Periodic discharges and prognostic significance
Pedersen GL et al, Clin Nphys 2013 (Denmark)

• 102 patients w/ PDs vs 102 age, gender and etiology matched controls, using ACNS nomenclature and read blinded to clinical info for this study
  – PDs: 46% LPDs, 16% GPDs, 4% BIPDs, 34% combos
• Mortality or acquired disability higher if PDs
  – O.R. 2.5, p=0.001
• Logistic regression: PDs were independent predictor of poor outcome (68% vs ~40%; p=0.034)
Periodic discharges and prognostic significance, cont’d

Pedersen GL et al, Clin Nphys 2013 (Denmark)

- Acute seizures: 97% in group with PDs vs 72% in group without (p<0.001)
- Newly developed subsequent epilepsy in survivors: 48% in group with PDs vs 16% without (p=0.002; O.R. 3.3)
  - No longer signif after controlling for clinical factors for overall PDs
- LPDs-plus had higher risk for later epilepsy (>4 weeks) than LPDs alone (p=0.034)
- Continuous or abundant LPDs were assoc’d w/ worse outcome than if frequent or occasional (p=0.006)
- Prolonged or very prolonged PDs assoc’d w/ worse outcome than if shorter duration (p=0.035)
• 4772 patients undergoing CEEG in 3 centers (Emory, Yale, Brigham and Women’s) over 2.5 years
Frequency matters

Figure. Model of Pattern Characteristics and Seizure Risk

Rodriguez Ruiz A et al, CCEMRC, JAMA Neurol 2017
# Prevalence matters

**etable 3: Prevalence of EEG Patterns and Association With Seizures**

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Number of Sessions w/o Seizures</th>
<th>Number of Sessions with Seizures</th>
<th>Odds Ratio (95% CI)</th>
<th>FDR-adjusted p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LRDA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rare/Occasional</td>
<td>159 (79%)</td>
<td>42 (21%)</td>
<td>1.91 (1.25-2.86)</td>
<td>0.006</td>
</tr>
<tr>
<td>Frequent</td>
<td>89 (68%)</td>
<td>42 (32%)</td>
<td>2.75 (1.72-4.36)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Abundant/Continuous</td>
<td>43 (67%)</td>
<td>21 (33%)</td>
<td>3.69 (1.92-6.87)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Not Recorded</td>
<td>6 (43%)</td>
<td>8 (57%)</td>
<td>5.79 (1.43-24.09)</td>
<td>0.04</td>
</tr>
<tr>
<td>GPD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rare/Occasional</td>
<td>172 (92%)</td>
<td>15 (8%)</td>
<td>0.54 (0.28-0.97)</td>
<td>0.10</td>
</tr>
<tr>
<td>Frequent</td>
<td>185 (85%)</td>
<td>32 (15%)</td>
<td>1.49 (0.94-2.29)</td>
<td>0.14</td>
</tr>
<tr>
<td>Abundant/Continuous</td>
<td>202 (77%)</td>
<td>61 (23%)</td>
<td>2.90 (2.00-4.16)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Not Recorded</td>
<td>27 (93%)</td>
<td>2 (7%)</td>
<td>0.78 (0.12-2.84)</td>
<td>0.79</td>
</tr>
<tr>
<td>LPD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rare/Occasional</td>
<td>127 (65%)</td>
<td>68 (35%)</td>
<td>6.26 (4.43-8.80)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Frequent</td>
<td>142 (63%)</td>
<td>85 (37%)</td>
<td>7.44 (5.35-10.31)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Abundant/Continuous</td>
<td>167 (46%)</td>
<td>194 (54%)</td>
<td>12.47 (9.61-16.22)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Not Recorded</td>
<td>13 (68%)</td>
<td>6 (32%)</td>
<td>6.56 (2.13-18.05)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Rodriguez Ruiz A et al, CCEMRC, JAMA Neurol 2017
SI does not seem to matter (same association w/ szs either way)

