Beaumont | CHILDREN'S HOSPITAL
OU WB School of MEDICINE

PEDIATRIC CONTINUOUS EEG MONITORING (CEEG)

Michigan Society of Electroneurodiagnostic Technologists

9.27.13

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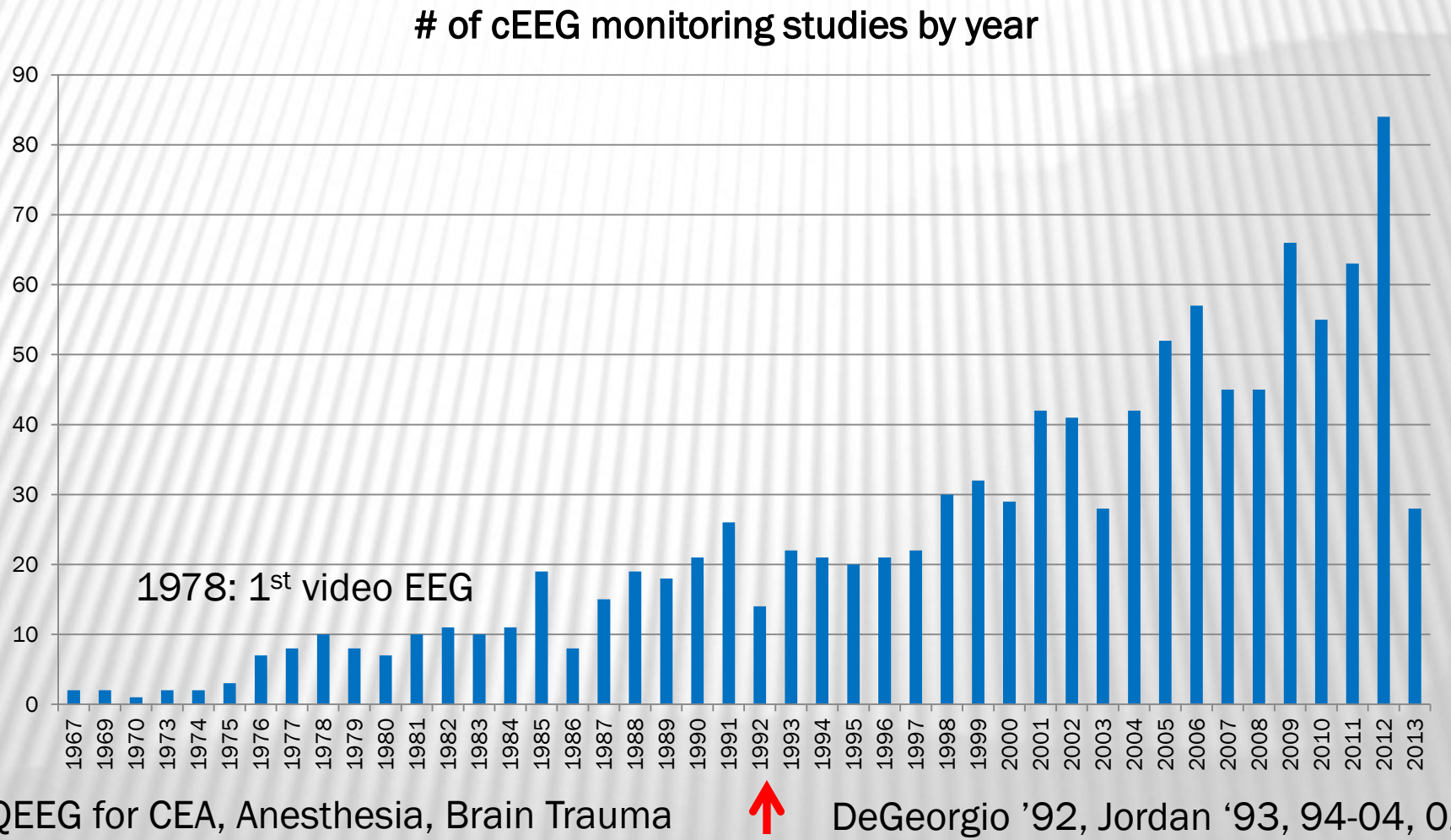
LEARNING OBJECTIVES

1. History of cEEG
2. What's what of common cEEG terminology
3. Update current practice of cEEG
   - NICU – Neonatal Intensive Care Unit
   - PICU – Pediatric Intensive Care Unit
4. Epidemiology – Subclinical Seizures / S.E.
5. Impact – Electrographic Seizures / S.E.
6. ACNS Guidelines & Critical Care EEG Terminology
   - Pediatric / Adult
   - Neonatal

CONTINUOUS EEG VIDEO MONITORING (CEEG)

- *Monitoring brain's electrical activity*
  - *Video correlation w/ clinical signs (reported sx's)*
- Indications:
  - Cerebral function monitoring (i.e. CEA, Anesthesia)
  - Event identification
  - Detect subclinical Sz *AND CLINICAL Sz*
  - EMU/Presurgical, ICU
- Where:
  - PICU, NICU, Floors

CEEG DEVELOPMENT OVER THE YEARS……



QEEG for CEA, Anesthesia, Brain Trauma

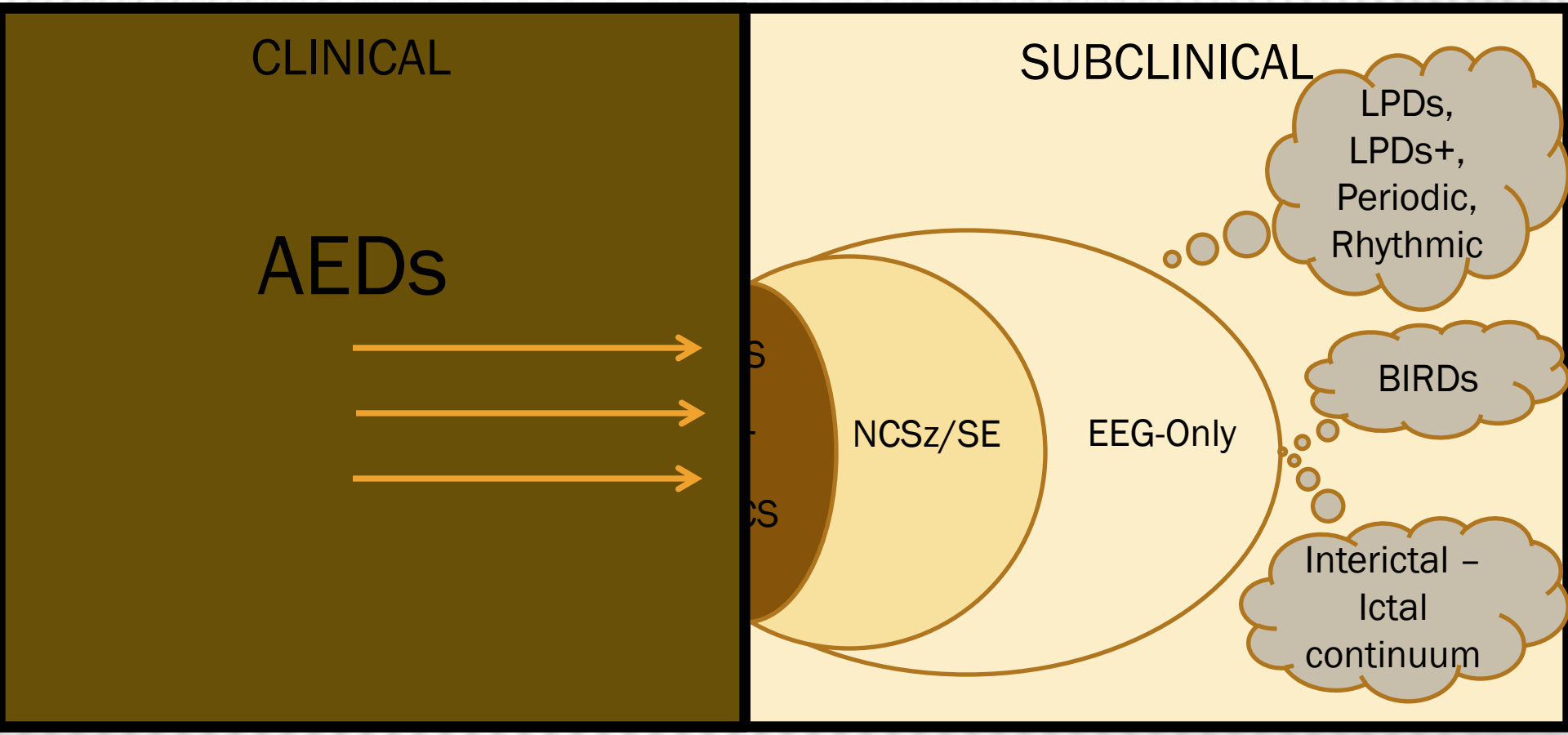
DeGeorgio ’92, Jordan ‘93, 94-04, 01-05

PubMed Search 9.25.13

WHAT'S WHAT – COMMON CEEG TERMINOLOGY

- Electroclinical Seizure = Clinical + EEG
- Clinical Seizure = Clinical +/- EEG
- Subclinical Seizure = Subtle Clinical or EEG-only
- Nonconvulsive Seizure = Subclinical Seizure
  - *Or = Subtle Clinical Seizure
- Electrographic Seizure = EEG +/- Clinical Signs
  - *Or = Subtle Clinical or Nonconvulsive Seizure
- Electrographic-Only Seizure = EEG only

DEMAND FOR CEEG MONITORING



DEMAND FOR CEEG MONITORING (CONT.)

