PART I MINIMALLY INVASIVE ANESTHESIA (MIA)® FOR MINIMALLY INVASIVE SURGERY

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Propofol Ketamine with Bispectral Index (BIS) Monitoring

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INTRODUCTION

Anesthesiologists are trained to administer anesthesia for surgery. Elective cosmetic surgery is commonly performed in an office-based facility with patients discharged to home. However, elective cosmetic surgery differs from elective or emergency surgery in many substantial aspects (see Tables 1-1 and 1-2).

"Cosmetic surgery is almost always elective, and patients are almost always in good health. The patient, however, is willing to risk this good health (at least to a limited extent) in order to experience improvements in physical appearance, and perhaps more importantly, self-esteem, body image, and quality of life."[1]

There is *no medical indication* for elective cosmetic procedures, excluding breast reconstruction post-mastectomy. One may consider risk-benefit ratios of differing anesthetic regimens in *medically* indicated surgery. However, surgery *without* medical indication should not accept *any* avoidable risk. Halogenated inhalation anesthetics are triggering agents for malignant hyperthermia (MH),[2] carry an increased risk of deep venous thrombosis with potential pulmonary embolism,[3] and are emetogenic.[4] If the patient is interested and the surgeon is willing, **all** cosmetic procedures *can* be performed under local *only* anesthesia. Therefore, *any* additional anesthetic agents should be subject to the highest justification.

Most patients desire *some* alteration of their level of consciousness from fully awake through completely asleep. Given that all known risks *should* be avoided, when possible, then which agents are best suited to the task, what monitors should be employed, and to what level

Table 1-1. Elective cosmetic procedures

Commonly performed cosmetic surgical procedures. All procedures have successfully been anesthetized with PK MAC/MIA™ technique in the office-based setting.

1. Rhinoplasty (closed or open)
2. Liposuction or suction assisted lipoplasty (SAL)
3. Blepharoplasty (open, transconjuctival, or endoscopic)
4. Rhytidectomy (open or endoscopic)
5. Breast augmentation, subglandular, subpectoral (via areaolar, inframammary, transaxilllary, or transumbilical approach)
6. Hair transplantation with or without scalp reduction
7. Facial resurfacing (laser, chemical peel, or mechanical dermabrasion)
8. Brow lift (coronoplasty or endoscopic)
9. Abdominoplasty (classical or simple skin)
10. Otoplasty
11. Genioplasty (mandibular advancement or recession)
12. Facial implants (malar and mandibular with silicone or autologous fat)
13. Lip enlargement (autologous fat transfer, radiated cadaver material [Alloderm®], Gortex® extrusions, Restylane,® Juvaderm,® etc.)
14. Platsyma band plication
15. Composite procedures; i.e., (a) endoscopic brow lift and endoscopic rhytidectomy, with open platysma band plication, (b) blepharoplasty, rhinoplasty, and rhytidectomy, or (c) breast augmentation with abdominoplasty

of anesthesia should be administered (i.e., minimal sedation ["anxiolyis"], moderate ["conscious"] sedation, deep sedation, or general anesthesia [GA])? (See Appendix 1-1, Defining Anesthesia Levels). If better outcomes are the goal, doesn't minimally invasive anesthesia for minimally invasive surgery make sense?[5] (See Table 1-3.)

WHY IS MINIMALLY INVASIVE ANESTHESIA® IMPORTANT?

"Less is more" is a Mies Vanderohe principle applied to the Bauhaus school of minimalist architecture. "Doing more with less" is a Buckminster Fuller concept of housing applied to his geodesic domes.

Table 1-2. Cosmetic procedures by type from PK MAC/MIA™ technique case log March 26, 1992 – March 26, 2002 [12]

	N	%
Liposuction	663	(25)
Breast augmentation	489	(18)
Facial resurfacing mechanical abrasion, chemical peel, or laser resurfacing	389	(14)
Rhytidectomy	305	(11)
Blepharoplasty	198	(7)
Rhinoplasty	81	(3)
Fat transfer	57	(2)
Abdominoplasty	54	(2)
Composite or misc. procedures	447	(18)
Total	2,683	(100)

"We hold the basic premise that the less the involvement of the patient's critical organs and systems (i.e., the lower the concentration of the agent, or the less 'deep' the anesthesia), the less will be the damage to the patient, whether this be temporary or permanent."[6]

"For the anesthetic itself, overall experiences indicate that the least amount of anesthetic that can be used is the best dose. Local and monitored anesthesia care (MAC) is preferable to regional. Regional techniques are preferable to general anesthesia."[7]

Table 1-3. Minimally invasive surgeries appropriate for BIS-monitored PK MAC, the MIA™ technique

1. *All* cosmetic procedures (see Table 1-1)
2. Gyn: laparoscopy (tubal ligation, fulgeration endometriosis)
3. Ortho: arthoscopy
4. Urology: lithotripsy
5. Gen. surg.: herniorraphy & breast cancer surgery
6. Neuro: microdiscectomy, microlaminectomy, carpal tunnel release
7. sedation for morbidly obese
8. peripheral injuries in U.S. Army field hospitals in Iraq, Afghanistan

