
The Traumatic Airway: The Anesthesiologist's Role in the Emergency Room

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A 1997 survey of nonoperating room emergency airway management and endotracheal intubation practices in the United States revealed that, in 45% of institutions, intubations in the emergency department (ED) were performed by emergency medical physicians, 32% by anesthesiology personnel, and 19% by both. Airway management in trauma patients, however, remains the domain of anesthesiologists.¹

The anesthesiologist, as one of the airway experts, should be consulted early when airway control may be required in trauma patients. In most major trauma centers, anesthesiologists have become part of the ED team evaluating the incoming trauma patient. As part of this role, the anesthesiologist needs to be aware of technical and medical issues specific to the trauma victim. Patients do not always require intubation initially; therefore, obtaining control of the at-risk or potentially at-risk airway is a problem not just in the ED but later in the intensive care unit and in the operating room as well. The trauma patient whose airway requires securing has, by assumption, a full stomach. This patient frequently must be regarded as having a concomitant cervical spine injury and, therefore, often has a cervical collar in place. The patient may also be intoxicated, have significant underlying medical disease, have had a difficult airway before the traumatic injury, present with a traumatic injury to the airway itself, or have significant neurological or other trauma. Indeed, the trauma patient may have many or all of these.

Qualities expected from the anesthesiologist as member of the ED team are swiftness and cooperation, both key to successful trauma care. Some anesthesiologists are intimidated when they are called to the ED to assess a trauma patient. It is not the usual environment of the anesthesiologist: In the OR the anesthesiologist is responsible for managing most of the pharmacological fluid, and supportive therapy. In the ED, however,

the role of the anesthesiologist is commonly restricted to the patient's airway. This, however, does not preclude the anesthesiologist from performing a brief assessment of the patient as expeditiously as the situation allows. Preparation of the OR for this patient will then be much more patient oriented (i.e., anticipated blood loss and need for rapid infusion systems, special monitoring).

Once the trauma team leader or an attending ED physician other than the anesthesiologist has determined that the trauma patient requires a secured airway, the anesthesiologist should not delay endotracheal intubation. This may be complicated if a problem is identified that is a contraindication to proceeding with a rapid sequence induction. Such a problem necessitates rapid determination of the best route of intubation in the most expedient manner. Discussions regarding whether endotracheal intubation is genuinely and immediately indicated should be deferred until the initial work-up of the patient has been completed. In a critical situation, the anesthesiologist has to trust the trauma team leader's decision. Indications to proceed with an emergent intubation in this setting include prophylactic airway control and are absolutely unrelated to airway compromise and respiratory insufficiency. Airway control may be indicated simply to facilitate rapid and safe work-up.

A rapid-sequence, or "crash," induction is the most common and fastest approach to airway control in patients with a putative full stomach. Complication rates and severity of complications, intubation attempts, and time to successful intubation are reduced by this approach compared with blind nasal intubation.² Experience has shown that endotracheal intubation after a rapid-sequence induction and stabilization of the cervical spine via in-line immobilization of the head does not magnify spinal cord injuries. Awake tracheal intubations by mouth, nares, or surgical approach should be rare and restricted to obvious contraindications to a standard induction. A controlled awake intubation of a patient with a cervical spine fracture may be warranted because such an approach permits continuous neurological monitoring. One should be aware, however, that even a well-sedated and topically anesthetized patient may still move his or her neck in the highly stressful emergency environment. In the retching patient, even in-line stabilization may not prevent forceful neck movements by the patient. Bronchoscopic intubation in the patient with traumatic airway bleeding is problematic because visualization of airway structures may be impossible.

■ Evaluation of the Airway

The patient arriving intubated in the ED requires assessment of correct positioning of the endotracheal tube and assessment of adequacy of ventilation. Important questions include: "To what depth has the endotra-

cheal tube been inserted?” “Can mainstem intubation be excluded?” “Are breath sounds equal bilaterally?” “Can a pneumothorax be excluded?” Provided a cuffed endotracheal tube has been inserted, mainstem intubation can be excluded if the cuff can be palpated just above the sternal notch. Palpation of the endotracheal tube cuff is indirectly possible by delicate compression of the pilot balloon with two fingers. If the cuff is above the sternal notch, external application of direct pressure where the cuff is located below the palpating finger will expand the pilot balloon and will be felt as vibration. This maneuver does not absolutely exclude an esophageal intubation. Auscultation of breath sounds, carbon dioxide (CO₂) detection, esophageal inflation bulb, repeat laryngoscopy, and even fiberoptic verification may occasionally be required to confirm correct endotracheal tube placement.

The patient requiring endotracheal intubation should be assessed to determine whether there are contraindications to a rapid-sequence technique. Even in an emergency situation, there is time to perform an expedited evaluation of the airway. This ideally includes a brief history and assessment of the patient’s neurological and respiratory status. In particular, it is important to determine whether the patient has protective airway reflexes and is capable of motion.