- Nonconvulsive seizures/NCSE occur commonly
- NCSz/NCSE potentially:
  - Worsen Acute Brain Injury
  - Increased Risk for Future Neurocognitive Morbidity
  - Increased Risk of Development of Epilepsy
- NCSE ⟶ ↑ Morbidity/Mortality

SUPPLY SOURCES FOR CEEG

- ↑ ↑ Utilization of cEEG in PICU/NICU over past 5yrs
- Resource Development & EEG reimbursement Δs

- Institutional Guidelines – Standardize Monitoring
- ACNS Guidelines: Neonatal, *Pediatric, & Adult*
- Research Consortia:
  - Pediatric Critical Care EEG Group (PCCEG)
  - Critical Care EEG Monitoring Research Consortium (CCEMRC)

CURRENT PRACTICE - OVERVIEW

- Sz are common in critically ill patients
- cEEG is required to diagnose Sz
- Most Sz are identified in 1-2 days monitoring
- cEEG findings change management
- Electrographic Sz probably worsen outcome
- Lack studies that identifying/managing Electrographic Sz improves outcome
- cEEG is increasingly being utilized
- Guidelines & position statements en vogue

SZ/CEEG - NEONATAL ICU (NICU)

- Sz in 1.5 - 5.5/1000 Neonates
  - 4% <30wks & 1.5% >30wks Scher '93, Scher '93
- Clinical data do not predict Sz Murray '06
- If you have 1 Sz.....usually many
  - S.E. diagnosed ~1/3
- Unique Patients/Brains & Unique EEG
  - Seizure incidence higher than any other time in life Sheth 99

SZ/CEEG – NEONATAL ICU (NICU) (CONT.)

- Unique Sz Semiology
  - Accurate recognition of Clinical Sz is challenging Scher 02
  - Experience clinicians frequently fail to recog Clin Sz
    - Staff ID 9% of 526 Clinical Sz Murray 08
    - 78% of 177 PBE (-EEG) Murray 08
    - Video Review: 50% accuracy (Poor interater agreement) Malone '09
  - Sz frequently Subclinical Connell 89, Hellstrom-Westas 85, Nash 11, Scher 03, Clancy 06

SZ/CEEG - NEONATAL ICU (NICU) (CONT.)

Continuous EEG monitoring of neonatal seizures: diagnostic and prognostic considerations.

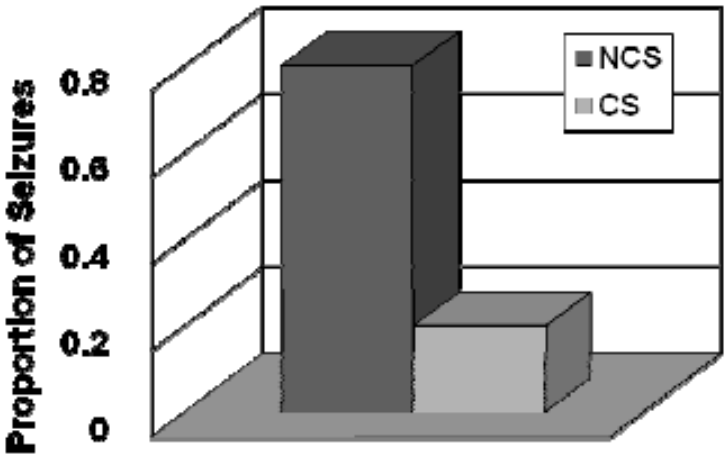
J Connell, R Oozeer, L de Vries, L M Dubowitz and V Dubowitz

Arch. Dis. Child. 1989;64;452-458
doi:10.1136/adc.64.4_Spec_No.452

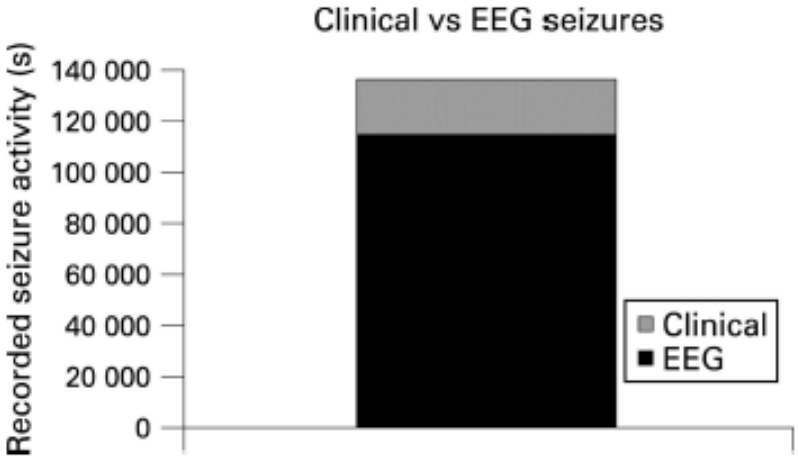
- Connell article – data
- 4 or 8 channel EEG – 275 Consecutive NICU adm
  - 25% critically ill
  - ¾ preterm infants
- 20% Sz
  - 42% Sz infants = Electrographic-only
    - 40% of E-only Sz infants were medically paralyzed
  - Additional 36% Sz infants = Electrographic onset preceded Clin Sz
    - Thus, 78% E-only portions of Sz (Would be similar to Clancy '06 #)
- 55% of Sz infants expired
  - No diff +/- Clinical Sz (Few had Clinical only Sz)

SZ/CEEG - NEONATAL ICU (NICU) (CONT.)

- Majority of seizures have no clinical correlate.



Clancy RR et al., Epilepsia, 1988.



Murray DM, et al., Arch. Dis. Child. Fetal Neo Ed. 2008.

- Nonparalyzed infants: Only 20% ESz provoke Clin Signs Clancy 06
  - >42% seen in Connell '89 – Better EEG
- Electroclinical uncoupling – Phenobarbital – 50%

Slide adapted Abend '13, PHB Data: Scher '03, Connell '89

CURRENT NICU CEEG INDICATIONS

- **HIE & THERAPEUTIC HYPOTHERMIA**
  - Nash ’11
    - N=41, 34% Sz → 10% S.E., 43% Subclinical
  - Wusthoff ’11
    - N=26, 65% Sz → 23% S.E., 47% Subclinical
- **Cardiac Surgery**
  - Peri-operative Subclinical Sz 6-20% Chock 06, Clancy 05, Helmers 97, Gaynor 05, Schmitt 05
- **ECMO**
  - Subclinical Sz 11-30% Campbell 91, Hahn 93, Horan 07

CURRENT NICU CEEG UTILIZATION

- Glass '12:
  - International Survey
  - Monitor "at risk" newborns: EEG 24%, aEEG 24%, Both 19%
    - None 34%
  - Seizure Diagnosis: Clinical 8%, EEG 58%, aEEG or EEG 38%
  - EEG Duration: <60min 31%, 24hrs 17%, Sz-free 24hrs 49%
- Boylan '10:
  - EEG Monitoring Access = 90% (EEG 27%, aEEG 22%, Both 51%)
  - Confident or Very Confident interpreting = 28%
- aEEG (Amplitude integrated EEG):
  - Sensitivity (single channel w/out raw EEG single channel for confirmation): <50%Rennie 04, Shellhaas 07
  - Addition of 2nd aEEG channel w/ ability to review raw EEG improves sensitivity to 76%, specificity 78%Shah 08
    - But Sz detection remains difficult with this tool
  - It has been shown to reduce total seizure duration in neonatesVan Rooij 10

CI

The American Clinical Neurophysiology Society's Guideline on Continuous Electroencephalography Monitoring in Neonates

Renée A. Shellhaas, Taeun Chang,† Tammy Tsuchida,† Mark S. Scher,‡ James J. Riviello,§ Nicholas S. Abend,‖ Sylvie Nguyen,¶ Courtney J. Wusthoff,# and Robert R. Clancy‖*