Cases being performed with PKR[a] TIVA
1. U.S. Army neurosurgery in Iraq.
[a]Propofol-Ketamine-Remifentanil

"When possible, procedures longer than three or four hours should be performed with local anesthesia and intravenous sedation because general anesthesia is associated with deep venous thrombosis at much higher rates under prolonged operative conditions."[3]

"Newer techniques for intravenous sedation that include the use of propofol, often in combination with other drugs, have made it possible to perform lengthy or extensive procedures without general anesthesia and *without the loss of the patient's airway protective reflexes.*"[9]

"When you can measure what you are speaking about, and express it in numbers, you know something about it; but when you cannot measure it, when you cannot express it in numbers, your knowledge is of a meager and unsatisfactory kind; it may be the beginning of knowledge, but you have scarcely, in your thoughts, advanced to the stage of science." (William Thompson, knighted Lord Kelvin. Popular lectures and addresses [*1891–1894*])

The bispectral index (BIS) monitor facilitates a numerical expression of the *hypnotic* component (anesthesia = hypnosis + analgesia) of the anesthetic state and may permit a reasonable inference about the analgesic state. Heart rate, blood pressure, and other clinical signs are notoriously unreliable indicators of anesthetic depth.[10] BIS provides new information about patients that is simply unavailable from any other vital or clinical sign.[11] BIS, as an index, has no units. The scale is 0–100, with 100 representing awake and zero representing isoelectric (or zero) brain activity. Hypnosis compatible with general anesthesia (GA) occurs between BIS 45–60. BIS 45–60 with *systemic* analgesia defines general anesthesia. BIS 60–75 with adequate *local* analgesia is a major part of the MIA™ technique. Patients who received MIA™ neither hear, nor feel, nor remember their surgical experience.[12]

Monk et al. published an associated 20% increase in the one-year mortality risk *associated* with every hour of BIS <45.[13] Therefore, BIS <45 for cumulative periods greater than one hour must be considered as overmedicating.

The routine practice of overmedicating for fear of undermedicating is no longer a desirable or acceptable practice (see Table 1-4).

Monk et al. postulated that the increase in one-year anesthetic mortality might be related to an inflammatory response from excessively deep anesthesia.[13] A more recent prospective, randomized controlled study demonstrated increased C-reactive protein levels with BIS <45 for more than 50% of the cases.[14]

Table 1-4. BIS levels and levels of sedation/anesthesia

BIS	Sedation/Anesthesia Level
98–100	Awake
78–85	Minimal Sedation ("Anxiolysis")
70–78	Moderate ("Conscious") Sedation[a]
60–70	Deep Sedation[b]
45–60 + *systemic analgesia*	General Anesthesia[c]
<45, >1 hr.	Overanesthetized![13]

[a] With moderate sedation, *passive* maneuvers like extension and rotation of the head or shoulder pillow may be all that are necessary to maintain the airway.
[b] With deep sedation, *active* maneuvers, like nasal airway or LMA, may be required to maintain airway patency.
[c] See Appendix 1-1.

The BIS monitor does not replace traditional vital-sign monitoring, that is, EKG, NIABP, SpO_2, (or $EtCO_2$ when indicated). When measured, the $EtCO_2$ typically runs between 38–42 with the MIA™ technique. The $EtCO_2$ offers the display of the waveform of the patient's respiration. Many experienced anesthesiologists are capable of assessing adequate respiratory movement without this information. Over 3,000 PK MAC cases have been safely anesthetized without $EtCO_2$ monitoring.

Titrating anesthesia with BIS trend is limited by the fact that the processing required for the BIS algorithm is delayed 15–30 seconds behind real time. This delay has given rise to the legitimate criticism that BIS does not predict patient movement. BIS, a measure of the hypnotic state, was not designed to predict patient movement (see Chapter 3).

EMG is the instantaneous display of the frontalis muscle activity if the XP software version of the BIS A2000, or later, is used. Inadequate analgesia leading to patient movement is predictable if the EMG is selected from the advanced screen menu to trend as a secondary trace. A spike in EMG (when BIS is 60–75, in spontaneously breathing patients) nearly always predicts inadequate analgesia, preceding patient movement (see Fig. 1-1). The anesthesiologist should utilize the 15–30 second delay in the change of the BIS value to simultaneously bolus propofol while encouraging the surgeon to supplement the local analgesia.

