From Vaping to Ventilation: Perioperative Challenges for Anaesthetists

Abstract:

The rapid and persistent rise of electronic cigarette (e-cigarette, EC) use introduces new challenges in perioperative medicine. While originally marketed as safer alternatives to tobacco smoking, vaping exposes users to nicotine, volatile organic compounds, aldehydes, and heavy metals that produce significant systemic effects[1][2]. This narrative review synthesises emerging evidence on the pathophysiological effects of vaping across pulmonary, cardiovascular, neurological, immune, and pharmacological domains, with a focus on perioperative risk. The implications for anaesthetic practice, including altered airway reactivity, haemodynamic instability, immune dysfunction, impaired wound healing, and modified drug metabolism is discussed.

Anaesthetists should approach chronic e-cigarette users with tailored preoperative assessment, vigilant intraoperative strategies, and structured postoperative monitoring. Evidence gaps are still, needing further research to establish evidence-based guidelines for perioperative care in this growing patient cohort.

Keywords:

Electronic cigarettes; Vaping; Anaesthesia; Perioperative management; Airway hyperreactivity; Cardiovascular risk; Anaesthetic pharmacology; Wound healing; Drug metabolism; Perioperative complications

Evidence gaps and research priorities:

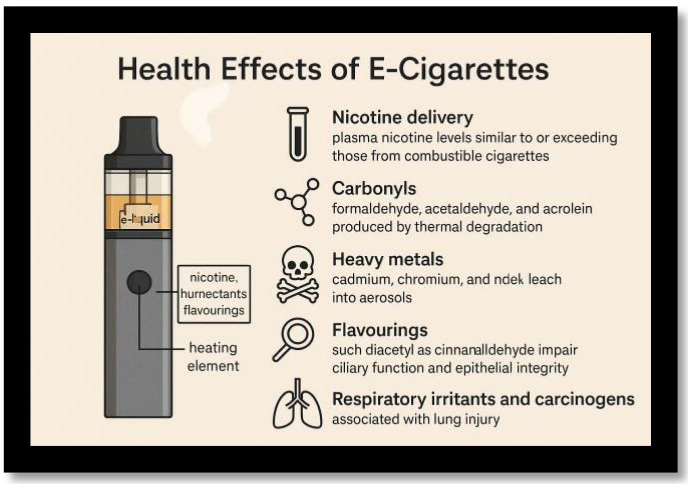
High-quality perioperative outcome data specifically in exclusive e-cigarette users remains sparse. Most recommendations extrapolate from mechanistic e-cigarette toxicology studies, observational cohorts, and the smoking literature. Priority research includes prospective cohorts of exclusive vapers, trials of peri-operative bronchodilator/lidocaine strategies in vapers, and studies defining the optimal pre-operative abstinence interval for e-cigarettes.

[1] Goniewicz et al., 2014; Williams et al., 2017, Jensen et al., Allen et al., 2016
[2] Warner et al., 2019; Aldwinckle & Carr, 2021

Introduction:

Over the past two decades, electronic cigarettes have emerged as one of the fastest growing nicotine delivery systems, with global use exceeding 80 million by 2023[3]. In Australia, prevalence has increased by more than 500% since 2018[4], with projections suggesting over 1.7 million users by 2024[5]. While initially promoted as harm-reduction tools for smokers, accumulating evidence shows that vaping is associated with unique toxicological exposures, including nicotine, propylene glycol, vegetable glycerine, volatile carbonyls, and heavy metals[6]. These compounds provoke inflammatory, oxidative, and immunomodulatory responses with clinical consequences for surgical patients[7]. Anaesthesia providers face increasing numbers of patients who vape, yet the perioperative implications stay under-recognised[8]. This paper reviews the composition of vaping products and liquids, the pathophysiological impacts of vaping products to patients, the anaesthetic implications for these patients, impacts and considerations for Anaesthesia Care, and recommendations integrating recent data to support clinical decision-making.

Composition of Vaping Products and Liquids:



E-cigarettes function by heating a liquid (commonly termed "e-liquid" or "vape juice") containing nicotine, humectants, and flavouring agents. The heating element aerosolises this solution, creating an inhalable vapor[9].

Nicotine delivery is highly variable, with plasma nicotine levels similar to or exceeding those from combustible cigarettes depending on device power and puff topography[10].

Carbonyls such as formaldehyde, acetaldehyde, and acrolein are produced by thermal degradation of propylene glycol and vegetable glycerine[11].

Heavy metals including cadmium, chromium, and nickel leach from atomisers into aerosols[12].

Flavourings such as diacetyl and cinnamaldehyde impair ciliary function and epithelial integrity[13]. While many flavourings are recognised as safe for ingestion, safety following inhalation has not been proven. Many of the compounds in vapes and their contents are known

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3 WHO, 2021; Wipfli et al., 2024
4 Royal Australian College of General Practitioners, 2024; Roy Morgan 2024; Department of Health and Aged Care, 2024
5 Ibid see 4
6 CDC, 2020; National Academies, 2018; Goniewicz et al., 2014; Williams et al., 2017
7 Sundar et al., 2016; Madison et al., 2019; Reidel et al., 2018; Corriden et al., 2020
8 Warner et al., 2019; Aldwinckle & Carr, 2021; Joseph et al., 2024
9 National Academies of Sciences, Engineering, and Medicine, 2018; CDC, 2020
10 Goniewicz et al., 2015; St Helen et al., 2016; Farsalinos and Polosa, 2014
11 Jensen et al., 2015; Sleiman et al., 2016
12 Williams et al., 2013; Olmedo et al., 2018
13 Allen et al., 2016; Clapp et al., 2017

respiratory irritants and carcinogens, with documented associations to lung injury and cardiovascular disease[14].

