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Sedation and Airway Anesthesia for Awake Fiberoptic Intubation

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Abstract: Awake fiberoptic intubation is the recommended technique for anticipated difficult airway management which requires effective local anesthesia for patient's comfort and co-operation. However, it is often an unpleasant procedure to the patient because sensation of passage of the instrument through the nose and larynx, pain and coughing is the main complaints, while endoscopists usually attribute difficulty in laryngeal visualization to secretions and inadequate local anesthesia. Spraying of local anesthetic can be achieved in different ways, including the use of commercially prepared aerosol spray cans, atomizers and nebulizers. Successful awake tracheal intubation (ATI) requires an adequate level of sedation, in which the patient is able to respond to verbal commands while maintaining spontaneous ventilation and hemodynamic stability. An ideal sedation technique for ATI should include anxiolysis, amnesia, appropriate analgesia, and suppression of the cough and gag reflexes. When attempting sedation for ATI, the Standard American Society of Anesthesiology monitors, oxygen via nasal cannula, and capnography should be in place. Several classes of drugs have been described, from benzodiazepines (e.g., diazepam and midazolam), to opioids (e.g., morphine, fentanyl, and remifentanil), to intravenous induction agents (e.g., ketamine and propofol), and to alpha2 agonists (e.g., clonidine and dexmedetomidine)

Keywords:*Airway Anesthesia, Sedation, Awake Fiberoptic Intubation*

Introduction

Awake fiberoptic intubation is the recommended technique for anticipated difficult airway management which requires effective local anesthesia for patient's comfort and co-operation. However, it is often an unpleasant procedure to the patient because sensation of passage of the instrument through the nose and larynx, pain and coughing is the main complaints, while endoscopists usually attribute difficulty in laryngeal visualization to secretions and inadequate local anesthesia **(1).**

The better local anesthetic technique would require a lower dose of local anesthetic and at the same time provide acceptable conditions for the endoscopists **(2).**

Methods of topicalization

Spraying of local anesthetic can be achieved in different ways, including the use of commercially prepared aerosol spray cans, atomizers and nebulizers **(3)**.

1. Atomization

Atomized lidocaine is frequently used to anesthetize the oropharynx. Local anesthetic can be directly sprayed onto the nasopharynx and oropharynx using McKenzie technique or a mucosal atomization device (MAD) **(4)**. The McKenzie technique uses a 20-gauge cannula attached to oxygen bubbling tubing via a three-way stopcock. The other end of bubbling tube is attached to an oxygen source, which delivers a flow of 2-4 L/min**.** As the local anesthetic is slowly administered via a 5-mL syringe attached to the top port of the cannula, a jet like spray effect is seen, which greatly increases the surface area of the local anesthetic and allows directed topicalization of the nasal and oral mucosa **(5).**

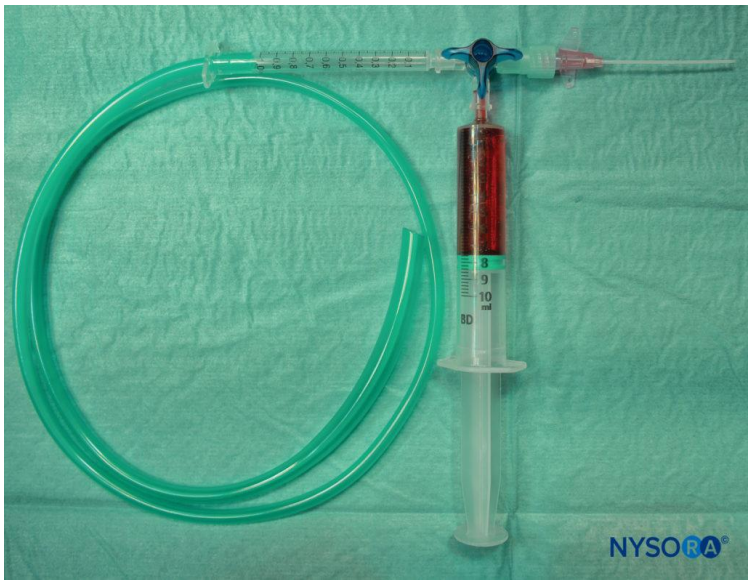

Fig. (1): Setup for McKenzie technique **(6)**

2. Spray-as-you-go' technique

The commonest method to anesthetize the larynx is to spray lidocaine directly down the fiberscope side port (spray-as-you-go technique).This is a technique is used when the larynx is identified using the fiberscope and anesthetized as visualized **(7).**

