Understanding and interpreting the EEG & CSA

The purpose of this document is to help gain a better (though basic) understanding of EEGs and the compressed spectral array (CSA). It should be read in conjunction with the Local Guideline for IntelliVue CSA / EEG. It contains some of the same information but goes more in depth on these revisited topics. Further, this document will focus on the indications for EEG in critical care settings, the evidence for its use and on how to interpret what you see. The appendices contain useful terms and definitions and give an outline on how to report the EEG.

Table of Contents

ABBREVIATIONS & GLOSSARY

ABI	Acute brain injury
AED	Anti-epileptic drug
cEEG	Continuous electroencephalogram
CBF	Cerebral blood flow
CSA	Compressed (compound) spectral array
DCI	Delayed cerebral ischaemia
EEG	Electroencephalogram
FFT	Fast Fourier Transformation
JHH	John Hunter Hospital
GCS	Glasgow Coma Score
Hz	Hertz (cycle per second; unit of frequency)
ICH	Intracerebral haemorrhage
ICP	Intracranial pressure
ICU	Intensive Care Unit
MDF	Mean dominant frequency
MO	Medical officer
NCSE	Non-convulsive status epilepticus
NCS	Non-convulsive seizure
NIRS	Near-infrared spectrometry
PbO_2	Brain tissue oxygen tension
PPF	Peak power frequency
qEEG	Quantitative EEG
RAS	Reticular activating system
SAH	Subarachnoid haemorrhage
SE	Status epilepticus
SEF	Spectral Edge Frequency
SSEP	Somatosensory evoked potential
Sz	Seizure
TP	Total power
TBI	Traumatic brain injury
TCD(I)	Transcranial Doppler Imaging

Introduction

Critical care patients with reduced level of consciousness can be difficult to monitor, and there are limited tools at our disposal to assess these patients and to determine changes in their clinical state. Generally, we depend on serial clinical / neurological examinations, intracranial pressure monitoring and neuroimaging with CT / DSA / MRI. Confined by this, we may at times miss significant pathology and, once recognised, the injury may have already become irreversible.

Over the last decade(s) novel technologies have become available beyond research use and are increasingly being used for monitoring in intensive care units. Some of these technologies include transcranial Doppler imaging (TCD), brain / CSF microdialysis, brain tissue oxygen tension (PbO_2), near-infrared spectrometry (NIRS) and continuous EEG monitoring (cEEG). In a consensus paper, The European Society of Intensive Care Medicine (ESCIM) and the Neurocritical Care Society recommend various forms of multi-modal monitoring for selected critical care patients with acute brain injury (ABI) [2]

It can be hard to obtain conventional EEGs due to resource limitation and costs. When performed, this bedside test usually records for only 30-60 minutes, potentially missing significant pathology. On the other hand, if monitored for 24 hours or longer, a vast amount of data would be generated and requires expert / neurologist review for its complex and time-consuming interpretation. To minimise the demand on resources, software manipulation of the raw EEG data has been found useful to allow for continuous EEG monitoring. These *quantitative* EEG (qEEG) techniques cannot replace the “gold standard” of conventional electroencephalograms but can serve as a valuable bedside tool for the evaluation of the intensive care patient with altered / reduced level of consciousness.

Basics of EEG and CSA

Electroencephalography (EEG) is the recording of spontaneous electrical activity generated by the cortical pyramidal cells and the thalamus with modulation by the reticular activating system (RAS). The electrical potential recorded on the surface is the summation of post-synaptic potentials and range from 0 to about 200 microvolts (µV). The frequency spectrum of these “brain waves” range from once every few seconds to 30 or more per second (Hz) and are divided into four bands for the purpose of interpretation [3]. An individual’s EEG varies with alterations in his / her state of consciousness/wakefulness, brain function, metabolism and blood flow.

Waveforms and frequency bands

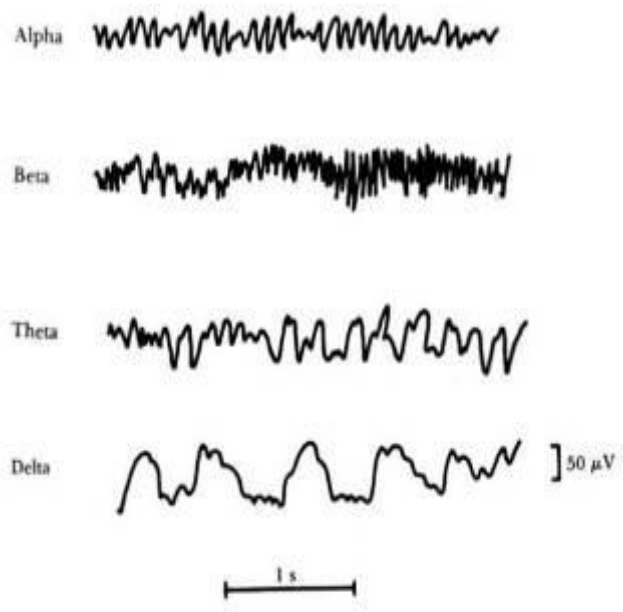

Fig. 1 *The four major frequency bands.*

Alpha waves (8-13 Hz band) predominantly arise from the occipito-parietal areas, are of lower voltage (30-50 µV) and are symmetrical (R sometimes > L) and usually found in normal, awake but resting (eyes shut) adults. Alpha waves are disrupted by activity and mental stimulation.

Beta waves (13-30Hz) usually have voltage <50 µV, are symmetrical, present in the frontal regions of awake and alert adults. Beta-activity depends on intact cortical input from the thalamus and RAS. The beta frequency band is sometimes subdivided into β1 (13-20 Hz) and β2 (20-30 Hz) for trend monitoring purposes.

Theta waves (4-8 Hz) normally occur in the parietal and temporal areas in children and sometimes during emotional stress or deep relaxation in adults.

Delta waves (<4Hz) are prominent during deep sleep and anaesthesia, in infants and with severe organic brain disease. These waves originate from central parts of the brain, are usually of higher amplitude / voltage (>50 µV), and signify minimal cortical influence by the lower brain centres.

Other defined waveforms and frequencies include gamma (30-100 Hz), lambda, kappa and mu waves, but these are less relevant for our use in critical care.

Frequencies and waveforms change with age, but details about this is beyond the scope of this paper. In general, the slower frequencies of infancy and childhood mature into adult patterns before there is an observed slowing of the background activity in the elderly.

Quantitative EEG and CSA

Conventional EEG requires accurate and standardised placement of 20-22 electrodes and yields great spatial resolution. Continuous traces can then be recorded and analysed according to location / symmetry, waveform frequency, voltage (amplitude), and rhythmicity.

Interpretation of EEGs is a specialist task and requires extensive experience. As a result, various forms of computerised manipulation of the raw EEG data have been developed to simplify recordings and minimise resource utilisation. By applying (fast) Fourier transformation (FFT), EEG can be *quantified* in terms of its amplitude, power, frequency and / or rhythmicity to generate numerical values, ratios or percentages – hence the term *quantitative EEG* (qEEG). The filtered and compressed data can then be graphically displayed to allow *trend monitoring* and interpretation by non-EEG trained personnel. Most qEEG software available today uses a limited montage with 2 to 6 channels. The technology is therefore less sensitive than conventional EEG and should be used as part of the overall assessment of the patient and as a "screening tool" that may allow for earlier investigation and expedite appropriate management [1,4]. Despite being available for a few decades, this technology is fairly "new" in ICU and more research is needed to determine the optimum number of channels, the best montage, the best combination of qEEG trends and the optimal duration and timing of monitoring [5].

