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REVIEW

Endotracheal Intubation in the Pharmaceutical-Poisoned Patient: a Narrative Review of the Literature

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Abstract

Introduction Endotracheal intubation (ETI) is an essential component of the supportive care provided to the critically ill patient with pharmaceutical poisoning; however, specific nuances surrounding intubation including techniques and complications in the context of pharmaceutical poisoning have not been well elucidated.

Discussion A search of the available literature on ETI in pharmaceutical-poisoned patients was undertaken using Medline, ERIC, Cochrane database, and PsycINFO using the following MeSH and keyword terms: ("toxicology" OR "poisons" OR "drug overdose" OR "poisoning") AND ("intubation, intratracheal" OR "intubation, endotracheal" OR "airway management" OR "respiration, artificial"). A hand-search was also performed when the literature in the above search required additional conceptual clarification, including using the "Similar Articles" feature of PubMed, along with reviewing articles' reference lists that discussed intubation in the context of a poisoning scenario. Articles with any discussion around the ETI process in the context of a pharmaceutical poisoning were then included. Intubation may be performed in patients poisoned with pharmaceuticals in the context of both single and multiple organ dysfunction including central and peripheral nervous system, pulmonary, or cardiovascular toxicity with hemodynamic instability, or localized effects resulting in mechanical airway obstruction. Certain classes of poisonings may require modifications to the standard rapid sequence induction airway management algorithm.

Conclusions ETI is a key component of the supportive care provided to the patient poisoned by a pharmaceutical agent. Clinicians should be aware of the spectrum of toxicities that can necessitate intubation, as well as airway management nuances that are specific to various poisoning presentations.

Keywords Endotracheal intubation · Airway management · Pharmaceutical poisoning · Supportive care

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Introduction

Endotracheal intubation (ETI) is an essential component of the supportive care provided to the critically ill patient with pharmaceutical overdose [1]. Every year, > 20,000 patients are intubated after overdose in the USA [2]. Approximately 16% of all adult intentional overdoses are intubated, most commonly after ingestions of atypical antipsychotics, benzodiazepines, antidepressants, and opioids [3]. Ten to twelve percent of pediatric overdose patients in the emergency department and ICU setting are intubated, most commonly after ethanol, clonidine, and acetaminophen ingestions [4, 5]. Despite these high rates of intubation after overdose, one analysis of overdose deaths suggested that 59% of cases had inadequate airway management, which suggests that there may be a gap in specialized knowledge concerning intubation after overdose [6].

Poisoned patients may present with altered mental status necessitating ETI due to loss of airway reflexes, or because the predicted clinical course anticipates imminent inability to protect airway and the risk of aspiration [7–11]. Patients may also require airway protection for management of overdose-related seizure, severe agitation, or delirium [12–14]. Toxic exposures may result in respiratory insufficiency due to direct lung or orotracheal injury, or decreased respiratory drive [15–17]. In our experience, failure of oxygenation can occur with poisoning-induced atelectasis, acute lung injury, and pulmonary edema. Failure of ventilation can occur with upper airway obstruction or hypoventilation due to CNS depression, respiratory muscle spasm, or paralysis. Acute lung injury may occur due to aspiration of gastric contents; hemodynamic instability; multi-organ dysfunction; and certain poisonings such as opioids, barbiturates, and salicylates.

Finally, we note that patients presenting with toxicological exposures requiring advanced or specialized therapies (e.g., pediatric subspecialists, hemodialysis, organ transplantation, ECMO) may be intubated for airway protection due to anticipated airway or CNS deterioration during transport to a facility capable of providing such care.

The purposes of this narrative review are to characterize airway management, particularly via ETI in the pharmaceutical-poisoned patient, and to identify issues in decision-making, management, and complications associated with airway management in this population.

