Rapid, Realtime ICU and ER EEG Monitoring

Kenneth G. Jordan, MD, FACP, FACNS, FAAN
President, Jordan NeuroScience, Inc.
Assoc. Clin. Prof of Biomedical Science, Neurology
University of California, Riverside CA
Disclosure

- Principal shareholder of Jordan NeuroScience, Inc., a medical device manufacturer in the field of acute EEG monitoring
- Certain JNS proprietary products and methods are included in this presentation.
- No money or fees from outside sources
What is Emergency EEG Monitoring (EmEEG)?

1. EEG that is done acutely for new onset or rapidly deteriorating brain dysfunction.

2. EEG done within a critical time-window to reverse, reduce or prevent brain damage.

3. EEG surveillance of intervention during acute or unstable brain injury.

“Dost thou love life? Then do not squander time, for that is the stuff life is made of.” Benjamin Franklin
### CEEG = “EKG MONITORING” OF THE BRAIN

<table>
<thead>
<tr>
<th>EKG-1903</th>
<th>EEG-1928</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. SENSITIVE TO CARDIAC ISCHEMIA</td>
<td>SENSITIVE TO CEREBRAL ISCHEMIA</td>
</tr>
<tr>
<td>2. DETECTS CARDIAC ISCHEMIA AT A REVERSIBLE STAGE</td>
<td>DETECTS CEREBRAL ISCHEMIA AT A REVERSIBLE STAGE</td>
</tr>
<tr>
<td>3. CORRELATES WITH CARDIAC BLOOD FLOW</td>
<td>CORRELATES WITH CEREBRAL BLOOD FLOW</td>
</tr>
<tr>
<td>4. RAPIDLY AND ACCURATELY DETECTS CARDIAC ARRHYTHMIAS</td>
<td>RAPIDLY AND ACCURATELY DETECTS EPILEPTIC ACTIVITY</td>
</tr>
</tbody>
</table>
WHY DO ICU-CEEG?

“To Detect and Protect”

1. To detect abnormalities at a reversible stage

2. To prompt timely and physiologically sound clinical decisions.

3. To monitor the benefit or harm of our interventions
ICU CEEG = The Rodney Dangerfield of Monitoring

With all this fancy technology, I hope someone is monitoring my brain!!
INDICATIONS FOR ICU-CEEG

1) Detection of subclinical seizures*
2) Differentiate non-seizure paroxysmal events
3) Management of burst-suppression medical coma
4) Detecting cerebral ischemia*
5) Monitoring level of sedation
6) Monitoring response to interventions*
Detection of Subclinical Seizures

The “Critical Care EMU”
## Incidence of NCS and NCSE

<table>
<thead>
<tr>
<th>PTS WITH NCS</th>
<th>43</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTS MONITORED</td>
<td>124</td>
</tr>
</tbody>
</table>

\[
\frac{43}{124} = 34\%
\]

<table>
<thead>
<tr>
<th>PTS WITH NCSE</th>
<th>33</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTS WITH NCS</td>
<td>43</td>
</tr>
</tbody>
</table>

\[
\frac{33}{43} = 76\%
\]

## Clinical Features of NCS (n = 43)

<table>
<thead>
<tr>
<th>Feature</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>COMA</td>
<td>37</td>
</tr>
<tr>
<td>Aphasia</td>
<td>18</td>
</tr>
<tr>
<td>Mental Dullness</td>
<td>14</td>
</tr>
<tr>
<td>Limb Posturing</td>
<td>12</td>
</tr>
<tr>
<td>Abn Eye Movements</td>
<td>12</td>
</tr>
<tr>
<td>Automatisms</td>
<td>5</td>
</tr>
<tr>
<td>Cortical Blindness</td>
<td>2</td>
</tr>
</tbody>
</table>

