EEG IN IOM

David Betts,
R.EEG/EP T., CNIM, CLTM, R.NCS T., CNCT
Beaumont Health System
Relationship of cerebral metabolism to surface EEG
Gross arterial blood supply
Indications for EEG in surgery
Value of EEG in OR
Effect of anesthesia on EEG
Mechanism of cortical damage
Derived EEG display – CSA
EEG signs of cortical distress
EEG revealing depth of anesthesia for neuroprotection
Adaptation for OR use - technique
Not discussing
direct cortical recording
in this presentation
nor the use of EEG in BIS or Entropy anesthesia monitors
Neuron Metabolism

- Constant need for glucose and oxygen
- Brain 2% of body weight, 20% of O2 consumption, 25% of glucose consumption
- Cerebral Blood Flow (CBF) 57ml per 100g of brain weight per minute

Mechanism Of Cortical Activity Resulting In EEG Waveforms

EEG is reflective of post-synaptic ion fluctuations in the outer radially orientated neurons.

**Figure 5-26.** Schematic model of cortical activity (polarity) in relation to cellular activity. Note the polarity of surface cortical activity is dependent upon the depth of the source and sink of the current flow. In A, thalamo-cortical input enter superficial layer of the cortex, while in B, it enters into deeper layer of the cortex.

Practical Guide for Clinical Neurophysiologic Testing,
Yamada & Meng
HYPOXEMIA

- Blood Brain Barrier Disruption
- Brain Edema
- SEIZURES
- STATUS EPILEPTICUS

- Ionic Shifts and Depolarization
  - $[\text{Na}^+]$
  - $[\text{K}^-]$
  - $[\text{Ca}^{2+}]$

- Energy Failure and Acidosis
  - ATP, PCR, glucose

- Energy Failure and Acidosis
  - FFA
  - Free Radical Lipid Peroxidation

- Neurotransmitter Release
  - (e.g., $\gamma$-Glutamate, monoamines)
  - Neurotransmitter Reuptake

- Ca$^{2+}$ Activated Phospholipid Degradation

- DISRUPTED CELL MEMBRANES AND METABOLIC MACHINERY
  - Focal Infarction
  - Delayed Neuronal Death
  - Transient Neuronal Dysfunction

www.nichd.nih.gov/.../pubs/acute/acute.cfm
REFRESH TERMINOLOGY

α  alpha  8-12 Hz
β  beta   13 Hz and above
θ  theta  4-7 Hz
δ  delta  3 Hz and below

Burst suppression = electrical inactivity punctuated by brief (1-4 seconds) of paroxysmal EEG activity.
### Relationship Between EEG Activity and Perfusion

<table>
<thead>
<tr>
<th>CBF (ml·100 g·min)</th>
<th>EEG Change</th>
<th>Cellular Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>35-50</td>
<td>Normal</td>
<td>• Decreased Protein Synthesis</td>
</tr>
</tbody>
</table>
| 25-35               | Loss of Faster Frequencies (8-14 Hz) | • Anaerobic Metabolism  
• Neurotransmitter Release (i.e. glutamate)  |
| 18-25               | Increasing Slower Frequencies (4-7 Hz) | • Lactic Acidosis  
• Declining ATP |
| 12-18               | Increasing Slower Frequencies (1-4 Hz) | • Sodium-Potassium Pump Failure  
• Increased Intracellular Water Content  |
| <10-12              | Suppression | • Calcium Accumulation  
• Anoxic Depolarization  
• Cell Death |

**Figure 1.** The relationship of cerebral blood flow to electroencephalogram (EEG) and pathophysiology. ATP, adenosine triphosphate (CBF). Data from [2,4].

*Foreman and Claassen Critical Care 2012, 16:216*  
[http://ccforum.com/content/16/2/216](http://ccforum.com/content/16/2/216)
“The most common and sensitive analog EEG change is attenuation of anesthetic-induced, fast activity (low beta, high alpha), which occurs in 14-47% of the patients following carotid cross-clamping [41, 42]. Increased delta activity is almost always associated with decreased amplitudes of higher frequency activity.”
ACNS’s intraoperative EEG guidelines (2000) defined three degrees of EEG changes caused by ischemia:

1) the first degree--a decrease in background fast activity, most apparent when using anesthetic agents that generate such fast activity (the diminution is considered significant if it exceeds 50-60% of baseline),

2) the second degree--an increase in slow (delta-theta) which should be considered clinically significant if it exceeds 50% of baseline (a decrease in fast activity may be simultaneous)

3) the third degree--all EEG activity progressively diminishes in amplitude and approaches iso-electricity
GROSS ARTERIAL DISTRIBUTION IN RELATIONSHIP TO EEG LEADS

Approximate locations in relationship to surface EEG.

