2 Propofol-Ketamine (PK) Anesthesia in Body Implant Surgery

Barry Friedberg

Poetry in Motion

All cosmetic surgery, including body implant surgery, can be performed under local anesthesia alone. When awake patients have pain during the case, they may move, but they also communicate verbally of their inadequate analgesia, i.e., "Ouch!" (Fig. 2.1).

More often than not, general inhalation anesthesia (GA) or propofol-opioid (i.e., alfentanil or remifentanil) total intravenous anesthesia (TIVA) is used for greater control of patient movement. Greater patient movement control obscures vital information about inadequate local analgesia to the (postoperative) detriment of the patient. No postoperative patient benefit could be determined when preemptive local analgesia was injected after induction of GA and prior to incision as seen in a meta-analysis of 80 studies [1].

Figure 2.2 clearly illustrates brain function is not necessary to produce movement. Movement in a sedated patient may also occur with or without brain involvement. Without the ability to discern brain-generated movement from that generated from the spinal cord, one remains stuck in the twentieth century mode of anesthesia wherein all patient movement had to be treated as if it might be a sign of patient awareness or recall.

Patient movement under sedation is almost always the patient's request for more local analgesia (Fig. 2.3). Brain-monitored propofol permits the differentiation between the need for more local analgesia (spinal cord-generated movement) and the need for more propofol (brain-generated movement). The ability to differentiate, and subsequently more appropriately treat, the two distinctly different types of patient movement results in less inappropriate types (and amounts) of adjuvant drugs being given to sedated patients.

Some surgeons direct their own diazepam (or midazolam)-ketamine anesthesia [2] with an impressive safety record. However, benzodiazepine sedation has no reliable, reproducible clinical signs for adequacy of brain protection from negative ketamine side effects. Currently available cerebral cortical monitors do not reliably measure benzodiazepine effect.

Direct measurement of anesthetic effect on the cerebral cortex has only been available since the 1996 FDA approval of the Bispectral Index® (BIS) (Aspect Medical Systems, Inc.) monitor. While

Fig. 2.1 Surgery without pain: an achievable PK goal

N.V. Chugay et al., *Body Sculpting with Silicone Implants*,
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Fig. 2.2 Headless chicken generating movement

Fig. 2.3 Adequate local analgesia is a critical element of PK anesthesia

cerebral cortical monitoring does not replace vital signs, like heart rate and blood pressure, vital signs only reflect brain stem activity (Fig. 2.4).

Brain stem activity (i.e., vital signs) simply cannot reliably guide the cerebral cortical effect, as was standard anesthetic practice prior to 1996. The net result of this void of cortical effect information was routine over-medication to prevent under-medication (anesthesia awareness). Complex activities like processing hearing, feeling, and recall occur in the cerebral cortex. Clearly, then direct cerebral cortical monitoring should be part of the twenty-first century anesthetic practice.

Over the past 21 years, propofol-ketamine (PK) monitored anesthesia care (MAC) has appeared as an alternative to both GA and propofol-opioid TIVA. From March 26, 1992 through December 25, 1997, PK anesthesia was more art than science. With the addition of BIS/EMG monitoring

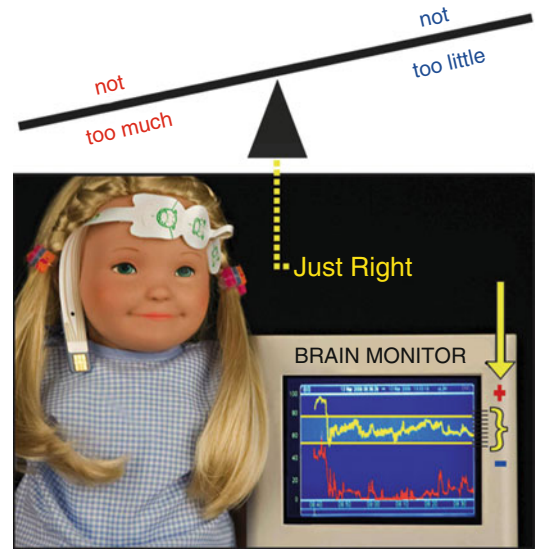

Fig. 2.4 BIS/EMG monitored PK MAC (aka "Goldilocks" anesthesia)

on December 26, 1997, numerical reproducibility was achieved [3].

Propofol-Ketamine TIVA [4] or "Ketofol"

There is no precise definition of what ketofol is. Generally ketofol refers to the 50:50 mixture of ketamine and propofol, 0.5 mg/kg of each

Fig. 2.5 Vodka martinis illustrating the difference between PK MAC and PK TIVA (aka ketofol)

(Fig. 2.5). However, a broader definition considered that ketofol is the combination of ketamine and propofol, regardless of the ratio to each other (the initial dose of each can be scaled up to 3 mg/kg). When they are used in infusion, the dose is 100 μg/kg/min.

