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Modernising Medical Careers

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WESLEYAN MEDICAL SICKNESS

Electroencephalography for the general physician

Introduction

Junior doctors in general medicine are often asked to request electroencephalograms or electroencephalographs (EEGs) on patients in their care. For many, however, the indications for an EEG, the nature of the test itself, and how the results obtained should influence patient management are poorly understood. Clinical neurophysiology is not usually included in the undergraduate curriculum, and under current training arrangements formal education in EEG will not occur unless doctors choose to specialize in neurology or clinical neurophysiology.

This article provides an introduction to the basis of the EEG, and gives some understanding and guidance as to when, and under what circumstances, the EEG is helpful and when it is not.

What is an EEG?

An EEG is a recording of electric field potentials generated by cerebral neuronal activity. More specifically, potentials arising as a result of thousands of summated pyramidal cell inhibitory and excitatory postsynaptic potentials, together with some intrinsic currents, are recorded as voltage differences between electrodes placed on the scalp, which are then amplified and plotted over time to produce the typical EEG trace (*Figure 1*). A routine recording lasts approximately 20 minutes but, depending on the clinical requirements, can be extended to include sleep, or ambulatory recordings.

Since the first animal studies by Richard Caton in 1875, and the first human studies by Hans Berger in the 1920s (Haas, 2003), the equipment and recording techniques have evolved, with paper records now replaced by digital recordings with high quality amplifiers and computerized data displays.

It is now standard practice to record with a minimum of 21 electrodes in adults, placed on specific locations on the scalp using bony landmarks as a guide (known as the international 10–20 system) (*Figure 2*). The voltage from each electrode may be recorded with reference to a specified reference potential (referential recording) or with reference to a sequence of adjacent electrodes (bipolar recording). The results are then displayed in various orderly arrangements of electrodes called montages. For simplicity, all the EEG examples in this article are dis-

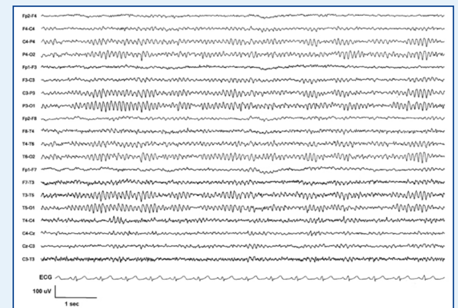

Figure 1. Normal waking adult electroencephalogram (EEG). Bipolar montage.

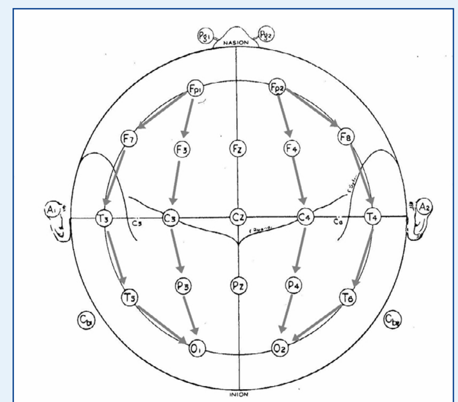

Figure 2. Diagrammatic representation of the international 10–20 system (modified from Jasper, 1958). The arrows represent how the channels are linked in the typical bipolar montage used in this article.

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played in a bipolar montage, with the electrodes linked as illustrated in *Figure 2*.

Types of EEGs

Most routine outpatient recordings now include a synchronized digital video recording. There are various types of EEG recordings, requested in different settings (*Table 1*). Discussion in this article will be limited to the use of routine video EEG.

Basic EEG nomenclature

The typical EEG report comprises a factual description of the trace followed by a clinical comment or conclusion. The following are some of the common terms used.

Cortical rhythms are classified according to their frequency range:

<4 Hz	Delta (or slow)
4 – <8 Hz	Theta
8–13 Hz	Alpha
14–40 Hz	Beta (or fast)
>40 Hz	Gamma

A spike is a suddenly appearing (paroxysmal) sharp component of <70 ms duration standing out from the background activity. A sharp wave is a broader sharp component lasting 70–200 ms.