Philips IntelliVue™ monitors offer *two-channel qEEG monitoring* with five electrodes through a technology called *compressed spectral array* (CSA). Here, the raw EEG signals are filtered and sampled over a period of time (epoch), and the generated output data yields a graphical snapshot of the amplitude / power (*y axis*) across the range of frequencies (*x axis*) as seen in figure 2.

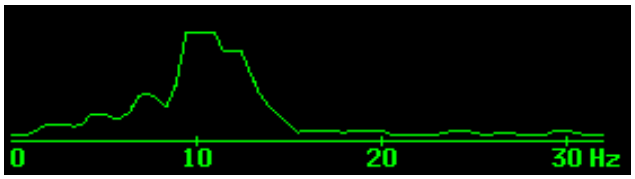

Fig. 2 *Fourier spectrum showing the contributions made by different frequencies for a given epoch time.*

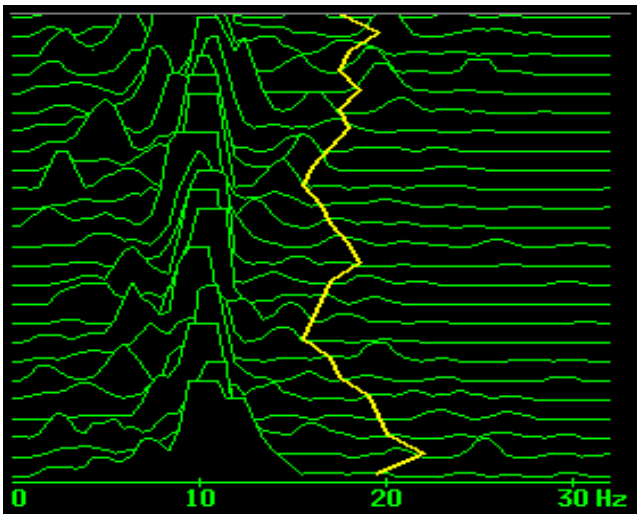

Fig.3 *Spectral array or Spectrogram – see text.*

The data from the next epoch will give another snapshot, which is placed in front of the former. Over time (*z axis*) a three-dimensional stack of these power spectrum snapshots will emerge with the newest at the front and the oldest at the back as in figure 3. The epochs on the IntelliVue™ can be changed from 2 seconds to 120 seconds to allow for more or less temporal detail in the window. One such display for each hemisphere will be visible to allow for assessment of symmetry. Numerical values including total power (TP) and spectral edge frequency (SEF) are also available and can be graphically displayed for trend to assist with the interpretation. Please refer to Appendix 1 for an explanation of these and other relevant terms.

CSA was one of the first forms of qEEG to be developed and is not commonly used today. A different form of time-frequency based qEEG includes density spectral array (DSA), which is available at the Calvary Mater Hospital with their Spacelabs monitors. Other forms of qEEG include amplitude integrated EEG (aEEG; commonly used in neonatology), enveloped trend analysis and several proprietary qEEG technologies. Please refer to Handbook of ICU EEG Monitoring [8] for more details.

Indications for the use of EEG and CSA monitoring

Evidence is sparse and recommendations for EEG monitoring in ICU is not well defined [2]. It is often recommended despite there being lack of hard evidence to support this. This is because EEG is non-invasive and carries low risk, so its use is justified by the potential harm of missed pathology, e.g. unrecognised seizure activity. It is imperative to note that most recommendations have been made for the use of conventional (continuous) EEG and not necessarily for CSA qEEG.

In an American survey [6], most responding intensivists utilise cEEG to identify non-convulsive seizures in critically ill patients. Only a minority uses cEEG for indications other than seizure detection and to titrate barbiturates / sedatives.

At JHH ICU the primary use of CSA EEG is for detection of electrical seizure activity and to document duration and response to treatment. Our 2-channel CSA is best at detecting generalised / global abnormalities and is not a great tool to detect regional and more subtle abnormalities seen with various encephalopathies. Great care must be taken if used for prognostication purposes.

Clinical indications

1. **Unexplained coma / unresponsiveness**

   International guidelines recommend cEEG for 48 hours in comatose patient to evaluate for NCS as 80% of seizures are detected by 24 hours and 87% when monitored up to 48 hours [2,6].

2. **Status epilepticus**

   Non-convulsive seizures and NCSE are frequent after generalised convulsive seizures (48% and 14%, respectively). Differentiating ongoing seizure activity from postictal or medication-induced encephalopathy can be challenging. Continuous EEG is required to guide treatment for refractory SE.

3. Post-cardiac arrest syndrome / coma

Seizures are documented in 10-40% of patients with coma after cardiac arrest, most being non-convulsive. Early seizure activity is generally a bad prognostic sign, as are burst suppression or generalised periodic discharges (see *Appendix 1*). EEG can also be used to differentiate myoclonic status from peripheral or subcortical myoclonus. Consensus guidelines suggest EEG during therapeutic hypothermia and within 24 hours after rewarming to rule out NCS in all comatose patients after cardiac arrest.

Emerging evidence suggests that cEEG and qEEG with simplified montage can be used reliably to identify *malignant patterns* and help with prognostication [16]. Nevertheless, at JHH ICU we should request conventional EEG (and SSEP) and neurologist input when assistance with prognostication is required.

4. Post severe TBI or intracerebral haemorrhage

Patients with severe TBI are at risk of seizures, and some datasets suggest a prevalence of 22-33%, of which half were non-convulsive seizures. Risk factors include depressed skull fractures, penetrating injuries and haemorrhages. Guidelines recommend the use of cEEG to rule out seizures. CSA should *not* be used to prognosticate after TBI. (Hyperlink to TBI guideline)

5. Severe subarachnoid haemorrhage

Continuous EEG can be used for comatose patients after SAH to detect seizure activity and delayed cerebral ischaemia (DCI) / vasospasm. Acute seizures have been reported in 3-26% of comatose aSAH patients, frequently non-convulsive seizures or NCSE. By monitoring trends in brain wave frequencies, it *may* be possible to detect DCI and vasospasm before clinical signs and much earlier than radiological signs. (Hyperlink to SAH guidelines)

6. Titration of sedation

Barbiturate coma is sometimes used for refractory intracranial hypertension and refractory status epilepticus. The thiopentone dose should be adjusted to the EEG pattern, aiming for burst suppression (hyperlink to TBI guidelines and Thiopentone guideline).

Electrode position and montages

The Philips IntelliVue™ EEG module uses an array of five EEG leads or electrodes – two electrode pairs and one reference lead. The latter is usually placed centrally / midline on the scalp, whereas the monitor software will guide you through the positioning of the two electrode pairs. There are five different montages A-E to choose from, and there is no consensus to which one is the preferred one.

Conventional EEG requires accurate placement of 20-22 electrodes, follows the International 10-20 system and is quite complex and time consuming. Each electrode represents a specific site of the cortex and is labelled by a letter and number (see fig. 4 and 5 below). By convention, the left sided electrodes have odd numbers and the right sided have paired numbers. The letters refer to the underlying lobes of the brain. The placement of electrodes for our 2-channel CSA EEG needs to be symmetrical but otherwise not as precise as for the conventional EEG.

