CEREBRAL OXIMETRY IN INFANTS WITH HIE

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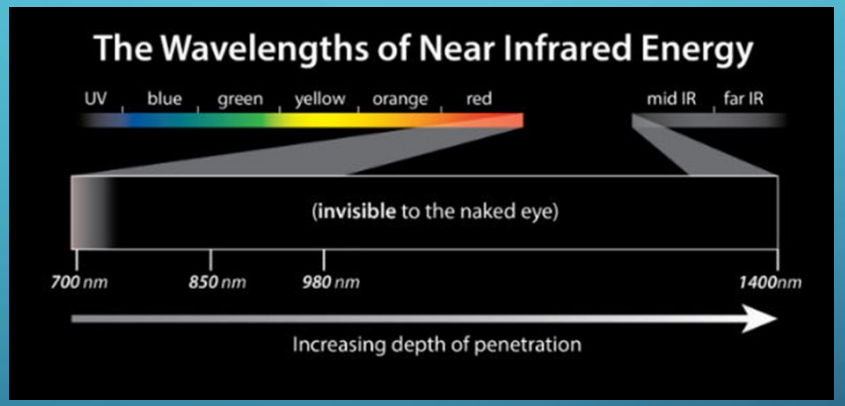
OBJECTIVES

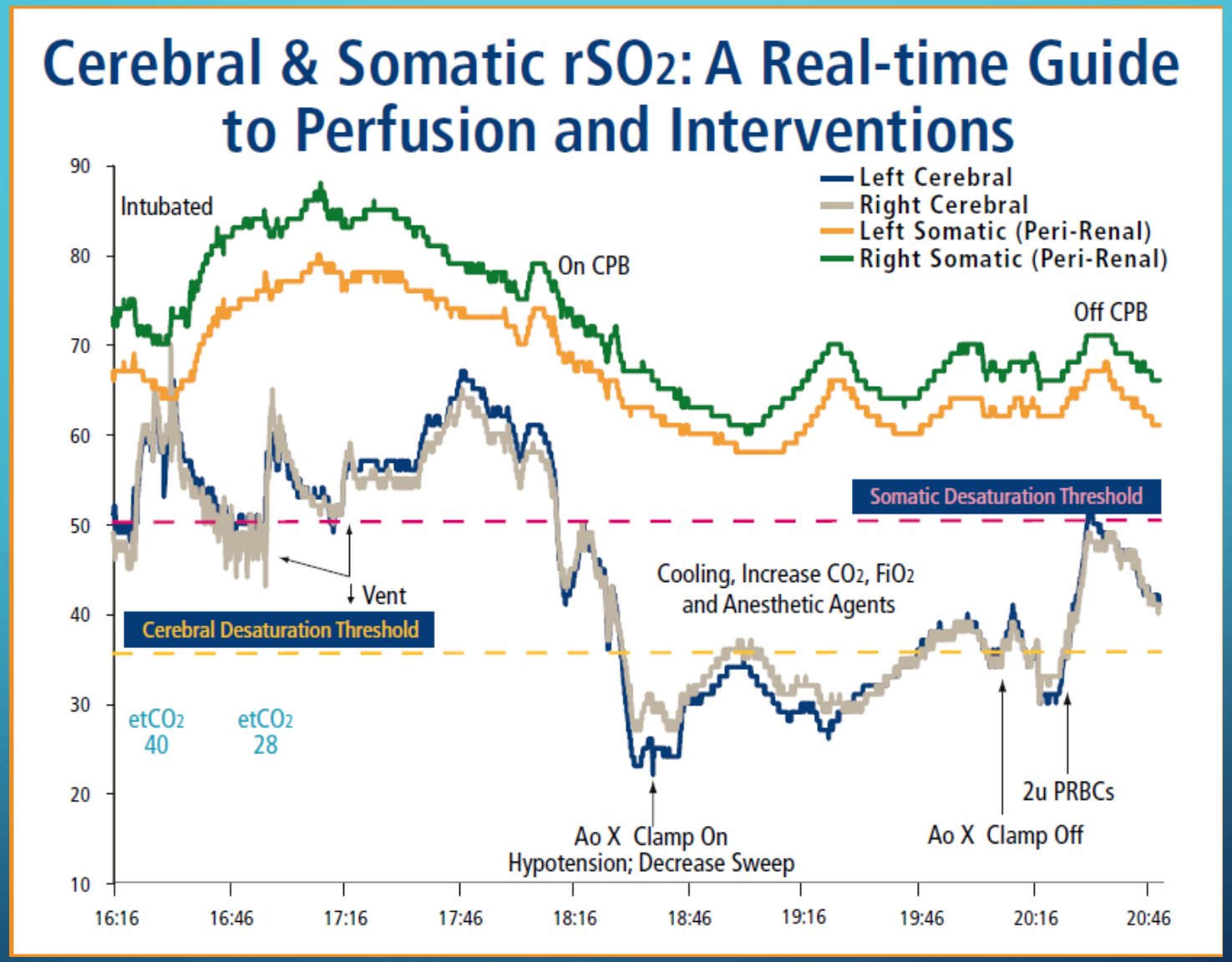
1. Understand how cerebral oximetry works
2. Understand how cerebral oximetry may guide intervention
3. Learn that cerebral oximetry trends may have prognostic value