The principle objection to ketofol is the inability to ascertain the amount of hypnosis (propofol effect) and the degree of NMDA block (ketamine effect) with induction and prior to the initial local anesthetic injection. The secondary objection is the potential for exceeding 200 mg ketamine during the case, potentially prolonging emergence. Conversely, PK MAC clearly defines hypnosis (BIS <75, baseline EMG) prior to dissociation (immobility with injection).

Anesthesia considerations for body implant cosmetic surgery revolve around the three parties' concerns – the patient, surgeon, and anesthesiologist. The key consideration is that the patient is the first priority! The patient wants (1) not to hear, feel, or remember their surgery, a cerebral cortical effect, and (2) to awaken promptly without pain, prolonged emergence, or postoperative nausea and vomiting (PONV) (Fig. 2.6), a function of anesthetic technique.

The surgeon wants a motionless patient during the surgery and the fewest possible postoperative concerns. Numb patients rarely move under sedation. Without a brain monitor to assure adequate

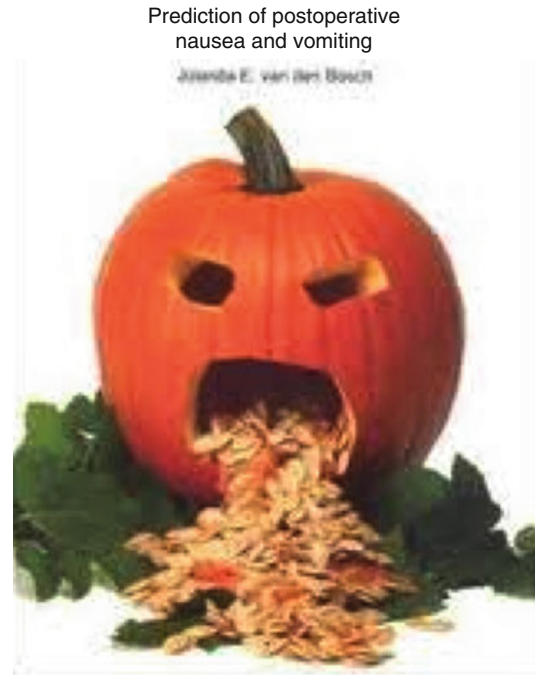

Fig. 2.6 Emesis is our nemesis

propofol sedation levels (i.e., BIS <75, EMG baseline vide infra), it is nearly impossible to encourage the surgeon to re-inject a vasoconstricted field.

The anesthesiologist wants reproducibility along with control of the patient's airway and movement during surgery. However, this is confounded by variations in patients' cerebral tolerance to medication effect in addition to their varying ability to metabolize and eliminate the anesthetic agents.

Friedberg's Triad answers the patients' desires, the surgeon's needs, and the anesthesiologist's quandary over what drug or intervention is most appropriate when facing patient movement under sedation.

1. Measure the brain
2. Preempt the pain
3. Emetic drugs abstain

"Measure the brain" means incrementally titrating propofol to BIS <75 with baseline electromyogram (EMG) (Fig. 2.7). Brain measurement provides numerically reproducible propofol levels to protect the brain from negative ketamine side effects.

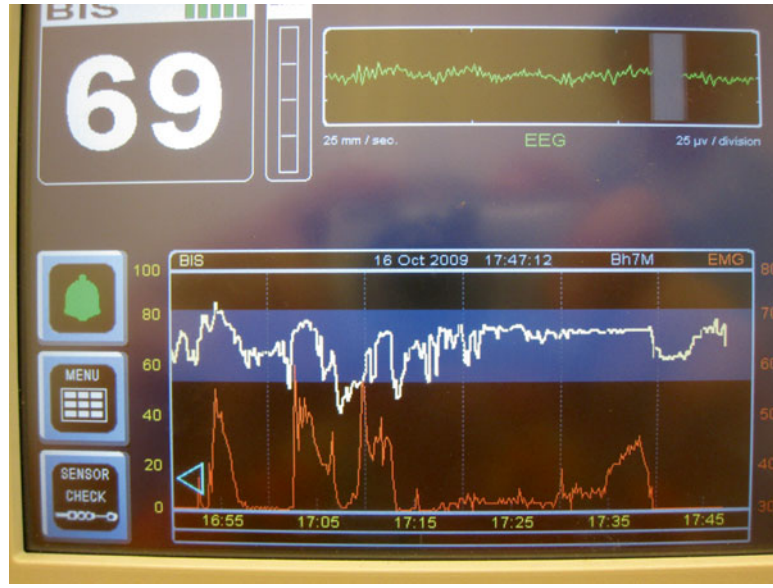

Fig. 2.7 EMG is the lower (*red*) trace

"Preempt the pain" means using a 50 mg dissociative dose of ketamine, independent of adult body weight, to completely saturate midbrain NMDA receptors 3 min prior to skin stimulation (i.e., injection of local analgesia).