\[
\frac{37 + 18 + 14 + 12 + 12 + 5 + 2}{43} = 100\%
\]
PERSISTING COMA WITH SMALL SDH

From: Jordan KG. JCN(1999):16:14-39
**EEG CRITERIA for NONCONVULSIVE SEIZURE**

**Guideline:** At least one primary criterion and the secondary criterion. Discharges are > 10 seconds in duration

**Primary criteria**

1. Repetitive generalized or focal spikes, sharp waves, spike-and-wave or sharp-and-slow wave complexes at > 3/second.
2. Repetitive generalized or focal spikes, sharp waves, spike-and-wave or sharp-and-slow wave complexes at < 3/second *and secondary criterion.*
3. Sequential rhythmic, periodic or quasi-periodic waves at >/= 1/sec with *unequivocal evolution* in frequency, location, or morphology.

**Secondary criterion**

Significant improvement in clinical state, resolution of epileptiform activity, and improvement of background EEG patterns (e.g. re-appearance of posterior dominant rhythm) temporally linked to intravenous administration of rapidly acting anti-epileptic drug.

*After Young GB, Jordan KG. Neurology, 1996, with modifications by Chong DJ, Hirsch LJ. JCN 2005*
1. Only 20 had clinical seizures at time of diagnostic EEG

2. 45% (31/67) non-anoxic patients improved in alertness on antiseizure drugs (including 15 who were comatose).

Drislane FW et al Neurology 1998;50 (suppl 1)
NCSE in Hepatic Encephalopathy

AMMONIA LEVEL=135

AMMONIA LEVEL UNCHANGED
### Generalized Status Epilepticus

#### Convulsive vs Nonconvulsive SE

<table>
<thead>
<tr>
<th></th>
<th>Convulsive (n=384/68% )</th>
<th>Nonconvulsive (n=184/32% )</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of epilepsy (%)</td>
<td>42.4</td>
<td>12.7</td>
</tr>
<tr>
<td>History of status epilepticus (%)</td>
<td>12.8</td>
<td>4.5</td>
</tr>
<tr>
<td>Median duration of status epilepticus at enrollment (hr)</td>
<td>2.8</td>
<td>5.8</td>
</tr>
<tr>
<td>Causal factors (%)*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Remote neurologic cause</td>
<td>69.5</td>
<td>34</td>
</tr>
<tr>
<td>Acute neurologic cause</td>
<td>27.3</td>
<td>37.3</td>
</tr>
<tr>
<td>Life-threatening medical condition</td>
<td>32.0</td>
<td>56.7</td>
</tr>
</tbody>
</table>

*1 Patient had more than one causal factor

At 11:30 p.m., a 76-year-old woman on theophylline for COPD, suddenly became bewildered with incoherent speech and suffered a generalized convulsive seizure (GCS). Paramedics observed four additional GCS without the patient awakening en route to the local hospital ED. The patient arrived at the ED at 12:05 a.m. comatose with stertorous respirations. The ED physician administered 4mg of Lorazepam I.V. concurrent with I.V. fosphenytoin 20 mg/kg at 150 mg/min. A CAT scan was normal for the patient’s age. No further convulsions were seen, but she remained unresponsive.
NCSE in Comatose Patients

(n = 610 patients)

1. 33% (40/119) of comatose children were in NCSE.

2. 22% (108/491) of comatose adults were in NCSE.

NCSE in Intracerebral Hemorrhage

1. 18-25% patients had NCS on CEEG
2. CEEG detected four times as many seizures as occurred clinically
3. NCS was associated with progressive cerebral edema, midline shift and clinical worsening
4. Early detection and control of seizures in ICH may improve clinical outcome.

