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Endotracheal Intubation and Medication Management: Evidence-Based Nursing Interventions for Airway Stabilization and Sedation

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Abstract

Background: Endotracheal intubation is a critical intervention in emergency and critical care settings, requiring rapid airway control to prevent hypoxemia, aspiration, and cardiovascular collapse. Nurses play a pivotal role in preparation, medication management, and post-intubation care.

Aim: This study aims to review evidence-based nursing interventions for airway stabilization and sedation during rapid sequence intubation (RSI), focusing on pharmacologic mechanisms, procedural safety, and team-based strategies.

Methods: A comprehensive literature review was conducted, synthesizing current guidelines and clinical evidence on RSI, including indications, drug selection, endotracheal tube (ETT) characteristics, and the "7 Ps" framework for safe administration.

Results: RSI is the standard of care for emergent airway management when difficulty is not anticipated. Induction agents such as etomidate, ketamine, and propofol provide rapid sedation, while neuromuscular blockers like succinylcholine and rocuronium ensure paralysis. Each drug has unique hemodynamic and neurologic profiles, influencing selection based on patient physiology. Nursing responsibilities include preoxygenation, equipment readiness, accurate dosing, monitoring for adverse effects, and initiating post-intubation sedation. Common complications include aspiration, esophageal intubation, hypotension, and medication-specific risks such as adrenal suppression (etomidate), emergence reactions (ketamine), and hypotension (propofol).

Conclusion: RSI success depends on meticulous preparation, pharmacologic literacy, and interprofessional collaboration. Nurses are integral to preventing complications and ensuring safe transitions to mechanical ventilation.

Keywords: Rapid sequence intubation, endotracheal tube, nursing interventions, airway management, sedation, neuromuscular blockade

Introduction

Mastery of endotracheal intubation is a foundational competency in nursing practice across time-sensitive and high-acuity environments, including pre-hospital care, emergency departments, intensive and critical care units, and peri-operative medicine. In these settings, rapid deterioration can occur within minutes, and the ability to recognize impending respiratory failure and support definitive airway management can determine clinical outcomes. Although endotracheal intubation is typically performed by physicians or advanced airway providers depending on local scope of practice, nurses play an essential role in patient assessment, preparation of medications and equipment, physiologic optimization before induction, continuous monitoring during the procedure, and immediate post-intubation stabilization. Success in airway emergencies depends on coordinated teamwork as well as clinical judgment regarding anticipated airway difficulty, the patient's underlying condition, and the physiologic risks associated with induction and paralysis. Rapid sequence intubation (RSI) is a structured method used to achieve prompt control of the airway. It involves the near-simultaneous administration of an induction agent and a neuromuscular blocking drug to create optimal conditions for endotracheal tube placement.[1] The rationale for RSI is that rapid onset sedation and paralysis facilitate visualization of the vocal cords, minimize patient resistance and airway reflexes, and reduce the risk of aspiration by shortening the time between loss of airway protective reflexes and cuff inflation within the trachea. For nursing and interprofessional teams, RSI is not merely a pharmacologic event; it is a safety-critical sequence

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that requires meticulous preparation, anticipation of complications, and adherence to evidence-based steps. This clinical activity, therefore, encompasses more than the act of tube placement itself and includes understanding indications, the pharmacologic mechanisms of commonly used sedatives and paralytics, equipment selection (including endotracheal tube considerations), the core sequence of intubation steps, potential adverse effects, and contraindications relevant to the medications most frequently used.[1]

FDA-Approved Indications

Emergent endotracheal intubation is typically indicated when a patient is unable to maintain oxygenation, ventilation, or airway protection, or when clinical trajectory suggests imminent failure of these functions. While individual cases differ, several high-yield scenarios are consistently recognized as core indications for emergent intubation. One of the most common is hypoxemic respiratory failure that persists despite maximal noninvasive support. When severe hypoxemia remains refractory to high inspired oxygen concentrations or to non-invasive positive pressure ventilation, invasive airway control and mechanical ventilation may become necessary to support oxygen delivery and reduce the work of breathing. From a nursing perspective, these patients often demonstrate escalating respiratory distress, tachypnea, accessory muscle use, fatigue, and deteriorating oxygen saturation trends that do not respond to escalation of noninvasive measures. In such cases, delayed transition to definitive airway control may increase the risk of cardiac arrest, aspiration, or multi-organ injury. Hypercapnic respiratory failure is another major indication, particularly when carbon dioxide retention leads to respiratory acidosis and increased work of breathing that signals impending ventilatory collapse. Patients with hypercapnic failure may present with somnolence, confusion, headache, or signs of respiratory muscle fatigue, and blood gas analysis may demonstrate worsening acidosis. Nursing surveillance is pivotal in identifying these trends early, because clinical decline can occur rapidly and may be underestimated if oxygen saturation is deceptively preserved with supplemental oxygen. When ventilation cannot be supported effectively with noninvasive measures or when mental status deteriorates, intubation becomes an essential intervention to restore adequate alveolar ventilation and reduce physiologic stress. Endotracheal intubation is also indicated when upper airway obstruction or injury threatens airway patency. Examples include thermal burns, caustic inhalation, facial or neck trauma, and progressive edema that may evolve unpredictably. In these scenarios, intubation is often performed early rather than late, because waiting for overt obstruction may convert a manageable airway into a “cannot intubate, cannot ventilate” emergency. Nursing assessment of voice changes, stridor, soot in the oropharynx, swelling, and escalating respiratory effort contributes to the rapid identification of deterioration risk. Inhalational injuries are particularly important because airway edema may progress even as initial presentation seems stable, and proactive airway stabilization can prevent catastrophic obstruction [1][2].

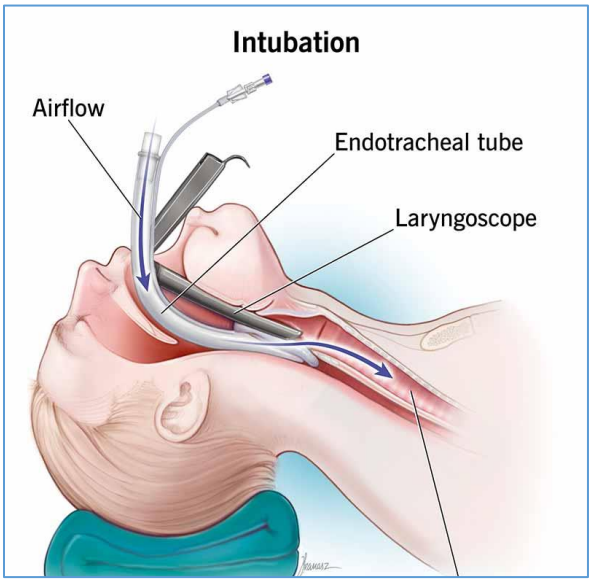

Fig. 1: Endotracheal Intubation.

Shock and hemodynamic instability represent another important indication when associated with altered mentation and increased work of breathing. Patients in shock may require intubation not only because of inadequate oxygenation or ventilation, but also to reduce metabolic demand and improve physiologic control while resuscitation proceeds. However, shock physiology increases the risk of peri-intubation cardiovascular collapse, particularly with sedative agents that may reduce sympathetic tone or myocardial contractility. Nursing preparation in these cases includes ensuring vascular access, optimizing volume status and vasopressor support where indicated, and anticipating abrupt blood pressure changes after induction. Intubation in shock therefore requires heightened vigilance, careful drug selection, and proactive hemodynamic monitoring. Clinical conditions associated with risk for airway compromise are also frequent indications, including stroke, drug overdose, and coma.[2] In these scenarios, the primary concern is often loss of protective airway reflexes, leading to aspiration risk and inability to maintain airway patency. A patient with decreased level of consciousness may not be able to protect against secretions or emesis, and neurologic deficits may impair breathing pattern and airway tone. Nurses contribute significantly by performing serial neurologic assessments, recognizing changes in airway protective responses, and communicating clinical deterioration to the airway team. Importantly, airway compromise risk is not restricted to neurologic etiologies; severe agitation, seizures, or intoxication

may also make oxygenation and ventilation unreliable and may necessitate intubation to stabilize the patient and allow safe diagnostic and therapeutic interventions [2].