At least two relevant and rapid methods for evaluation of potential intubation difficulties have been described. However, some components of these evaluations require patient cooperation and are not feasible in the obtunded patient. In the first approach, four areas are evaluated:

1. The distance between the thyroid cartilage and the symphyseal portion of the mandible (tip of the chin). A distance of less than about 6 cm (or three of the patient’s fingers side by side) is considered potentially problematic because it implies the presence of an anterior larynx, potentially resulting in difficulty with visualization of the vocal cords during direct laryngoscopy.
2. Mouth-opening ability. If fewer than three fingers can be placed in between the teeth, laryngoscopy may be problematic.
3. Distance from the ramus to the symphysis of the mandible. A distance of less than 5.5 to 6 cm is suggestive of difficulty in visualizing the airway.
4. The motion of the neck may be evaluated in a qualitative manner, provided the patient does not have a known or potential neck injury. The patient who cannot flex and hyperextend the neck is considered at risk. The patient in a cervical collar is an extreme example of one in whom such testing is obviously not indicated.

Thus, the patient with a small mouth opening, decreased distance from the thyroid cartilage to mandibular symphysis, decreased ramus to

symphyseal distance, and decreased range of motion of the neck is considered to be at risk for a difficult laryngoscopy.

In 1985 Mallampati and colleagues³ described a more quantitative manner of evaluation. This system classifies the airway in terms of difficulty to be expected in visualizing the airway based on the structures seen when the patient opens his or her mouth as widely as possible and protrudes the tongue as far as possible. A class I airway is one in which the soft palate, faucial pillars, and uvula are seen with the patient maneuver. A class II airway is one in which the soft palate and faucial pillars are visualized, and a class III airway is one in which only the soft palate is seen. Samssoon and Young's⁴ modification of this system adds a class IV airway in which the tongue completely obstructs vision of the soft palate such that only the hard palate is visible. There is some correlation between the airway class and the ease of exposure of the laryngeal inlet via direct laryngoscopy, but with every grading system difficult airways will be missed. Each anesthesiologist's clinical experience is probably the most important factor in the decision process of whether an airway should be labeled as difficult.

Patients with massive upper airway swelling, obvious dislocation of the mandible, disruption of the airway, and an obvious foreign body in the airway may be best treated with a surgical airway. Direct trauma to the airway is relatively rare. In one series evaluating 106 consecutive patients admitted to a single institution over a period of almost 20 years, direct trauma to the airway was predominantly penetrating rather than blunt (100 penetrating to six blunt). Overall mortality was 17%. Fifty-three percent of 13 patients with principal injuries of the thoracic trachea died; cervical tracheal injuries were associated with a mortality of approximately 14%. All patients presented with signs of airway compromise such as tachypnea, dyspnea, cyanosis, subcutaneous emphysema, or an abnormal respiratory pattern. Severe airway compromise was evident in almost half the patients. Hemoptysis was present in less than one third of patients. Patients with major vascular injuries plus tracheal involvement were generally not salvageable. Twenty-four (23%) were treated with oral or nasal intubation, 19 (18%) with emergency tracheostomy, and three (2%) with intubation of the injured trachea.⁵

■ Preparation for Intubation

Once the patient's airway has been evaluated for the feasibility and difficulty of endotracheal intubation, careful preparations must be made for the procedure. In the patient for whom a rapid-sequence intubation (RSI) is necessary, often little is possible in the way of psychological preparation. Nonetheless, if the patient is cognizant of his or her surroundings, the anesthesiologist should calmly explain why the intubation is required.

Intravenous catheters require confirmation that they are neither infiltrated nor placed inadvertently into an artery. Next, the anesthesiologist should confirm that all the equipment and personnel necessary to establish a surgical airway are present at the bedside should intubation and ventilation attempts fail. Emergency cricothyroidotomy—in experienced hands—is a relatively safe and quick means of obtaining an airway when endotracheal intubation fails or is contraindicated.⁶ Approximately 5% of trauma victims will require an emergency cricothyroidotomy to establish an airway. A second person will need to be assigned to assist the anesthesiologist, preferably one trained to give cricoid pressure. Cricoid pressure reduces both the likelihood of aspiration of stomach contents and the amount of air insufflated into the stomach from positive-pressure ventilation. The weight applied with a three-finger technique to the cricoid ring should approximate 8 kg (2 gallons of water). Cricoid pressure may be problematic in a patient with a potential tracheal or laryngeal injury. In a patient with an unstable cervical spine, cricoid pressure should not be initiated before in-line traction and stabilization of the head and neck so as to minimize neck movement. If there is any possibility of cervical spine injury, a third person should perform in-line stabilization of the cervical spine to minimize head and neck extension as well as flexion and cord compression during laryngoscopy.