Comments

In general, in the alert awake adult, slow activity is abnormal, and indicates underlying cerebral dysfunction, but is not specific for aetiology. Theta activity is a much less specific finding and interpretation is highly dependent on the clinical context. In patients with the appropriate clinical history, rhythmic bursts of slow or theta activity may sometimes represent ictal (seizure) activity (e.g. temporal intermittent rhythmic delta activity, or rhythmic theta in mesial temporal lobe seizures as a result of hippocampal sclerosis).

Although spikes and sharp waves are often seen as part of epileptiform discharges, they need not always represent pathology, and may be seen in some patterns recognized as normal or benign variants (e.g. small sharp spikes in sleep), or variants with doubtful or uncertain association with epilepsy (e.g. 6 Hz spike and wave, wicket spikes, 6–7 and 14 Hz positive spikes) (Westmoreland, 2003). The reporting electrophysiologist should normally comment on the significance of the findings, and the clinician should take care not to over-interpret factual reports as representing confirmation of an epileptic tendency. If there is uncertainty, the reporting electrophysiologist should be contacted and the findings discussed.

Table 1. Types of EEG recordings

Type of EEG recording	Indication for request
Routine inpatient or outpatient video EEG	Episodes of loss of consciousness, suspected seizures
	Syndromic classification of epilepsy
	Impaired responsiveness, suspected subclinical seizures
Sleep recording (video EEG with the patient either sleep-deprived or given a sedative)	High clinical suspicion of epilepsy, but routine EEG either normal or non-diagnostic
Prolonged video EEG (day case)	As part of presurgical workup
	Suspected non-epileptic attacks
Ambulatory EEG (no video, the patient presses an event button and keeps a diary)	Multiple recurrent mild attacks, or attacks with aura/ 'warning' of uncertain aetiology
Video telemetry (using routine surface electrodes)	Diagnosis of epilepsy type/syndrome
	Presurgical workup (intractable epilepsy)
	Suspected non-epileptic attacks
Multiple sleep latency tests and polysomnography (often only in specialized sleep laboratories)	Suspected rapid eye movement or non-rapid eye movement sleep disorders, obstructive sleep apnoea
Procedures in epilepsy surgery centres only: (i) Video telemetry using intracranial electrodes (e.g. foramen ovale, depth, or subdural electrodes)	Presurgical workup
(ii) Electrocorticography (EEG recorded using grids of electrodes placed directly on the cortex)	
(iii) Magnetoencephalography	

EEG = electroencephalogram

Factors affecting the normal EEG

Age

The normal EEG alters with maturation until adulthood; this is of particular importance when studying premature babies and neonates, where correct information regarding gestational age is important for accurate interpretation of the findings. Detailed discussion of the use of EEG in paediatrics would be inappropriate here in an introductory article, because familiarity with the normal maturational changes in childhood would be required, and for that the interested reader should refer to the standard paediatric neurophysiology texts.

State of arousal

The normal posterior alpha rhythm of the relaxed alert state is replaced by slower rhythms in drowsiness, and progression to the deeper stages of sleep is accompanied by various sleep phenomena and further slowing of the background activities. Sedation produces similar background slowing, and in intensive care patients, the residual effects of intravenous sedation may interfere with interpretation of the EEG, if the record is taken too early after sedation is withdrawn.

In what situations is the EEG useful?