Materials and Methods

Data Sources and Searches

We searched Medline, ERIC, Cochrane database, and PsycINFO using the following MeSH and keyword terms: ("toxicology" OR "poisons" OR "drug overdose" OR "poisoning") AND ("intubation, intratracheal" OR "intubation, endotracheal" OR "airway management" OR "respiration, artificial"). All available years were included. The search strategy was limited to humans and English language studies. Publications were included if there was a specific discussion of ETI for the management of poisoning by a pharmaceutical agent, and they were excluded if there was no specific discussion of ETI for a particular poisoning; if the poisoning was due to a non-pharmaceutical agent; or if airway management was discussed but did not address a poisoning. We also supplemented our strategy by hand-searching the references of included studies or when additional conceptual clarifications were required after review of the above literature. The authors also used the "Similar Articles" feature of PubMed to search for additional articles of interest. Hand-searched articles performed for clarification of concepts were included by the authors where we felt the published content facilitated improved understanding of the topic.

Study Selection

We included a selection of relevant studies and review articles regardless of design. Pertinent articles regarding airway management for pharmacologically poisoned patients were included, and non-pertinent (obscure/uncommon xenobiotics, etc.) articles were omitted for brevity. This narrative review combines expert opinion with a referenced description of the available literature and proposes potential best practices based on the available data or experiences described in published articles. Clinical circumstances, expert consultation, and provider experience should inform patient care in individualized patient encounters.

Results and Discussion

We found 567 articles that met inclusion criteria. Four hundred sixty-seven were excluded because there was no specific discussion of ETI for a particular poisoning; the poisoning was due to a non-pharmaceutical agent; or because airway management was discussed but did not address a poisoning, leaving 100 pertinent articles. Three additional articles were included following editorial review. The authors found a dearth of specific information around the management of poisoned patients with ETI. Most articles mentioned the process of ETI in the context of a particular pharmaceutical poisoning but did not clearly discuss the nuances of this intervention. Thus, in this narrative review article, the authors describe pertinent literature in combination with expert opinion and extrapolation from experience.

Preparation for Intubation and the Decision to Intubate

Conditions that increase the challenge of ETI and complicate the decision to definitively manage the airway in poisoned patients include both pre-existing and poisoning-induced physical and anatomic abnormalities of the airway; emesis; altered mental status; presence/absence of gag reflex; physiologic and metabolic derangement; failure of oxygenation or ventilation; and lack of response to antidotal therapy such as naloxone or flumazenil [18–20]. The decision to intubate should be made deliberately and with care since the procedure of intubation, the subsequent change in cardiopulmonary physiology as a result of positive pressure ventilation, and the medications used to facilitate airway management and procedural sedation may increase morbidity by exacerbating hypoxia, hypotension, oropharyngeal damage, and acidosis [18]. While potentially lifesaving, intubation is not without

risks, as poisoned patients who develop hypoxemia and hypercapnia during ETI have an increased incidence of serious post-intubation complications during their admission [21]. Early complications may include laryngeal injury, glottic edema, right mainstem intubation, vomiting, aspiration, and hypotension [21]. Delayed complications can include vocal cord paralysis, laryngeal scarring, adhesions, and subglottic granuloma formation [21]. Diligent planning and foresight can mitigate the worsening of these conditions including consideration of the underlying physiologic state of the poisoned patient, optimization of oxygenation and ventilation, and careful attention to drug selection [18].

Poisoned patients may be at high risk of several specific peri-intubation complications that may require additional preparation. Overdose patients have an increased risk of vomiting and laryngeal injury and should be intubated in a location with rapid access to adequate and functioning suction to clear the airway [22]. Overdose patients may develop peri-intubation hypotension due to the ingested medications, acidosis, volume depletion, and decreased cardiac preload as a result of positive pressure ventilation. In our opinion, pre-intubation fluid resuscitation prior to ETI should be considered. Specific overdoses (e.g., salicylates, tricyclic antidepressants) may require manipulation of pH due to sensitivity to acid-base derangements particularly during intubation, and use of IV sodium bicarbonate in the preparatory stage of intubation is recommended [23].