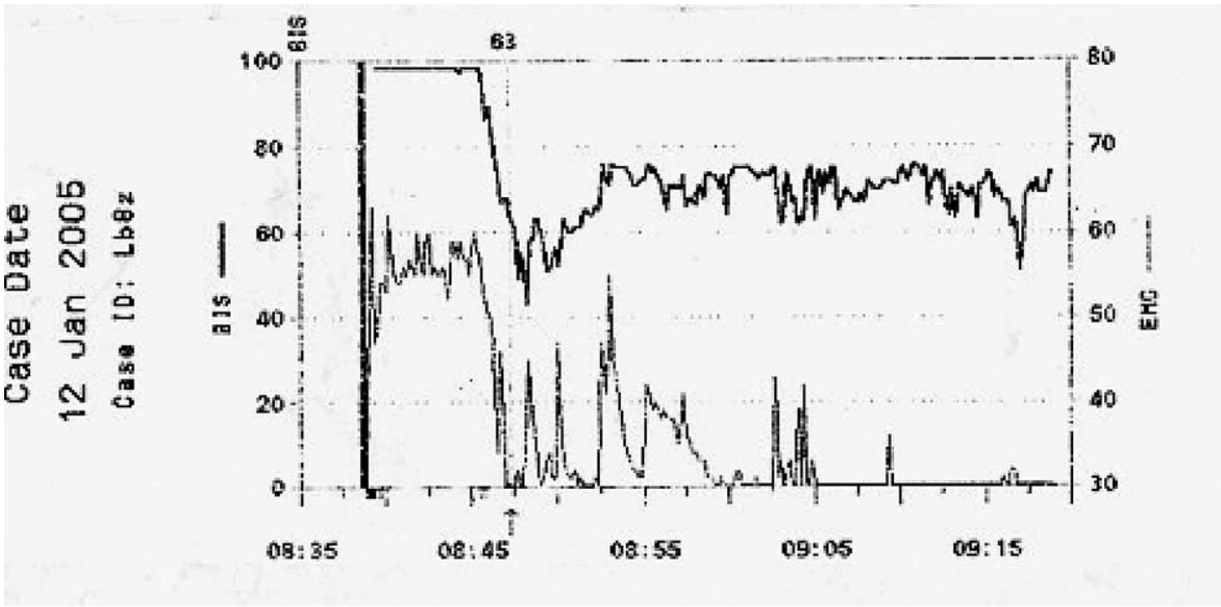

Figure 1-1. Incremental propofol induction began 08:45. Ketamine 50 mg IV administered 08:47, BIS = 63. In this particular case, BIS increases post-ketamine dose. However, the increase does **not** defeat the ability to titrate propofol to BIS 60–75!

Postoperative Nausea and Vomiting (PONV)

Macario et al. conducted a statistically validated survey of a panel of expert anesthesiologists on what postoperative anesthetic outcome *they* believed patients most wanted to avoid.[15] The anesthesiologists concluded that **pain** was the number one anesthesia outcome patients most desired to avoid. A follow-up, similarly statistically validated survey of patients' anesthesia outcomes they most desired to avoid was **emesis**![16] Clearly, a disconnect exists between what anesthesiologists believe about their patients and what the patients actually want most to avoid. A potential explanation could be that patients who consent for elective surgery *expect* to have some postoperative discomfort but do not want their pain to be compounded by emesis.

How are PONV, preemptive analgesia, and postoperative pain management related?

There is a consensus among PONV authorities like Apfel, Chung, Gan, Scuderi, and White, that both inhalational anesthetics and opioids are emetogenic agents. "In the *context* of [emetogenic] anesthesia, postoperative pain management and opioid related PONV remain problems."[17] In the context of emetogenic anesthesia, experts advise "multimodal" prophylaxis in the highest risk group.[18]

Apfel's recent NEJM article identifies the highest PONV risk group of patients as nonsmoking females, with a history of previous PONV and/or motion sickness, having emetogenic (i.e., elective cosmetic) surgery of two or more hours.[4] Apfel's criterion of high risk applies *exceptionally* well to Friedberg's previously referenced series of 2,683 patients.[12]

Elective cosmetic surgery anesthesia for the "rich and famous" of Beverly Hills and Newport Beach is the highest risk PONV population! This conclusion reflects the southern California geographic bias of the author. There are many other such communities worldwide.

The MIA™ technique is not perfect but *contextually* nonemetogenic. Without ***any*** antiemetic prophylaxis, this highest risk group of patients experienced a total of thirteen PONV events for an unprecedented 0.5% PONV rate![12] A 50 mg dissociative dose of ketamine at BIS <75 propofol levels eliminates the noxious input of the injection of local analgesia while avoiding emetogenic agents like the halogenated inhalational vapors and intravenous opioids.

Lidocaine provides intraoperative analgesia with bupivicaine providing postoperative analgesia. In this *context*, it has been extremely rare for patients to require (emetogenic) opioid relief of their postoperative discomfort.

Elimination of all emetogenic triggers defines nonopioid, preemptive analgesia (NOPA). NOPA is the hallmark of the MIA™ technique. In Friedberg's fifteen-year experience, no patients have been admitted to the hospital following PK MAC/MIA™ technique for either PONV or unmanageable pain.

Beware Laryngospasm

No technique is perfect. Classical laryngospasm can be diagnosed by the characteristic "crowing" sound generated by a small gap in the vocal cords owing to their incomplete closure. With ketamine-associated laryngospasm, the vocal cords most commonly close completely. Hence, only rarely will crowing noise alert the anesthesiologist to impending desaturation. Additionally, the usual remedy of positive pressure ventilation combined with anterior jaw thrust is *completely* ineffective. The anesthesiologist must pay particular attention to *sneezing* or *coughing* as the only prodrome warning him of impending laryngospasm.