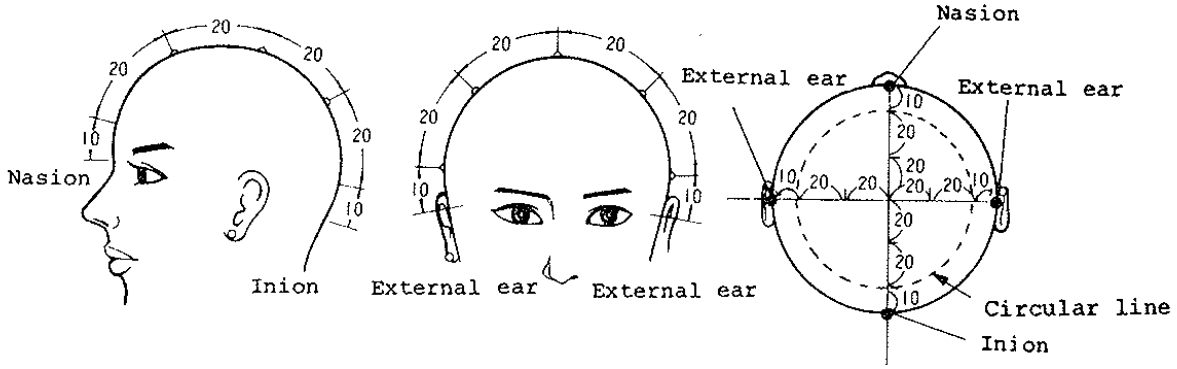

Fig. 4 *The International 10-20 system for electrode placement where the skull is divided into 100 equal units in the sagittal and coronal plane measured from the nasion to the inion and from the left to right external ear, respectively. (Fig. from [9])*

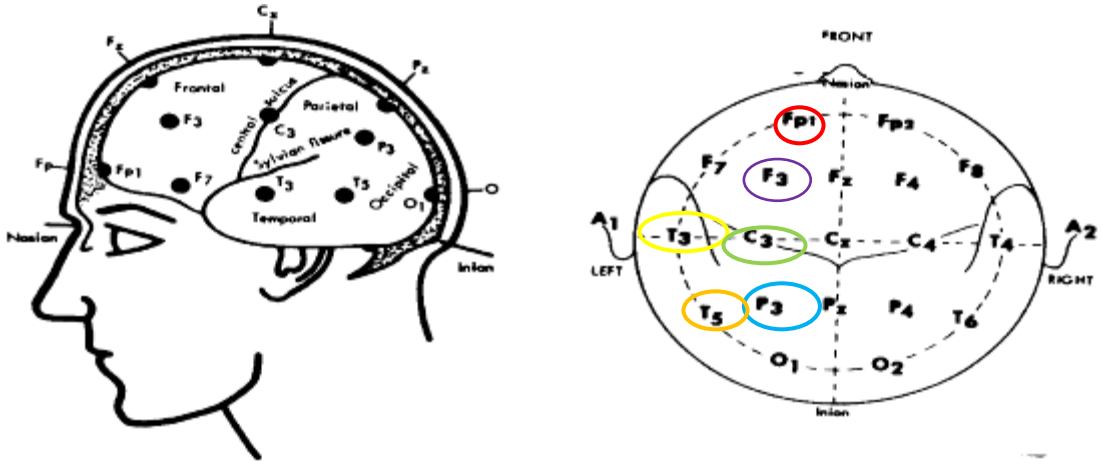

Fig. 5 *Relevant landmarks and anatomy of the brain and their relationship to standard electrode placement. The encircled sites on the picture on the right are the left-sided electrode positions used with the Philips IntelliVue®. (Fig. from [9])*

As a rough and simplified guide in *adult* ICU, do the following for the electrode placement used in montages A-E:

- Fp1/2 (Frontal) – Approximately 3.5 cm above the nasion and 3 cm lateral to this midline point (usually over medial part of eyebrows).
- C3/4 (Central) – Approximately two-thirds up from the external auditory meatus to the (sagittal) midline.

- T3/4 (Temporal) – Approximately 4 cm above the external auditory foramen.
- F3/F4 (Frontal) – Approximately midway between Fp1/2 and C3/4.
- P3/4 (Parietal) – About 5 cm behind C3/4 or 5 cm above T5/6 towards the midline.
- T5/6 – midway between a point 3 cm above the external occipital protuberance (inion) and T3/4.

There are no studies to suggest that one montage is superior to the others. Most significant seizures in the comatose critical care patients are generalised, so any of the five montages A-E are equally likely to detect global epileptic activity.

When choosing a montage is seems logical to consider and avoid drains / probes, bandages, wounds etc. and perhaps use areas of the scalp free of hair to optimise skin contact. Posterior leads are more likely to lose skin contact with any head movement and increase the chance of pressure areas with prolonged monitoring. Keep in mind that montages involving Fp1/2 are prone to ocular artefacts and should be avoided if there are lots of eye or forehead muscle movement. Further, consider the underlying pathology and use, for instance, temporal electrodes if there is an underlying temporal haemorrhage or suspected herpes simplex encephalitis.

When monitoring for DCI or ischaemia it has been suggested that F3-C3 / F4-C4 roughly correspond to ACA territory, C3-T3 / C4-T4 correspond to MCA territory, and P3-O1 / P4-O2 corresponds to PCA territory [13].

If no abnormality has been detected within 12-24 hours and the patient's clinical status remains unchanged, consider changing the montage to monitor / screen over a different anatomical region.

Interpretation and patterns to recognize

People spend years to learn how to interpret EEGs so this can't be taught in a few paragraphs. Also, there are a lot of controversies and uncertainties with definitions / terminology and their interpretation, and the interrater agreeability is at times poor.

Diagnostically, the most important use of the CSA EEG is to rule in / out epileptiform activity, and our focus at JHH should be on this. Therapeutically, we should use this technology to titrate antiepileptic and sedative agents.

Please read Appendix 1 for an understanding of important terminology before continuing reading.

Seizures

- **An electrographic seizure is an abnormal, sustained, rhythmic electrical discharge (that evolves).**
- An epileptiform pattern has been defined as "transients distinguishable from background activity, with a characteristic spiky morphology, typically, but neither exclusively nor invariably, found in interictal EEGs of people with epilepsy." [Noachtar et al]

- Both the raw ("live") EEG trace and the CSA can be used to help diagnose epileptiform activity. CSA, being a condensed display of EEG based on power (amplitude) and frequency, can be a great tool to "find the needle in the haystack" but can never *replace* conventional EEG.
- Seizures can be very easy or incredibly difficult to recognise.
- In the ICU environment, there can be significant false positives related to artefacts.
- Electrographic seizures in ICU patients often have a different morphology to other patient groups. The seizures are often more *slowly evolving* and can be of *low frequencies and amplitudes*. This increases the potential for missed cases (false negatives) [8].
- **Many different EEG patterns can be observed in comatose patients and some may be confused with epileptiform activity, others are considered borderline epileptiform or of uncertain significance [1]. Examples of the latter include generalised or lateralised period discharges with a frequency < 2.5Hz or rhythmic discharges faster than 0.5 Hz. Expert / neurologist's opinion is needed for these cases.**

EEG criteria for determination of an electrographic seizure (modified from [8])

1. Repetitive or rhythmic focal or generalised epileptiform discharges (e.g. sharp waves and spikes) at greater than 3 Hz and lasting for longer than 10 seconds.
2. Similar discharges at less than 3 Hz with clear evolution in frequency, location or waveform / morphology OR clinical motor manifestations.
3. Clinical improvement and improvement or normalisation of the EEG after acute administration of a rapid-acting IV anti-epileptic drug (AED), typically benzodiazepines.
4. Suspect seizure (NCS) if a patient returns rapidly to a baseline state (after AED or spontaneously) and the EEG becomes normal.

