Episode 154: EEG monitoring with Dr. Emery Brown

On this episode: Dr. Jed Wolpaw and Dr Emory Brown

In this 154th episode I welcome Dr. Emery Brown to the show to discuss how we monitor the depth of anesthesia. We discuss the drawbacks to BIS and why Dr. Brown thinks the EEG itself is the best way to go.

Table of Contents

Hyperlinks to section of notes.

CLASSIC MONITORING OF DEPTH OF ANESTHESIA?	2
MONITORING COMPONENTS OF ANESTHESIA	2
HOW DOES BIS WORK?	2
HOW TO USE EEG RATHER THAN BIS #?	3
HOW TO INTERPRET EEG WITH DRUG COMBINATIONS?	3
REFERENCES	4

Classic monitoring of depth of anesthesia?

- Typically ballpark with MAC, then use HR, BP
- Target controlled infusions based on mathematical models to reach desired state
- Definition of MAC may be incorrect? More like giving reasonable starting dose of drug, and change based on demographics
 - Dr Brown's definition: drug-induced, reversible state that consists of anti-nociception, unconsciousness, amnesia, immobility/akinesia/muscle relaxation, w/ maintenance of physiologic stability – break it down to these parts and think about it individually

Monitoring components of anesthesia

- Muscle relaxation Electromyogram or Train of Four
- Consciousness like EEG, which has been shown to change systematically w/ dose/class/age
 Can develop level of unconsciousness schema using EEG in real time. More reliable
- **Nociception** more challenging. Typically BP and HR, and more recently monitors using HR and electrodermal monitors skin impedance. Pain is perception of nociception. Less progress than level of consciousness.
 - Scenario 1: Surgical incision while patient on propofol drip and sevoflurane. HR goes up without change in EEG = nociceptive response, but not arousal.
 - Scenario 2: Same anesthetic as above, but incision so potent, patient is gaining consciousness.
 - Former scenario would give fentanyl, latter would increase propofol or sevo. Importance of identifying cause of physiologic response.
 - If nociception is experienced but not causing arousal, does it need to be treated?
 - Yes stimulation can cause nociception + inflammation + stress
 - Example of having surgery with just propofol, and patient would wake up in pain.
- Amnesia no good monitor. Inferred from EEG signals consistent w/ unconsciousness.
 - Isoelectric EEG = highly unlikely that patient is conscious. Based on studies in rats, and more recently in primates: isoelectric to slow firing shows sparse neurons firing in brain. Very difficult for neurons to fire synchronously as with slow oscillations.
 - \circ EEG signals under anesthesia are very strong. Less subject to artifact (eg movement) as in other study environments. Oscillations 5µV in gamma band when awake, 20-50µV in alpha band.. much larger and noise free under anesthesia.

How does the BIS monitor work?

- **Proprietary algorithm** suggests that signal strong enough to approximate systematic changes in EEG through level of consciousness.
- Bispectrum looks at coupling of oscillations. With Hz 1 and Hz2, how tightly are they coupled?
- Nonlinear coupling: one wave form = sine of omega 1; sine wave of omega 2. Multiply by each other. Why multiply? If adding, would see two signals on a spectrogram, looking at amplitudes. If multiply, will see signaling at base frequencies as well as (omega 1 omega 2). Triplet of amplitude has more information about coupling.

How to use EEG rather than BIS #?

- Signal decomposition by looking at amplitudes of components as function of frequency (increase in amplitude, decrease in frequency). EEG patterns change specifically w/ administration of 1) drug dose; 2) drug class; and 3) age.
 - Putting all this info on a scale from 0-100 will lose nuance. Proprietary algorithm adds element of uncertainty in care that can be otherwise avoided
 - See EEG reading tutorials: <u>https://eegforanesthesia.iars.org/</u>
- How to use this in practice?
 - TIVA propofol in patient in mid 20s: presence of alpha and delta oscillations on spectrogram = unconscious. Investigated with subjects and mathematical models.
 - If see burst suppression, then suggests going down on anesthesia. If HR/BP change, alpha waves increase in frequency, slow oscillations start disappearing = gaining consciousness

How to interpret EEG with drug combinations?

- <u>2015 pape</u>r shows hierarchy: specific oscillations on sevo or prop. Adding remifentanil will not change oscillations because masked. If dialing back sevo/prop, remi still maintains level of unconsciousness.
- Ketamine? Low dose → active brain by inhibiting inhibitory interneurons. Level where
 patients might see hallucinations. At higher doses, will see fast and slow oscillations. Infusion
 w/ ketamine + propofol will show oscillations at halfway point because competing for
 interneuron.
 - Propofol works by enhancing inhibitory interneurons (GABAergic)

References

EEG reading tutorials: https://eegforanesthesia.iars.org/

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Dental Anesthesia info:

Contacts:

Thomas Whitmer: thomas.whitmer.tw@gmail.com

Mana Saraghi: msaraghi@gmail.com

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http://www.ada.org/~/media/CODA/Files/anes.ashx

American Society of Dentist Anesthesiologists website <u>https://www.asdahq.org</u>

Virtual tour https://www.youtube.com/watch?v=ZJ1Mo65ah4w

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