These exposures translate to multi-systemic impacts to e-cigarette users, relevant in perioperative pathophysiology.

Pathophysiological Impacts:

The increasing prevalence of vaping has prompted growing concern over its systemic pathophysiological consequences, particularly within pulmonary, cardiovascular, central nervous, immune, musculoskeletal, and pharmacological domains.

Pulmonary Implications:

Vaping has been shown to increase airway resistance and bronchial hyperreactivity, even after short-term exposure[15]. The inhalation of heated aerosolised substances provokes inflammatory responses in the airway epithelium, impairing mucociliary clearance and disrupting alveolar macrophage function. These alterations contribute to increased susceptibility to infection and diminished pulmonary defence mechanisms. Clinically significant pulmonary diseases such as bronchiolitis obliterans, exacerbations of chronic obstructive pulmonary disease (COPD), and e-cigarette or vaping product use-associated lung injury (EVALI) have been documented in association with chronic use[16]. Alveolar surfactant depletion has also been suggested, potentially leading to alveolar collapse and hypoxaemia. Furthermore, the inhalation of vaping aerosols has been shown to induce oxidative stress and increase the release of pro-inflammatory cytokines (e.g., IL-6, IL-8, CCL2, CXCL10)[17], further worsening lung tissue damage and inflammation[18].

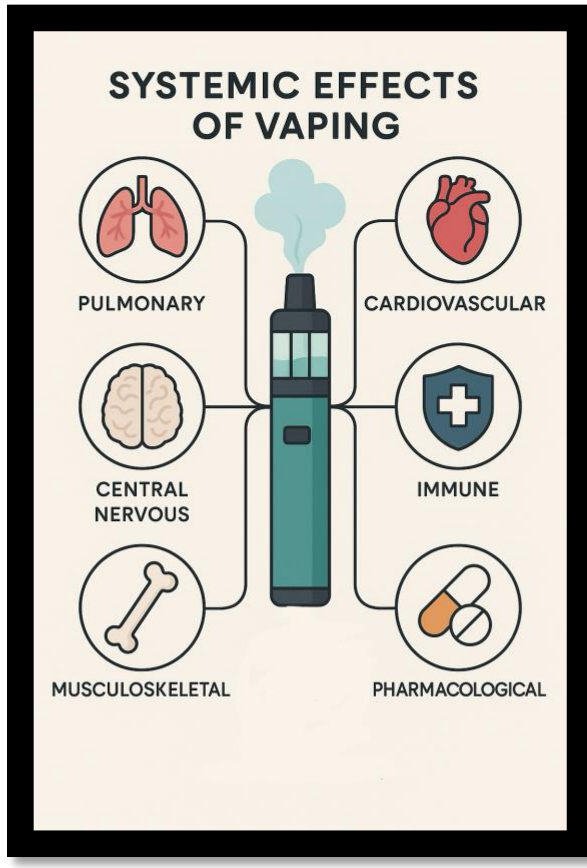
Cardiovascular Implications:

The cardiovascular consequences of vaping are increasingly well-documented. Acute use is associated with endothelial dysfunction, arterial stiffness, and a transient rise in blood pressure and heart rate, thereby increasing myocardial oxygen demand. These haemodynamic changes are accompanied by impaired coronary perfusion, increased risk of arrhythmias, and atherogenic processes mediated through inflammation and oxidative stress. Observational and interventional studies have reported elevated arterial stiffness markers, such as pulse wave velocity and augmentation index, following vaping, even in otherwise healthy individuals[19]. Additionally, the exposure to nicotine and other aerosol constituents may contribute to insulin resistance and dyslipidaemia, compounding cardiovascular risk profiles[20].

[14] Gotts et al., 2019; Glantz and Bareham, 2018; National Academies of Sciences, Engineering, and Medicine, 2018
[15] Chatterjee et al., 2020; Reidel et al., 2018
[16] Madison et al., 2019; El-Boghdadly et al., 2024
[17] Corroded et al., 2020; Sundar et al., 2016
[18] Gotts, J.E., Jord, S-E., McConnell, R. & Tarran, R., 2019, *BMJ*, 366, l5275, Madison, M.C., Landers, C.T., Gu, B-H., Chang, C-Y., Tung, H-Y., You, R. et al., 2019, *Journal of Clinical Investigation*, 129(10), 4290–4304
[19] Carnevale et al., 2016; Benowitz & Burbank, 2016; Qasim et al., 2017
[20] Middlekauff, H.R., 2020, *Trends in Cardiovascular Medicine*, 30(3), 133–140, Polosa et al., 2020; Qasim, H., Karim, Z.A., Rivera, J.O., Khasawneh, F.T. & Alshbool, F.Z., 2017, *Journal of the American Heart Association*, 6(9), e006353

Central Nervous System Implications:

Though less extensively studied, the central nervous system (CNS) may also be affected by compounds present in vaping aerosols. Volatile organic compounds (VOCs) have the potential to exert CNS depressant effects. Of particular concern is the use of vaping products among adolescents, a population in which neurodevelopmental processes remain ongoing[21]. Nicotine exposure during this critical developmental window has been implicated in long-term cognitive and behavioural impairments. Moreover, chronic exposure to CNS-active substances may alter responses to anaesthetic agents, although this relationship remains incompletely understood[22].