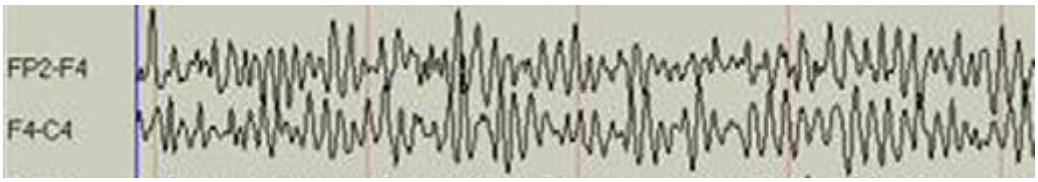

Fig. 6 *Spikes and polyspikes with frequency around 12 Hz [8]*

Fig. 7 (below) *Patient with status epilepticus with a regular, rhythmic spike and wave pattern at a frequency of around 15 Hz. Note the SEF 90% (yellow line) in the CSA showing minimal fluctuation over the last 12 * 2 seconds (set buffer period). Note the moderately high total power (TP), supporting the diagnosis of seizures.*

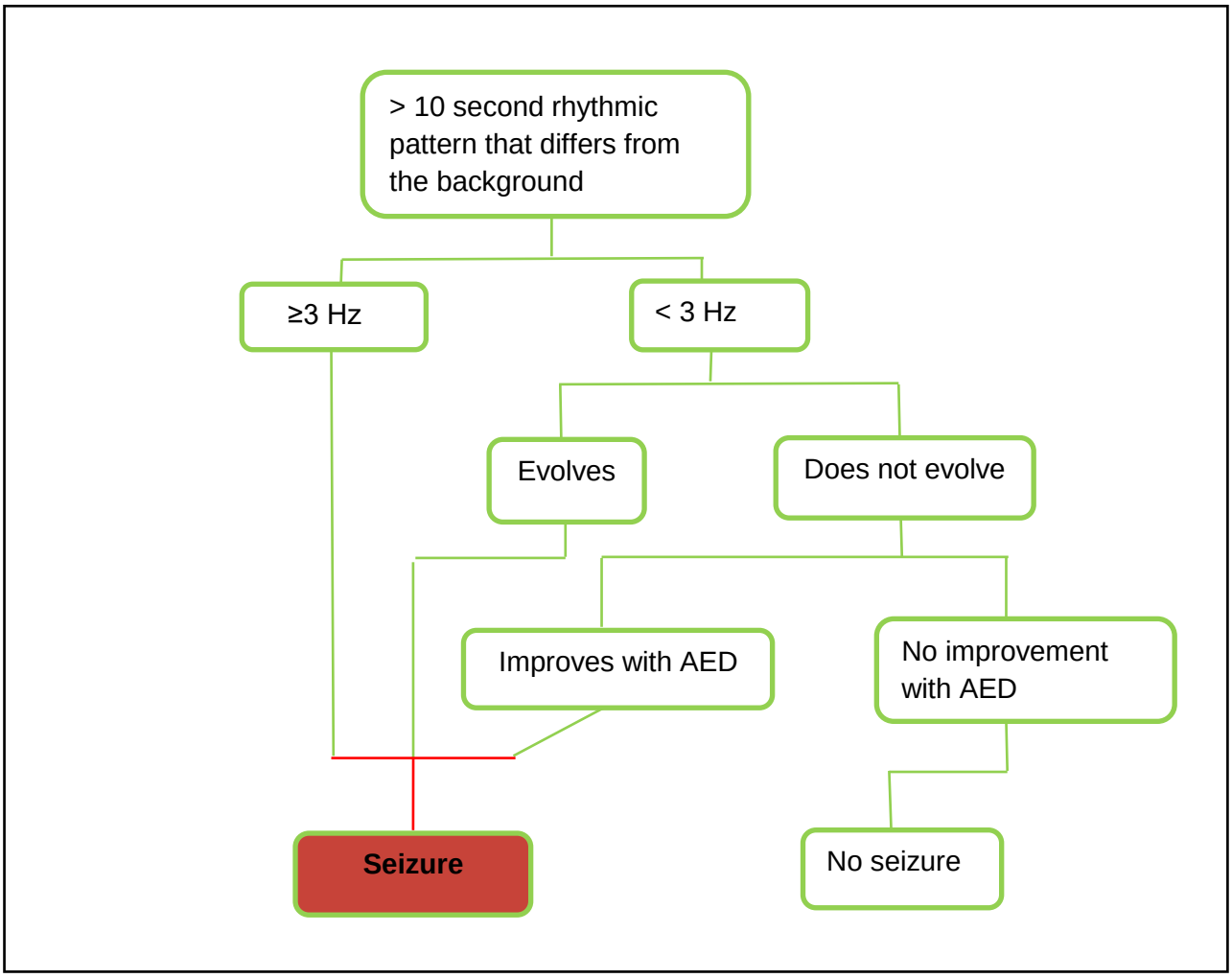

Fig. 8 *Schematic approach to the diagnosis of electrographic seizures [20].*

Typical features with regards to NCSE [1,8]

1. Rhythmic, generalised, symmetric spike-and-wave or polyspikes and waves at 2-3.5 Hz (fig. 9).
2. Atypical spike-and-wave with lower frequency and less symmetry.
3. Multiple spike-and-wave.
4. High voltage, repetitive, rhythmic delta activity with intermixed spikes, sharp waves or sharp components.

Fig. 9*. NCSE with spikes and spike-and-wave pattern day 2 post cardiac arrest in patient with myoclonus. The patient died after withdrawal of active treatment.*

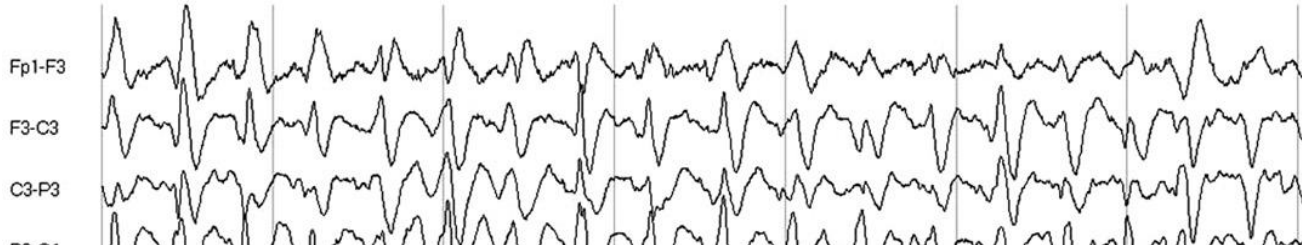

Fig. 10 *Generalised 2.5 Hz spike and slow wave activity with later clinical and electrographic improvement with lorazepam [8].*

Fig. 11 *Comatose patient with subtle facial muscle contractions and periodic generalised discharges signifying the possibility an inter-ictal state and NCS. This is one of the tricky patterns that even experts often disagree about. Suppressing the pattern pharmacologically usually doesn't change the outcome.*

Seizures and CSA

- The four most important CSA trends for seizure detection are based on the following characteristics:
  - a change / evolution in amplitude (power)
  - a change / evolution in frequency
  - asymmetry (unless generalised)

  } when compared to background activity

*Artefacts are typically represented by a prolonged or sudden change (no evolution).