Rapid Sequence Intubation as the Standard of Care

Regardless of the underlying indication, rapid sequence intubation is widely considered the standard of care for conditions requiring quick airway control when the airway is not anticipated to be difficult.[3] This principle reflects the balance between speed, safety, and physiologic control. RSI's defining feature is the combination of a sedative induction agent and a neuromuscular blocker. Together, these medications render the patient unconscious and produce flaccid paralysis, facilitating endotracheal tube placement and minimizing aspiration risk by reducing coughing, gagging, and uncontrolled movement during laryngoscopy.[4] For nursing practice, understanding this pharmacologic synergy is essential because nurses often prepare, administer, and monitor these medications, and they must be prepared to recognize adverse effects such as hypotension, apnea, arrhythmias, or allergic reactions. Common induction agents used during RSI include etomidate, ketamine, and propofol, while commonly used neuromuscular blocking agents include succinylcholine and rocuronium. Each agent has distinct onset characteristics, hemodynamic effects, and contraindications, and the optimal selection is shaped by patient physiology. For example, an induction agent that is hemodynamically well tolerated may be preferred in shock, whereas agents with bronchodilatory effects or favorable neurologic profiles may be advantageous in other contexts. Similarly, succinylcholine offers rapid onset and short duration but has specific contraindications, while rocuronium provides a longer duration of paralysis that may be beneficial or problematic depending on the scenario. The effectiveness and safety of these drugs are therefore not uniform across patient populations, and careful selection is part of high-quality airway management. In addition, nurses play a major role in the broader RSI workflow, including preparation of suction, oxygenation strategies, positioning, preoxygenation, monitoring setup, equipment checks, medication labeling, and readiness for rescue ventilation or alternative airway devices. The pharmacologic phase cannot be separated from these procedural safety measures, because the period after induction but before successful tube placement is one of the highest-risk moments in acute care. Accordingly, the indications for intubation are inseparable from readiness to execute RSI safely and to manage immediate complications. These considerations are explored in greater detail in subsequent discussions of specific clinical scenarios, as well as the adverse effects and contraindications associated with commonly used sedatives and paralytic agents.[1][2][3][4]

Mechanism of Action

Rapid sequence intubation (RSI) is a coordinated pharmacologic and procedural intervention designed to create optimal conditions for rapid endotracheal tube placement while minimizing the physiologic stress and aspiration risk associated with airway manipulation. The central pharmacologic objective of RSI is to achieve immediate unconsciousness, suppression of airway reflexes, and skeletal muscle relaxation, thereby enabling successful laryngoscopy and tracheal intubation on the first attempt whenever possible. To accomplish this, clinicians commonly employ an induction agent—selected for its ability to rapidly produce hypnosis and amnesia—and a neuromuscular blocking agent (NMBA) to induce flaccid paralysis. Although airway equipment and tube devices are indispensable, medications largely determine the patient's physiologic trajectory during the most vulnerable phase: the transition from spontaneous breathing to an apneic, mechanically supported state. For nursing and interprofessional teams, understanding the mechanism of action of RSI medications is clinically significant because the pharmacodynamics of these agents influence hemodynamics, intracranial physiology, bronchial tone, and the risk of adverse events such as hypotension, arrhythmias, or prolonged paralysis. In practice, medication selection is rarely "one size fits all." Instead, it is shaped by patient physiology, suspected intracranial pathology, airway reactivity, and the anticipated course of post-intubation sedation and ventilation [3][4].

Etomidate

Etomidate is frequently described as the most commonly used induction agent in RSI because it provides rapid hypnosis with comparatively favorable hemodynamic stability. Mechanistically, etomidate is a non-barbiturate sedative-hypnotic that produces central nervous system depression primarily through potentiation of gamma-aminobutyric acid (GABA) neurotransmission. Specifically, etomidate enhances the activity of the GABA-A receptor complex, increasing inhibitory synaptic signaling and thereby producing rapid sedation and loss of consciousness. The pharmacologic advantages of etomidate are closely linked to its speed and predictability. It typically has a short onset of action—often within 30 to 60 seconds—and a brief duration of hypnotic effect, with a short half-life commonly described in the range of minutes.[5] These properties make etomidate well suited to RSI, where rapid onset is needed to avoid prolonged airway manipulation and brief duration is desirable to facilitate transition into maintenance sedation that can be titrated after the airway is secured. From a physiologic standpoint, the defining advantage of etomidate is that it produces minimal direct cardiovascular depression at standard induction doses, and it is often characterized as having limited effect on systemic blood pressure.[5] This feature is particularly important in patients with pre-existing hypotension,

hemorrhagic shock, or trauma, where induction-related vasodilation or myocardial depression can precipitate peri-intubation cardiovascular collapse. Etomidate also demonstrates central nervous system effects that may be advantageous in certain neurocritical contexts, including reductions in cerebral blood flow and cerebral metabolic demand for oxygen.[5] These effects can be clinically relevant when intubation is performed for airway protection in patients with traumatic brain injury or raised intracranial pressure, where minimizing surges in intracranial dynamics is a key objective. For nursing practice, the clinical significance lies in anticipating etomidate's rapid onset and brief duration, ensuring continuous monitoring through the induction window, and preparing for the transition to post-intubation sedation so that awareness does not occur as etomidate wears off [5].

Drug	Dose range,[a] mg/kg	Onset, seconds	Duration, minutes	Analgesia	Hemodynamic response	Observations/adverse effects
Etomidate	0.2-0.3, 0.15	15-45	3-12	No	Minimal change in cardiac output	Causes adrenal insufficiency
Propofol	2, 0.5	15-45	5-10	No	Sympathetic depression, hypotension common	Reduces airway resistance
Dexmedetomidine	0.5-1 μg/kg (slow intravenous bolus)[b]	10-30 minutes	4 hours	Yes	30% hypotension with rapid administration; may also cause hypertension	Does not depress respiratory drive
Methohexital	0.75-1, 0.5	<30	4-6	No	Peripheral vasodilatation, negative inotropic effect, positive chronotropic effect (less hypotension than thiopental)	Can cause laryngospasm
Thiopental	3, 1.5	<30	5-10	No	Peripheral vasodilatation; negative chronotropic effect; hypotension very common	Decrease in systemic blood pressure less than decrease in intracranial pressure, so preserves cerebral perfusion pressure; may cause histamine release and bronchospasm
Ketamine	1-2, 0.25-0.5	45-60	10-20	Yes	Sympathetic stimulation, may observe hypertension	Dissociative anesthesia with preserved airway; bronchodilatation

Fig. 2: Endotracheal Tube Medication.

Ketamine

Ketamine occupies a distinct pharmacologic category among RSI induction agents because it provides dissociative anesthesia with analgesic and sympathomimetic properties. Chemically derived from the phencyclidine class, ketamine acts primarily as a noncompetitive antagonist at the N-methyl-D-aspartate (NMDA) receptor, a glutamate receptor subtype central to excitatory neurotransmission and pain processing.[6] By reducing excitatory signaling, ketamine produces a dissociative state in which the patient is functionally disconnected from sensory input, with accompanying amnesia and analgesia. Unlike many sedative-hypnotics that may reduce sympathetic tone, ketamine often increases catecholamine release and inhibits catecholamine reuptake, producing a sympathomimetic effect that can increase heart rate and blood pressure in patients with preserved sympathetic reserves. This physiologic profile may be beneficial in patients at risk of hypotension during induction, although the clinical effect can vary depending on the patient's baseline catecholamine status and overall physiologic reserve. Ketamine's onset for induction is typically rapid, often occurring within 1 to 2 minutes, and it has a short-to-moderate duration, with a half-life commonly described in the range of several minutes.[6] These kinetics allow clinicians to achieve timely conditions for intubation while maintaining the option to provide ongoing sedation after the airway is secured. A particularly important clinical feature of ketamine is its bronchodilatory effect, which is attributed to a combination of sympathetic stimulation, direct smooth muscle relaxation, and attenuation of airway reflex hyperreactivity. This makes ketamine a preferred agent in patients with acute severe asthma or bronchospasm, where airway manipulation and stress can worsen obstruction and where bronchodilation can support ventilation after intubation. For nurses, ketamine's mechanism is relevant to both respiratory and hemodynamic management: teams should anticipate potential increases in secretions, changes in blood pressure, and the need for careful monitoring of respiratory mechanics in patients with reactive airway disease [6].

