Chapter 39. Seizures

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Abstract

Seizures are common in intensive care patients generally. Cardiac surgery or post-cardiac arrest patients are at particular risk of brain injury. Seizures are an important and potentially reversible cause of prolonged unconsciousness as well as being associated with poorer ICU outcome, although the latter may be multifactorial. Cerebral metabolic rate is greatly increased during seizure activity that may lead to energetic crisis and neuronal injury. Non-convulsive seizures are particularly common in ICU patients and are therefore under-recognised. Electroencephalography (EEG) is therefore an essential ICU investigation the diagnosis of seizures, epileptiform activity and for distinguishing these from other disorders of consciousness, which may have characteristic EEG signatures. Continuous EEG is particularly sensitive for clinically occult seizures and is also helpful for managing seizures refractory to simple treatment. However EEG requires specific expertise to perform and interpret which can be a barrier to its use.

Keywords

1. Seizures
2. Status epilepticus
3. Electroencephalography
4. Cardiac surgery
5. Cardiac arrest
6. Hypoxic-Ischaemic Encephalopathy

Learning points (5)

1. Seizures are common in both in general ICU and in cardiac ICU in particular. Seizures are associated with poorer outcomes.
2. Seizures are an important reversible cause of unexplained unconsciousness.
3. Many seizures in the critically ill are non-convulsive and therefore may go unrecognised.
4. Electroencephalography is required for the diagnosis of seizures and a range of seizure-like pathologies.
5. EEG equipment is complex to set up and interpretation requires specialist expertise.

1.   The burden and characteristics of seizures in intensive care

Seizures are common but under-recognised in the Intensive Care Unit (ICU). Sub-clinical electrographic seizure activity is common in neurosciences patients but may also occur in critically ill patients without a past history of seizure. Most studies show

a prevalence of seizures of 15-40% and identify the presence of seizures and status epilepticus as independent predictor of poorer outcome. To what extent this represents a modifiable pathology or an epiphenomenon of worse underlying neurological injury is uncertain. However prolonged seizure activity is associated with neuronal energetic failure and injury. Therefore recognition and treatment of seizures is important. Furthermore, seizures are also an important reversible cause of unconsciousness and should be considered in the differential diagnosis of a patient who does not wake appropriately from sedation / anaesthesia.

Clinically apparent seizures are due to focal or global disorganized brain electrical activity and may be manifest as changes in behavior / level of consciousness or abnormal movements. However in sedated or critically ill patients, none of these findings may be clinically obvious or apparent. The diagnosis of true seizures is complicated by other types of abnormal movements that may be seen in intensive care patients (such as shivering, myoclonus, tremor, emergence from neuromuscular blockade). A systematic approach to distinguishing seizures from non-seizure movements is helpful (Figure 1).

Most clinical seizures are self-limiting. Patients will regain consciousness although a post-ictal period of altered mental state is common due to neurotransmitter depletion, changes in receptor concentration / inhibition or altered cerebral blood flow. Sometimes clinical seizures may be prolonged or repeated. Status epilepticus (SE) is defined as 'an acute epileptic condition characterized by continuous generalized convulsive seizures for at least five minutes, or by two seizures without full recovery of consciousness between them'. Electrographic seizure activity may also occur without movement, and this is particularly important in ICU patients. Such non-convulsive seizures (NCS) are a more elusive diagnosis that is frequently unrecognised. Its incidence after cardiac surgery is probably particularly high due to exposure of the patient to some degree of sub-clinical embolic phenomena, metabolic derangement, hypotension, or pro-convulsive drugs. Non-convulsive SE (NCSE) is further defined as an electrographic diagnosis of "continuous or intermittent ictal discharges without the patient regaining consciousness, and no overt clinical signs of convulsive activity". NCSE is often suspected in patients who were in convulsive SE and subsequently do not fully regain consciousness. Hence, if coma persists after SE, EEG is necessary to identify the cause of unconsciousness (NCSE or a post-ictal state). However it may occur de novo in ICU patients and this represents a particular diagnostic challenge.