Historical and Examination Characteristics Which Influence Airway Management

Patients with pharmacologic toxicity primarily require airway management for two indications that can exist independently or concomitantly: respiratory failure, defined as failure to ventilate, oxygenate, or both; and need for airway protection, defined as risk of aspiration, inability to protect airway, airway damage, and CNS depression. Thus, assessment of the entire clinical scenario is ideal (both current and anticipated course), as reliance on a single indicator of respiratory insufficiency, such as pulse oximetry, may result in delay to management and increased risk of respiratory failure and aspiration [24–27].

Respiratory Failure

Among patients with altered respiratory status due to poisoning, waveform capnography may be used to monitor for developing respiratory failure and is essential if supplemental oxygen is utilized, due to risk of unrecognized hypoventilation or apnea [28]. In our opinion, monitoring of trends in blood gas analysis and waveform capnography, in addition to physical examination findings such as bradypnea, hypopnea, and respiratory fatigue, can support the decision to intubate.

Airway Protection

Poisoning is the most common cause of non-trauma-induced coma in patients under the age of 35 years [25]. Presence of gag and cough reflexes has been used to inform the need for intubation in patients with altered mental status but is not an adequate predictor of aspiration risk. The presence of the gag reflex varies, even among patients with normal mental status, and testing gag or cough reflexes may actually increase the risk of vomiting and aspiration [29–35]. Therefore, in our opinion, the practice of attempting to elicit a gag reflex in a poisoned patient should be abandoned.

In many clinical scenarios, an assessment of cerebral function and the predicted clinical course may guide the decision to intubate. While Glasgow Coma Scale (GCS) is commonly used in head injury patients, the use of the GCS in poisoned patients has not been found to reliably predict the need for intubation, given the difference in the underlying pathophysiology of altered mental status [8, 20, 25, 36, 37]. There is conflicting data and no clear consensus on this topic. One 12-month prospective study of overdose patients supports intubation with the GCS of 8 or less (sensitivity of 90% and a specificity of 95% for predicting need for ETI), while another study found that no patient with a GCS of 8 or less had an aspiration event or required ETI, suggesting that carefully selected patients may be candidates for close observation and monitoring [7, 37]. In our opinion, physical assessment and clinical gestalt, clinical trends, and expected clinical course, rather than over-reliance on calculated variables such as the GCS, can be more helpful in determining the need for intubation. The GCS should be a consideration, but not the only factor in the decision to intubate [20]. While aspiration risk remains high in obtunded patients, objective indicators of the need to perform ETI remain unclear. Intubation, when performed, should be done early to reduce risk of complications and aspiration [38].

Medications to Facilitate Airway Management

Using both a sedating and paralyzing agent has been shown to increase successful intubation and decrease complication risk [39, 40]. However, the drug choice for induction and paralysis will change, depending on the pharmaceutical ingested and the clinical condition of the patient.

The use of sedating agents such as benzodiazepines, barbiturates, etomidate, or propofol as induction agents can assist with the management of underlying agitation, seizures, or sympathomimetic overdrive, which can be from toxicant effect or underlying withdrawal, but may cause myocardial depression [41]. Ketamine has several desirable qualities as a

sedative agent. Because of its dissociative properties, it tends to maintain airway reflexes, including spontaneous respiratory drive and protection of the airway from secretions [42–44]. It may be a good choice for use in patients with bradycardia, hypotension, or depressed cardiovascular status [42–45]. As ketamine has been found to increase heart rate, blood pressure, and myocardial oxygen demand, it should be used with caution in poisoned patients with underlying coronary artery disease, heart failure, or sympathomimetic overdrive [46, 47].