WHAT IS CEREBRAL OXIMETRY?

- Cerebral oximetry is a non-invasive tool based on near-infrared spectroscopy (NIRS) that can monitor the regional hemoglobin oxygen saturation (rScO2) of the frontal cortex.



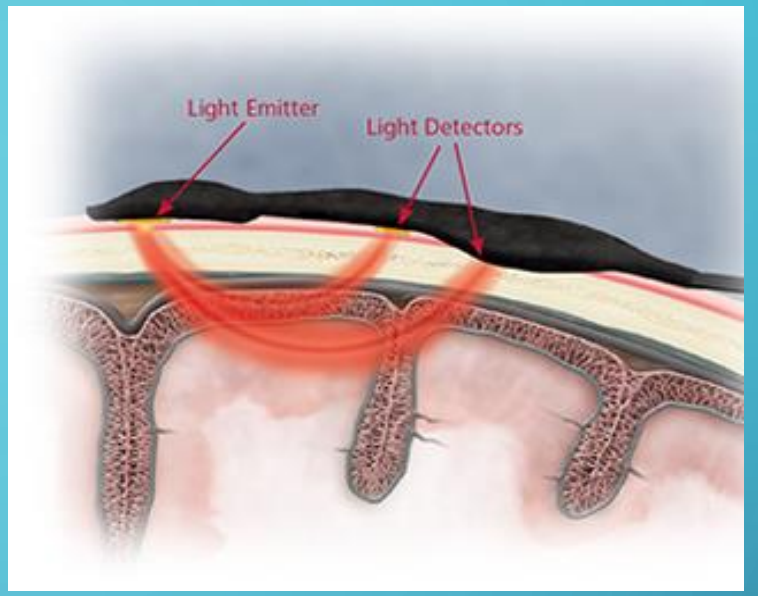
- It provides continuous information about brain oxygenation and it provides a measurement of the brain as a sentinel organ indexing overall organ perfusion and injury.



Left and right peri-renal NIRS

Left and right cerebral NIRS

HOW DOES IT WORK?



- An emitter sends light of the near-infrared spectrum (wavelength of 700-1100nm) through cerebral tissue in a semi-curved shape to a detector, approximately 2–3 cm in depth.
- Differences in NIR light absorption are detected by the sensor and the ratio between O_2Hb and HHb is expressed as the $rScO_2$ or tissue oxygenation index (TOI), depending on the manufacturer of the NIRS device.

CEREBRAL OXIMETRY VALUES

- Felt to be consistent with a mixed venous measurement.
- Good correlation with jugular venous oxygen saturation.
- rSCO2/ systemic arterial oxygen saturation = cerebral oxygen utilization
- $rScO_2$ is between approximately 40 and 56% directly after birth
  - increases up to 78% in the first 2 days after birth
  - stabilizes during 3–6 weeks after birth with values between 55 and 85%
- Trend is more useful than single absolute value

MODIFIERS OF CEREBRAL OXYGENATION

- Ventilation impacts cerebral circulation
  - High mean airway pressures can reduce oxygen saturation
- pCO2: hypercapnia induces cerebral vasodilation and hypocapnia induces vasoconstriction
  - Increased pCO2 increases oxygen saturation and decreases oxygen extraction
- SGA infants: much higher $rScO_2$ in first few postnatal days
  - Likely a function of intra-uterine preservation of brain blood flow

MODIFIERS OF CEREBRAL OXYGENATION

- Hypotension: true hypotension will affect $rScO_2$
  - Consider permissive hypotension unless cerebral saturation affected
- Significant PDA: shunting away from the brain can have a profound effect on rScO2
  - rScO2 rarely used as a marker of PDA significance
- Blood transfusions: anemic infants who undergo transfusion have resultant increase in rScO2
- Dysfunction cerebral autoregulation: RDS, surgery, high concentrations of pressors
  - Impaired autoregulation linked to poor ND outcomes.