"Emetic drugs abstain" simply means scrupulous avoidance of opioids (narcotics) like morphine, meperidine, fentanyl, alfentanil, or remifentanil as well as inhalational agents like forane, sevoflurane, or desflurane (Fig. 2.8). Assurance of adequate local analgesia obviates the need for these troublesome agents. Abstaining from emetic agents also eliminates the need for antiemetic drugs.

Propofol's advantages over inhalational agents include the following:

1. Not a malignant hyperthermia (MH) trigger.
2. Not needing to stock dantrolene, an MH antidote.
3. Antiemetic qualities.
4. Antioxidant qualities: halogenated inhalational agents like forane, desflurane, or sevoflurane are oxidizing agents.
5. Rapid, pleasant emergence likely due to rapid metabolism.
6. Preserved REM sleep patterns.

Unlike benzodiazepines, propofol clinical signs (i.e., loss of lid reflex and loss of verbal response) are reliable and clinically reproducible, and cerebral cortical effect can be measured and, therefore, is numerically reproducible.

Bispectral Index (BIS) Monitor

With the 1983 introduction of pulse oximetry (SpO_2) monitoring, anesthetic mortality declined from 1 in 10,000 in the 1950s to about 1 in 250,000 patients. Additional vital signs of blood pressure, EKG and $EtCO_2$, while important, still only provide a reflection of brain stem activity. However, the part of the brain that processes hearing, feeling, and memory is the cerebral cortex.

For anesthesiologists practicing prior to 1996, there was no direct measure of patient cerebral anesthetic response. To compensate for this lack of information about cortical effect, the anesthesiologist was obliged to over-medicate for fear of not giving enough anesthetic. In 1996, the Food and Drug Administration (FDA) approved Aspect Medical Systems' Bispectral Index (BIS) monitor to directly measure the patients' cerebral drug response.

While the BIS technology has been validated in over 3,500 published scientific studies and found in over 75 % of US hospitals, BIS utilization remains at only about 25 %. There are sev-

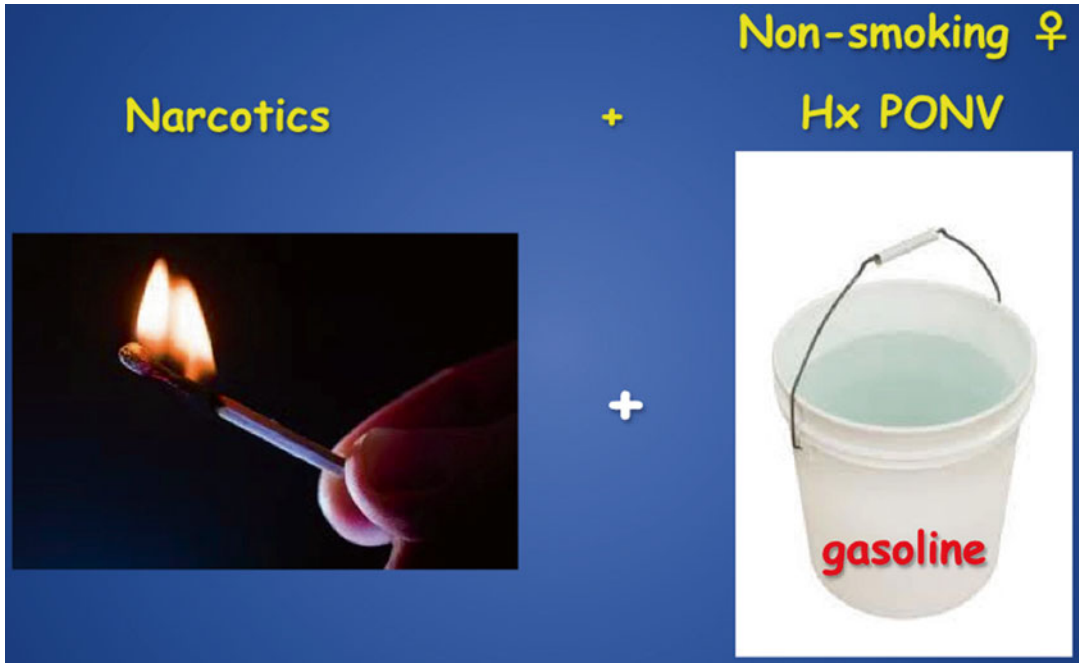

Fig. 2.8 "As long as emetogenic agents are part of the anesthetic regimen, the use of anti-emetics is of limited utility" – Christian Apfel MD, PhD

eral reasons for the underutilization of BIS monitoring. First, the BIS value is a unit-less, derived value that is 15–30 s behind real time (vital signs, like heart rate and blood pressure, are measured in real time). Titrating anesthetics with only the BIS value is akin to trying to drive an automobile with only the rearview mirror information. The factory default setting displays only the BIS value and tracing. A tool that does not provide useful, real-time information is not often used. BIS values delayed from real time are not especially helpful to titrate anesthetics.