The treatment of choice is a rapid IV bolus of lidocaine 1 mg · lb^{-1} or 2 mg · kg^{-1}.

Concern about adding more lidocaine in patients receiving relatively large amounts of lidocaine local analgesia has led other anesthesiologists to prefer to deepen the propofol level by adding a 50 mg propofol bolus to break the laryngospasm. However, when IV lidocaine *has* been administered for laryngospasm, no stigmata of lidocaine toxicity have been observed. The BIS showed no decrease in response to the IV lidocaine bolus. There was no transient hypotension or widening of the EKG complex during the case. No patient complained of tinnitus, tremulousness, or metallic taste on the tongue after emergence.

Administering succinylcholine (SCH) to break the spasm is suboptimal because SCH adds unnecessary (and avoidable) risk as an MH triggering agent. (Neither propofol nor ketamine are MH triggering agents.) Further, the myalgias associated with SCH make the agent totally unacceptable in the elective cosmetic surgery patient.

Waiting until desaturation occurs after the prodrome will add a substantial amount of time until the lidocaine can circulate to anesthetize (and open) the vocal cords. Desaturation increases the physiologic stress to the patient. The alarm of the pulse oximeter, accompanied by the bluish discoloration of the patient, increases the psychological stress to the anesthesiologist, surgeon, and operating room nursing staff. This disturbing scenario is best minimized by promptly giving IV lidocaine when the patient coughs or sneezes.

WHAT IS CLONIDINE-PREMEDICATED, BIS-MONITORED PK MAC, OR THE MIA™ TECHNIQUE?

Something old (ketamine), something new (BIS-monitored propofol hypnosis), something borrowed (diazepam ketamine technique[19]), no one blue (SpO_2 >90% on room air).

Why Ketamine?

The brain cannot respond to stimuli it does not receive. *Critical concept*: GA does not reliably block all incoming noxious stimuli! The "wind-up" phenomenon,[20] mediated by the NMDA receptors, is often invoked to explain acute postoperative pain after general anesthesia, as well as the formation of chronic pain states.

"Dissociation" refers to a patient who, under the influence of ketamine, remains motionless in response to noxious stimuli.

Based on clinical observation, the NMDA receptor block from a 50 mg dissociative dose of ketamine reliably blocks all incoming noxious stimuli to the cortex (the so-called mid-brain spinal) for a period of 10–20 minutes. After obtaining an equal dissociative effect with a 50 mg ketamine dose in both 90-pound female and 250-pound male patients, the author concluded that *the number of NMDA receptors does not vary with patient body weight in adults.*

Preemptive analgesia is most consistently observed when the NMDA receptors are saturated *prior* to noxious stimulation. Acetaminophen 1,000 mg po is adequate for postoperative pain management (for the few patients who request it) in the context of clonidine-premedicated, BIS-monitored PK MAC patients.[12] (See Table 1-5.)

Making Ketamine Predictable

In *other* contexts, ketamine has a well-deserved reputation for causing hypertension, tachycardia, and an unpredictable 20% of patients experiencing hallucinations or dsyphorias.[21] Hypnotic doses of propofol block ketamine-induced hallucinations as well as undesirable hemodynamic sequellae.[22] Being able to assign a numerical value

Table 1-5. Ketamine tips

1. 80% patients achieve dissociative effect with 25 mg ketamine, 98% with 50 mg ketamine. No "down side" to 50 mg dose as long as BIS <75. Wait 2–3 min. before injecting local. Wait an additional min. if patient is reactive before administering more ketamine.
2. Preemptive analgesia effect is variable when inadequate dissociative effect is obtained. Saturate NMDA receptors!
3. *All adult patients, independent of body weight, require 50 mg ketamine initial dose to saturate NMDA receptors.*
4. Reinjection of previously injected field does NOT require more ketamine.
5. Consider injecting both sides with initial ketamine dose.
6. If prep. is cold, consider injecting 25 mg ketamine 2–3 min. before prep. or consider warming prep. solution!
7. With experience, less ketamine is administered. Friedberg's case log of the last 500 cases (of 2,683 patients) showed 80% performed with either one or two 50 mg doses of ketamine.[12]
8. Mixing propofol with ketamine is TIVA[23] not MAC.
9. Do not exceed an aggregate total of 200 mg ketamine.
10. Do not give ketamine in the last 20–30 minutes of a case.

with BIS to the level of propofol hypnosis, *prior* to administering the ketamine, was an enormous breakthrough in making ketamine a predictable agent. Not only could the initial ketamine dose be administered without problems, but also subsequent doses, when needed, could be given with assurance.

First, create a *stable* level of propofol in the brain by performing an incremental, not bolus, induction. The incremental induction maintains spontaneous ventilation, commonly maintains masseter tone, avoids propofol waste, and is less apt to produce induction hypotension. Incremental propofol induction provides hypnosis with a minimal physiologic and pharmacologic trespass to the patient. *Lesser trespass increases patient safety.*

Lesser trespass increases the probability of maintaining the SpO_2 >90% on room air (i.e., room air, spontaneous ventilation, or RASV). *Key concept*: Titrate propofol to BIS <75 *before* giving the ketamine! Do NOT give ketamine at BIS >75.