NCS have been reported in up to to 37% of critically ill patients undergoing cEEG monitoring. The highest proportion of NCS is seen in patients who underwent emergency EEGs for altered mental status and clinically suspected NCSE. Studies prospectively performing EEGs in unselected comatose patients without clinical signs of seizures found either NCS or NCSE in up to 8% of cases.

2.   Electroencephalography

Given the diagnostic difficulties presented by critically ill patients, the unequivocal detection / exclusion of seizures often requires electroencephalography (EEG). EEG is a technique that measures the spatial distribution of voltage fields on the scalp and their variation over time. The origin of this activity is thought due to reflect the

fluctuating sum of excitatory and inhibitory postsynaptic potentials that arise primarily from apical dendrites of pyramidal cells in the outer layer of the cerebral cortex under the input from subcortical structures.

The potentials recorded by the EEG are very small (of the order of microvolts) and are therefore easily contaminated by electrical noise and other artefacts such as the electromyogram when trying to record from areas with larger underlying muscles. Furthermore, EEG changes may be relatively localised meaning that an array of electrodes is needed to cover the whole scalp adequately. Finally, seizures may occur intermittently and so continuous recordings (cEEG) over 12-24 hours or even longer are needed to realise the highest diagnostic sensitivity. Such considerations make recording a clinically useful EEG much more complex and involved than, say, an ECG. The American Clinical Neurophysiology Society has published recommendation for recording ICU cEEG. Equipment, length of recording and electrodes montages should meet the technical standards defined in these consensus statements (Figure 2).

Even in the absence of seizures, the EEG shows stereotypical changes with consciousness, which can be diagnostically useful. EEG activity can be classified by frequency, amplitude, distribution or location, symmetry, synchrony, reactivity (to external stimulation), morphology, rhythmicity and regulation. The signal frequencies detected by a standard clinical EEG are divided into four standard frequency ranges or "bands" : alpha (8 to <13 Hz, usually occipital), beta (>13 to 25 Hz, usually frontal and central), theta (4 to <8 Hz, usually central or diffuse) and delta (<4Hz, focal or diffuse).

The most important EEG pattern of wakefulness is a posterior dominant rhythm (PDR). The PDR is located predominantly at the occipital poles but becomes anterior as the patient becomes drowsy (normal sleep). This activity is normally in the alpha range and is symmetrical. Slower rhythms such as generalised continuous delta are always abnormal and are usually associated with diffuse or multifocal cortical injury or metabolic derangement. Intermittent slowing, such as Intermittent Rhythmic Delta Activity (IRDA) and Frontal IRDA (FIRDA) or Occipital IRDA (OIRDA) are thought to be caused by dysfunction in subcortical centres influencing cortical activation and may represent a manifestation of a more generalised process not limited to the frontal or occipital lobes.

Reactivity refers to a clinical (electromyographic activity, respiratory pattern change) and/or EEG response (increased continuity, amplitude reduction, frequency change) to external stimulation (pain, passive eyes opening, auditory stimuli). In cardiac arrest patients, a nonreactive EEG background after rewarming is associated with poor neurological outcome. However the association is complex: this pattern is still compatible with good recovery when observed during therapeutic hypothermia and patients with myoclonus and no EEG reactivity within 72 h from cardiac arrest subsequently have occasionally had good outcomes.

Electrographic seizures are characterized by repetitive or rhythmic focal or generalised epileptiform discharges at greater than 3 Hz, and lasting more than 10 seconds or at less than 3 Hz but with clear evolution in frequency, location, waveform or field or clinical manifestation. They usually resolve or improve after

administration of rapid-acting intravenous antiepileptic drugs such as benzodiazepines. The electrographic features of SE are highly variable including rhythmic, generalised and symmetric spike-and-waves or polyspikes and waves at 2 to 3.5 Hz, or atypical spike and wave with lower frequency and less symmetry, or multiple spike-and-wave, or high-voltage, repetitive, rhythmic (focal or generalized) delta activity with interspersed spikes, sharp waves, or sharp components. (Figure 3)

If continuous EEG is not available, multiple intermittent serial recordings are desirable as seizures may otherwise be missed, especially as high-dose anaesthetics are weaned. Whilst the EEG does not change significantly at body temperatures between 32 to 34°C, sedative drugs commonly used during therapeutic hypothermia can markedly affect the EEG background.

