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REVIEW ARTICLE

Rapid sequence intubation

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Establishing an airway is a stressful, high-risk maneuver for a physician. The rapid sequence intubation procedure is used to minimize complications, achieve a high success rate, and reduce stress. The sequence involves the following 7 steps: 1) planning and preparation, 2) preoxygenation, 3) pretreatment, 4) sedation and neuromuscular relaxation, 5) protection and positioning, 6) tube placement inside the trachea with proof, and 7) postintubation management. This review describes the steps and explains how to carry them out. [Emergencias 2012;24:397-409]

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Introduction

Rapid sequence intubation (RSI) involves the administration of an induction agent immediately followed by a neuromuscular blocking agent (virtually simultaneously) as well as other procedures in order to facilitate intubation in critically ill patients and to minimize the risk of vomiting and aspiration[1,2]. RSI is indicated in serious cases requiring immediate isolation of the airway (severe multiple injury with compromised airway, decreased level of consciousness, severe dyspnea, etc.) where it has been shown to have a high success rate in final intubation[3]. Furthermore, the use of RSI reduces the level of operator stress and uncertainty[4].

It is often applied in surgery, especially emergency interventions, and when orotracheal intubation is required in both pre-hospital and hospital emergencies. The emergency physician must therefore master this procedure to gain access and isolate the airway with minimal risk of complications[5-7].

Process of rapid sequence intubation

RSI usually consists of seven steps: 1) planning and preparation, 2) pre-oxygenation, 3) pre-treatment, 4) sedation (induction) with neuromuscular block, 5) protection and patient position, 6) checking the endotracheal tube and 7) post-intubation management. These steps can be modified according to the characteristics of the emergency and the peculiarities of each case[1,5].

1. Planning and Preparation

Once the indication for RSI has been established (Table 1), the physician responsible should ensure he/she has all the necessary equipment in working order: source of oxygen, suction system, auto-inflatable bag, laryngoscope, endotracheal tubes, equipment for difficult intubation (laryngeal mask, cricothyrotomy device, etc.), resuscitation kit, pharmacological drugs and patient monitoring equipment (oxygen saturation, heart rate, blood pressure and ECG recording). The mnemonic "SOAPME" is used to help remember all the necessary equipment for intubation: Suction, Oxygen, Airway, Pharmacology, Monitoring, Equipment[1].

At this stage, a quick anatomical examination is required to gauge whether intubation and/or ventilation is likely to be difficult or not[5]. There are three very useful methods, known in the medical literature by mnemonics that help as-

Table 1. Indications for rapid sequence intubation

- Recent or unknown food intake.
- Intestinal obstruction, lower esophageal sphincter incompetence, ileus obstruction.
- Pregnancy.
- Obesity.
- Central nervous system depression (low level of consciousness/coma).
- Multiple trauma with compromised airway.
- Severe respiratory failure.
- Major burns.

sess potentially difficult intubation: MOANS, LEMON (described below) and TMD (thyromental distance). The latter seems to be the least effective predictor of difficult intubation[6]. There are another two for predicting cricothyrotomy (SHORT) and for positioning supraglottic devices (RODS)[9], but these are not discussed in this review since they only refer to handling difficult intubation.

LEMON Method

1. Look externally: a brief look at the neck, mouth and internal airway. This should identify anatomical features suggesting difficult intubation such as morbid obesity, or macro- or micrognathic jaw, macroglossia, very large teeth, beard and facial or cervical trauma[10,11].

2. Evaluate: using the 3-3-2 rule, evaluate the degree of mouth opening by introducing three fingers between the upper and the lower teeth, 3 fingers between the chin and the start of the neck, and two fingers between the thyroid cartilage and floor of the jaw (dimensions suitable for intubation)[12,13] (Figure 1).

3. Mallampati: viewing the hypopharynx. This is performed with the patient sitting upright; he/she is asked to open the mouth and extend the tongue. A torch or light is used to visualize the hypopharynx. Upon observation, the airway is classified as one of 4 grades, with grades III and IV indicating difficult airway (Figure 2). In critically ill patients, sometimes supine and with a low level of consciousness, the hypopharynx may be displayed by manual mouth opening and extraction of the tongue, which makes the Mallampati maneuver more difficult and questionable in the critically ill[14].

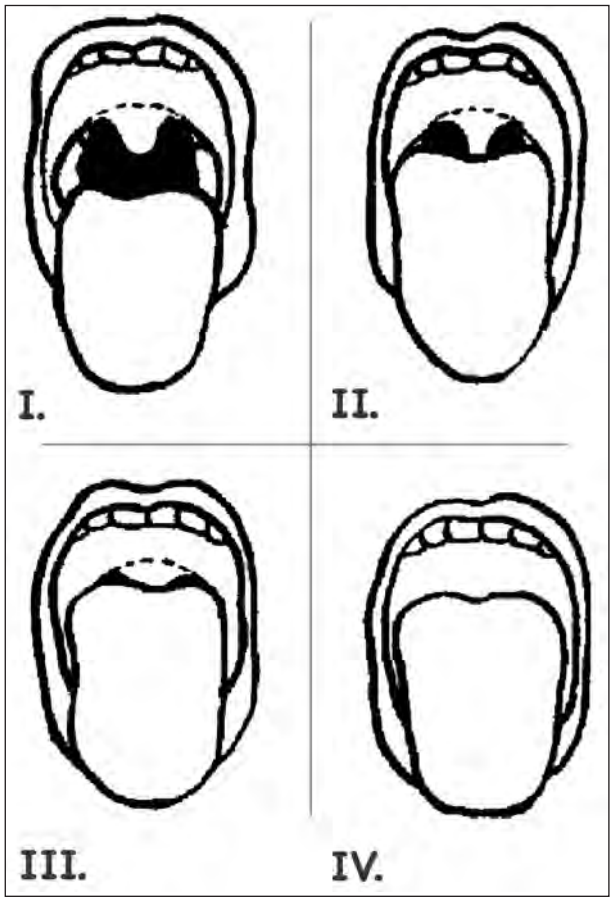

Figure 2. Mallampati classification.

4. Obstruction of the airway: presence of infection in the upper airway (peritonsillar abscess, epiglottitis, etc.), laryngeal masses or tumors, extrinsic compression, direct neck trauma, etc. that hinder or impede visualizing the lower airway.

5. Neck mobility: limited flexion and extension complicating head-neck alignment makes for difficult intubation: cervical arthritis, whiplash, etc.

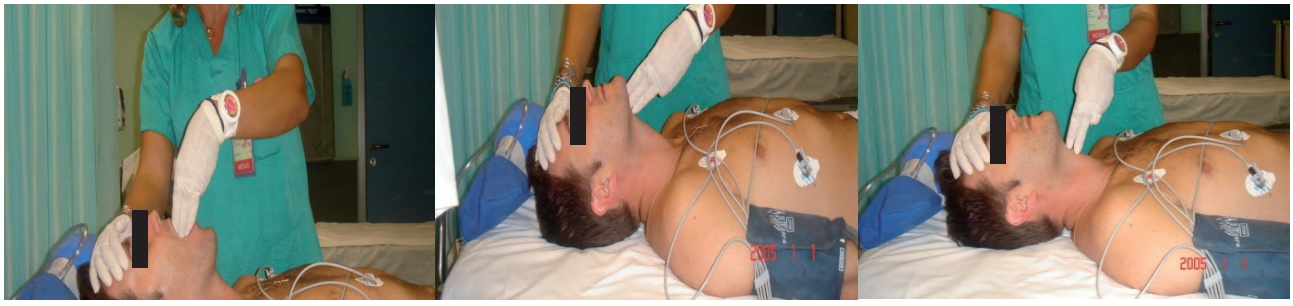

Figure 1. 3-3-2 evaluation.

For a patient who does not present external anomalies of the face or neck, the 3-3-2 rule applies, and for those with Mallampati grade I-II, no upper airway abnormalities or problems of cervical mobility, a priori, easy access for intubation is predicted[15].