The optimal use of BIS is by trending the frontalis muscle electromyogram (EMG) as the secondary trace [5] and responding to EMG spikes as if they were heart rate or blood pressure changes. EMG is to the frontalis muscle what the EKG is to the myocardial muscle, i.e., a real-time, physiologic parameter (Fig. 2.9). While some allege the use of ketamine invalidates the ability to titrate propofol with BIS, there is evidence to the contrary [6], along with 15 years of reproducible clinical practice.

Premedication

Between March 1992 and June 1997, the addition of midazolam premedication was undertaken in the hope of reducing the cost of the average 3–20 ml bottles of propofol (Diprivan® Zeneca). Three groups of patients were informally studied – 0, 2, and 4 mg midazolam premedication, with the 4 mg group selected for cases of 4+ h. Review of the comparative propofol rates revealed no cost-effective reduction with either 2 or 4 mg midazolam premedication [7].

Midazolam premedication was subsequently abandoned in favor of no pharmacologic agents from June 1997 through December 1998. In September 1997, Oxorn published a double-blind Level I RCT showing no propofol-sparing effect from 2 mg midazolam versus none, but a 3× increase in postoperative pain medication was required in the midazolam patient group [8].

In December 1998, oral clonidine premedication was added to the PK regimen. The therapeutic level is 2.5–5.0 mcg/kg [9]. Patients weighing

Fig. 2.9 Botox does not eliminate EMG spikes

between 95 and 175 lb require 0.2 mg clonidine to achieve the therapeutic level. For patients with systolic blood pressure <100 mmHg, clonidine should be avoided. An explanation for the salutary postoperative effect on patients' pain has been suggested recently [10].

Two clonidine caveats:

1. Never give clonidine for patients to take at home prior to surgery. In the event of postural hypotension, one is unlikely to have someone to either start an intravenous or place the patient in Trendelenburg. Also, patients are not likely to have their scheduled surgery.
2. Clonidine is available in 0.1, 0.2, and 0.3 mg doses. Only stock 0.1 mg formulation of clonidine to avoid medication dose errors.

Lastly, clonidine premedication is not critical to the success of PK anesthesia. If one has recurrent hypotension with the clonidine, eliminating it from the regimen will not negatively affect the reproducible success of PK anesthesia. While propofol is generic and dramatically less expensive than in the 1990s, given the recent problems sourcing propofol, it may still be worth including propofol-sparing clonidine premedication.

Induction

All patients receive 0.2 mg glycopyrrolate (Robinul®) in the same syringe as 1.5 mL 1 % lidocaine prior to starting the propofol infusion. Glycopyrrolate counteracts the tendency of ketamine to produce excessive salivation. Saliva touching the vocal cords frequently results in laryngospasm and oxygen desaturation. Lidocaine often eliminates the discomfort patients report from the propofol infusion.

Hypnosis first, then dissociation follows Vinnik's concept using propofol instead of diazepam [2] Most anesthesia providers emulate the speedy hare in their approach (i.e., 1,000–2,000 mcg/kg bolus) to starting a case. However, the winning example of the slow and steady tortoise (i.e., sequential 50 mcg/kg doses) (Fig. 2.10) is demonstrated in this YouTube clip at http://www.youtube.com/watch?v=GlQ3Do3b3_I.

Three distinct benefits derive from the incremental type of induction:

1. Patient's airway and drive to breathe are infrequently disturbed, simplifying airway management.

Fig. 2.10 Slow and steady wins the race: airway, breathing, and blood pressure preserved

Table 2.1 PK anesthesia progressive airway algorithm (assumes incremental propofol induction): errors to avoid [13]

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

2. Blood pressure is maintained, unlike that observed with propofol bolus induction that often necessitates ephedrine treatment.
3. Stable brain level of propofol provides protection from the historically reported ketamine side effects of hallucinations [11], dysphorias, and flashbacks.

The induction time of 2.5 min shown in the YouTube clip approximates the more traditional preoxygenation, bolus propofol induction, muscle relaxation, laryngoscopy, endotracheal tube placement, and tube position confirmation times for GA.

Airway Management

Three muscles, the temporalis, orbicularis oris, and genioglossus, are responsible for maintaining the tongue from retrograde airway occlusion. Snoring is one sign of partial airway obstruction while pre-tracheal, sternal notch retraction is more often associated with total airway obstruction.

BIS 60–75 is not a measure of airway patency or brain oxygenation. Only the SpO_2 will measure oxygenation. When incremental propofol induction is administered, the $EtCO_2$ most commonly remains between 38 and 42. If one is not able to observe ventilation, $EtCO_2$ monitoring can display a waveform of the patient's breathing. This author has not used $EtCO_2$ monitoring for BIS/EMG monitored PK MAC and does not mandate its use for others.