Paralytic agents improved intubating conditions and intubation success rates in a prospective study in poisoned patients [39]. Clinical context must always dictate care, but addition of a paralytic in the appropriate patient may help to facilitate successful ETI and should be considered in all cases without profound hypotonia unless otherwise contraindicated. The use of neuromuscular blockade may be particularly helpful in patients in whom cardiovascular instability precludes the use of high-dose sedation; in patients with hypertonicity from serotonin toxicity or sympathomimetic poisoning; in patients with hyperthermia due to motor hyperactivity; and in patients with toxin-induced status epilepticus [48]. Rapid-onset, short-acting depolarizing agents such as succinylcholine are contraindicated in the patients who may have underlying hyperkalemia, such as patients with acute digoxin toxicity, or severe rhabdomyolysis after a prolonged period of toxin-induced immobility or increased motor activity due to sympathomimetic drug intoxication [49]. In such cases, the use of a non-depolarizing paralytic may be preferred [50].

Adjunctive and Antidotal Therapies Prior to Intubation

In patients presenting with CNS depression in the context of poisoning, reversal of CNS depression with antidotal therapy may obviate the need for intubation. While employing naloxone to rapidly reverse the CNS depression associated with opioid overdose, severe adverse effects are rare and using the lowest effective dose minimizes risk (0.04 mg IV initial dose, then escalating doses) [51].

Flumazenil should be used with caution, as use of high doses of flumazenil in benzodiazepine-dependent patients may produce seizures by unmasking epileptogenic co-ingestions [52–54]. A large review determined that there is a 2.85 times increased risk of adverse events when using flumazenil compared with placebo in suspected benzodiazepine intoxication, including convulsions and supraventricular tachydysrhythmia [55]. However, several more recent studies have reported that the use of flumazenil in small increments (0.25–0.50 mg intravenous) in mixed overdoses is likely safe, with a low risk of seizures associated with flumazenil administration, from 0.6 to 1.4% [52, 55, 56]. The risk of flumazenil-induced benzodiazepine withdrawal resulting in seizures is exceedingly rare. In our opinion, it is reasonable to attempt treatment with a 0.25–0.50 mg IV while pre-oxygenating for intubation. This may be enough to prevent the need for ETI. If a seizure occurs, proceed with sedation, paralysis, and ETI. Supportive care with ETI is likely safer given potential seizure risk, especially with epileptogenic co-ingestions.

In a retrospective review of poisoned patients managed with intravenous lipid emulsion therapy (ILE), the majority of patients also managed with ETI had no significant change in the GCS after receiving ILE, suggesting that intravenous lipid emulsion was not effective at reducing the need for ETI [9]. In a randomized controlled trial of ILE in the setting of non-local anesthetic drug overdoses, there was no significant effect in rate of ETI; however, the GCS was significantly improved at 6 hours post-infusion in patients receiving ILE [57]. In our opinion, more research is needed to determine if ILE may reduce the availability of sedation medications such as etomidate and propofol, which has potential to reduce the efficacy of sedative use in ETI.

Decontamination in Conjunction with Airway Management

The potential for aspiration of gastric contents as well as activated charcoal (AC) should be considered in all poisoned patients. Patients with a high risk of aspiration should be intubated, and AC may be administered via gastric tube with a low risk of resulting aspiration pneumonitis. However, AC administered to the stomach via gastric tube may be aspirated into the lung in up to 25% of patients and has led to other adverse events such as obstructive laryngitis [58, 59]. There are rare cases of charcoal aspiration pneumonitis, including one case demonstrating bronchoscopic and CT evidence of extensive endobronchial charcoal deposition and bilateral confluent areas of lung consolidation in the setting of acute respiratory distress syndrome after AC aspiration [60]. A retrospective study of patients receiving AC following ETI for poisoning found a 4% rate of aspiration pneumonitis as determined by the development of a new infiltrate on chest x-ray within the 48-hour period following intubation [61]. In a prospective randomized trial, the rate of aspiration after AC was < 1% and no different than the control group [62]. Gastric lavage may be considered in the intubated patient who presents within an hour of a life-threatening ingestion for which no antidote or effective treatment is available, but should be considered only when the benefits of the procedure significantly outweigh the risks of gastric or esophageal perforation, or aspiration. Routine use of lavage is not recommended [63, 64]. In our opinion, among patients who meet criteria for ETI, airway management should take priority and be performed prior to decontamination. Overall, we found a scarcity of recent data indicating the risk reduction of aspiration related to ETI in poisoned patients, and whether ETI reduces risk of

aspiration in the context of interventions such as gastric lavage, whole bowel irrigation, or AC.