MOANS

Difficult bag-mask ventilation may be predicted by the presence of certain features[16], identified by the mnemonic MOANS. The M represents "mask" to indicate difficult mask seal; O refers to "obesity" and "obstruction"; the A refers to aged patients (older than 55 years); the N refers to "no teeth," and S refers to "stiff lungs or stiff chest wall." In general, these predictive methods are highly accurate, with a sensitivity of 86.6% and specificity 96.0%[15]. After completing the examination, if difficult intubation is predicted, the necessary resources must be prepared in addition to standard intubation material.

2. Pre-oxygenation

This must be performed simultaneously with the planning and preparation steps. It involves administering oxygen via a reservoir mask (FiO_2 = 1) for 5 minutes to replace functional residual capacity nitrogen with oxygen (washing-out); this allows maintaining apnea in a patient for 3-8 minutes without hypoxemia[17,18]. Desaturation time is directly related with weight: a healthy 70 kg patient will maintain oxygen saturation ($SatO_2$) over 90% for 8 minutes, an obese patient weighing 127 kg for 3 minutes and a healthy child of 10 kg less than 4 minutes[19].

There are measures such as raising the head 25° during pre-oxygenation which can lengthen saturation time in obese patients, or the use of CPAP at 7.5-10 cm H_2O, which increases saturation time by 1 minute[20-22].

Importantly, ventilation should not be performed manually with a mask and self-inflatable bag, since it increases gastric pressure and the possibility of regurgitation and/or vomiting. However, it is sometimes necessary to pre-oxygenate with manual ventilation in patients with $SatO_2$ less than 90% and not yet intubated when it is necessary to apply high oxygen flow and the cricoids or Sellick technique to prevent regurgitation[23].

3. Pretreatment

This involves administering drugs before the induction-relaxation stage in order to mitigate the adverse adverse effects of orotracheal intubation (hypotension, bradycardia or tachycardia, increased intracranial pressure and airway resistance). Pretreatment has its benefits and risks associated with the drug used. Only those drugs with proven benefit are currently used. Those unsupported by clear evidence are avoided, such as depolarizing neuromuscular blockers used previously.

The drugs used in RSI pre-treatment are atropine, lidocaine and short-acting opiates (the most widely used is fentanyl). They are generally not always used. The current consensus[24] suggests the use of these drugs in the following clinical situations:

– Severe head injury with signs of intracranial hypertension: lidocaine[25] and fentanyl[26-29].

– Dissection vascular, ischemic heart disease and with hypertension and increased heart rate: fentanyl[30-32].

– Acute asthma or severe bronchospasm: lidocaine[24] (although some recent studies have questioned this[33,34]).

– Children under 1 year of age, under-5 years receiving succinylcholine, those older than 5 years who need a second dose of succinylcholine, and bradycardia patients going to receive succinylcholine: atropine[35,36].

Pre-treatment, to be most effective, should be applied 3 minutes before induction. In an emergency that does not allow intubation delay, pretreatment may be applied sooner or skipped[24].

The characteristics of the drugs used in pretreatment are shown in Table 2.

4. Sedation with neuromuscular paralysis

In RSI, sedation and neuromuscular relaxation are induced simultaneously to produce unconsciousness and muscle relaxation to facilitate intubation and minimize the risk of aspiration. Currently the most widely used sedatives are: etomidate, ketamine, midazolam and propofol (Table 3), the choice depending on the specific clinical circumstance[41].

– **Etomidate:** This is a derivative of imidazole. It binds to GABA receptors and inhibits neuronal excitation causing sedation. It is the most widely used agent for RSI in the ED[42] due to its hemodynamic stability (it does not produce hypotension and histamine release) and the absence of absolute contraindications[43,44]. It is rapidly metabolized in the liver with neutral hemodynamic effects. Renal elimination is less than 2% of the active drug. Repeated boluses or continuous infusion are not recommended to maintain sedation[45].

Table 2. Characteristics of the drugs used in pre-treatment

- Lidocaine:
  - Presentation: lidocaine hydrochloride injection solution 1%/10 ml = 100 mg, 2%/10 ml = 200 mg, 5%/10 ml = 500 mg, 5%/50 ml = 2500 mg.
  - Mechanism of action: anti-arrhythmic class Ib.
  - RSI pretreatment dose: 1.5 mg/kg IV[5,24].
  - Onset of effect: iv bolus: 45-90 seconds. Duration: 10-20 minutes. Metabolism: 90% liver. Elimination (urine): 1-2 hours (increased in renal failure, congestive heart failure, shock).
  - Effect-indication as pretreatment in RSI: decreases airway resistance and decreases intracranial pressure, so it is indicated in patients with bronchospasm and disease with intracranial hypertension (epidural hematoma, cerebral hemorrhage, etc.)[5,24].
  - Adverse effects: mainly arrhythmia.
  - Precautions: Epilepsy. Sinus bradycardia. Heart failure. Hepatic and renal failure.
  - Absolute contraindications: Allergy. 2nd or 3rd degree AV block (without pacemaker).
- Fentanyl:
  - Presentation: Blister 0.150 mg/3 ml.
  - Mechanism of action: opioid derivative. Mu receptor agonist.
  - Dose RSI pre-treatment: 3 mcg/kg iv. slow bolus (30-60 seconds). In patients with moderate hemodynamic instability 1 ug/kg[37].
  - Onset of effect: less than 1 minute (highly liposoluble). Duration: 1-2 h. Metabolism: Hepatic. Elimination: Renal (no active metabolites). No histamine release: hemodynamic stability[31].
  - Effect-indication RSI pre-treatment: pain control attenuating neurovegetative responses to laryngoscopy, which increases pressure and heart rate. Associated with muscle relaxants, decreases intracranial pressure in patients with head injury and signs of intracranial hypertension[24].
  - Adverse effects: bradycardia, mild hypotension, respiratory depression and stiff chest, which is produced by the administration of rapid boluses and high doses (> 500 mg), resolved by administering succinylcholine[38].
  - Contraindications: Allergy. Shock.
- Atropine:
  - Presentation: Blister 1 mg/1 ml.
  - Mechanism of action: anti-cholinergic agent.
  - RSI pretreatment dose: 0.02 mg/kg iv. rapid bolus (minimum total dose: 0.1 mg-dose and maximum: 0.5 mg. Less than 0.1 mg associated with paradoxical bradycardia)[39].
  - Onset: 1 min. Maximum effect 2-4 minutes. Duration: infants < 2 years: 4-9 h. > 2 years: 2-4 h. Metabolism: liver. Elimination: Renal.
  - Effect-indication RSI pre-treatment: laryngoscopy in children produces bradycardia by vagal stimulation and use of succinylcholine increases respiratory secretions, atropine reduces these effects[39,40].
  - Adverse effects: rare, very high doses may produce arrhythmia.
  - Contraindications: Allergy. Glaucoma.

The most important adverse effects are: 1) "transient myoclonus" (not seizure) that some authors claim to prevent by prior administration of benzodiazepines[46], although in the context of RSI this is not necessary because of simultaneous administration of a muscle relaxant, 2) a priori, a pro-epileptogenic effect so it should be used with caution in epileptics, which should be controlled with the use of propofol or midazolam[43] for sedation maintenance and 3) corticoadrenal suppression, which lowers cortisol and aldosterone levels in the blood, especially in continuous infusion[47]. The current debate centers on the use of etomidate in septic shock, where corticoadrenal suppression in continuous infusion is associated with increased mortality. In patients with severe sepsis administered a single dose of etomidate to induce anesthesia there are sufficient data to affirm that it does not increase mortality, so it can be used in RSI patients with sepsis grave[48-52]. On empirical grounds, some authors recommend the administration of corticosteroids (hydrocortisone 100 mg intravenously) in the first 24 h in patients with severe sepsis and refractory hypotension when etomidate was used to induce anesthesia[53]. If in doubt about etomidate, an acceptable alternative is ketamine[54].

With respect to the use of etomidate in pediatric patients and corticoadrenal suppression, there are studies supporting its safety[55] and it has no absolute contraindications.