Bolus propofol induction produces rapid decreases in all three airway muscles, frequently leading to the need to support the airway with chin lift and positive pressure ventilation. Incremental propofol induction frequently, but not always, tends to maintain tongue muscle support. Propofol, incrementally titrated to BIS 60–75, may still produce airway obstruction. However, obstruction occurs far more infrequently than with bolus induction. Observationally, the mouth, more often than not, tends to remain closed with incremental propofol, while the so-called "O" sign is more characteristic of bolus propofol.

Patient safety with sedation absolutely demands scrupulous attention to airway patency. Management of the airway follows a progressive algorithm (Table 2.1) that depends on what is required to keep the tongue from occluding the airway.

1. Head position: extend and laterally rotate ~30 %, rhytidectomy or facelift position (Fig. 2.11).
2. Shoulder pillow (or 1,000 mL IV bag) (Fig. 2.12): ~30 % increased extension force on genioglossus muscle.
3. Nasal airway #28 FR (Fig. 2.13) ~30 %.
4. LMA #4 (Fig. 2.14): ~5–10 %. No intubation required 21 years, >5,000 patients.
5. Oxygen, supplemental: ~10–20 %.

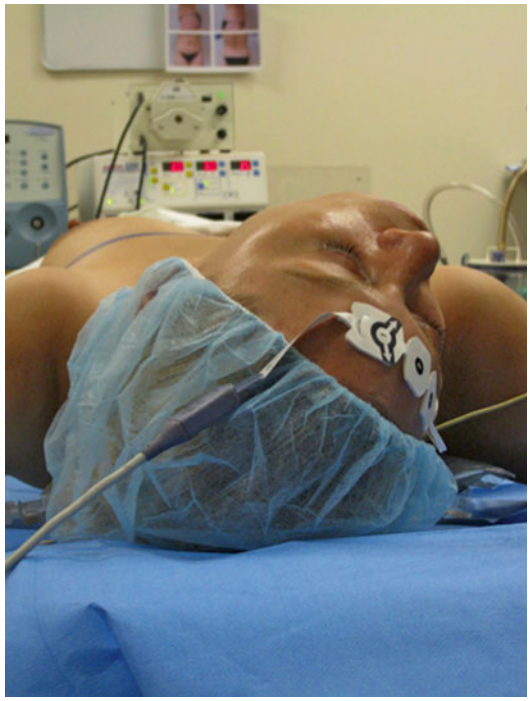

Fig. 2.11 Head position

Dissociation (NMDA Receptor Block)

Although Vinnik's initial ketamine dose is 75 mg [2], this has been reduced to 50 mg independent of adult body weight [5]. The 50 mg ketamine dose will provide complete *N*-methyl-D-aspartate (NMDA) block in ~98 % of adults (Fig. 2.15). A 25 mg dose will produce NMDA block in ~80 % of patients. However, there are no negative consequences to using the 50 mg dose once the brain has been protected with a stable propofol level. Stable propofol brain level will not be produced with a bolus propofol induction. The practical upper aggregate ketamine dose has been found to be 200 mg for day surgery cases. It is useful, therefore, to inject as many of the surgical fields with the initial dissociating 50 mg ketamine dose. Ketamine dissociation is only used to render the patient unresponsive to the local anesthetic injection of a virgin field.

When patient movement (without EMG spike) occurs, necessitating additional local anesthetic

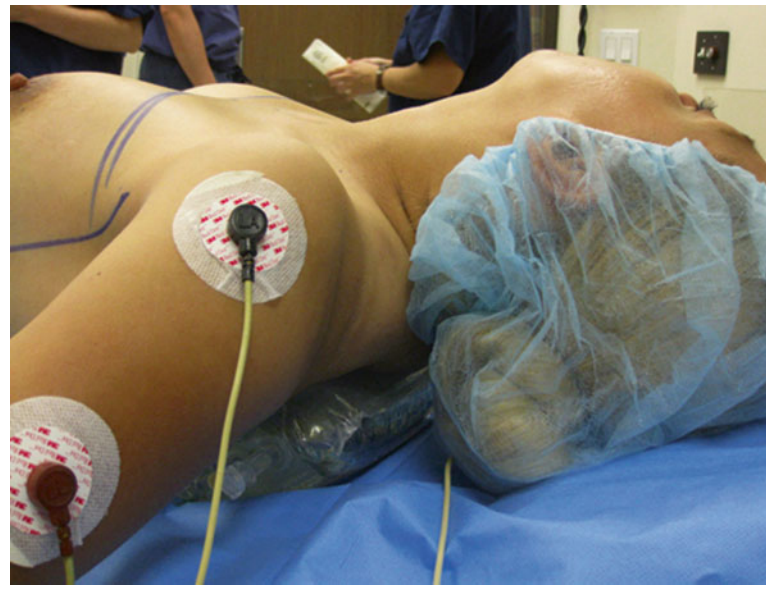

Fig. 2.12 Shoulder pillow (or 1,000 mL IV bag)