Regular electrode maintenance, scalp inspections, keeping the scalp dry, and reducing pressure on scalp from the electrodes is important to avoid scalp injuries. Sweating and scalp breakdown are more common in patients with fever, sepsis and prolonged systemic disease requiring frequent electrodes changes. Hypothermia presents a theoretically increased risk of coagulopathy. However, scalp breakdown with bleeding or infection has not been reported among post-arrest patients treated with therapeutic hypothermia (even up to several days of cEEG with either disposable or reusable versions of disc electrodes (Figure 2).

The EEG is typically recorded for later off-line analysis by a neurophysiologist. To this end the use of simultaneous video recording is recommended to identify sources of artefact and must be correctly repositioned when the staff moves either the patient or the camera of the EEG acquisition unit. Routine ICU care such as physiotherapy, suctioning, and oral care can create rhythmic artefacts that mimic electrographic seizures but are easily identified with video analysis.

EEG electrode technology is sophisticated: The low voltages involved mean that very low electrical impedances are required. Poor contact renders the electrodes susceptible to electrical interference. Electrical noise from intravenous pumps, electrical beds, dialysis machines, and other medical devices can obscure the EEG recording and requires proper identification and trouble-shooting.

Specialised hardware and software increases the utility of cEEG for monitoring at the bedside. Options include the ability to enter nursing notes, pushbuttons for seizures and other clinical events, software to integrate physiologic data, and quantitative EEG software for graphical display of quantitative EEG trends.

3. Causes and prognostic significance of seizures in intensive care

The dominant aetiology and prognostic significance of seizures in the cardiothoracic intensive care unit varies from patient to patient. Three distinct patient populations are of particular importance in the cardiac ICU; those who are post cardiac surgery (where embolic phenomena are likely to be important), patients successfully resuscitated from cardiac arrest (where hypoxic-ischaemic injury dominates) and those patients suffering from general critical illness.

3.1. Post-cardiac surgery

Postoperative seizures are known to complicate cardiac surgery with an incidence up to 7% and recurrence rates of around 50%. This is almost certainly an underestimate, with NCS probably frequently missed and not necessarily benign. The manifestations and causes of seizures in the context of cardiac surgery are diverse, as are the expected outcomes. Both convulsive and non-convulsive seizures may contribute to prolonged reduced levels of consciousness, an increased length of stay in the ICU, and possible increase morbidity and mortality. Furthermore, convulsive seizures lead to cardiovascular strain and metabolic derangement in patients already in a fragile state. Perioperative seizures may be caused by thromboembolic ischaemic stroke, cerebral air embolism, antibiotic toxicity or other perioperative drugs administration such as tranexamic acid (TXA).

Cardiac surgery is associated with the risk of injury to the central and peripheral nervous system with open-chamber procedures, including valve and aortic repairs and cardiac transplants, having a higher incidence of complications than does coronary artery bypass graft surgery. Perioperative seizures are frequently associated with thromboembolic ischaemic stroke or cerebral air embolism following open-chamber and valve surgery but antibiotic toxicity or other perioperative drugs administration such as tranexamic acid (TXA) may also be implicated.

Several risk factors have been directly associated with the presence of postoperative seizures. The combination between TXA administration, especially high doses (> 80 mg/kg) and open-chamber surgery has been confirmed in several studies as an important perioperative factor. Higher preoperative creatinine (>120 μmol/L), thoracic aortic surgery and early seizures onset (<4 hours) were associated with recurrent seizures (RS) after cardiac surgery have also been found to increase risk of seizures. Despite longer ICU stay and mechanical ventilation duration of patients with RS compared to those with isolated seizures, the presence of RS per se was not associated with significantly increased long-term morbidity or mortality.