Fig. 12 *CSA trace with marked change in power spectrum and an impressive spike in SEF 90%. Clinical correlation is necessary as this rather sudden change could also be due to artefacts.*

Fig. 13 *Comatose patient with NCSE / myoclonic status. No marked change / evolution on this trace but relatively high dominant frequency in a non-responsive patient raises suspicion.*

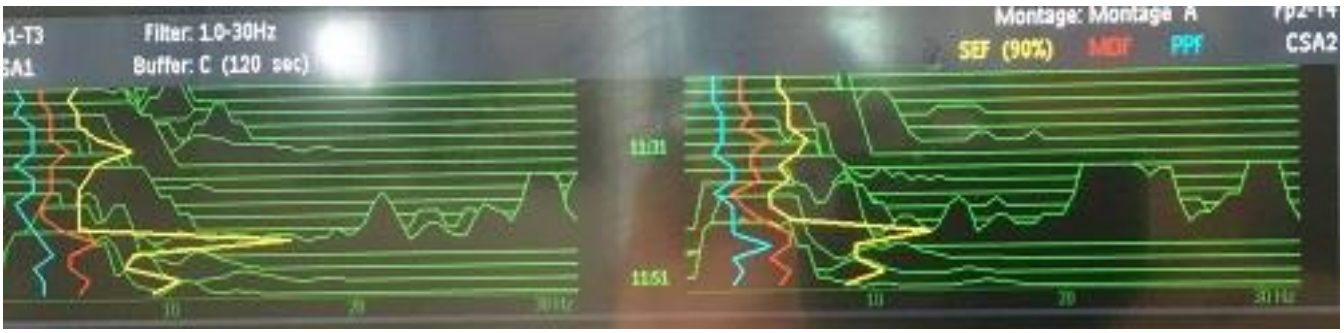

Fig. 14 *Concerning CSA patterns in a patient with hepatic encephalopathy and intermittent seizures. Note the marked increase in frequency represented by a shift of the 'hills and valleys' to the right and by a step-up in the SEF 90 (yellow line) evolving over 1-2 time lines. Clinical correlation and assessment of the raw EEG are important.*

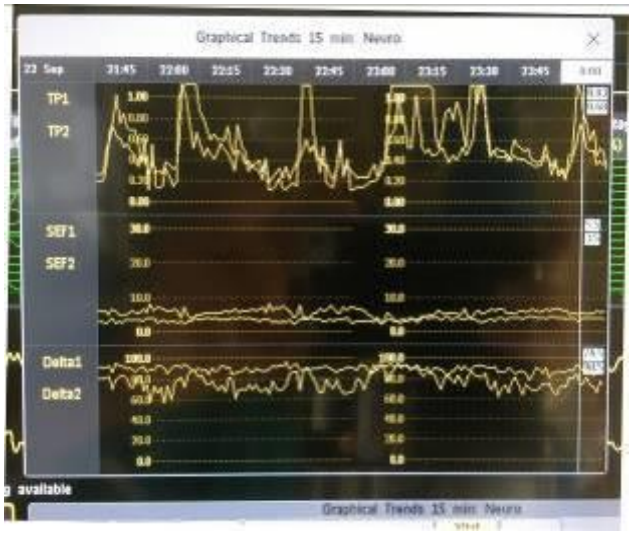

Fig. 15 ***Fig. 16***

Figs 15-17 *are from the same patient / recording; both numerical and graphical trends are shown. She reportedly exhibited some right-sided tonic activity the night before. Retrospective review of the CSA suggested an evolution of frequencies and total power over the left hemisphere around 23:00, lock stepping with the clinical event. Note the gradual increase in TP1 and Delta1 with subsequent "normalization" to baseline values. The background extreme slow-wave activity on the right side (Delta2) correlated well with her extensive right-sided infarct. In this case it would be very important to review the raw EEG to determine if the clinical findings and CSA represented true electrographic seizure activity.*

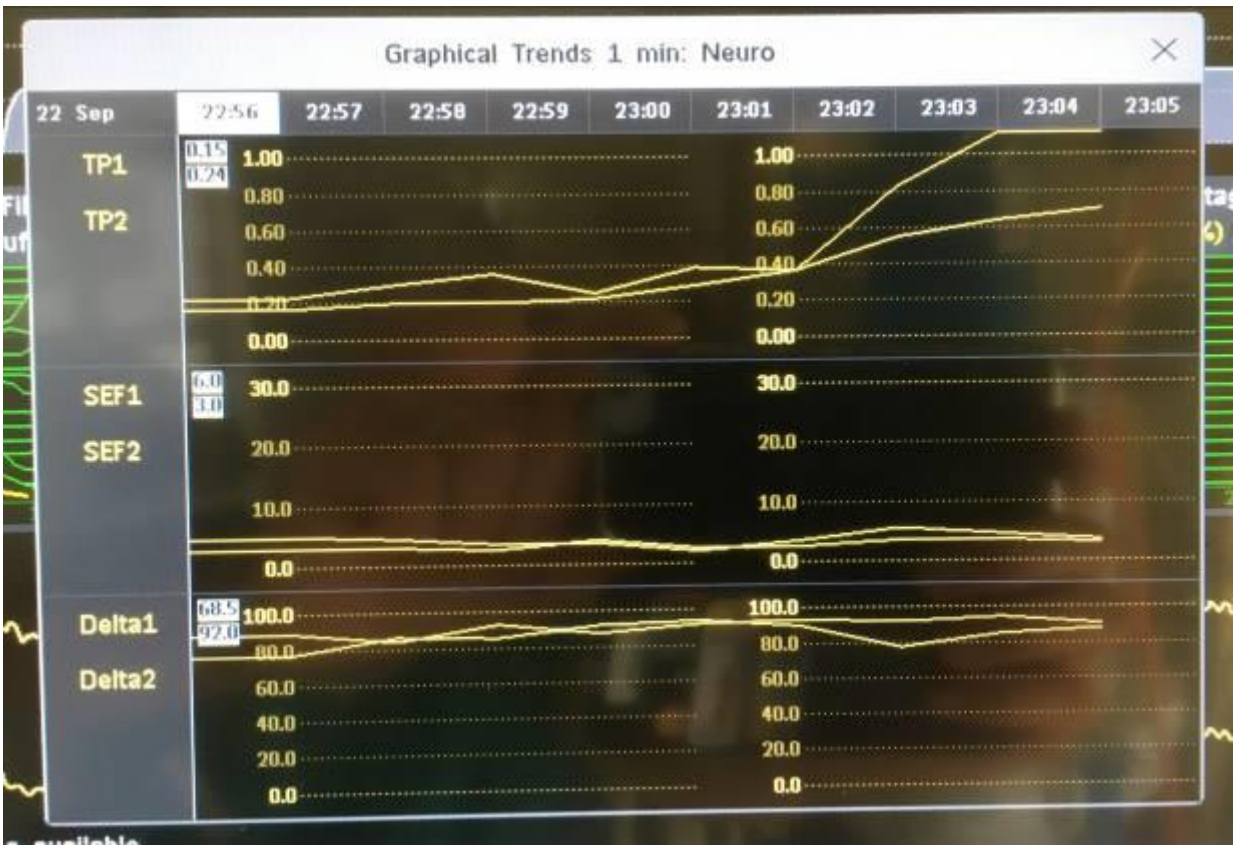

Fig. 17

Subarachnoid haemorrhage and ischaemia

1. Seizures

Acute seizures have been reported in 3-26% of comatose aSAH patients, frequently non-convulsive seizures or NCSE. See above on how to recognize seizures on the EEG.

2. Ischaemia and DCI

- It is a well-established fact that there is a relationship between cerebral ischaemia and spectral changes in the EEG.
- The pyramidal neurons found in the deeper cortical layers are exquisitely sensitive to ischaemia and low oxygen levels – any reduction in blood flow and oxygen supply will cause early changes on the EEG. A significant reduction of CBF and / or change in metabolism can sometimes be seen within 60-90 seconds in the form of paucity of faster frequencies, followed by an increase in slow frequency waveforms (theta and delta). Ongoing ischaemia leads to inadequate ATP generation and eventually Na/K ATPase failure, resulting in infarction and suppression of the EEG.
- Serial exams and imaging are only capable of detecting DCI once the damage becomes clinically or radiologically apparent. EEG may be useful to <u>detect and treat before</u> irreversible injury occurs. I.e. we can potentially titrate vasopressors and set blood pressure targets based on changes on the ECG.

