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Complications of Emergency Tracheal Intubation: Hemodynamic Alterations - Part I

Thomas C. Mort, MD

Emergency airway management outside the elective operating room presents considerable risks to the patient and significant challenges to the practitioner. Complications and adverse consequences are commonplace, yet they have not received their justified discussion or scrutiny in the literature. This review will discuss potentially life-threatening complications partitioned into 2 broad categories: hemodynamic and airway. Part 1 will focus on alterations in the heart rate and blood pressure, new onset cardiac dysrhythmias and cardiac arrest. Part 2 will explore airway related consequences such as hypoxemia, esophageal intubation, multiple intubation attempts, and aspiration.

Key words: intubation, hypotension, bradycardia, cardiac arrest, tachycardia, hypertension, airway management

The Response to Airway Manipulation

Laryngoscopy, tracheal intubation, and other oropharyngeal-laryngotracheal manipulations are potent stimulators of the sympathetic and parasympathetic nervous systems, with variable and sometimes unpredictable responses in any patient but, in particular, the critically ill patient requiring emergency tracheal intubation. Airway manipulation under emergency circumstances may be plagued by an increased risk of airway management difficulties and critical events such as multiple laryngoscopy attempts, esophageal intubation, and aspiration. Each may lead to severe hypoxemia, culminating in some with medullary induced bradycardia with subsequent hypotension, decreased cardiac output, and if left uncorrected, cardiovascular collapse. Extreme fatigue, hypoxia, hypercarbia, acidosis, and other stresses may push the patient’s physiologic system to nearly maximal sympathetic outflow.

The acute marked attenuation of the sympathetic tone associated with resolution of hypoxia and hypercarbia may exacerbate hypotension through a decrease in cardiac preload/afterload, especially when accompanied by hypovolemia. Positive-pressure ventilation and positive end-expiratory pressure (PEEP) may further compromise ventricular preload by impeding venous return.

Most clinical studies investigating these physiologic alterations related to airway management have been performed in hemodynamically stable patients. The ensuing discussion is based on currently available literature, so one may assume that any physiologic effects will likely be exaggerated in degree and more numerous in occurrence, and thus more poorly tolerated in the critically ill patient.

Preparing the patient for airway management often requires a substantial dosage reduction from standard dosage regimens of sedative hypnotics and opioids. Titration of induction medications to desired effect is more time consuming but should be considered as a component of a best-practice strategy. Treating hemodynamic perturbations, which may be extreme in character, with vaspressors, vasodilators, short-acting selective β-blockers,
and inotropic agents based on the patient’s individual clinical picture coupled with the practitioner’s experience is an additional component of a best-practice strategy.

The hyperdynamic increase in blood pressure or heart rate, or both, during airway manipulation has been the focus of a tremendous amount of time and energy in the elective operating room in an attempt to temper such a response with the goal of reducing its impact on cardiovascular risk and patient morbidity [1-6]. Moreover, postinduction hypotension, bradycardia, and cardiac arrest have been reviewed in the elective operating room environment [7-12] but have only recently been highlighted in the setting of emergency airway management [13-18]. Thus, extreme bradycardia as a result of vigorous, repetitive laryngoscopy coupled with hypoxemia may lead to hypotension, decreased cardiac output, and cardiac arrest if left uncorrected. This response, fortunately, is rare during the elective surgical setting but can represent a devastating cardiovascular insult in a critically ill patient who requires emergency airway management [19].

An excellent review of the functional anatomy, physics, airway pharmacology, and more detailed explanation of the physiologic response to intubation can be found in the Basic Clinical Science Considerations section, Part 1 (pages 1-120) in Airway Management, Principles and Practice, edited by JL Benumof (Mosby Publishing). Also, chapter 9 in the same text expertly covers preparation of the patient for awake intubation.