Anterior Cerebral Artery – FZ, CZ

Middle cerebral Fpz, F3,4 C3,4, P3,4 F7,8 T7,8, P7,8

Posterior cerebral O1,2

Neuroscience. 2nd edition.
Purves D, Augustine GJ, Fitzpatrick D. et al., editors.
Cortical vascular territories

Fz  Cz  Pz
F3  C3  P3
F7  T7  P7  O1

- Anterior cerebral artery
- Middle cerebral artery
- Posterior cerebral artery

Line drawing of brain by Patrick Lynch (patricklynch.net)
Note that anterior cerebral artery is mostly medial brain and along the vertex.
EEG is a good indicator of cortical neurological activity, BUT does that translate into usefulness in surgery?

The evidence is not conclusive.

What works in rats doesn’t mean routine human use.
Results: The mean reported perioperative stroke rate for CEAs with routine shunting was 1.4% and for routine nonshunt was 2%. Meanwhile, the mean perioperative stroke rates for selecting shunting were 1.6% using EEG, 4.8% using TCD, 1.6% using SP, 1.8% using SSEP, and 1.1% for CBA [regional anesthesia]. Similar results were noted for perioperative stroke and death rates.

Conclusions: The use of routine shunting and selective shunting was associated with a low stroke rate. Both methods are acceptable, and the individual surgeon should select the method with which they are more comfortable. (J Vasc Surg 2011;54:1502-10.) Ali F. AbuRahma, MD, Albeir Y. Mousa, MD, and Patrick A. Stone, MD,
Tumor resection near major arteries
Unintentional occlusion of artery
Cerebral aneurysm clipping
Gage anesthesia for “Burst suppression”
Inadvertent clipping supply to brain
Neuroprotection
Drug induced coma
Hypothermia
Neuroprotection

Hypothermia

“We included four trials of cooling for cerebral protection during brain surgery, involving a total of 1219 patients. We did not find any evidence that hypothermia for neuroprotection in patients undergoing brain surgery is either effective or unsafe when compared to normothermia”.

Cooling for cerebral protection during brain surgery

Wilson Roberto Oliveira Milani¹,*
Pedro L Antibas¹,
Gilmar F Prado²

Published Online: 5 OCT 2011 Cochrane Reviews
Electroencephalographic burst suppression surgery with propofol during cardiac valve replacement did not significantly reduce the incidence or severity of neurologic or neuropsychologic dysfunction. The authors' results suggest that neither cerebral metabolic suppression nor reduction in cerebral blood flow reliably provide neuroprotection during open heart surgery.

Anesthesiology. 1999 May;90(5):1255-64.


Ischemia Research and Education Foundation, San Francisco, California, USA. dtb@crucis.iref.org
IF YOU ARE ASKED:

DESPITE THE FOREGOING
CONSIDER THE TECHNICAL
ASPECTS OF DOING EEG IN OR
Anesthesia effects on EEG
LOOKING SOLELY AT EEG, THE EFFECTS OF ANESTHETIC DRUGS CAN MIMIC GLOBAL HYPOXIA.
ANESTHETIC DRUG EFFECTS ON EEG


Found on Epilepsy.com/Professionals
### Isoflurane (Desflurane similar)

<table>
<thead>
<tr>
<th>Effect on EEG Frequency</th>
<th>Effect in EEG Amplitude</th>
<th>Burst Suppression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sub-anesthetic</td>
<td>Loss of α, ↑ frontal β</td>
<td>↓</td>
</tr>
<tr>
<td>Anesthetic</td>
<td>Frontal 4- to 8-Hz activity</td>
<td>↑</td>
</tr>
<tr>
<td>Increasing dose &gt;1.5 MAC</td>
<td>Diffuse θ and δ → burst suppression → silence ↑ → 0</td>
<td></td>
</tr>
</tbody>
</table>

### Nitrous oxide (alone)

- Frontal fast oscillatory activity (>30 Hz) ↑, especially with inspired concentration >50% No
<table>
<thead>
<tr>
<th>Drug</th>
<th>Effect Frequency</th>
<th>Effect Amplitude</th>
<th>Burst Suppression</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Barbiturates</strong></td>
<td></td>
<td></td>
<td>Yes, with high doses</td>
</tr>
<tr>
<td>Low dose</td>
<td>Fast frontal $\beta$ activity</td>
<td>Slight ↑</td>
<td></td>
</tr>
<tr>
<td>Moderate dose</td>
<td>Frontal $\alpha$ frequency spindles</td>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>Increasing high dose</td>
<td>Diffuse $\delta \rightarrow$ burst suppression $\rightarrow$ silence↑↑↑↑ → 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Etomidate</strong></td>
<td></td>
<td></td>
<td>Yes, with high doses</td>
</tr>
<tr>
<td>Low dose</td>
<td>Fast frontal $\beta$ activity</td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>Moderate dose</td>
<td>Frontal $\alpha$ frequency</td>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>Increasing high dose</td>
<td>Diffuse $\delta \rightarrow$ burst suppression $\rightarrow$ silence↑↑ → 0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## Benzodiazepines

<table>
<thead>
<tr>
<th>Effect on EEG Frequency</th>
<th>Effect in EEG Amplitude</th>
<th>Burst Suppression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low dose</td>
<td>Loss of α,</td>
<td>NO</td>
</tr>
<tr>
<td></td>
<td>increased frontal β activity</td>
<td>↓</td>
</tr>
<tr>
<td>High dose</td>
<td>Frontally dominant δ and θ</td>
<td>↑</td>
</tr>
</tbody>
</table>
### IV ANESTHESIA DRUGS