While preparations are being made, the spontaneously breathing patient should be receiving 100% oxygen (O₂) via an Ambu bag or Mapleson D bag. The patient may not require bag-valve-mask (BVM) ventilation, but the utilization of one of these two devices with a tight mask fit will improve denitrogenation. The Mapleson D bag, a non-self-inflating bag, is preferable because it provides evidence of patient-generated airflow as well as correct placement of the endotracheal tube after intubation. Even though RSIs are originally described without positive-pressure ventilation between administration of induction agent plus muscle relaxant and intubation, most anesthesiologists in the ED perform modified RSIs. In a modified RSI, a patient is ventilated *under cricoid pressure* after induction and before attempts at direct laryngoscopy. Although this maneuver may increase the risk of inflating the stomach and subsequent regurgitation, it provides knowledge of the patient's airway status in case an initial laryngoscopy and intubation attempt is unsuccessful and decreases the incidence, severity, and consequences of hypoxemia. In head trauma patients, hypercarbia and its deleterious consequences on cerebral perfusion pressure may be reduced if positive-pressure ventilation is successful. Under no circumstance would we recommend withholding positive-pressure ventilation in an apneic and hypoxic patient. The consequences of prolonged hypoxia outweigh by far the risk of aspiration through cricoid pressure. However, chest injury or blood aspiration may cause poor pulmonary compliance and make prolonged BVM inadequate and dangerous.

Before anesthetic induction the patient should have appropriate

monitoring. This usually entails a noninvasive blood pressure device, electrocardiography, pulse oximetry, and, when possible, exhaled gas analysis. The latter, although often not available, will assist in ensuring that, before induction, denitrogenation has occurred ($F_I O_2 \approx F_E O_2$ [expired oxygen concentration]). $F_E O_2 > 90\%$ is achieved after approximately 3 minutes of regular tidal volume breathing, provided there is a good mask fit. The PaO_2 achieved after 3 minutes is higher than that achieved after five deep breaths in a cooperative patient. Unfortunately, the trauma patient may be too unstable to afford adequate denitrogenation or may not be able to cooperate because of central nervous system (CNS) injuries or severe distress. After intubation, gas analysis (capnography) of patients with an adequate circulation confirms whether the trachea has been accessed successfully. Capnography may be noncontributory in patients with circulatory collapse. Single-use CO_2 detectors may be used when an ED does not provide capnography. If neither is available, an esophageal bulb detector, a self-inflating bulb connected compressed to the endotracheal tube, may provide further guidance as to the location of the tip of the endotracheal tube. The principle behind the esophageal bulb detector is that the walls of the esophagus will be aspirated into the distal openings of the endotracheal tube by the negative pressure generated by the compressed bulb, thereby forming an effective seal. With an esophageal intubation, the bulb, therefore, would stay collapsed.

Airway Equipment

A typical setup includes a BVM device with supplemental O_2 , at least two laryngoscope handles, and standard straight (Miller) and curved (MacIntosh) blades. We recommend the availability of size 4 MacIntosh blades, because they can be used in smaller as well as larger mouths. Other essential equipment includes the appropriate-sized endotracheal tube plus smaller sized tubes (because patients with tracheal stenosis may be encountered). A stylet, oral and nasopharyngeal airways, lubricant, and suction should be checked and ready to use at all times. The batteries in the laryngoscope handles and the light bulbs on the blades must be checked before they are needed. Other backup equipment, dependent on local preference, should be available and may include the Combitube, fiberoptic bronchoscope, cricothyrotomy kit, jet ventilation kit powered by wall oxygen (at 50 psi) or an E cylinder, laryngeal-mask airway (LMA), and lighted stylet. The intubating laryngeal mask was introduced in the United States as a modification of the classic LMA. In addition to serving as an elective or emergency ventilating device, it is designed to allow blind intubation and may be an excellent alternative in the traumatized patient when a cannot-intubate/cannot-ventilate situation occurs.⁷ Use of this device in patients with a cervical spine fracture may have the additional advantage that neck movement is kept at a minimum. Avoidance of dan-

gerous hyperextension of the occipitoatlantoaxial complex with the intubating LMA has been verified by lateral cervical spine fluoroscopy during intubation in two patients with cervical spine fractures.⁸

Before the initial administration of drugs, the operator must have clearly considered backup plans for securing the airway if direct laryngoscopy fails. The operator must also know whether anyone is available to provide assistance if the attempt at securing the airway fails. In the trauma center ED, this is usually not a concern because of the presence of trauma surgeons.

Personal protective gear such as gloves, mask, gown, and eye protection must be immediately available in the trauma rooms and must be used to prevent transmission of hepatitis, human immunodeficiency virus, and other infectious agents.

Drugs and Technique

What agents should be used for trauma patients? To some extent the answer is related to the agents with which the operator is most familiar and comfortable. With very few exceptions (see later discussion), the most appropriate drugs for induction and neuromuscular blockade are those whose pharmacokinetic and pharmacodynamic profiles are best known by the operator.