The Hyperdynamic Response to Airway Management

The hyperdynamic response that frequently accompanies direct laryngoscopy and endotracheal intubation reflects a number of physiologic factors, including wakefulness, the magnitude, vigor, and extent of the airway manipulation, underlying hypertension and cardiovascular disease, intravascular volume status, and other factors that often culminate in hypertension and tachycardia, or both, with airway manipulation [19-23] (Table 1). Laryngoscopy and tracheal intubation, alone, are potent stimulants and commonly lead to an increased heart rate or a hypertensive blood pressure response, or both [1-5,24]. Persistent elevation in blood pressure after intubation may reflect an exaggerated response often seen in the high-risk individual with significant preexisting comorbid conditions such as diabetes and renal and cardiovascular disease, as well as the extremes of age [23,24], and may be accentuated in those with a central nervous system insult, the intoxicated, and the traumatized patient [19,25].

This hypertensive response may be treated with intravenous induction agents titrated to a desired end point; vasodilators such as nitroglycerine, sodium nitroprusside, or diltiazem; or β-antagonists such as esmolol or labetalol [26-31]. Careful titration of these agents is imperative, because overly aggressive treatment may quickly introduce further hemodynamic compromise when the therapy outlasts a self-limited phase of postintubation stimulant. Select groups of critically ill patients may be prime candidates for aggressive therapy in the hopes of blunting the hyperdynamic response to airway instrumentation; for example, head injury, intracerebral hemorrhage, cerebral vascular accident, or seizure disorder [19,25].

Regardless of the underlying comorbid conditions, graceful or aggressive attempts to advance the endotracheal tube (ETT) into the trachea, balloon inflation, ETT suctioning, mainstem bronchial/carinal impingement, coughing, bucking, or fighting ventilation are additional factors that may potentiate an acute alteration in hemodynamics, especially the hyperdynamic response [19,25,32]. The aggressive administration of anesthesia induction agents is literally a double-edged sword, one that is capable of limiting the upper limits of blood pressure and heart rate elevation during airway manipulation and oppositely, the quiescent, stimulation-free period that usually follows airway manipulation may lead to a sharp reduction in the blood pressure [1,13,33-35].

Table 1. Potential Morbidity Due to Acute Hypertension and Tachycardia

<table>
<thead>
<tr>
<th>Condition / Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial strain, ischemia, infarction</td>
</tr>
<tr>
<td>Acute left ventricular, right ventricular failure</td>
</tr>
<tr>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Dysrhythmia 2’ strain, ischemia</td>
</tr>
<tr>
<td>Post-hypertensive cardiovascular collapse</td>
</tr>
<tr>
<td>Increased cerebral blood flow/pressure</td>
</tr>
<tr>
<td>Cerebral vascular bleeding</td>
</tr>
<tr>
<td>Aortic dissection-propagation of injury</td>
</tr>
<tr>
<td>Aneurysm stress/leak/rupture thoracic, abdominal, cerebral, optic</td>
</tr>
<tr>
<td>Accelerated bleeding (ie, trauma)</td>
</tr>
<tr>
<td>Alteration in valvular flow dynamics</td>
</tr>
</tbody>
</table>

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The Challenge to Control the Hyperdynamic Response

The adult patient with any combination of current and past medical and surgical conditions may experience mild, moderate, or severe increases in heart rate or blood pressure, or both, during laryngoscopy and ETT placement [1,19,25]. Patients may still break through regardless of the anesthesia induction agents, adjuncts, and vasoactive medications that can be administered in an attempt to prevent such a hyperdynamic response [30-33].

Being mindful of the preexisting comorbidities and the current clinical deterioration prompting intubation, the airway manager’s judgment and experience will influence the medication choices and techniques to prepare the patient for airway instrumentation. The major challenge is to select the agents that will achieve the goal of blunting, attenuating, or blocking the postlaryngoscopy hyperdynamic response for only a brief amount of time with minimal subsequent influence.