– **Ketamine:** along with etomidate, considered by some authors to be the main hypnotics for RSI in adults and pediatric patients[56,57]. Ketamine is a liposoluble derivative of phencyclidine with high hypnotic and analgesic potency: it acts on GABA and opioid receptors. It produces dissociative anesthesia characterized by catalepsy and amnesia, as well as being a powerful painkiller, while preserving spontaneous breathing and laryngeal reflexes. The patient after induction is rigid, with no voluntary mobility, no pain, open eyes (nystagmus) and amnesia of what happened. Ketamine acts on the cholinergic receptors. It releases catecholamines that trigger increased heart rate, increased cardiac contractility, increased mean blood pressure, increased cerebral blood flow, intracranial hypertension and bronchial smooth muscle relaxation producing bronchodilation. Ketamine also reduces the production of vascular nitric oxide which decreases its vasodilator effect. These characteristics make ketamine ideal for RSI induction in hypotensive patients or those in shock (excluding cardiogenic)

Table 3. Hypnotics used in rapid sequence intubation

Drug	Presentation	Dose	Time to onset	Duration	Indications	Adverse effects	Precautions	Contraindications
Etomidate	20 mg/10 ml	0,3 mg/kg	15-45 sg	3-12 min.	Hemodynamic instability.	Transient Myoclonus. Cortical suppression.	Epilepsy. Sepsis.	None.
Ketamine	500 mg/10 ml	1-2 mg/kg	30-60 sg	10-15 min.	Shock and severe hypotension. Severe asthma.	Tachycardia. Hypertension. Intracranial hypertension.	Schizophrenia. Ischemic cardiopathy. Intracranial hypertension.	Hypertension. Intracranial hypertension. Cardiogenic shock Cerebral hemorrhage.
Midazolam	15 mg/3 ml	0,2 mg/kg	30-60 sg	15-30 min.	Status epilepticus. (STABLE)	Hypotension. Decreased cardiac output. Tachyphylaxis.	Elderly. Kidney or liver failure.	Hypertension. Shock.
Propofol	200 mg/20 ml	1,5-2,5 mg/kg	15-45 sg	5-10 min.	Epilepsy. Intracranial hypertension. Asthma. Emergency arterial hypertension (STABLE)	Hypotension. Bradycardia. Low output. Anaphylaxis.	Aseptic manipulation.	Hypotension. Bradycardia. Shock.
Thiopental	500 mg/1000 mg powder	3-5 mg/kg	30-60 sg	5-30 min	Intracranial hypertension. Status epilepticus (STABLE)	Hypotension Bradycardia Bronchospasm Laryngospasm Anaphylaxis	Hypotension Kidney or liver failure.	Porphyria Shock. Bronchial asthma. Severe ischemic cardiopathy.

and severe asthma[46,58]. Its metabolism is hepatic and renal elimination of the active drug is below 4%[59].

Ketamine is indicated for anesthetic induction. It has some important considerations: First, awakening from anesthesia there may be delirium, agitation, visual, auditory and proprioceptive hallucinations and possible progression to delirium. It must therefore be used with caution in patients with psychiatric history as it may induce acute psychotic episodes. These effects are reduced significantly when used as a single dose and in combination with midazolam[60]. Second, ketamine improves myocardial contractility and cardiac output, resulting in increased oxygen demand which mean it should be used with caution in patients with coronary artery disease and the risk/benefit balance should be assessed due to the probability of ischemic myocardial events. Third, as mentioned, ketamine increases intracranial pressure and therefore should a priori not be used in patients with suspected brain injury and intracranial hypertension. Current evidence shows that ketamine mildly increases intracranial pressure in patients with TBI and especially if associated with fentanyl and/or midazolam. Ketamine can used with caution in patients with brain damage and intracranial hypertension with arterial hypo-or normotension, but is contraindicated if there is arterial hypertension[59].

Absolute contraindications of ketamine include cardiogenic shock, hypertensive emergencies and cerebral hemorrhage with intracranial hypertension and arterial hypertension[59,61].

– **Midazolam:** The best of the benzodiazepines for RSI[1]. It acts on GABA receptors causing sedation and amnesia, and no analgesic effect[44].

Midazolam acts at different levels[5,62]: 1) central nervous system (CNS), with anticonvulsant activity, and is an ideal agent for epileptic status[63] and does not modify intracranial pressure (ICP)[26], 2) it acts on bronchial smooth muscle, causing bronchodilation, so it is optimal for patients with severe asthma[34]; 3) at the cardiovascular level, upon intravenous bolus administration, it produces moderate hemodynamic alteration and cardiac output and mean arterial pressure by 10 to 25% secondary to vasodilation, which limits its use in hypotensive patients and/or shock[64], but if administered by continuous infusion these effects are mitigated and it is considered an optimal agent (as discussed below) for the maintenance of sedation and 4) it acts on the respiratory apparatus; at does higher than 0.1 mg/kg, respiratory depression occurs.

If midazolam is used, some precautions apply to hypovolemic patients, where blood volume should be restored and the induction dose reduced to 0.1 mg/kg. When used simultaneously with an opiate, synergism occurs so the dose should be reduced to the lower limit allowed in

both drugs to reduce adverse effects and maintain the same effect.

In elderly patients with renal and liver failure (which decreases the elimination of midazolam), reduced doses are recommended to avoid major adverse effects and tachyphylaxis (after 72 h of sedation it is necessary to increase the dose). Its hemodynamic effects and inter-individual variability limits its use as a single agent for induction in RSI with critical patients[5,44].

– **Propofol:** This is an alkylphenol which acts on CNS and GABA receptors, causing sedation and amnesia. It comes in a highly lipid emulsion which gives it great power and speed of diffusion to the brain (rapid unconsciousness when administered in intravenous bolus form), and its distribution is high which explains the rapid recovery from its effects. These characteristics make propofol the ideal sedative in the ED for emergency diagnostic and short-term therapeutic processes[65,66].

Propofol acts at multiple levels on the cardiovascular system; when administered in bolus form, it reduces systemic vascular resistance and produces myocardial depression causing hypotension (10% decrease in mean blood pressure), decreased cardiac output and bradycardia. It also reduces the sympathetic response to the act of intubation and aggravates its hemodynamic effects. So, a priori, propofol is an ideal agent for RSI in critically ill patients. If continuously infused, hemodynamic changes are minimal[5]. One study reported achieving similar intubation conditions and minimal hemodynamic changes with low-dose propofol induction (0.5 mg/kg) associated with rocuronium[67]. At the CNS level, it reduces blood flow and cerebral oxygen consumption with a decrease in ICP, and it is an acceptable agent in hemodynamically stable patients with severe head injuries[68,69]. Furthermore, it has anticonvulsant and antiemetic effects[70]. Propofol also acts on the respiratory system, causing bronchial smooth muscle relaxation and hence bronchodilation, and should be considered for induction in patients with severe asthma. It improves intubation conditions and facilitates direct laryngoscopy since it inhibits reaction to pharyngeal and laryngeal stimulation. This means that a muscle relaxant can be omitted if necessary[71]. Finally, it also lowers intraocular pressure[72].

The most common adverse effects of propofol are pain at the site of injection (reduced if the injection site is in the forearm and with 40 mg of 2% lidocaine added)[73]; the need for safe aseptic precautions since it is presented as a lipid emulsion and there is a risk of bacteria and fungi proliferation which has led to cases of sepsis[74].

– Propofol may induce anaphylaxis, hypertriglyceridemia, acute pancreatitis, lactic acidosis (if used in continuous infusion, etc.)[75].

– Despite its anticonvulsant effect[76], there have been cases of seizure[76] although not clinically significant in the context of RSI due to the simultaneous use of a neuromuscular blocking agent[77].

Propofol is not authorized for use in children under 3 years of age and in obstetrics, although it has been used with caution in real clinical practice[46]. If administered to adult patients by continuous infusion (due to the need for neurological reassessment and/or short sedation) at doses higher than 5 mg/kg/hr and during more than 48 hours, it can produce propofol infusion syndrome consisting of heart failure, metabolic acidosis, rhabdomyolysis and renal failure[78]. Continuous infusion is contraindicated in patients under 17 years of age[46].