3.2. Post-cardiac arrest

Clinical seizures occur in up to 40% of patients following cardiac arrest, mostly in the first 3 to 5 days. Seizures most commonly present as diffuse or multifocal myoclonus or focal and generalized tonic-clonic seizures, contribute to decreased level of consciousness and have implications for prognosis.

Non-convulsive seizures are prevalent and often present as NCSE. Therefore EEG is required for the diagnosis. Electrographic seizures may be focal, multifocal or generalized: they may arise from a continuous background, a suppressed background, or a burst suppression pattern. They may be of any frequency, and typically shows evolution of frequency, voltage, morphology, and/or field.

NCSE after cardiac arrest is very difficult to treat, and it is not clear if treatment improves neurologic outcome and mortality. However it is common: Post-arrest patients treated with therapeutic hypothermia and receiving routine EEG or cEEG monitoring have a prevalence of NCSE/SE around 30%. Clinical seizures typically begin within the first 2 days following cardiac arrest but it is not uncommon to see earlier electrographic seizures on the EEG. Several studies have shown that initial electrographic seizures are typically not associated with any clinical correlate of myoclonus or other motor seizure types by day 2 to 3.

Myoclonus can also occur in patients with hypoxic injury of the brain and may be either acute or chronic. Acute post-hypoxic myoclonic status epilepticus (MSE), occurs soon after a hypoxic insult and is a clinical diagnosis consisting of unrelenting diffuse myoclonus involving the face, limbs and torso that are often precipitated by stimulation. It is strongly associated with poor outcome if onset is during the first 24 hours post-arrest, and this association remains true with therapeutic hypothermia, although the onset may be delayed 3 to 5 days after cardiac arrest. It is important to appreciate that although rare (less than 5%) there have been reports of survivors of MSE with good cognitive outcome. Most of these patients were treated with at least three to four antiepileptic drugs (including high-dose anaesthetics), had preserved brainstem reflexes, intact cortical somatosensory evoked potentials responses and reactive EEG background.

Sub-cortical structures are involved in MSE and therefore the EEG does not show the same electrographic features. Instead the EEG may show generalised periodic discharges (GPDs) and/or a burst suppression pattern. A brainstem origin of post-anoxic myoclonus may be suspected when there is sequential activation of muscles innervated by cranial nerves (reticular reflex myoclonus) and characterised by unspecific EEG changes (non-epileptiform patterns such as diffuse alpha activity). By contrast, cortical action-reflex myoclonus instead presents with epileptiform discharges and/or polyspikes and typically (but not necessarily) affects a few muscles in a localised distribution.

MSE may be confused with chronic post hypoxic myoclonus (Lance-Adams Syndrome, LAS) LAS is a distinct entity that may also appear after a period of cerebral hypoxia in patients with a more prolonged coma or period of sedation. In contrast to MSE, LAS develops later; days or weeks after the initial hypoxic event and does not respond to anticonvulsant treatment. Crucially however such patients regain consciousness, but may exhibit muscle jerks affecting face, trunk or limbs often provoked by sensory stimuli and strikingly elicited by the willed voluntary action. LAS is rare, but its differentiation from MSE is important to avoid incorrect prognostication and for the institution of timely rehabilitation.

3.3. General intensive care considerations

In the cardiac ICU, anoxic brain injury is a common cause of severe encephalopathy as well as seizures, and ischaemic or haemorrhagic stroke must be suspected in patients who had cardiac surgery and develop postoperative seizures or SE. However, there are many possible causes for seizures in the critically ill and a single cause may often never be identified.

Sepsis is the most frequent cause of altered mental status in the general ICU as sepsis-related encephalopathy has been associated with neuronal damage, mitochondrial and endothelial injury, and disturbances in neurotransmission. Metabolic alterations due to hepatic and/or renal dysfunction can lead to a background of encephalopathy.

Other primary systemic diseases can present as altered mental status and are associated with seizures (such as posterior-reversible leukoencephalopathy

secondary to malignant hypertension, eclampsia, metabolic and electrolyte disturbances, embolic cerebral infarcts or mycotic aneurysms from endocarditis, central nervous system vasculitis from an underlying autoimmune disease such as polyarteritis nodosa, systemic lupus erythematosus, and Sjøgren's syndrome, paraneoplastic syndromes and limbic encephalitis).