The literature does not convincingly support one method of induction over another in the critically ill patient who requires emergency airway management. Strategies are best tailored to the individual patient’s needs, based on the experience and judgment of the airway manager rather than a standard intubation protocol. The administration of a rapid-onset, relatively short-duration agent to assist in the hyperdynamic response to airway manipulation in the emergency setting is similar to medications that are commonly incorporated in the operating room (intravenous β-blockers, calcium-channel blockers, vasodilators).

The addition of a neuromuscular blocking agent (NMB) may have a major impact on the dosing of induction agents and the subsequent need for vasoactive medications, especially in the critically ill patient who is maintaining blood pressure by virtue of concurrent agitation, struggling, and muscle contraction. After cessation of muscle contraction and vigorous activity, systemic vasodilatation may follow the administration of NMB agents, independent of any direct or indirect vasodilatory effects that may take place by NMB-induced histamine release or other similar pathways.

Currently available critical care airway literature, for the most part, neglects the reporting of hypertension and the hyperdynamic response to airway manipulation. Those who have reported it have suggested it is uncommon, falling in the range of 0.3% to 11% [21,22,32] compared with more recent literature that reported 1 in 5 patients undergoing emergency tracheal intubation experiences hypertension postintubation [13-17].

High-Risk Groups for Hyperdynamic Response

Neurologic injury, trauma, the septic populations, and younger patients (<50 years) who require emergency tracheal intubation appear to be at higher risk for developing a hyperdynamic response [19,20,36-38]. The hyperdynamic response to airway manipulation is well known in seizure disorders, head trauma, intracerebral hemorrhage, aneurysmal vasospasm, cerebral vascular accidents, and hydrocephalus in the elective setting of the operating room, yet these responses seem to be exaggerated in the emergency setting, with or without aggressive prelaryngoscopy preparation [20,25]. Likewise, the trauma victim, exclusive of head injury, has a higher heart rate and blood pressure elevation with laryngoscopy that appears to be inversely proportionate to age [19,20].

Airway management with the goal of optimizing ventilation and oxygenation is obviously important to the well-being of the patient, but some reports have suggested that out-of-hospital tracheal intubation was associated with adverse outcomes after severe traumatic brain injury. The implications for these findings remain undefined [19]. Unfortunately, to date, no well-designed, randomized controlled trials have been conducted to guide the clinician with evidence-based strategies for the patient population that requires emergency intubation.

Hypotension

The incidence of postintubation hypotension in the emergency setting is the most common of the hemodynamic alterations and may be based on many factors, singly or combined. The institution of positive-pressure ventilation and PEEP, plus any vasodilatation and myocardial depression from anesthesia agents, may contribute to postintubation hypotension [1,33-35]. This response is accentuated in incidence and magnitude in the critically ill patient who is struggling with underlying cardiopulmonary deterioration, acid-base imbalance, septic-induced hemodynamic alterations, hemorrhage, hypovolemia, and other maladies [20,33].

Hypotension, even for a brief duration, has been suggested as a significant contributing factor to patient morbidity. A prolongation of hospital stays and poorer outcomes, especially in trauma patients...
and those with neurologic injuries have been associated with postintubation hypotension [20,34,37,39].

Very few published reports that evaluate complications of emergency airway management mention hemodynamic alterations, particularly hypotension. Tayal et al [40] and Sakles et al [41], in 2 separate studies, reported that less than 0.3% (4 patients in nearly 1200) developed hypotension (systolic blood pressure <90 mm Hg) associated with emergency department intubation. Conversely, emergency airway services outside the operating room (including the emergency department) provided by an anesthesiology department reported an incidence of 42% of hypotension, and more than 50% of those patients experiencing hypotension required vasoactive medications to elevate the mean arterial pressure (MAP) to more than 65 mm Hg [13-15].