Immune and Musculoskeletal Implications:

Vaping-related immunomodulation may impair wound healing and musculoskeletal integrity. In vitro studies suggest that exposure to vaping aerosols reduces fibroblast activity and disrupts collagen deposition[23]—two processes critical to effective tissue repair. Although clinical data is limited, the observed dysregulation of immune responses and chronic low-grade inflammation may contribute to a heightened incidence of postoperative wound infections and delayed healing. Additionally, altered osteoblast function has been postulated, with potential implications for bone remodelling and skeletal strength[24].

Pharmacological Implications:

Vaping may influence the metabolism and efficacy of various pharmacologic agents through the induction of hepatic cytochrome P450 enzymes, particularly CYP1A2[25]. This has implications for perioperative care, including increased dosage requirements for neuromuscular blocking agents such as rocuronium and vecuronium[26]. Furthermore, the CNS depressant effects of certain vaping constituents may reduce the minimum alveolar concentration (MAC) needed for volatile anaesthetics, though empirical data is currently limited. Chronic nicotine exposure may also elevate perioperative opioid requirements due to altered nociceptive processing[27].

[21] Glantz & Bareham, 2018
[22] Polosa, R., O'Leary, R., Caponnetto, P., Bustamante, G., Perricone, C. & Puglisi, C., 2020, *BMC Medicine*, 18, 79
[23] Moller et al., 2002; Reidel et al., 2018
[24] Clapp, P.W., Pawlak, E.A., Lackey, J.T., Keating, J.E., Reeber, S.L., Glish, G.L. & Jaspers, I., 2017, *American Journal of Physiology – Lung Cellular and Molecular Physiology*, 313(2), L278–L292
[25] Hukkaen et al., 2005
[26] Naguib et al.,2018
[27] Hukkanen, J., Jacob, P. & Benowitz, N.L., 2005, *Pharmacological Reviews*, 57(1), 79–115

Pathophysiological effects of vaping extends beyond the pulmonary system, encompassing a broad array of cardiovascular, neurological, immunological, and pharmacological domains. While some effects are well-substantiated by emerging clinical and preclinical data, others remain theoretical for lack of substantive data and limited research. Continued investigation is warranted to find the full spectrum of health implications associated with vaping, particularly in the perioperative and long-term disease contexts[28].

Implications and Considerations for Anaesthesia Care:

Use of electronic cigarettes is associated with acute sympathetic activation; airway inflammation and hyperreactivity; and patterns of nicotine exposure that can influence analgesic needs. Direct peri-anaesthetic evidence in exclusive e-cigarette users is limited; however, convergent data from human exposure studies, physiologic investigations, and perioperative literature support several practical implications for anaesthetic management. Where only cigarette or combination data exists, it is noted below[29].

Pharmacological Responses:

Neuromuscular Blockers:

Although e-cigarette-specific data are lacking, cigarette smokers often need higher rocuronium doses[30] and show altered responses to steroidal neuromuscular blockers, likely due to nicotinic receptor upregulation rather than enzyme induction[31]. Heavy or dual e-cigarette users can achieve nicotine levels equal to or greater than smokers[32], making altered blockade responses biologically plausible[33].

Cytochrome Induction:

Cigarette smoke induces CYP1A2 via PAHs, accelerating metabolism of many drugs. e-cigarette aerosols hold far fewer PAHs and do not consistently induce CYP1A2[34], meaning the classic “smoker’s” interactions are unlikely in exclusive e-cigarette users. Rocuronium/vecuronium are not CYP1A2-cleared, though nicotine may influence CYP2E1 activity and hepatic blood flow, with possible but unproven effects on anaesthetic metabolism[35].

Volatile Anaesthetic Requirement (MAC):

Data on e-cigarette and MAC are limited. While some e-cigarette constituents may have CNS depressant effects, nicotine’s sympathomimetic activity could counteract this[36]. Current evidence

28 Benowitz, N.L. & Burbank, A.D., 2016, *Trends in Cardiovascular Medicine*, 26(6), 515–523
29 Benowitz, N.L. & Burbank, A.D., 2016, *Trends in Cardiovascular Medicine*, 26(6), 515–523
30 Brull & Kopman, 2017
31 Feldman et al., 1993; Kudoh et al., 1994
32 Goniewicz et al., 2019
33 Benowitz, N.L. & Burbank, A.D., 2016, *Trends in Cardiovascular Medicine*, 26(6), 515–523
34 Hukkanen et al., 2005
35 Benowitz & Burbank, 2016, Benowitz, N.L. & Burbank, A.D., 2016, *Trends in Cardiovascular Medicine*, 26(6), 515–523
36 Warner et al., 2019; Alderwinckle & Carr, 2021

does not support assuming reduced MAC in e-cigarette users; dosing should be titrated to effect[37].