- Relative delta percentage (delta power / total power) appears to provide the most robust correlation with CBF and $CMRO_2$ during focal ischaemia.
- The suggested changes and trends to look out for suggesting DCI and / or vasospasm are:
  i. Change in total power
  ii. Increased power in slower frequencies (i.e. increased relative delta percentage)
  iii. Reduced power in faster frequencies (beta and alpha)
  iv. Reduced relative alpha percentage
  v. Reduced alpha / delta ratio (ADR)
  vi. Reduced %relative alpha variability (very difficult to assess without more sophisticated software)

  * As stated above, we're looking for trends and changes, not absolute numbers. The changes may be symmetrical or asymmetrical. To detect significant changes, it is essential to first establish the background activity and baseline values. Most patients with comatose SAH will have predominant slow-wave activity from the initial acute brain injury (and sedation), so further slowing can be very difficult to detect. Ideally, assessments are done post stimulation and with the same level of sedation to exclude confounding effects of sleep and spontaneous changes. The baseline can change gradually and not be significant clinically. However, according to previous studies any reduction in ADR of more than 10% six or more times in a row (e.g. with 15-minutly assessments) OR any reduction in ADR greater than 50% are considered significant and strongly suggestive of DCI.

  Keep in mind that studies that have specifically looked for DCI and spreading cortical depolarisation have used the complete 10-20 EEG montage and sophisticated software. At this stage, it will be very difficult and labour intensive for us to pick up ischaemic changes electrographically. Nevertheless, the bedside nurses will document %alpha and %delta hourly and at times we may be able to detect significant trends.
- The emergence of new discrete or periodic epileptiform discharges has been reported as a potential indicator of ischaemic as well. There is an association between (late) epileptiform discharges and cortical spreading depolarisation and ischaemia.
- Certain patters on qEEG have been suggested to predict later vasospasm – e.g. the development of continuous polymorphic, rhythmic or unreactive delta predicted vasospasm 100% of the time.

Post-cardiac arrest syndrome / coma

- There have been attempts in classifying EEG patterns post cardiac arrest to help predict outcome.
  - **Simplified classification of EEG patterns post CA:**
    - Continuous pattern – associated with good outcome.
    - Flat pattern – not uncommon initially, but associated with poor outcome if present after initial 24 hours or after re-warming.
    - Burst suppression pattern – associated with poor outcome, especially if associated with myoclonic activity. Effect of sedatives must be excluded.

- Electrographic status epilepticus – associated with poor outcome.

- **Malignant vs. benign EEG patterns post CA:**

  Malignant: Post-anoxic status epilepticus, alpha coma, burst suppression, generalized suppression beyond first 24 hours.

  Benign or uncertain significance: Generalised slowing activity, generalised alpha/theta activity/frequencies, epileptiform discharges.

- Friberg et al [16] have shown that this level of prognostication can be performed with simplified / limited montage qEEG (amplitude-derived qEEG instead of CSA).
- Good neurological outcome has been reported following aggressive anti-epileptic treatment for seizures occurring in the rewarming phase after therapeutic hypothermia (TH).
- In patients treated with TH monitored with cEEG, a non-reactive EEG was strongly associated with poor outcome (better than SSEP). On the other hand, a reactive EEG is often associated with a good recovery. NB! Reactivity needs to be assessed with conventional EEG.
- **Myoclonus** – Elmer et al. [17] recently classified four EEG phenotypes of early post-anoxic multifocal myoclonus. All but one of the patterns, although rate, were associated with unfavorable outcome. This pattern was recognized by continuous background (various frequencies) with narrow, vertex spike-wave discharges in lockstep with myoclonic jerks (see fig 18). The continuous background suggests an intact cortical component. These discharges appeared to be more responsive to antiepileptic drugs than seen with other patterns and reduced with time.

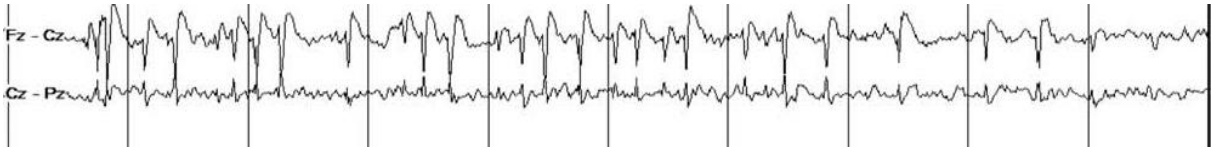

Fig. 18 *Pattern with background activity and intermittent spikes associated with myoclonic activity.*

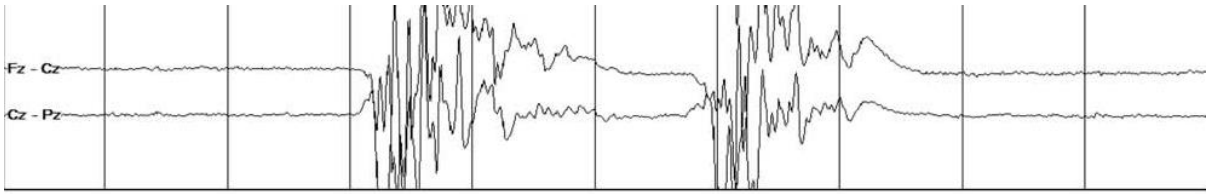

Fig. 19 *Burst-suppression pattern with clinical myoclonic activity in lockstep with the bursts. Invariably associated with poor outcome in the study by Elmer et al [17].*

NB! Despite some convincing evidence, we must be very careful when using CSA EEG as an aid to prognostication after cardiac arrest. Most studies have utilised other forms of qEEG, used more electrodes than our 2-channel technique and had significant neurology input and expertise. When clinically indicated, prognostication should still be done with conventional EEG (and/or SSEP) and review by one of our neurologists.

Barbiturate titration and burst suppression

- EEG and CSA can be used to titrate general anaesthetics / sedatives including Propofol and thiopentone. The two most likely clinical scenarios are
  - Titration of general anaesthetics vs. (refractory) status epilepticus.
  - Titration of thiopentone when used for refractory intracranial hypertension (Hyperlink to thiopentone guideline).
- The literature is unclear about how "deep" sedation one should aim for. Some sources suggest aiming for no more than 2-5 bursts per minute for the best ICP-lowering and therapeutic effect. Alternatively, aim for a burst-suppression ratio (BSR) greater than 75% (i.e. at least 75% of trace to be suppression pattern).
- See figs. 19 and 22 for examples of the burst-suppression pattern.

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19. Noachtar S. et al. A glossary of terms most commonly used by clinical electroencephalographers and proposal for the report form for the EEG findings. Recommendations for the Practice of Clinical Neurophysiology: Guidelines of the International Federation of Clinical Physiology (EEG Suppl. 52). Elsevier Science BV. 1999.
20. *Recognizing seizure in a comatose patient by* Jeremy Moeller. Available through www.youtube.com via *EEG Basics*.

Also, check out EEGucation (www.neurophysiology.com) on www.YouTube.com for numerous useful lectures on EEG.

Appendices

Appendix 1 – Definitions and numerics

A. TERMS SPECIFICALLY RELATED TO DSA [8,9]

Power – The area under the Fourier spectrum amplitude curve within a given frequency range. For example, the alpha power would be the area under the curve from 8-13 Hz. May be expressed as absolute power (the actual value) or relative power (ratio of power in a given frequency to the total power over all frequency ranges)

Total Power (TP1 / TP2) – Total power content (all frequencies) of a channel, i.e. the voltage squared over an impedance of 1 ohm. Unit nW.

Power ratio – Ratio of power in two frequency bands. For example, the alpha to delta ratio (ADR) is the ratio of power in the alpha frequency band to that in the delta frequency band. Use as a potential useful tool for monitoring for ischaemia.