– **Thiopental:** A barbiturate with great liposolubility that acts on GABA and CNS receptors causing sedation and hypnosis. At high doses it also has anticonvulsant properties[46].

In the cardiovascular system, it causes vasodilation and myocardial depression which results in severe hypotension at anesthetic induction doses, and is more pronounced in patients with unstable hemodynamics, so its use is discouraged in unstable or potentially unstable patients[44]. In the CNS, it produces a reduction of cerebral blood flow and metabolic demand and decreases ICP, so it is an ideal agent for patients with suspected or confirmed raised ICP as long as they are hemodynamically stable. Moreover, high doses suppress neuronal activity which means it has anticonvulsant properties. In the respiratory system, thiopental liberates histamine and can induce and/or exacerbate bronchospasm (so it is contraindicated in asthma patients) as well as laryngospasm[1]. Thiopental can suppress the recruitment, activation and activity of leukocytes responsible for immunosuppression, so it should not used in cases of sepsis.

The most common adverse effects are hypotension, bradycardia, bronchospasm, laryngospasm and local dermal necrosis if not injected intravenously. Contraindications are porphyria, asthma, shock and severe heart disease[44].

Due to its hemodynamic effects and the existence of other safer hypnotics, thiopental is currently less used than in the past[5,15]. The choice of hypnotic for induction in RSI depends on the clinical situation, the most common being:

– 1st scenario: severe head injury or suspected hemorrhagic stroke vs signs of intracranial hy-

pertension. Etomidate is recommended. If the patient presents severe hypotension, ketamine can be used but not when there is spontaneous cerebral hemorrhage. Midazolam and propofol are acceptable alternatives, but due to hypotension secondary to brain injury the clinical condition could worsen so low doses are recommended.

– 2nd scenario: status epilepticus. The first choice is midazolam. If the patient is hemodynamically unstable, etomidate is preferable, or alternatively propofol.

– 3rd scenario: severe bronchospasm. Ketamine or propofol are preferred in hemodynamically stable patients. Other valid options are midazolam and etomidate. If the patient is hemodynamically unstable, ketamine or etomidate are recommended.

– 4th scenario: cardiovascular disease (ischemic heart failure, aortic dissection, etc.). The first choice is etomidate, for its great hemodynamic stability.

– 5th scenario: shock (except cardiogenic shock). Etomidate and ketamine are recommended.

– Neuromuscular blockers (Table 3): These drugs act on the nerves and muscles, blocking cholinergic transmission and inhibiting body motions, resulting in muscle paralysis. Together with sedatives, they improve intubation conditions. Note that the inclusion of these drugs in RSI must be preceded by a pre-assessment of the airway 5 (LEMON and MOANS) and, if possible, direct visualization of the glottis (Cormack-Lehane grading system, Figure 3), although the latter can skipped by skilled and experienced staff. If the assessment indicates difficult intubation, the risk/benefit must be weighed before using these agents[24].

They are classified as:

– Depolarizing neuromuscular blockers (DPNMB) which are agonists of postsynaptic receptors of acetylcholine with prolonged action. They act by inhibiting acetylcholinesterase and cause higher concentrations of acetylcholine. This initial hyperstimulation occurs (which explains transient fasciculations) until it reaches a point where the phenomenon of desensitization occurs, resulting in muscle paralysis. The main agent representing this group is succinylcholine.

– Non-depolarizing neuromuscular blockers (NDPNMB) which are competitive antagonists (without extrinsic activity) of postsynaptic receptors. They occupy these receptors preventing acetylcholine binding and inhibiting contraction, resulting in muscular paralysis. The most widely used NDPNMB are atracurium, vecuronium, cisatracurium and rocuronium[79].

The ideal neuromuscular blocker for RSI must meet four requirements: a) rapid onset of action, b) brief recovery time, c) minimal hemodynamic effects d) absence of significant adverse effects6. There is no neuromuscular blocker that meets these criteria, but succinylcholine and rocuronium come closest[80].

1. Succinylcholine: an agent used over a century ago. In 1906 it was used in animals and until 1951 was not applied to humans[81] (Table 3). It is rapidly hydrolyzed in plasma by acetylcholinesterase. It virtually bypasses the liver, and less than 10% of the drug is excreted unchanged via the kidney. The effect is prolonged in patients with low levels of acetylcholinesterase: pregnancy, hypothyroidism, liver cirrhosis, malnutrition, burns and cancer[79]. The advantages of succinylcholine over NDPNMB is rapidity of onset, higher the degree of relaxation and very short action time, which makes an ideal agent for RSI. However, an important disadvantage with respect to NDPNMB is its adverse effects and contraindications[82] (see Table 4)[83,84].

Low doses of succinylcholine (0.3-0.6 mg/kg) for induction have been shown to achieve optimal conditions for intubation, with reduced fasciculations and apnea time. There is always the option of a repeat dose if intubation is unsuccessful, without hemodynamic implications and almost no serious adverse effects. But further studies are needed to confirm the optimal dose[85].

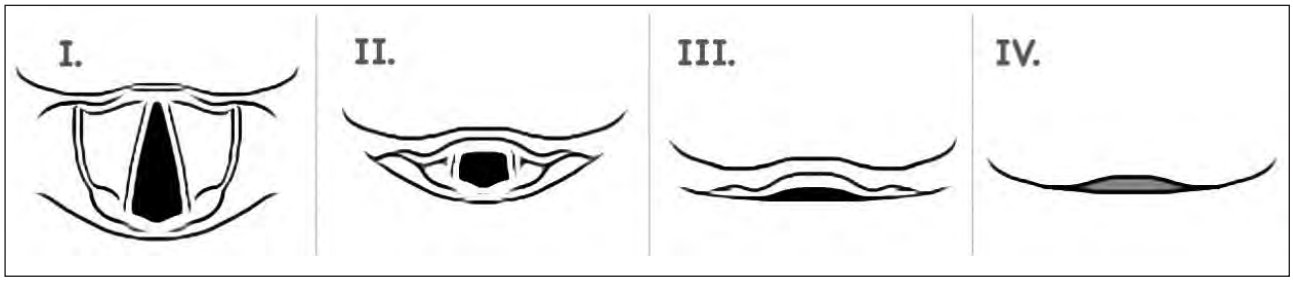

Figure 3. Cormack-Lehane grading system.

Relative contraindications:

a) Severe head injury, although fasciculations increase mild and transient ICP, some studies have observed that it does not affect cerebral perfusion and some authors recommend premedication with small doses of rocuronium (0.06 mg/kg) to mitigate fasciculations[86]; b) glaucoma and ocular surgery, where propofol can mitigate the decrease in intraocular pressure[87]; c) hypotension, d) acute or chronic renal failure[5] and e) in patients treated with statins for hypercholesterolemia, succinylcholine produces more fasciculations and higher concentrations of myoglobin, although still below renal toxicity threshold[88,89].

Absolute contraindications are: a) personal or family history of malignant hyperthermia; b) patients with severe hyperkalemia (changes in the ECG) and/or potentially high risk of severe hyperkalemia (more than 72 hours after a spinal cord injury or stroke or multiple trauma or severe burn, multiple sclerosis or any other disease with denervation injury, congenital myopathy, prolonged immobilization and sepsis); c) severe muscle trauma d) extreme bradycardia; e) allergy to succinylcholine[5,82-84].

2. Rocuronium: A NDPNMB formulated in rocuronium bromide. It is characterized by being dose-time dependent: the higher the dose the faster and longer its action. It is the fastest of all NDPNMB, which allows its use in RSI of critically ill patients[79]. It is eliminated by liver metabolism (it does not produce active metabolites) and feces (its effect is prolonged in renal failure) and 10% via the kidney in unchanged form (not prolonged in renal failure patients)[90]. It has no relevant adverse hemodynamic effects due to low direct release of histamine, mild vagolisis and sympathetic activation[79,82]. Its adverse effects are rare and not significant (mild hypotension, tachycardia, and urticaria) except that there are some reported cases of anaphylactic shock. It has no absolute contraindications, except for known allergy[79]. The advantages of rocuronium over succinylcholine for RSI are: similar intubation conditions, hemodynamic stability, infrequent adverse effects and lack of contraindications. The disadvantage is the effect lasts over 45 minutes, which a priori limits its use for expected difficult intubation[91].