This local anesthetic is administered through the working channel of a flexible intubating endoscope while advancing the tip through the upper and lower airway with the goal of intubating the patient's trachea **(8).**

An epidural catheter can be inserted through the flexible bronchoscope channel and distal end of the catheter extending 1 to 2 cm beyond the FIS. Insertion of an epidural catheter through the working channel allows precise application of LA to the desired parts of the airway. LA is placed onto the vocal cords under direct visualization. Once the vocal cords are sprayed, the solution can also be instilled into the trachea following passage through the rima glottidis **(5).**

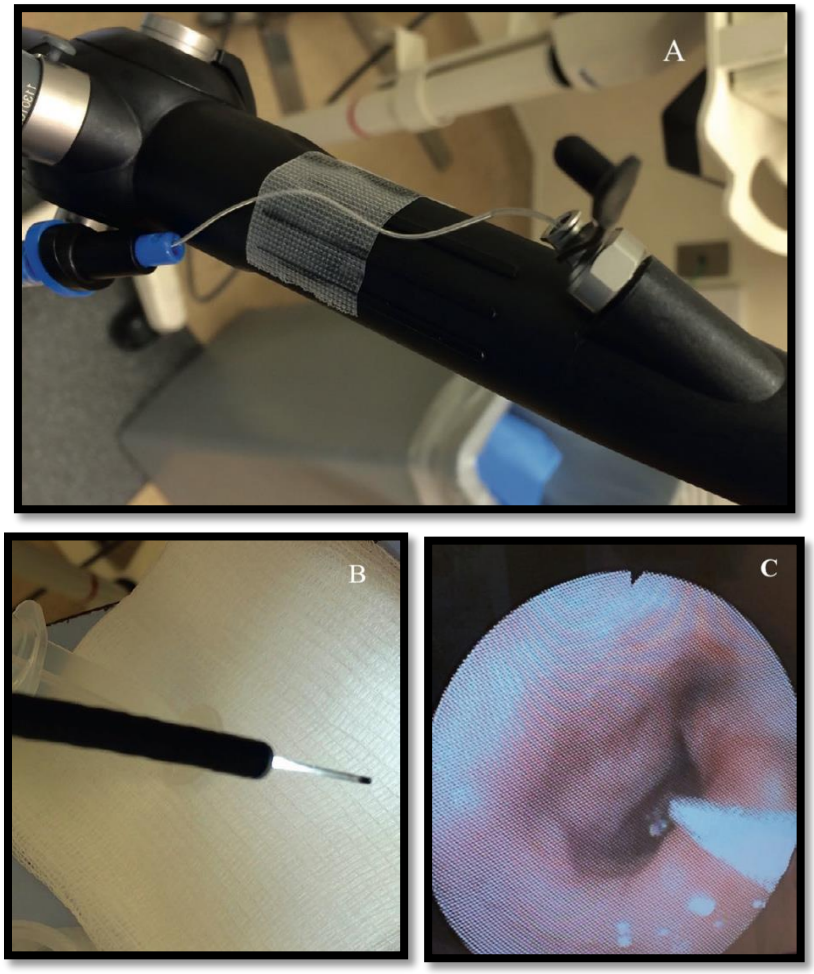

Fig. (2): (A) Proximal epidural catheter taped to handle by working channel. (B) Distal catheter 1 cm out from tip. (C) Drop of lidocaine from initial "spray as you go." **(9)**