CEREBRAL OXIMETRY IN HIE

- Infants with HIE have increased rScO2 and decreased cFOE during days following asphyxia.
  - Cerebral hyperoxygenation likely a result of decreased metabolism leading to low oxygen utilization, impaired cerebral autoregulation despite hyperperfusion after injury.
  - Higher rScO2 have correlated with adverse outcomes at 2 years (both with and without TH)

CAN CEREBRAL OXIMETRY BE OF PROGNOSTIC VALUE?

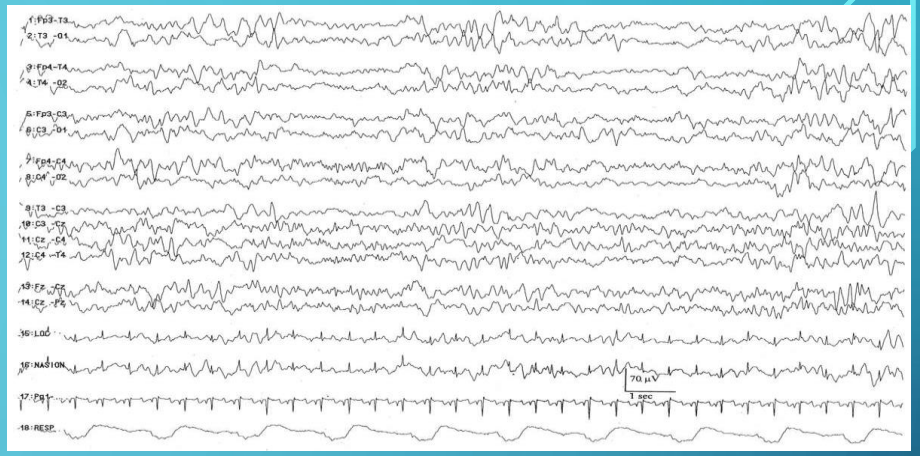
- Retrospective review, N=38 neonates with HIE with rScO2 data during cooling between 2013-2016 (total N of babies cooled during that time: 62).
- data: continuous vEEG, CO values throughout cooling, post rewarming MRI
- Hypotheses:
  - Persistently abnormal vEEG tracings would correlate with severe injury.
  - Higher rScO2 during cooling will correlate with greater severity of the hypoxic-ischemic injury as seen on brain MRI.
  - Cerebral oximetry values and vEEG results can be combined to construct an injury prediction model.

VEEG PATTERNS

- Continuous:

normal continuity, or amount of uninterrupted activity,

for age with only discontinuous periods during quiet sleep.

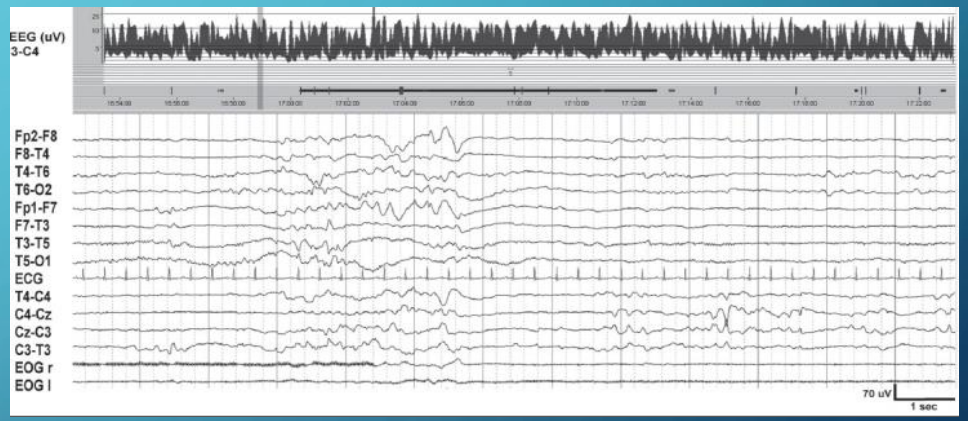


- Discontinuous:

at least one hour of burst activity (with some normal features)