Patients with altered mental status

The EEG is helpful in differentiating altered mental status arising from an acute psychosis, in which the EEG is typically within normal limits, from that caused by an encephalopathy or encephalitis which is associated with diffuse slowing of cortical rhythms, with or without focal abnormalities. It is also helpful in identifying cases where impairment of consciousness is caused by non-convulsive status epilepticus (*Figure 3*). Normal alpha activity during an attack of apparent unresponsiveness would suggest a non-epileptic/

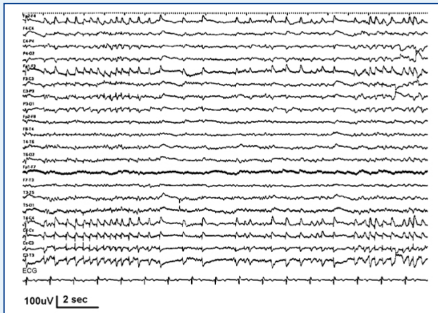


Figure 3. Non-convulsive status epilepticus in an unresponsive 69-year-old man in the intensive care unit following a cardiac arrest. Note the evolving nature of the discharges.

Table 2. Electroencephalogram patterns and commonly associated conditions

Pattern	Clinical associations	
Triphasic waves	Metabolic encephalopathies (e.g. hepatic or uraemic encephalopathy, electrolyte abnormalities, anoxia), lithium intoxication	
Periodic complexes	Generalized	Creutzfeldt–Jakob disease, subacute spongiform panencephalitis, severe anoxia
	Lateralized	Acute or subacute destructive lesions (including cerebral infarcts), focal epileptogenic lesions, bitemporal periodic epileptiform discharges in herpes simplex virus encephalitis
Frontal intermittent rhythmic delta activity	Initially described in association with deep midline lesions, raised intracranial pressure, or subcortical dysfunction, but is now recognized as being much less specific, being more often found as a non-specific finding in diffuse encephalopathies	

psychogenic cause (provided the history does not suggest paralysis as a result of neuromuscular disease, or a brainstem stroke, e.g. locked-in syndrome). An EEG containing an unusual amount of generalized fast activity in a poorly responsive or comatose patient would raise the possibility of benzodiazepine or barbiturate poisoning.

Because it reflects neuronal activity, the EEG is helpful in providing some indication of the severity of cerebral dysfunction. However, it is not helpful in determining aetiology, since there are only a limited number of ways the brain can react to injury; thus similar electrographic changes can arise as a result of insults of various aetiologies. Nevertheless, some patterns, when present, may be helpful in pointing towards a broad aetiological category (*Table 2*).

Although herpes simplex virus (HSV) encephalitis is characteristically associated with periodic lateralized epileptiform discharges (PLEDs) over the temporal regions, this typical abnormality may not appear if the patient has received early treatment with acyclovir, in which case less specific temporal, frontal or generalized slowing may occur. PLEDS are not specific for aetiology (*Table 2*) and may also be seen in cerebral infarction (*Figure 4*), again emphasizing the importance of the clinical context when interpreting EEG findings.

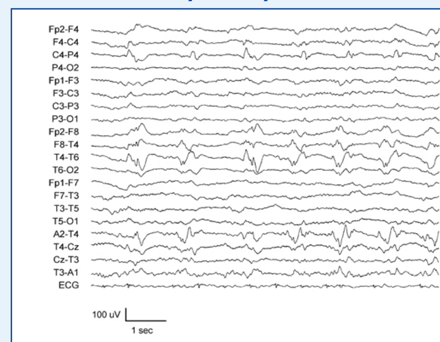
In patients with psychiatric disorders, it is important to note that a variety of medications can alter the EEG. Sedative or recreational drugs, and some antipsychotic drugs, particularly lithium and clozapine, may give rise to slowing and epileptiform changes. It is therefore important that full and accurate information on medication is always provided with the EEG request.

Unresponsive patients on the intensive care unit

The EEG can provide helpful prognostication in patients who have suffered a severe cerebral anoxic insult. Provided all other factors likely to be contributing to the EEG pattern (such as metabolic abnormalities, sedation, hypothermia, sepsis) have been excluded, certain patterns are generally accepted as associated with a poor outcome (Chatrian and Turella, 2003; Hui et al, 2005) (*Table 3*).