Respiratory Considerations:

Airway reactivity: e-cigarette exposure acutely increases airway resistance and provokes cough/wheeze; observational and human-exposure studies show airway inflammation (elevated nitric oxide, cytokines) and hyper-responsiveness[38]. This translates peri-operatively to a higher risk of bronchospasm and laryngospasm at induction and emergence[39].

Choice of volatile agent: Desflurane is a known airway irritant with higher rates of cough/laryngospasm than sevoflurane; thus, sevoflurane is generally preferred in patients with reactive airways[40].

Mitigating hyper-reactivity: Pre-induction inhaled bronchodilators in patients with reactive airways reduce peri-operative bronchospasm; intravenous lidocaine can attenuate cough and airway reflexes around extubation, though evidence for preventing intra-induction bronchospasm is mixed[41].

Cardiovascular Considerations:

Sympathetic activation and haemodynamic lability. Acute e-cigarette use increases heart rate and blood pressure, and impairs endothelial function. Systematic reviews and controlled exposure studies document heightened sympathetic tone and vascular dysfunction shortly after vaping sessions. Conservative vasoactive titration and close e-cigarette monitoring are recommended, particularly in patients with underlying cardiovascular disease[42].

Current Preoperative Assessment:

The preoperative assessment of patients who vape is constrained by significant limitations. Unlike tobacco smoking, there are no validated or standardised tools to quantify vaping exposure in terms of frequency, duration, device type, or chemical constituents inhaled[43]. Patients may underreport or mischaracterise their use due to a lack of awareness on the clinical relevance of vaping, further reducing the reliability of history-taking[44]. Biomarkers such as cotinine and nicotine capture exposure but do not reflect the broader toxicological burden unique to vaping aerosols[45]. The rapid evolution of devices and liquid formulations creates wide variability in physiological effects, limiting the ability to generalise perioperative risk[46]. Current guidelines

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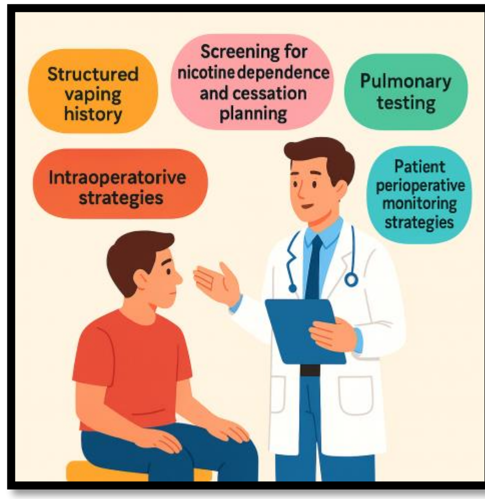
[37] Caruso et al., 2019, Benowitz, N.L. & Burbank, A.D., 2016, *Trends in Cardiovascular Medicine*, 26(6), 515–523
[38] Reidel et al., 2018; Chatterjee et al., 2020
[39] Benowitz, N.L. & Burbank, A.D., 2016, *Trends in Cardiovascular Medicine*, 26(6), 515–523
[40] Ibid see 39
[41] Ibid see 39
[42] Ibid see 39
[43] Yingst et al., 2019
[44] Polosa et al., 2020
[45] Williams et al., 2017; Hukkanen et al., 2005
[46] Chatterjee et al., 2020

extrapolate from cigarette smoking data, leaving knowledge gaps in accurately predicting pulmonary, cardiovascular, and immunological complications specific to vaping[47][48].

Recommendations for Anaesthesia Care:

Use of electronic cigarettes produces variable toxicant exposures and accumulative physiological effects that are relevant to perioperative risk and anaesthesia management[49]. A structured perioperative assessment that includes vaping-specific variables allows more exact risk stratification and tailored optimisation[50]. Recommendations are broken down into the following categories:

- Structured vaping history
- Screening for nicotine dependence and cessation planning
- Pulmonary testing
- Patient perioperative monitoring strategies
- Intra-operative strategies
- Pharmacotherapy



Structured vaping history:

A focused, structured e-cigarette history should be obtained for all patients who report current or recent vaping[51]. Key items and why they should be addressed in assessment:

Device type and settings (pod, mod, “heat-not-burn”, coil resistance, wattage/temperature): higher power/temperature increases formation of reactive carbonyls (formaldehyde, acrolein) and particulates that irritate airways and promote inflammation[52].

E-liquid composition: nicotine concentration (mg/mL), PG:VG ratio and identified solvents — these determine nicotine delivery kinetics and aerosol physicochemistry[53].

Flavourings: several flavouring chemicals (eg, cinnamaldehyde, diacetyl) are linked to cytotoxicity, impaired mucociliary function and airway inflammation[54].

Use pattern/puff topography: frequency, typical puff duration/volume and time-to-first-vape on waking (analogous to cigarette “time to first cigarette”) predict dependence and recent nicotine exposure[55].