Propofol

Propofol is a widely used sedative-hypnotic with rapid onset and short duration that makes it valuable both for induction and for maintenance sedation through continuous infusion. Its mechanism of action is multifactorial, but its predominant effect is potentiation of GABA-A receptor activity, enhancing inhibitory neurotransmission in the central nervous system and producing rapid hypnosis.[7][8] Because propofol is highly lipid soluble, it crosses the blood–brain barrier quickly and produces sedation within seconds—often described as 9 to 50 seconds—making it among the fastest induction agents used in RSI.[7][8] Propofol's duration is brief, with a short half-life commonly described as minutes, which contributes to its titratability and suitability for infusion-based sedation in mechanically ventilated patients.[7][8] Clinically, propofol has properties beyond hypnosis that influence its role in airway management. It has anticonvulsant effects and antiemetic properties, and it can reduce intracranial pressure through decreases in cerebral metabolic rate and cerebral blood flow.[7][8] These characteristics can be advantageous in patients with traumatic brain injury, elevated intracranial pressure, or status epilepticus, where controlling cerebral physiology is a priority. However, propofol's rapid onset and potent vasodilatory and myocardial depressant effects mean that it can cause clinically significant hypotension, particularly in hypovolemic patients or those with limited cardiovascular reserve. Therefore, while propofol can be an excellent choice in hemodynamically stable patients—especially those who benefit from intracranial pressure reduction—it requires careful selection and close monitoring in shock states. From a nursing perspective, the key mechanistic implication is that propofol can change hemodynamics quickly; therefore, pre-induction blood pressure optimization, readiness to administer fluids or

vasopressors when indicated, and vigilant post-induction monitoring are essential components of safe practice [7][8].

Neuromuscular Blocking Agents

Neuromuscular blocking agents are central to RSI because they provide the skeletal muscle paralysis that facilitates laryngoscopy, improves glottic exposure, and reduces airway trauma by limiting coughing, gagging, and reflexive movement. NMBAs act at the neuromuscular junction to interrupt transmission of motor signals to skeletal muscle. Importantly, they do not provide sedation, analgesia, or amnesia. Consequently, they must always be used in conjunction with an induction agent to ensure that the patient is unconscious and unaware of the procedure; otherwise, the patient may experience awareness with paralysis, a severe and ethically unacceptable outcome. For nursing teams, this principle has practical implications: medication preparation must ensure that induction drugs are administered effectively and that post-intubation sedation is initiated promptly, particularly when the induction agent has a short duration and the NMBA persists for a longer interval. Succinylcholine is the prototypical depolarizing NMBA and is widely used in RSI because of its very rapid onset and short duration. Mechanistically, succinylcholine acts as an acetylcholine receptor agonist at the motor endplate, producing sustained depolarization that prevents repolarization and blocks subsequent neuromuscular transmission. The initial depolarization typically produces visible fasciculations, followed quickly by flaccid paralysis as the neuromuscular junction becomes unable to generate additional action potentials. Succinylcholine's onset is commonly within 30 to 60 seconds, and its duration is relatively short—often approximately 5 to 15 minutes—making it highly compatible with the rapid sequence goal of achieving immediate paralysis while allowing recovery if intubation fails and rescue ventilation becomes necessary.[9][10] This short duration can be operationally beneficial during difficult airway scenarios because it offers a theoretical pathway back to spontaneous respiration, although clinical reliance on this recovery is limited and rescue oxygenation strategies remain essential. Rocuronium is a non-depolarizing NMBA that produces paralysis through competitive antagonism at nicotinic acetylcholine receptors at the neuromuscular junction. By blocking acetylcholine binding, rocuronium prevents depolarization and thereby inhibits muscle contraction. Its onset is slower than succinylcholine, typically about 1 to 2 minutes, but it can still be suitable for RSI when dosed appropriately. The key difference is duration: rocuronium commonly lasts far longer—approximately 45 to 70 minutes—resulting in prolonged paralysis.[9][10] Because of this, rocuronium is often selected when succinylcholine is contraindicated or unavailable, recognizing that the longer duration commits the patient to continued ventilatory support and requires reliable post-intubation sedation and analgesia. The prolonged effect also has safety implications in failed airway scenarios, since spontaneous recovery is not imminent, and providers must be prepared for rescue airway strategies. For nursing practice, this longer duration underscores the importance of continuous sedation, close ventilator monitoring, and frequent reassessment for hemodynamic changes during the post-intubation period [9][10].

In summary, the mechanism of action of RSI medications reflects a deliberate pairing: induction agents rapidly suppress consciousness through modulation of central neurotransmission—most commonly via GABA potentiation (etomidate and propofol) or NMDA antagonism with dissociative anesthesia (ketamine)—while neuromuscular blockers interrupt neuromuscular transmission to produce flaccid paralysis and optimize intubating conditions.[5][6][7][8][9][10] The clinical significance for nursing and interprofessional teams lies in understanding how these mechanisms translate into physiologic effects and operational risks. Etomidate's relative hemodynamic neutrality and CNS protective features often support its use in hypotensive or trauma patients.[5] Ketamine's dissociative analgesia and bronchodilatory effects can be valuable in severe asthma and in patients who may benefit from sympathomimetic support.[6] Propofol's ultra-rapid onset and intracranial pressure–lowering properties may be advantageous in neurocritical presentations, while its cardiovascular depressant potential demands caution in unstable patients.[7][8] Succinylcholine provides rapid, short-lived depolarizing paralysis suited to RSI, whereas rocuronium provides non-depolarizing paralysis with longer duration, commonly reserved for cases in which succinylcholine is unsuitable.[9][10] Understanding these pharmacologic mechanisms supports safer drug selection, anticipatory monitoring, and prompt post-intubation care, all of which are essential for optimizing outcomes during emergent airway management.

Endotracheal Tubes

Endotracheal tubes (ETTs) are fundamental airway devices designed to secure a patent airway, facilitate positive-pressure ventilation, protect against aspiration, and support oxygenation and ventilation across a wide spectrum of clinical emergencies. In the context of rapid sequence intubation and other emergent airway interventions, the selection, preparation, and maintenance of an endotracheal tube are as clinically consequential as the pharmacologic components of airway management. For nursing practice, competence with ETT characteristics is essential because nurses frequently prepare airway equipment, assist during tube placement, monitor cuff pressures, evaluate ventilatory adequacy, and identify complications related to malposition or mucosal injury. Understanding the structural design and

functional rationale of ETT components strengthens procedural safety and supports timely clinical decision-making during airway stabilization and subsequent mechanical ventilation. Standard ETTs are most commonly manufactured from polyvinyl chloride (PVC), a material selected for its balance of flexibility, transparency, and cost-effective production, although alternative materials such as silicone, metal, or rubber are used in specialized clinical situations depending on intended duration, procedural demands, or anatomical constraints. Material composition influences tube rigidity, biocompatibility, and the ability to maintain lumen patency under bending or patient movement. Regardless of material, the central purpose remains consistent: to provide a conduit for airflow between the ventilator or resuscitation device and the tracheobronchial tree. Standard tubes are produced in a range of internal diameters to accommodate neonatal, pediatric, and adult airway sizes. Typical ETT internal diameters span from approximately 2.0 mm in very small pediatric patients to 12.0 mm in larger adults, with selection guided by patient age, anthropometry, airway anatomy, and the presence of co-existing pulmonary or airway disease that may alter resistance or increase the need for suctioning and bronchoscopy access. In adult practice, common size conventions support initial tube selection while acknowledging the need for flexibility. Tubes in the range of 7.5 mm to 8.5 mm internal diameter are frequently appropriate for average adult males, while 7.0 mm to 7.5 mm tubes are often preferred for females. These ranges reflect typical adult airway dimensions and balance the competing goals of ease of passage through the glottis, adequate airflow with minimal resistance, and sufficient internal caliber to permit airway suctioning and bronchoscopy when necessary. However, airway anatomy varies considerably across individuals, and emergent circumstances may limit the ability to predict the optimal size with certainty. For this reason, clinicians should prepare multiple tube sizes during airway setup, including a backup size smaller than the intended choice. From a safety perspective, this redundancy is crucial because unexpected glottic narrowing, edema, anatomic variants, or restricted mouth opening can make a selected tube size unpassable, and delays in securing the airway can quickly lead to hypoxemia or hemodynamic compromise [9][10][11].