Transplant patients in the post-surgical ICU are another population at risk for altered mental status and seizures. Seizures in these patients are often multifactorial with causes including severe metabolic derangements, toxicity from immunosuppressive therapy, and other post-operative complications. Central nervous system infections are an important cause of seizures, and this is of particular significance in patients who are immunosuppressed.

4.   Management of seizures

SE is an emergency and must be treated aggressively and early as the longer an episode of SE persists, the more refractory the patient will become to treatment. Also, clinically evident seizures should be treated immediately without waiting for the EEG but cEEG should ideally be started if a patient requires continuous neuromuscular blockade after initial treatment with antiepileptic drugs (AEDs).

It is important to assess and manage patients' airway because apnoea or airway soiling can occur with generalised seizures and intubation may be required. The use of short-acting paralytics is preferable for intubation so that on-going seizure activity will not be masked. Autonomic instability may accompany seizures and cardiovascular support may be required.

Once SE has become established, rapid administration of intravenous therapy is required to control seizures. Whilst any benzodiazepine may be effective, lorazepam is the drug of choice as it has a long redistribution half-life (3-10 hours) reducing the likelihood of rebound seizures. If the first bolus (usually 2-4 mg in adults) fails, then a second bolus can be given followed by a loading dose of phenytoin (20 mg/kg infused at a maximum rate of 50 mg/min).

A significant minority of patients with SE may be refractory to adequate doses of two AEDs. If SE has not responded to this first line treatment and lasts 60-90 minutes, patients will risk physiological compromise, neuronal damage and increasing drug resistance. The patient should be anaesthetised and transferred to ICU to stop seizure activity. Propofol is likely to be the most familiar agent and is an effective anticonvulsant, although midazolam may be employed. More rarely barbiturates such as thiopentone may be required but specialist advice should be sought.

Laboratory tests to pursue include; blood glucose, full blood count, metabolic profile, liver function test, magnesium, phosphate, urine toxicology, serum ethanol levels, troponin, creatine kinase (CK), urinalysis, urine and blood cultures and AEDs levels. If vitamin B1 deficiency is suspected (history of dietary insufficiency/inadequacy), administer intravenous thiamine before giving glucose to avoid precipitation of Wernicke's encephalopathy. Acid-base alterations and hyperthermia are common during SE and should be treated as soon as possible. A degree of rhabdomyolysis is common and can lead to acute kidney injury or even renal failure. Depending on CK

levels, urine output should be maintained and alkalinisation with intravenous sodium bicarbonate considered. If CK is severely elevated, then renal replacement should be commenced from the outset to remove circulating myoglobin although this is rare in the context of seizures alone.

A CT head must be performed once the patients is stabilised and clinically apparent seizures have stopped. If the aetiology is unknown and the CT head is negative, an MRI head should be performed. In patients with SE of unknown origin, a lumbar puncture is indicated and empirical cover with CNS-penetrating antibiotics and antivirals should be considered while CSF analysis, microscopy, culture and PCR studies are being obtained. This is particularly important in febrile or immunocompromised patients.

Phenytoin has gained widespread acceptance as a first-line anti-epileptic drug (AED) due to its availability, intravenous formulation, effectiveness and familiarity. However its use in cardiac intensive care unit must be carefully considered. In patients with post-cardiac arrest SE and after cardiac surgery, special attention should be given to the risk of cardiac dysrhythmia (bradycardia occasionally progressing to heart block) or hypotension in this very susceptible population. In addition, phenytoin and warfarin have complex and poorly understood interactions that vary with cytochrome p450 genetic polymorphisms, which may limit its use in patients requiring anticoagulation.