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[47] Polosa, R., O’Leary, R., Caponnetto, P., Bustamante, G., Perricone, C. & Puglisi, C., 2020, *BMC Medicine*, 18, 79
[48] Chatterjee, S., Tao, J.Q., Johncola, A., Guo, W., Caporale, A., Langham, M.C. & Wehrli, F.W., 2020, *American Journal of Physiology - Lung Cellular and Molecular Physiology*, 319(1), L129–L138
[49] Tomaszek, L., et al., 2021, *BJA Education*, 21(10), 348–354
[50] ANZCA, 2025, *Australian and New Zealand College of Anaesthetists*, Accessed 2025
[51] Royal Australian College of General Practitioners (RACGP), 2024, *RACGP Guideline*, Version October 2024
[52] Møller, A.M., Pedersen, T., Villebro, N. & Munksgaard, A., 2002, *Journal of Bone & Joint Surgery (Br)*, 84-B(2) 178-181
[53] Ibid see 35, 36, 37
[54] Ibid see 35, 36, 37
[55] Ibid see 35, 36, 37

Dual use / other inhaled substances: concurrent cigarette or cannabis use markedly increases toxicant burden and perioperative risk and should be recorded[56].

Practical documentation can be brief (device, nicotine mg/mL, daily sessions/puffs, dual use, recent symptoms) but should be retrievable in the anaesthesia record because these items map to physiologic effects that change perioperative management[57].

Screening for nicotine dependence and cessation planning:

Tooling:

Use an e-cigarette-adapted, validated instrument such as the Penn State Electronic Cigarette Dependence Index (PSECDI) to quantify dependence and anticipate withdrawal or perioperative nicotine requirements. The PSECDI has shown psychometric validity and practical scoring bands for clinical use[58].

Cessation timing:

Although direct randomised evidence in exclusive vapers is limited, peri-operative smoking-cessation literature shows reduced postoperative complications when abstinence is achieved weeks before surgery; major agencies therefore continue to recommend a pre-operative cessation interval (commonly cited ≥3–8 weeks) when feasible[59]. Treat the perioperative period as a teachable moment for cessation and arrange support[60].

Pharmacotherapy:

When nicotine replacement is indicated to prevent withdrawal (and thereby reduce sympathetic surges and agitation), perioperative Nicotine Replacement Therapy (NRT) or other pharmacotherapy could be used in accordance with local guidelines. However, it is notable that available observational data and guideline analyses does not support routine withholding of NRT preoperatively when nicotine dependence necessitates treatment[61]. Clinicians should weigh each patients individual risks (including wound healing in some contexts) and follow institutional policies[62].

Pulmonary testing and investigations (selective, not routine):

Current perioperative guidance (and contemporary PFT practice reviews) recommends selective use of preoperative pulmonary function tests (PFTs) and arterial blood gases (ABGs) rather than routine ordering for all e-cigarette users. PFTs/ABGs should be reserved for patients whose

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56 Ibid see 35, 36, 37
57 Ibid see 35, 36, 37
58 Yingst, J.M., Hrabovsky, S., Nichols, T.T., Wilson, S.J., Blank, M.D., & Foulds, J., 2019, *Nicotine & Tobacco Research*, 21(12), 1690–1698, Royal Australian College of General Practitioners (RACGP), 2024, *RACGP Guideline*, Version October 2024
59 Royal Australian College of General Practitioners, 2024; Moller et al., 2002
60 Ibid see 59
61 Royal Autralian College of General Practitioners, 2024; Australian & New Zealand College of Anaesthetists 2025
62 Royal Australian College of General Practitioners (RACGP), 2024, *RACGP Guideline*, Version October 2024, Møller, A.M., Pedersen, T., Villebro, N. & Munksgaard, A., 2002, *Journal of Bone & Joint Surgery (Br)*, 84-B(2), 178–181

symptoms, medical history, or planned surgery (eg, major thoracic resections) would change management. Uncomplicated, asymptomatic vapers without known lung disease evidentially have a lower risk potential therefore baseline PFTs solely because of e-cigarette use is not recommended. Clinician individual assessment and guided decision making should be utilised when assessing necessity of testing[63].

Imaging / higher-acuity testing such as; Chest radiography, CT, or further testing, should be indicated if there is reasonable clinical suspicion of the patient having EVALI, chemical pneumonitis, unexplained dyspnoea, hypoxaemia, or when preoperative imaging would provide confirmation of perioperative risk to allow anaesthetic planning or impact surgical planning[64].

Patient Perioperative Monitoring Strategies:

Patients who report regular e-cigarette (e-cigarette) or vaping use should be considered at increased risk of perioperative complications due to documented effects on airway reactivity, cardiovascular lability, and altered drug metabolism[65]. Evidence shows that e-cigarette exposure is associated with increased airway hyper-responsiveness and impaired mucociliary clearance[66], endothelial dysfunction[67], and sympathetic activation from nicotine[68][69].

So, intraoperative monitoring should extend beyond standard ASA minimum requirements and include:

Enhanced respiratory monitoring: Continuous capnography, quantitative waveform analysis, and vigilant assessment for bronchospasm given the heightened risk of perioperative airway reactivity. Consideration of intraoperative spirometry or airway pressure monitoring is warranted in high-risk users[70][71][72][73].