separated by low voltage intervals with no discernible activity

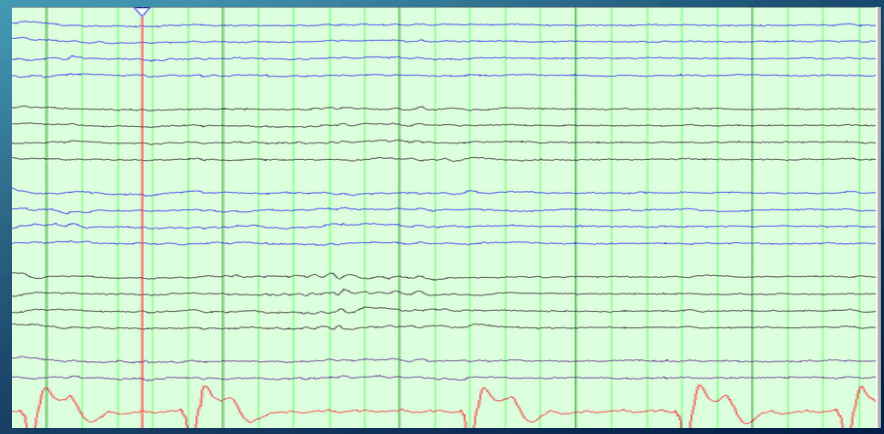
and is not explained by *tracé alternant* (quiet sleep)



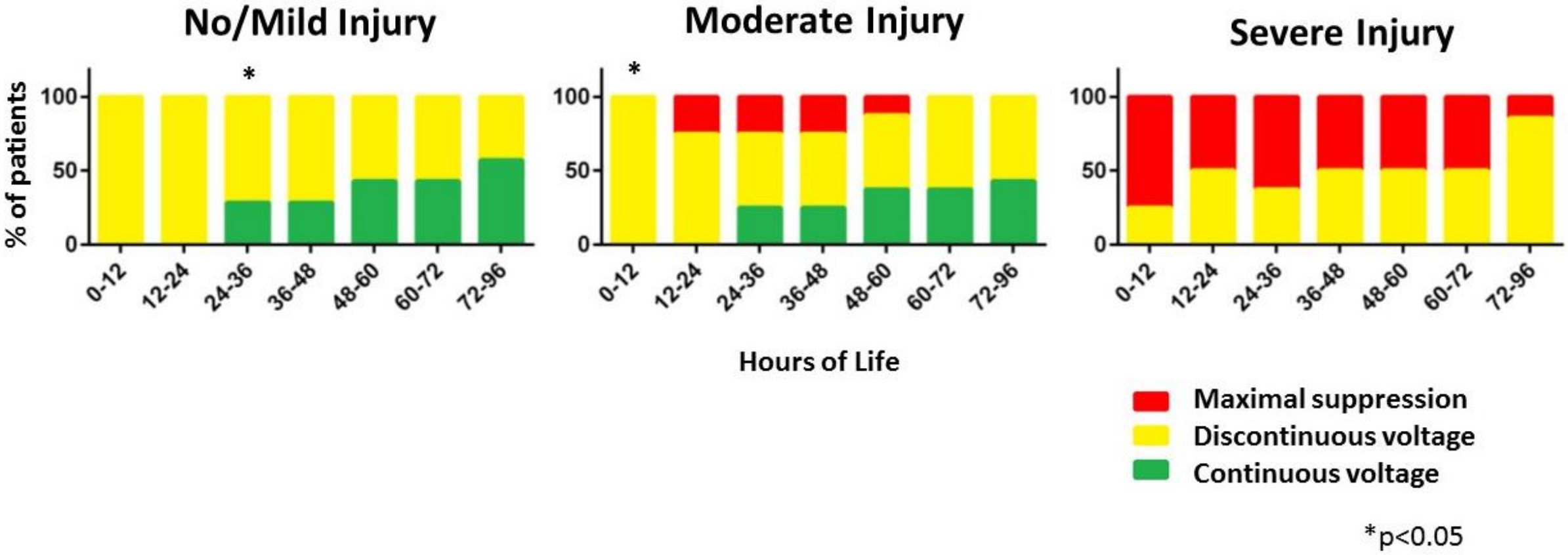
- Maximal suppression:

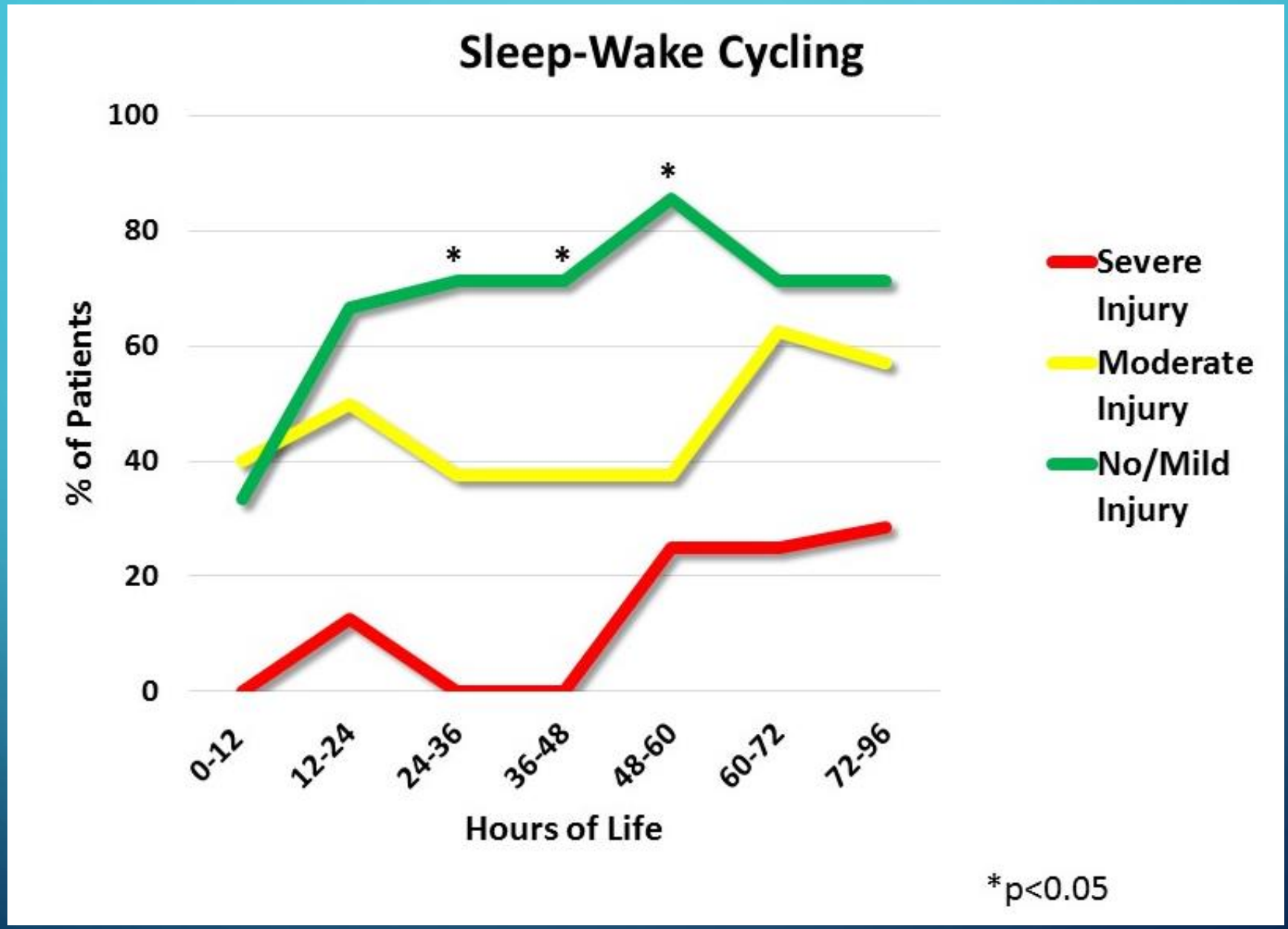
No discernible background activity for at least one hour