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Number of Sessions/Total Sessions with Seizures</th>
<th>% of Sessions with Seizures</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRDA – No SI</td>
<td>95/770</td>
<td>12%</td>
</tr>
<tr>
<td>GRDA – SI</td>
<td>27/157</td>
<td>17%</td>
</tr>
<tr>
<td>LRDA – No SI</td>
<td>102/356</td>
<td>28%</td>
</tr>
<tr>
<td>LRDA – SI</td>
<td>11/54</td>
<td>20%</td>
</tr>
<tr>
<td>GPD – No SI</td>
<td>89/569</td>
<td>17%</td>
</tr>
<tr>
<td>GPD – SI</td>
<td>21/127</td>
<td>17%</td>
</tr>
<tr>
<td>LPD – No SI</td>
<td>323/701</td>
<td>49%</td>
</tr>
<tr>
<td>LPD – SI</td>
<td>30/101</td>
<td>30%</td>
</tr>
<tr>
<td>BIPD – No SI</td>
<td>28/93</td>
<td>36%</td>
</tr>
</tbody>
</table>

Rodriguez Ruiz A et al, CCEMRC, JAMA Neurol 2017
Highly Epileptiform Bursts

“Present if multiple epileptiform discharges (traditional definition) are seen within the majority (>50%) of bursts and occur at an average of 1/s or faster. Also present if a rhythmic, potentially ictal-appearing pattern occurs at 1/s or faster within the majority (>50%) of bursts”
Highly Epileptiform Bursts Are Associated With Seizure Recurrence

Stephen A. Thompson* and Stephen Hantus†

# TABLE 3. Classification of Burst Suppression Versus Subsequent Activity

<table>
<thead>
<tr>
<th></th>
<th>SZ</th>
<th>CS</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEB</td>
<td>11</td>
<td>6</td>
<td>17</td>
</tr>
<tr>
<td>Non</td>
<td>0</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Total</td>
<td>11</td>
<td>22</td>
<td>33</td>
</tr>
</tbody>
</table>

A total of 33 examples of burst suppression were dichotomized into HEB or nonepileptiform, compared against whether seizure (SZ) or continuous slow (CS) occurred in the subsequent 24 hours (Fisher’s exact test, $P = 0.0001$).
Highly epilept bursts; sz within 24 h of wean

Non-highly epilept burst; no sz within 24 h of wean

THOMPSON AND HANTUS, J CLIN NPHYSIOL 2016
Resolution of NCSE over 11 Hours
Struck A et al, Ncrit Care 2016 (MGH)
61% of 18 patients on the IIC showed hypermetabolism on PET
Unified EEG criteria for nonconvulsive status epilepticus
“The Salzburg Criteria”

Sándor Beniczky¹,²,*, Lawrence J. Hirsch³, Peter W. Kaplan⁴, Ronit Ressler⁵, Gerhard Bauer⁶, Harald Aurlien⁷,⁸, Jan C. Brøgger⁷,⁸, Eugen Trinka⁹; Epilepsia SEP 2013

- **EDs > 2.5 Hz, or**
- **EDs ≤ 2.5 Hz or rhythmic delta/theta activity (>0.5 Hz) AND one of the following:**
  - EEG and clinical improvement after IV AED, or
  - Subtle clinical ictal phenomena during the EEG patterns mentioned above, or
  - Typical spatiotemporal evolution**

*If EEG improvement occurs without clinical improvement, or if fluctuation without definite evolution, this should be considered possible NCSE.*

**Incrementing onset (increase in voltage and change in frequency), or evolution in pattern (change in frequency >1 Hz or change in location), or decrementing termination (voltage or frequency).
Conclusion: “The Salzburg criteria for diagnosis of NCSE have high diagnostic accuracy and excellent inter-rater agreement, making them suitable for implementation in clinical practice.”
Value of benzo trial for possible NCSE
Hopp JL et al, The Neurologist 2011 (U. Maryland)

• 62 patients with benzo trial for impaired consciousness
  with epileptiform pattern “potentially consistent with NCSE”
  – Examined immediately and for 1 hour
• 22 (35%) had improved consciousness, and all survived
  and had good functional outcome
• 40 (65%) did not respond clinically, and only 14 (35%)
  recovered consciousness
• EEG response w/out clinical also correlated w/ higher
  chance of awakening prior to discharge, but less strongly,
  and no correlation with survival
11 experts blindly reviewed 20 continuous EEG studies containing GPDs

Inter rater agreement for TWs: fair (k=0.33); IRA for gen’d and periodic were near perfect (k=0.81)

Patients with “Triphasic Waves” were as likely to develop seizures (25%) as those without TWs (26%).