Table 1-6. Clinical pathway for MIA™ technique

1. Clonidine 0.2 mg PO 30–60 min preop (Systolic >100, body weight >100 pounds).
2. Glycopyrrolate 0.2 mg IV with 2 ccs 1% lidocaine plain.
3. Incrementally *titrate propofol to BIS <75* with multiple, sequential *150 ug · kg^{-1} · 20 sec.* mini-boluses. *N.B.* If pump does not have a bolus feature, set initial rate to *450 ug · kg^{-1} · min^{-1}* and reduce the rate toward 50 as soon as the EMG begins to decrease.
4. Basal propofol infusion rate *50 ug · kg^{-1} · min^{-1}*.
5. Ketamine 50 mg IVP @ *BIS <75* 2–3 minutes *prior* to injection local anesthesia.
6. Adjust basal propofol rate upward to maintain BIS 60–75 if ketamine causes an increase.
7. Inject adequate local analgesia.
8. Administer more ketamine only after two reinjections of the field fail to eliminate patient movement.
9. Maintain propofol at BIS 60–75, EMG 0 on BIS scale, 30 on EMG scale.
10. Bupivicaine in field before closure, especially for browlift, subpectoral breast augmentation, and abdominoplasty.

Because the elective cosmetic surgical patient tends to be healthy, cardiac output and redistribution from the brain tend not to be significant factors in altering established brain levels of propofol. However, the nineteenfold interpatient variation in propofol hydroxylation may play a significant role in the ability to maintain a stable level of propofol in the brain.[23] Measuring an individual patient's *brain* response to propofol with BIS would appear to be a more effective strategy than employing target controlled infusions (TCI) to achieve specific *blood* levels of propofol (see Table 1-6).

Premedication

PK MAC was derived from diazepam ketamine MAC technique, which was first published in 1981.[19] Vinnik clearly enumerated that only ***after*** the patient was soundly asleep from the diazepam was the ketamine to be administered.[19] Diazepam hypnosis, followed by ketamine dissociation, followed by local anesthetic injection was Vinnik's clinical pathway. Although Guit was the first to publish the combination of propofol and ketamine, the technique was described as a total intravenous anesthetic (TIVA).[24] TIVA

strongly implies that the local analgesia injected by the surgeon is not essential for the success of the TIVA technique. In contradistinction, the surgeon's local analgesia *is* essential for the success of PK MAC.

Guit's TIVA technique was unknown to Friedberg in 1992 when Friedberg embarked on *replacing* Vinnik's diazepam with propofol. The surgeons quickly complained about the cost of the propofol and pleaded for relief. Friedberg added midazolam in an effort to reduce the amount of propofol. From March 26, 1992 through March 26, 1997, the case log Friedberg maintained contained patient's names, dates, surgeons, patient age, gender, weight, surgical procedure(s) (see Table 1-2), midazolam, propofol, ketamine, and anesthesia times.[8] Propofol rates, mg · min^{-1} and ug · kg^{-1} · min^{-1}, were calculated *retrospectively*.

If 2 mg midazolam was good, perhaps 4 mg midazolam could be better for propofol-sparing purposes. In the aforementioned case log, a total of 354 patients received 0 mg midazolam, 316 patients received 2 mg, and another 303 patients received 4 mg midazolam premedication from 1992–97. No consistent, incremental relationship could be established in propofol savings between the 0, 2, and 4 mg midazolam groups.[8] In June 1997, Friedberg eliminated the midazolam from PK MAC.

In September 1997, Oxorn published a very elegant Level I study confirming Friedberg's uncontrolled, clinical experience in 973 patients.[25] Oxorn reported that there was no statistical difference in either induction or maintenance doses of propofol between those patients who received 2 mg midazolam premedication and those who received none.[25] *However, the unexpected finding was that a statistically significant threefold number of patients who received midazolam required pain medication in the PACU.*[25]

From July 7, 1997, through December 21, 1998, 268 patients received BIS-monitored PK MAC *without* premedication, midazolam, or other benzodiazepine. During BIS-monitored propofol hypnosis, there were no patients who suffered from hallucinations or a lack of amnesia. This experience led Friedberg to conclude that benzodiazepine premedication was superfluous to provide amnesia or to prevent hallucinations *in the presence of BIS monitoring.* Some of these patients were included in a subsequent publication.[26]

Patients continued to request premedication to calm them. After attending the New York Postgraduate Assembly (PGA) in December 1998, Friedberg returned with the renewed notion of adding po clonidine as a premedication. Like Vinnik's concept of administering sleep doses of diazepam to block ketamine hallucinations, clonidine for premedication had also been previously reported in the plastic surgery literature. [27,28]

Inconsistent propofol sparing results were observed with 0.1 mg po clonidine. A therapeutic clonidine dose should be in a range between 2.5–5.0 ug · kg^{-1}.[29] Clonidine 0.2 mg mg po achieves that range in patients weighing between 95–175 pounds. The higher dose of clonidine provided consistent propofol sparing results and further refinement of BIS-monitored PK MAC.[30]

From January 26, 2001 to September 2002, rofecoxib 50 mg po was added to the clonidine. When the drug was voluntarily withdrawn from the market, rofecoxib was deleted from the premedication. While the *addition* of the rofecoxib appeared to benefit the patient, the *deletion* of the agent did not appear to increase (the already few) postoperative patient complaints of discomfort.

