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Dexmedetomidine sedation for awake fiberoptic intubation

Richard J. Unger, MD,[a] and Christopher J. Gallagher, MD[b]

[a]*From the Scripps Memorial Hospital, La Jolla, California; and the*
[b]*Department of Anesthesiology, University of Miami/Jackson Memorial Hospital, Miami, Florida*

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The awake fiberoptic intubation (AFI) is an important part of the anesthesiologist's armamentarium. As well as being technically challenging, it is also uncomfortable and stressful for all involved. We discuss the use of an alpha-2 agonist, dexmedetomidine, to help the clinician and patient through this procedure. The advantages of dexmedetomidine are that it produces a unique "Cooperative Sedation," which reduces discomfort in the patient and assists in the topicalization of the airway. Most importantly, dexmedetomidine does not produce significant respiratory depression, so the airway may be secured in the safest manner possible. We discuss the history of sedation for AFI, other medications, as well as guidelines and pitfalls in the use of dexmedetomidine.

"In dreams begin responsibility."
William Butler Yeats
Responsibilities, 1914

And as our patients face the dreamland of general anesthesia, we take on the responsibility for keeping their airway safe and secure. In case of doubt, the safest, surest route is the awake intubation. Just look at the ASA Difficult Airway Algorithm[1] and the ASA Difficult Airway Algorithm Modified for Trauma.[2] Awake intubation keeps popping up again and again, like some beachball you try to push under the water. There's no keeping it down!

OK fine, so we do the awake intubation. No problem, right? No sooner said than done?

Who hasn't seen this scenario:

"AAAAAAGH! GAAAAAAAAAAAAAAAGH!"

Damn! I thought he was topicalized.

"Hold still Mr. Smith!" Turning to the cart, maybe *just a little more midazolam. Just a touch, a touch of fentanyl, you know, zap that cough reflex.*

"Gaaaaaaaaaaaaaaaak!"

"Um, doctor, his heart rate is pretty. . ."

"THANK YOU, I KNOW, LET ME JUST GET THIS!"

Thank God, he's finally quieted down. Where are those cords, I saw them just a second ago.

BOOP
BOOP
BOOP
BOOP.

"S#&∗!!! Gimme the mask, gimme the mask!"

Sound familiar?

Telling someone, "Just intubate them awake" is like most advice. You can't argue with it, and it just plain makes sense. But then implementing this great advice can be a real headache. On the tennis court, for example, people give me this advice: "Just hit it in all the time." Well, shucks, great advice. Wonderful advice. If I would just follow that advice, I'd beat Roger Federer at Wimbledon every time.

But it's hard to "hit it in every time."

And it's hard to "just intubate them awake".

Awake intubation can give you adverse hemodynamic

Address reprint requests and correspondence: Richard J. Unger, MD, 7372 Eads Avenue, La Jolla, CA 92037.
E-mail: richard.unger@gmail.com.

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Table 1 Site of action of Dexmedetomidine

History of difficult intubation or mask ventilation
Anticipated difficult laryngoscopy on physical examination
Aspiration risk in patients unsuitable for rapid sequence inductions
Need for neurological assessment after intubation
Potential loss of airway secondary to mass effect, infection, bleeding

consequences[3] and cause trauma—epistaxis, for example. And a number of scenarios make awake intubation nearly impossible: intoxicated and uncooperative patient, C-spine injury where the patient may thrash around and worsen neurologic injury. Some patients with difficult airways may not cooperate due to developmental delays, closed head injury sequelae, or chromosomal abnormalities.

Sedation for awake intubation has its own list of misery, as illustrated above.[4] Mix and match some narcotic and benzodiazepine, and the synergistic effect may convert your "awake" intubation into an "asleep, oh hell, what do I do now?" intubation.

Any magic bullet out there?

Is dexmedetomidine (who are we kidding, you read the chapter title, what else were we going to talk about?) the magic bullet?

Well, not exactly, but it gets pretty *close* to being that magic bullet. Let's see how.

There are a multitude of indications for awake fiberoptic intubation (AFI)[5] (Table 1): history of difficult airway, anticipated difficult direct visualization of larynx, trauma, mass, or infection in the upper airway, and need for neurologic assessment after intubation. There are but a few, but absolute contraindications to AFI do exist (Table 2). Local anesthesia is an integral part of awake airway management, but that will not be discussed in this essay. Instead, we will focus on the safe methods of sedating patients who have indications for AFI. Patient cooperation is paramount when attempting to do AFI, and in addition to informed consent and patient acceptance, judicial administration of intravenous agents can greatly and safely aid the anesthesiologist when facing a patient in need of AFI.

Typically, the better a patient understands and cooperates with topical and regional anesthesia of the airway, the more effective will be the local anesthetics and the better the patient and staff will tolerate the procedure! Our focus in this essay will be to explain the advantages of dexmedetomidine to facilitate these procedures and explain its advantage over more traditional agents.

How does dexmedetomidine compare with other sedative agents? To better understand the advantages of dexmedetomidine, it will be useful to understand the other agents which have been used before dexmedetomidine was available.

Premedication

Premedication is an essential component of AFI. We use premedicants to reduce secretions, enhance hemodynamic stability, create amnesia, and reduce anxiety. Benzodiazepines including midazolam and lorazepam will effectively reduce anxiety and create amnesia. *Caution's the byword when you use these drugs along with dexmedetomidine because dexmedetomidine is synergistic with all sedative medications*.[6]

How about narcotics? No need to use them. Dexmedetomidine plus a little bit of benzodiazepine provides you all the sedation your need. Plus, by avoiding narcotics, you avoid their respiratory depression.

Vagolytic and antisialogogues such as glycopyrrolate are essential premeds to patients undergoing AFI. A dry mouth enhances the efficacy of local anesthetics,[7] enhances visualization of the airway, and helps prevent the bradycardia seen with alpha-2 agonists. Recent work has shown that younger patients with more predominant vagal tone will have a safer journey with alpha-2 agonists if pre-treated with glycopyrrolate.[8]

Other antisialogogues such as hyoscine and atropine have been used, but in themselves have potent CNS side effects. What is the one symptom of atropine poisoning? "Mad as a hatter." Atropine crosses into the brain and you get a central cholinergic effect. Now you've converted your awake intubation into a "madhouse" intubation! Glycopyrrolate is a quaternary ammonium and does not cross the blood–brain barrier, therefore causing no sedation and CNS side effects. Typically, adults given 0.2-0.3 mg of glycopyrrolate will have effective drying of the mucosal membranes with no adverse effects on heart rate. That is, the glycopyrrolate will not *cause* a tachycardia and will not *allow* a bradycardia.

Conscious sedation

Almost all patients require some form of intravenous sedation before AFI. This makes the term AWAKE fiberoptic intubation a bit of a misnomer. Sedation is needed for anxiety and pain associated with the procedure. Important goals of intravenous sedation include maintenance of airway patency and ventilatory drive. Dexmedetomidine is a medication that provides these unique characteristics. The term "Cooperative Sedation" has been aptly coined in relation to the unique sedation achieved with dexmedetomidine.

Table 2 Contraindications for awake fiberoptic intubation (AFI)

Refusal
Uncooperative patient
Bleeding (unable to visualize)
Local anesthetic allergy or toxicity