3. Nebulization

Nebulizers are a non-invasive technique that is used to deliver very fine particles of drugs to the distal airway. As a sole technique, it may provide adequate anesthesia for instrumentation of the airway. However, combined with other techniques, it provides better patient acceptance and hemodynamic stability. Facemask nebulization can be used for nasal or oral topicalization (**10).**

4 - 5ml of 2%, 4% or 10% lidocaine can be nebulized for 10 -15 minutes *(doses will vary depending on weight and techniques employed).* The patient is asked to take shallow breaths to tropicalize the more proximal airway and to take slow deep breaths for the more distal airway. This process may need repeating to ensure adequate airway anesthesia **(5).**

For nasal intubation, a facemask that covers the mouth and the nose can be utilized. It is important to ensure the patient does not speak during nebulizer application. If oral intubation is planned, a nebulizer attached to a mouthpiece is sufficient **(7).**

4.Airway Nerve blocks

When topical anesthesia has failed or is ineffective for awake tracheal intubation (ATI), nerve blocks in the form of a glossopharyngeal nerve (GPN) block, superior laryngeal nerve (SLN) block, and transtracheal block are indicated. These blocks are contraindicated in patients with coagulopathies or those who are actively anticoagulated. Aspiration should be instituted prior to LA deposition to avoid intravascular injection, which can lead to nerve injury, seizures, and trauma **(5).**

These nerve blocks may be inadequate in some patients because of bad technique, lack of experience and anatomical variations which can disturb the patient cooperation and can abort AFOI **(11)**.

However, ultrasound can be an excellent tool to increase the success rate of airway blocks by optimizing the accuracy of LA deposition. Ultrasound can be most useful in identifying the greater cornu of the hyoid bone for the SLN block and the cricothyroid membrane for the transtracheal block **(5).**

Glossopharyngeal Block:

This block anesthetizes the oropharynx by LA administration along the IXth cranial nerve, which provides sensation to the posterior third of the tongue, the anterior surface of the epiglottis, the vallecula, the tonsils, and the pharyngeal walls. The block techniquecan be done through intraoral or peristyloid approach **(12).**

Superior Laryngeal Block

The internal branch of the SLN provides sensory innervation to the tongue base, the posterior surface of the epiglottis, the arytenoids, and the aryepiglottic folds. This block anesthetizes the larynx above the vocal cords **(5).**

The superior laryngeal nerves are blocked bilaterally by infiltrating 1.5 ml of lidocaine 2% on each side at the lateral and inferior aspect of the hyoid bone **(13).**

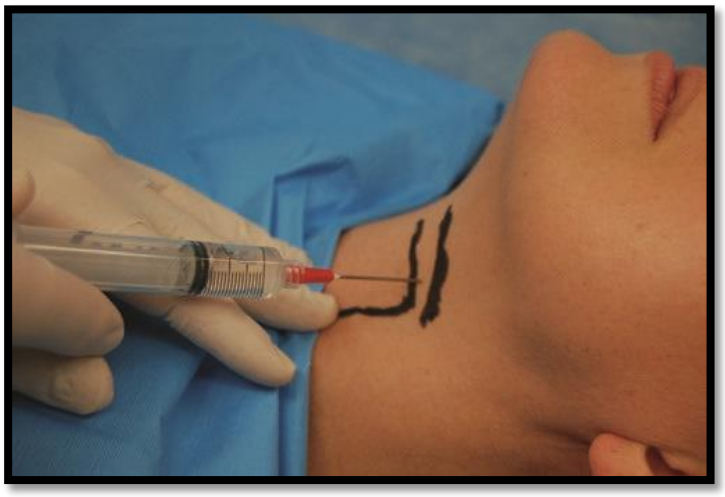

Fig. (3): Superior laryngeal nerve block **(13).**

Transtracheal Block

The transtracheal block anesthetizes the recurrent laryngeal nerve (RLN), which provides sensation to the trachea below the level of the vocal cords. The RLN provides motor innervation to all intrinsic muscles of the larynx except the cricothyroid, supplied by the external branch of the SLN.