At the present time, only clonidine 0.2 mg po (30–60 minutes preoperatively) and glycopyrrolate 0.2 mg with 2 cc 1% lidocaine IV are given as premedication (see Table 1-6).

Fluid Management

The long-standing teaching that patients who are NPO after midnight are at least 500–1,000 ccs behind on their fluids is not especially relevant for elective cosmetic surgery patients. As stated earlier, these are by and large essentially healthy patients who are far different from the debilitated ward patients on whom most anesthesia trainees learn about anesthesia. Elective cosmetic surgical patients are not "dry." Vasodilating anesthetics are no longer being administered. Lastly, large fluid shifts and blood loss are atypical experiences in most elective cosmetic surgery.

Other authors have analogized the insult produced by liposuction to that of a burn injury. However, burn patients do not have compression garments applied to obliterate the "third space" created by the aspiration of subcutaneous fat.

Fluid replacement regimens based on experience in burn patients areinappropriate for liposuction patients.

Especially for cases up to 5,000 ccs of liposuction, fluid replacement should remain modest, that is, not more than 1,000 ccs. Otherwise, one may risk fluid overload,

Table 1-7. MIA™ airway algorithm (assumes incremental propofol induction)

1. Extend and laterally rotate head, one side may have better gas exchange than the other.
2. Insert shoulder (not neck) pillow to increase force of extension.
3. Insert lubricated nasal airway (#28 FR most commonly).
4. Insert lubricated LMA (#4 most commonly).
5. No ET required: >15 yrs, >3,000 patients; no opioids, benzodiazepines, or muscle relaxants.

Table 1-8. Local anesthesia tips

1. PDR limit of 500 mg lidocaine with epinephrine (7 mg · kg^{-1}) is outdated and overly conservative. Neither the 2005, 2006 nor the 2007 (print or electronic) editions of PDR have *any* entry for injectable lidocaine!
2. 200 ccs of 0.5% lidocaine (1,000 mg) with epinephrine is well tolerated and without sequellae of toxicity
3. Tumescent or "wetting" solution = 500 mg lidocaine, 1 mg epinephrine in 1,000 ccs NSS (Klein) or LR (Hunstead)
4. 5,000 ccs of tumescent solution = 2,500 mg lidocaine
5. 5,000 ccs of tumescent solution in a 60 kg female patient = 42 mg · kg^{-1}
6. Avoid >50 cc 0.25% (125 mg) bupivicaine for postoperative analgesia.

pulmonary edema, and dilution of platelets and other coagulation factors.

Another unaesthetic consequence of 2,000–4,000 ccs fluid replacement in this patient population is enuresis on the operating room table. This will embarrass the patient and annoy the nurse who had to clean it up. Catheterizing the patient to compensate for inappropriate fluid administration exposes the patient to the risk of an unnecessary bladder infection.

Patients who experience caffeine withdrawal headache without their morning caffeine are encouraged to drink their cup of coffee black or with non-dairy creamer, if necessary. Apple juice or water is permitted up until an hour before surgery. Patients who are hungry upon awakening are encouraged to have toast and jam. Simple carbohydrates and sugars are rapidly absorbed by the stomach and pose no real threat to patient safety. It is far better to have the patient arrive without hypoglycemia. Patients are encouraged to void before getting on the operating table. (See Table 1-7.)

Major Confounding Principle

A blanched surgical field does *not* guarantee adequate surgical analgesia. More local analgesia resolves the patient movement 99% of the time. Administer more ketamine only after two reinjections of the field fail to eliminate patient movement.

BIS becomes much more than a simple tool with which to titrate propofol. BIS becomes a case management tool. By being able to demonstrate adequate propofol levels (i.e., BIS 60–75) *during* patient movement, the surgeon can be educated to inject more analgesia. In addition to the *initial* injection of the local analgesia, the patient is spared noxious, painful input during the surgery. The brain cannot respond to stimuli it does not receive. *Postoperative pain management begins intraoperatively!* Reproducible preemptive analgesia occurs under conditions of adequate dissociation secondary to the saturation of the NMDA receptors. (See Table 1-5.)

BIS as Fianchetto

From Italian, *fianchetto* is a chess term meaning a "double move." In a "binary" system of anesthesia (hypnosis + analgesia = anesthesia), being able to measure hypnosis permits an inference about the adequacy of analgesia. Adequate analgesia produces de facto muscle relaxation for minimally invasive surgery. BIS 60–75 with EMG = 0 (on the BIS scale, 30 on the EMG scale) defines adequate hypnosis for the MIA™ technique. Therefore, *adequate* hypnosis in the presence of patient movement (usually preceded by a spike in EMG) infers *inadequate* analgesia!