Induction Agents

Propofol This alkyl phenol is an intravenous (IV) anesthetic agent unrelated to barbiturates, steroids, or imidazole agents.⁹ One of the most unique aspects of propofol is its rapid-onset/rapid-offset characteristics. The dose of propofol needed to induce general anesthesia in 95% of healthy, unpremedicated patients is 2.5 mg/kg; the induction time (onset) will range between 22 and 125 seconds. After a single bolus, the drug will have an offset time of about 5 to 10 minutes. The offset time is so short because the drug redistributes after a bolus injection into muscle, fat, and other poorly perfused tissues. Thus, once the drug bolus has been administered, redistribution occurs rapidly from the central compartment (i.e., CNS) into the peripheral compartment (e.g., muscle, fat) and the patient rapidly awakens. A 30% decrease in the systemic arterial pressure is commonly seen on IV injection of the drug in healthy patients. This is related to vasodilation rather than negative inotropic action of the drug. The drug does not cause arrhythmias or ischemia. After the injection of 2 mg/kg, respiratory depression may be so profound as to cause apnea for about 30 seconds. Tidal volume, minute volume, functional residual capacity, and respiratory response to CO₂ or hypoxia are blunted. Opioids augment each of these effects. Neither hepatic nor renal function is impaired by propofol. Intracranial pressure, cerebral metabolic rate, and

cerebral blood flow appear to be decreased by the drug. In the presence of hypovolemia, the capability of a standard dose of propofol to reduce systemic vascular resistance may produce significant hypotension. Depending on the extent of suspected hypovolemia, we, therefore, recommend a reduction of the induction dose of propofol from 1–2 mg/kg to 0.5 mg/kg or less. No induction dose may be safe in the severely hypotensive patient.

Etomidate This imidazole derivative is an IV anesthetic agent unrelated to barbiturates, steroids, or phenolic agents.¹⁰ The only formulation of etomidate available in the United States is a solution of 2 mg/ml in a 35% propylene glycol vehicle. While the formulation is stable, it should not be diluted or mixed with other drugs. Only the (–) stereoisomer has hypnotic activity. Like propofol, etomidate has rapid-onset/rapid-offset characteristics. The dose of etomidate needed to induce general anesthesia in 95% of healthy, unpremedicated patients is about 0.3 mg/kg; the induction time (onset) is usually within one arm-brain circulation, less than 60 seconds. After a single bolus, and assuming no other drugs have been administered, etomidate will have an offset time of about 5 minutes.

After a bolus injection, redistribution occurs into muscle, fat, and other relatively poorly perfused tissues. Thus, after a bolus is injected, redistribution occurs rapidly from the central compartment into the peripheral compartment, and the patient rapidly awakens. After an induction dose of etomidate, cerebral blood flow (CBF), cerebral metabolic rate for oxygen (CMRO₂), and intracranial pressure are significantly decreased. Cerebral perfusion pressure (CPP) is not altered. The myoclonic movements commonly seen after induction doses of etomidate are not associated with electroencephalographic evidence of seizure activity; however, seizures have been reported after use of etomidate.

In general, an approximate 10% decrease in the systemic arterial pressure may be seen in any patient going from the awake to the unconscious state, simply because the change in resting sympathetic tone noted as one progresses from wakefulness to unconsciousness. After an induction dose of 0.3 mg/kg, a slight increase in cardiac index, and a slight decrease in heart rate, arterial blood pressure, and systemic vascular resistance are noted. The coronary vascular resistance is decreased, and the coronary blood flow is increased by about 19%. In patients with mild hypovolemia, etomidate causes less hypotension than thiopental or propofol. Myocardial function, as measured by dP/dT, appears to be unimpaired after etomidate. Patients with valvular heart disease showed a 10–20% decrease in systemic arterial pressure, pulmonary artery pressure, and pulmonary artery wedge pressure (PAWP) with no alteration in central venous pressure, heart rate, or electrocardiogram.

After injection of 0.3 mg/kg ideal body weight, etomidate causes a brief period of hyperventilation, with a small increase in both tidal volume

and respiratory rate; this is followed by a short-lived period of respiratory depression or apnea. Apnea with etomidate is less common than is apnea after an induction dose of propofol or thiopental. Apnea becomes more common in the elderly and in patients pretreated with opioids. Bronchomotor tone is not significantly affected by etomidate.

No significant hepatic or renal impairments are caused by etomidate. Of minor significance, however, etomidate impairs both 17α - and 11β -hydroxylase and by these mechanisms decreases cortisol, 17-hydroxyprogesterone, aldosterone, and corticosterone production. Although this effect is seen after even a single dose of etomidate, it is only clinically relevant with continuous infusion of the agent. In fact, Wagner and colleagues¹¹ reported patient deaths resulting from continuous infusion of etomidate. For this reason, we use this agent only for induction. The induction dose of etomidate is 0.25 to 0.5 mg/kg ideal body weight IV (about 20 mg in a normal sized adult).

