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Pharmacological Modulation of Hemodynamic Responses During Tracheal Extubation: A Narrative Review of Intravenous Magnesium Sulphate and Esmolol

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ABSTRACT:

Background:

Tracheal extubation is frequently accompanied by sympathetic stimulation, resulting in tachycardia, hypertension, coughing, and airway irritation. Although these responses are usually transient, they may precipitate serious cardiovascular, neurological, or surgical complications in high-risk patients. Pharmacological modulation is therefore an important component of safe extubation, with magnesium sulphate and esmolol commonly used due to their favourable haemodynamic profiles.

Methods:

A narrative review of published clinical trials, randomized controlled studies, meta-analyses, and review articles was conducted to evaluate the efficacy and safety of intravenous magnesium sulphate and esmolol for attenuation of haemodynamic responses during tracheal extubation. Evidence on mechanisms of action, dosing strategies, comparative effectiveness, and use in special patient populations was synthesized.

Results:

Both magnesium sulphate and esmolol effectively attenuate extubation-related haemodynamic responses. Magnesium maintains heart rate and blood pressure closer to baseline while providing bronchodilation and analgesic sparing, with minimal respiratory depression. Esmolol produces rapid and pronounced suppression of heart rate and blood pressure, offering superior control of tachycardia and hypertension but with a higher risk of bradycardia. Comparative studies suggest differing clinical advantages based on patient profile and surgical context.

Conclusion:

Intravenous magnesium sulphate and esmolol are effective agents for controlling haemodynamic responses during tracheal extubation. Agent selection should be individualized, balancing the need for haemodynamic stability, airway protection, and patient-specific comorbidities.

Introduction

Tracheal extubation—the removal of the endotracheal tube (ETT) when an airway is no longer needed—marks a critical transition between anaesthesia and recovery. Although often routine, extubation can provoke abrupt sympathetic activation via stimulation of laryngeal and tracheal mucosa, leading to transient increases in heart rate (HR) and blood pressure (BP), cough and airway irritation [1,2]. Many patients tolerate these responses without consequence; however, those with cardiovascular or neurosurgical disease, raised intracranial or intra ocular pressure, or significant surgical wounds may experience deleterious outcomes such as myocardial ischaemia, intracranial haemorrhage, wound dehiscence, and pulmonary oedema [3]. Extubation related cardiovascular and respiratory complications occur three times more frequently than complications at intubation (12.6 % vs 4.6 %) [4], emphasising the need for effective attenuation strategies.

Pharmacologic interventions have been extensively studied to modulate these extubation responses. Intravenous (IV) magnesium sulphate and esmolol—two drugs with distinct pharmacodynamic profiles—are increasingly used as adjuncts to blunt the sympathetic surge. This narrative review synthesises available research on magnesium and esmolol for peri extubation haemodynamic control, compares their efficacy to other pharmacologic agents and non pharmacologic techniques, discusses their role in special populations, and highlights gaps for future research. The outline follows the pathophysiology of extubation responses, pharmacologic classes used, individual analysis of magnesium and esmolol, comparative effectiveness and practical considerations.

Physiological basis of haemodynamic response during tracheal extubation

Airway reflexes during extubation originate from mechanoreceptors in the laryngeal and tracheal mucosa. Stimulation increases afferent input via the vagus and glossopharyngeal nerves, activating the medullary vasomotor centre and the sympathetic–adrenal axis. The resulting catecholamine release produces tachycardia, systemic and pulmonary hypertension, increased myocardial oxygen demand, arrhythmias and elevated intracranial or intra ocular pressure [5]. Bucking and coughing further increase intrathoracic and intra abdominal pressures which can impair venous return, promote negative pressure pulmonary oedema, and precipitate wound dehiscence [6,7]. The magnitude of haemodynamic response depends on airway stimulation intensity, depth of anaesthesia, patient factors (e.g., age, cardiovascular status, opioid tolerance) and surgical context.