- Shellhaas '11:
  - Idealized Goals – NOT Mandated Standard of Care
  - Indications:
    - Differential Diagnosis – Abnormal Paroxysmal Events
    - Detection of Electrographic Sz in High Risk Populations
    - Monitoring Burst Suppression
    - Judge severity of encephalopathy
  - Procedures for monitoring
  - Duration of monitoring: 24hrs – Routine EEG little value
  - Training of caretakers
  - EEG interpretation & reporting
    - 1st hour reported asap & ≥2x in 24hrs
  - Data Retention & Storage
  - Digital trending & analyses

The American Clinical Neurophysiology Society's Guideline on Continuous Electroencephalography Monitoring in Neonates

Renée A. Shellhaas, Taeun Chang,† Tammy Tsuchida,† Mark S. Scher,‡ James J. Riviello,§ Nicholas S. Abend,‖ Sylvie Nguyen,¶ Courtney J. Wusthoff,# and Robert R. Clancy‖*

TABLE 1. Examples of Sudden, Stereotyped Clinical Events That May Raise the Suspicion for Neonatal Seizures

Focal clonic or tonic movements
Intermittent forced, conjugate, horizontal gaze deviation
Myoclonus
Generalized tonic posturing
"Brainstem release phenomena" such as oral–motor stereotypes, reciprocal swimming movements of the upper extremities or bicycling movements of the legs
Autonomic paroxysms such as unexplained apnea, pallor, flushing, tearing, and cyclic periods of tachycardia or elevated blood pressures

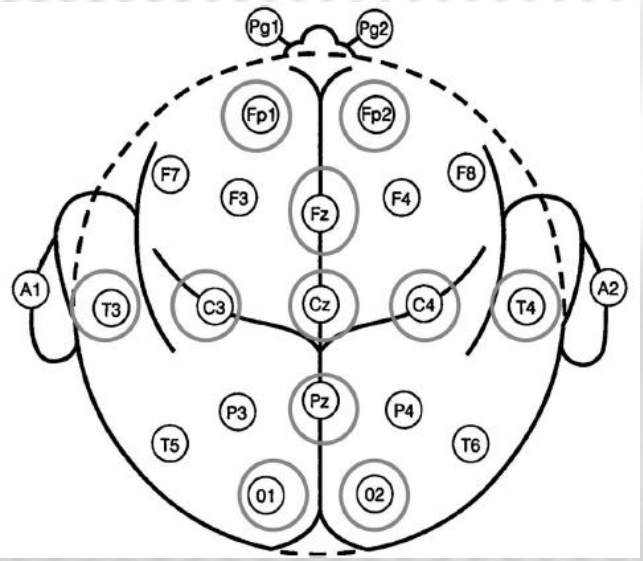


TABLE 2. Examples of High-Risk Clinical Scenarios Which May Lead to Consideration of Long-Term Neonatal EEG Monitoring

Examples of Clinical Scenarios Conferring High Risk of Neonatal Seizures
Clinical syndrome of acute neonatal encephalopathy
Neonatal depression from suspected perinatal asphyxia (chronic or acute)
After cardiopulmonary resuscitation
Cardiac or pulmonary risks for acute brain injury and clinical encephalopathy
Significant respiratory conditions such as severe persistent pulmonary hypertension
Need for ECMO
Congenital heart defects requiring early surgery using cardiopulmonary bypass
CNS infection
Laboratory confirmed meningoencephalitis
Suspected CNS infection, such as clinical evidence in setting of maternal chorioamnionitis, funisitis, group B streptococcus or HSV colonization
CNS trauma
Intracranial subarachnoid, subdural, or intraventricular bleeding
Clinical encephalopathy and suspicion for CNS injury, for example, maternal trauma, traumatic delivery, prolonged second stage of labor, or suspected nonaccidental trauma
Inborn errors of metabolism (suspected or confirmed)
Perinatal stroke (suspected or confirmed)
Sinovenous thrombosis (suspected or confirmed)
Premature infants with additional risk factors
Acute high-grade intraventricular hemorrhages
Very low birth weight with clinical concern for encephalopathy
Genetic/syndromic disease involving CNS
Cerebral dysgenesis on neuroimaging
Dysmorphic features or multiple anomalies with microcephaly

SZ/CEEG – PEDIATRIC ICU (PICU)

- PICU cEEG Experience more ~ Adult Experience than that of neonates
  - 2-4yo – Significant myelination
  - Still quite different than adults:
    - Incidence
    - Treatment
    - Significance/Outcome

1ST PAPER – CEEG PICU

Frequency and Predictors of Nonconvulsive Seizures During Continuous Electroencephalographic Monitoring in Critically Ill Children

Nathalie Jette, MD, MSc; Jan Claassen, MD; Ronald G. Emerson, MD; Lawrence J. Hirsch, MD

- 44% Sz (75% EEG-only)
- 23% S.E. (89% NCSE)

SUMMARY – ELECTROGRAPHIC SZ INCIDENCE PICU

- Prior single-studies – varying incidences of ESz:
  - 7 - 48% Abend 11, Hosain 05, Jette 06, Abend 07, Alehan 01, Tay 06, Saengpattrachai 06, Shahwan 10, Abend 09, Williams 11, Greiner 12, Kirkham 12
    - Variability:
      - Small sample size
      - Case mix variability across institutions
      - Interinstitution variabilities in cEEG indications
      - Range of studies performed over a decade – cEEG/Crit Care evolved

PCCEG Epidemiologic Study

- Retrospective
- 11 sites
- 550 subjects
- PICU (1mo-21yrs)
- Clinically indicated cEEG

Site	PCEEG Member
Boston	Tobias Loddenkemper Ivan Sanchez Fernandez
Chicago	Joshua Goldstein
DC	Jessica Carpenter
Denver	Kevin Chapman
Duke	William Gallentine
Los Angeles	Christopher Giza Jason Lerner Joyce Matsumoto
Miami	Ann Hyslop
Michigan	Daniel Arndt
Philadelphia	Nick Abend Dennis Dlugos
Phoenix	Korwyn Williams
San Francisco	Kendall Nash
Toronto	Cecil Hahn Eric Payne

Electrographic seizures in pediatric ICU patients

Cohort study of risk factors and mortality

Nicholas S. Abend, MD
Daniel H. Arndt, MD
Jessica L. Carpenter, MD
Kevin E. Chapman, MD
Karen M. Cornett, MT
William B. Gallentine, DO
Christopher C. Giza, MD
Joshua L. Goldstein, MD
Cecil D. Hahn, MD, MPH
Jason T. Lerner, MD
Tobias Loddenkemper, MD
Joyce H. Matsumoto, MD
Kristin McBain, MS
Kendall B. Nash, MD
Eric Payne, MD
Sarah M. Sánchez, BA
Iván Sánchez Fernández, MD
Justine Shults, PhD
Korwyn Williams, MD, PhD
Amy Yang, BS
Dennis J. Dlugos, MD

ABSTRACT

Objectives: We aimed to determine the incidence of electrographic seizures in children in the pediatric intensive care unit who underwent EEG monitoring, risk factors for electrographic seizures, and whether electrographic seizures were associated with increased odds of mortality.

Methods: Eleven sites in North America retrospectively reviewed a total of 550 consecutive children in pediatric intensive care units who underwent EEG monitoring. We collected data on demographics, diagnoses, clinical seizures, mental status at EEG onset, EEG background, interictal epileptiform discharges, electrographic seizures, intensive care unit length of stay, and in-hospital mortality.

Results: Electrographic seizures occurred in 162 of 550 subjects (30%), of which 61 subjects (38%) had electrographic status epilepticus. Electrographic seizures were exclusively subclinical in 59 of 162 subjects (36%). A multivariable logistic regression model showed that independent risk factors for electrographic seizures included younger age, clinical seizures prior to EEG monitoring, an abnormal initial EEG background, interictal epileptiform discharges, and a diagnosis of epilepsy. Subjects with electrographic status epilepticus had greater odds of in-hospital death, even after adjusting for EEG background and neurologic diagnosis category.

Conclusions: Electrographic seizures are common among children in the pediatric intensive care unit, particularly those with specific risk factors. Electrographic status epilepticus occurs in more than one-third of children with electrographic seizures and is associated with higher in-hospital mortality. *Neurology*® 2013;81:383–391

GLOSSARY

CEEG = continuous EEG; **CI** = confidence interval; **IQR** = interquartile range; **OR** = odds ratio; **PICU** = pediatric intensive care unit.