Cardiovascular surveillance: Continuous electrocardiography with heightened vigilance for arrhythmias, beat-to-beat blood pressure monitoring (arterial line) in patients with chronic or heavy use due to nicotine-induced sympathetic effects and potential endothelial dysfunction[74].

Neuromuscular and anaesthetic depth monitoring: Processed EEG or BIS monitoring should be considered, as nicotine and other vaping constituents may alter anaesthetic drug requirements and MAC values, although current data remains inconclusive[75].

Postoperative monitoring: Extended pulse oximetry and capnography in the PACU, with consideration of overnight oximetry in heavy users, due to increased risk of hypoxia, atelectasis, and impaired mucociliary clearance[76].

[63] Royal Australian College of General Practitioners (RACGP), 2024, *RACGP Guideline*, Version October 2024
[64] Ibid.
[65] Joseph et al., 2024
[66] Gotts et al., 2019
[67] Carnevale et al., 2016
[68] Benowitz & Fraiman, 2017, Gotts, J.E., Jord, S-E., McConnell, R. & Tarran, R., 2019, BMJ, 366, I5275
[69] Middlekauff, H.R., 2020, Trends in Cardiovascular Medicine, 30(3), 133-140
[70] Benzowitz, N.L. & Burbank, A.D., 2016, Trends in Cardiovascular Medicines, 26(6), 515-523
[71] Carnevale, R., Sciarretta, S., violi, F., etal., 2016, CHEST, 150(3) 606-612
[72] El-Boghdadly, K., et al., 2024, Canadian Journal of Anesthesia, 71, 1-9
[73] Flood et al. 2020
[74] Ibid see 52,53,54
[75] Ibid

Intra-operative strategy:

The intra-operative plan should be organised in reflection with carried out risk identification for each e-cigarette patient, with thorough consideration to airway reactivity and cardiovascular effects[77].

Airway management:

In patients with recent vaping exposure and symptoms of airway excitability (e.g., cough, wheeze, sputum production), an intravenous induction strategy such as propofol or total intravenous anaesthesia (TIVA) should be strongly considered. Propofol is well documented to blunt airway reflexes[78] and reduce the incidence of laryngospasm and bronchospasm compared with volatile induction, an effect particularly valuable in patients with airway hyper-reactivity[79][80][81].

Vaping-related airway inflammation, impaired mucociliary clearance, and heightened cough reflex sensitivity have been proven in both clinical and experimental studies, suggesting a lowered threshold for bronchospasm during perioperative stimulation[82]. When possible, minimising repeated airway instrumentation (e.g., multiple laryngoscopy attempts, unnecessary suctioning) is recommended to reduce further mucosal irritation and airway compromise[83][84].

Deepening the plane of anaesthesia during critical periods of intubation and extubation significantly reduces the risk of laryngospasm, bronchospasm, and desaturation events[85]. Conversely, airway manipulations performed under light planes of anaesthesia have been shown to increase adverse airway responses, particularly in patients with baseline reactive airways such as vapers, who exhibit bronchial epithelial dysfunction and increased eosinophilic airway inflammation[86][87].

So, intravenous induction, maintenance of adequate anaesthetic depth, and cautious, well-planned airway manipulation represent best practice strategies to mitigate airway complications in vaping patients[88].

Volatile agent choice:

Desflurane is well documented to provoke upper airway irritation, coughing, breath-holding, and sympathetic activation, with significantly higher incidence rates compared to sevoflurane, particularly during induction and emergence[89]. In patients who vape, this risk is magnified due to

[76] Ibid
[77] Ibid
[78] Hirshman et al., 1999
[79] O'Donnell & Dolan, 2018; Hirshman et al., 1999[7]
[80] Hirshman, C.A., McCullough, J., et al., 1999, *Anesthesiology*, 91(3), 750–755
[81] O'Donnell, J. & Dolan, R., 2018, *Current Opinion in Anaesthesiology*, 31(1), 28–35
[82] Gotts et al., 2019; Madison et al., 2019
[83] Gotts, J.E., Jord, S-E., McConnell, R. & Tarran, R., 2019, *BMJ*, 366, l5275
[84] Madison, M.C., Landers, C.T., Gu, B-H., Chang, C-Y., Tung, H-Y., You, R. et al., 2019, *Journal of Clinical Investigation*, 129(10), 4290–4304
[85] Holley et al., 2019
[86] Muthumalage et al., 2019
[87] Ibid see 64, 65
[88] Ibid see 64, 65
[89] Eger et al., 1997; Ebert et al., 1998

airway epithelial inflammation, increased cough reflex sensitivity, and heightened bronchial reactivity associated with chronic exposure to propylene glycol, glycerol, aldehydes, and flavouring compounds[90][91][92].

Studies have shown that desflurane can increase sympathetic outflow, leading to tachycardia and hypertension—effects that may compound nicotine-induced sympathomimetic activity in vapers[93]. Conversely, sevoflurane has a more favourable airway profile, producing less bronchospasm, reduced coughing, and smoother induction, making it the volatile agent of choice in patients with reactive airways[94][95][96].

Given that vapers show increased baseline airway resistance and impaired mucociliary clearance, use of desflurane may worsen perioperative respiratory complications[97]. Therefore, sevoflurane should be the preferred volatile agent in this population, balancing both haemodynamic stability and minimising airway hyperreactivity. When desflurane is unavoidable, it should be titrated cautiously with adequate premedication (e.g., short-acting β_2 agonists, lidocaine) to mitigate airway irritation[98][99].