Fig. 2.13 Nasal airway #28 FR

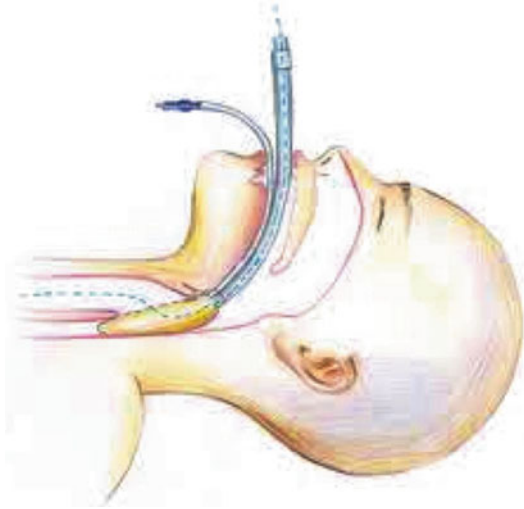

Fig. 2.14 LMA #4

injection, additional ketamine dissociation is rarely required. Paradoxically, there is usually sufficient residual analgesia to block the noxious signal from follow-up local analgesia injection but not enough to continue surgery without that supplementation. Also, once the surgical field has been injected, continuing to administer more ketamine will more often produce an aggregate ketamine dose in excess of 200 mg and a somnolent emerging patient.

Additional ketamine dissociation may be required once the surgical field has been injected a total of three times (i.e., the initial and two subsequent injections to eliminate patient movement). However, cognizance of the aggregate dose must be taken to preserve the rapid emergence and ability to efficiently discharge the patient from the surgical facility. Brain measurement of propofol levels assures continued brain protection for subsequent ketamine doses, should they be necessary.

Ketamine increases the laryngeal or so-called life-preserving reflexes (Fig. 2.16) mentioned in CA AB595 requiring office certification whenever anxiolytics or analgesics are administered in doses likely to depress those reflexes. Recognition of the prodrome of laryngospasm, along with prompt therapy, is essential to the best conduct of the case.

Ketamine-Associated Laryngospasm

The "crowing" sound of laryngospasm is created by the partial vocal cord closure. However, ketamine-associated laryngospasm produces complete vocal cord closure. Hence, no readily recognizable sound occurs with ketamine-associated laryngospasm. The prodrome is a cough or sneeze. Therapy consists of the prompt intravenous bolus injection of lidocaine 1 mg/lb. Like benzodiazepines, propofol hypnosis elevates the seizure threshold of lidocaine. Seizures have not been seen over the 21 years of administering lidocaine 1 mg/lb for ketamine-associated laryngospasm.

Maintenance

The art of the technique lies in defining the basal infusion rate necessary (i.e., "surfing" the level of consciousness) to maintain BIS 60–75 (with