STABLE ICH WITH DECLINING GCS

PLEDS on Standard EEG

NCSE on ICU-CEEG
NCS/NCSE OCCUR COMMONLY IN ALL TYPES OF ABI
(Table shows %NCSE / %NCS)

<table>
<thead>
<tr>
<th>Study</th>
<th>CSE</th>
<th>INF</th>
<th>TBI</th>
<th>ICH</th>
<th>SAH</th>
<th>NSG</th>
<th>ALOC</th>
<th>ASBL</th>
<th>ME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Towne et al (2000)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>8/8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Young and Doig (2005)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>?/32</td>
</tr>
<tr>
<td>Treiman et al (1998)</td>
<td>32/?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

*Monitored 48-72 hours to R/O NCSE
“HIERARCHY” OF RISK FOR NCS/NCSE

1. CSE>INF>NSG>SAH>TBI>ME>ALOC>ICH>AIS
2. Incidence of NCS is 8-48%, depending on Dx
3. Incidence of NCSE is 8-20%, depending on Dx
4. NCSE occurs in av. 47% of patients with NCS.
Mortality Increases With the Duration of NCSE

- **Etiology:**
  - Remote symptomatic: 16% (4/25) \( p = 0.009 \)
  - Acute symptomatic: 46% (11/24) \( OR = 6.0 \)

- **NCS only vs. NCSE**
  - 12% vs. 54% \( OR = 10.0 \) \( p = 0.002 \)

- **Seizure Duration:**
  - <10 h: 10% (3/30) \( p = 0.0006 \)
  - 10-20 h: 33% (2/6)
  - >20 h: 85% (11/13) \( OR = 1.093/h \)

- **Delay to Diagnosis**
  - <0.5 h: 36% (5/14)
  - >1 <24 h: 39% (7/18)
  - ≥24 h: 75% (6/8) \( OR 1.039/h \) \( p = 0.00001 \)

* Young GB, Jordan KG., Doig G. Neurology, 1996
NCSE and Excitotoxicity in Head Trauma

[Glutamate]

SEIZURES

Time (hrs)

 Courtesy of Paul Vespa, MD
 UCLA School of Medicine
INDETERMINATE PATTERNS

SI-GRDA: Stimulus-induced generalized rhythmic delta activity. In this case, the pattern was elicited by succinylcholine.

PERIODIC TRIPHASIC WAVES

SIRPIDS

GPEDS
DETECTING AND MONITORING CEREBRAL ISCHEMIA

Based on: Jordan KG. JCN(2004);21:341-352
# Ischemic EEG Changes in Carotid Clamping

<table>
<thead>
<tr>
<th></th>
<th>Before clamping</th>
<th>After clamping</th>
<th>4 min after shunting</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CBF</td>
<td>Time in sec</td>
<td>CBF</td>
</tr>
<tr>
<td>♂ Age: 50 Yr</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Left)</td>
<td>72</td>
<td>150</td>
<td>17</td>
</tr>
<tr>
<td>T₃ - C₃</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Right)</td>
<td>48</td>
<td>50</td>
<td>15</td>
</tr>
<tr>
<td>C₄ - T₄</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>♂ Age: 53 Yr</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Left)</td>
<td>35</td>
<td>50</td>
<td>5</td>
</tr>
<tr>
<td>T₃ - C₃</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>♂ Age: 77 Yr</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Left)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T₃ - C₃</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1-70 Hz

50 μv

2 sec
<table>
<thead>
<tr>
<th>CBF LEVEL (ml/100gm/min)</th>
<th>EEG CHANGE</th>
<th>DEGREE OF NEURONAL INJURY</th>
</tr>
</thead>
<tbody>
<tr>
<td>35-70</td>
<td>NORMAL</td>
<td>NO INJURY</td>
</tr>
<tr>
<td>25-35</td>
<td>LOSS OF FAST BETA FREQUENCIES</td>
<td>USUALLY REVERSIBLE</td>
</tr>
<tr>
<td>18-25</td>
<td>SLOWING OF BACKGROUND TO 5-7HZ THETA</td>
<td>POTENTIALLY REVERSIBLE</td>
</tr>
<tr>
<td>12-18</td>
<td>SLOWING TO 1-4HZ DELTA</td>
<td>POTENTIALLY REVERSIBLE</td>
</tr>
<tr>
<td>&lt; 8-10</td>
<td>SUPPRESSION OF ALL FREQUENCIES</td>
<td>NEURONAL DEATH</td>
</tr>
</tbody>
</table>
CBF-EEG CORRELATION IN AIS