Bronchodilators and lidocaine:

Pre-induction administration of a bronchodilator, such as a short-acting β_2 agonist (e.g., salbutamol 200–400 μg inhaled), should be considered a reasonable adjunct in symptomatic or reactive airway patients[100]. Chronic e-cigarette use is associated with increased airway hyperresponsiveness, heightened vagal tone, and bronchial epithelial inflammation, leading to a higher risk of peri-intubation bronchospasm compared with non-users[101][102][103].

Intravenous lidocaine (0.5–2 mg/kg i.v.) administered peri-induction has been shown in multiple RCTs and meta-analyses to reduce the incidence of intubation- and extubation-related cough, blunt sympathetic responses, and attenuate airway reflexes[104]. This is particularly relevant in vapers, who demonstrate increased cough reflex sensitivity and impaired mucociliary clearance secondary to propylene glycol, glycerol, and flavouring chemical exposures[105][106][107].

Given that vaping is associated with eosinophilic airway inflammation and reduced exhaled nitric oxide (indicative of airway reactivity), standard bronchospasm algorithms should be prepared in advance, with inhaled β_2 agonists immediately available for intra-operative rescue[108]. Intraoperative bronchospasm stays a significant concern, as e-cigarette users exhibit higher

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[90] Gotts et al., 2019; Madison et al., 2019
[91] Ebert, T.J. & Muzi, M., 1993, *Anesthesiology*, 79(3), 444–453
[92] Ebert, T.J., et al., 1998, *Anesthesiology*, 89(6), 1294–1299
[93] Ebert & Muzi, 1993
[94] Lohser & Slinger, 2006
[95] Eger, E.I. II, et al., 1997, *Anesthesia & Analgesia*, 84(3), 479–485
[96] Lohser, J. & Slinger, P., 2006, *Anesthesia & Analgesia*, 102(2), 302–315
[97] Ebert & Muzi, 1993; Eger et al., 1997; Ebert et al., 1998
[98] Eger, E.I. II, et al., 1997, *Anesthesia & Analgesia*, 84(3), 479–485
[99] Lohser, J. & Slinger, P., 2006, *Anesthesia & Analgesia*, 102(2), 302–315
[100] Clivio et al., 2025
[101] Gotts et al., 2019; Madison et al., 2019
[102] Tanaka, Y., Nakayama, T., Nishimori, M., et al., 2017, *Cochrane Database of Systematic Reviews*, 2017(7), CD004081
[103] Wang, Y., et al., 2020, *British Journal of Anaesthesia*, 125(2), 238–250
[104] Sun et al., 2014; Tanaka et al., 2017
[105] Clapp et al., 2017
[106] Clivio, S., Putzu, A. & Tramèr, M.R., 2025, *Anesthesia & Analgesia*
[107] Clapp, P.W., Pawlak, E.A., Lackey, J.T., Keating, J.E., Reeber, S.L., Glish, G.L. & Jaspers, I., 2017, *American Journal of Physiology – Lung Cellular and Molecular Physiology*, 313(2), L278–L292
[108] O'Donnell & Dolan, 2018

perioperative airway reactivity rates comparable to, and in some cases exceeding, those of chronic smokers[109][110].

Thus, an anticipatory strategy combining pre-induction bronchodilator, peri-induction intravenous lidocaine, and vigilant preparation for bronchospasm aligns with best practice for optimising airway safety in vaping patients undergoing anaesthesia[111].

Neuromuscular management:

Because residual neuromuscular blockade increases the risk of airway compromise and pulmonary complications, quantitative neuromuscular monitoring (objective TOF measurement) is recommended whenever non-depolarising neuromuscular blockers are used, with confirmation of a TOF ratio ≥0.9 prior to extubation[112]. This aligns with contemporary ASA and AAGBI guidance, which both emphasise the role of objective monitoring in reducing residual paralysis–related airway events[113].

Emerging evidence suggests that patients who vape may show altered pharmacokinetics and higher metabolic clearance of neuromuscular blocking agents. Nicotine has been shown to induce cytochrome P450 (particularly CYP1A2 and CYP2E1) activity and increase hepatic blood flow, thereby enhancing the metabolism of several anaesthetic agents. In vapers, this pharmacological effect may shorten the duration of action of non-depolarising neuromuscular blockers, resulting in unpredictable recovery profiles and a higher risk of residual weakness if quantitative monitoring is not employed[114].

So, vigilant quantitative monitoring is essential in this patient cohort, with reversal guided by objective neuromuscular function rather than elapsed time. Use of sugammadex for aminosteroid blockers (e.g., rocuronium, vecuronium) or appropriate anticholinesterases should be tailored to patients with a well monitored recovery, as vape-associated variability in metabolism may otherwise predispose to incomplete reversal[115].