Several single-center studies have reported electrographic seizures in 10%–40% of children who

PCCEG EPIDEMIOLOGIC DATA ABEND '13

- Electrographic Sz – 30% (162/550)
  - Electrographic S.E. – 38%
    - Sz >30min – 46%
    - Recurrent Sz >50% of 1hr Epoch – 56%
  - Sz w/ Clinical correlate?:
    - All – Only 27%
    - Some – 34%
    - None – 36%
  - Sz risk factors: (multivariate analysis)
    - Younger Age
    - Clinical Sz prior to cEEG
    - Abnormal initial EEG background
    - IEDs
    - Epilepsy Diagnosis
  - NCSz risk factors: (reported elsewhere)
    - Younger Age Abend 11, Williams 11, Schreiber 12
    - Convulsive SE Williams 11
    - Acute Seizures McCoy 11, Greiner 12, Schreiber 12
    - Structural Brain Injury & TBI McCoy 11, Greiner 12, Williams 11
    - EEG: Lack of Reactivity Jette 06, Epileptiform D/C Williams 11, J 06, McCoy 11, Abend 09, Background Discont Abend 09

Table 1 Electrographic seizure characteristics

Electrographic seizure characteristic	n (%)
Typical seizure duration (n = 158)	
10–59 s	60 (38)
1–5 min	63 (40)
6–30 min	25 (16)
>30 min	10 (6)
Clinical correlate (n = 162)	
All (100%)	43 (27)
Most (50%–99%)	22 (14)
Some (1%–49%)	33 (20)
None (0%)	59 (35)
Unknown	5 (3)
Seizure onset localization (n = 162)	
Focal	86 (53)
Multifocal	30 (19)
Generalized	39 (24)
Unknown	7 (4)
Seizure maximal spread localization (n = 162)	
Focal-unilateral	80 (49)
Bilateral	76 (47)
Unknown	6 (4)

Table 2 Electrographic seizure occurrence by diagnosis

Diagnosis (n)[a]	Electrographic seizures present, %	Electrographic seizures absent, %
Sepsis (19)	58	42
Epilepsy (159)	48	52
Brain malformation (24)	38	62
CNS inflammation or autoimmune disorder (24)	33	67
Stroke (33)	30	70
Traumatic brain injury (61)	30	70
Metabolic (59)	29	71
CNS infection (28)	29	71
Unknown (14)	21	78
Tumor/oncologic (21)	19	81
Hypoxic-ischemic encephalopathy (73)	18	82
Pharmacologic sedation—no known neurologic problem (15)	13	87
Toxin (8)	13	87
Paralytic administration (26)	8	92

[a] Subjects could have more than one diagnosis.

PCCEG EPIDEMIOLOGIC DATA ABEND '13 (CONT.)

Diagnosis (N)	Seizure(s) 30% (162)
Sepsis (19)	58%
Epilepsy (159)	48%
Brain Malformation (24)	38%
Central Nervous System Inflammation or Autoimmune Disorder (24)	33%
Stroke (33)	30%
Traumatic Brain Injury (61)	30%
Metabolic (59)	29%
Central Nervous System Infection (28)	29%
Unknown (14)	21%
Tumor/oncologic (21)	19%
Hypoxic-Ischemic Encephalopathy (73)	18%
Pharmacologic sedation – no known neurologic problem (15)	13%
Toxin (8)	13%
Paralytic Administration (26)	8%

CURRENT PICU INDICATIONS

- ACNS Guidelines – PICU: Pending

- Follow Neurocritical Care Society Guidelines for S.E.:
  - Recent clinical Sz or S.E. w/out RTB >10min
  - Coma, including post-cardiac arrest
  - Epileptiform activity or Periodic discharges initial 30m
  - Intracranial hemorrhage including TBI, SAH, ICH
  - Suspected NCSz/NCSE in pts w/ AMS
- Post-cardiac arrest - Hypothermia
- Traumatic Brain injury
- ECMO
- Cardiac Surgery

EXAMPLE – SPECIFIC PATIENT TYPE
CEEG – ACUTE BRAIN INJURY

- 1st Report – Adult Neuro ICU: Jordan '93 & '95
  - Varied BI
  - Sz o NICU Course: 35%
    - *75% EEG-only Sz (~25%)*
- Similar reports – Adult Neuro-ICU:
  - *EEG-only Sz*: 11% Litt 94 - 55% Claassen 04
- 2 TBI-specific reports early:
  - Vespa '99 Prospective
  - Ronne-Engstrom '06 Retrospective

BREAKTHROUGH PAPER

J Neurosurg 91:750–760, 1999

Increased incidence and impact of nonconvulsive and convulsive seizures after traumatic brain injury as detected by continuous electroencephalographic monitoring

PAUL M. VESPA, M.D., MARC R. NUWER, M.D., PH.D., VALERIY NENOV, PH.D., ELISABETH KONNE-ENGSTROM, M.D., PH.D., DAVID A. HOVDA, PH.D., MARVIN BERGSNEIDER, M.D., DANIEL F. KELLY, M.D., NEIL A. MARTIN, M.D., AND DONALD P. BECKER, M.D.

Division of Neurosurgery and Department of Neurology, University of California at Los Angeles School of Medicine, Los Angeles, California; and Department of Neurosurgery, Uppsala University, Uppsala, Sweden

- Prospective
- cEEG mod-sev TBI (GCS 3-12) – 94pts
  - Standardized care protocols: ICP, CPP, Ventilation, PHT (10-20) ER + ≥7d
- EPTS: 22%
  - *EEG-only* = 52%
  - EPTSz >48hrs: 2/21
    - Clinical Literature: 56-100% PTSz ≤24hrs
- Non-Sz Group: 10% Epileptiform d/c's

FOLLOW-UP PAPER

Acta Neurol Scand 2006: 114: 47–53 DOI: 10.1111/j.1600-0404.2006.00652.x

ACTA NEUROLOGICA SCANDINAVICA

Continuous EEG monitoring in patients with traumatic brain injury reveals a high incidence of epileptiform activity

Ronne-Engstrom E, Winkler T. Continuous EEG monitoring in patients with traumatic brain injury reveals a high incidence of epileptiform activity.
Acta Neurol Scand 2006: 114: 47–53. © Blackwell Munksgaard 2006.

E. Ronne-Engstrom[1], T. Winkler[2]

Departments of [1]Neurosurgery and [2]Clinical Neurophysiology, Division of Neuroscience, University Hospital, Uppsala, Sweden

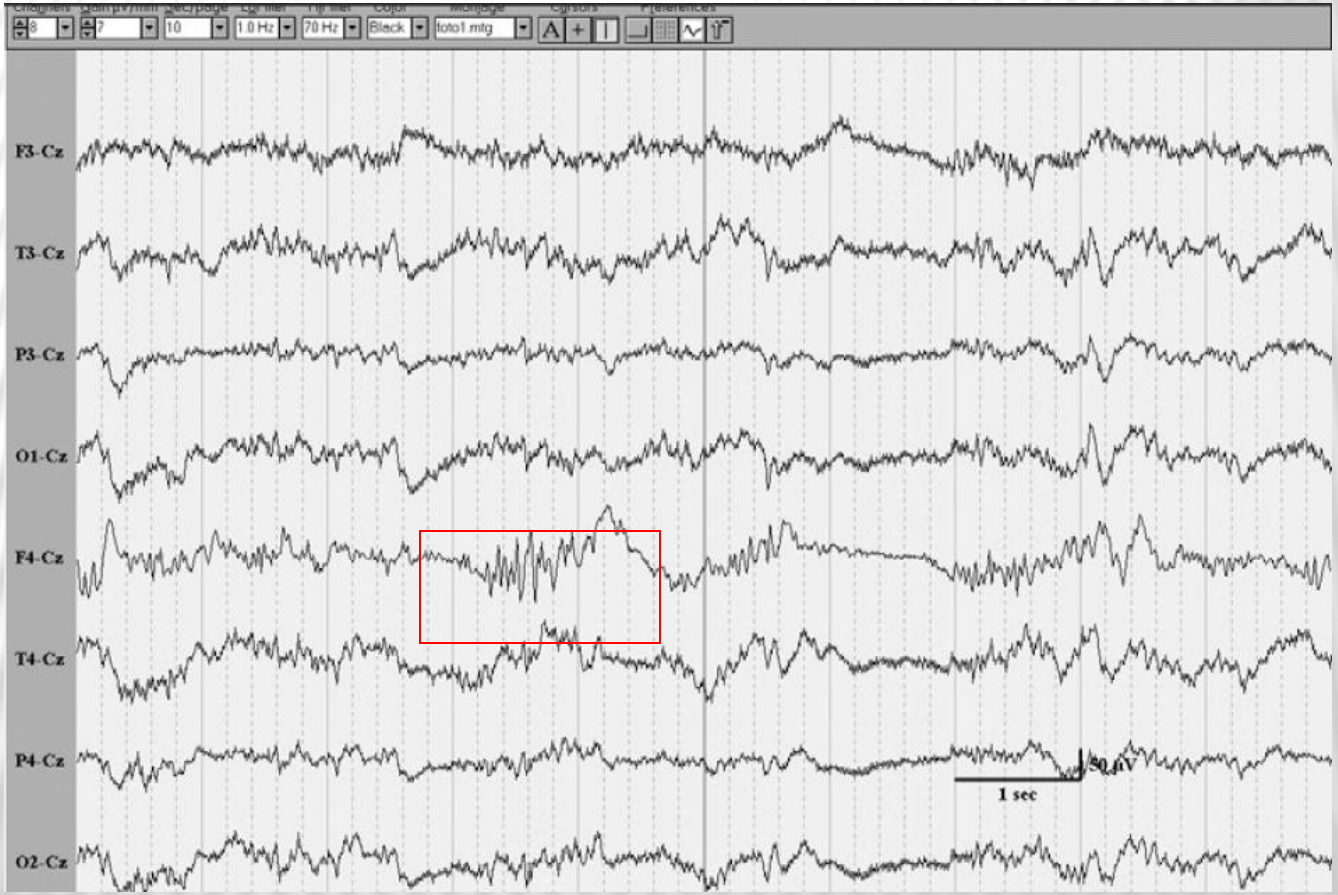
- Retrospective
- cEEG >24hrs, Standardized care protocols: ICP, CPP, Ventilation
- EPTS: 33%
  - *"Significant %" EEG-only*
- Non-Sz Group: 16% epileptiform d/c's