A

CBF=16.1

B

CBF=16.1

C

50μV x 1sec  LF=1Hz HF=70Hz
RCBF = 79

Normalized EEG; Resolved Deficits
Alpha Variability And Alpha-delta Ratio In SAH Vasospasm

DAY 1: APHASIC & DROWSY
TCD=220; BP=160/110 (MAP=110)

Bilaterally Abnormal Bilaterally Abnormal EEG L>R*

*Inferred RCBF: LFT: critical at 10-15 ml/100g/min
RHem: moderate at 18-25
DAY 3: NML
TCD=180; BP=200/110 (MAP=150)

DAY 12: NML
TCD=112; BP=140/80 (MAP=100)

*EEG worsens with ↓ in MAP
Impaired cerebral autoregulation

**EEG stable with ↓ in MAP
Return of normal cerebral autoregulation
<table>
<thead>
<tr>
<th>Impact</th>
<th>No. of PTS</th>
<th>(%) Total</th>
<th>ACI</th>
<th>HEM</th>
<th>SZ</th>
<th>MC</th>
<th>BT</th>
<th>INF</th>
<th>HT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decisive</td>
<td>109</td>
<td>(54)</td>
<td>28</td>
<td>22</td>
<td>36</td>
<td>9</td>
<td>8</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Contributing</td>
<td>64</td>
<td>(32)</td>
<td>16</td>
<td>16</td>
<td>5</td>
<td>10</td>
<td>5</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>None</td>
<td>27</td>
<td>(14)</td>
<td>13</td>
<td>5</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>200</td>
<td>(100)</td>
<td>57</td>
<td>43</td>
<td>44</td>
<td>20</td>
<td>16</td>
<td>13</td>
<td>7</td>
</tr>
</tbody>
</table>
Rationale for Em-EEG:

Timeline of Secondary Brain Injury

1. Hypoxia; hypotension; status epilepticus, cerebral ischemia, ICP
2. Status epilepticus, cerebral ischemia, ICP
3. Status epilepticus, cerebral ischemia, ICP
4. Status epilepticus, ICP, cerebral ischemia, ICP, vasospasm, infection, PE

“The chief function of the body is to carry the brain around.” Thomas Edison.”
Time Window for Successful Treatment of Status Epilepticus

“As acute brain injury progresses, human nervous tissue is rapidly and irretrievably lost. Therapeutic interventions should be emergently pursued.”  

J. Saver*

Why is EmEEG Important?:
Because “Time is Brain”

1. Delay in Stroke: 2 million neurons die per minute*

2. Delay in NCSE: 1-2%/hr increased mortality.**

“The dogmas of the quiet past are inadequate to the present. As our case is new, so we must act anew.”  *Abraham Lincoln*

<table>
<thead>
<tr>
<th>Category</th>
<th>Conventional EEG</th>
<th>EmEEG</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Kinds of patients</strong></td>
<td>Chronic Brain Problem</td>
<td>Acute Brain Injury</td>
</tr>
<tr>
<td><strong>2. Consequences of delay</strong></td>
<td>Inconvenient</td>
<td>Damaging or Deadly</td>
</tr>
<tr>
<td><strong>2. Location of test</strong></td>
<td>OP Lab, EMU</td>
<td>EMS, ER, ICU, Hosp</td>
</tr>
<tr>
<td><strong>3. Technical priorities</strong></td>
<td>Diligence and precision</td>
<td>Speed and accuracy</td>
</tr>
<tr>
<td><strong>5. Test Availability</strong></td>
<td>Work hours</td>
<td>24/7</td>
</tr>
<tr>
<td><strong>6. Results needed</strong></td>
<td>24-48 hours</td>
<td>Real-time/Emergent</td>
</tr>
</tbody>
</table>
“Human beings are almost unique in having the ability to learn from the experience of others, but are disinclined to do so.” Douglas Adams