Currently the agent of choice for RSI is succinylcholine[80,91] due to its speed of action, short duration and excellent intubation conditions, but it has important adverse effects and contraindications which have made some authors doubt whether it is optimal for critically ill patients, especially after the appearance of rocuronium in 1994 (currently 2nd choice, if succinylcholine is contraindicated). These authors argue that critical patients who need RSI are often hemodynamically unstable or potentially unstable, and suggest that severity worsens with the use of succinylcholine. They recommend the use of rocuronium as the NMB of choice for RSI since, at high doses, rocuronium acts just as rapidly, intubation condi-

Table 4. Neuromuscular blockers

Drug	Presentation	Dose	Time to onset	Duration	Adverse effects	Precautions	Contraindications
Succinylcholine	100 mg/2 ml or 200 mg/10 ml	1,5 mg/kg	30-60 sg	5-10 min.	Bradycardia. Hypotension. Cardiorespiratory arrest. Severe hyperkalemia (arrhythmia). Fasciculations. Trismus. Malignant hypothermia. Rhabdomyolysis (acute renal failure). Hypersalivation. Increase of intracranial, intraocular or gastric pressure. Anaphylaxis.	Pregnancy. Hypothyroidism. Liver cirrhosis. Malnutrition. Cancer.	*Relative:* Severe TBI, glaucoma, eye surgery, acute/chronic kidney failure treatment with statins. *Absolute:* Severe/probable hyperkalemia, Patients > 72 h with: CVA severe multiple trauma, prolonged immobilization, major burns, sepsis. Multiple sclerosis. Congenital myopathy.
Rocuronium	50 mg/5 ml .	0,9-1,2 mg/kg	45-60 sg	45-60 min.	Mild hypotension. Tachycardia. Anaphylactic shock (very rare).	Liver failure.	None

TBI: traumatic brain injury, CVA: cerebrovascular accident.

tions are similar and, most importantly, it has virtually no hemodynamic effect, no significant adverse effects and no relative or absolute contraindications[90,92].

Other authors defend succinylcholine as a first-choice NMB, claiming that its adverse effects are controllable. Furthermore the RSI induction dose of the alternative, rocuronium, has effects lasting more than 45 minutes which limits its use in patients with a high probability of difficult intubation since failure to achieve successful intubation means the patient is relaxed and the airway cannot be definitively isolated over 45 minutes with the attendant risks involved[93]. Since the advent of sugammadex (Table 5)[94,95] this situation is resolvable. It reverses the effects of rocuronium in 1-2 minutes after administration, faster than the spontaneous reversion of succinylcholine[96,97]. In conclusion, both options are safe and effective for RSI in the ED, but rocuronium would probably be preferred in most circumstances[98-100].

Table 5. Characteristics of sugammadex

- Presentation: 200 mg/2 ml, 500 mg/5 ml.
- Mechanism of action: binds to rocuronium-vecuronium forming a complex and decreases NDPNMB concentration in plasma without causing hemodynamic changes.
- RSI Dose: 16 mg/kg.
- Onset of action: immediate.
- Duration: 1.8 hours on average.
- Metabolism: no active metabolites.
- Elimination: Renal, < 5% unchanged.
- Indication: immediate reversal of neuromuscular blockade by rocuronium, vecuronium (16 mg/kg administered 3 minutes after administration of rocuronium at 1.2 mg/kg achieves reversal in 1.5 min).
- Adverse effects: urticaria and dysgeusia.
- Precautions: Not recommended in severe renal failure or in children < 2 years.
- Contraindications: None.

NDPNMB: non-depolarizing neuromuscular blocker, RSI: rapid sequence intubation.

5. Patient positioning, protection using Sellick's maneuver and optimal visualization of the airway using the BURP maneuver

In this phase the patient is positioned to facilitate intubation. Cricoid pressure may be applied (the Sellick maneuver) to prevent regurgitation during endotracheal intubation and optimal display of the airway may be achieved with the BURP maneuver.

The ideal position for intubation is called "sniffing the morning air" or "sipping English tea"[1], in which the neck is flexed and there is extension at the cranio-cervical (atlanto-axial) junction (except in the patient with suspected cervical spinal cord injury).This aligns the structures of the upper airway in the optimum position for laryngoscopy and provides the best view of the larynx and facilitates intubation[1]. This is facilitated by slightly raising the head of the bed or by use of a small pillow[5]. This position ensures correct alignment of the three axes (oral, pharynx and larynx) for optimal viewing of the glottis and facilitates intubation[1]. During direct laryngoscopy and visualization of the glottis, the operator can assess the probability of difficult intubation using the Cormack-Lehane classification, where grades I-II predict easy intubation and grades III-IV predict difficult intubation. In grades III-IV, the BURP maneuver (backward, upward, rightward, pressure) is recommended[5]. The maneuver was termed BURP as an acronym for "backward, upward, rightward, pressure". This procedure displaces the thyroid cartilage dorsally in such a way that the larynx is pressed against cervical vertebrae, two centimeters in cephalic direction, until resistance appears. Subsequently, it should be displaced 0.5 cm -2.0 cm to the right. This allows better viewing and possible modification of the Cormack-Lehane grades. This technique is incompatible with the Sellick maneuver[101] exclusively used to prevent gastric reflux[102].

First described in 1961 by Sellick, the maneuver decreases the risk of pulmonary aspiration of gastric contents during induction of anesthesia[103]. The technique involves the application of backward thumb and middle or forefinger pressure on the cricoid cartilage to compress the esophagus between the cricoid cartilage and the anterior vertebral body, resulting in occlusion of the esophageal lumen and thus prevent gastric reflux[5]. There is controversy about its usefulness. Some studies show it does not completely prevent aspiration, that if not correctly performed it may hinder visualization of the glottis. Studies are inconclusive. It is recommended, a priori, in RSI from the time of induction until definitive intubation but subject to operator judgement. It may be used with caution, if ventilation (positive pressure) with a self-inflating bag is necessary, to prevent passage of air into the stomach and the adverse effects of excessive ventilation[102-105]. So both the Sellick technique and BURP should be performed by another physician with expertise, and subject to the judgement of the operator responsible for the RSI[5].

6. Checking for correct placement of the endotracheal tube

Verification of correct endotracheal tube placement is performed immediately after intubation.

Accidental intubation of the esophagus or a bronchus can result in serious injuries. There are various methods to verify correct endotracheal tube placement:

1. Direct visualization of the introduction of the endotracheal tube through the vocal cords.
2. Inspection, palpation and auscultation (5 points: both mid-infra-clavicular areas, bilateral axillary area, axillary midline at the level of the 5th intercostal space and the epigastrium).
3. Measurement of ET depth from the incisors.
4. Capnography.
5. Chest x-ray.
6. Fiberoptic bronchoscopy[106].

No single method alone guarantees correct ET placement. Direct visualization of the ET passing through the vocal cords confirms its entry into the upper airway, but does not rule out subsequent displacement. Lung auscultation is the most common method but has low sensitivity due to the noise of air passing other structures and environmental noise. Measuring the depth of ET insertion is unreliable and it depends on whether the patient has a long or short neck. Capnography is a very reliable method, measuring the elimination of CO_2 via the airway. The presence of CO_2 rules out esophageal intubation, but is not viable in situations of hypoperfusion.

Chest radiography is performed routinely to check for ET placement in the trachea or main bronchus, not does not rule out esophageal location. Fiberoptic bronchoscopy is very reliable, almost 100%, but is not always available in emergency situations[5,102,106]. In short, it is recommended that all available resources be used immediately after ET placement[102].