Moreover, a study of intubation and the initiation of mechanical ventilation in emergency department patients observed an incidence of 28.6%, with nearly half requiring vasoactive agents to support hemodynamics after intubation [35]. The simple recognition of such events, the variable definitions of complication parameters, and self-reporting practices may be the underlying differences between these reports [42,43]. Nonetheless, the extent and degree of hypotension will be influenced by any administered induction agents, the patient's volume status, preexisting comorbidities, and the reason for the clinical deterioration that prompted the need for airway control, plus numerous other factors [44-46].

Patients with acute cardiovascular dysfunction who require emergency intubation experienced a higher incidence of postintubation hypotension (54%) compared with all noncardiac groups (36%) coupled with a commensurate need for vasoactive medications to treat the hypotension [47]. Patients with pulmonary (61%) and abdominal (73%) sepsis are particularly prone to postintubation hypotension, and more than 60% may require vasoactive agents to support hemodynamics [unpublished data, Hartford Hospital].

**Induction Agents and Their Influence on the Rate of Hypotension**

The choice of the method to prepare the patient for airway intervention will vary with the patient’s clinical situation and the practitioner’s experience, and most importantly, the interpretation of the patient's needs. The use of topical anesthesia—or administering no medication—may be appropriate for some patients owing to their dire clinical circumstances, their reduced level of responsiveness, or concerns about airway management difficulties. Whether the patient being readied for an airway intervention truly requires medication for sedation-analgesia-hypnosis, with or without muscle relaxation, almost all patients do receive intravenous induction agents. Sedative-hypnotics, such as the benzodiazepines, propofol, and etomidate, are frequent choices of emergency airway managers.

Several emergency medicine groups have compared etomidate with midazolam for prehospital rapid-sequence intubation (RSI). Swanson et al [48] found a low incidence of hypotension with both agents (6% to 10%), although the mean dose of midazolam was relatively small. Conversely, Choi et al [44] used midazolam (2-4 mg) or etomidate (0.2-0.3 mg/kg) and observed 1 in 5 patients administered midazolam had hypotension compared with etomidate (4%). Davis et al [49] also noted that midazolam provided for prehospital RSI was associated with a dose-related incidence of hypotension.

Many prefer the hemodynamic stabilization offered by etomidate, but it should not be considered a panacea [50-53]. Its role as a single-dose induction agent is currently being questioned owing to transient depression of the adrenal axis. Recent evidence suggests that the adrenal suppression, once regarded as a minor concern, may be much more influential in patient well being. Suppression of glucocorticoid synthesis may potentially confound the diagnosis of adrenal insufficiency, and some authors have expressed caution with its administration, especially in patients with sepsis or trauma. Some authorities have suggested that etomidate should be avoided completely, avoided in select populations such as the septic population, or if it is used, empirical adrenal replacement therapy for at least 24 hours should be considered [54-56]. Other alternatives to etomidate may offer improved intubating conditions [57]. Perhaps well-designed clinical trials should be performed to determine the relevance of these published precautions.

Barbiturates, which were mainstay agents decades ago, have been displaced by etomidate, midazolam, and propofol in the elective and the emergency settings; however, thiopental remains a useful agent in central neurologic injury states because of its valuable effects on cerebral metabolism [58,59]. Its low cost, wide availability, lack of adrenal axis influence, and well-known hemodynamic profile may bring it back into mode sometime in the future.
The use of NMB for emergency airway management is to some controversial and to others a normal component of the intubation process. The literature seems divided into 2 camps: routine use of NMB typically in a rapid-sequence technique (commonly emergency medicine/prehospital personnel) [36,40-42,48,49,60-65], or selective use of NMB with maintenance of spontaneous ventilation by titration of sedative-hypnotics-analgesics to effect allowing intubation (some anesthesia personnel) [46,66]. The justification for using NMB to ease the effort of intubating the trachea may not effectively be balanced by the loss of spontaneous ventilation in the high-risk patient with a potential or known difficult airway.