A key design distinction in endotracheal tubes is the presence or absence of a cuff. In general, uncuffed tubes are used in newborns, reflecting neonatal airway anatomy and the clinical practice traditions that emphasize minimizing mucosal pressure injury in very small airways. In contrast, cuffed ETTs are used in most pediatric patients beyond the neonatal period and in essentially all adults, because a cuff allows clinicians to create a seal within the trachea that supports effective positive-pressure ventilation and reduces air leakage around the tube. The cuff, positioned near the distal end of the tube, is an inflatable balloon that expands outward when filled with air. When appropriately inflated, it forms a circumferential seal against the tracheal wall that improves tidal volume delivery, stabilizes ventilation parameters, and limits the escape of gas during inspiration. This seal is not merely a mechanical convenience; it has direct clinical implications for oxygenation, carbon dioxide clearance, and the ability to deliver consistent airway pressures in patients with respiratory failure. Beyond ventilation efficiency, the cuff contributes to airway protection by reducing the passage of oropharyngeal and gastric secretions into the lower respiratory tract. While cuff inflation does not eliminate aspiration risk entirely—particularly in the presence of micro-aspiration or cuff pressure variation—it provides a meaningful barrier that supports infection prevention and reduces the burden of aspiration-related complications. The cuff also helps maintain tube position by providing frictional stability within the trachea, although definitive fixation still depends on external securement devices and careful monitoring because tube migration can occur with patient movement, repositioning, or transport. To support safe cuff management, standard ETTs include a pilot balloon located at the proximal end of the tube outside the patient. The pilot balloon is connected to the cuff via a thin inflation line and includes a one-way valve through which air can be introduced or withdrawn. Clinically, the pilot balloon provides a tactile and visual indicator of cuff inflation status, but it does not reliably quantify cuff pressure; therefore, best practice is to measure cuff pressure using a manometer when available. Maintaining an appropriate cuff pressure is critical because both underinflation and overinflation carry clinically significant risks. A commonly recommended cuff pressure range is 20 to 30 cm H_2O, which is generally sufficient to achieve an adequate seal for ventilation while minimizing excessive pressure on the tracheal mucosa that could impair perfusion and contribute to ischemic injury. Excessively high cuff pressures are associated with tracheal mucosal damage, ulceration, and, in severe cases, long-term complications such as tracheal stenosis. Conversely, insufficient cuff pressure may permit air leak, impair ventilation efficacy, and increase aspiration risk, particularly during positive-pressure ventilation [11].

Another important safety feature of standard ETTs is radiopacity. Most tubes incorporate a radiopaque line running along the length of the tube, enabling clinicians to identify the tube and its distal tip on plain radiographs. Radiographic confirmation remains an important adjunct to bedside assessment, particularly after emergent intubation, during patient transport, or when clinical changes raise concern for tube migration. Correct tube position is typically

assessed using clinical markers such as chest rise, auscultation, and end-tidal carbon dioxide, but imaging can provide additional confirmation of depth and identify complications such as right mainstem intubation or significant malposition. The radiopaque marker thus supports post-procedure verification and patient safety, especially in complex or unstable cases where physical examination findings may be ambiguous. During the intubation process, standard practice commonly involves the use of an indwelling stylet inserted within the ETT prior to placement. The stylet increases tube rigidity and allows the operator to shape the distal tube curvature—often into a gentle "hockey stick" configuration—to facilitate passage through the vocal cords during laryngoscopy. However, because increased rigidity can increase the risk of mucosal trauma if the tube is advanced forcefully, careful technique and appropriate shaping are essential. Nursing staff assisting with intubation play a key role in ensuring the stylet is positioned correctly, does not protrude beyond the distal tube tip, and is removed promptly once the tube is placed and the cuff is inflated. These steps reduce trauma risk and support immediate ventilation. Route selection is another key consideration in emergent airway management. Oral intubation is generally recommended in emergency and rapid sequence contexts because it provides a more direct pathway to the glottis, permits larger tube sizes, and can be executed rapidly with standard laryngoscopy equipment. Nasal intubation is more limited in emergent scenarios and is typically considered only when oral access is compromised, such as in severe oral or mandibular trauma or when facial deformities make oral placement impractical. Even in such cases, nasal intubation carries additional risks, including epistaxis and potential complications in the setting of facial fractures, and therefore requires careful clinical judgment.[11][12]

In summary, endotracheal tubes are engineered devices whose design features directly support the physiologic goals of airway management: effective ventilation, airway protection, and stable tube positioning. Tube material, internal diameter, cuff presence, pilot balloon design, cuff pressure targets, radiopaque markings, and the use of a stylet all contribute to safe and effective intubation practice. For nurses and interprofessional teams, competence in these device characteristics enables more reliable preparation, faster troubleshooting during difficult airways, and safer ongoing management after intubation, ultimately reducing complications and improving patient outcomes in emergent and critical care settings.[11][12]

Administration

Rapid sequence intubation (RSI) is a time-critical, protocolized process that integrates pharmacology, airway equipment, physiologic optimization, and coordinated team performance to achieve rapid endotracheal intubation with minimal complications. In emergent settings, RSI is designed to establish definitive airway control quickly while reducing the risk of aspiration, hypoxemia, hemodynamic collapse, and airway trauma. Although the visible procedure is tube placement, the safety of RSI depends primarily on preparation and the disciplined execution of sequential steps that ensure correct medication selection, accurate dosing, and appropriate timing while confirming that both the patient and the equipment are ready for immediate intervention. Importantly, RSI using neuromuscular blocking agents (NMBAs) is widely regarded as the standard of care for urgent airway control when difficulty is not anticipated, and it is associated with fewer complications compared with using sedatives alone. The structured sequence of RSI is commonly summarized as the "7 Ps": preparation, pre-oxygenation, pretreatment, paralysis with induction, positioning, placement with confirmation, and post-intubation management.[13] For nursing practice and interprofessional teams, this framework is clinically useful because it provides a shared mental model that improves communication, reduces omissions, and supports consistent performance under stress [13].

Preparation

Preparation is the foundation of RSI and should begin with rapid assessment of both the patient and the anticipated airway difficulty. Even when an airway appears straightforward, the team must assume that failure is possible and that rescue strategies may be required. Preparation includes evaluating features associated with difficult mask ventilation, difficult laryngoscopy, or aspiration risk, while simultaneously ensuring the patient is physiologically optimized to tolerate apnea and induction. Establishing reliable intravenous access is essential because RSI medications must be delivered rapidly and predictably; ideally, at least one large-bore intravenous line is secured, with a second line available when feasible in high-risk patients. Continuous monitoring must be initiated before induction, including telemetry for rhythm surveillance, noninvasive blood pressure measurement at frequent intervals, and continuous pulse oximetry. Capnography equipment should also be ready because end-tidal carbon dioxide is central to confirming tracheal placement after intubation. Equipment readiness during the preparation phase is non-negotiable. Multiple endotracheal tube sizes must be prepared because anatomic variation and airway swelling can make the initial selection incorrect. Tubes should be checked for cuff integrity before use to ensure there are no leaks that could compromise ventilation after placement. Laryngoscopy equipment must be available in multiple sizes and configurations, including curved and straight blades, since the best visualization strategy can vary by patient anatomy and operator preference. All laryngoscopes should be checked for a functioning light source to avoid preventable failure at the moment of laryngoscopy.