7. Post-intubation management

This last phase, in addition to treating the underlying disease, involves adequate sedation, with analgesic and relaxation maintenance, adjustment of mechanical ventilation parameters and exhaustive monitoring (oxygen saturation, capnography, heart rate and blood pressure) and chest x-ray to quickly detect the most common complications in RSI.

All patients connected to mechanical ventilation experience pain and anxiety. Inadequate sedation and analgesia inherently produces cholinergic release in the patient, resulting in tachycardia, hypertension and tachypnea, as well as body movement and maladjustment to mechanical ventilation. Hence the importance of correct analgesia and sedation. Fentanyl is the analgesic of choice due to its rapid action, slight release of histamine (which favors hemodynamic stability) and the absence of active metabolites. Patients attended in the ED or by out-of-hospital EMS who need emergency intubation are going to stay for a relatively short period of time; after the initial dose of intravenous fentanyl (2-3 ug/Kg) it is preferable to continue with repeated intravenous boluses until the desired effect is achieved, since continuous infusion favors build-up of the drug and increases elimination time.

After good analgesia, adequate sedation is required to reduce anxiety and produce amnesia. The sedatives used for maintenance are midazolam, lorazepam (not marketed in Spain) and propofol. The latter is increasingly used because of its speed of action and short duration. In addition, infused propofol decreases mechanical ventilation time compared with lorazepam. In the ED, it is safe because the undesirable "propofol infusion syndrome" only appears after 48 hours.

Neuromuscular blockers are known to favor adaptation to mechanical ventilation, but their use in continuous perfusion increases the development of poly-neuromyopathy in critical patients, which extends their time on mechanical ventilation and intensive care unit (ICU) stay, and is therefore associated with increased mortality (so only short periods are recommended). For maintenance, the most generally used NMB are cisatracurium and rocuronium because of their optimal stability characteristics. The choice of NMB depends on the induction drug. If succinylcholine was used initially, it is preferable to continue with cisatracurium. If rocuronium was used initially, it is preferable to continue with the same agent[107]. Cisatracurium is ideal for maintenance of anesthesia in the ICU and in the transport of critical patients in mobile ICU units, due to its hemodynamic stability effect, and the absence of histamine release. It does not affect cerebral perfusion, can be used in patients with renal and liver failure (because its metabolism is organ-independent, by Hofmann degradation, i.e. pH and physiological temperature hydrolysis), does not produce active metabolites and has no significant adverse effects. In the ED it is recommended for intravenous boluses at doses of 0.1-0.15 mg/kg (presentation: 10 mg/5 ml). It lasts 45-60 minutes, and the dose can be repeated if necessary during transfer to the UCI[90,108,109].

Precise adjustment of mechanical ventilation parameters is necessary to prevent ventilator induced lung injury due to the combined effects of volutrauma, atelectrauma, barotrauma and bio-

trauma. These complications typically occur with the use of high tidal volumes, high pressure and high oxygen concentrations. The most characteristic signs are hypotension and increased airway resistance. To avoid these complications, lung-protective strategies are recommended, involving the use of low volumes to avoid volume/barotrauma, positive end expiratory pressure (PEEP) and alveolar recruitment maneuvers to prevent atelectrauma, and reduced inspired oxygen concentration to decrease biotrauma[107].