Alternative Approaches in the Difficult Intubation of Poisoned Patients

Supraglottic devices have a role for both pre/reoxygenation and as a rescue device, and may be helpful for rapid airway control if multiple patients present simultaneously with poisoning, but do not replace ETI for definitive airway security [65–67]. Video laryngoscopy or fiberoptic bronchoscopy may be useful for those with predicted difficult airway anatomy, but the latter requires considerable skill and training to use effectively, which may necessitate anesthesiology consultation if the clinician is not facile with these techniques [66].

Prior studies of patients intubated using a nasotracheal approach over a 5-year period found that one of the most frequent primary diagnoses was drug overdose. Nasal dilation, topical vasoconstrictors, and sedation improved nasotracheal intubation success [68]. While an uncommon approach, in toxicological exposures that result in severe oral or laryngeal edema without pharyngeal or tracheal damage, nasotracheal intubation can be considered if time allows, as an alternative to surgical airway options [69].

Delayed-sequence intubation is performed by administering sedating agents that do not block or blunt spontaneous ventilation, or prevent the maintenance of airway reflexes. Agents that are often considered in delayed-sequence intubation include the dissociative ketamine and dexmedetomidine [70]. We feel that in cases with a severe sympathomimetic toxidrome, the use of ketamine may worsen the patient's toxicity, and agents like propofol or etomidate should be utilized. However, in patients with poisoning without sympathomimetic effects, ketamine can provide dissociation to facilitate preintubation resuscitation and preoxygenation via face mask, or noninvasive positive pressure ventilation (NIPPV) systems such as continuous positive airway pressure and bilevel positive airway pressure.

Minimizing risk of deoxygenation may be especially important in patients in an agitated or hypermetabolic state secondary to toxicant effect, as cellular oxygen demand will be higher and precipitous desaturation a possibility. Preoxygenation and nitrogen washout can be performed by face mask, high flow nasal cannula, or the NIPPV systems [71, 72]. Early utilization of the NIPPV may facilitate preoxygenation and may be able to obviate the need for ETI in appropriately selected patients, but caution should be utilized when selecting the NIPPV as primary method of ventilation and oxygenation in a poisoned patient [73]. While the NIPPV may be considered in patients with rapidly reversible triggers for acute lung injury, it cannot replace ETI as a definitive airway and does little to protect against aspiration. Delay to intubation in patients due to the use of the NIPPV may cause adverse outcomes in patients with prolonged effects from poisoning, or advanced stage of acute lung injury [74].

Apneic oxygenation is another important consideration during ETI, whether during the rapid sequence intubation or delayed-sequence intubation process, particularly in patients with limited physiologic reserve, where apnea or hypoventilation during ETI would be poorly tolerated [75, 76]. Apneic oxygenation works via the passive diffusion of oxygen from the pharynx into the lungs down the oxygen concentration gradient, and can be accomplished via standard or high flow nasal cannula devices [70, 71, 75, 76]. In cases where severe hypoxemia is a concern or where prevention of respiratory acidosis is a priority (e.g., salicylates, tricyclic antidepressants), bag-valve-mask ventilation may be implemented during the apneic period of ETI [77].