Extubation responses often peak immediately and persist for 5–10 min but may last longer in high risk patients [4]. Patients undergoing neurosurgery, ophthalmic surgery, carotid endarterectomy and cardiac surgery are particularly vulnerable to hypertensive surges and arrhythmias. Conversely, extubating under deep anaesthesia reduces sympathetic activation yet may increase the risk of airway obstruction, laryngospasm and aspiration [8]. The need to balance haemodynamic stability and airway patency has driven the search for pharmacologic agents that provide smooth extubation without compromising respiratory function.

Pharmacological strategies for attenuation of extubation response

Multiple drug classes have been employed to attenuate extubation induced haemodynamic and airway responses. Sodium channel blockers (lidocaine) inhibit nerve conduction and suppress airway reflexes [9]; calcium channel blockers (diltiazem, verapamil) decrease vascular smooth muscle tone and heart rate [10]; β adrenergic blockers (esmolol, metoprolol, labetalol) blunt sympathetic output and tachycardia; α2 adrenergic agonists (dexmedetomidine) provide sedation and sympatholysis; opioids (fentanyl, remifentanil) offer analgesia and blunt catecholamine release; and NMDA antagonists or vasodilators (magnesium sulphate) attenuate catecholamine release and provide bronchodilation.

Local anaesthetics

Intravenous and intratracheal lidocaine decrease cough reflex and haemodynamic surges. A placebo controlled trial comparing intravenous lidocaine (1 mg kg^{-1}) alone versus diltiazem–lidocaine combinations showed that combined diltiazem and lidocaine provided greater reductions in HR, systolic and diastolic BP and mean arterial pressure from 1 to 10 min post extubation compared with lidocaine alone [10]. Intratracheal lidocaine may be more effective than intravenous administration. In a randomized study of breast surgery patients, intratracheal 2 % lidocaine 3 mg kg^{-1} suppressed post extubation cough and significantly reduced HR and BP compared with placebo, whereas intravenous lidocaine 1.5 mg kg^{-1} provided similar but slightly less pronounced benefits [9]. The authors concluded that intratracheal lidocaine is a simple, cost effective method for suppressing cough and haemodynamic responses [9]. Intracuff lidocaine similarly reduces postoperative sore throat and cough, although systemic absorption is unpredictable [11].

Calcium channel blockers

Diltiazem and verapamil block L type calcium channels, lowering vascular tone and HR. A randomized trial of 105 adults compared diltiazem 0.1 mg kg^{-1} + lidocaine versus diltiazem 0.2 mg kg^{-1} + lidocaine versus lidocaine alone. Both diltiazem–lidocaine groups exhibited lower HR and BP than lidocaine alone from 1 min through 10 min post extubation [10]. Although effective, calcium channel blockers may cause hypotension or bradycardia, limiting their routine use.

Opioids

Short acting opioids (fentanyl, remifentanil) suppress sympathetic responses but carry risk of respiratory depression. In a double blind trial of 120 laparotomy patients comparing magnesium sulphate 50 mg kg^{-1}, remifentanil 1 µg kg^{-1} and placebo, both magnesium and remifentanil groups had significantly lower HR and MAP immediately after extubation and at 3, 5 and 10 min compared with placebo [4]. Remifentanil produced more rapid recovery from neuromuscular blockade than magnesium but was associated with hypotension and bradycardia [4].

α2-adrenergic agonists (dexmedetomidine)

Dexmedetomidine activates α2 adrenoreceptors, reducing norepinephrine release and providing sedation without respiratory depression. In a randomized trial of 50 patients, an infusion of dexmedetomidine 0.75 µg kg^{-1} given over 15 min before extubation resulted in significantly lower HR, systolic, diastolic and mean arterial pressures from 5 min after infusion until 20 min after extubation compared with placebo [12]. Extubation quality scores indicated smoother extubation and higher sedation in the dexmedetomidine group, although bradycardia and hypotension were more frequent [12]. The narrative review on smooth extubation techniques reports that dexmedetomidine 0.5–0.75 µg kg^{-1} reduces cough and agitation but prolongs emergence and may cause bradycardia [11].