[109] Muthumalage et al., 2019
[110] Ibid see 85, 86
[111] Ibid see 84, 85, 86
[112] Naguib et al. 2018
[113] Brull, S.J. & Kopman, A.F., 2017, *Anesthesiology*, 126(1), 173–190
[114] Naguib, M., Brull, S.J., Kopman, A.F., et al., 2018, *Anesthesia & Analgesia*, 127(1), 71–80
[115] Ibid see 91

Clinical Checklist for Anaesthesia Care:

Phase	Recommendations
■ Pre-operative	• Obtain structured vaping history: device type/settings, nicotine concentration, PG:VG ratio, solvents, flavourings, use pattern, dual use. • Screen dependence: **PSECDI** tool. • Aim ≥3–8 weeks abstinence; arrange cessation support. • Selective PFTs/ABGs only if symptomatic/high-risk. • Imaging (CXR/CT) if suspicion of EVALI, pneumonitis, hypoxaemia.
■ Intra-op: Airway	• Prefer **IV induction** (propofol/TIVA). • Avoid repeated airway instrumentation. • Maintain deep anaesthetic plane for intubation/extubation. • Prepare bronchospasm rescue (β■-agonist ready).
■ Intra-op: Agents & Adjuncts	• Volatile choice: **Sevoflurane preferred**. • Avoid/cautiously titrate desflurane. • Pre-induction bronchodilator (short-acting β■ agonist). • Consider IV lidocaine (0.5–2 mg/kg). • Quantitative NM monitoring; confirm TOF ≥0.9. • Reversal with sugammadex/anticholinesterase per monitoring.
■ Monitoring	• **Respiratory:** Capnography, airway pressures, spirometry (high-risk). • **Cardiac:** Continuous ECG; arterial line in heavy users. • **Anaesthetic depth:** BIS/EEG if altered MAC suspected. • **Post-op:** Oximetry & capnography in PACU; consider overnight monitoring.
■ Post-operative	• Monitor for bronchospasm, hypoxia, MI, pulmonary infection. • Continue NRT if indicated. • Provide cessation counselling & support pre-discharge.

Future Clinical Research Recommendations[116]:

Prospective perioperative outcome studies:

Most existing data derive from observational studies, dual users, or extrapolation from cigarette smoking. Controlled cohort studies are needed to quantify the true incidence of complications such as bronchospasm, cardiovascular events, and altered analgesic requirements[117].

Dose–response and device-specific investigations:

Variability in device type, power settings, e-liquid composition, and puff topography influences toxicant exposure and perioperative risk. Research linking these parameters to clinical outcomes will allow more precise risk stratification and management[118].

Pharmacokinetic and pharmacodynamic studies:

The effect of chronic vaping on drug metabolism, neuromuscular blocker response, and analgesic requirements stays poorly defined. Mechanistic studies and clinical trials are needed to guide dosing adjustments and anaesthetic planning.

116 Ibid see 91
117 Warner et al., 2019; Tomaszek et al., 2021
118 Sleiman et al., 2016; Goniewicz et al., 2014

Interventional studies on perioperative optimisation:

Evidence for strategies such as pre-induction bronchodilators, intravenous lidocaine, and nicotine-replacement therapy in vapers is limited. Randomised or pragmatic trials could clarify efficacy, safety, and optimal protocols.

Long-term health and recovery outcomes:

The impact of vaping on wound healing, infection rates, and post-discharge morbidity is largely unknown. Longitudinal studies tracking postoperative recovery in e-cigarette users would inform both anaesthetic management and long-term care planning[119].

Educational initiatives and guideline development:

As the prevalence of vaping continues to rise, structured training for anaesthetists and multidisciplinary perioperative teams is needed. Evidence-based guidelines tailored to e-cigarette users will support consistent risk assessment, intraoperative management, and postoperative care[120].

Conclusion:

The rapid growth of e-cigarette use presents anaesthetists with an increasingly common but poorly defined clinical challenge. Vaping introduces a complex interplay of pulmonary, cardiovascular, neurological, immunological, and pharmacological effects that influence perioperative risk. Although parallels can be drawn from cigarette smoking literature, unique toxicological exposures in e-liquids—including aldehydes, metals, and flavouring agents—creates vaping-specific considerations[121].

This review highlights that patients who vape may be more vulnerable to airway hyper-reactivity, haemodynamic instability, impaired wound healing, and unpredictable anaesthetic drug responses. Structured preoperative assessment, vigilant intraoperative management, and enhanced postoperative monitoring are therefore essential. Practical measures include detailed vaping histories, targeted use of pulmonary and cardiovascular testing, and anticipatory strategies such as intravenous induction with propofol, preference for sevoflurane, and quantitative neuromuscular monitoring[122].

Despite emerging mechanistic and observational evidence, high-quality prospective outcome studies are still urgently needed. Future research should define dose–response relationships, device-specific risks, and evidence-based optimisation strategies. Until such data become available, clinicians must adopt a cautious, individualised approach grounded in pathophysiological understanding and extrapolated smoking literature.

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119 Joseph et al., 2024
120 El-Boghdadly et al., 2024
121 Gotts et al., 2019; Madison et al., 2019; Clapp et al., 2017
122 Ibid see 94

Vaping should not be regarded as benign in the perioperative setting. Proactive risk assessment, multidisciplinary collaboration, and integration of perioperative cessation strategies are key to improving outcomes for this expanding patient population. Continued awareness, education, and research are imperative to ensure anaesthetists remain prepared to meet the evolving challenges posed by vaping.

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