Acute Cardiac Injury

EmEKG
Freq VPC’s = 20%*
VT/VF = 8%**

Acute Brain Injury

EmEEG
NCS = 23%ª
NCSE = 13%ª

Indications for Emergency EEG: ACEP 2004

1. Refractory status epilepticus
2. Persistent altered consciousness
3. NCSE after GCSE
4. Pharmacological paralysis-sedation
5. Coma
1. EmEEG is rarely done for status epilepticus

2. EmEEG availability in <10% hospitals ERs.

3. EmEEG response time from request to interpretation is >4x longer than the treatment window for SE. (4.5 hours)

4. Why? 1) Unawareness and 2) Limited access to EEG expertise

“Our Age’s anxiety is the result of trying to do today's jobs with yesterday's tools.” Marshall McLuhan.
Scope of Emergency Seizures in the U.S.

1. 2,300,000 Sz patients seen in ER's/year
2. 1,900,000 required IV's, cardiac monitoring, blood tests.
3. 1,140,000 were given anti-seizure medications
4. 600,000 were admitted to hospital
5. 750,000 had new onset seizures
6. 185,000 had status epilepticus.
7. Diagnosis of seizure based on hx and clinical observation. Rare EEG confirmation

1. Non-convulsive Status Epilepticus (NCSE)

1. NCSE often goes unrecognized or is mistaken for behavioral or psychiatric disturbance. (1)

2. 31% of patients with convulsive SE persist in NCSE after “adequate” treatment. (2)

3. 7-14% of ED Sz pts had NCSE by EEG (3,4,5)

4. 16%-27% of ED ALOC pts had definite NCSE or active epileptic spikes on EEG (6)

2. **Non-Epileptic (“Pseudo”) Seizures**

1. Range of 8-26% incidence of ED Sz pts have unsuspected NES found by EEG (1,2,3)

2. 10% pts with presumed epileptic status have pseudo-status by EEG (3)

3. Risk: AEDs, IV sedation, 50% of NESE intubated (4); death from unrecognized NESE (5).

*Normal EEG activity with movement artifact in actively “seizing” patient with NES (rectangle)*

EmEEG in ED Patients With Seizures or ALOC

Does EmEEG change initial ED Dx/Rx?

237 Patients: 164 Sz; 73 ALOC:

Jordan K, Schneider A. Neurocritical Care 2004; 1:257 (abstr)
### Impact of EmEEG in 273 Sz/ALOC Patients

<table>
<thead>
<tr>
<th>EmEEG Dx</th>
<th>NCSE</th>
<th>NES</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (%)</td>
<td>23 (8)</td>
<td>30 (11)</td>
<td>53 (19)</td>
</tr>
<tr>
<td>Change Rx</td>
<td>+ AED</td>
<td>- AED</td>
<td>19% reversal</td>
</tr>
</tbody>
</table>

- **EmEEG Dx**: EmEEG Diagnosis
- **NCSE**: Neuroleptic-Care Syndrome
- **NES**: Neuroleptic-Induced Syndrome
- **TOTAL**: Total
- **N (%)**: Number and Percentage
- **Change Rx**: Change in Treatment
- **+ AED**: Treatment with AED
- **- AED**: Treatment without AED
- **19% reversal**: Percentage of reversal
EmEEG in Acute Behavior Change

14 yo Tourette’s Syndrome: Aggressive and Fighting

After IV fosphenytoin, pleasant and cooperative

Also see: Kaplan PW.. Epilepsia.1996;37:643-650
The Dangerous Delays in Emergency EEG

"In delay there lies no plenty."
--William Shakespeare
**Sources of Delay in EmEEG**