Suction must be immediately accessible at the bedside, because secretions, blood, or emesis can rapidly obscure the view of the glottis and complicate oxygenation. A critical concept in RSI preparation is that a backup plan should be present even when no obvious difficulty is identified. Backup plans may include alternative airway devices, a second laryngoscope type, supraglottic airways, and clear escalation pathways to more advanced interventions if intubation fails. This phase also highlights the central role of nursing staff and respiratory therapists. Adequate staffing is required not only to pass equipment but also to support the cognitive load of the primary airway operator. Nurses frequently calculate and prepare medications, ensure monitoring and intravenous access, document time points, and watch for early signs of hypotension or desaturation. Respiratory therapists commonly assist with preoxygenation strategies, bag-mask ventilation when required, and immediate ventilator preparation once intubation is completed. The presence of appropriately trained team members is therefore a core safety intervention, because RSI is rarely successful as a "single-operator" event.[14]

Pre-oxygenation

Pre-oxygenation aims to maximize the patient's oxygen reserves so that they can tolerate the apneic period that occurs after induction and paralysis. During RSI, apnea is intentional: the patient's spontaneous breathing ceases, and the team relies on the oxygen reservoir created by pre-oxygenation until the airway is secured. Standard practice involves delivering the highest possible oxygen concentration at high flow for approximately 3 to 5 minutes. This approach increases alveolar oxygen stores and delays the onset of hypoxemia during apnea. For nursing and respiratory care teams, pre-oxygenation is not simply "placing oxygen" but ensuring the patient is actually receiving high-concentration oxygen effectively, which depends on mask seal, flow rate, patient cooperation, and airway patency. Maintaining upper airway patency during preoxygenation is essential. Simple maneuvers such as the chin lift or jaw thrust can improve airway openness and facilitate oxygen entry, particularly in patients with decreased level of consciousness or obstructive anatomy. Some patients, however, cannot achieve adequate oxygen saturation despite standard approaches, such as those with severe pneumonia, pulmonary edema, acute respiratory distress syndrome, or status asthmaticus. In such cases, pre-oxygenation may require non-invasive positive pressure ventilation masks to improve alveolar recruitment and support ventilation. Positive end-expiratory pressure (PEEP) valves may also be added to a bag-valve mask system to prevent alveolar collapse and improve oxygenation.[15] These strategies are particularly important in high-risk patients because desaturation during RSI can occur rapidly and may precipitate bradycardia, cardiac arrest, or neurologic injury.

Pretreatment

Pretreatment is a variable step that involves administering medications or supportive interventions to optimize physiologic conditions prior to induction and paralysis. Although not required in every case, pretreatment can be clinically valuable when it reduces predictable complications associated with airway instrumentation or induction drugs. Pretreatment may include intravenous fluid administration to support preload in patients at risk of hypotension, anxiolysis using benzodiazepines when agitation compromises preoxygenation, or analgesia when pain and sympathetic surges are anticipated. A commonly used approach involves administering a short-acting opioid, such as intravenous fentanyl, to blunt the sympathetic response to laryngoscopy and intubation. This can be particularly relevant in patients in whom surges in heart rate and blood pressure may be harmful, such as those with intracranial pathology or significant cardiovascular disease. Pretreatment may also be tailored to pulmonary physiology. In patients with reactive airway disease, a short-acting beta-agonist such as albuterol may be administered to reduce bronchospasm and minimize airway resistance during and after intubation. This intervention aims to improve ventilation and reduce the risk of severe airflow obstruction after induction. In selected shock states, pretreatment with vasopressor or alpha-adrenergic support may be considered to mitigate the anticipated reduction in mean arterial pressure that can occur when sympathetic tone is lost after induction, especially with agents that cause vasodilation or myocardial depression. While these decisions are individualized and context-dependent, they reflect a central principle: pretreatment is intended to stabilize physiologic vulnerability before the high-risk transition into apnea and mechanical ventilation [14].

Paralysis and induction

Paralysis with induction is the defining step of RSI and involves administering the chosen induction agent and neuromuscular blocker in immediate sequence to produce rapid unconsciousness and flaccid paralysis. The selection of drugs should have been made during preparation based on clinical status, allergies, contraindications, and the anticipated need for hemodynamic stability or bronchodilation. A key principle is that RSI medications are given as intravenous boluses rather than titrated gradually. The rationale is to achieve a predictable onset and depth of effect within seconds, minimizing prolonged airway manipulation while the patient transitions into apnea. Induction agents are typically administered first, followed immediately by the paralytic agent. Etomidate is often dosed intravenously at 0.15 mg/kg to 0.3 mg/kg, with the dose adjusted based on clinical judgment and patient factors. Ketamine is commonly administered at 2 mg/kg intravenously. Propofol may

be given in a range of 0.5 mg/kg to 2 mg/kg intravenously, with dose selection heavily influenced by hemodynamic stability because propofol can cause significant hypotension. Immediately after the induction agent, the paralytic is administered. Succinylcholine is typically given as 1.5 mg/kg, while rocuronium is administered at approximately 1 mg/kg. These dosing strategies reflect the need for rapid onset and adequate paralysis to maximize first-pass success. From the nursing perspective, this phase is especially safety-sensitive because medication dosing must be accurate, syringes must be clearly labeled, and administration must occur in the correct sequence. Nurses also monitor for rapid changes in blood pressure, oxygen saturation, and heart rhythm that can occur immediately after induction. Because neuromuscular blockade eliminates visible signs of distress, monitoring becomes the primary method for detecting instability. A parallel priority is planning for post-intubation sedation, because many induction agents have short durations, and a paralyzed patient must not be allowed to regain awareness without analgesia and sedation [13][14].

Positioning

Positioning and airway protection occur during the brief interval after paralysis and induction and before tube placement. At this stage, the patient is apneic and unable to protect the airway, so the risk of aspiration becomes clinically significant. Positioning also influences the ease of laryngoscopy and the probability of first-pass success. While the original "7 Ps" framework lists positioning as one of the steps, it is best understood as a continuous optimization of airway alignment and patient safety immediately before and during laryngoscopy. During this phase, bag-mask ventilation should be minimized when possible to reduce gastric insufflation and aspiration risk, assuming that pre-oxygenation has created sufficient oxygen reserves. However, patient safety remains the priority; if oxygen saturation is falling, controlled bag-mask ventilation may be necessary. Some clinicians may perform the Sellick maneuver—applying pressure over the cricoid cartilage to occlude the esophagus—although its routine use is debated and is typically reserved for specific scenarios where aspiration risk is considered high. Regardless of whether cricoid pressure is used, the patient must be monitored closely for desaturation and bradycardia, and the team must be prepared to pause and re-oxygenate if necessary [13][14].

Placement and confirmation

Placement should occur once adequate sedation and paralysis have been achieved. Direct laryngoscopy is performed, and once the glottis is visualized clearly, an appropriately sized ETT—with a stylet in place—is advanced through the vocal cords under direct visualization. After the tube passes the cords, the cuff is inflated, commonly with approximately 10 mL of air depending on tube size and cuff characteristics, and the stylet is removed. However, placement is only the beginning; confirmation is essential because esophageal intubation is a life-threatening complication that must be recognized immediately. The most reliable bedside confirmation method is end-tidal carbon dioxide detection, using quantitative waveform capnography when available or colorimetric detectors when not. Auscultation remains an important adjunct: clinicians should listen over both lung fields to confirm bilateral breath sounds and over the epigastrium to ensure that air entry is not occurring in the stomach. Unequal breath sounds may suggest right mainstem bronchus intubation, whereas absent breath sounds with gastric insufflation signals esophageal placement. After immediate clinical confirmation, a chest radiograph is used to verify depth and position. On imaging, the tip of the ETT is typically expected to be approximately 2 to 5 cm above the carina, balancing the risk of endobronchial intubation if too deep and accidental extubation if too shallow. These confirmation steps are central to nursing responsibilities as well, because nurses frequently monitor capnography, document confirmation findings, and observe for changes that may indicate tube migration during transport or repositioning [14].

