responses in patients with reduced cardiopulmonary reserve.

12.17 Absence of Clinically Important Endpoints

The available literature remains inadequate for determining whether electronic-cigarette use affects the incidence of atrial fibrillation, ventricular arrhythmia, heart-failure hospitalization, progression of pulmonary hypertension, right ventricular failure, cardiovascular mortality, or sudden cardiac death in adults with chronic pulmonary disease. Most studies were designed to measure short-term physiological changes rather than clinical outcomes. Until sufficiently powered longitudinal studies are conducted, the relationship between electronic-cigarette use and major cardiovascular events in this population will remain uncertain.

13. Interpretation of Relative Harm

Electronic cigarettes may expose users to fewer combustion-derived toxicants than combustible cigarettes [1-3]. Studies showing smaller acute changes after vaping or improved vascular function after switching should be interpreted as evidence of possible relative harm reduction under specific conditions [16,21,25,31].
Three distinctions are essential:

13.1 Less harmful is not harmless

A product can produce a smaller effect than combustible cigarettes while still causing measurable autonomic or vascular changes.

13.2 Switching is not dual use

Potential benefit depends largely on complete replacement of combustible cigarettes. Continuing to smoke even a smaller number of cigarettes may preserve considerable risk.

13.3 Switching is not abstinence

The appropriate reference group differs according to the clinical question. For a current smoker unable to stop, complete switching may reduce some exposures. For a non-smoker or former smoker, initiating electronic-cigarette use introduces avoidable exposure.

The 2026 COPD cohort illustrates this distinction: switching was associated with fewer major cardiovascular and cerebrovascular events than continued smoking, but complete cessation produced broader benefits and electronic-cigarette switching did not provide equivalent respiratory improvement [44].

14. Clinical Implications

14.1 Routine Screening

There is no current basis for routine ECG or echocardiographic screening solely because an adult with chronic pulmonary disease uses electronic cigarettes. Decisions regarding cardiac testing should instead reflect the patient's symptoms, physical examination findings, severity of the underlying pulmonary disorder, presence of hypoxemia, medication exposure, cardiovascular history, and established diagnostic indications. This approach helps avoid unnecessary investigation while ensuring that clinically significant cardiac abnormalities are not overlooked.

14.2 Clinical History

A comprehensive clinical assessment should cover both combustible-cigarette and electronic-cigarette exposure. Clinicians should document current and previous cigarette use, cumulative exposure in pack-years, the date on which the patient switched products, and whether the individual is an exclusive electronic-cigarette user or a dual user. The history should also record the device type, nicotine concentration, nicotine-salt or free-base formulation, frequency of vaping, approximate daily liquid consumption, flavouring substances, and any cannabis or tetrahydrocannabinol exposure. The source of the products, recent changes in device or liquid, and any temporal relationship between vaping and symptoms such as palpitations, chest discomfort, dyspnea, dizziness, or exercise intolerance should also be discussed.

14.3 Indications for a Resting ECG

A standard 12-lead ECG is reasonable when a patient presents with palpitations, syncope or presyncope, unexplained persistent tachycardia, chest discomfort, or newly developed exercise intolerance. ECG assessment should also be considered in the presence of a suspected electrolyte abnormality, significant hypoxemia, pulmonary hypertension, known structural heart disease, use of QT-prolonging medications, or frequent administration of sympathomimetic bronchodilators. In these settings, the ECG may help identify conduction abnormalities, repolarization changes, ischemic patterns, atrial arrhythmias, ventricular ectopy, or evidence of right-heart strain.

14.4 ECG Interpretation

An abnormal corrected QT interval or prolonged Tpeak–Tend interval should not automatically be blamed on electronic-cigarette exposure. The clinician should evaluate serum potassium, magnesium, and calcium concentrations and analuse all prescribed, over-the-counter, and non-prescribed medications. Renal and hepatic function should be assessed because impaired drug clearance may increase the risk of QT prolongation. Other considerations include acute myocardial ischemia, heart rate at the time of recording, the formula used for QT correction, the possibility of congenital long-QT syndrome, and comparison with previous ECGs. Where tachycardia is present, reliance on the Bazett formula alone may overestimate QTc prolongation, and an alternative correction such as the Fridericia formula may provide additional information.