References

1 Elizabeth Mace S. Challenges and Advances in Intubation: Rapid Sequence Intubation. Emerg Med Clin N Am. 2008;26:1043-68.
2 Bair A. Rapid sequence intubation in adults. Uptodate 2011. (Consultado 8 Septiembre 2011). Disponible en: http://www.uptodate.com/contents/rapid-sequence-intubation-in-adults.
3 Cudnik MT, Newgard CD, Daya M, Jui J. The impact of rapid sequence intubation on trauma patient mortality in attempted prehospital intubation. J Emerg Med. 2010;38:175-81.
4 Eich C, Timmermann A, Russo S, Cremer S, Nickut A, Strack M, et al. A controlled rapid-sequence induction technique for infants may reduce unsafe actions and stress. Acta Anaesthesiol Scand. 2009;53:1167-72.
5 Palencia Herregón E, Borrallo Pérez J, Pardo Rey C. Intubación del enfermo crítico. Med Intensiva. 2008;(Supl. 1):3-11.
6 Mateos Rodríguez AA, Navalpotro Pascual JM, Pardillos Ferrer L, Pelayo Martínez E. Validez de la distancia tiromentoniana como predictor de vía aérea difícil en medicina extrahospitalaria. Emergencias. 2011;23:246.
7 Herrerías Lloréns J. Inducción anestésica de secuencia rápida. Rev Esp Anestesiol Reanim. 2003;50:87-96.
8 Fakhry S, Scanlon J, Robinson L, Askari R, Watenpaugh R, Fata P, et al. Prehospital Rapid Sequence Intubation for Head Trauma: Conditions for a Successful Program. J Trauma. 2006;60:997-1001.
9 Walls RM, Murphy MF. The difficult airway in adults. Update 2011. (Consultado 8 Septiembre 2011). Disponible en: http://www.uptodate.com/contents/the-difficult-airway-in-adults?
10 Reed MJ, Dunn MJG, McKeown DW. Can an airway assessment score predict difficulty at intubation in the emergency department? J Emerg Med. 2005;22:99-102.
11 Hyuk Joong C, Hyung-Goo K, Tae Ho L, Hyun Soo C, Junho C, Young-Min O, et al. Endotracheal intubation using a GlideScope video laryngoscope by emergency physicians: a multicentreanalysis of 345 attempts in adult patients. J Emerg Med. 2010;27:380-2.
12 Nee PA, Benger J, Walls RM. Airway management. J Emerg Med. 2008;25:98-102.
13 Matthew JR. Intubation training in emergency medicine: a review of one trainee's first 100 procedures. J Emerg Med. 2007;24:654-6.
14 Bair A, Caravelli R, Tyler K, Laurin E. Feasibility of the preoperative mallampati airway assessment in emergency department patients. J Emerg Med. 2010;38:677-80.
15 Reynolds S, Heffner J. Airway management of the critically ill patient. Rapid-sequence intubation. Chest. 2005;127:1397-412.
16 Dargin J, Medzon R. Emergency department management of the airway in obese adults. Ann Emerg Med. 2010;56:95-104.
17 Weingart SD. Preoxygenation, reoxygenation, and delayed sequence intubation in the emergency department. J Emerg Med. 2011;40:661-7.
18 Davis DP, Douglas DJ, Koenig W, Carrison D, Buono C, Dunford J. Hyperventilation following aero-medical rapid sequence intubation may be a deliberate response to hypoxemia. Resuscitation. 2007;73:354-61.
19 Benumof J, Dagg R, Benumof R. Critical hemoglobin desaturation will occur before return to an unparalized state following 1 mg/kg intravenous succinylcholine. Anestesiology. 1997;87:979-82.
20 Levitan RM, Chudnofsky C, Sapre N. Emergency airway management in a morbidly obese, noncooperative, rapidly deteriorating patient. Am J Emerg Med. 2006;24:894-6.
21 El Solh AA. Airway Management in the Obese Patient. Clin Chest Med. 2009;30:555-68.
22 Loder WA. Airway Management in the Obese Patient. Crit Care Clin. 2010;26:641-6.
23 Baskett P JF, Baskett TF. Brian Sellick, Cricoid Pressure and the Sellick Manoeuvre. Resuscitation. 2004;61:5-7.
24 Elizabeth Mace S. Challenges and Advances in Intubation: Airway Evaluation and Controversies with Intubation. Emerg Med Clin N Am. 2008;26:977-1000.
25 Mower WR, Knopp RK. Clinical controversies: lidocaine administration before rapid sequence intubation in patients with traumatic brain injuries. Ann Emerg Med. 2007;49:84-6.
26 ilber SH. Rapid Sequence Intubation in adults with elevated intracranial pressure: A survey of emergency medicine residency programs. Am J Emerg Med. 1997;15:263-7.
27 Juul N, Duch B, Rasmussen M. Clinical management of patients with head injury. Curr Anaesth Crit Care. 2009;20:132-7.
28 Thomas SH, Benevelli W, Brown DFM, Wedel S. Safety of fentanyl for analgesia in adults undergoing air medical transport from trauma scenes. Air Med J. 1996;15:2:57-9.
29 Bernard S, Nguyen V, Cameron P, Masci K, Fitzgerald M, Cooper DJ, et al. Prehospital rapid sequence intubation improves functional outcome for patients with severe traumatic brain injury. Ann Surg. 2010;252:959-65.
30 Taylor EJS, Feneck RO, Chambers DJ. Fentanyl and myocardial protection: is there a preconditioning mechanism? Crit Care. 2001;5:(Supl 4).
31 Mularski RA. Pain management in the intensive care unit. Crit Care Clin. 2004;20:381-401.
32 Gindre S, Ciais JF, Levraut L, Dellamonica J, Guerin J, Grimaud D. Rapid sequence intubation in emergency: is there any place for the fentanyl? Ann Fr Anesth Reanim. 2002;21:760-6.
33 Brenner B, Corbridge T, Kazzi A. Intubation and mechanical ventilation of the asthmatic patient in respiratory failure. J Allergy Clin Immunol. 2009;124:19-28.
34 Brenner B, Corbridge T, Kazzi A. Intubation and mechanical ventilation of the asthmatic patient in respiratory failure. J Emerg Med. 2009;37:S23-S34.
35 Bottor LT. Rapid Sequence Intubation in the Neonate. Adv Neonatal Care. 2009;9:111-7.
36 Agrawal D. Rapid sequence intubation in children. Uptodate 2011. (Consultado 21 Septiembre 2011). Disponible en: http://www.uptodate.com/contents/rapid-sequence-intubation-inchildren? source=search_result&selectedTitle=1%7E33#H15.
37 Devlin JW, Roberts RJ. Pharmacology of commonly used analgesics and sedatives in the ICU: benzodiazepines, propofol, and opioids. Crit Care Clin. 2009;25:431-49.
38 Thomas SH, Shewakramani S. Prehospital trauma analgesia. J Emerg Med. 2008;35:47-57.
39 Bledsoe GH, Schexnaydery SM. Pediatric rapid sequence intubation. A Review. Pediatr Emerg Care. 2004;20:339-44.
40 Gerardi MJ, Sacchetti AD, Cantor RM, Santamaria JP, Gausche M,Lucid W, et al. Rapid-sequence intubation of the Pediatric Patient. Ann Emerg Med. 1996;28:55-74.
41 Horvath II PR, Mayberry R, Franklin K, Ekbla G. The Medication-Assisted Intubation Matrix©: A Literature Review and Evidence-Based Guidelines. Ann Emerg Med. 2007;50:111.
42 Sivilotti MLA, Filbin MR, Murray HE. Does the Sedative Agent Facilitate Emergency Rapid Sequence Intubation? Acad Emerg Med. 2003;10:612-20.
43 Forman SA. Clinical and molecular pharmacology of etomidate. Anesthesiology. 2011;114:695-707.
44 Pandit JJ. Intravenous anaesthetic agents. Anaesth Intensive Care. 2010;12:144-50.
45 Hool AJ, Kitson RM. Induction of anaesthesia. Anaesth Intensive Care. 2009;11:25-31.
46 Huter L, Schreiber T, Gugel M, Schwarzkopf K. Low-dose intravenous midazolam reduces etomidate-induced myoclonus: a prospective, randomized study in patients undergoing elective cardioversion. Anesth Analg. 2007;105:1298-302.
47 Hohl CM, Kelly-Smith CH, Yeung TC, Sweet D, Doyle-Waters M, Schulzer M. The effect of a bolus dose of etomidate on cortisol levels, mortality, and health services utilization: a systematic review. Ann Emerg Med. 2010;56:105-13.
48 Jones AE. The etomidate debate. Ann Emerg Med. 2010;56:490-1.
49 Dmello D, Taylor S, O'Brien J, Matubchak GM. Outcomes of etomidate in severe sepsis and septic shock. Chest. 2010;138:1327-32.
50 Ehrman R, Wira III C, Hayward A, Lomax A, Mullen M. Etomidate use in sepsis does not increase mortality. Ann Emerg Med. 2010;56:117.
51 Feldman J. Etomidate, sepsis, and informed consent. Ann Emerg Med. 2011;57:706.
52 Majesko A, Darby JM. Etomidate and adrenal insufficiency: the controversy continues. Crit Care. 2010;14:328.
53 Tekwani KL, Watts HF, Sweis RT, Rzechula KH, Kulstad EB. A comparison of the effects of etomidate and midazolam on hospital length of stay in patients with suspected sepsis: a prospective, randomized study. Ann Emerg Med. 2010;56:481-9.
54 Jabre P, Combes X, Lapostolle F, Dhaouadi M, Ricard-Hibon A, Vivien