Postoperative Pain Management

In the context of clonidine-premedicated, BIS-monitored PK MAC, now formally known as the MIA™ technique, postoperative pain is minimal to nonexistent. Part of this phenomenon may be explained by having patients emerge

from propofol with the clonidine still in effect. Patients who have lower anxiety levels, secondary to lowered catecholamines from the clonidine, tend to have less pain complaints. In the diethyl ether era, "stormy induction, stormy emergence" was the common rationale for premedicating surgical patients. Preoperatively, a clonidine-premedicated patient may not appear drowsy but, upon questioning, usually admits to feeling "calmer." A further explanation for the remainder of the observation of minimal-to-no postoperative pain appears to be the phenomenon of preemptive analgesia.

With the dissociative effect of ketamine, no noxious signals reach the cortex during the injection of local anesthesia. *GA does NOT reliably block all incoming noxious stimuli.* Use the BIS to not only maintain hypnosis at 60–75 but also to assure inadequate local analgesia is dealt with appropriately (i.e., more local) and not by subterfuge (i.e., more ketamine, propofol, or opioids). Lastly, bupivicaine, especially for browlift, breast augmentation, and abdominoplasty, provides long-lasting nonopioid relief. Do not exceed a total of 125 mg bupivicaine (or 50 ccs 0.25%) for postoperative analgesia. Because the bupivicaine quickly binds to tissue, it is necessary only to splash it into the operative field. Some surgeons prefer to close the wound and inject the bupivicaine retrograde up the suction drainage tube(s). Both approaches with bupivicaine are effective.

All of the anesthesiologists' efforts to prevent PONV and effect adequate pain management may be for naught if the surgeon discharges the patient home with an opioid-containing analgesic (i.e., Vicodin® or Tylenol #3®). Darvocet® or other similar nonopioid analgesics may provide an increment of relief greater than 1,000 mg acetaminophen every six hours. Oral diazepam is especially effective for decreasing the muscle spasm associated in subpectoral breast implant patients. *N.B. This is also a useful strategy for any other submuscular implants; i.e., gluteal.*

The few patients who do complain of pain present a differential diagnosis of "central" (or supratentorial) versus "peripheral" (infratentorial) pain. *Both complaints are real.* Some patients may complain of pain when they had been predominantly immobile for the surgery. This pain is more likely to be "central" in origin. This type of patient may respond better if 50 mg po diphenhydramine (Benadryl®) is added to the 1,000 mg acetaminophen (Tylenol P. M.®). More experience with the MIA™ technique will eliminate most of the patient movement seen with inadequate local analgesia. These patients may require ketorolac 30–60 mg IV to deal with "peripheral" pain issues. As the surgeon becomes more willing to inject additional local analgesia *during* the case when patient movement occurs at BIS 60–75, fewer issues of "peripheral" pain will be manifest. None of the more than 3,000 PK MAC patients has ever required hospital admission for intractable pain. (See Table 1-9.)

Table 1-9. Errors to avoid

1. Ketamine before propofol: NO
2. Ketamine at BIS >75: NO
3. Bolus propofol induction: NO
4. Inadequate local analgesia: NO
   BIS as *fianchetto* for adequate propofol *and* lidocaine
5. Opioids instead of more lidocaine: NO
6. Ketamine instead of more lidocaine: NO
7. >200 mg *total* ketamine or any in last 20 min. of case: NO
8. Tracheostomize patient for laryngospasm instead of IV lidocaine: NO
9. SCH instead of lidocaine for laryngospasm: NO

CONCLUSION

One must empathize with those who, understandably, have difficulty believing that a subpectoral breast augmentation in combination with a classical abdominoplasty can be performed as an office-based or day surgery without PONV or postoperative pain management issues. "Cognitive dissonance" is the psychological principle that precludes individuals from believing what they observe when it sharply contradicts what they have been taught to believe.

The On-Q® pump may have some additional value; but in the context described in this chapter, it offers little pain management benefit to offset the additional $280 cost (in 2005 dollars). While dexmedetomidine may possess 8 times the alpha$_2$ agonist potency of clonidine, it is 400 times more expensive (2005 dollars) and more tedious to

administer. There are no current plans to replace clonidine with dexmedetomidine in the MIA™ technique.

The MIA™ technique reproducibly provides preemptive analgesia and is not technically difficult to execute. It does, however, require the active cooperation of the surgeon. Surgeons have become more interested in the use of local anesthesia to diminish PONV and postoperative pain management problems they perceive to be produced by the emetogenic agents the anesthesiologist chooses to administer.

Although initially developed for office-based, elective cosmetic surgery, the MIA™ technique is by no means limited to these types of cases (see Table 1-3). The MIA™ technique offers superior outcomes to alternative forms of anesthesia (see Part II) for cosmetic surgery (i.e., essentially **zero** PONV *without* the use of anti-emetics and minimal postoperative pain management).