Post-intubation management

Post-intubation management begins immediately after airway confirmation and includes securing the ETT, connecting the patient to a mechanical ventilator, initiating ongoing sedation and analgesia, and monitoring for complications. Tube securement is a critical safety step because even a correctly placed tube can become dislodged during movement, transport, or patient agitation. Once connected to the ventilator, ventilator settings must be selected to match the patient's physiology and underlying disease process, and continuous monitoring must confirm that oxygenation and ventilation goals are achieved. A key post-intubation principle is that induction agents used during RSI often have short half-lives, and neuromuscular blockade—especially when rocuronium is used—may persist for a prolonged period. Therefore, clinicians must initiate appropriate sedation and analgesia promptly to prevent awareness and distress. This typically involves continuous infusions or scheduled dosing strategies that can be titrated to clinical goals, ensuring patient comfort, ventilator synchrony, and hemodynamic stability. Nurses are central in this phase, as they assess sedation depth, manage infusion titration based on protocols, monitor blood pressure and respiratory parameters, and identify early complications such as hypotension, arrhythmias, bronchospasm, ventilator dyssynchrony, aspiration, or barotrauma. Post-intubation management also includes ongoing evaluation for secondary complications related to the procedure itself, such as dental trauma, airway bleeding, laryngeal edema, or tube obstruction from secretions. Regular reassessment of cuff pressure is important to maintain

the recommended range, minimize tracheal injury, and prevent air leaks that impair ventilation. Documentation of tube depth at the teeth or gums and periodic verification during care activities supports early detection of migration. Finally, the team must consider the broader clinical trajectory: intubation is a stabilization intervention, not definitive treatment for the underlying cause of respiratory failure or airway compromise. Accordingly, post-intubation care includes diagnostic evaluation, initiation of disease-specific therapy, and reassessment of goals of care when appropriate. In sum, RSI administration is best conceptualized as a structured sequence designed to reduce preventable harm while securing the airway. The "7 Ps" framework provides an operational checklist that supports safe medication selection and delivery, optimizes oxygen reserves, reduces physiologic stress, and ensures reliable confirmation and post-intubation stabilization.[13] When these steps are executed with disciplined teamwork—supported by nursing vigilance, respiratory therapy expertise, and a clear backup plan—RSI can be performed with improved first-pass success and reduced complications compared with sedatives alone, thereby strengthening patient outcomes in emergent airway management.[14][15]

Adverse Effects

Rapid sequence intubation (RSI) is designed to secure the trachea rapidly while preserving oxygenation and hemodynamic stability, thereby reducing the time a critically ill patient spends in an unprotected airway state.[16] Despite its clear clinical benefits, RSI remains a high-risk intervention because it intentionally transitions a patient from spontaneous breathing to apnea while simultaneously exposing them to potent sedative and neuromuscular blocking medications and the mechanical risks of airway instrumentation. As a result, complications may arise from three broad sources: the patient's underlying condition and physiologic vulnerability, adverse effects of the medications used, and technical or procedural problems related to endotracheal tube placement and confirmation.[17] For nurses and interprofessional teams, adverse effect awareness is clinically significant because early recognition and rapid mitigation often determine whether a complication remains reversible or becomes catastrophic. The goal is therefore not only to understand what complications can occur, but also to anticipate them, implement prevention strategies during the "7 Ps," and respond effectively when warning signs emerge. A fundamental procedural risk begins during tube selection. Choosing an inappropriate endotracheal tube size may directly injure airway structures. If an endotracheal tube is too large, it may cause trauma to the vocal cords during passage, promote mucosal abrasion, and increase the risk of post-intubation laryngeal edema. Such injury can manifest as difficult tube advancement, bleeding, or later complications such as hoarseness and airway swelling. In severe cases, edema can impair ventilation after extubation or contribute to stridor and the need for re-intubation. Because airway anatomy can be unpredictable, the preparation phase should include multiple tube sizes and a willingness to downsize promptly rather than repeatedly forcing an oversized tube. From a nursing perspective, assisting in equipment readiness and supporting timely decision-making—such as handing the backup tube without delay—helps prevent repeated traumatic attempts [17].

Aspiration of gastric contents is another major complication and is a risk for virtually all patients undergoing RSI because airway protective reflexes are rapidly abolished by induction agents and paralysis. Fasting reduces aspiration risk, and fasting is encouraged when there is clinical suspicion that intubation is likely; however, emergency care often does not allow sufficient time for gastric emptying. Aspiration can occur before intubation, during laryngoscopy, or after tube placement if cuff inflation is delayed or inadequate. The clinical consequences range from mild chemical pneumonitis to severe aspiration pneumonia and acute respiratory distress syndrome. Clinically, aspiration may be suspected when there is visible emesis or gastric material in the oropharynx, sudden oxygenation worsening during or after the procedure, or the appearance of a new infiltrate on post-intubation chest radiography.[18] Suction readiness is therefore a critical preventive strategy, and nursing teams are often central in ensuring suction is functional and used promptly when emesis occurs. Cricoid pressure, sometimes applied as an aspiration-reduction maneuver, has potential disadvantages in emergent practice. While it is intended to reduce gastric insufflation and regurgitation, it may impair laryngoscopic visualization and thus delay intubation, paradoxically increasing hypoxemia risk. In addition, if active vomiting occurs, cricoid pressure has been associated with concerns for esophageal injury, including theoretical risk of rupture. Because RSI safety is driven by first-pass success and swift confirmation, any maneuver that worsens the view or delays placement must be reassessed in real time. For interprofessional teams, this underscores that aspiration prevention must be balanced against the immediate need to secure the airway efficiently, with decisions guided by the patient's condition and the operator's view [17][18].

Among the most dangerous procedural complications is esophageal intubation. Placement of the tube into the esophagus prevents effective ventilation, leading quickly to severe hypoxemia and potentially cardiac arrest and death. This risk is magnified in urgent settings with poor visualization, secretions, blood, or anatomic difficulty. The critical safety response is strict adherence to confirmation

protocols. End-tidal carbon dioxide detection, using colorimetric devices or preferably waveform capnography, is strongly recommended to confirm tracheal placement, as it provides rapid objective evidence that ventilation is occurring in the lungs rather than the stomach. Failure to promptly confirm placement, or reliance solely on auscultation in noisy environments, can lead to delayed recognition of esophageal intubation and catastrophic deterioration. Nursing vigilance is essential here: nurses often monitor the capnography signal, document confirmation, and alert the team immediately if end-tidal carbon dioxide is absent or inconsistent with effective ventilation. Pneumothorax is a less common but potentially severe complication associated with endotracheal intubation and subsequent ventilation. It may occur due to barotrauma in vulnerable lungs, particularly when high airway pressures are required, and it has been associated with situations such as mainstem bronchus intubation or severe reactive airway disease where dynamic hyperinflation can occur.[18] A pneumothorax can cause sudden hypoxemia, hypotension, and increased ventilator pressures, and in its tension form, it can rapidly become life-threatening. Post-intubation chest radiography is therefore clinically important not only to confirm depth but also to evaluate for pneumothorax.[18] Because radiography is not instantaneous, teams must also rely on bedside cues—such as unilateral breath sounds, abrupt oxygen desaturation, tachycardia followed by hypotension, or a sudden rise in peak inspiratory pressures—to identify a possible pneumothorax early. Nurses frequently detect these changes first due to continuous monitoring responsibilities and should be empowered to activate emergent evaluation pathways when these signs occur [18].