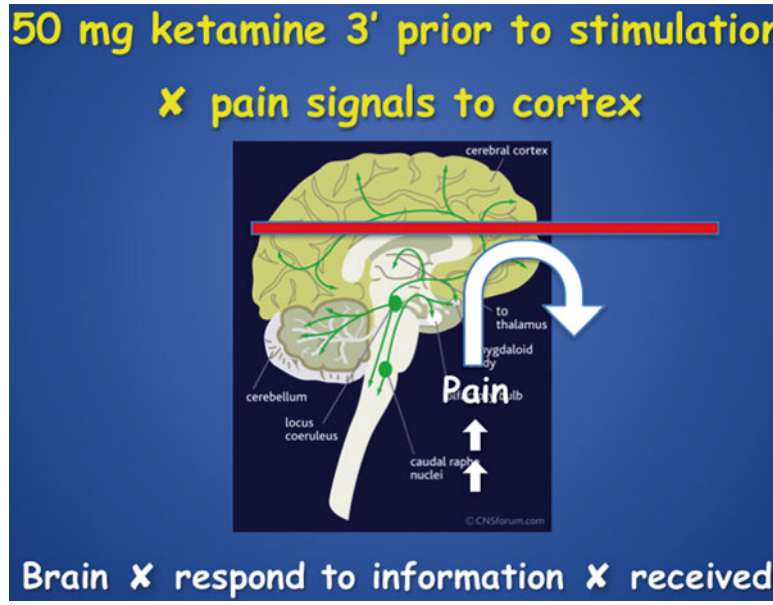

Fig. 2.15 Windup phenomenon eliminated with NMDA block prior to injection

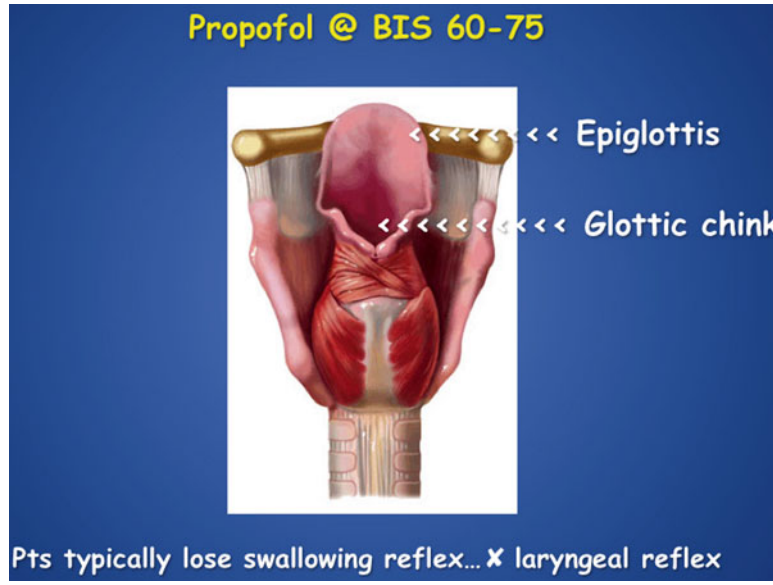

Fig. 2.16 Laryngeal reflexes protect the lungs from aspiration and are life preserving. Ketamine increases laryngeal reflexes, while opioids (narcotics) decrease them

baseline EMG) as well as defining the optimal bolus rate required to return the patient to BIS 60–75 when EMG spike activity is observed. Recall is not dependent on the absolute BIS value observed, but rather on the area under the BIS curve >3–5 min.

The empirically derived initial bolus of 50 mcg/kg^{-1} and basal rate of 25 mcg/kg^{1}/min^{-1} are not "set in stone" but have been found to produce the least trespass to patient airway, breathing, and blood pressure. For instance, when preparing to anesthetize a very elderly or frail patient, one might consider decreasing the bolus and basal rate in half and taking 3–4 min, instead of the customary 2 min, to induce propofol to BIS <75, EMG baseline.

Adjusting Expectations

One cannot provide BIS/EMG PK MAC without prior discussion to secure the surgeon's understanding for the need to supplement local analgesia during the case. Some surgeons appear to perceive the need to supplement local during the case as an affront to their ability to inject local analgesia. Other surgeons mistakenly believe the presence of vasoconstriction guarantees adequate local analgesia. While these are untrue presumptions, care needs to be taken to not insult the surgeon's ego or intellect when the need for additional local analgesia becomes manifest.

BIS/EMG monitoring can explain the differentiation of spinal – versus brain-driven patient movement to the surgeon (Fig. 2.17). Vasoconstriction observed in the surgical field does not guarantee adequate analgesia, especially when there is no EMG activity and propofol BIS levels are 60–75!

"Perfect" local analgesia with the initial injection is not required, but subsequent persistence with local analgesia when required during the case is an absolute requirement for PK success. One then sets the stage for poetry in motion. The anesthesiologist measures the level of hypnosis while the surgeon controls surgical analgesia. The patient benefits enormously by not being hurt during the procedure.

Fig. 2.17 The tango: poetry in motion

Postoperative Pain Management

Patients do not typically exhibit the traumatized "look" of surgery. The dissociative effect precludes intraoperative pain. The major pain signal of violating the skin is never sent to the brain (i.e., dissociation or NMDA block). Hence, patients typically have little or no postoperative discomfort after body implant surgery. The brain cannot wind up from information it never receives. Over the past 15 years of BIS/EMG monitored PK MAC, a minority of >3,000 patients have required postoperative opioids prior to discharge from the office facility.

Much of the postoperative discomfort is a function of muscle spasm secondary to increasing muscle fiber length by the implant placement. This issue can readily be dealt with by prescribing 5–10 mg oral diazepam every 4–6 h. Patients recover quickly and are able to swallow oral medications after BIS/EMG monitored PK MAC. Midazolam (Versed®) 1–2 mg intravenously has infrequently been given for the rare patient. Postoperative opioids like hydrocodone carry the (avoidable) risks of PONV and constipation. Sometimes, patients can avoid postoperative opioids by taking not more than 1,000 mg acetaminophen (Tylenol®) every 8 h to supplement the diazepam relaxation.

Patients will benefit with prolonged postoperative analgesia by placing bupivacaine (Marcaine®) in the implant pockets prior to closing or injecting retrograde in the drains. Since bupivacaine rapidly binds to tissues, it is not necessary to inject the agent with a needle. Depending on the pocket size, 10–25 mL of bupivacaine per pocket is deposited. To avoid cardiotoxicity, never exceed a total of 125 mg or 50 mL 0.25 % bupivacaine. If greater than 50 mL is required, simply dilute 0.25–0.125 % with normal saline (NSS) to have 100 mL to distribute among multiple pockets.

On occasion, there are patients who have been predominantly immobile during surgery but complain bitterly of pain after surgery. These patients are given two "Big Blue" pain pills (i.e., Tylenol® PM) for relief of nonphysical pain. Unless specifically asked for the name of this drug, staff does not customarily mention the proprietary name as the therapeutic efficacy is greatly diminished by doing so.