14.5 Ambulatory Rhythm Monitoring

Ambulatory ECG monitoring may be useful when palpitations are intermittent, symptoms are not captured on a resting ECG, syncope remains unexplained, clinically significant ectopy is suspected, or paroxysmal atrial fibrillation is considered possible. Monitoring may also be useful when symptoms appear consistently after vaping or following

bronchodilator administration. The monitoring method should be selected according to symptom frequency. A 24- to 48-hour Holter monitor may be suitable for daily symptoms, whereas extended patch monitoring or event recording may be more appropriate when symptoms occur less frequently.

14.6 Indications for Echocardiography

Echocardiography may be appropriate when dyspnea appears disproportionate to the degree of lung-function impairment or when the patient develops unexplained exercise limitation, peripheral edema, elevated jugular venous pressure, or clinical suspicion of pulmonary hypertension. Additional indications include syncope, abnormal cardiac biomarkers, newly detected ECG abnormalities, signs of heart failure, unexplained worsening hypoxemia, or suspected right or left ventricular dysfunction. Echocardiography can help distinguish cardiac causes of breathlessness from progression of the underlying pulmonary disorder.

14.7 Recommended Echocardiographic Components

In adults with chronic pulmonary disease, a clinically informative echocardiographic examination should include a comprehensive evaluation of both the left and right sides of the heart. Left-heart assessment should include left ventricular dimensions and volumes, ejection fraction, regional wall motion, mitral inflow velocities, tissue-Doppler e' velocities, the E/e' ratio, left atrial volume, and global longitudinal strain when clinically appropriate. These measurements can help identify systolic dysfunction, diastolic impairment, elevated filling pressures, ischemic abnormalities, and subclinical myocardial dysfunction.

Right-heart assessment should include right ventricular basal and mid-cavity dimensions, right atrial area or volume, tricuspid annular plane systolic excursion, tissue-Doppler S' velocity, fractional area change, and right ventricular free-wall strain when technically feasible. Tricuspid regurgitation velocity, estimated right atrial pressure, estimated pulmonary artery systolic pressure, and inferior vena cava size and respiratory variation should also be assessed. These parameters are particularly important in chronic pulmonary disease because pulmonary hypertension and right ventricular dysfunction may contribute substantially to exercise intolerance, peripheral edema, hypoxemia, and adverse clinical outcomes [14,15].

14.8 Attribution of Abnormalities

Dyspnea, tachycardia, ECG changes, or right-heart abnormalities in a person who uses electronic cigarettes should not be blamed on vaping without a careful evaluation of other potential causes. Clinicians should consider progression of the underlying pulmonary disease, an acute respiratory exacerbation, pulmonary embolism, infection, anemia, left ventricular disease, sleep-disordered breathing, medication effects, physical deconditioning, and continuing combustible-cigarette smoking. Several of these conditions may coexist, and a temporal association with vaping does not by itself establish causality.

14.9 Cessation Counselling

Patients should be advised that complete cessation of combustible cigarettes remains the primary clinical objective. Dual use of combustible and electronic cigarettes should not be viewed as an adequate substitute for cessation because continued cigarette smoking may preserve a substantial proportion of tobacco-related risk. Patients should also be informed that electronic cigarettes are not physiologically neutral and that their long-term cardiac safety in individuals with chronic pulmonary disease has not been established.

Patients who have completely switched from combustible cigarettes to electronic cigarettes should be supported in eventually discontinuing electronic-cigarette use when feasible. Cessation counselling should be individualized and should include evidence-based behavioural support, assessment of nicotine dependence, and appropriate pharmacological cessation options according to the patient's respiratory condition, cardiovascular risk, previous quit attempts, treatment preferences, and potential contraindications.

15. Proposed Research Framework

15.1 Study Populations

Future studies should recruit separate and adequately powered cohorts representing the major chronic pulmonary disorders in which electronic-cigarette exposure may have clinically important cardiovascular consequences. These

populations should include patients with COPD stratified according to the severity of airflow limitation, severe asthma, bronchiectasis, interstitial lung disease, chronic hypoxemic respiratory failure, pulmonary hypertension, and COPD–sleep apnea overlap syndrome. Recruiting these groups separately would allow researchers to identify disease-specific differences in autonomic, electrocardiographic, myocardial, and pulmonary vascular responses rather than treating chronic pulmonary disease as a single homogeneous condition.