Phenytoin has a long half-life and dosing must be carefully considered. It is also strongly protein-bound and levels should be checked regularly and be adjusted for serum albumin levels to avoid inadvertent overdose;

$$[\text{Corrected phenytoin}]=[\text{Measured phenytoin}]/(0.29 \times \text{Albumin}+0.1)$$

For CrCl <10 ml/min or in renal replacement therapy use;

$$[\text{Corrected phenytoin}]=[\text{Measured phenytoin}]/(0.1 \times \text{Albumin}+0.1)].$$

Valproate is an alternative to phenytoin. It is not associated with significant cardiac side effects, can be loaded quickly intravenously (30 to 60 mg/kg infused over 30 to 60 minutes followed by maintenance 15 to 20 mg/kg in divided doses) and is very well tolerated. It is hepatically metabolised but is safe even in patients with mild-to-moderate liver dysfunction. Valproate has been demonstrated to be at least as effective as phenytoin in treating SE. A rare side effect that must be suspected in prolonged recovery after seizures in patients loaded with valproate is hyperammonemia, an idiosyncratic metabolic response. Another option that is gaining popularity in all ICU settings is levetiracetam, due to its minimal drug-drug interactions and unchanged excretion such as hepatic disease does not preclude its use. The dose should be adjusted in renal failure to allow for decreased clearance. A starting dose of 500 mg twice a day is thought to achieve therapeutic levels in most patients although this may rapidly be increased over a few days to 1500 mg twice daily if required. Valproate, benzodiazepines, and levetiracetam have shown some efficacy in reducing post-anoxic myoclonus.

In the rare situations where moderate or high-doses barbiturates have been used, any neurological prognostication should be delayed because of their prolonged sedative effect. A randomized trial of refractory SE found a median duration of intubation of 14 days in survivors in the thiopental or pentobarbital infusion arm, versus 4 days for survivors in the propofol arm with no differences in mortality. Laboratory barbiturate assay is possible but not locally available in most hospitals.

Once the patient has been sedated, EEG (ideally cEEG) is helpful in establishing on-going AED effectiveness, whether a second AED is needed and what maintenance therapy will be required after sedation withdrawal. Once all seizure activity has stopped for more than 24 hours and provided there are adequate blood levels of AEDs, then the anaesthetic can be slowly weaned. Should SE recur, other drugs may be tried, but good clinical trials are lacking to date.

5.   Seizures vs epileptiform activity: To treat or not to treat?

In addition to definitive seizure activity, the EEG may also reveal abnormal epilepsy-like activity. Although strictly speaking such epileptiform discharges are not seizures, they may be important as their presence is highly associated with both convulsive and non-convulsive seizures. Periodic discharges (PDs) are stereotyped repetitive discharges with diverse electrographic morphology that occur at regular intervals and may be localised to one hemisphere (Lateralised PDs, LPDs) (Figure 2) or bilateral (Bilateral Independent PDs, BIPDs and Generalised PDs, GPDs).

The clinical significance and management of epileptiform activity is controversial, especially in comatose patients. In the general ICU more than 20% of patients are estimated to suffer electrographic seizures or periodic discharges (PDs). The presence of PDs is associated with a higher incidence of death or severe disability at hospital discharge (20), however, the clinical significance of PDs and their association with underlying structural injury is still controversial. Whether epileptiform activity is a true inter-ictal pattern or instead simply represents a predisposition to seizures is unclear. Evidence for improvement in outcome from treatment with AEDs is lacking. Nevertheless these patterns are pathological: the clinical significance and management of such epileptiform findings should therefore be considered on a case-by-case basis.