Postoperative Nausea and Vomiting (PONV)

Avoidance of both opioids and inhalational vapors (i.e., emetogenic agents) has allowed the lowest published rate of PONV (0.6 %) in a high-risk patient population without the need of antiemetic use [7].

Discussion

Inasmuch as GA and TIVA remain the most commonly performed anesthetics for body implant surgery, the question remains, "Why are more anesthesiologists not offering BIS/EMG PK MAC?"

Because the academic centers rarely expose their trainees to BIS/EMG PK MAC, many anesthesiologists are reluctant to try a technique they have not become familiar with in their residency training. Without being exposed to the real-time use of BIS with EMG as the secondary trace, many anesthesiologists have discarded the idea of using this device. Also, BIS monitors are not always physically available in cosmetic surgery practices. For too many anesthesiologists, the idea of buying their own BIS monitor remains a totally foreign concept they need to shed.

Academic anesthesia centers are still not routinely having residents titrate anesthetics with BIS/EMG. Without being able to differentiate spinal-cord-generated movement (i.e., movement without EMG spike) from cortically generated movement (i.e., movement with EMG spike), it is difficult to convince the surgeon for the need of additional local analgesia as well as spinal cord movement not being a sign of awareness and recall. BIS/EMG PK MAC without dealing with the need for adequate local analgesia will not provide dramatically better outcomes.

Lectures in anesthesia training programs continue to emphasize hallucinations, hypertension, and tachycardia as ketamine's side effects when the drug is administered as a solo agent. Rarely is cognizance taken of the satisfactory elimination of those troublesome side effects when pretreatment with either benzodiazepines [2] or propofol [11] is provided.

BIS monitoring has been FDA approved since 1996. Advocacy of universal BIS monitoring could reduce anesthetic use by 30 % [3], potentially jeopardizing the American Society of Anesthesiologists (ASA) of millions of various forms of drug company (Big Pharma) support dollars. From 1983 to 1990, the ASA also resisted advocacy of pulse oximetry as a standard of care until Medicare mandated the additional SpO_2 fee being bundled as a base charge.

Surgeons who appreciate the value of good local analgesia tend to be more receptive to PK MAC for their patients. They also understand the difference between intraoperating room process and postsurgical *outcomes*. Why should more anesthesiologists seek to offer BIS/EMG PK MAC for elective cosmetic surgery like body implants? Patient safety is improved with a minimally invasive anesthetic regimen. Preservation of native airway, reduced need for supplemental oxygen, and blood pressure stability self-evidently minimize the potential for needing to perform CPR or call 911.

Ketamine does not depress respiration. Respiratory embarrassment is far less likely when only a single respiratory depressant (i.e., propofol) is titrated against both brain stem and cortical response. Patients who experience desaturation with PK MAC frequently recover normal SpO_2 without the need for bag-mask ventilation. Often a simple, transient chin lift will suffice.

Patient satisfaction is dramatically improved for several reasons. Patients emerge from propofol feeling refreshed as opposed to "hung over." Propofol permits REM sleep patterns as well as providing an anti-inflammatory effect. The absence of emetic drugs also provides a nearly zero PONV rate [7], especially important

in patients with a previous history of PONV. Ketamine's anti-inflammatory effect supplements that of propofol [12].

Cost-effectiveness is improved when drugs are administered in the optimal fashion. Propofol usage is reduced by 30 % with BIS/EMG monitoring and clonidine premedication [3]. Ketamine usage is most often only one or two 50 mg doses. However, most importantly is that local analgesia instead of excessive propofol, ketamine, or even opioids is employed to accurately deal with the patient's need for intraoperative pain relief. Providing a lack of information to the patient's brain (i.e., dissociation or complete NMDA block) for both the initial local analgesia injection and during the case for supplemental analgesia has led to >3,000 BIS/EMG PK MAC patients not needing opioid relief upon emergence since December 26, 1987.

Outliers are eliminated using BIS/EMG monitoring with an incremental approach to anesthetic induction. Clonidine premedicated patients, on average, require 25–50 mcg/kg/min^{-1} to maintain BIS 60–75. Patients have been observed to require as low as 2.5 mcg/kg/min^{-1} and as high as 185 mcg/kg/ min^{-1} to maintain BIS 60–75 with the same excellent recovery times. Responding to EMG spikes as if they were heart rate or blood pressure changes is the key to avoiding over – and under-medication [5].

Conclusions

Patients are universally happy with BIS/EMG PK MAC anesthesia. Happy patients tend not to file medical liability lawsuits. According to a leading medical liability insurer, anesthesiologists get sued every 8 years on average. For the past 21 years of PK MAC, no patients have filed such an action. Happy patients are also more often likely to return for additional procedures and are more likely to have friends who observe their positive anesthesia outcome and seek out that surgeon for their surgeries. PK MAC anesthesia patients are a positive advertisement and an asset to the cosmetic surgeon.

References

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