Beta-adrenergic blockers

Ultra short acting β1 blockers such as esmolol and longer acting agents like metoprolol provide rapid sympatholysis. The 2025 meta analysis of 31 randomized controlled trials involving 1,803 patients undergoing extubation found that beta blockers significantly reduced systolic, diastolic and mean arterial pressures and mean HR at 1, 2, 5, 10 and 15 min compared with placebo [12]. Beta blockers also lowered the incidence of hypertension and tachycardia by 72 % and 80 %, respectively [12]. Metoprolol and esmolol reduce coughing by blocking sympathetic activation of vagal afferent C fibres [12]. However, calcium channel blockers like diltiazem or verapamil may be less favourable because of higher rates of hypotension and bradycardia [10].

Magnesium sulphate

As a polyvalent cation, magnesium antagonizes calcium influx at presynaptic nerve terminals, inhibits catecholamine release from adrenal medulla and peripheral nerves, blocks NMDA receptors, and causes smooth muscle relaxation. These properties confer vasodilation, anti arrhythmic effects and bronchodilation [1]. Magnesium reduces depolarising neurotransmission and enhances the effect of neuromuscular blocking agents, but high doses can lead to hypotension or respiratory depression [1].

Intravenous magnesium sulphate

Mechanisms and pharmacokinetics

Magnesium's attenuation of extubation responses arises from its ability to block presynaptic voltage dependent calcium channels, reducing catecholamine release and decreasing smooth muscle contractility. It also antagonizes NMDA receptors and decreases acetylcholine release at the neuromuscular junction, producing mild muscle relaxation [13]. Infusions produce vasodilation and negative chronotropic effects, lowering systemic BP and HR. Magnesium has a rapid onset (~3–5 min) and elimination half life of ~4 h; sedation may be prolonged with large bolus doses, especially in renal impairment [14].

Clinical evidence

A multitude of randomized trials have evaluated magnesium for extubation. In a prospective double blind study (40 patients per group), 40 mg kg^{-1} magnesium sulphate or 0.6 mg kg^{-1} esmolol was administered 3 min before extubation. Both agents significantly reduced HR and mean arterial pressure compared with baseline; however, magnesium maintained HR and BP closer to baseline while esmolol produced >20 % falls in HR, systolic BP and MAP. Magnesium provided smoother extubation with fewer episodes of coughing or laryngospasm. The authors concluded that magnesium is more effective than esmolol in preventing haemodynamic fluctuations [15].

Appagalla et al. randomized 60 ASA I–II adults undergoing elective surgery to receive magnesium 30 mg kg^{-1} or saline. HR, systolic and diastolic BP and MAP were significantly lower in the magnesium group from extubation through 15 min post extubation, and extubation quality scores were better with magnesiumjcdronline.org [16]. Ramsay sedation scores were higher but without respiratory depressionjcdronline.org [16]. In a dose response study, Honarmand et al. compared magnesium 30, 40 and 50 mg kg^{-1}. All doses below 50 mg kg^{-1} reduced haemodynamic responses during laryngoscopy; doses ≥50 mg kg^{-1} produced bradycardia and hypotension [17]. Based on these findings, doses of 30–40 mg kg^{-1} are most commonly used.

Randomized trials also evaluated magnesium as an adjunct to other agents. The remifentanil–magnesium study described above found that magnesium (50 mg kg^{-1}) decreased HR and MAP at immediate and 3 min after extubation compared with placebo [18]. The double lumen tube trial by Hur et al. infused magnesium 15 mg kg^{-1} h^{-1} after a 30 mg kg^{-1} bolus. Although magnesium did not reduce the incidence of emergence cough, it significantly reduced the severity of cough and lowered HR without causing hypotension or delayed recovery [19]. Another study in smokers undergoing surgery compared magnesium to dexmedetomidine and found that both drugs reduced coughing; however, dexmedetomidine provided deeper sedation and more pronounced haemodynamic suppression whereas magnesium had delayed onset [20].

Safety profile

Magnesium's side effects are dose related. Hypotension, bradycardia and respiratory depression may occur at high plasma concentrations [14]. It potentiates neuromuscular blockers and may prolong recovery, though standard doses (30 mg kg^{-1} bolus and 15 mg kg^{-1} h^{-1} infusion) have been shown not to delay extubation [19]. Magnesium should be used cautiously in patients with renal impairment because elimination is renal. Overall, magnesium appears safe when titrated appropriately and has the advantage of preserving respiratory function compared with opioids and sedatives.

