Translational Perioperative and Pain Medicine

ISSN: 2330-4871

Editorial | Open Access Volume 5 | Issue 4

How Does Routine Anesthesia Care Impact Today's Opioid Crisis: The Rationale for Opioid Free Anesthesia (OFA)

Barry L Friedberg, MD*

President, Goldilocks Anesthesia Foundation, A Nonprofit Corporation, USA

As a 40-year practicing, board-certified anesthesiologist, I am concerned much of the current opioid crisis revolves around how anesthesia is routinely administered.

"More than 75% of heroin overdoses begin with prescription drugs", said Dr. Toby Cosgrove, Cleveland Clinic CEO. Many patients get prescription opioids for relief of postoperative pain. Opioid Free Anesthesia (OFA) not only uses no opioids during surgery but also radically reduces the need for them after surgery.

Many people originally become addicted to opioids after taking them for postoperative pain relief. By reducing or even eliminating the need for opioid medications after surgery, an entire population of addicts may be prevented.

Routine opioid use began with premedication to minimize secretions and induction excitement [1]. If opioids are so effective mitigating postoperative pain, why does postoperative pain management remain an issue? [2,3] Opioids are for anesthesiologists that don't know how to preemptively saturate infra-tentorial NMDA receptors to block pain from the first stroke of the scalpel.

As long as one violates the integument, *all* surgery (i.e. thoracic, neuro, ortho, ENT facial or other cosmetic) is the same information for the sedated/anesthetized brain. The worst possible message for the surgery patient's brain is the knowledge of the penetration (invasion) of the protected world of self. Although other pain receptors exist within the body, no other cortical signal is more critical to postoperative outcomes than the 'invasion' of self.

A surgeon's scalpel is no different to the medicated brain than a mugger's knife. The use of pre-incision subcutaneous local anesthesia as well as the pre-closure splash of bupivacaine prolongs the 'deception' or the brain's ability to perceive the violation of the skin.

Prior to the advent of direct cortical monitors, anesthesiologists relied on the absence of heart rate (HR) and blood pressure (BP) changes at incision to determine adequate anesthesia was achieved. The ASA Awareness study revealed half of the patients who experienced awareness with recall under anesthesia had *no* HR or BP changes with which to alert the anesthesiologist [4].

With the perspective of direct brain monitoring information, it is now understood that HR & BP changes most accurately reflect brain stem signs and are notoriously unreliable guides to cortical responses. However, pain and the awareness of consciousness are processed at higher, cortical levels [5].

In 1996, the Food & Drug Administration (FDA) approved the bispectral (BIS) index to *directly* measure the cortical anesthetic response to hypnotic agents like propofol. The BIS monitor has been validated in more than 3,500 studies and is found in most hospitals and ambulatory centers where anesthesia for major surgery is given. Other brain monitors like Entropy are also available but there is no literature claiming superiority to the BIS.

The BIS brain monitor generates a number between 0-100 from a two lead EEG forehead sensor. The lower the number, the more hypnotized is the patient (Table 1) [6].

BIS below 40 is considered over medicated. Use of the sensor and monitor is cost effective [7]. Neither cost nor difficulty reading the monitor should impede its regular use for major surgery under anesthesia, the goal of the nonprofit Goldilocks Anesthesia Foundation (www.goldilocksfoundation.org).

Free-standing BIS A-2000 and VISTA units have software that enables the selection of the electromyogram (EMG) as a secondary trend. EMG is to the facial *frontalis* muscle what EKG is to the cardiac muscle; i.e. a di-

Table 1: BIS and levels of sedation/anesthesia [9].

Awake	98-100
Minimal sedation	78-85
Moderate sedation	70-78
Deep sedation	60-70
General anesthesia	40-60 with systemic analgesia
Over medicated	< 40

rectly measured *real time* signal. EMG spikes persist in the presence of neuromuscular blocking agents as well as Botox. EMG spikes signal incipient arousal [8,9]. BIS without concomitant EMG trending is like trying to drive a car with only rearview mirror information.

Postulates

1st Postulate: Without nocioceptive arousal the cortex cannot process pain.

2nd Postulate: Opioids fail to block cortical input.

3rd Postulate: Measurement is the basis for scientific practice.

4th Postulate: EMG spikes *absence* with skin violation is prima facie evidence of infra-tentorial receptor saturation.

5th Postulate: Infra-tentorial NMDA receptor saturation with 50 mg IV ketamine 2-5minutes pre-incision blocks cortical input.

6th Postulate: NMDA receptor saturation creates opioid free, preemptive analgesia.

On March 26, 1992, 26 years of opioid free anesthesia for office-based elective cosmetic surgery anesthesia was initiated using propofol ketamine (PK) sedation; i.e. hypnosis first, then dissociation [10]. During the first 15 years of a 40-year private practice, anesthesia career, never once did patients' need for postoperative opioid pain therapy occur to this author.

By the spring of 1993, 50 *consecutive* PK Apfel-defined postoperative nausea & vomiting (PONV) high risk patients (i.e. non-smoking, females, history of PONV or motion sickness, having emetogenic surgery) [11] emerge *entirely devoid* of opioid pain relief need. Also observed was the virtual absence of PONV *without* anti-emetic drugs. PONV is patients' #1 outcome to avoid after surgery [12]. Only after adding the real-time BIS brain monitor was the PK paradigm *numerically reproducible*.

The lack of need for postoperative opioid pain relief led this author to conclude patients had pain upon awakening because they were having pain *during* surgery! This was an astonishing insight considering the cardinal function of anesthesia is the *prevention* of pain during surgery.