Surprisingly, those with TWs were actually less likely to have toxic-metab encephalopathy (55% vs 79%, p<0.01)
Response rates to anticonvulsant trials in patients with triphasic wave EEG patterns of uncertain significance
O’Rourke D, Chen, PM, Gaspard N, Foreman B, McClain L, Karakis I, Mahulikar A, Westover MB, for the CCEMRC, Neurocrit Care 2015

- 3 institutions (Yale, Columbia, MGH), retrospective, N=64 w/ TW pattern who got benzo trial for possible NCSE
  - 72% had metabolic derangement or infection
  - Excluded postanoxia and those with definite status epilepticus (clinical or on EEG)
- Response: resolution of EEG and either unequivocal improvement in encephalopathy or appearance of previously absent normal EEG patterns
  - Divided into immediate (<2 hours) or delayed
- Benzo trials: 83% loraz, mean dose 2.5 mg; 17% midaz, mean 4 mg.
- Non-benzo trials: 69% levetiracetam, 44% pht, 7% lacos, 4% vpa
Response rates to anticonvulsant trials in patients with triphasic wave EEG patterns of uncertain significance

RESULTS

O’Rourke D, Chen, PM…Westover MB, and CCEMRC, Neurocrit Care 2015

• Positive response in 10/53 (19%, all immediate) benzo trials and 19/45 (42%; 7% immed, 20% delayed but definite, 16% delayed and possible) of trials with nonsedating AED
  – Overall 34% definite positive response and 11% possible
  – No difference in metab status in responders and nonresponders
  – No difference in benzo doses between responders and nonresponders
  – Complications of trial: one only: bradycardia, then PEA arrest after load of fosph; recovered.

• Suggests these trials are useful and that many patients with metabolic encephalopathy and “triphasic waves” have an “AED responsive” condition.
Results of the **TRENds** trial: A prospective, randomized, multicenter trial of lacosamide vs fosphenytoin for treatment of recurrent nonconvulsive seizures.

Aatif Husain, MD, Duke Univ, Study PI

and

the CCEMRC
• Multicenter Phase 3 RCT (ClinicalTrials.gov NCT01458522)
• Critically Ill Adults
• Frequent NCS on cEEG monitoring
• IV lacosamide vs. IV fosphenytoin
• Primary Outcomes:
  – Efficacy using cEEG
  – Secondary outcomes: adverse event profiles, length of ICU stay, outcomes
TRENds trial: Methods

• Prospective, randomized, active-control, non-inferiority, open-label study with blinded EEG review (primary outcome blinded)
• Surrogate consent
• Inclusion: nonconvulsive seizures on cEEG despite at least 2 medications.
• Intervention: IV lacosamide 400 mg bolus, then 200 bid versus IV fosphenytoin 20 mg/kg, then 2.5 mg/kg bid.
  – Rebolus of LCM 200 or fPHT 5 mg/kg was given if electrographic szs were seen between 2 and 8 hours after the bolus
TRENds trial: Methods, cont’d

- Primary endpoint: no recurrence of electrographic szs (w/ or w/out clinical correlate) for 24 hours following bolus (or re-bolus if given), via blinded central review.
TRENDS trial: Results

<table>
<thead>
<tr>
<th></th>
<th>LCM</th>
<th>fPHT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Response rate (sz freedom for 24 hrs)</td>
<td>19/30 (63.3%)</td>
<td>16/32 (50%)</td>
</tr>
</tbody>
</table>

P value for noninferiority of LCM: p=0.02
Risk ratio for response w/ LCM vs fPHT: 1.27 (90% conf int: 0.875-1.833)
## TRENdS Trial: Results, cont’d