PRECLINICAL PTSz Detection

- Animals:
  - Prince et al. '09: (Lit Review)
    - *Electrographic only (no clinical signs)* focal Sz in TBI simulated Rats
      - Depth electrodes & video EEG
  - Pitkanen et al.

OTHER POTENTIAL DX BENEFIT?

- Preepileptic signatures? Damaged brain - Image Neg TBI?
  - Ronne-Engstrom '06: Focal high frequency d/c's + slow wave
    - 66% (12/18) FHFDs → Epileptiform activity
    - 44% (8/18) had Sz



1. Vespa '02: Impaired α variability - Px value - GCS 3-8
2. Vespa '97 & Claassen: α/Δ ratio - Impending vasospasm - SAH

ELECTROGRAPHIC (SUBCLIN/NONCONV) PTSZ ↑ *ICP & METABOLIC STRESS*

Nonconvulsive electrographic seizures after traumatic brain injury result in a delayed, prolonged increase in intracranial pressure and metabolic crisis

Crit Care Med 2007 Vol. 35, No. 12

Paul M. Vespa, MD, FCCM; Chad Miller, MD; David McArthur, PhD; Mathew Eliseo, BS; Maria Etchepare, BSN, RN; Daniel Hirt, BS; Thomas C. Glenn, PhD; Neil Martin, MD; David Hovda, PhD

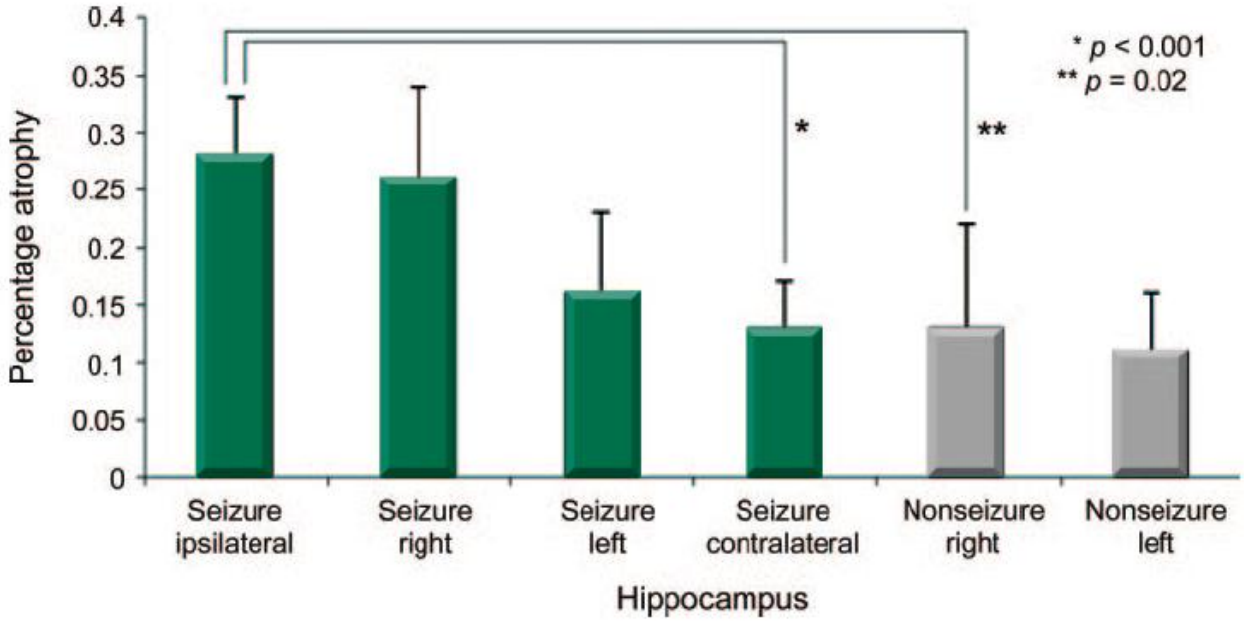
- Results:
  - +EPTSz group: Overall ↑ ICP & Lactate
    - ↑ >100hrs
- Conclusions:
  1. PTSz *NOT just benign epiphenomina!*
     - Direct evidence: ↑ ICP, Δ IC Hemodynamics, & potentiate metab stress
  2. PTSz *Therapeutic target for TBI patients*
  3. Consider cEEG in TBI pts w/ ICP refractory to conventional measures
     - ↑ICP >96hrs post-injury
     - Detecting & Rx Sz (E/C) may improve ICP control

Nonconvulsive seizures after traumatic brain injury are associated with hippocampal atrophy

P.M. Vespa, D.L. McArthur, Y. Xu, M. Eliseo, M. Etchepare, I. Dinov, J. Alger, T.P. Glenn and D. Hovda

Neurology 2010;75;792-798

Figure 2 Long-term brain atrophy in hippocampal regions are shown for the seizure and nonseizure groups

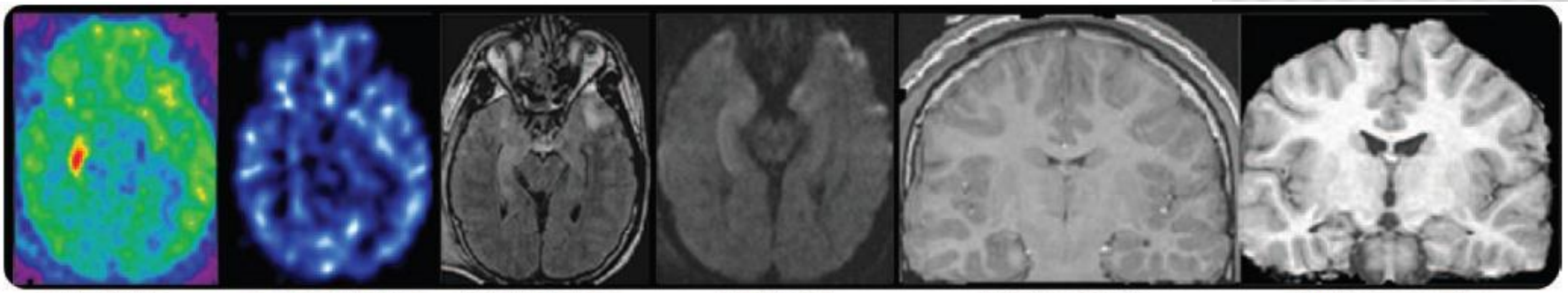


Mod-Sev TBI w/ cEEG
N=140

+ acute & chronic MRI
N=29

6/29 had Sz & were compared w/
10 controls w/o Sz

Slide courtesy of Chris Giza, MD



Subclinical early posttraumatic seizures detected by continuous EEG monitoring in a consecutive pediatric cohort

*[1]Daniel H. Arndt, †‡[1]Jason T. Lerner, †‡Joyce H. Matsumoto, §Andranik Madikians, †‡Sue Yudovin, ‡¶Hannah Valino, ‡¶David L. McArthur, †‡Joyce Y. Wu, ‡¶Michelle Leung, ‡¶Farzad Buxey, †Conrad Szeliga, #Michele Van Hirtum-Das, †‡Raman Sankar, **††Amy Brooks-Kayal, and †‡¶‡‡Christopher C. Giza

Epilepsia, **(*):1–9, 2013
doi: 10.1111/epi.12369

Summary

Purpose: Traumatic brain injury (TBI) is an important cause of morbidity and mortality in children, and early posttraumatic seizures (EPTS) are a contributing factor to ongoing acute damage. Continuous video-EEG monitoring (cEEG) was utilized to assess the burden of clinical and electrographic EPTS.