The cricothyroid membrane lies between these two structures, just superior to the cricoid cartilage. A 22-gauge intravenous catheter is injected through the cricothyroid membrane until air is aspirated while attached to a syringe filled with 4% lidocaine**.** It is important to stop advancing the needle once tracheal positioning is confirmed, as puncture of the posterior laryngeal wall should be avoided. Continuous aspiration of the syringe is important, as air bubbles offer confirmation of proper tracheal placement. Local anesthetic is then injected as coughing facilitates lidocaine distribution to ensure the blockade of the RLN **(12).**

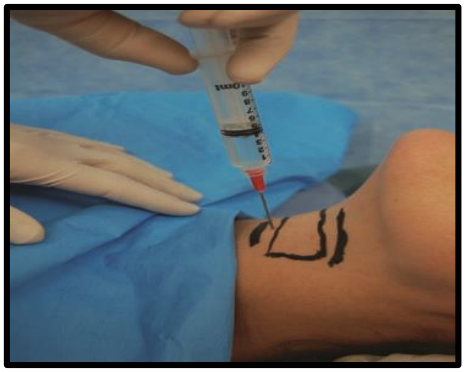

Fig. (4): Transtracheal injection of local anesthetic **(13).**

Gargling:

Most commonly, several milliliters of a 2-4% lidocaine solution are placed in the mouth and the patient is instructed to gargle with this solution. While this method can provide adequate anesthesia to the oral mucosa, it does not often cover the larynx or trachea adequately **(3)**.

Sedation for Awake Fiberoptic Intubation

Successful awake tracheal intubation (ATI) requires an adequate level of sedation, in which the patient is able to respond to verbal commands while maintaining spontaneous ventilation and hemodynamic stability. An ideal sedation technique for ATI should include anxiolysis, amnesia, appropriate analgesia, and suppression of the cough and gag reflexes **(5).**

When attempting sedation for ATI,the Standard American Society of Anesthesiology monitors, oxygen via nasal cannula, and capnography should be in place **(14).**

Several classes of drugs have been described, from benzodiazepines (e.g., diazepam and midazolam), to opioids (e.g., morphine, fentanyl, and remifentanil), to intravenous induction agents (e.g., ketamine and propofol), and to alpha$_2$ agonists (e.g., clonidine and dexmedetomidine). **Johnston and Rai** reviewed multiple studies on solo drug uses; average doses and their characteristics. **(Table 1) (14).**

Table 1: Doses and characteristics of sedative drugs **(14)**

Drug	Dose	characteristics
Midazolam	0.015 to 0.03 mg/kg boluses	amnesia/sedation
Fentanyl	0.7 to 1.5 µg/kg boluses	analgesia/antitussive
Remifentanil	0.05 to 0.1 µg/kg bolus 0.03 to 0.1 µg/kg/ minute infusion	analgesia
Ketamine	0.07 to 0.15 mg/kg boluses	sedation/analgesia
Propofol	25 to 75 µg/kg bolus; 25- to 75 µg/kg/minute infusion	sedation
Dexmedetomidine	0.5 to 1.0 µg/kg bolus over 10 minutes; followed by 0.2- to 0.7-µg/kg/hour infusion	sedation/analgesia/ antisialogogue/ hemodynamic stability/ amnesia

Midazolam:

Midazolam is a benzodiazepine that can be injected in boluses of 0.5–1 mg, usually not exceeding 0.05 mg kg^{-1}. Because of it has no analgesic properties, it is commonly used in conjunction with fentanyl boluses up to 1.5 ug kg^{-1}. The main advantages of this technique are its simplicity and widespread experience of its use. The added advantage of amnesia may improve patient experience and result in improved compliance with any future awake intubations. Bolus injection can result in oversedation, and so it should be remembered that its effects can be antagonized with flumazenil **(15).**

Remifentanil:

It is a potent and ultra-short-acting opioid which is metabolized by non-specific plasma and tissue esterases. It can effectively minimize both the patient's cough (antitussive) and gag reflexes, with a context-sensitive half-time of three minutes and an elimination half-time of six minutes. Its unique pharmacokinetic characteristics make it easy to titrate, while it provides profound analgesia, suppresses airway reflexes, and has minimal effect on cognitive function This makes it an attractive drug of choice for the intensely stimulating but usually brief airway manipulation during an AFOI. It is increasingly being used either as the primary agent or in conjunction with midazolam **(16).**

Propofol:

Propofol is a highly lipid-soluble alkylphenol derivative that can be injected in boluses, as a simple infusion, or as a target controlled infusion (TCI). The vast majority of studies looking at its use for sedation during awake intubation have been with TCI. Achieving the correct balance between under sedation and oversedation can be extremely challenging when using propofol as a sole sedative agent, with effect-site concentrations of >3 *ug* ml^{-1} seemingly associated with increased likelihood of oversedation. Concomitant administration of opioids or benzodiazepines can improve efficacy. Some evidences suggest that propofol is best used as TCI with effect-site concentrations up to 1 *ug* ml^{-1}, in conjunction with remifentanil **(15).**

Dexmedetomidine:

Dexmedetomidine, which has found favor for sedation in critical care, has a shorter half-life and eight-fold greater selectivity for α_2 over α_1 receptors than clonidine. It has been enthusiastically advocated for AFOI on the grounds of its ability to produce profound sedation without causing the respiratory depression associated with other anxiolytic-hypnotic drugs and opioids. When respiratory compromise is seen, it occurs as a result of profound oversedation following very large initial bolus doses **(17).**

Mechanism of action:

DEX is a highly selective α_2 adrenoceptor (AR) agonist which produces clinical effects after binding to G-Protein-coupled α_2-AR, of which there are three subtypes (α_2A, α_2B, and α_2C). These receptor subtypes are found ubiquitously in the central, peripheral and autonomic nervous systems, as well as in vital organs and blood vessels **(17)**.

Locus coeruleus of the brain stem is the principal site for the sedative action and spinal cord is the principal site for the analgesic action, both acting through α_2A-AR. Higher affinity to α_2 receptor selectively leads to vagomimetic action on heart (bradycardia) and VD. DEX has a low affinity for β adrenergic, muscarinic, dopaminergic and serotonin receptors. It suppresses shivering possibly by activity at α_2B-AR receptors in the hypothalamic thermoregulatory center of the brain **(18).**

Pharmacokinetics:

1. Absorption:

Oral bioavailability is poor because of extensive first-pass metabolism. However, bioavailability of intranasal (IN) and sublingually administered DEX is high (84%), offering a potential role in pediatric sedation and premedication **(19)**.

2. Distribution:

DEX is a highly protein-bound drug. In plasma, 94% of DEX is bound to albumin and α_1-glycoprotein. The distribution phase is rapid, with a distribution half-life ($t_{1/2}$) of approximately 6 minutes **(20)**.

3. Metabolism and elimination:

DEX undergoes almost complete biotransformation through direct N-glucuronidation, and cytochrome P-450 (CYP 2A6)-mediated aliphatic hydroxylation to inactive metabolites. Metabolites are excreted renally (95%), fecally (4%), and less than 1% are excreted unchanged **(21)**.

Doses:

Table (2): Doses of dexmedetomidine **(22)**:

Route	Dose
Intravenous(IV)	Loading dose of 1 *ug*/kg over 10-20 minutes followed by a maintenance infusion in the range of 0.2- 0.7 *ug*/kg/hr.
Intramuscular (IM)	IM injection (2.5 *ug*/kg) of DEX has been used for premedication.