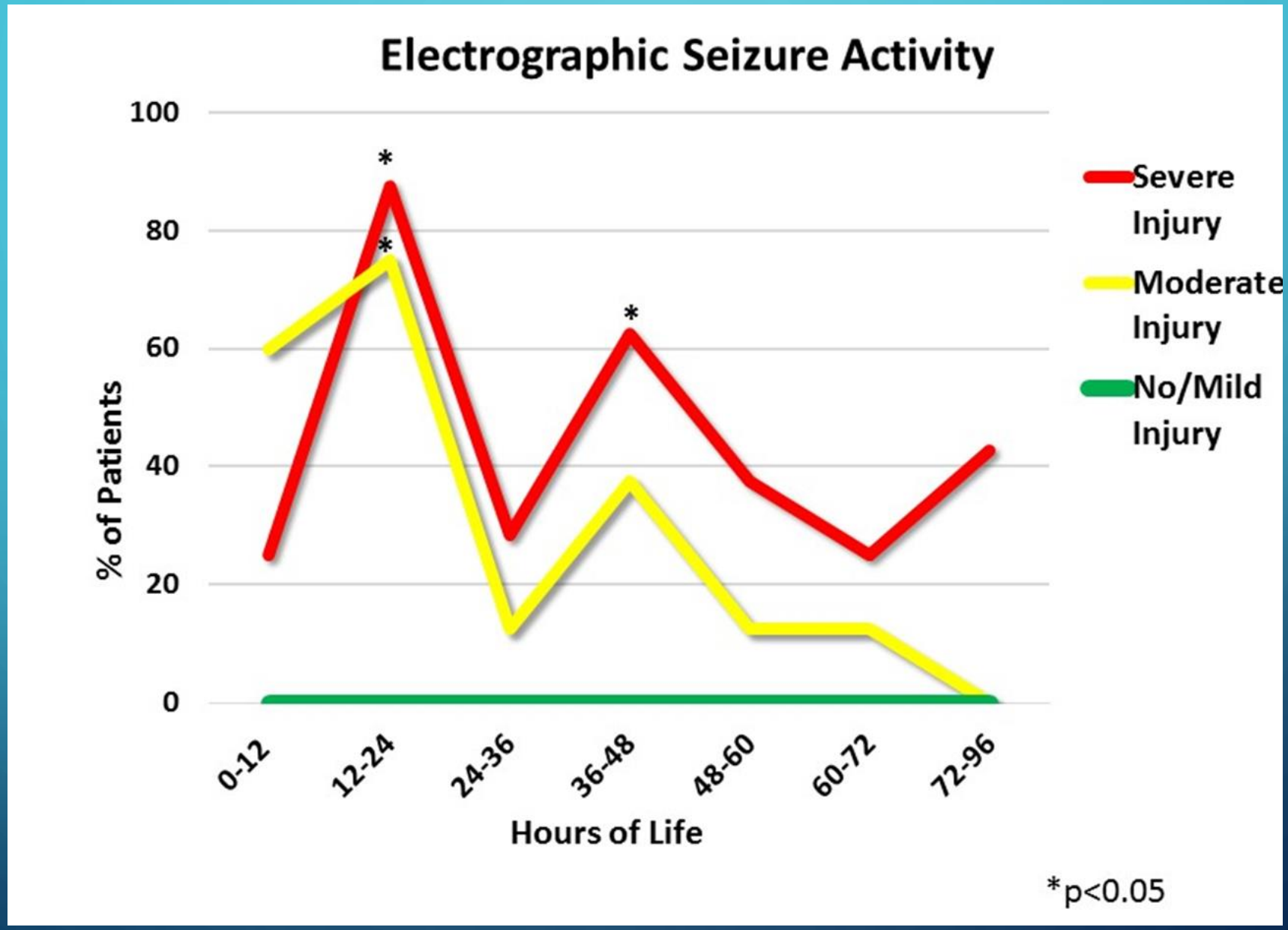
OR seizure activity on a maximally depressed background