Poisoned patients with potential for progressively worsening airway edema, direct oropharyngeal or laryngeal damage, or severe toxin-induced hemodynamic instability may be managed with increased probability of success via awake intubation such as by using local airway anesthesia, low-dose sedation, and intubation under direct visualization such as with fiberoptic scope or video laryngoscopy [70]. In our opinion, if intubation is not possible due to large volume emesis, high volume suction devices should be used, and alternative airway access such as surgical airway may be considered. While aspiration is rare in nil per os (NPO) operative patients using supraglottic airways, these devices provide limited protection from aspiration when high volume emesis is present, compared with ETI [78, 79].

Post-Intubation Care

The use of dexmedetomidine for post-intubation sedation may lead to bradycardia, so should not be used in patients with ingestions of that cause negative inotropy or chronotropy, such as beta blockers, calcium channel blockers, or alpha-2 agonists [80]. Longer acting agents, such as benzodiazepines, may be beneficial for toxicological cases with a presumed long course, such as alcohol withdrawal, but have been associated with over-sedation [80–82].

Patients with repeated muscular contractions due to serotonin toxicity, antimuscarinic toxicity, or stimulant use with psychomotor agitation may develop hyperthermia, rhabdomyolysis, and acidosis. In cases where motor movement exacerbates toxicity, paralysis with long-acting non-depolarizing agents (e.g., vecuronium) or cisatracurium may be used to stop motor activity, or decrease rhabdomyolysis and heat production. Cisatracurium may be advantageous in these cases as it can be stopped to perform a neurologic examination and allows for repeated evaluations of motor activity. In patients with existing renal or hepatic failure, it is preferred over other agents that may develop prolonged effects due to delayed clearance, such as vecuronium [83–85].

Cholinergic agents that block acetylcholinesterase may pose an increased risk when intubating patients who have received these medications. Succinylcholine is metabolized by acetylcholinesterase. Acetylcholinesterase inhibitors include physostigmine, which may have been given IV in an attempt to reverse anticholinergic delirium, and several pharmaceuticals used for dementia. When intubating patients with dementia who are treated with donepezil, rivastigmine, or galantamine, an interaction with succinylcholine resulting in prolonged neuromuscular blockade should be anticipated [86–88].

Considerations Regarding Specific Pharmaceutical Toxicants

Sympathomimetic Toxicity

Patients with severe sympathomimetic toxicity can present with hyperadrenergic manifestations including seizures, dysrhythmias, depressed myocardial contractility, hyperthermia, tachycardia, hypertension, diaphoresis, and agitation [89, 90]. Patients may develop complications that interfere with ETI such as increased mean arterial pressure; increased heart rate; increased cerebral and myocardial oxygen demand; and increased intracranial and intraocular pressure, laryngospasm, bronchospasm, and dysrhythmia [70]. Pre-treatment adjunctive medications such as fentanyl may be used to anticipate and blunt these effects [91]. Sedation may be used during intubation to limit excitatory neurotransmission within the CNS. Benzodiazepines are recommended due to favorable therapeutic index and for secondary reduction of seizure risk, as well as for high efficacy in treatment of sedative-hypnotic withdrawal, which can mimic sympathomimetic toxicity [92]. Patients with agitated delirium and hyperthermia (e.g., methamphetamine, cocaine, other stimulants) may have severe acidosis, hypoxemia, and hypovolemia, and these patients have a high risk of cardiac arrest and hypotension in the period surrounding ETI, so it is our recommendation that pre-, peri-, and post-ETI fluid resuscitation and optimization of ventilation and oxygenation be considered [93–96].

Sodium Channel Blockade

Patients with severe prolongation of the QRS from pharmaceutical overdose, particularly tricyclic antidepressants, should be treated with sodium bicarbonate (100 mL 8.4% sodium bicarbonate in adults or more) as part of the pre-intubation resuscitation to optimize cardiac conduction, reduce risk of dysrhythmia, and reduce the risk of peri-intubation acidosis [49].