Ketamine This arylcycloalkylamine is an intravenous anesthetic agent unrelated to barbiturates, steroids, or phenolic agents.¹⁰ Agents of this class induce a state of sedation, immobility, amnesia (although the profundity of the amnesia varies), and marked analgesia. The term *dissociative anesthesia* comes from the feeling of strong dissociation from the environment that patients experience when given this agent. The formulations of ketamine available in the United States are solutions of 10, 50, and 100 mg/ml in an aqueous acidic (pH 3.5–5.5) solution containing a preservative. Although the formulation is stable, it should not be mixed with alkaline solutions such as the barbiturates or with diazepam. Although the (+) stereoisomer is three to four times as potent as the (–) isomer, ketamine is marketed as a racemic mixture; the (–) isomer, in addition to being less potent, has more clinically important side effects, including the disturbing emergence reaction.

Ketamine has rapid-onset/rapid-offset characteristics. Unlike the barbiturates, benzodiazepines, and etomidate, the mechanism of the action of ketamine is not through the modulation of γ -aminobutyric acid transmission. Rather, it is thought that ketamine acts as an antagonist to CNS muscarinic receptors and as an agonist to opioid receptors. The primary site of action of ketamine appears to be the thalamoneocortical projection system; it depresses neuronal function in parts of the cortex and thalamus while stimulating parts of the limbic system, including the hippocampus.¹² This is thought to create functional disorganization of nonspecific pathways in the midbrain and thalamic areas.

The IV dose of ketamine needed to induce general anesthesia in healthy, unpremedicated patients is between 1 and 3 mg/kg ideal body weight, injected over 1 minute; the induction time is usually within one arm-brain circulation, less than 60 seconds. A sense of dissociation is usually evident within 15 seconds, and consciousness is lost within about

30 seconds. After a single bolus, and assuming no other drugs have been administered, ketamine will have an offset time of 10 to 15 minutes for unconsciousness, about 40 minutes for analgesia, and as long as 1 to 2 hours for amnesia. It is important to remember that the amnestic properties of ketamine are variable and unreliable. A similar induction profile, with somewhat slower onset, may be seen with intramuscular injection when between 3 and 6 mg/kg ideal body weight is used. After an induction dose of ketamine, CBF, CMRO₂, intracranial pressure, CPP, and intraocular pressure are significantly increased. Thus, ketamine has no role in the treatment of patients with potential or actual intracranial disease. Muscle tone may be increased, and purposeless movements occur relatively commonly; on occasion, violent irrational responses to stimuli are observed. After an induction dose of 0.5 to 3 mg/kg ideal body weight, a 25% increase in arterial blood pressure is noted as well as lesser increases in heart rate and cardiac output. In vitro, ketamine is a negative inotropic agent; the augmentation in hemodynamic indices noted in vivo are related to the increased sympathetic activity. In patients whose sympathetic nervous system is maximally stimulated, induction with ketamine may result in hypotension. Ketamine is antiarrhythmogenic and counteracts both epinephrine- and digitalis-induced arrhythmias.

Lower doses of ketamine cause few significant alterations in the respiratory system. Pharyngeal and laryngeal reflexes are intact and, although the cough response is somewhat blunted, airway obstruction does not occur. Mild respiratory depression is occasionally seen in the elderly and in those patients in whom the drug is rapidly pushed; apnea is rare. Ketamine, because of its sympathomimetic effect and direct effect on smooth muscle, is often effective in reducing bronchospasm.¹³ The response curves to hypercarbia and hypoxia are not seriously affected after ketamine administration. The agent does act as a sialagogue, so an anticholinergic, such as nebulized or intravenous glycopyrrolate, may be needed before administering the agent to prevent excess secretions from developing in the airway.

No significant hepatic or renal impairments are caused by ketamine. If hypotension occurs, decreased hepatic blood flow will follow. An induction dose of 0.5 to 3 mg/kg ideal body weight intravenously (3–6 mg/kg intramuscularly) is usually appropriate. Emergence dysphoria seen with ketamine can be reduced through the use of a small dose of benzodiazepine after induction, provided the patient can tolerate this hemodynamically.

Scopolamine This belladonna alkaloid is a classically used amnestic agent, indicated in those rare cases when hemodynamic stability is so tenuous that even etomidate is considered problematic. Scopolamine, like atropine, is a competitive inhibitor of acetylcholine and other muscarinic agonists. They compete for a common binding site on the muscarinic

receptor. At clinically relevant doses, 0.005 mg/kg ideal body weight (about 0.3–0.5 mg in a normal individual) IV, the drug will produce anterograde and retrograde amnesia. Additional effects of scopolamine at this dose range include its antisialagogue properties and a minimal increase in heart rate. Onset is very rapid. Additions of benzodiazepines and narcotics increase the amnestic properties of scopolamine.