In addition to procedural complications, medication-related adverse effects are a major contributor to RSI risk. Adequate sedation is a cornerstone of safe RSI because neuromuscular blockade alone does not produce unconsciousness or analgesia. When sedation is inadequate, patients may experience awareness during paralysis, a profound and ethically unacceptable outcome. Conversely, excessive sedation or inappropriate drug selection can precipitate hemodynamic collapse, prolonged unconsciousness, or neurologic compromise. Therefore, the adverse effects of each sedative agent must be weighed against its anticipated benefits and the patient's physiologic reserve. Etomidate is commonly favored for induction because it is typically associated with relative hemodynamic stability, yet it is not free of adverse effects. Potential complications include myoclonus and nausea or vomiting. A clinically important consideration is adrenal suppression. A single induction dose of etomidate may cause transient adrenocortical suppression through reversible inhibition of 11-β-hydroxylase, an enzyme involved in cortisol synthesis. When suppression occurs, it is generally reversible and typically does not persist beyond approximately 24 hours, and cortisol levels are usually maintained within physiologic ranges.[19] While this effect is often clinically limited after a single RSI dose, it remains a consideration in critically ill patients where adrenal reserve may already be strained. Etomidate has also been noted to potentially lower seizure threshold in patients with known seizure disorders.[19] For nursing teams, the implication is to remain attentive to post-intubation hemodynamics, signs of endocrine instability in critically ill patients, and neurologic status trends, particularly when seizure risk exists. Ketamine has a distinct adverse effect profile related to its dissociative mechanism. One well-recognized phenomenon is the emergence reaction, characterized by disturbing dreams, hallucinations, and emotional distress as sedation wears off. This can be unsettling for patients during recovery phases and may complicate post-intubation sedation transitions. Concurrent use of benzodiazepines can reduce the likelihood or severity of this phenomenon by providing anxiolysis and smoothing emergence. Ketamine also influences cerebral physiology by increasing cerebral blood flow, and therefore it should be avoided in patients with elevated intracranial pressure where increased cerebral blood volume may worsen intracranial dynamics.[6] In practice, this means ketamine selection should be individualized: it can be particularly valuable in severe bronchospasm due to bronchodilatory effects, yet it may be undesirable in scenarios where intracranial pressure is a primary concern [18][19].

Propofol provides rapid induction and is commonly used for both intubation and ongoing ventilator sedation, but its adverse effects can be clinically significant. Propofol is contraindicated in patients with soybean or egg allergies because some formulations may contain components derived from these products, raising concern for allergic reaction. Hemodynamically, propofol decreases sympathetic activity and produces peripheral vasodilation along with myocardial depression, which can cause hypotension. In many cases, this hypotension can be treated by restoring intravascular volume with intravenous fluid boluses and, when necessary, using vasopressor or inotropic support transiently to maintain perfusion. However, in patients with limited cardiovascular reserve or profound shock, propofol-associated hypotension may be severe and can precipitate end-organ hypoperfusion. Propofol may also worsen an existing neurologic injury by reducing cerebral perfusion pressure, particularly if systemic hypotension occurs.[7] For nursing care, this underscores the importance of close blood pressure monitoring, readiness to administer fluids or vasopressors per protocol, and careful titration of any ongoing propofol infusion after intubation. Overall, adverse effects in RSI must be conceptualized as a predictable risk landscape rather than rare mishaps. Procedural complications such as aspiration,

esophageal intubation, pneumothorax, and airway trauma can occur even with skilled operators, particularly when patients are physiologically unstable or airways are contaminated by blood or secretions.[16][17][18] Medication-related complications, including etomidate-associated myoclonus and transient adrenal suppression, ketamine emergence phenomena and intracranial concerns, and propofol-associated allergy risk and hypotension, require careful selection and vigilant monitoring.[6][7][19] The practical safety implication for nursing and interprofessional teams is that RSI success is defined not only by tube placement but also by preventing deterioration during the peri-intubation window, confirming placement objectively with end-tidal carbon dioxide detection, and initiating immediate post-intubation stabilization strategies that address oxygenation, hemodynamics, and continuous sedation.[16][17]

Contraindications

Contraindications to rapid sequence intubation (RSI) medications are clinically significant because RSI is frequently performed under extreme time pressure, in physiologically unstable patients, and with limited opportunity to "test dose" or titrate agents slowly. In this setting, drug selection must be deliberate and individualized, balancing the urgency of airway control against predictable adverse physiologic effects. While there are few absolute contraindications to securing an airway itself when a patient is in imminent respiratory failure, there are important contraindications and cautions that apply to specific induction agents and neuromuscular blockers. For nursing and interprofessional teams, understanding these contraindications is essential because nurses frequently prepare and administer medications, monitor for early signs of toxicity, and provide critical safety checks—such as verifying allergy history or reviewing conditions that increase the risk of catastrophic complications like malignant hyperthermia or life-threatening hyperkalemia. Contraindications therefore function as risk filters that shape the safest possible medication plan in high-risk, high-acuity situations [17][18][19].

Etomidate

Etomidate is often selected for RSI because it is commonly associated with relative hemodynamic stability; however, its endocrine and neurologic effects can make it inappropriate or risky in certain populations. One concern is that etomidate may lower the seizure threshold in patients with a known seizure disorder.[19] Although etomidate is not universally contraindicated in all patients with seizures, this effect warrants caution in individuals with poorly controlled epilepsy or those in whom seizure provocation could worsen outcomes, particularly in settings where seizure activity could compromise ventilation, intracranial physiology, or hemodynamic stability. Etomidate can also cause nausea and vomiting, which has practical relevance to airway management because emesis in the peri-intubation window increases aspiration risk—especially after loss of protective reflexes. A more specific contraindication-related issue is etomidate's effect on adrenal cortisol synthesis. Etomidate can inhibit adrenal cortisol production in a dose-dependent manner, with suppression reported for up to approximately 12 hours.[19] Mechanistically, this relates to reversible inhibition of 11-β-hydroxylase, a key enzyme in the cortisol synthesis pathway. In many patients receiving a single induction dose, the clinical consequences may be limited, but in patients who already have impaired adrenal reserve, the additional suppression could be clinically meaningful. Therefore, clinicians should exercise caution in patients with known adrenal insufficiency, including those with primary adrenal failure or chronic steroid dependence.[19] In critically ill or septic patients, where endogenous cortisol response may contribute to hemodynamic stability, the decision to use etomidate is often weighed carefully against alternatives. In practice, this does not mean etomidate is never used in critically ill patients; rather, it highlights that adrenal insufficiency and seizure disorders represent situations where etomidate selection should be more conservative and where post-intubation monitoring for hemodynamic instability may need to be intensified.[19]

Ketamine

Ketamine has a unique role in RSI because it provides dissociative anesthesia with analgesia and tends to preserve airway reflexes more than other sedatives; however, its sympathomimetic and cerebral physiologic effects create important contraindications. Ketamine's sympathomimetic properties can increase cardiac output, heart rate, and blood pressure by stimulating catecholamine release and reducing reuptake.[6] In patients with limited coronary reserve or established coronary artery disease, this increase in myocardial oxygen demand may precipitate ischemia, particularly during a physiologically stressful event such as intubation. Consequently, ketamine is contraindicated in patients with known coronary artery disease when the risk of ischemia is clinically significant.[6] Similarly, ketamine may be contraindicated in patients with uncontrolled hypertension because its pressor effects can worsen hypertensive crises and potentially contribute to end-organ injury.[6] Ketamine's effects on cerebral and ocular physiology are also central to its contraindications. As a cerebral vasodilator, ketamine can increase cerebral blood flow, which may raise intracranial pressure in susceptible patients.[6] For this reason, ketamine is contraindicated in patients with closed head injury or conditions where intracranial pressure is already elevated and further increases could worsen neurologic injury.[6] In parallel, ketamine may increase intraocular pressure, making it contraindicated in patients with raised intraocular

pressures, such as those with certain ocular injuries or glaucoma-related risks.[6] These contraindications underscore an important safety principle: ketamine is often beneficial in bronchospasm and hypotension-prone patients, but it may be harmful in patients with cardiovascular ischemia risk, significant hypertension, or intracranial and ocular pressure vulnerability.