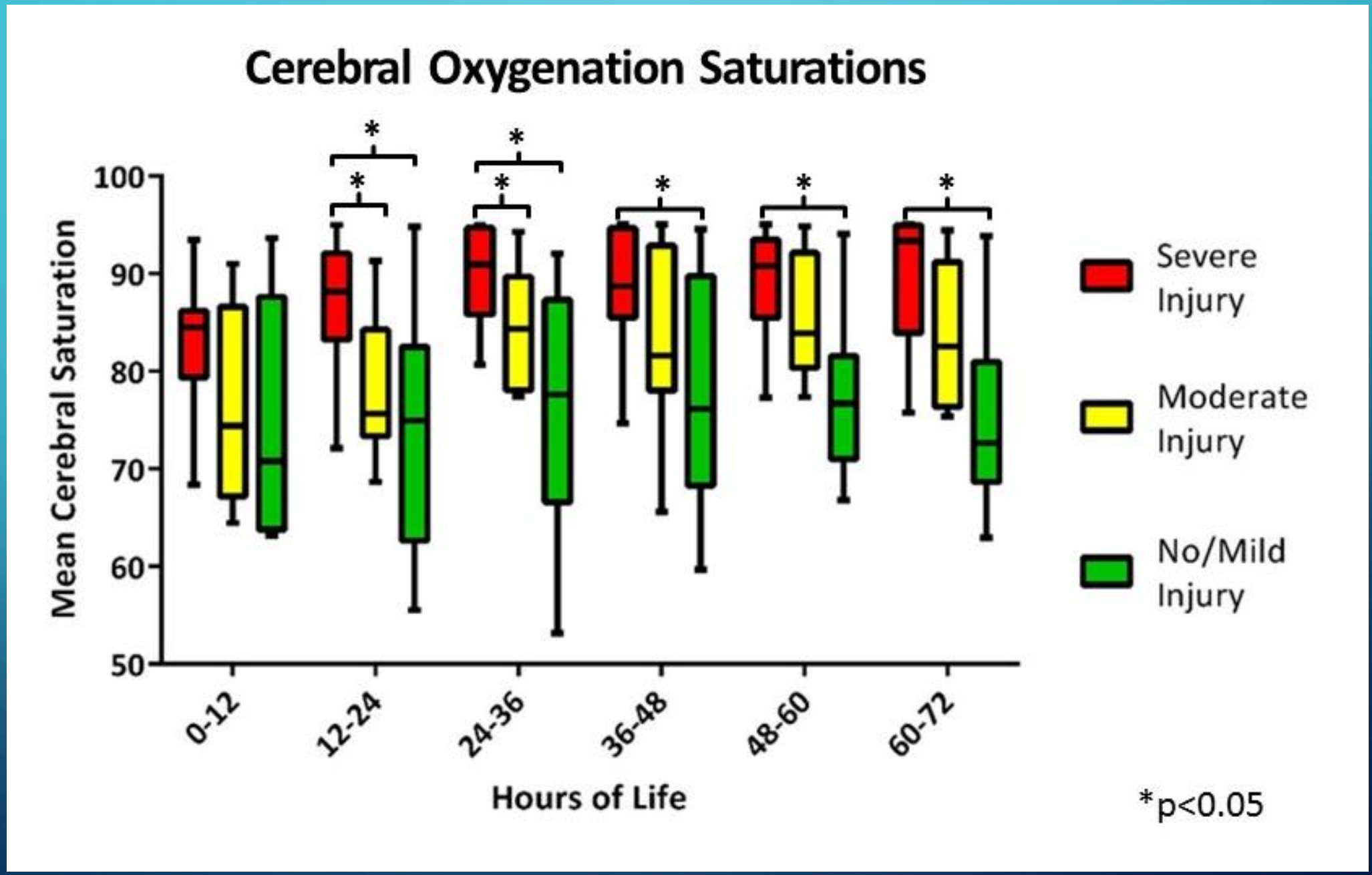


EEG Background Pattern




Sleep-Wake Cycling
100
80
60
40
20
0
% of Patients
*
*
*
0-12
12-24
24-36
36-48
48-60
60-72
72-96
Hours of Life
Severe Injury
Moderate Injury
No/Mild Injury
*p<0.05


Electrographic Seizure Activity
100
80
60
40
20
0
% of Patients
*
*
*
0-12
12-24
24-36
36-48
48-60
60-72
72-96
Hours of Life
Severe Injury
Moderate Injury
No/Mild Injury
*p<0.05


Cerebral Oxygenation Saturations
Mean Cerebral Saturation
100
90
80
70
60
50
0-12
12-24
24-36
36-48
48-60
60-72
Hours of Life
Severe Injury
Moderate Injury
No/Mild Injury
*p<0.05

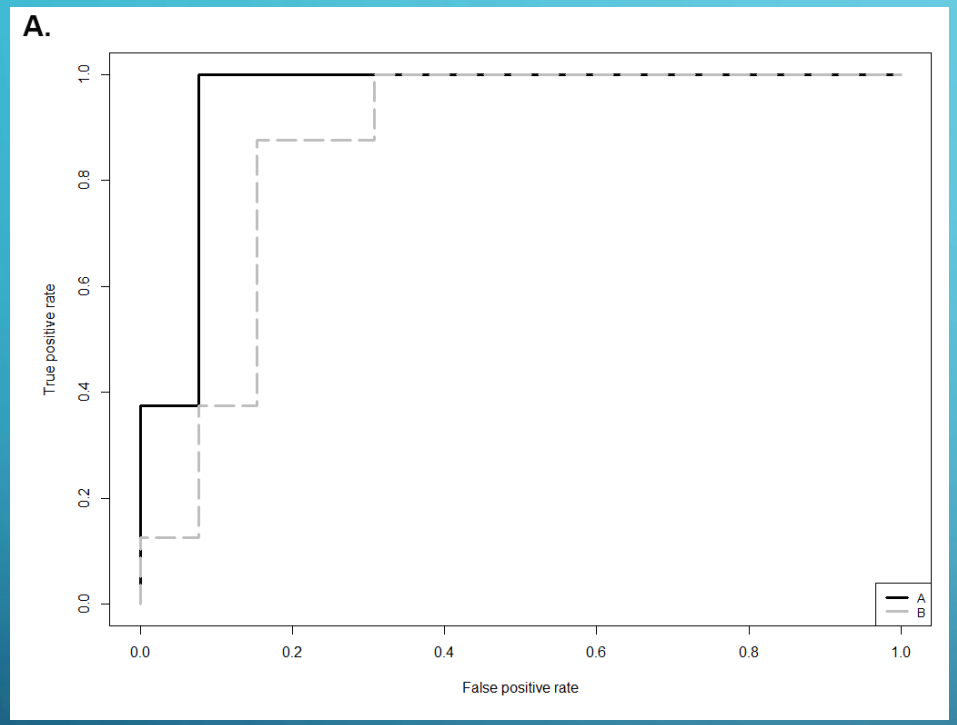
No SWC	0-12 hr	12-24 hr	24-36 hr	36-48 hr	48-60 hr	60-72 hr	72-96 hr
Sensitivity	100	88	100	100	75	75	71
Specificity	38	57	53	53	60	67	64
PPV	44	54	50	53	50	55	50
NPV	100	89	100	100	82	83	82