Figures

Figure 1. The diagnosis and initial management of seizures after cardiac surgery (after Hunter And Young, with permission (10)).
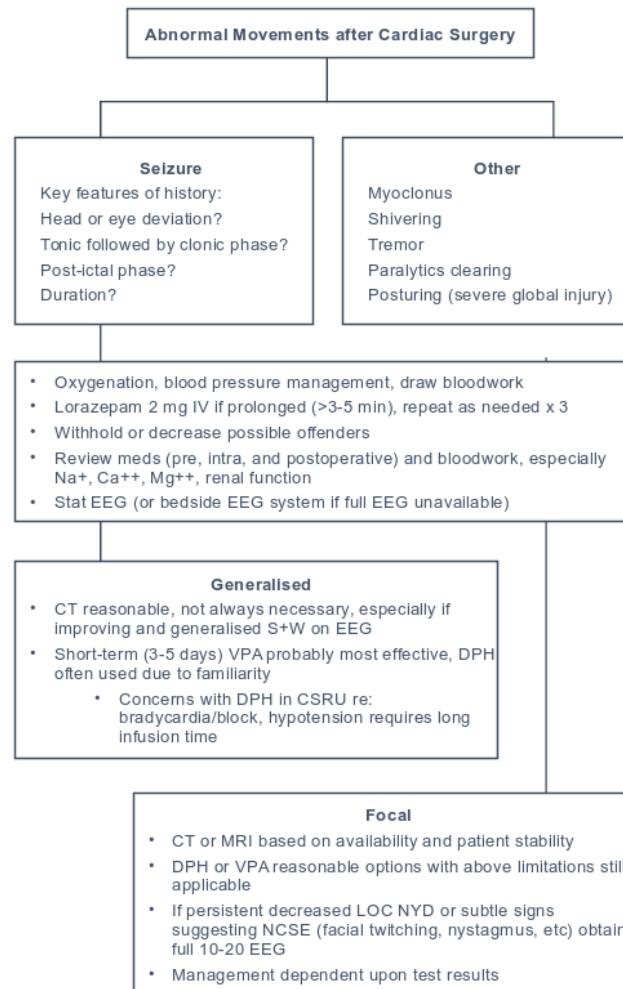
Figure 2. Left panel: typical EEG scalp electrodes for clinical use. Right panel: International 10-20 system for standard scalp electrode placement. The distances between adjacent electrodes are either 10% or 20% of the total anterior/posterior or left/right distance. Left and right are distinguished by odd and even numbers respectively. Each channel of the EEG is obtained by measuring the voltage between adjacent electrodes (bipolar montage, such as in figure 2) or of individual electrodes compared to a reference electrode or combination of electrodes (referential montage).
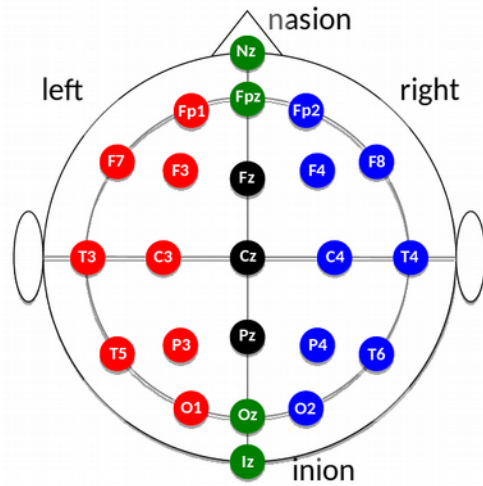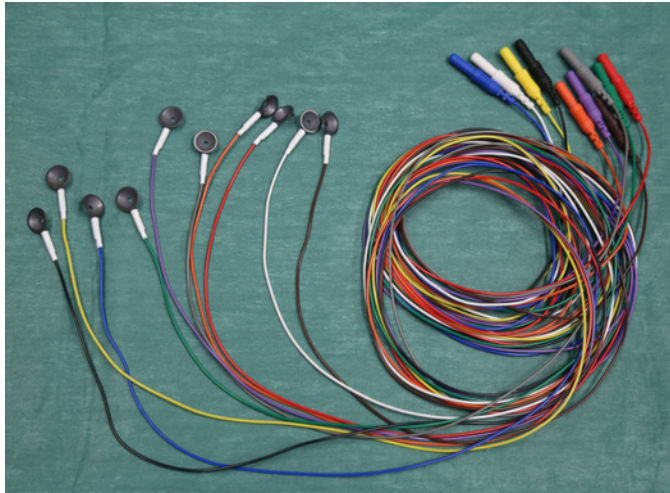
Figure 3. Examples of EEGs from ICU. Top left: normal wakeful EEG- high frequencies with alpha wave activity dominant in posterior / occipital channels. Top right: Encephalopathic EEG illustrating prominent slow (delta-wave) activity. Bottom left: Generalised seizure activity with sharp spikes and waves in all channels. Bottom right: LPDs- periodic spike discharges over the left hemisphere (odd numbered channels).
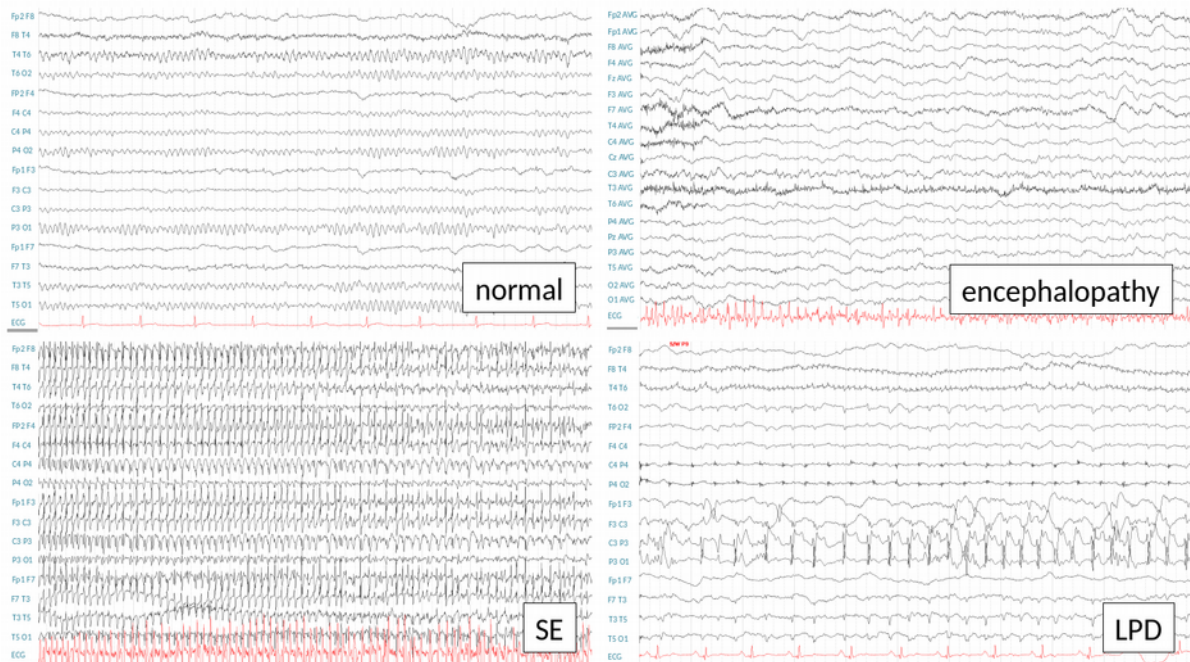
Further reading