Methods: Eighty-seven consecutive, unselected (mild – severe), acute TBI patients requiring pediatric intensive care unit (PICU) admission at two academic centers were monitored prospectively with cEEG per established clinical TBI protocols. Clinical and subclinical seizures and status epilepticus (SE, clinical and subclinical) were assessed for their relation to clinical risk factors and short-term outcome measures.

Key Findings: Of all patients, 42.5% (37/87) had seizures. Younger age ($p = 0.002$) and injury mechanism (abusive head trauma – AHT, $p < 0.001$) were significant risk factors. Subclinical seizures occurred in 16.1% (14/87), while 6.9% (6/87) had only subclinical seizures. Risk factors for subclinical seizures included younger age ($p < 0.001$), AHT ($p < 0.001$), and intraaxial bleed ($p < 0.001$). SE occurred in 18.4% (16/87) with risk factors including younger age ($p < 0.001$), AHT ($p < 0.001$), and intraaxial bleed ($p = 0.002$). Subclinical SE was detected in 13.8% (12/87) with significant risk factors including younger age ($p < 0.001$), AHT ($p = 0.001$), and intraaxial bleed ($p = 0.004$). Subclinical seizures were associated with lower discharge King's Outcome Scale for Childhood Head Injury (KOSCHI) score ($p = 0.002$). SE and subclinical SE were associated with increased hospital length of stay ($p = 0.017$ and $p = 0.041$, respectively) and lower hospital discharge KOSCHI ($p = 0.007$ and $p = 0.040$, respectively).

Significance: cEEG monitoring significantly improves detection of seizures/SE and is the only way to detect subclinical seizures/SE. cEEG may be indicated after pediatric TBI, particularly in younger children, AHT cases, and those with intraaxial blood on computerized tomography (CT).

KEY WORDS: Clinical neurophysiology, Children, Epilepsy, ICU.

- 43% Sz rate
  - RF: Younger age, AHT
- 16% Subclinical Sz (6.9% only Subclinical Sz)
  - RF: Younger age, AHT, & Intraaxial bleed
- 18.4% S.E.
  - RF: Younger age, AHT, & Intraaxial bleed
- 13.8% Subclinical S.E.
  - RF: Younger age, AHT, & Intraxial bleed

- Subclinical Sz:
  - Lower Hospital D/c KOSCHI score
- S.E. & Subclinical S.E.
  - Increased Hospital LOS
  - Lower Hospital D/c KOSCHI score

Continuous EEG Monitoring for the Detection of Seizures in Traumatic Brain Injury, Infarction, and Intracerebral Hemorrhage: "To Detect and Protect"

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- Acute Symptomatic Sz after brain injury ARE NOT BENIGN
  - *Vulnerable State*
- HOWEVER, No clinical class I/II trials:
  - Sz provoked injury affects outcome
  - Absolutely Chg Mgmt for these Sz
- HOWEVER, Substantial evidence mounting
  - Acutely injured brains
  - *EEG-only Sz*

CURRENT PICU CEEG UTILIZATION

- Sanchez et al. – PCCEEG ‘ 13:
  - Surveyed 61 institutions - Retrospective
  - 47/50 US centers & 11/11 Canadian
  - 31 questions (5-10 min)
  - Significant increase (~30%) over 1 year period
  - US – median 10 pts/month
  - Technologists: Available 24/7 87% (often call-back)
    - Screen EEG: 50%
  - Most institutions utilize EEG screening by physicians & Techs 2-3x/day
  - 60% have formal qualifications to interpret EEG
  - 31% have clinical pathways addressing cEEG use

cEEG Indication		%
Event Characterization (movement, Δvital signs)		**100%**
Altered Mental Status	After seizure or status epilepticus	**96%**
	With acute primary neurologic disorder	**89%**
	Unknown etiology	**89%**
Specific Conditions	Resuscitation from cardiac arrest	68%
	Traumatic Brain Injury	60%
	ECMO	36%

CURRENT PICU CEEG UTILIZATION(CONT.)

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FULL-LENGTH ORIGINAL RESEARCH

Electroencephalography monitoring in critically ill children: Current practice and implications for future study design

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Summary

Purpose: Survey data indicate that continuous electroencephalography (EEG) (CEEG) monitoring is used with increasing frequency to identify electrographic seizures in critically ill children, but studies of current CEEG practice have not been conducted. We aimed to describe the clinical utilization of CEEG in critically ill children at tertiary care hospitals with a particular focus on variables essential for designing feasible prospective multicenter studies evaluating the impact of electrographic seizures on outcome.

Methods: Eleven North American centers retrospectively enrolled 550 consecutive critically ill children who underwent CEEG. We collected data regarding subject characteristics, CEEG indications, and CEEG findings.

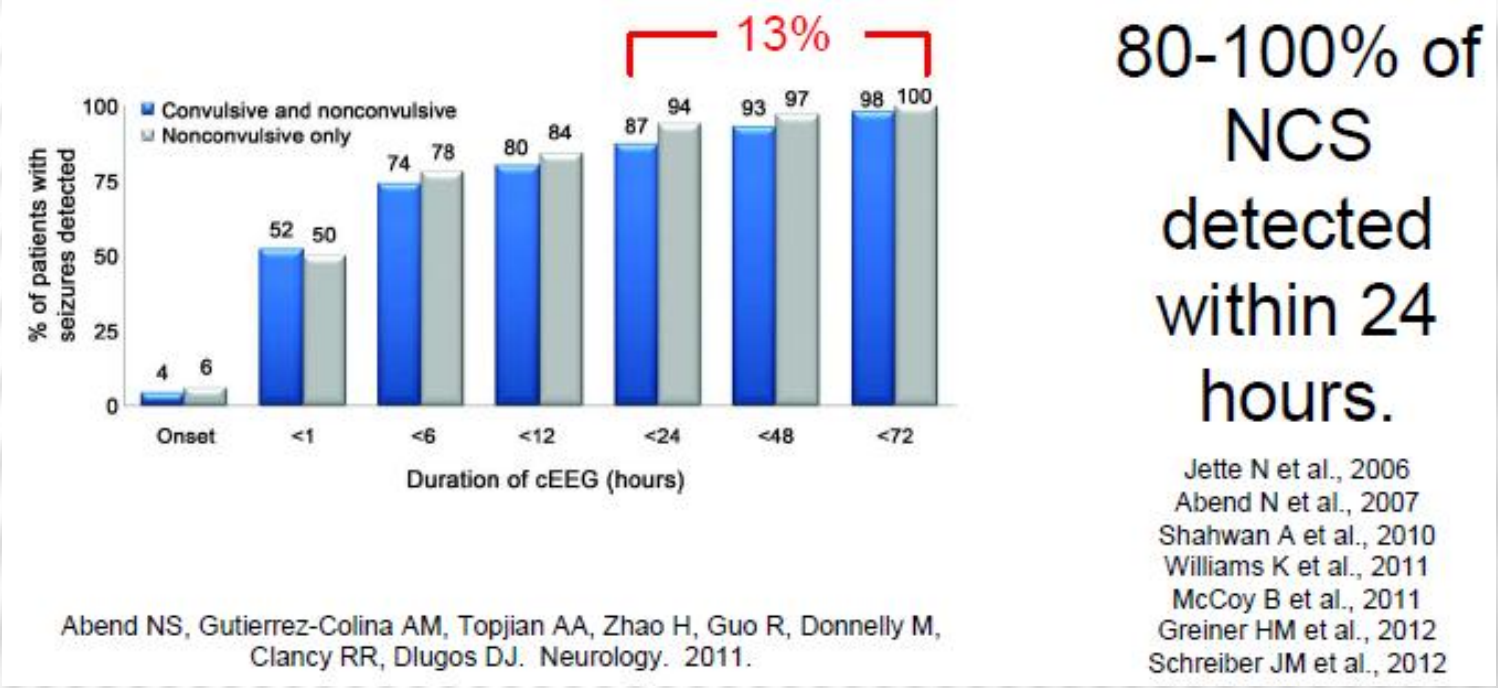
Key Findings: CEEG indications were encephalopathy with possible seizures in 67% of subjects, event characterization in 38% of subjects, and management of refractory status epilepticus in 11% of subjects. CEEG was initiated outside routine work hours in 47% of subjects. CEEG duration was <12 h in 16%, 12–24 h in 34%, and >24 h in 48%. Substantial variability existed among sites in CEEG indications and neurologic diagnoses, yet within each acute neurologic diagnosis category a similar proportion of subjects at each site had electrographic seizures. Electrographic seizure characteristics including distribution and duration varied across sites and neurologic diagnoses.

Significance: These data provide a systematic assessment of recent CEEG use in critically ill children and indicate variability in practice. The results suggest that multicenter studies are feasible if CEEG monitoring pathways can be standardized. However, the data also indicate that electrographic seizure variability must be considered when designing studies that address the impact of electrographic seizures on outcome.