15.2 Comparator Groups

At minimum, future studies need clearly defined comparator groups consisting of never-smokers who have never used electronic cigarettes, former smokers who currently use no nicotine-containing product, exclusive electronic-cigarette users, exclusive combustible-cigarette smokers, dual users of electronic and combustible cigarettes, and individuals who have completely switched from combustible cigarettes to electronic cigarettes. These groups are necessary to distinguish the effects of current vaping from those of previous smoking, continuing combustible-cigarette exposure, dual use, and complete nicotine abstinence.

15.3 Exposure Verification

Electronic-cigarette exposure should be characterized using both self-reported information and objective biomarkers. Researchers should document the specific device, nicotine concentration, nicotine formulation, estimated liquid consumption, frequency and duration of use, and puff topography. Objective exposure assessment should include plasma nicotine, serum or salivary cotinine, exhaled carbon monoxide, and tobacco-specific biomarkers when feasible. Repeated exposure assessments should be carried out during follow-up because electronic-cigarette products and patterns of use may change substantially over time.

15.4 ECG Protocol

A standardized ECG protocol would improve comparability across studies. Participants should undergo a defined period of pre-exposure abstinence, with caffeine consumption and medication timing controlled as far as clinically appropriate. ECG recordings should be obtained after a standardized supine resting period, with simultaneous documentation of respiratory rate. A digital 12-lead ECG should be used, and interpretation should preferably be undertaken by a blinded central laboratory. Measurements should include the uncorrected QT interval as well as QTc calculated using both Bazett and Fridericia formulas. The methodology used to measure the Tpeak–Tend interval should be prespecified, measurements should be averaged across repeated cardiac cycles, and primary ECG outcomes should be defined before data analysis.

15.5 Ambulatory Monitoring

Longer-term studies should include ambulatory rhythm monitoring because a brief resting ECG may fail to detect intermittent arrhythmias. Depending on the study population and research objective, monitoring may include 24- to 48-hour Holter recording, 7- to 14-day patch monitoring, symptom-triggered event recording, or data obtained from implanted cardiac devices when clinically available. Relevant outcomes should include premature atrial complex burden, premature ventricular complex burden, non-sustained ventricular tachycardia, supraventricular tachycardia, atrial fibrillation, overall heart-rate profile, and correlation between reported symptoms and documented rhythm abnormalities.

15.6 Echocardiographic Protocol

Future echocardiographic studies need comprehensive and reproducible imaging protocols. These should include standard chamber quantification, left ventricular global longitudinal strain, right ventricular free-wall strain, and three-dimensional right ventricular volume assessment where technically feasible. Pulmonary pressure estimation, left ventricular diastolic assessment, and detailed evaluation of right ventricular systolic function should be included. Interobserver and intraobserver reproducibility should be reported, echocardiographic analysis should be carried out by blinded readers or a central laboratory, and serial imaging should be carried out to determine whether abnormalities are transient, persistent, progressive, or reversible after cessation of electronic-cigarette use.

15.7 Pulmonary Measurements

Cardiac findings should be interpreted together with objective measurements of pulmonary disease severity. These assessments should include spirometry, lung volumes, diffusing capacity, resting and exertional oxygen saturation,

and arterial blood gas analysis when clinically indicated. Functional assessment should include the six-minute walk test and, where feasible, cardiopulmonary exercise testing. Investigators should also document exacerbation frequency, validated respiratory symptom scores, and the use and timing of respiratory medications. Combining these measures would help clarify whether ECG or echocardiographic abnormalities are associated with electronic-cigarette exposure, underlying pulmonary impairment, hypoxemia, medication effects, or interactions among these factors.