Spectral Edge Frequency (SEF) – Represents the frequency below which a certain percentage of the total power resides. The percentage is configurable. For example, SEF 90 = frequency below which 90% of the total power resides. SEF is a potential parameter for cerebral ischaemia or level of sedation.

Mean dominant frequency (MDF)

Peak power frequency (PPF) – The frequency with the highest power.

DELTA1 / DELTA2 – The percentage of the total power lying in the delta band

THETA1 / THETA2 – The percentage of the total power lying in the theta band

ALPHA1 / ALPHA2 – The percentage of the total power lying in the alpha band

BETA1 / BETA2 – The percentage of the total power lying in the beta band

B. USEFUL TERMS RELATED TO EEG [8,10]

Background – Refers to underlying waveform activity, symmetry, variability, reactivity (<u>not</u> muscle or eye blink artefacts), voltage and continuity.

Discharges – Waveforms with no more than three phases OR *any* waveform lasting < 0.5 sec regardless of the number of phases.

Amplitude – Refers to the height of the EEG discharges (peak to peak) and is measured in microvolts.

Very low	< 20 µV
Low	20-49 µV
Medium	50-199 µV
High amplitude	> 200 µV

Prevalence – Refers to the percent of record or epoch that incudes the observed pattern. Ranges from continuous → abundant → frequent → occasional → rare.

Duration – Refers to the typical duration of a single occurrence of the pattern (if not continuous). Very brief (<10 sec), Brief (10-59 sec), Intermediate (1-4.9 min), Long (5-60 min), Very long (>1 hour).

Symmetry – The EEG is generally symmetrical over both hemispheres. Where there is loss of symmetry there may be pathology or ischaemia in one of the hemispheres.

Generalised – any bilateral, symmetric and synchronous pattern.

Lateralised – Unilateral hemispheric or focal patterns.

Periodic discharges (PDs)

- Repeating waveforms or discharges (<0.5 sec) with relatively uniform morphology and occurring at nearly regular intervals (fig. 20A). There must be a quantifiable inter-discharge interwall between the consecutive waveforms.
- Lateralized periodic discharges (LPDs, formerly PLEDs) - EEG patterns seen in some comatose patients. May represent ictal, inter-ictal or semi-ictal patterns [11]. Highly associated with seizures. Most commonly associated with focal destructive lesions (e.g. viral encephalitis), but can also be seen with other neuronal pathologies.
- Generalised periodic discharges (GPDs, formerly GPEDs) – Associated with severe bihemispheric dysfunction of various aetiologies. Also *associated* with seizure activity.

Rhythmic – Repetition of a waveform with relatively uniform morphology and duration, and without an interval between consecutive waveforms.

Rhythmic delta activity (RDA) – Less than 4 Hz; (fig. 20B).

*Considered *rhythmic* or *periodic* if it continues for at least six cycles.

Sharpness – Descriptive term for the duration of phases of the EEG discharges (periodic discharges and spike/sharp waves only).

- Blunt – smooth or sinusoidal morphology
- Sharply contoured (typically seen with delta or theta waves)
- Sharp – 70-200 ms
- Spiky - < 70 ms

Spike wave (SW) refers to a spike or sharp wave following by a slow wave (fig 20C)

Evolving – At least two unequivocal, sequential changes in frequency, morphology, or location. To qualify, one change in frequency must last for at least three cycles. To qualify as an evolution in morphology, each different morphology must last at least three cycles. A change in amplitude alone would not qualify as evolving.

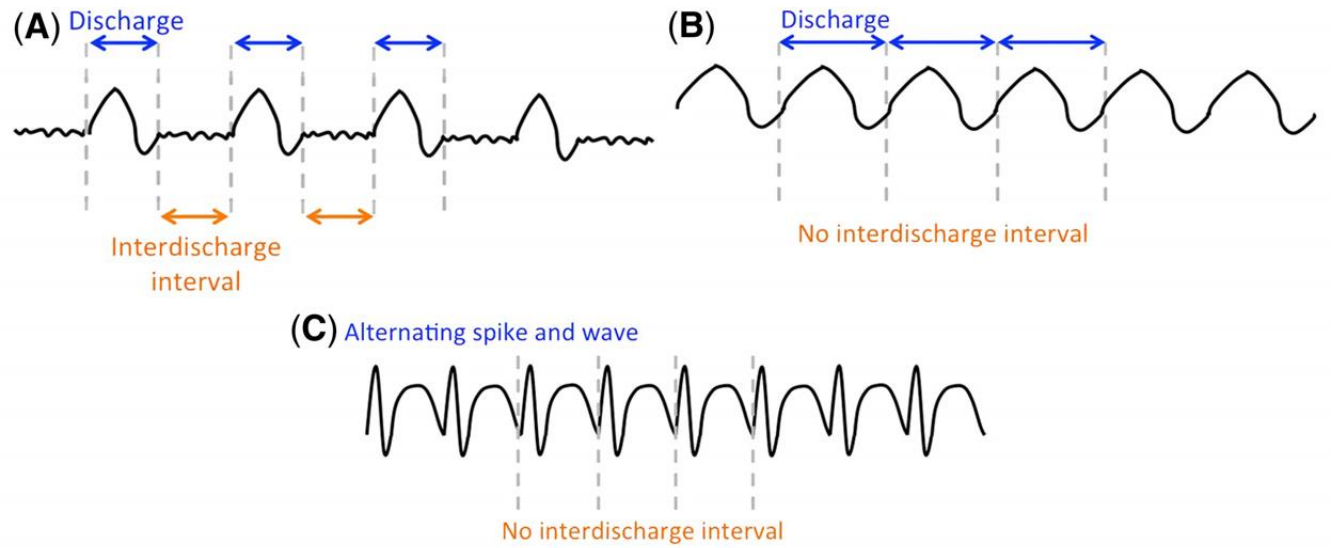

Fig 20 *See text above.*

Triphasic waves – Old term for "continuous 2-Hz generalised periodic discharges with triphasic morphology". Moderately high-amplitude and predominantly seen frontally. Seen in comatose patients, e.g. metabolic (hepatic) or toxic encephalopathies. Can be difficult to distinguish from epileptiform patterns. Have been associated with seizures but do not denote this.

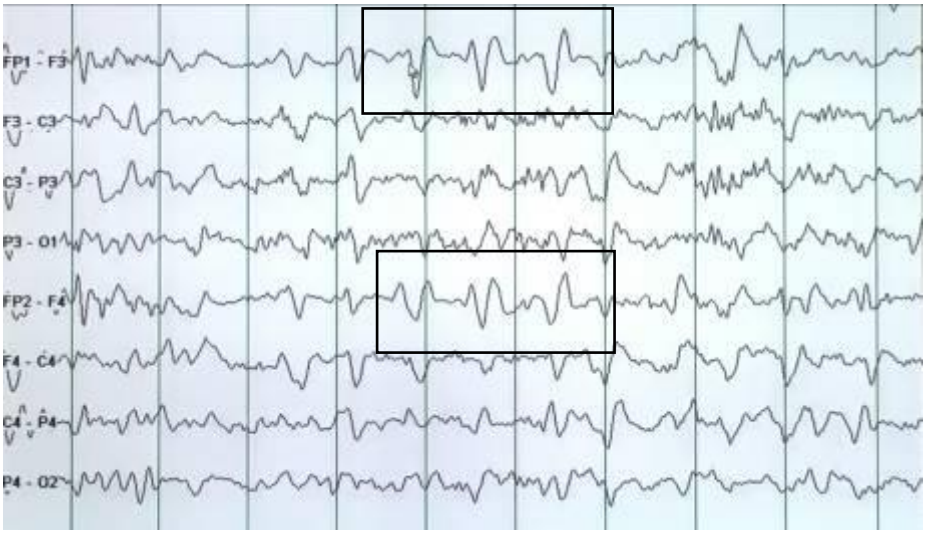

Fig. 21 *"Triphasic waves"*

Burst suppression patterns – Periodic high voltages, sharply contoured waveforms incl. spikes and polyspikes, alternating with periods of severe suppression or even isoelectricity (fig. 22). The bursts last from less than 1 sec to 10 seconds, the suppressions from 1 sec to more than 10 seconds and often correlates with the degree of cerebral dysfunction. Bursts are defined as waveforms lasting > 0.5 sec and having at least four phases.

