

The Failed Sedation Solutions for the Oral and Maxillofacial Surgeon



Robert C. Bosack, DDS^{a,b,*}

KEYWORDS

• Failed sedation • Complication • Office-based anesthesia • Patient selection

KEY POINTS

- Failed sedation is the inability to satisfactorily complete a planned procedure (with sedation).
- Failed sedation is an inexorably entwined, multifactorial event, which can be due to unsuitable patient, procedure, or facility choices; inappropriate drug selection/dose; adverse drug response; and/or lack of a shared, structured, and realistic anesthetic plan.
- The true incidence of failed sedation in the oral and maxillofacial surgery office is unknown, but can approach *1 case per month*.
- Failed sedation can best be prevented by communication and realistic alignment of patient expectations.
- Management of failed sedation will most often include lightening anesthetic depth and/or aborting the planned procedure, with patient safety remaining the top priority.

INTRODUCTION AND DEFINITION

Open airway office “anesthesia” can be typically characterized as moderate to deep sedation (with occasional descent into general anesthesia) combined with local anesthesia, with a retreat to moderate sedation for longer cases.¹ Anesthetic protocols in the oral and maxillofacial surgery office routinely include intravenous midazolam and fentanyl, often supplemented with ketamine, nitrous oxide, and/or incremental small propofol boluses. Oral premedication with a benzodiazepine or alpha-2 agonist is sporadically entertained. The 3-fold purpose of these medications is to (1) permit, facilitate, and expedite painful or threatening procedures by blocking pain, and decreasing or eliminating perception; (2) mitigate physical and emotional reactions to painful stimuli; and (3) improve patient satisfaction. These three

objectives are best accomplished by providing safe, reversible, drug-induced analgesia, amnesia, anxiolysis, and behavior control in a “stress minimized” setting.² Sedation success can be loosely defined as the ability to safely complete the intended procedure, while minimizing pain, anxiety, recall, and the neuroendocrine stress (fight or flight) response (**Box 1**), and maintain patient satisfaction. The event of inability to successfully complete the planned procedure, to the satisfaction (expectation) of the provider and/or patient/guardian, is termed *failed sedation*. Failed sedation could also include patient combativeness, the (unplanned) use of physical restraint, unexpected postanesthetic recall of intraoperative awareness, and adverse drug effects (benzodiazepine disinhibition and ketamine delirium). “Bad anesthesia begets bad surgery” (Robert Campbell,

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^a Private Practice, 16011 South 108th Avenue, Orland Park, IL 60467, USA; ^b University of Illinois at Chicago College of Dentistry, 801 S. Paulina, Chicago, IL 60612, USA

* Corresponding author. 16011 South 108th Avenue, Orland Park, IL 60467.

E-mail address: r.bosack@comcast.net

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Box 1**The neuroendocrine stress response**

The neuroendocrine stress response is an expected, predictable reaction to any disruption in homeostasis. It involves the activation of the sympathetic (fight or flight) reaction (neuro) and the activation of the hypothalamic-pituitary axis (endocrine), ultimately resulting in the release of glucocorticoids, which facilitates the former, among many other physiologic mechanisms. Clinically, as a reaction to pain, this response can include tachycardia, hyperventilation, pupillary dilation, peripheral vasoconstriction, central vasodilation, hypertension, agitation, and combativeness, among others.

Data from Miller DB, O'Callaghan JP. Neuroendocrine aspects of the response to stress. Metabolism 2002;51(6 Suppl 1):5-10.

DDS, personal communication, 2015). Compromised surgery includes torn flaps, damage to adjacent structures, and so forth. For purposes of this discussion, failed sedation will not include complications of anesthesia, such as adverse cardiac and pulmonary responses, nausea, vomiting, pulmonary soiling, and myocardial ischemia, among others. Certainly, however, anesthetic complications cannot be isolated from failed sedations.

CAUSE AND SPECTRUM OF FAILED SEDATION

Failed sedation is a multifactorial event that can be due to and compounded by suboptimal (1) drug choices, doses, adverse side effects, and/or technique; (2) patient selection, preparation, and drug responses; and (3) surgical case selection. Practitioner training, experience, preparation, ability, and willingness to progress to general anesthesia can also play a role in inability to successfully complete a planned procedure.

Drug choices and anesthetic technique play a large role in the success or failure of sedation in the oral and maxillofacial surgery office. It is useful to recall the definition of deep sedation/analgesia, which is a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully following repeated or painful stimuli and general anesthesia, where patients are unarousable. Therefore, when in a state of deep sedation, if local anesthesia is inadequate, or if systemic analgesia is inadequate, one would anticipate patient arousal, movement (combativeness), increases in blood pressure and heart rate, and so forth: the neuroendocrine sympathetic stress response. Many would interpret this as failed sedation. The most obvious scenario for this circumstance is not waiting for

administered drugs to take full effect, or rushing the medication. Possible strategies for remediation include waiting for drug action, ensuring profound local anesthesia, redosing or introducing alternative drugs, progression to full general anesthesia (an attendant side effects), or sedation/case abandonment.

Two other “failed sedation” scenarios deserve mention. A one-drug, benzodiazepine regimen can, at times, prove to be less than satisfactory to obtund perception and reaction to the administration of local anesthesia, and can, in fact, increase cold, heat, and electrical pain perception.³ Finally, stage II excitement (between deep sedation and general anesthesia) can occasionally be experienced, with attendant patient movement and adverse physiologic changes interfering with case completion.

Ketamine delirium⁴ is not always predictable, but may be predisposed at higher doses, in adolescent female patients, and in those with psychiatric illness. Because there is no reversal for ketamine, management includes minimizing external visual, auditory, and tactile stimulation and waiting for drug redistribution and elimination. Paradoxical benzodiazepine disinhibition (psychomotor and cognitive dysfunction) is also a possibility that tends to occur in younger patients, perhaps seen more often in those who struggle to internalize their pre-sedation anxiety (impulse control). Treatment options include benzodiazepine reversal, use of other agents, or waiting for drug diffusion away from central receptors. Benzodiazepine redosing is rarely successful in this instance. Nelson and colleagues⁵ concluded that sedation outcome was significantly associated with high scores over the temperament domains of effortful control, attention focusing, and inhibitory control, as determined by a questionnaire. However, this begs the question that successful attempts at sedation would exclude those very patients who might benefit the most from it.

Anesthetic technique depends not only on drug choices but also on real-time, robust, moment by moment monitoring of drug effect.⁶ Intense monitoring oftentimes will provide not only a window of safety but also invite possibilities of alternative drug strategies, as interventions become proactive rather than reactive. Similarly, rapid detection of adversity can limit the duration and severity of adverse side effects, minimizing their contribution to failed sedation.

Patient selection and responses to anesthetic drugs can also contribute to failed sedation. Virtually all anesthetic drugs have adverse side effects, which include not only disinhibition and delirium but also loss of upper airway tone, ventilatory

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