Pharmacodynamics:

1. Cardiovascular system (CVS):

DEX produces a biphasic BP response; a short hypertensive phase and subsequent hypotension. The two phases are considered to be mediated by two different α_2-AR subtypes; the initial hypertensive phase is most likely due to VC induced by activation of α_2B-AR, whereas hypotension is mediated by the α_2A-AR. The dose-dependent bradycardia seen with DEX is mediated primarily by a decrease in sympathetic tone, and partly by baroreceptor reflex and enhanced vagal activity **(23)**.

2. Respiratory effects:

DEX induces minimal respiratory depression, even when higher doses are used. This property provides great protection in specific situations such as awake craniotomy, awake fiberoptic intubation and weaning and extubation in intensive care unit (ICU) patients **(24)**.

3. Central nervous system (CNS):

DEX administration produces sedation and anxiolysis, thought to be mediated by its inhibitory effect on noradrenergic neurons in the locus coeruleus. The sedative effects are unique, as an arousable sedation state is induced similar in nature to natural sleep **(25)**.

Clinical applications of dexmedetomidine:

1.Adjuvant to general anesthesia:

Intraoperative administration of DEX in lower concentrations has been associated with:

- Reduction of the requirement of inhaled and IV anesthetic agents.
- Reduction in the dose of opioids required perioperatively and postoperatively.
- Attenuating hemodynamic stress response to intubation and extubation.
- Fewer interventions to treat tachycardia.
- Reduction in the incidence of myocardial ischemia.

(26)

2. Prevention of emergence agitation:

DEX decreased the incidence of emergence agitation in children aged 1-10 years, without prolonging the time to extubate. It also reduces the postoperative agitation in elderly patients **(27)**

3. Analgesia:

DEX provides intense analgesia during the perioperative period. Analgesic effect is thought to be mediated by α_2-receptor that suppress pain transmission is by hyperpolarization of interneurons and reduction of the release of pronociceptive transmitters such as substance P and glutamate. Neuraxial DEX has anti-nociceptive effects on somatic and visceral pains. It also reduces postoperative pain and prolongs analgesic duration. It can be used for labor analgesia, gastrointestinal endoscopy analgesia and cancer pain **(28)**.

4. Sedation:

Sedation for Awake fiberoptic intubation:

Dexmedetomidine for it sedative, anxiolytic, analgesic and sympatholytic properties may be considered as a useful drug during awake intubation, reducing participants' discomfort, without depressing respiratory function and having a negligible impact on the cardiovascular system **(29)**.

Sedation for other short-term procedural sedation: such as; Transesophageal echocardiography (TEE), colonoscopy and awake craniotomy **(30)**.

Sedation in the ICU:

DEX has become a popular sedative agent in ICU, because of its ability to produce cooperative sedation, reduces the duration of mechanical ventilation, opioids requirements and length of ICU stay **(23)**.

Adverse effects of dexmedetomidine(22):

The most frequently observed adverse effects include hypotension, bradycardia, dry mouth and nausea. Other reported adverse effects include fever, rigors, cyanosis and muscle weakness.

It may lead to arrhythmias, atrioventricular (AV) block, cardiac arrest, T-wave inversion, angina pectoris, pulmonary edema, bronchospasm, respiratory depression, syncope, neuropathy, paresthesia, paresis, hyperkalemia, lactic acidosis and hyperglycemia.

Abrupt discontinuation after long-term use of DEX may lead to a withdrawal syndrome of nervousness, agitation, headaches and hypertensive crisis.

Contraindications of dexmedetomidine:

Dexmedetomidine is not recommended in patients with uncontrolled hypertension, bradycardia, hemodynamic instability, advanced heart block and ventricular dysfunction. It should be used with extreme caution in pregnant women **(27).**

Dexmedetomidine antagonist:

Atipamezole is a highly selective α_2-adrenoceptors antagonist widely used in veterinary medicine to reverse dexmedetomidine.In humans, it is not recommended because the effective reversal of dexmedetomidine sedation needed atipamezole dosages 40–100-fold higher than that of dexmedetomidine. Such high dose causes unwanted side effects, such as emesis, motor restlessness, and an increase in blood pressure **(31)**.

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