<table>
<thead>
<tr>
<th>Source of Delay</th>
<th>Time (hours/minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrodes Set-Up by on call EEG tech</td>
<td>1.5-2.3 hr *</td>
</tr>
<tr>
<td>Time to run study</td>
<td>30-45 min</td>
</tr>
<tr>
<td>Neurologist’s interpretation of study</td>
<td>1.5 hr-48 hours (av 3hrs.)**</td>
</tr>
</tbody>
</table>

*Jordan K, Crit Care Med., 2004,*  
**Quigg et al. J. Clin Neurophys. 1999**
## On-call EEG TechTime-Motion Study

<table>
<thead>
<tr>
<th>Task</th>
<th>Duration (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Page…get ready…leave</td>
<td>15-30</td>
</tr>
<tr>
<td>Drive Time/parking</td>
<td>15-30</td>
</tr>
<tr>
<td>To Lab for supplies</td>
<td>15</td>
</tr>
<tr>
<td>Set up equipment at</td>
<td>15</td>
</tr>
<tr>
<td>Chart review. Enter history</td>
<td>10-20</td>
</tr>
<tr>
<td>Measure head:10/20</td>
<td>10-20</td>
</tr>
<tr>
<td>Apply electrodes</td>
<td>10</td>
</tr>
<tr>
<td><strong>Total Set-Up Time</strong></td>
<td><strong>1.5 Hr – 2.3hr</strong></td>
</tr>
</tbody>
</table>
“We struggle with the complexities and avoid the simplicities”  Norman Vincent Peale

Conventional EEG: A Profusion and Confusion of Paste, Tape and Wires
Simplifying EmEEG

1. Electrode placement template for EEG set-up

2. “Plug and play” EEG machines located in the ER/ICU

3. Online connectivity to remote EEG reader

4. Abbreviated 10-15 minute recording for triage (“Go-No Go”) decisions:
   1) Seizure?
   2) Structural process?
   3) Encephalopathy?
   4) Medication/sedation?
   5) Brain Death?
   6) Normal?
“Imagination plus innovation equals realization.”
Dennis Waitley

**Simplifying EmEEG**

1. **5-minute ER EEG:** 2/25 ALOC patients in NCSE
   
   *(Bautista et al. JCN 2007;24:16-21)*

2. **Inpatient 24/7 EmEEG:** 8%-10% NCSE in ALOC patients in hospital
   
   *(Towne et al., Neurology 2000; 54:340-5)*

3. **Wireless EmEEG:** 11% NCS
   

4. **EmEEG Set-Up Devices:** BraiNet, Electro-Cap, E-Net, Subhairline electrodes.

5. **EEG “Critical Care Monitoring Tech”** for 24/7 EmEEG monitoring and pre-reading.
EmEEG Set-Up Template

<table>
<thead>
<tr>
<th></th>
<th>Conventional</th>
<th>BraiNet®</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Set-Up Time</strong></td>
<td>1.5-2.3 hr</td>
<td>10-15 min</td>
</tr>
</tbody>
</table>

"You may delay, but time will not."  
Benjamin Franklin
EmEEG in ER:

EEG Set-Up with BraiNet®

Reader paged and EEG Sent by Internet
ICU: EEG Set-up with BraiNet®
EmEEG Remote Monitoring Team

EmEEG set up and started in ICU or ER

EEG CCMT at home or “Central Station” monitors/adjusts EEG by remote control. Pages expert for significant abnormalities or questions.

Expert checks EEG in office or reviews it on Blackberry
The Future: Anywhere, Anytime, Realtime EmEEG

Brain Injuries happen anywhere, anytime

Connectivity is anywhere, anytime

Expert Reader Network available anywhere, anytime
As Two Great American Philosophers Remind Us:

“If your determination is fixed, I do not counsel you to despair. Few things are impossible to diligence and skill. Great works are performed not by strength, but perseverance.”  
Samuel Johnson

“We have brains in our heads. We have feet in our shoes. We can steer ourselves any direction we choose.”  
Dr. Seuss
Emergency EEG
Anywhere, Anytime
Saves Brains!