Midazolam This is one of a family of benzodiazepines used in the ED as a sedative agent. In the ED, midazolam is frequently used both as an anesthetic induction agent and for conscious sedation. Of the three commonly used benzodiazepines, diazepam, lorazepam, and midazolam, the latter is the most lipid soluble. The lipophilicity of midazolam accounts both for its rapid onset of action and large volume of distribution. The dose of midazolam needed intravenously to induce general anesthesia in healthy, unpremedicated patients is between 0.15 and 0.3 mg/kg ideal body weight; the induction time is usually within 30 to 60 seconds. After a single bolus, and assuming no other drugs have been administered, midazolam will have an offset time of 6 to 15 minutes for unconsciousness and produce possible amnesia for as long as 1 to 2 hours. Midazolam has no analgesic properties and thus must be used with an analgesic when a painful procedure is to be performed. A decrease in CBF and $CMRO_2$ ¹⁴ and an increase in the seizure threshold characterize the CNS effects of midazolam. Midazolam also provides some protection, although less than that seen with sodium thiopental (STP) (Pentothal), in the presence of cerebral hypoxia. After a dose of 0.15 mg/kg ideal body weight, one will consistently see a decrement in systolic (5%) and diastolic (10%) blood pressure. Additionally, the heart rate increases by approximately 18%. These changes are similar to those seen in patients with coronary artery disease administered 0.2 to 0.3 mg/kg midazolam. Other changes in cardiovascular state seen with midazolam include an approximate 15–33% decrement in systemic vascular resistance. The vasodilatory properties of midazolam result in a decrement in PAWP and augmentation in cardiac index in those patients with indices compatible with significant congestive cardiomyopathy; the induced alterations in hemodynamics move the values toward normal. Mean arterial pressure and central venous pressure are decreased irrespective of left ventricular function. The respiratory effects of small doses of midazolam are minimal. Midazolam produces some respiratory depression, with a decrement in the ventilatory response to CO_2 . Unlike the right-shifted CO_2 response curve seen with opioids, the curve is merely flattened after midazolam administration. The effect appears to be related to dose given and the speed of injection. Patients previously medicated with opioids are more likely to become apneic. Dosing of midazolam depends on the effect desired. For sedation, intermittent doses of 0.5 to 1 mg IV may be administered to a normal sized adult. Alternatively, the agent may be given as a continuous infusion, without a

loading dose, at a rate of 0.5 to 1 $\mu\text{g}/\text{kg}/\text{min}$. An anesthetic induction dose of midazolam ranges from 0.1 to 0.3 mg/kg ideal body weight; thereafter, if a maintenance dose is required, it will approximate 1 $\mu\text{g}/\text{kg}/\text{min}$.

Sodium Thiopental Sodium thiopental (STP) is the prototypic fast-on, fast-off barbiturate induction agent.¹⁵ The redistribution half-life, $T_{1/2\alpha}$, for STP is 5 to 8 minutes, whereas the $T_{1/2\beta}$ ranges from 5 to 17 hours. The IV dose of STP needed to induce general anesthesia in healthy, unpremedicated patients is between 1 and 4 mg/kg ideal body weight; the induction time is usually within 30 to 60 seconds. STP has no analgesic properties. Indeed, STP is said to have an antinociceptive effect, making a stimulus more painful. Clinical signs of hyperalgesia include tearing, tachycardia, hypertension, diaphoresis, and tachypnea.

STP causes venodilation and is negative inotropic. The agent causes a centralized respiratory depression, with decreased responsiveness to hypoxia and hypercarbia. There are no significant effects on the kidney or liver. Patients with preexisting liver disease and hypoproteinemia tend to have a higher fraction of nonbound (hence active) STP; in these patients, a reduced dose should be administered. CBF and intracranial pressure, as well as the cerebral metabolic rates for oxygen and glucose, are decreased after administration of STP. Finally, in elderly patients, because of increased circulation time, the onset of the drug tends to be delayed. In this situation, the clinician is wise to allow a few extra seconds for the drug to achieve its effect and avoid administering an extra, unnecessary dose. Because of its potent negative effects on cardiac contractility and systemic vascular resistance, normal doses of this agent should be used only in the ED in patients with stable hemodynamics. In patients with a moderately tenuous hemodynamic status, 1 mg/kg may be the appropriate dose. When administered to patients with suspected increased intracranial pressure, especially if hypovolemia is present, the blood pressure must be monitored closely and catecholamines or fluids may need to be coadministered to maintain a normal or high normal cerebral perfusion pressure.

Neuromuscular Blocking Agents

Rapid-Sequence Endotracheal Intubation The technique of rapid-sequence endotracheal intubation, in contrast to straightforward emergency intubation of the airway, implies that a muscle relaxant is administered with an anesthetic induction agent. When emergency intubations were compared with RSIs, complications were greater in number and severity in the group that did not receive neuromuscular blocking agents and included aspiration (15%), airway trauma (28%), and death (3%). None of these problems were observed in the rapid-sequence group.¹⁶ To

a certain extent, the neuromuscular blocking (NMB) agent used for RSI is less important than is the rationale behind the choice.