Prior to ketamine, patients were premedicated with 0.2 mg glycopyrrolate then had propofol *incrementally* titrated to 60 < BIS < 75 *with* baseline EMG [13]. Ketamine 50 mg *independent* of adult body weight was administered 2-5 minutes pre-incision. Pre-incision lidocaine was infiltrated. Prior to closure, bupivacaine (not to exceed a total of 125 mg or 50 ml of 0.25%) was splashed into the wound.

Table 2: Friedberg's Triad.

Measure the brain
Preempt the pain
Emetic drugs abstain

Dexamethasone was not given for the first 1,264 patients when the lowest published PONV rate was published in an Apfel-defined high risk patient population *without* anti-emetic administration [14]. Beginning in May 2009, dexamethasone 10 mg IV was given at induction with no change in PONV incidence.

Validation of Postulates

From 1998 through 2018, over 4,000 painful outpatient patient surgeries (i.e. subpectoral breast augmentation and classical abdominoplasty) were performed under BIS/EMG monitored, propofol hypnosis using preemptive ketamine (i.e. midbrain NMDA receptor saturation), followed by injection of subcutaneous local anesthesia without postoperative opioid rescue or a single hospital admission for pain. Patients rarely used postoperative opioids. No reports of opioid addiction were observed [15].

Preemptive ketamine prevents pain *during* surgery with dramatic reductions and, often elimination, of the need for opioids after surgery. No hospitalizations for postoperative pain management or PONV occurred during 20 years of opioid free anesthesia (OFA) for more than 4,000 patients.

Preemptive ketamine tricks the brain by denying it the information the body has been 'invaded' by the surgeon. During the magic interval between surgical intrusion and the 'discovery' of intrusion, healing takes place. That interval permits a degree of healing with a dramatic reduction, not infrequently the elimination, of opioid requirements for pain.

Opioid free anesthesia (OFA) patients look like surgery never even happened! OFA embodies Friedberg's Triad (Table 2). Real time brain monitoring should be the standard of care for major surgery under anesthesia.

Disclaimer

Neither Dr. Friedberg nor the nonprofit Goldilocks Anesthesia Foundation receive BIS maker financial support.

References

1. Buxton D: The use of morphine, scopolamine, atropine and similar drugs before inhalation anesthesia. *Proc Roy Soc Med* 1911;4:53.

2. Hopf HW, Weitz S: Postoperative pain management. *Arch Surg.* 1994;129(2):128-132.

3. Chou R, Gordon DB, de Leon-Cassola OA, et al: Management of Postoperative Pain: A Clinical Practice Guideline From the American Pain Society, the American Society of Regional Anesthesia and Pain Medicine, and

the American Society of Anesthesiologists' Committee on Regional Anesthesia, Executive Committee, and Administrative Council. *J Pain* 2016;17:131-57.

4. Domino KB, Posner KL, Caplan RA, Cheney FW: Awareness under anesthesia: a closed claims analysis. *Anesthesiol* 1999;90:1053-61.
5. Garcia-Larrea L, Bastuji H: Pain and consciousness. *Prog Neuropsychopharmacolog Biol Psychiatry* 2018;87:193-99.
6. Friedberg BL: Propofol ketamine with bispectral (BIS) index monitoring ch in Friedberg BL, ed.: *Anesthesia in Cosmetic Surgery*. Cambridge University Press, New York, 1-13, 2007.
7. Klopman MA, Sebel PS: Cost-effectiveness of bispectral index monitoring. *Curr Op Anesthesiol* 2011;24:177-81.
8. Practice Advisory for Intraoperative Awareness and Brain Function Monitoring *Anesthesiol* 2006; 104:847–64.
9. Mathews DM, Clark L, Johansen J, et. al: Increases in Electroencephalogram and Electromyogram Variability Are Associated with an Increased Incidence of Intraoperative Somatic Response. *AnesthAnalg* 2012;114:759-770.
10. Friedberg BL: Hypnotic doses of propofol block ketamine induced hallucinations. *Plast Reconstr Surg* 1993;91:196.
11. Apfel CC, Laara E, Koivuranta M, *et al*: *A Simplified Risk Score for Predicting Postoperative Nausea and Vomiting*: Conclusions from Cross-validations between Two Centers. *Anesthesiol* 1999;91:693-700.
12. Macario A, Weinger M, Carney S, Kim A: Which clinical anesthesia outcomes are important to avoid? The perspective of patients. *AnesAnalg* 1999;89:652-8.
13. Propofol Induction Using BIS Monitor without Instrumenting Airway.
14. Friedberg BL: Propofol-ketamine technique, dissociative anesthesia for office surgery: a five-year review of 1,264 cases. *AesthPlast Surg* 1999;23:70-74.
15. Friedberg BL: Can Friedberg's Triad solve persistent anesthesia problems? Over-Medication, Pain Management, Postoperative Nausea and Vomiting. *PlastReconstr Surg Global Open* 2017;5:e1727-1734.

Corresponding Author: *Barry Lynn Friedberg, M.D., President, Goldilocks Anesthesia Foundation, A Non-profit Corporation, P.O. Box 10336, Newport Beach, CA 92658, USA, Tel: 011-1-949-233-8845, Email: drbarry@goldilocksfoundation.org*

Editor: *Renyu Liu, MD, PhD, Associate Professor, Department of Anesthesiology and Critical Care, Perelman School of Medicine at the University of Pennsylvania, 336 John Morgan building, 3620 Hamilton Walk, Philadelphia, PA 19104, Phone: 2157461485, Email: liur@uphs.upenn.edu*

Additional publication details

Journal short name: Transl Perioper & Pain Med

Received Date: August 27, 2018

Accepted Date: December 19, 2018

Published Date: December 21, 2018

Citation: Friedberg BL. How Does Routine Anesthesia Care Impact Today's Opioid Crisis: The Rationale for Opioid Free Anesthesia (OFA). Transl Perioper & Pain Med 2018; 5 (4):98-100