Treatment emergent adverse effects within 24 hours of study drug administration

<table>
<thead>
<tr>
<th></th>
<th>LCM</th>
<th>fPHT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment emergent adverse effects</td>
<td>9/35 (25.7%)</td>
<td>9/37 (24.3%)</td>
</tr>
<tr>
<td>Serious adverse effects</td>
<td>5/35 (14.3%)</td>
<td>4/37 (10.8%)</td>
</tr>
<tr>
<td>Treatment discontinuation</td>
<td>2/35 (5.7%)</td>
<td>3/37 (8.1%)</td>
</tr>
<tr>
<td>Arrhythmias</td>
<td>2/35 (5.7%)</td>
<td>3/37 (8.1%)</td>
</tr>
<tr>
<td>Acute resp failure</td>
<td>1/35 (2.9%)</td>
<td>0/37</td>
</tr>
<tr>
<td>Hypotension</td>
<td>2/35 (5.7%)</td>
<td>2/37 (5.4%)</td>
</tr>
</tbody>
</table>
TRENDS Trial: Conclusion

- LCM was non-inferior compared to fPHT in controlling nonconvulsivse seizures noted on cEEG.
- Treatment emergent adverse effects were comparable with both AEDs.
- LCM can be considered an alternative to fPHT in the treatment of NCS detected on cEEG monitoring in critically ill patients.
Proposed practical working definitions of NORSE, FIRES, related syndromes, and Status Epilepticus (SE) of different severities: consensus panel

5 April 2017
Hotel Imlauer
Salzburg Austria

Sponsored by NORSE INSTITUTE
Objective

• To standardize terms, even if somewhat arbitrary or “semantic”
  • Allows multicenter studies
  • Improves communication, including via the literature
  • Gives a name to a clinical scenario/condition so that families can learn about it, and so that physicians know what to look up on the web, etc.
  • Helps with fundraising

• Identifying NORSE/FIRES early might help with treatment and identifying more specific diagnoses
The players:

- **Consensus team:**
  - 19 people from 8 countries; 7 pediatric—in red; * = attended the live consensus meeting in Salzburg (9 from 6 countries)
  - *Lawrence Hirsch, USA [chair]
  - *Nicolas Gaspard, Belgium
  - *Andreas van Baalen, Germany
  - *Rima Nabbout, France
  - *Sophie Demeret, France
  - *Tobias Loddenkemper, USA
  - *Vincent Navarro, France
  - *Nicola Specchio, Italy
  - *Eugen Trinka, Austria
  - Lieven Lagae, Belgium
  - Andrea Rossetti, Switzerland
  - Sara Hocker, USA
  - Teneille Gofton, Canada
  - Nicholas Abend, USA
  - Emily Gilmore, USA
  - Cecil Hahn, Canada
  - Houman Khosravani, Canada
  - Marissa Kellogg, USA
  - Felix Rosenow, Germany
Proposed definition of NORSE

• New-Onset Refractory Status Epilepticus: A clinical presentation, in a patient without active epilepsy, with new onset of refractory status epilepticus without a clear acute or active structural, toxic or metabolic cause.

  • Most of the common acute or active structural, toxic or metabolic etiologies can be identified in the first few hours, but it may take up to 72h to rule out acute strokes, brain masses, drug overdoses, etc.
  • Includes viral infections; autoimmune syndromes of new onset, even if clear in the first 72h, e.g. classic anti-NMDA encephalitis; and allows remote brain injuries or resolved epilepsy.
  • Requires imaging, CSF analysis, toxicology and blood tests as recommended for evaluation of status epilepticus in other guidelines (e.g., Neurocritical Care Society).
  • Subgroup: Cryptogenic after extensive workup; referred to as “cryptogenic NORSE”.
Proposed definition of FIRES

- **FIRES**: Febrile illness-related epilepsy syndrome: a subcategory of NORSE that requires a prior febrile illness with fever starting between 2 weeks and 24h prior to onset of refractory status epilepticus.
  - Note: has to be refractory SE; no age cutoff; can be infant, child or adult.
  - Note: can be with or without fever at the time of onset of SE (about 50% have fever in prior literature)
  - Excludes most or all of febrile SE as fever in febrile SE is usually acute onset (few hours or less).
SE of different severities: Definitions

• **Refractory SE (RSE):** SE persisting despite administration of at least 2 appropriate parenteral medications including a benzodiazepine.
  
  Note: no specific duration required.

• **Super-Refractory SE (SRSE):** SE persisting at least 24 hours after onset of anesthesia, either without interruption despite appropriate treatment with anesthesia; recurring while on appropriate anesthetic treatment; or recurring after withdrawal of anesthesia and requiring its re-introduction.