In reports supporting NMB use in a rapid-sequence technique, it is highly touted as being nearly universally successful, with few adverse effects or patient morbidity [40-42]. Unfortunately, NMBs are administered in many cases almost without regard for the patient’s airway status [40-42]. Rationalization that the use of NMB to ease the effort of intubating the trachea may not effectively be balanced by the loss of spontaneous ventilation in the high-risk patient with a potential or known difficult airway.

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Table 2. Medication and the Rate of Postintubation Hypotension

<table>
<thead>
<tr>
<th>Agent(s) Use (%)</th>
<th>Dosage</th>
<th>Overall Use (%)</th>
<th>Hypotension (%)</th>
<th>VP (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Topical only or no meds</td>
<td>14</td>
<td>46</td>
<td>53</td>
<td></td>
</tr>
<tr>
<td>Propofol 0.4-1.5 mg/kg, 30-200 mg</td>
<td>19</td>
<td>35</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td>Etomidate 0.1-2 mg/kg, 5-20 mg</td>
<td>40</td>
<td>42</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>Thiopental 1-3 mg/kg, 75-400 mg</td>
<td>6</td>
<td>31</td>
<td>36</td>
<td></td>
</tr>
<tr>
<td>Morphine alone 0.05-0.1, 2-8 mg</td>
<td>4</td>
<td>45</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Midazolam 0.02-0.08 mg</td>
<td>13</td>
<td>41</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Morphine + Midazolam</td>
<td>4</td>
<td>39</td>
<td>46</td>
<td></td>
</tr>
</tbody>
</table>

a. Data from Hartford Hospital database of 3224 patients undergoing emergency intubation.

b. Vasopressor agent (dopamine, norepinephrine, phenylephrine, ephedrine).

Bradycardia

Opposite the commonly seen hyperdynamic response to intubation is an abrupt slowing of the heart rate. It is an undesirable response, because it nearly ubiquitously ushers in hemodynamic instability. Patients who have been administered medications that slow SA-node, AV-node, or ventricular conduction, such as digoxin, calcium-channel blockers, amiodarone, procainamide, and β-blockers, in addition to the aggressive use of fentanyl or other medications that possess vagotonic properties, may be at increased risk for a further slowing of the heart rate. Laryngoscopy and tracheal intubation may counter these effects, yet may lead to hemodynamic compromise if the sympathetic-parasympathetic balance leans toward increased vagal tone. Currently, a paucity of supporting data exists for evidence-based recommendations for emergency airway management in patients receiving medications with SA or AV nodal influence, such as β-blockers, digoxin, calcium-channel blockers, and amiodarone in adult population, and caution is advised.

Preexisting bradycardia caused by medication, an intrinsically slow heart rate in hypertensive disease in the elderly patient, the physically fit, and occasionally with severe hypoxemia or elevated intracranial pressure, may place the patient at a lower threshold to experience further bradycardia. Other significant contributing factors such vagotonic stimulation via vigorous or prolonged laryngoscopy and tracheal manipulation, esophageal intubation, and airway related complications that lead to severe or prolonged hypoxemia have resulted in bradycardia and cardiac arrest [13]. Progressive bradycardia has also been noted to precede intraoperative cardiac arrests in most cases [7-11]. A common factor leading to infantile bradycardia in the operating...
room is hypoxemia; hence, bradycardia is associated with significant morbidity and even mortality during the administration of anesthesia in both children and adults [8,9,11]. Propofol has been suggested as a contributing factor for bradycardia, although its role remains ill defined [67].