Intravenous esmolol

Mechanisms and pharmacokinetics

Esmolol is an ultrashort acting, cardio selective β1 adrenergic blocker with rapid onset (within 2 min) and very short elimination half life (~9 min) [21]. By blocking β1 receptors in the heart, esmolol reduces HR, myocardial contractility and cardiac output, thereby lowering BP and myocardial oxygen consumption. It does not exert intrinsic sympathomimetic or membrane stabilizing activity and is hydrolysed by plasma esterases, making its effects quickly reversible [21].

Clinical evidence

A single bolus of esmolol 2 mg kg^{-1} administered 2 min before extubation has been shown to attenuate tachycardia and hypertension. In a randomized double blind trial of 45 adults undergoing abdominal surgery, the incidence of tachycardia (>20 % increase) was only 2 % in the esmolol group versus 49 % in the placebo group, and hypertension (>20 % increase) was 4 % versus 31 % [21]. Patients receiving esmolol had smoother extubation with less coughing [21]. Another study in craniotomy patients receiving esmolol 2 mg kg^{-1} 5 min before extubation reported significant reductions in systolic, diastolic and mean arterial pressures and HR after extubation and improved extubation scores compared with saline [21].

Comparative trials with other β blockers demonstrate esmolol's superiority during extubation. Patel and Shashank randomized 60 patients to receive esmolol 1.5 mg kg^{-1} or labetalol 0.25 mg kg^{-1}. Both drugs attenuated extubation responses, but esmolol was more effective at controlling SBP, DBP and MAP immediately post extubation, whereas labetalol provided better HR control at 5–15 min [21]. A prospective study combining esmolol 0.5 mg kg^{-1} with lidocaine 1 mg kg^{-1} showed superior reduction in HR compared with diltiazem–lidocaine or lidocaine alone from 1 min to 10 min post extubationhealthcare-bulletin.co.uk [21].

Esmolol has also been compared with magnesium. In the trial of Lokhande et al., esmolol lowered HR and BP more rapidly than magnesium but induced >20 % reductions in haemodynamic variables whereas magnesium maintained them near baseline [15]. A study evaluating esmolol 1 mg kg^{-1} versus magnesium 30 mg kg^{-1} found that esmolol prevented haemodynamic surges better than magnesium, though both drugs reduced HR and MAP compared with controlresearchgate.net [21]. The difference may be attributed to esmolol's faster onset and more pronounced sympatholysis; however, magnesium offered smoother extubation with less risk of bradycardia.researchgate.net [21]

Esmolol's utility extends beyond healthy adults. The randomised trial of 207 patients investigated metoprolol, another β blocker, but provides insight into β blocker class effects. Metoprolol 5 mg intravenously administered before extubation reduced the risk of bucking (43 % vs 65 %) and moderate or severe coughing (6 % vs 31 %) compared with placebo [21], supporting the notion that β blockers attenuate airway reflexes and haemodynamic responses via sympatholytic mechanisms [21].

Safety profile and adverse effects

Esmolol's rapid metabolism confers an excellent safety profile. Hypotension and bradycardia may occur but are quickly reversible due to short half life [21]. In the single dose studies mentioned, only minor adverse events were reported and no patient required vasopressors [21]. Esmolol should be titrated cautiously in patients with heart block, heart failure or reactive airway disease. Continuous infusions (1–2 mg kg^{-1} h^{-1}) effectively attenuate haemodynamic responses during intubation and extubation and reduce intraoperative opioid requirementspdfs.semanticscholar.org [21]. However, high maintenance doses (≥2 mg kg^{-1} h^{-1}) may be required to suppress extubation responses, which could increase risk of hypotensionpdfs.semanticscholar.org [21].

Comparative analysis: magnesium vs esmolol and other agents

Both magnesium and esmolol reduce haemodynamic responses to extubation, yet their efficacy and profiles differ. Magnesium acts via calcium antagonism and NMDA blockade, providing vasodilation and bronchodilation without profound bradycardia. Esmolol exerts rapid β1 blockade producing strong suppression of HR and BP [22].