#### Opiates

<table>
<thead>
<tr>
<th>Dose</th>
<th>Effect on EEG Frequency</th>
<th>Effect in EEG Amplitude</th>
<th>Burst Suppression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low dose</td>
<td>Loss of $\beta$, $\alpha$ slows ↔</td>
<td>↑</td>
<td>No</td>
</tr>
<tr>
<td>Moderate dose</td>
<td>Diffuse $\theta$, some $\delta$</td>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>High dose</td>
<td>$\delta$, often synchronized</td>
<td>↑↑</td>
<td></td>
</tr>
</tbody>
</table>
### IV ANESTHESIA DRUGS

#### Propofol

<table>
<thead>
<tr>
<th>Effect on EEG Frequency</th>
<th>Effect in EEG Amplitude</th>
<th>Burst</th>
<th>Suppression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low dose</td>
<td>Loss of α, ↑ frontal β</td>
<td>↓</td>
<td>Yes</td>
</tr>
<tr>
<td>Moderate dose</td>
<td>Frontal δ, waxing-waning α</td>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>Increasing high dose</td>
<td>Diffuse δ → burst suppression → silence</td>
<td>↑↑ → 0</td>
<td></td>
</tr>
</tbody>
</table>
### IV ANESTHESIA DRUGS

**Ketamine**

<table>
<thead>
<tr>
<th>Dose Level</th>
<th>Effect on EEG Frequency</th>
<th>Effect in EEG Amplitude</th>
<th>Burst Suppression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low dose</td>
<td>Loss of α, ↑ variability</td>
<td>↑↓</td>
<td>No</td>
</tr>
<tr>
<td>Moderate dose</td>
<td>Frontal rhythmic θ</td>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>High dose</td>
<td>Polymorphic δ, some β↑</td>
<td>↑ (β is low amplitude)</td>
<td></td>
</tr>
</tbody>
</table>
EFFECT OF ANESTHESIA ON EEG
DEMONSTRATED DURING INTUBATION

Starts with patient awake and proceeds to burst suppression
Right frontal leads omitted because that was the area of surgery.
O1 omitted in early recording because of artifact.
AWAKE

- Alpha
- Beta
- Eye blinks
AWAKE TENSE

- Eye
- Blinks
- EMG Activity From Tense Muscles
- Lack of Posterior Alpha
PROPOFOL INDUCTION OF ANESTHESIA IMMEDIATELY POST BOLUS

Higher amplitude frontal beta

Alpha replaced by beta

Bi-frontal 2-3 Hz
ISOFLURANE AND PROPOFOL
PRE-INTUBATION
EEG

ISOFURANE 0.9%

SUFENTA 0.2 MCG/KG/HR
COMPRESSED SPECTRAL ARRAY (CSA)

Converts EEG from time domain to frequency domain (x-axis)

EEG 10 seconds

CSA displays amount of frequency in 8 seconds
COMPRESSED SPECTRAL ARRAY (CSA)

Condense information

EEG 10 seconds per screen

CSA 20 lines shows 160 seconds
COMPRESSED SPECTRAL ARRAY (CSA)
COMPARE DISPLAY OF INFORMATION AWAKE EEG
COMPRESSED SPECTRAL ARRAY (CSA)
BURST – SUPPRESSION EEG
COMPRESSED SPECTRAL ARRAY (CSA)

Be Aware – Artifact can look like EEG on the CSA

What does the raw EEG look like?
ELECTRODES IN WATER
CSA 30 MINUTES OF DATA
Spectral edge = frequency at which 95% (or another frequency of choice) power occurs below.
CSA & SPECTRAL EDGE FREQUENCY (SEF)
SSEP sensitivity
SSEP localization

UE SSEP – Middle Cerebral Artery
LE SSEP – Anterior Cerebral Artery

SSEP will be maintained during burst-suppression EEG
Can monitor for ischemia when EEG is not dependable
EEG is quickly effected by ischemia
SSEP take the time of the average about 1-2 minutes
SPECIFIC APPLICATIONS - CEA

Carotid endarterectomy – CEA

1. ischemic changes from clamping
SPECIFIC APPLICATIONS -
SPECIFIC APPLICATIONS - CEA

Baseline Median SSEP
CLAPING
SSEP DURING CEA

FINAL TRACING – RETURN TO BASELINE
Goal – total suppression of cerebral activity

EEG – Iso-electric $18^\circ C$

Burst Suppression $23^\circ C$
SPECIFIC APPLICATIONS – ANEURYSM

1. Neuroprotection by deep anesthesia – burst suppression

2. Ischemic changes secondary to clipping
Possible Surgical Sites - Aneurysm Circle of Willis
What are progressive EEG changes with reduced blood flow?

- Loss of fast
- Increase of slow
- Flat
What is the significant of these blood flow values?

Normal: 57 ml/100gm/min
EEG changes: 35
Ischemic threshold: 18
Infarction: 12
A FIM