Despite the range of clinical problems that may potentiate bradycardia, primarily hypoxemia and vagotonic influences related to airway manipulation appear to be major factors. The incidence of bradycardia dramatically increases as the number of airway-associated complications mount during difficult laryngoscopy and intubation [13,14]. The reduction in heart rate is typically accompanied by a significant lowering in blood pressure, often requiring aggressive therapy. The incidence of bradycardia, occurring in 1.5% to 3.5% of emergency intubations, is notably increased in the face of specific airway-related complications, namely esophageal intubation, hypoxemia, regurgitation and aspiration, and 3 or more attempts with the laryngoscope [13,14] (Table 3). When confronted with an acute reduction in heart rate during or after intubation, it behooves the practitioner to optimize oxygen delivery and support the airway, provide pharmacologic intervention, and rule out any common factors that may lead to bradycardia, including unrecognized esophageal intubation, tension pneumothorax, or main-stem bronchus intubation [18,69]. Of note, a progressive bradycardia preceded a cardiac arrest in 90% of the cases in several databases [7,8,13,14].

The aggressive teaching of airway management techniques and incorporating the standards of the American Association of Anesthesiologists Difficult Airway Algorithm outside of the operating room may decrease the incidence of hypoxemic-related bradycardia. One strategy involving direct laryngoscopy, supplemented by end-tidal carbon dioxide detection combined with immediate access to a tracheal tube introducer (bougie), the laryngeal mask airway, the Combitube (Kendall-Sheridan Corp, Argyle, NY), and fiberoptic bronchoscopy, reduced the incidence of bradycardia and cardiac arrest episodes by 50% [13,68-72].

### Dysrhythmias

The acute onset of a new dysrhythmia during the manipulation of the airway or immediately after completion of securing the airway is infrequently reported. Preexisting rhythm disturbances may be exaggerated by even straightforward airway manipulation but may pale in comparison to the response initiated by a vigorous laryngoscopy, especially if it is associated with multiple attempts, inadequate sedation, or additional myocardial compromise. Patients may experience a new onset of a dysrhythmia, most typically a supraventricular tachycardia, atrial fibrillation, or flutter with a rapid ventricular response that is usually poorly tolerated by the critically ill patient. These rhythm alterations are often complicated by varying degrees of hypotension.

Succinylcholine is a well-known causative factor in contributing to a multitude of atrial and ventricular rhythm disturbances [73]. The critically ill, especially those with cardiac-based clinical deterioration such as acute myocardial ischemia or infarction are prone to experience ventricular arrhythmias (bigeminy,
trigeminy, or frequent or multifocal premature ventricular contractions (PVCs). Likewise, a prolonged, vigorous, or traumatic manipulation of the airway can potentiate life-threatening dysrhythmias [13-15].

The incidence of new-onset cardiac dysrhythmias occurring during or immediately after airway manipulation is scarcely reported, with an overall incidence of 5.8% in the Hartford Hospital database. Discounting severe bradycardia, complete heart block, or asystole, the remaining 60% of the patients who sustained a new-onset dysrhythmia were categorized by supraventricular disturbances (50%, primarily atrial fibrillation or flutter) and ventricular disturbances (10%) that included multifocal PVC, bigeminy, and ventricular tachycardia. These rhythm disturbances were poorly tolerated: two thirds of the patients required vasoactive agents to counter the reduction in the mean arterial blood pressure.

Cardiac Arrest

The risk of anesthesia-related cardiac arrest in the operating room is relatively low, on the order of 1 per 10,000 cases, and most of these are not related to airway mishaps and difficulties [7-11]. The risk of cardiac arrest in the remote location during emergency management of the airway may be as high as 2% outside the operating room [13,14,18]. Profound hypoxemia (pulse oximetry oxygen saturation <70%) proved to be a primary contributing factor because its presence was overwhelming during the airway procedure of those who arrested [13]. Additional risk factors contributing to cardiac arrest during airway manipulation included multiple intubation attempts (3 or more), aspiration, bradycardia, and esophageal intubation, often with one or more of these complications cascading from one to another [13,14]. Non-airway-related cardiac arrests included ETT obstruction due to secretions, tension pneumothoraces, massive pulmonary thromboembolism, medication-induced cardiovascular thromboembolism, and acute myocardial infarction with cardiogenic shock [13]. Table 4 outlines a varied list of etiologic factors that may contribute singly or in combination to the risk of cardiopulmonary arrest and cardiovascular collapse related to tracheal intubation [18].