Salicylate Toxicity

Decision for ETI in salicylate toxicity should be considered carefully because toxicity can worsen with the loss of centrally driven hyperventilation [97]. In many of these patients, the combination of tachypnea; confusion; and pale, diaphoretic skin may lead the practitioner to intubate for presumed respiratory failure [97]. In these cases, a blood gas may be used to evaluate for ventilatory failure and a low pCO_2 may confirm that these signs are due to CNS effects of salicylate—intravenous dextrose and sodium bicarbonate should be administered to manage neuroglycopenia and to decrease CNS salicylate concentrations, respectively [98]. Sedation and paralysis leading to hypoventilation or apnea during ETI lead to acute hypercarbia and respiratory acidosis [23]. In salicylate toxic patients, this respiratory acidosis in addition to the primary metabolic acidosis leads to a decrease in serum pH and an increase of salicylate transfer into the brain, leading to worsening toxicity. For this reason, we recommend the following approach to a salicylate toxic patient. First, determine if the patient truly requires intubation and avoid intubation based solely on initial patient appearance of confusion, diaphoresis, and tachypnea, in the context of salicylate ingestion. During preparation for intubation, administer 100 mL (2 ampules) of sodium bicarbonate prior to sedative and paralytic. Consider the NIPPV or bag-valve-mask to match minute ventilation and avoid falling pH due to hypercarbia until intubating conditions are optimized. Intubate as quickly as possible and set ventilator settings to be slightly higher than the minute ventilation that the patient was producing prior to intubation. This often requires ventilatory rates of greater than 20 breaths/minute in adults, with sometimes lower tidal volumes at high ventilatory rates to avoid breath stacking and barotrauma, but to maximize highest possible minute ventilation. Blood gases should be monitored every 15 minutes in the first hour after intubation and appropriate ventilatory changes are made [23, 97].

Other Specific Toxidromes

Anticholinergic toxicity, as occurs with large ingestions of antihistamines, antipsychotics, and some medications used in the treatment of Parkinson's disease, may be managed with benzodiazepines or physostigmine. However, physostigmine should be avoided if ETI is needed for airway protection and succinylcholine use is planned as it interferes with succinylcholine metabolism [99].

Ingestion of alpha-2-agonists, such as clonidine or tizanidine, may require management with ETI which may be complicated by bradycardia, hypotension, and emesis [100]. Clonidine ingestions are the most common indication for ETI in pediatric overdoses [4]. In cases with bradycardia and hypotension, intravenous fluid boluses prior to intubation may

mitigate post-intubation severe hypotension. Atropine may be used to prevent vagally stimulated bradycardia, particularly in children with bradycardia-inducing poisonings [101]. Limited evidence suggests that the CNS depression from clonidine overdose may respond to high-dose intravenous naloxone and obviate the need for intubation [102]. Once the decision to intubate is made, it may be reasonable in opioid-naive children to attempt to reverse the CNS toxicity by treating with up to 10 mg intravenous naloxone [102].

Other overdoses may lead to bradycardia and hypotension, including calcium channel blockers, beta blockers, and other antihypertensives. In most of these cases, intubation is performed for airway protection, not respiratory failure, and may be performed in a delayed or awake fashion to optimize hemodynamics. Intravenous fluid boluses and push-dose vasopressors may be used to increase blood pressure; insulin bolus to increase inotropy; and atropine, glucagon, and beta agonists to increase chronotropy in the peri-intubation period [103].

Conclusion

Management of the airway in the critically ill pharmaceutical-poisoned patient comes with numerous challenges. The existing literature does not elucidate one specific management strategy for the poisoned patients. Instead the clinical circumstances should guide the decision to intubate; the specific approach to ETI; the administration of any antidote, paralytic, or sedating medication and decontamination; and the selection of induction or paralytic medications. The specific properties of the pharmaceuticals that were ingested should be taken into consideration when developing an airway management strategy. Risk of hemodynamic compromise, altered mental status, seizure, agitation, and aspiration should be considered, and a strategy chosen to mitigate risk of decompensation.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflicts of interest.

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