Succinylcholine (SUX), the prototypic depolarizing NMB agent, is the NMB agent of first choice in trauma patients because of its unsurpassed rapid onset. SUX evokes repetitive discharges at the motor nerve terminals, and transmission of these discharges results in visible fasciculations.¹⁷ The use of a nondepolarizing NMB agent before administering SUX is discouraged in trauma patients because these agents prolong the onset time of SUX, blunt fasciculations, and, therefore, may make it difficult to clinically time the onset of SUX paralysis, increase the dose requirement of SUX, and, furthermore, prolong paralysis, which may be detrimental in a cannot-ventilate/cannot-intubate scenario. The brevity of action of this agent is related to its rapid degradation by plasma pseudocholinesterase. The dose of SUX required for RSI is 0.5–1 mg/kg ideal body weight; clinical duration is 5–10 minutes in normal individuals, and full recovery may be expected in 12–15 minutes. The lower dose range should be used if there are any concerns with the airway maintenance, because recovery to a spontaneously breathing patient is then theoretically shorter. The use of a muscle relaxant to facilitate intubation is, of course, discouraged if there are serious doubts as to the feasibility of laryngoscopic endotracheal intubation. In this case, alternate avenues to airway control rather than an RSI should be sought. In the presence of a “defasciculating” dose of a nondepolarizing NMB agent, 0.04 mg/kg ideal body weight d-tubocurarine (about 3 mg in a 70-kg individual); or pancuronium 0.02 mg/kg ideal body weight (about 1 mg in a 70-kg individual), the dose of SUX will need to be increased to approximately 1.5 mg/kg.

The potential cardiovascular effects include occasional bradycardia (more likely encountered in children) and more likely seen after a second dose. Bradycardia is related to muscarinic acetylcholine receptor stimulation and may be prevented by pretreating with atropine. A nodal rhythm or ventricular arrhythmias may also be seen. Hyperkalemia after SUX administration is not a concern during the first day of trauma unless the patient has a history of CNS disease, malignant hyperthermia, renal failure, or extensive muscle damage. Late but clinically relevant hyperkalemia may be seen in patients treated with SUX after burns, trauma, nerve damage/neuromuscular disease, closed head injury, and intra-abdominal infections. This effect may be seen from 1 week (burns, trauma, intra-abdominal infection) to within 6 months (nerve damage/neuromuscular disease) after injury.

If an anesthesiologist chooses not to use SUX as muscle relaxant to intubate a trauma victim (e.g., because of concern of hyperkalemia, elevation of intragastric pressure, open eye injury, SUX muscle pain), there is a wide selection of nondepolarizing muscle relaxants. Three nondepolarizing muscle relaxants are devoid of cardiovascular side effects: cisatracurium, vecuronium, and rocuronium. Rapacuronium, which has been

Table 1. *An Approach to the Airway*

1. Rationale for intubation
Hypoventilation
Hypoxia
Shock
Altered mental status (Glasgow Coma Scale Score <9)
Noncooperative or intoxicated patient requiring rapid work-up
2. Preoxygenation
Ensure, if possible, via $FE_{O_2} \approx FI_{O_2}$ (>90% if time permits)
$SpO_2 \geq 97\%$
3. Induction
STP 0.5 to 4 mg/kg or etomidate 0.25 to 0.5 mg/kg
Cricoid pressure to prevent passive aspiration as soon as drug is pushed
When patient becomes apneic, ensure ability to ventilate using BVM device
4. NMB agent administration
After ensuring ability to ventilate with BVM device and maintaining cricoid pressure, SUX 1 mg/kg without defasciculation
Perform DL after fasciculation (SUX) or in 60 seconds with a nondepolarizing muscle relaxant
5. If successful, ensure appropriate placement
Visualize ET tube pass through vocal cords
Chest movement with ventilation
Mist in ET tube on exhalation
End-tidal CO_2
Esophageal bulb detector
6. If unsuccessful
Maintain cricoid pressure
Reoxygenate with BVM device
Perform second DL
7. If unsuccessful
Maintain cricoid pressure
Oxygenate with BVM device
Initiate call for assistance (if available)
Initiate back-up plan

FE_{O_2} = fraction of expired oxygen; FI_{O_2} = fraction of inspired oxygen; SpO_2 = oxygen saturation measured by pulse oximetry; STP = sodium thiopental; BVM = bag-valve-mask; NMB = neuromuscular blocking; SUX = succinylcholine; DL = direct laryngoscopy; ET = endotracheal; CO_2 = carbon dioxide.

introduced in the United States, may be a useful alternative to SUX because of its rapid onset and offset with induction.