Reported Hemodynamic Alterations

The currently available reports describing the various hemodynamic changes as outlined in this article are relatively sparse when considering the ramifications that these associated consequences of emergency airway management inflict on patients and their safety. Many data collections tend to emphasize 1 or just a few of the hemodynamic alterations, and those that do report them describe an extremely low rate of complications (Table 5) [17,40-42,66,61-65,72]. Unfortunately, many of the references reported complications without describing in detail the categoric definitions of the individual complication, hence complications may vary depending on their definition and the interpretation of such definitions by the clinical gathering body [43]. Although many of these pathophysiologic factors are seen in the elective operating room setting, the acutely ill patient often will respond to emergency airway management in an exaggerated fashion.

<table>
<thead>
<tr>
<th>Author</th>
<th>Study Type</th>
<th>Patients (N)</th>
<th>Hypo (%)</th>
<th>Hyper (%)</th>
<th>Tachy (%)</th>
<th>Brady (%)</th>
<th>Dysrh (%)</th>
<th>Arrest (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mateer et al [72]</td>
<td>PRO-ED</td>
<td>113</td>
<td>11</td>
<td></td>
<td>1.4</td>
<td>0.9</td>
<td></td>
<td></td>
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<tr>
<td>Dufour et al [61]</td>
<td>RET-ED</td>
<td>219</td>
<td>11</td>
<td></td>
<td>1.4</td>
<td>0.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Norwood et al [62]</td>
<td>RET-ED</td>
<td>229</td>
<td>11</td>
<td></td>
<td>1.4</td>
<td>0.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rotondo et al [63]</td>
<td>RET-ED</td>
<td>198</td>
<td>11</td>
<td></td>
<td>1.4</td>
<td>0.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gnauck et al [64]</td>
<td>RET-ED</td>
<td>60</td>
<td>11</td>
<td></td>
<td>1.4</td>
<td>0.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sakles et al [41]</td>
<td>OB-ED</td>
<td>610</td>
<td>0.5</td>
<td></td>
<td></td>
<td>0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tayal et al [40]</td>
<td>OB-ED</td>
<td>596</td>
<td>0.3</td>
<td></td>
<td></td>
<td>0.5</td>
<td>0.7?</td>
<td></td>
</tr>
<tr>
<td>Vijayakumar et al [65]</td>
<td>PRO-ED</td>
<td>160</td>
<td>0.3</td>
<td></td>
<td></td>
<td></td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>Schwartz et al [66]</td>
<td>PRO-A</td>
<td>297</td>
<td>0.3</td>
<td></td>
<td>0.5</td>
<td>0.7?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mort [17]</td>
<td>PRO-A</td>
<td>1298</td>
<td>41</td>
<td>19</td>
<td>29</td>
<td>3.5</td>
<td>6.5</td>
<td>1.9</td>
</tr>
</tbody>
</table>

RET = retrospective; OB = observational; PRO = prospective; Hypo = hypotension; Hyper = hypertension; Tachy = tachycardia; Brady = bradycardia; Dysrh = dysrhythmia; Arrest = cardiac arrest; ED = emergency department; A = anesthesia.
Summary

The primary goal of emergency airway management is to provide adequate oxygenation and ventilation. If tracheal intubation is required, securing the airway should be completed as quickly as feasible while fostering a safe environment for the patient. The airway manager should formulate a primary airway plan and a contingency plan if conventional attempts fail to secure the airway [70-72]. Understanding the varied hemodynamic changes that may accompany tracheal intubation of the critically ill patient is the first step toward improving airway care and advancing patient safety in this high-risk population.

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Emergency Intubation Complications