Randomised trials reveal that magnesium maintains haemodynamic variables closer to baseline while esmolol causes deeper reductions. In Lokhande et al., magnesium preserved HR and MAP within ±20 % of baseline whereas esmolol decreased them by >20 % [22]. Appagalla et al. reported that magnesium significantly attenuated HR and BP and improved extubation quality without major side effects [23]. Conversely, the Brazilian trial showed that esmolol reduced tachycardia incidence from 49 % to 2 % and hypertension from 31 % to 4 % [24]; extubation quality was smoother, but bradycardia occurred in some patients [24].

When combined with lidocaine, esmolol provided better HR control than diltiazem–lidocaine combinations [25]. However, magnesium also demonstrates additive effects when used with opioids or anaesthetics; it reduces intraoperative opioid consumption and enhances analgesia [26]. The meta analysis of beta blockers suggests that while β blockers more consistently reduce hypertension and tachycardia, magnesium may offer broader benefits such as bronchodilation and anti nociception [27].

Other agents such as dexmedetomidine and remifentanil also provide effective attenuation. Dexmedetomidine yields smoother extubation and better cough suppression than magnesium but prolongs emergence and increases bradycardia [28]. Remifentanil suppresses HR and MAP similar to magnesium but allows faster recovery [29]. Lidocaine, whether intravenous or intratracheal, reduces cough and haemodynamic surges [30]. Calcium channel blockers attenuate BP and HR but risk hypotension [31]. Overall, the choice between magnesium and esmolol depends on patient comorbidities, desired depth of attenuation, and potential side effects.

Special patient populations and clinical scenarios

Cardiac and hypertensive patients

Patients with ischemic heart disease or poorly controlled hypertension may not tolerate tachycardia or hypertension at extubation. β blockers are especially beneficial in this population [27]. The Brazilian trial demonstrated that esmolol significantly reduced tachycardia and hypertension without provoking severe hypotension [24]. Metoprolol reduced bucking and moderate/severe cough in a diverse cohort and may be preferable for longer procedures [27]. Magnesium also offers haemodynamic stability and may be used when β blockers are contraindicated or when bronchodilation is desired. However, caution is required in patients with reduced ejection fraction or conduction delays because magnesium can cause bradycardia and hypotension at higher doses [22].

Neurosurgical and ophthalmic procedures

In neurosurgery and ophthalmic surgery, preventing increases in intracranial or intraocular pressure is paramount. Studies in craniotomy patients show that esmolol 2 mg kg^{-1} administered 5 min before extubation significantly lowers HR and BP and improves extubation scores compared with saline [24]. Dexmedetomidine has also been used successfully to blunt hypertensive surges in neurosurgical patients but may delay neurological assessment [28]. Magnesium provides vasodilation without excessive bradycardia and could be considered, but data in neurosurgical patients are limited [22].

Obstetric and pediatric patients

Research on magnesium and esmolol during extubation in obstetric and pediatric populations remains limited. The sympatholytic effects of β blockers may risk uterine relaxation and fetal bradycardia, so their use should be cautious. Magnesium is commonly used for eclampsia prophylaxis and may provide additional benefit of haemodynamic control during caesarean extubation; however, further studies are

required. In pediatrics, the airway is particularly reactive, and non pharmacologic techniques such as deep extubation, LMA exchange and careful suctioning are usually favored [32]. The review on smooth extubation techniques highlights that pharmacologic aids may have different efficacy in high risk or pediatric patients [32].