15.8 Acute-Exposure Design

Randomized crossover studies could help separating the effects of nicotine from those of the electronic-cigarette aerosol. Such studies could compare nicotine-containing electronic-cigarette aerosol, nicotine-free aerosol, nicotine administered without aerosol exposure, combustible-cigarette smoking, sham exposure, and a no-exposure control condition. Whenever possible, nicotine delivery should be matched between exposure conditions or objectively measured and statistically accounted for. Adequate washout periods should also be incorporated to minimize carryover effects.

15.9 Longitudinal Design

Prospective multicentre cohorts should track participants for several years to evaluate persistent and clinically meaningful outcomes. Relevant endpoints should include changes in ECG repolarization indices, incident atrial and ventricular arrhythmias, changes in right and left ventricular structure and function, development or progression of pulmonary hypertension, heart-failure hospitalization, respiratory exacerbations, major adverse cardiovascular events, cardiovascular mortality, and all-cause mortality. Repeated assessment of both exposure status and cardiopulmonary outcomes would be essential because participants may switch between exclusive vaping, dual use, combustible smoking, and complete abstinence during follow-up.

15.10 Sample-Size Considerations

Studies should be adequately powered according to a prespecified primary outcome. Small physiological studies may be sufficient to detect immediate changes in heart rate, blood pressure, or selected ECG intervals, but they are not suitable for evaluating infrequent outcomes such as sustained arrhythmia, heart-failure hospitalization, pulmonary-hypertension progression, cardiovascular death, or all-cause mortality. Sample-size calculations should account for anticipated effect size, outcome variability, participant attrition, subgroup analyses, and repeated measurements.

15.11 Confounder Control

Future studies also need to account carefully for factors that may independently influence cardiac and pulmonary outcomes. Important covariates include age, sex, socioeconomic status, cumulative smoking exposure, current smoking intensity, second-hand smoke exposure, cannabis use, alcohol consumption, caffeine intake, physical activity, body mass index, blood pressure, diabetes, lipid status, renal function, pre-existing cardiovascular disease, pulmonary disease severity, oxygen therapy, sleep apnea, medication use, and electrolyte status. Accounting for these variables is essential to reduce residual confounding and to avoid incorrectly attributing abnormalities to electronic-cigarette exposure.

16. Suggested Minimum Dataset for Future Studies

Table 2. Minimum dataset for cardiopulmonary electronic-cigarette research

Domain	Recommended variables
Participant characteristics	Age, sex, ethnicity, body mass index, socioeconomic status
Pulmonary diagnosis	Confirmed diagnosis, disease duration, severity, exacerbation history
Smoking history	Current status, pack-years, quit date, cigarettes per day
Electronic-cigarette exposure	Device, power, nicotine concentration, formulation, flavour, frequency, duration, liquid consumption

Exposure biomarkers	Nicotine, cotinine, exhaled carbon monoxide, tobacco-specific biomarkers
Medications	Bronchodilators, corticosteroids, macrolides, fluoroquinolones, QT-prolonging drugs, cardiovascular medication
Pulmonary physiology	Spirometry, lung volumes, diffusing capacity, resting and exertional oxygen saturation
Resting ECG	Heart rate, PR, QRS, QT, QTc, Tp-e, Tp-e/QT, Tp-e/QTc
Ambulatory ECG	Ectopy, atrial fibrillation, supraventricular tachycardia, ventricular tachycardia
Autonomic measures	Time-domain and frequency-domain HRV using standardized methodology
Left-heart echocardiography	Ejection fraction, volumes, diastolic indices, left atrial volume, longitudinal strain
Right-heart echocardiography	RV dimensions, TAPSE, S', fractional area change, RV free-wall strain
Pulmonary circulation	TR velocity, estimated pulmonary pressure, pulmonary acceleration time
Clinical outcomes	Exacerbations, hospitalization, heart failure, MACCE, mortality
Timing	Baseline, acute post-exposure, intermediate follow-up and longitudinal follow-up