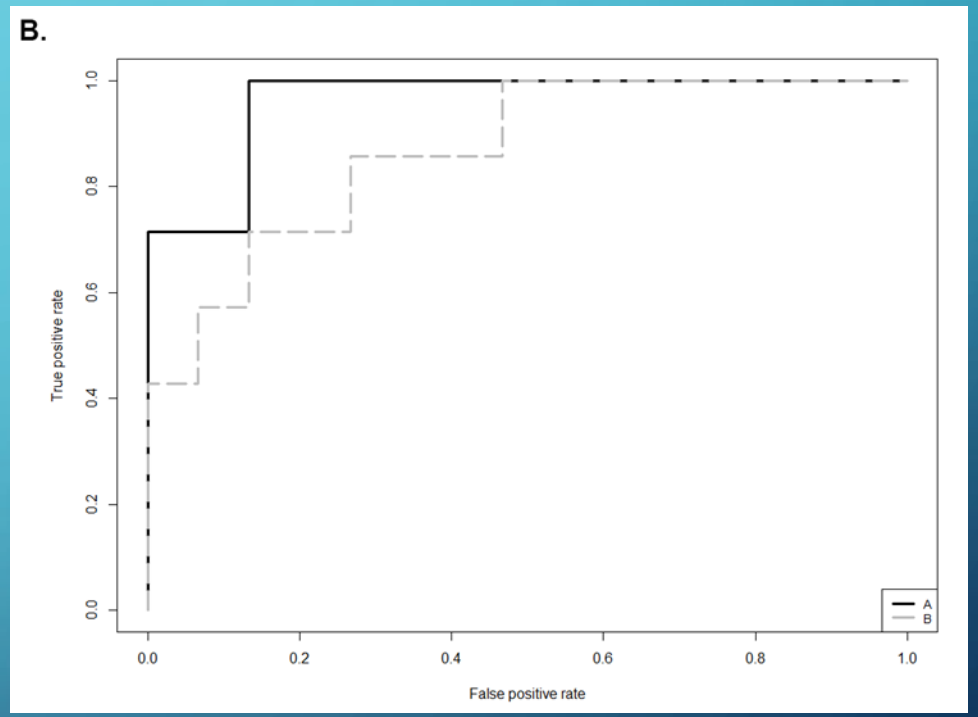
Maximal Suppression	0-12 hr	12-24 hr	24-36 hr	36-48 hr	48-60 hr	60-72 hr	72-96 hr
Sensitivity	75	50	50	50	50	50	14
Specificity	100	86	87	87	93	100	100
PPV	100	67	67	67	80	100	100
NPV	89	75	76	76	78	79	70

Seizures	0-12 hr	12-24 hr	24-36 hr	36-48 hr	48-60 hr	60-72 hr	72-96 hr
Sensitivity	25	88	29	63	38	25	43
Specificity	63	57	93	80	93	93	100
PPV	25	54	67	63	75	67	100
NPV	63	89	74	80	74	70	78

RECEIVER OPERATOR CURVES

Epoch:
12-24 hours





Epoch:
24-36 hours

Black line= model A (cerebral oximetry + seizures + SWC + vEEG background pattern)
Grey line= model B (cerebral oximetry only)

QUESTIONS?