In the final analysis, the MIA™ technique provides safety, simplicity, and satisfaction for all parties involved in the surgical experience: patients, their at-home caregivers, surgeons, nurses, and anesthesiologists.

REFERENCES

1. Goldwyn RM: Psychological aspects of plastic surgery: A surgeon's observations and reflections, in Sarwer DB, Pruzinsky T, Cash TF, et al. (eds.), *Psychological aspects of reconstructive and cosmetic plastic surgery*. Philadelphia, Lippincott, Williams & Wilkins, 2006; p13.
2. www.mhaus.org
3. McDevitt NB: Deep venous thrombosis prophylaxis. *Plast Reconstr Surg* 104:1923,1999.
4. Apfel CC, Korttila K, Abdalla M, et al.: A factorial trial of six interventions for the prevention of PONV. *N Engl J Med* 350:2441,2004.
5. Friedberg BL: Minimally invasive anesthesia for minimally invasive surgery. *Outpatient Surgery Magazine*. Herrin Publishing Partners LP, Paoli, PA. 2:57,2004.
6. Cullen SC, Larson CP: *Essentials of Anesthetic Practice*. Chicago, Year Book Medical Publishers, 1974; p82.
7. Laurito CE: Anesthesia provided at alternative sites, in Barasch PG, Cullen BF, Stoelting RK (eds.), *Clinical Anesthesia*, 4th ed., Philadelphia, Lippincott, Williams & Wilkins, 2001; p1343.
8. Friedberg BL: Propofol-ketamine technique, dissociative anesthesia for office surgery: A five-year review of 1,264 cases. *Aesth Plast Surg* 23:70,1999.
9. Lofsky AS: Deep venous thrombosis and pulmonary embolism in plastic surgery office procedures. *The Doctors' Company Newsletter*. Napa, CA, 2005. www.thedoctors.com/risk/specialty/anesthesiology/J4254.asp
10. Flaishon R, Windsor A, Sigl J, et al.: Recovery of consciousness after thiopental or propofol. *Anesthesiol* 86:613, 1997.
11. www.aspectms.com/resources/bibliographies
12. Friedberg BL: Propofol ketamine anesthesia for cosmetic surgery in the office suite, chapter in Osborne I (ed.), *Anesthesia for Outside the Operating Room. International Anesthesiology Clinics*. Baltimore, Lippincott, Williams & Wilkins, 41(2):39,2003.
13. Monk TG, Saini V, Weldon BC, et al.: Anesthetic management and one-year mortality after non-cardiac surgery. *Anesth Analg* 100:4,2005.
14. Kersssens C, Sebel P: Relationship between hypnotic depth and post-operative C-reactive protein levels. *Anesthesiol* 105:A578,2006.
15. Macario A, Weinger M, Truong P, et al.: Which clinical outcomes are both common and important to avoid? The perspective of a panel of expert anesthesiologists. *Anesth Analg* 88:1085,1999.
16. Macario A, Weinger M, Carney K, et al.: Which clinical anesthesia outcomes are important to avoid? The perspective of patients. *Anesth Analg* 89:652,1999.
17. White PF: Prevention of postoperative nausea and vomiting—A multimodal solution to a persistent problem. *N Engl J Med* 350:2511,2004.
18. Scuderi PE, James RL, Harris L, et al.: Multimodal antiemetic management prevents early postoperative vomiting after outpatient laparoscopy. *Anesth Analg* 91:1408, 2000.
19. Vinnik CA: An intravenous dissociation technique for outpatient plastic surgery: Tranquility in the office surgical facility. *Plast Reconstr Surg* 67:199,1981.
20. Thompson SWN, King AE, Woolf CJ: Activity-dependent changes in rat ventral horn neurons in vitro, summation of prolonged afferent evoked depolarizations produce a D-2-amino-5-phosphonovaleric acid sensitive windup. *Eur J Neurosci* 2:638,1990.
21. Corssen G, Domino EF: Dissociative anesthesia: further pharmacologic studies and first clinical experience with the phencylcidine derivative CI-581. *Anesth Analg* 45:29, 1968.
22. Friedberg BL: Hypnotic doses of propofol block ketamine induced hallucinations. *Plast Reconstr Surg* 91:196,1993.
23. Court MH, Duan SX, Hesse LM, et al.: Cytochrome P-450 2B6 is responsible for interindividual variability of propofol hydroxylation by human liver microsomes. *Anesthesiol* 94:110,2001.
24. Guit JBM, Koning HM, Coster ML, et al.: Ketamine as analgesic with propofol for total intravenous anesthetic (TIVA). *Anaesthesia* 46:24,1991.
25. Oxorn DC, Ferris LE, Harrington E: The effects of midazolam on propofol-induced anesthesia: propofol dose requirements, mood profiles and perioperative dreams. *Anesth Analg* 85:553,1997.
26. Friedberg BL, Sigl JC: Bispectral (BIS) index monitoring decreases propofol usage in propofol-ketamine office based anesthesia. *Anesth Analg* 88:S54,1999.