17. Overall Certainty of Evidence

Table 3. Qualitative certainty of evidence

Outcome	Evidence quantity	Consistency	Directness to chronic pulmonary disease	Overall certainty
Acute heart-rate increase	Moderate	Generally consistent	Very indirect	Low to moderate
Acute blood-pressure increase	Moderate	Generally consistent	Very indirect	Low to moderate
Sympathetic predominance/HRV alteration	Limited to moderate	Fairly consistent for nicotine-containing exposure	Very indirect	Low
QT/QTc alteration	Limited	Inconsistent	Very indirect	Very low
Tp-e and related ratios	Limited	Direction generally suggests prolongation, but heterogeneous	Very indirect	Very low
Clinically documented arrhythmia	Minimal	Not established	Absent	Very low
Acute LV systolic dysfunction	Minimal	No significant effect in one principal study	Very indirect	Very low
Acute LV diastolic dysfunction	Minimal	Less effect than combustible smoking in one study	Very indirect	Very low
Chronic LV remodeling	Absent	Not assessable	Absent	Very low
RV dysfunction	Absent	Not assessable	Absent	Very low
Pulmonary artery pressure	Absent	Not assessable	Absent	Very low
Cardiovascular events in COPD	One large observational cohort plus indirect evidence	Suggests lower risk than continued smoking but greater uncertainty than abstinence	Direct population, indirect cardiac testing	Low

18. Limitations of This Analysis

This analysis has several limitations.

First, it was a focused analysis rather than a prospectively registered systematic review. Formal duplicate screening, duplicate extraction and standardized risk-of-bias scoring were not undertaken.

Second, PubMed/MEDLINE was the principal database. Relevant reports indexed exclusively in other databases may have been missed.

Third, electronic-cigarette terminology and technology evolve rapidly. Studies may have been indexed under product-specific terms not captured by the search.

Fourth, a large proportion of the available evidence involved acute laboratory exposure rather than habitual long-term use.

Fifth, direct evidence from adults with chronic pulmonary disease was extremely limited. Much of the interpretation necessarily relied on indirect evidence from healthier populations.

Sixth, the absence of an identified study should be understood as an evidence gap rather than absolute proof that no relevant report exists in any database or language.

Seventh, no quantitative synthesis was performed because of marked heterogeneity in devices, exposure protocols, comparator groups and outcome measurements.

Finally, evidence published after January, 2026, was outside the search period.

19. CONCLUSIONS

Overall, human studies suggest that nicotine-containing electronic-cigarette use can acutely increase heart rate and blood pressure and shift cardiac autonomic balance toward sympathetic predominance. Several small studies have also reported changes in ventricular repolarization indices, including QTc, Tpeak–Tend, Tpeak–Tend/QT and Tpeak–Tend/QTc.

These findings demonstrate physiological activity but do not establish clinically significant arrhythmia. The available studies were small, heterogeneous and predominantly conducted in healthy adults or combustible-cigarette smokers. Clinically important rhythm outcomes, including atrial fibrillation, ventricular tachycardia, syncope and sudden cardiac death, remain inadequately evaluated.

Human echocardiographic evidence is exceptionally limited. One acute study did not demonstrate significant immediate deterioration in measured left ventricular function after electronic-cigarette use, whereas conventional cigarette smoking adversely affected several myocardial performance and diastolic indices. That study cannot exclude chronic myocardial effects, effects of modern devices or greater susceptibility among patients with chronic pulmonary disease.

The largest evidence gap concerns the right ventricle and pulmonary circulation. No adequate human study has determined whether electronic-cigarette use influences right ventricular function, pulmonary artery pressure, ventricular strain or pulmonary-hypertension progression in COPD, asthma, bronchiectasis or interstitial lung disease.

Routine ECG or echocardiographic screening based solely on electronic-cigarette exposure is not currently evidence-based. Cardiac evaluation should be guided by symptoms, clinical examination, pulmonary disease severity, oxygenation, medication exposure and established cardiovascular indications.

Clinicians should avoid two unsupported extremes: electronic cigarettes should not be described as harmless, but abnormal cardiac findings should not automatically be blamed on vaping. Complete cessation of combustible tobacco remains the most important intervention. When electronic cigarettes are used as a substitute, complete switching is preferable to dual use, and eventual cessation of all inhaled nicotine products should remain a clinical objective where feasible.

Prospective, adequately powered and disease-specific studies integrating electronic-cigarette exposure assessment, pulmonary physiology, ambulatory ECG and comprehensive right- and left-heart echocardiography are urgently required.

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