KEY WORDS: EEG monitoring, Seizure, Status epilepticus, Pediatric, Nonconvulsive seizure.

HOW LONG DO WE MONITOR PATIENTS??

- Most patients will have Sz 1-2 days monitoring



- ACNS Neonate Guidelines:
  - High Risk Neonates – Conventional EEG x 24hrs
  - If Sz detected – EEG monitoring >24hrs Sz-free

HOW LONG DO WE MONITOR PATIENTS?? (CONT.)

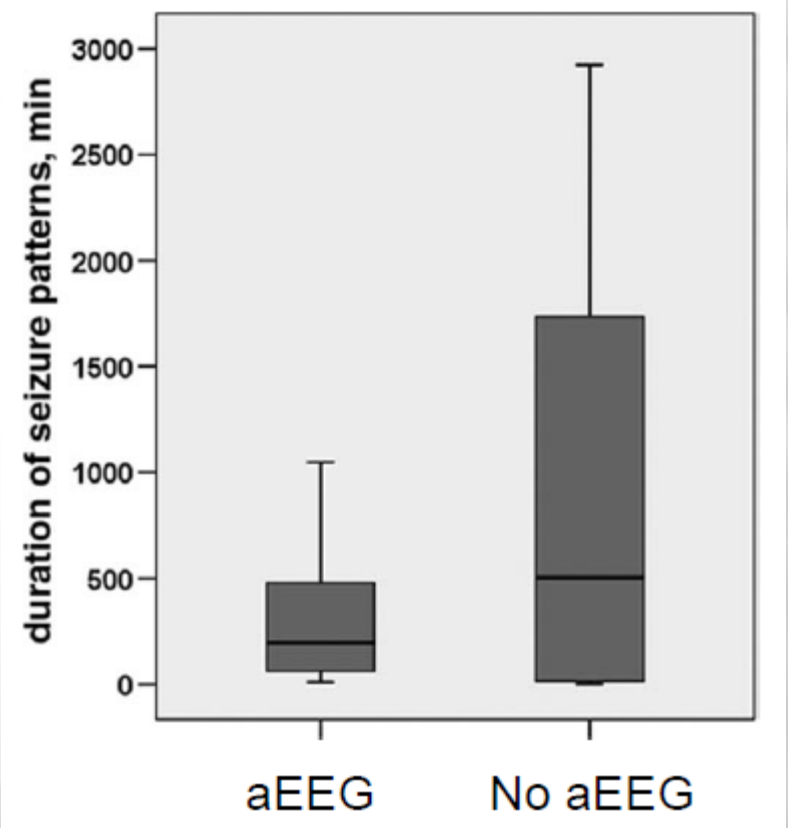
- Abend '10: Survey – cEEG Duration if No Sz
  - 24hrs if:
    - Comatose: 47%
    - Obtunded/Lethargic: 48%
    - Periodic EDs: 40%
- Specific Patients:
  - NICU Cooling/Hypothermia/HIE: 4 days (3 cool, 1 warm)
    - Maximum seizure burden 22hrs Lynch 12
    - Sz occur any day Wusthoff 11
    - S.E. tends to occur days 1-2 Wusthoff 11
  - Neurocritical Care Society Guideline: 48hrs if comatose

CEEG IMPACT ON MANAGEMENT/OUTCOME

- Little data
- Mostly Sz impacts/does not impact outcome
- Few electrographic Sz occurrence papers show it impacts outcome

CEEG IMPACT ON MANAGEMENT/OUTCOME NEONATAL ICU (NICU)

- Van Rooj '10:
  - 33 HIE Neonates
  - aEEG = ↓ Sz Burden



CEEG IMPACT ON MANAGEMENT/OUTCOME NEONATAL ICU (NICU) (CONT.)

- Outcome predictors
  - Interictal EEG (serial)[Clancy/Legido 87, Holmes 93, Monod 72, Watanabe 99, Mariani 08]
- Sz predictors – High Risk Neonates (i.e. HIE)
  - Significantly AbNL Background EEG + Sz = 81%
  - NL Background EEG + Sz = 4%[Laroia 98]

CEEG IMPROVES CLINICAL DECISION MAKING ADULT/PEDS DATA

- Jordan ’93 & ’95:
  - 1st report in NeuroICU:
    - TBI, Stroke, Coma, etc.
    - Decisions: (1) Start/Δ AED
      (2) Get neuroimaging: CT/MRI
      (3) Adjust CPP or MAP
  - cEEG decisive in 51% pts
  - Significant contribution in additional 31%
    → cEEG detected subclinical pathophysiology that could be treated in 82%
- Vespa ‘99b: Goal directed Sz Rx improved outcome (All Neuro-ICU)
  - NO additional cost (↓Hosp cost), ↓LOS, ↑GOS, Guides care >90%
- Abend ‘11: cEEG led to Mgmt Chgs in 60%
  - AED Chg = 47%
  - Paroxysmal Event Not Sz = 21%
  - Urgent Neuroimaging = 3%
- Kilbride ‘09: cEEG led to AED Px Chg in 52%

EVIDENCE MOUNTING THAT ESZ ARE NOT GOOD

- ADULT:

1. Vespa '99a: Sz did NOT affect outcome: (1) LOS (2) 1 mo GOS (TBI specific)
   - 1 month GOS? ≤36% improvement in 6-12 month GOS Corral '07
   - BUT, +PTSE = death (vs isolated Sz → no Δ mortality rate)
   - AND f/u 315 pts: 27% had PTSz → Factor ↑ mortality Shields/Vespa 04
2. Vespa '07: EEG-only Sz ↑ICP & metabolic stress → ↑Morbidity
3. Hirsch '08: "EEG-only Sz can hurt you"
4. Vespa '10: Focal MRI ipsilateral Hippocampal Atrophy with EEG-only Sz

- NEONATAL: Kwon 11, Glass 09, Gluckman 05, Van Rooj 07, Glass 11, McBride 00, Painter 12

- PEDIATRIC: Arndt 13, Greiner 12, Schreiber 12, Gwer 12, Kirkham 12, Topjian 12

- *Still waiting for evidence that treating ESz improves outcome*

Nonconvulsive Seizures in Traumatic Brain Injury: What You Don't See Can Hurt You

Epilepsy Currents, Vol. 8, No. 4 (July/August) 2008 pp. 97–99
Wiley Periodicals, Inc.

by Lawrence J. Hirsch, MD

Nonconvulsive Electrographic Seizures after Traumatic Brain Injury Result in a Delayed, Prolonged Increase in Intracranial Pressure and Metabolic Crisis. Vespa PM, Miller C, McArthur D, Eliseo M, Etchepare M, Hirt D, Glenn TC, Martin N, Hovda D. *Crit Care Med* 2007; [Epub ahead of print]. OBJECTIVE: To determine whether nonconvulsive electrographic post-traumatic seizures result in increases in intracranial pressure and microdialysis lactate/pyruvate ratio. DESIGN: Prospective monitoring with retrospective data analysis. SETTING: Single center academic neurologic intensive care unit. PATIENTS: Twenty moderate to severe traumatic brain injury patients (Glasgow Coma Score 3–13). MEASUREMENTS AND MAIN RESULTS: Continuous electroencephalography and cerebral microdialysis were performed for 7 days after injury. Ten patients had seizures and were compared with a matched cohort of traumatic brain injury patients without seizures. The seizures were repetitive and constituted status epilepticus in seven of ten patients. Using a within-subject design, post-traumatic seizures resulted in episodic increases in intracranial pressure (22.4 ± 7 vs. 12.8 ± 4.3 mm Hg; $p < .001$) and an episodic increase in lactate/pyruvate ratio (49.4 ± 16 vs. 23.8 ± 7.6; $p < .001$) in the seizure group. Using a between-subjects comparison, the seizure group demonstrated a higher mean intracranial pressure (17.6 ± 6.5 vs. 12.2 ± 4.2 mm Hg; $p < .001$), a higher mean lactate/pyruvate ratio (38.6 ± 18 vs. 27 ± 9; $p < .001$) compared with nonseizure patients. The intracranial pressure and lactate/pyruvate ratio remained elevated beyond postinjury hour 100 in the seizure group but not the nonseizure group ($p < .02$). CONCLUSION: Post-traumatic seizures result in episodic as well as long-lasting increases in intracranial pressure and microdialysis lactate/pyruvate ratio. These data suggest that post-traumatic seizures represent a therapeutic target for patients with traumatic brain injury.