Smokers and patients with reactive airways

Smokers often have a heightened cough reflex. In a randomized trial of heavy smokers, dexmedetomidine provided better cough suppression than magnesium, although both reduced cough frequency [28]. Magnesium's bronchodilatory properties may still be advantageous in patients with reactive airway disease, but dosing should be considered alongside potential sedative effects [22]

Practical considerations for clinical use

Drug selection

Choice of pharmacologic agent should be tailored to patient comorbidities, surgical type, and available resources. Esmolol is ideal when rapid, titratable β blockade is needed and in patients with high risk of tachycardia or hypertension [24]. Magnesium sulphate is advantageous when bronchodilation and analgesia are desired, or when β blockers are contraindicated [22,26]. Dexmedetomidine offers sedation and sympatholysis but may delay recovery [28]. Lidocaine is useful for suppressing cough and sore throat with minimal haemodynamic impact, particularly via intracuff or intratracheal routes [30]. Calcium channel blockers may be reserved for patients intolerant of β blockers and magnesium but require caution due to potential hypotension [31]. Opioids are effective but risk respiratory depression and delayed recovery; remifentanil may be preferred due to short half life [29].

Timing and dosing

Both magnesium and esmolol should be administered shortly before anticipated extubation. Most studies administered magnesium 30–40 mg kg^{-1} bolus 3–5 min before extubation; continuous infusion (15 mg kg^{-1} h^{-1}) can be considered during emergence [23]. Esmolol dosing ranges from 0.5 to 2 mg kg^{-1} bolus given 2–5 min pre extubation, with or without maintenance infusion (0.5–2 mg kg^{-1} h^{-1}) [24,26]. Higher doses provide more pronounced haemodynamic control but risk hypotension and bradycardia; thus incremental titration is advised [24]. Dexmedetomidine is usually given as 0.5–0.75 µg kg^{-1} over 10–15 min prior to extubation [28].

Multimodal approaches and non-pharmacologic techniques

Combining agents with different mechanisms may provide synergistic benefits. For example, esmolol plus lidocaine improved HR control compared with diltiazem–lidocaine or lidocaine alone [25]. Magnesium can be paired with opioids or dexmedetomidine for enhanced analgesia and cough suppression [29]. Non pharmacologic techniques such as deep extubation, insertion of a laryngeal mask airway (LMA) before removing the ETT, and limiting airway stimulation complement pharmacologic aids. The narrative review on smooth extubation emphasises developing an extubation strategy, selecting low risk vs high risk patients, and employing adjuncts like intracuff lidocaine, topical steroids and careful suctioning [32].

Gaps in current evidence and future directions

Despite numerous trials, heterogeneity in study design, dosing regimens and outcome measures limits cross-comparison. Many studies are single-centre with small sample sizes and enrol ASA I–II patients undergoing uncomplicated surgery. There is a need for larger randomised trials involving high-risk populations (e.g., elderly, cardiac, obese, obstetric and paediatric patients) to determine optimal dosing and safety. Comparative trials directly assessing magnesium vs esmolol vs dexmedetomidine using standardized outcomes (e.g., composite cardiovascular events, extubation quality scores, patient-centred outcomes) would guide clinical decision-making. The effect of combining these agents with enhanced recovery protocols and opioid-sparing strategies warrants further study. Additionally, the long-term impact of attenuating extubation responses on postoperative morbidity, mortality and resource utilisation remains unclear and should be explored.

Tracheal extubation often provokes significant haemodynamic and airway responses that can harm vulnerable patients. Pharmacologic modulation forms a cornerstone of extubation management. Intravenous magnesium sulphate attenuates HR and BP surges by blocking calcium influx, inhibiting catecholamine release and providing bronchodilation, with minimal respiratory depression and improved extubation quality. Esmolol, an ultrashort-acting β1-blocker, rapidly suppresses sympathetic activity, markedly reducing tachycardia and hypertension but risks bradycardia and hypotension__. Comparative studies suggest that magnesium maintains haemodynamic parameters closer to baseline whereas esmolol produces deeper reductions; each has unique advantages depending on patient profile and surgical context. Beta-blockers overall reduce cough, hypertension and tachycardia, while magnesium may offer additional analgesia and bronchodilation_. Dexmedetomidine, lidocaine, calcium channel blockers and opioids provide alternative or adjunctive options but carry specific drawbacks.

Clinicians should individualise pharmacologic strategies based on patient comorbidities, procedure type and extubation risk. Future research should focus on standardised dosing protocols, multimodal combinations, and outcomes relevant to high-risk populations. Achieving a smooth extubation not only prevents immediate cardiovascular and respiratory complications but may also reduce postoperative morbidity and improve recovery trajectories.

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