B, et al. Etomidate versus ketamine for rapid sequence intubation in acutely ill patients: a multicentre randomised controlled trial. Lancet. 2009;374:293-300.
55 Ching KY, Baum CR. Newer agents for rapid sequence intubation etomidate and rocuronium. Pediatr Emer Care. 2009;25:200-10.
56 Adnet F, De La Coussaye JE, Jabre P. Intubation en séquence rapide: quels médicaments utiliser en préhospitalier? Réanimation. 2010;19:622-6.
57 Ray D, McKeown D. Emergency tracheal intubation: More than just technical skill. Resuscitation. 2011;82:505-6.
58 Svenson J, Abernathy M. Ketamine for prehospital use: new look at an old drug. Am J Emerg Med. 2007;25:977-80.
59 Strayer R, Nelson L. Adverse events associated with ketamine for procedural sedation in adults. Am J Emerg Med. 2008;26:985-1028.
60 Sener S, Eken C, Schultz C, Serinken M, Ozsarac M. Ketamine with and without midazolam for emergency department sedation in adults: a randomized controlled trial. Ann Emerg Med. 2011;57:109-4.
61 Green S, Roback M, Kennedy R, Krauss B. Clinical practice guideline for emergency department ketamine dissociative sedation: 2011 update. Ann Emerg Med. 2011;57:449-61.
62 Nordt S, Clark R. Midazolam: a review of therapeutic uses and toxicity. J Emerg Med. 1997;15:357-65.
63 Shearer P, Riviello J. Generalized convulsive status epilepticus in adults and children: treatment guidelines and protocols. Emerg Med Clin N Am. 2011;29:51-64.
64 Frölich MA, Arabshahi A, Katholi C, Prasain J, Barnes S. Hemodynamic characteristics of midazolam, propofol, and dexmedetomidine in healthy volunteers. J Clin Anesth. 2011;23:218-23.
65 Miner J, Burton J. Clinical practice advisory: emergency department procedural sedation with propofol. Ann Emerg Med. 2007;50:182-7.
66 Weaver C, Hauter W, Brizendine E, Cordell W. Emergency department procedural sedation with propofol: is it safe? J Emerg Med. 2007;33:355-61.
67 Kwon M, Kim S, Jeon D, Song J, Kim W. The effect of additional propofol on intubation conditions. J Clin Anesth. 2010;22:603-7.
68 Subhas K, Appleby J. Traumatic brain injury: initial resuscitation and transfer. Anaesth Intensive Care. 2011;12:201-3.
69 Hayward E, Hunt K. Clinical neuroprotection and secondary neuronal injury mechanisms. Anaesth Intensive Care. 2011;12:198-200.
70 San-Juan D, Chiappa K, Cole A. Propofol and the electroencephalogram. Clin Neurophysiol. 2010;121:998-1006.
71 García Vicente E, Sandoval Almengor JC, Díaz Caballero LA, Salgado Campo JC. Ventilación mecánica invasiva en EPOC y asma. Med Intensiva. 2011;35:288-98.
72 Hanna S, Ahmad F, Pappas AL, Mikat-Stevens M, Jellish WS, Kleinman B, et al. The effect of propofol/remifentanil rapid-induction technique without muscle relaxants on intraocular pressure. J Clin Anesth. 2010;22:437-42.
73 Salman A, Salman M, Saricaoglu F, Akinci SB, Aypar U. Pain on injection of propofol: a comparison of methylene blue and lidocaine. J Clin Anesth. 2011;23:270-4.
74 Muller AE, Huisman I, Roos PJ, Rietveld AP, Klein J, Harbers JBM, et al. Outbreak of severe sepsis due to contaminated propofol: lessons to learn. J Hosp Infect. 2010;76:225-30.
75 Mallory M, Baxter A, Yanosky D, Cravero J. Emergency physician–administered propofol sedation: a report on 25,433 sedations from the pediatric sedation research consortium. Ann Emerg Med. 2011;57:462-8.
76 Power KN, Flaatten H, Gilhus NE, Engelsen BA. Propofol treatment in adult refractory status epilepticus. Mortality risk and outcome. Epilepsy Res. 2011;94:53-60.
77 Hickey K, Martin D, Chuidian F. Propofol-induced seizure-like phenomena. J Emerg Med. 2002;29:447-9.
78 Laquay N, Prieur S, Greff B, Meyer P, Orliaguet G. Le syndrome de perfusion du propofol. Ann Fr Anesth Reanim. 2010;29:377-86.
79 Farooq K, Hunter J. Neuromuscular blocking agents and reversal agents. Anaesth Intensive Care. 2011;12:266-70.
80 Perry JJ, Lee JS, Sillberg VAH, Wells GA. Rocuronium versus succinylcholine for rapid sequence induction intubation. Cochrane Database of Systematic Reviews 2008, Issue 2. Art. No.: CD002788. DOI:10.1002/14651858.CD002788.pub2.
81 Caldwell J. The Continuing Search for a Succinylcholine Replacement. Anesthesiology. 2004;100:763-4.
82 Pollard B. Neuromuscular blocking agents and reversal agents. Anaesth Intensive Care. 2005;6:189-92.
83 Orebaugh S. Succinylcholine: adverse effects and alternatives in emergency medicine. Am J Emerg Med. 1999;17:715-21.
84 Booij L. Is succinylcholine appropriate or obsolete in the intensive care unit? Crit Care. 2001;5:245-6.
85 Pérez-Moreno J, Rodríguez M, González P, Sánchez-Elduayen MT. Dosificación actual de la succinilcolina. Rev Esp Anestesiol Reanim. 2007;54:54-5.
86 Hernández-Palazón J, Noguera-Velasco J, Falcón-Araña L, Doménech-Asensi P, Burguillos-López S, Nuño de la Rosa-Carrillo V. La precurarización con rocuronio previene las fasciculaciones y los cambios bioquímicos tras la administración de succinilcolina. Rev Esp Anestesiol Reanim. 2004;51:184-9.
87 Zimmerman A, Funk K, Tidwell J. Propofol and alfentanil prevent the increase in intraocular pressure caused by succinylcholine and endotracheal intubation during a rapid sequence induction of anesthesia. Anesth Analg. 1996;83:814-7.
88 Turan A, Mendoza M, Gupta S. Consequences of succinylcholine administration to patients using statins. Anesthesiology. 2011;115:28-35.
89 Lee C. Succinylcholine should be avoided in patients on statin therapy. Anesthesiology. 2011;115:6-7.
90 Chamorro C, Romera MA, Pardo C, Silva JA. Nuevos bloqueadores neuromusculares. Med Intensiva. 2001;25:340-3.
91 Mallon WK, Keim SM, Shoenberger JM, Wall RM. Rocuronium *vs* succinylcholine in the emergency department: a critical appraisal. J Emerg Med. 2009;37:183-8.
92 Lee C, Katz R. Clinical implications of new neuromuscular concepts and agents: So long, neostigmine! So long, sux! J Crit Care. 2009;24:43-9.
93 Seupaul R, Jones J. Does succinylcholine maximize intubating conditions better than rocuronium for rapid sequence intubation? Ann Emerg Med. 2011;57:301-2.
94 Yang L, Keam S. Sugammadex. A review of its use in anaesthetic practice. Drugs. 2009;69:919-42.
95 Ren W, Jahr J. Reversal of neuromuscular block with a selective relaxant-binding agent: sugammadex. Am J Ther. 2009;16:295-9.
96 McDonagh D, Benedict P, Kovac A, Drover D, Brister N, Morte J, et al. Efficacy, safety, and pharmacokinetics of sugammadex for the reversal of rocuronium-induced neuromuscular blockade in elderly patients. Anesthesiology. 2011;114:318-29.
97 Lee C, Jahr J, Candiotti K, Warriner B, Zornow M, Naguib M. Reversal of profound neuromuscular block by sugammadex administered three minutes after rocuronium. A Comparison with Spontaneous Recovery from Succinylcholine. Anesthesiology. 2009;110:1020-5.
98 Strayer R. Rocuronium versus succinylcholine: cochrane synopsis reconsidered. Ann Emerg Med. 2011;58:217-8.
99 Marsch S, Steiner L, Bucher E, Pargger H, Schumann M, Aebi T, et al. Succinylcholine versus rocuronium for rapid sequence intubation in intensive care: a prospective, randomized controlled trial. Crit. Care. 2011;15:R199.
100 Souto Mata F, Martínez Melgar J, Katcher W, Rama Sorribas JM, Paz Esquete J. Experiencia con el uso de rocuronio en la intubación orotraqueal en una unidad de soporte vital avanzada móvil (USVA). Puesta al día en urgencias, emergencias y catástrofes. 2009;9:1-7.
101 Carrillo-Esper R, Vinay-Ramírez B, Bahena A. Maniobra de BURP. Revista Mexicana de Anestesiología. 2008;31:63-5.
102 Paal P, Herff H, Mitterlechner T, Goedeckeb A V, Brugger H, Lindner K, et al. Anaesthesia in prehospital emergencies and in the emergency room. Resuscitation. 2010;81:148-54.
103 Vanner R. Cricoid pressure. Int J Obstet Anesth. 2009;18:103-5.
104 Harris T, Ellis D, Foster L, Lockey D. Cricoid pressure and laryngeal manipulation in 402 pre-hospital emergency anaesthetics: Essential safety measure or a hindrance to rapid safe intubation? Resuscitation. 2010;81:810-6.
105 Ellis D, Harris T, Zideman D. Cricoid pressure in emergency department rapid sequence tracheal intubations: a risk-benefit analysis. Ann Emerg Med. 2007;50:653-65.
106 Rigini N, Boaz M, Ezri T, Evron S, Jackobashvilli S, Izakson A. Prompt correction of endotracheal tube positioning after intubation prevents further inappropriate positions. J Clin Anesth. 2011;23:367-71.
107 Wood S, Winters M. Care of the intubated emergency department patient. J Emerg Med. 2011;40:419-27.
108 Schram W, Jesenko R, Bartune A, Gilly H. Effects of cisatracurium on cerebral and cardiovascular hemodynamics in patients with severe brain injury. Act Anaesthesiol Scand. 1997;41:1319-23.
109 De Boer H. Neuromuscular transmission: new concepts and agents. J Crit Care. 2009;24:36-42.

Secuencia de intubación rápida

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El aislamiento de la vía aérea de emergencia es una técnica de alto riesgo y estresante para el facultativo. Para minimizar las complicaciones, tener alto porcentaje de éxito de intubación y disminuir el estrés del responsable de la intubación se utiliza la secuencia de intubación rápida (SIR). Consta de 7 pasos: planificación y preparación, preoxigenación, pretratamiento, sedación y relajación neuromuscular, posición y protección, comprobación del tubo endotraqueal y manejo postintubación. En esta revisión realizamos una descripción y actualización de las fases de SIR. [Emergencias 2012;24:397-409]

Palabras clave: Secuencia de intubación rápida. Intubación endotraqueal. Opioide. Hipnótico. Relajante neuromuscular.