• **Prolonged SRSE (PSRSE):** SRSE that persists or recurs after at least 7 days of appropriate treatment, including ongoing need for anesthetics.

• Prolonged RSE (PRSE): RSE that persists or recurs after at least 7 days of appropriate treatment, but without use of anesthetics.
Conclusions, I

- Nonconvulsive seizures are common in the critically ill, including those without known brain disease
  - Esp if Coma or prior seizures (past or recent, short or long)
- Most seizures in the ICUs are nonconvulsive
  - >50% of patients with szs in the ICU have purely nonconv szs
  - A 30-60 minute EEG will pick up seizures in about half of these patients
- There is extensive evidence (though no prospective treatment trials) that seizures, including nonconvulsive ones, are harmful for the brain, esp when superimposed on acute brain injury.
  - Assoc’d w/ increased mass effect, metabolic crisis, HC atrophy, worse short and long term outcome including QOL, neurological worsening, later epilepsy
  - “dose-response” effect in recent studies looking at sz burden
Conclusions, II

- There are equivocal patterns and there is an ictal-interictal continuum in encephalopathic patients
  - 2 Hz or faster more likely to be associated with neuronal injury
- Use IV AED diagnostic trials
  - Including in patients with “triphasic waves”
- LRDA (lateralized rhythmic delta activity) has similar clinical significance as LPDs.
Conclusions III: Critical Care epilepsy/EEG is a rapidly evolving area

- Standardized nomenclature (ACNS guideline) with good interrater reliability
  - Adult/peds and neonatal versions
  - Learning about meaning of LRDA, “plus”, other modifiers, highly epileptiform bursts, etc

- Guidelines
  - Neurocrit Care Society
  - CCEMRC (>45 centers) and multicenter trials
    - E.g., TRENds: fospht vs lacos for nonconv seizures

- Regular symposia
- ICU-EEG Fellowships
Conclusions, IV

- Take advantage of video and quantitative EEG software
- Take advantage of free database software (acns.org; or S.C.O.R.E.)
- Do not keep patients on too long, over-interpret ICU EEGs, or over-treat the findings

Too Much Flomax??
Conclusions, Last ones:

• DO obtain EEGs rapidly with timely initial interpretation
  • Need fast, convenient method of hookup and interpretation at all hospitals

• Much to learn
  – Attend at least one talk on this topic every year
  – Great career opportunity
THANK YOU!
Brief Potentially Ictal Rhythmic Discharges in Critically Ill Adults

Original Investigation

Ji Yeoun Yoo, MD; Nishi Rampal, MD; Ognen A. Petroff, MD; Lawrence J. Hirsch, MD; Nicolas Gaspard, MD, PhD

B(I)RDs
Yoo J et al, JAMA Neurol 2014
B(I)RDs
Yoo J et al, JAMA Neurol 2014

- <10 seconds, rhythmic activity >4 Hz, with or without evolution
  - Usually theta (70%), sharply contoured, and most commonly 1-3 seconds in duration
- \(N=20\) patients (2% of all ICU-EEG patients)
- All had cerebral injury
- Associated with electrographic seizures
  - \(15/20\) (75%) vs \(10/40\) (25%) of matched controls (\(p<0.001\))
B(I)RDs, cont’d
Yoo J et al, JAMA Neurol 2014

- 9/15 with seizures had only nonconvulsive/subclinical seizures
- B(I)RDs preceded seizures in 14/15 cases
- B(I)RDs ceased in 12/12 cases in which seizures were controlled
B(I)RDs: 80 yo woman, left temp stroke, clinical seizure, then not fully awake after
Seizure in a patient with B(I)RDs
55% of patients with B(I)RDs also had LPDs
**B(I)RDs and outcome:**
LPDs and seizures associated with worse outcome, but not B(I)RDs.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Poor</th>
<th>Good</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>B(I)RDs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>16 (80)</td>
<td>4 (20)</td>
<td>.24</td>
</tr>
<tr>
<td>Absent</td>
<td>25 (63)</td>
<td>15 (37)</td>
<td></td>
</tr>
<tr>
<td>LPDs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>21 (84)</td>
<td>4 (16)</td>
<td>.047</td>
</tr>
<tr>
<td>Absent</td>
<td>20 (57)</td>
<td>15 (43)</td>
<td></td>
</tr>
<tr>
<td>Seizures</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>21 (84)</td>
<td>4 (16)</td>
<td>.047</td>
</tr>
<tr>
<td>Absent</td>
<td>20 (57)</td>
<td>15 (43)</td>
<td></td>
</tr>
</tbody>
</table>
Proposed definition of B(I)RDs:

- Focal or generalized rhythmic activity greater than 4 Hz (at least 6 waves at a regular rate) lasting 0.5-10 seconds, not consistent with a known normal pattern or benign variant, and that has at least one of the following features:
  - 1. evolution (definite B(I)RD)
  - 2. similar morphology and location as interictal epileptiform discharges or seizures in the same patient (definite B(I)RD)
  - 3. sharply contoured but without 1 or 2 (possible B(I)RDs)

Proposed categories of B(I)RDs:

- B(I)RDs with versus without evolution
- Definite versus possible B(I)RDs as defined above
Detecting Ischemia with EEG

• Cortical layers 3 & 5 particularly sensitive to ischemia and generate most of EEG
  Jordan 1999

• **EEG detects neuronal dysfunction at a reversible stage = window of opportunity**
  Chiappa 1993, Astrup 1981
  – EEG changes at CBF < 25-30 ml/100g/min
  – Synaptic transmission preserved down to 17-18 ml/100g/min, when EEG changes are prominent.
  – Cell death at <10-12 mg/100ml/min

• Progression of EEG changes with infarction:
  – Polymorphic delta, then loss of fast activity and sleep spindles, then attenuation
    Cohn 1948
  – RAWOD: predictive of massive infarct, high risk of malig edema
    Jordan 1999
<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>REVERSIBLE</td>
</tr>
<tr>
<td>18-25</td>
<td>SLOWING OF BACKGROUND TO 5-7HZ THETA</td>
<td>REVERSIBLE</td>
</tr>
<tr>
<td>12-18</td>
<td>SLOWING TO 1-4HZ DELTA</td>
<td>REVERSIBLE</td>
</tr>
<tr>
<td>&lt; 8-10</td>
<td>SUPRESSION OF ALL FREQUENCIES</td>
<td>NEURONAL DEATH</td>
</tr>
</tbody>
</table>
Monitoring CBF in acute stroke

• Quant EEG changes correlated with CBF and clinical exam during BP, hemodilution and triple H therapy (Wood 1984; Jordan 1991; Suzuki 1990)
Ischemia and Vasospasm after SAH

- DCI occurs in 25-40% of SAH patients
- Decreased relative alpha variability: 32 SAH patients. Vespa 1997
  - QEEG was 100% sensitive for angiogram-defined vasospasm (19/19); ppv=76%.
  - QEEG changes preceded TCD or angio detection of vasospasm by at least 2 days in 10/14 patients.

- CPMC protocol: SAH grade IV-V, monitored x 7 days, N=43
  - Post-stim alpha/delta ratio was best predictor of ischemia from vasospasm
  - Patients with ADR falling >10% below baseline in 6 consecutive recordings: sensitivity 100%, specificity 76%
  - Or a single recording >50% below baseline: sensitivity 89%, specificity 84% Claassen 2004
TCD R MCA 144 cm/sec

Decreased LOC, left arm drift, dysarthria

Angio: R MCA vasospasm, infusion of Papaverin and Verapamil

Alpha/Delta Ratio and delayed cerebral ischemia

Claassen, 2004
Rathakrishnan R et al, 2011, Neurocrit Care

- 12 SAH patients at high risk for DCI; 8 had DCI
- Composite alpha index (CAI)
  - Combined relative alpha power and variability
    - 6 hour calculation repeated every 30 minutes
    - Done after removal of artifact-laden sections of EEG
- Done simulating real time, but not used clinically
- By adding the CAI, sensitivity for clinical deterioration improved from 40 to 67%
  - Sens for improvement improved from 8 to 50%
- In 3/8 patients with DCI, CEEG change occurred >24 hrs prior to clinical change
• Alpha/delta ratio was the best single measure
• Even better when combined with alpha variability
• QEEG changes preceded clinical changes (by mean of 7 hours) and CT scan changes (by mean of 44 hours)