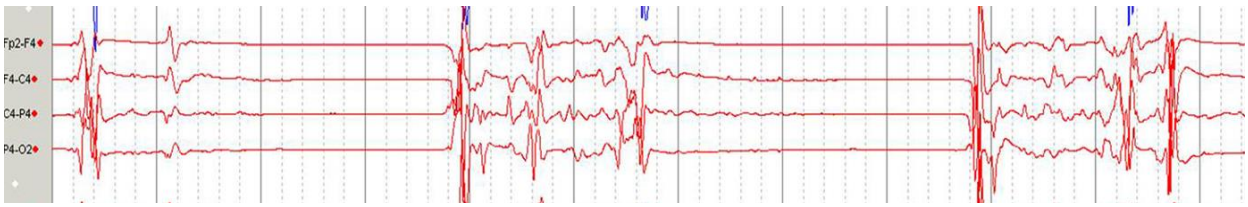

Fig. 22 *Burst suppression pattern*

Non-convulsive status epilepticus (NCSE) – "Comatose" NCSE is defined by coma accompanied by continuous or periodic epileptiform or rhythmic discharges with or without minor motor activity. I.e. EEG is crucial for diagnosis [1].

Appendix 2 – Reporting the EEG

- EEG / CSA should be reviewed and reported 2-4 times / 24-hour period. The daytime intensivist or senior registrar should at least review once after morning handover and once before night shift handover.
- Any concern or uncertainties identified should be discussed with senior staff, a neurologist or the neurosurgical team. Consider the need for supplementary investigation such as CT, MRI, DSA or a change of treatment –e.g. increased circulatory support or use of AEDs.
- When reporting the CSA and EEG it is helpful to use standardised terms and a report template. As suggested by the American Clinical Neurophysiology Society (ACNS), the nomenclature for rhythmic or periodic patterns includes **Main term #1** followed by **Main term #2**, with **modifiers** added as appropriate. **Background activity** needs to be described as well. See Appendix 3 for ACNS terminology [8].

Suggested format

Review period / epoch

Date and time

Pertinent medications

I.e. sedatives (type and doses), antiepileptic drugs, vasopressors.

Montage and technical issues

Document the montage used (A-E). Report if excessive artefacts or inadequate skin connection / impedance.

Clinical events reported incl. times

Take note from handover and nurses' report incl. seizure log and try to lock-step significant clinical events with the CSA EEG.

Background EEG activity

Comment on dominant power spectrum / band and voltage. Comment on asymmetry and focal changes.

Seizures or epileptiform discharges (e.g. rhythmic or periodic discharges)

a. Raw EEG – currently not able to retrospectively review.
b. CSA

Changes in alpha / delta ratio (ADR) or delta / total power ratio

This applies when specifically monitoring for ischaemia or DCI post aSAH. It is important that the assessments of ratios are done under similar conditions – e.g. similar level of sedation and post stimulation. Use the CSA and scroll through the timeline for trend assessment. Take note of numerical or graphical changes in the spectrogram and calculate ratios if suggestive of significant changes.

Appendix 3 – ACNS Standardised terminology [10]

ACNS Standardized Critical Care EEG Terminology: 2012 version
Reference Chart

Main term 1
G *Generalized* *- Optional : Specify frontally, midline or occipitally predominant*
L *Lateralized* *- Optional: Specify unilateral or bilateral asymmetric* *- Optional: Specify lobe(s) most involved or hemispheric*
BI *Bilateral Independent* *- Optional: Specify symmetric or asymmetric* *- Optional: Specify lobe(s) most involved or hemispheric*
Mf *Multifocal* *- Optional: Specify symmetric or asymmetric* *- Optional: Specify lobe(s) most involved or hemispheric*

Main term 2
PD *Periodic Discharges*
RDA *Rhythmic Delta Activity*
SW *Rhythmic Spike and Wave* *OR* *Rhythmic Sharp and Slow Wave* *OR* *Rhythmic Polyspike and Wave*

Plus (+) Modifier
No +
+F *Superimposed fast activity – applies to PD or RDA only*
+R *Superimposed rhythmic activity – applies to PD only*
+S *Superimposed sharp waves or spikes, or sharply contoured - applies to RDA only*
+FR *If both subtypes apply – applies to PD only*
+FS *If both subtypes apply – applies to RDA only*

Major modifiers

Prevalence	Duration	Frequency	Phases[1]	Sharpness[2]	Absolute Amplitude	Relative Amplitude[3]	Polarity[2]	Stimulus Induced	Evolution[4]
Continuous ≥90%	**Very long** ≥1h	**≥4/s**	**>3**	**Spiky** <70ms	**High** ≥200µV	**>2**	**Negative**	**SI** *Stimulus Induced*	**Evolving**
Abundant 50-89%	**Long** 5-59min	**3.5/s** **3/s**	**3**						
		2.5/s	**2**	**Sharp** 70-200ms	**Medium** 50-199µV	**≤2**	**Positive**	**Sp** *Spontaneous only*	**Fluctuating**
Frequent 10-49%	**Intermediate duration** 1-4.9min	**2/s** **1.5/s**	**1**				**Dipole**	**Unk** *Unknown*	
Occasional 1-9%	**Brief** 10-59s	**1/s** **0.5/s**		**Sharply contoured** >200ms	**Low** 20-49µV		**Unclear**		**Static**
Rare <1%	**Very brief** <10s	**<0.5/s**		**Blunt** >200ms	**Very low** <20µV				

Minor modifiers

Onset	Triphasic[5]	Lag
Sudden ≤3s	**Yes**	**A-P** *Anterior-Posterior*
Gradual >3s	**No**	**P-A** *Posterior-Anterior*
		No

NOTE 1: Applies to PD and and SW only, including the slow wave of the SW complex
NOTE 2: Applies to the predominant phase of PD and the spike or sharp component of SW only
NOTE 3: Applies to PD only
NOTE 4: Refers to frequency, location or morphology
NOTE 5: Applies to PD or SW only

Sporadic Epileptiform Discharges

Prevalence
Abundant ≥1/10s
Frequent 1/min-1/10s
Occasional 1/h-1/min
Rare <1/h

Background

Symmetry	Breach effect	PDR	Background EEG frequency	AP Gradient	Variability	Reactivity	Voltage	Stage II Sleep Transients	Continuity
Symmetric	**Present**	**Present** Specify frequency	**Delta**	**Present**	**Present**	**Present**	**Normal** ≥20µV	**Present and normal**	**Continuous**
Mild asymmetry ≤50% Amp. 0.5-1/s Freq.	**Absent**	**Absent**	**Theta**	**Absent**	**Absent**	**SIRPIDs only**	**Low** 10-20µV	**Present but abnormal**	**Nearly continuous:** ≤10% **periods of suppression** (<10µV) or **attenuation** (≥10µV but <50% of background voltage)
Marked asymmetry >50% Amp. >1/s Freq.	**Unclear**		**≥Alpha**	**Reverse**	**Unclear**	**Absent**	**Suppressed** <10µV	**Absent**	**Discontinuous:** 10-49% **periods of suppression** or **attenuation**
					Unclear				**Burst-suppression** or **Burst-attenuation:** 50-99% **periods of suppression** or **attenuation**
									Suppression