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MCQs (5)

1. Any seizure lasting longer than what period of time is defined as status epilepticus?
   - A)   1 minute
   - B)   5 minutes
   - C)   30 minutes
   - D)   60 minutes

2. When we are awake and very alert, the EEG normally shows:
   - A)   Beta waves (13-30 Hz)
   - B)   Theta waves (5-7 Hz)
   - C)   Alpha waves (8-12 Hz)
   - D)   Delta waves (1-4 Hz)

3. Which one of the following cardiac surgery risk factors would be more likely present in patients with postoperative seizures?
   - A)   High doses of tranexamic acid
   - B)   Open-chamber procedures
   - C)   Stroke
   - D)   All of the above

4. Which one of the following regarding neurological outcome after cardiac arrest is true?
   - A)   The Lance Adams syndrome is associated with a good neurological outcome
   - B)   It can be predicted at 24 hours in patients who have undergone therapeutic hypothermia
   - C)   Targeted-temperature management to 36 C is not associated with better neurological outcome
   - D)   EEG/cEEG should be recorded only in patients with clinical signs of seizures/SE

5. Which of the following is the drug of first choice in a patient with generalized convulsive status epilepticus?
   - A)   Propofol
   - B)   Phenytoin
   - C)   Pentobarbital
   - D)   Lorazepam