Propofol

Propofol is widely used for induction and post-intubation sedation because of its rapid onset and short duration, but its hemodynamic effects create strong cautions and, in some scenarios, functional contraindications. Propofol commonly causes hypotension by reducing systemic vascular resistance through vasodilation and by producing myocardial depression.[7] In hemodynamically stable patients, this effect can often be managed with intravenous fluid boluses and, if needed, transient vasopressor support. However, in patients with pre-existing hypotension, shock, or limited cardiovascular reserve, propofol-induced reductions in blood pressure can be profound and may precipitate end-organ hypoperfusion. Therefore, propofol should be used cautiously or avoided in patients who are already hypotensive or at high risk for peri-intubation cardiovascular collapse.[7] This consideration is especially relevant in trauma, septic shock, and massive hemorrhage, where maintenance of perfusion pressure during induction is critical. Propofol also carries a risk of hypersensitivity reactions in patients with allergies to eggs or soy products, as some formulations contain components derived from these substances.[7] In emergency airway management, allergy history can be incomplete, and nursing assessment becomes important in identifying documented allergies and clarifying reaction types when time permits. While not every egg or soy allergy predicts severe reaction to propofol, the presence of a known hypersensitivity history should prompt careful risk assessment and consideration of alternative agents. Consequently, propofol's main contraindication themes are hemodynamic vulnerability and potential allergic sensitivity, both of which can have immediate and severe consequences during the RSI window.[7]

Succinylcholine

Neuromuscular blocking agents must also be assessed for contraindications because paralysis can amplify or precipitate life-threatening physiologic derangements. Succinylcholine is a depolarizing neuromuscular blocker valued for its rapid onset and short duration, but it has several important absolute contraindications. A personal or family history of malignant hyperthermia is an absolute contraindication to succinylcholine use because exposure can trigger a catastrophic hypermetabolic reaction with rapid progression to hyperthermia, acidosis, muscle rigidity, rhabdomyolysis, and cardiovascular collapse.[9] In emergency settings, family history may be unknown, but any documented prior malignant hyperthermia episode or strong suspicion should lead to avoidance of succinylcholine. Conditions predisposing to life-threatening hyperkalemia are also absolute contraindications to succinylcholine.[9] Succinylcholine can raise serum potassium through depolarization-related potassium efflux from skeletal muscle. In most healthy individuals, this increase is modest, but in patients with upregulated extrajunctional acetylcholine receptors—such as those with certain neuromuscular disorders, severe burns, prolonged immobilization, or other high-risk conditions—the potassium rise can be large enough to provoke fatal arrhythmias. Because the consequence can be immediate cardiac arrest, these hyperkalemia-prone conditions represent critical exclusion criteria when selecting succinylcholine for RSI.[9]

Clinical implications for nursing and interprofessional teams

In summary, medication contraindications in RSI require rapid but systematic evaluation. Etomidate warrants caution in patients with seizure disorders and adrenal insufficiency because of seizure threshold effects and dose-dependent adrenal cortisol suppression.[19] Ketamine is contraindicated in patients with closed intracranial trauma or elevated intracranial and intraocular pressures and in those with coronary artery disease or significant hypertension due to sympathomimetic and vasodilatory effects.[6] Propofol should be used cautiously or avoided in patients with pre-existing hypotension because of vasodilatory and myocardial depressant effects and requires attention to potential hypersensitivity in those with egg or soy allergies.[7] Succinylcholine is absolutely contraindicated in individuals with personal or family history of malignant hyperthermia and in patients with conditions that predispose to life-threatening hyperkalemia.[9] These considerations emphasize that safe RSI is not solely a technical airway procedure; it is a pharmacologically complex intervention that depends on precise drug selection, rapid safety screening, and vigilant monitoring—domains where nursing and interprofessional team performance directly contributes to preventing avoidable harm.

Enhancing Healthcare Team Outcomes

Optimizing outcomes during endotracheal intubation depends on disciplined interprofessional collaboration, because airway management is not solely a procedural task but a high-risk clinical transition that integrates pharmacologic induction, neuromuscular blockade, physiologic monitoring, and post-intubation stabilization. Although endotracheal intubation is typically performed by airway-trained clinicians such as nurse anesthetists, anesthesiologists, emergency physicians, intensivists, and other credentialed providers depending on the clinical setting, safe execution requires that every member of the healthcare team understands the purpose, timing, and potential adverse effects of the medications used. Induction agents and neuromuscular blockers produce

profound and immediate changes in consciousness, respiratory drive, airway reflexes, and hemodynamics. If team members do not share a common understanding of expected drug effects, early warning signs of complications may be missed, resulting in preventable hypoxemia, hypotension, arrhythmias, or unrecognized awareness in paralyzed patients. Therefore, medication literacy across disciplines—nursing, respiratory therapy, pharmacy, and physician providers—functions as a core patient safety intervention. A second determinant of success is structured preparedness before the first medication is administered. Resuscitative equipment must be immediately available in the room, including suction, oxygen delivery devices, bag-valve-mask capability, advanced airway adjuncts, and emergency medications and defibrillation resources if deterioration occurs. RSI and emergency intubation frequently occur in unstable patients whose physiologic reserve is limited; even when airway placement is technically successful, peri-intubation decompensation can still occur due to loss of sympathetic tone, apnea-induced hypoxemia, or underlying shock. Equipment readiness is therefore not optional redundancy but an essential safeguard that enables rapid rescue ventilation, treatment of hypotension, or correction of life-threatening dysrhythmias without delay. During emergency intubation, continuous monitoring is indispensable, and assigning a dedicated nurse to monitor vital signs strengthens team performance and reduces cognitive overload for the airway operator. This nurse's role extends beyond passive observation; it includes anticipating trends, communicating changes in oxygen saturation or blood pressure in real time, confirming medication administration times, and helping coordinate immediate interventions such as fluid boluses, vasopressor initiation per protocol, or re-oxygenation if desaturation occurs. Respiratory therapists contribute critical expertise in preoxygenation, ventilation support, capnography use, and ventilator setup, while pharmacists can assist with dosing verification, contraindication screening, and rapid preparation of post-intubation sedation and analgesia plans. Clear role delineation, closed-loop communication, and shared situational awareness allow the team to execute intubation efficiently while maintaining patient safety throughout the peri-intubation window. Ultimately, improved outcomes are achieved when the interprofessional team functions as a cohesive unit: preparing equipment and medications proactively, monitoring continuously, anticipating complications, and communicating openly. With vigilant teamwork and appropriate pharmacologic selection, endotracheal intubation can be performed with fewer adverse events, faster stabilization, and more reliable transition to definitive respiratory support, thereby improving the likelihood of favorable patient outcomes [19].

Conclusion:

Rapid sequence intubation represents a high-stakes clinical procedure where seconds determine outcomes. While the technical act of placing an endotracheal tube is often emphasized, the true safety margin lies in preparation and teamwork. Nurses are central to this process, bridging pharmacology and physiology with vigilant monitoring and precise execution. Their role extends beyond medication administration to anticipating complications, ensuring equipment readiness, and maintaining situational awareness during the peri-intubation window. Drug selection is not uniform; it must be tailored to patient-specific factors such as hemodynamic stability, intracranial pressure, and airway reactivity. Understanding the mechanisms and contraindications of agents like etomidate, ketamine, propofol, succinylcholine, and rocuronium is essential for preventing catastrophic outcomes such as cardiovascular collapse or awareness under paralysis. Post-intubation care is equally critical, requiring prompt initiation of sedation and analgesia, continuous reassessment of ventilatory parameters, and proactive management of complications like aspiration or pneumothorax. Ultimately, RSI should be viewed as a structured, team-driven intervention rather than a single operator skill. When executed with disciplined adherence to evidence-based protocols and interprofessional collaboration, RSI can transform a life-threatening airway crisis into a controlled, stable transition to definitive respiratory support.

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