- Cited prior evidence NCSz are harmful:
  - NCSz or Periodic d/c's → Independ predictors worse outcome in multiple populations
  - Epilepsy (w/out TBI) + Prolonged NCSz → Permanent neurologic injury, albeit rarely
  - NSE (neuronal injury) ↑ p NCSE (even w/out brain injury)
  - Pericontusional elect d/c's → 2° brain injury
  - Preclinical rat MCA occlusion stroke → NCSz → ↑ infarct & mortality
  - Preclinical rat pilocarpine-induced NCSE → Long-term motor & behav deficits
  - Hemorrhagic stroke + NCSz → ↑ ML shift (28% incidence)
- Mitchell '02: Pediatric SE paper cited similar reasons to argue for treating NCSE
- NCSE: Delayed Dx & Duration - Independent predictors of worse outcome Shneker 03
  - Duration: <10hrs (10% death) >20hrs (85%)
  - Delay in Dx: <30min (36% death) >24hrs (75%)
  - Etiology: Epilepsy related (3%) & Cryptogenic (18%) Acute Symp (27% death)
- In contrast, Aggressive Rx often required in critically ill to stop NCSz
  - Potentially harmful → Ongoing controversy → "Rx or No Rx?"

NEUROCRITICAL CARE SOCIETY GUIDELINE FOR STATUS EPILEPTICUS BROPHY 12

- S.E.: ≥5min (1) continuous clinical and/or electrographic Sz activity (2) recurrent Sz activity w/out recovery (baseline) between Sz
- S.E. Treatment: Should occur rapidly & continue sequentially until electrographic Sz are halted
- cEEG is usually required for treatment of S.E.
- cEEG should be initiated <1hr S.E. onset:
  - If ongoing Sz suspected
- Duration of cEEG monitoring: 48hrs in comatose

PHENYTOIN VS LACOSAMIDE - NCSZ

Epilepsia, 54(Suppl. 6):84–88, 2013
doi: 10.1111/epi.12287

STATUS EPILEPTICUS 2013

Treatment of Recurrent Electrographic Nonconvulsive Seizures (TRENdS) Study

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Summary

Nonconvulsive seizures (NCS) and nonconvulsive status epilepticus (NCSE) are electrographic seizures (ESz) that are not associated with overt clinical seizure activity. NCS are distinct ESz, whereas NCSE has ongoing, continuous electrographic seizure activity. Both are common in critically ill patients admitted to hospital intensive care units (ICUs), and studies have shown that about 20% of ICU patients undergoing continuous electroencephalography (cEEG) monitoring will have NCS/NCSE. Although the treatment for convulsive SE is well established, there is no clear consensus for the treatment of NCS/NCSE. Antiepileptic drugs (AEDs), such as phenytoin (PHT) and fosphenytoin (fPHT), used in convulsive SE are also used to treat NCS/NCSE despite lack of data for their appropriateness for these conditions. Recent studies have shown that very aggressive treatment of NCSs/NCSE can lead to worse outcomes because the AEDs used can have significant adverse effects. Recently, several intravenous (IV) AEDs have become available for substitution therapy when their oral use is not possible. There are retrospective case reports and case series that suggest that these AEDs may be beneficial for treatment of NCS/NCSE. The Treatment of Recurrent Electrographic Nonconvulsive Seizures (TRENdS) Study will compare the efficacy and tolerability of fPHT and lacosamide in patients having NCS as noted by cEEG monitoring. The study is currently open to recruitment and has 13 sites in the United States. A total of 200 subjects will be randomized, 100 to each treatment arm.

KEY WORDS: Nonconvulsive seizures, Electrographic seizures, Continuous EEG monitoring, Fosphenytoin, Lacosamide.

ACNS GUIDELINES

- Critical Care EEG Terminology
  - Adult: J Clin Neurophys Volume 30, Number 1, 2013
  - Neonate: Volume 30, Number 2, 2013
- cEEG Monitoring Guidelines
  - Neonate
  - Pending: Children & Adult

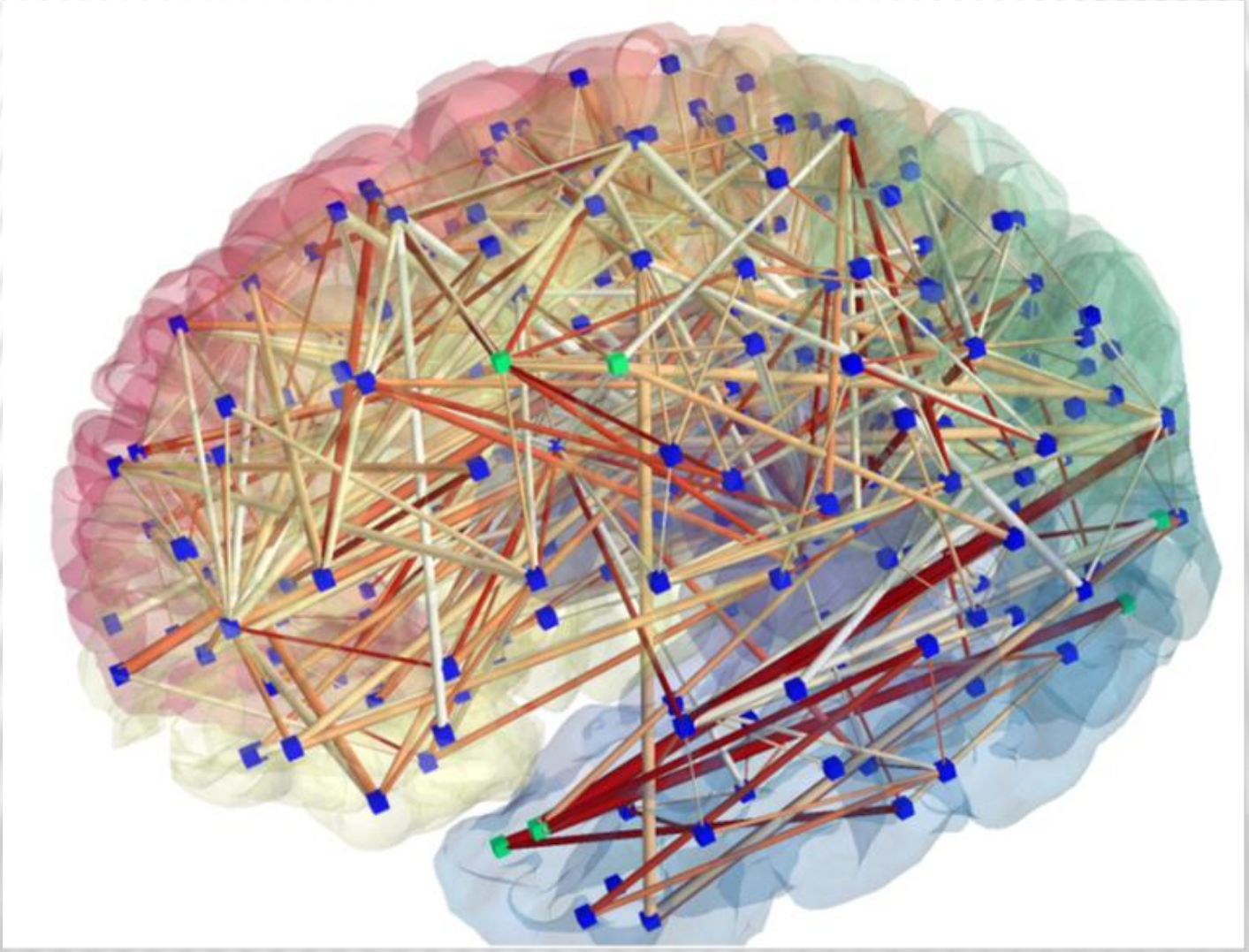
*Update cEEG monitoring PICU / NICU

J Clin Neurophys Volume 30, Number 2, 2013

ADDITIONAL ICU EEG ISSUES

- Ictal-Interictal Continuum
  - Nomenclature, Significance
- EEG background / prognosis
  - Guide for real-time Mgmt
- Quantitative EEG / Persyst / Trending
  - Efficient Sz identification
  - Identification of interval interictal background chgs

THANK YOU!



Electroencephalography and clinical Neurophysiology, 79 (1991) 94–100
 0013-4649/91/$03.50
ADONIS 001346499100117X

EEG 90513

Long-term EEG monitoring in the early premature: developmental and chronobiological aspects

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(Accepted for publication: 16 November 1990)

Summary Long-term cassette EEG monitoring in the neonatal intensive care unit has established prognostic criteria regarding the developmental outcome by quantifying seizure activity. The clinical significance of the organization of continuous and discontinuous EEG patterns in the early premature is still an open question. This report presents quantified EEG data from repeated 24 h records during the first week of life in premature infants (conceptional age < 32 weeks) with and without ultrasound evidence of intracerebral hemorrhage. The repartition and evolution of EEG background activity is not a reliable parameter regarding pathology. The continuity index is rather a maturational variable and its ultradian fluctuation is an early expression of the "basic rest activity cycle" (BRAC) rhythm.

Key words: Long-term EEG monitoring; Continuous/discontinuous EEG patterns; Early prematures; Cerebral maturation; BRAC rhythm