Rapacuronium This is an aminosteroidal drug with the fastest onset of action of nondepolarizing muscle relaxants. It further differs from other nondepolarizing muscle relaxants in that it can be immediately antagonized with neostigmine and then has a duration similarly short as

Table 2. Summary of Induction and NMB Agents for RSI

Drug	Dose	Onset	Offset	T _{1/2α}	T _{1/2β}
Propofol					
Infusion	1–2 mg/kg	22–125 seconds	5–10 minutes	2–2.3 minutes	29–44 minutes
Etomidate	10–100 µg/kg/minutes				
Infusion	0.25–.5 mg/kg	<60 seconds	5 minutes	2.7 min 2.9 ± 1.1 hour	
Ketamine	1–3 mg/kg	<60 seconds	10–15 minutes	17 minutes	3 hours
Infusion	0.5–3 mg/kg/hour				79 minutes
Scopolamine	0.005 mg/kg	Rapid	30 minutes		
Midazolam	0.1–.3 mg/kg	30–60 seconds	6–15 minutes	6–15 minutes	1.7–4 hours
Infusion	0.5–1 µg/kg/min				
Thiopental	1–4 mg/kg	30–60 seconds	10–15 minutes	5–8 minutes	5–17 hours
			NMB Agents		
Succinylcholine	1–1.5 mg/kg	30–60 seconds	5–10 minutes		
Rapacuronium	1.5 mg/kg	50 seconds	10 minutes		
Cisatracurium	0.4 mg/kg	60–90 seconds	75–100 minutes		
Vecuronium	0.3 mg/kg	60–90 seconds	>120 minutes		
Rocuronium	0.9–1.2 mg/kg	60 seconds	>60 minutes		

Table 3. Summary of Intravenous Induction Agents and Clinical Scenarios

Indication	Agents*
Cardiac arrest, GCS score <5	Intubate without agents
Hemodynamically stable patient; "feasible" airway	STP Propofol Ketamine Midazolam
Hemodynamically stable patient; potentially difficulty airway	Ketamine STP Propofol
Hemodynamically unstable patient	Ketamine Etomidate Scopolamine Midazolam (0.05–0.1 mg/kg) Low-dose STP (0.5 mg/kg) Low-dose propofol (0.25 mg/kg)
CHI, hemodynamic instability	Low-dose STP Low-dose propofol Scopolamine
CHI, hemodynamic stability	STP Propofol Midazolam

*Listed in order of preference. GCS = Glasgow Coma Scale; CHI = closed head injury; STP = sodium thiopental.

SUX. When 1.5 mg/kg rapacuronium (1.5 times its ED₉₀) were compared with SUX and intubation was started 50 seconds later, clinicians unaware of the drug used judged intubating conditions as excellent or good in 89.4% of patients after rapacuronium and in 97% after SUX. After intubation, the maximum increase in heart rate averaged 23.1% after rapacuronium but only 9.4% after SUX. Bronchospasm and increased airway pressure were observed in 10.7% of patients given rapacuronium, which is approximately twice as frequent as in patients who received SUX. The authors concluded that after RSI of anesthesia in adults, clinically acceptable intubating conditions were achieved less frequently after rapacuronium 1.5 mg/kg than after SUX 1 mg/kg. They further concluded that higher doses may be necessary to achieve intubation within 60 seconds under intubating conditions similar to those after SUX.¹⁸

Cisatracurium This is a highly potent nondepolarizing muscle relaxant. It releases no histamine and is 80% degraded by Hofmann elimination. This is a purely chemical process at physiological pH and temperature in which molecular fragmentation occurs. Ester hydrolysis may also occur. The onset of the drug after an intubating dose of 0.4 mg/kg ideal body weight (eight times the ED₉₅) is usually within 60 to 90 seconds.

Clinical duration at this dose is 75–100 minutes and is not altered by renal failure.

Vecuronium At a dose of 0.3 mg/kg, vecuronium bromide has an onset within 60 to 90 seconds. The clinical duration at this dose, however, is >120 minutes.

Rocuronium Even though less potent than vecuronium, rocuronium at a dose of 0.9–1.2 mg/kg has a fast onset of paralysis. The clinical duration at such an “almost rapid-sequence dose,” however, is also >60 minutes.

Summary

An approach to the airway is addressed in Table 1. A summary of induction/NMB agents and doses is given on Table 2; indications for the different agents are noted on Table 3. The central pharmacological issue is not that any one drug is universally preferred over another. Rather, it is key that one develop a thoughtful rationale for the drugs used, and a plan to get out of trouble if one is suddenly in the sinking situation of a patient with a difficult airway who cannot breathe on his or her own. The backup plan might involve the use of BVM ventilation, blind digital intubation, fiberoptic bronchoscope-aided intubation, retrograde techniques, light wand intubation, laryngeal mask airway techniques, posterior pharyngeal endotracheal tube placement ventilation, or a surgical airway. Most of these approaches are reviewed elsewhere.¹⁹

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