It is important that all the relevant clinical information should be provided when requesting an EEG on a patient on the intensive care unit, since insufficient clinical information may lead to wrong conclusions being drawn. For example, a burst-suppression pattern carries a quite different prognosis when associated with coma following cerebral anoxia, compared with coma owing to drug intoxication, severe hypothermia or anaesthetic agents. Spontaneous fluctuation in cerebral activity or evidence of responsiveness of the cerebral activity to external stimuli is a favourable prognostic indicator.

Figure 4. Periodic lateralized epileptiform discharges over the right centrotemporal region following an acute cerebral infarct in the right middle cerebral artery territory.



In patients in whom paroxysmal movements, autonomic fluctuations, or sudden changes in conscious level raise the possibility of seizure activity, a video EEG, with appropriate monitoring (surface electromyography, oximetry, electrocardiogram, blood pressure), is useful for differentiating ictal from non-ictal events.

The EEG is also useful for monitoring seizure control in patients admitted in status epilepticus, and to assess progress in patients with drug intoxication or encephalitis.

Patients with suspected epileptic attacks

An interictal EEG is often helpful in providing support for a diagnosis of epilepsy in patients with a highly suggestive clinical history, and may assist in syndromic classification of patients with recurrent sei-

Table 3. Post anoxic patterns associated with an unfavourable outcome

Flat trace, <20 uV, unresponsive	
Pseudoperiodic patterns:	Burst suppression pattern
	Pseudoperiodic generalized epileptiform discharges, bilateral periodic lateralized epileptiform discharges
Post-anoxic myoclonus status epilepticus	
Alpha coma	
Theta coma	

zures. This subject will be dealt with in more detail in a subsequent article in the next issue of this journal.

In patients with frequent non-epileptic attacks (previously also known as psychogenic seizures), recording a habitual attack can be very helpful in establishing the nature of the episodes. It should be remembered, however, that it is not uncommon for non-epileptic attacks and genuine seizures to co-exist.

Dementia

The EEG is of only limited diagnostic usefulness in dementia. It is helpful when sporadic Creutzfeldt–Jakob disease is suspected, when generalized one per second periodic complexes may be seen (*Figure 5*); repeated recordings are often required (Markand and Brenner, 2003). This finding is not specific to sporadic Creutzfeldt–Jakob disease, but is helpful if the history and clinical findings are highly suggestive. In moderately severe Alzheimer's disease, the record may show slowing of the posterior alpha activity, followed in the later stages by more widespread slowing (Gordon and Sim, 1967). The EEG has largely been superseded by imaging for the diagnosis of multi-infarct dementia.

The EEG report

The 'normal' EEG

A record is reported as normal if EEG patterns recognized as associated with clinical pathology are not seen during the period of the recording.

Since the typical recording lasts 20 minutes, this does not exclude the possibility of epileptiform or other abnormal activity occurring at other times. A single routine EEG therefore can never exclude the presence of epilepsy. The importance of the clinical history cannot be overemphasized.

In addition, it should be remembered that a normal EEG does not exclude the presence of cerebral pathology, since not all abnormalities of brain structure and function produce EEG abnormalities.

The 'abnormal' EEG

An EEG is reported as abnormal if it contains specific features recognized as being associated with pathology, such as epileptiform activity, photoparoxysmal activity (paroxysmal activity induced by photic stimulation), slow activity (generalized or focal), amplitude asymmetries, specific abnormal patterns, or if there is no recordable cerebral activity.

However, an abnormal EEG does not always indicate the presence of clinically significant cerebral pathology. Epileptiform changes may be seen in 10% of patients who have undergone intracranial surgery, and in about 3% of individuals with psychiatric disorders who do not have epilepsy (Bridges, 1987). Furthermore, the presence of photoparoxysmal responses induced in a laboratory setting does not necessarily imply that the patient has photosensitive epilepsy; it may well be an incidental finding if there are no features to suggest photosensitivity in the history. It cannot be overemphasized that EEG findings should never be interpreted in isolation.

Conclusions

The EEG remains a useful investigation in specific clinical conditions, particularly where there is impairment of consciousness. The findings should always be interpreted in the context of the patient's history and clinical signs. The EEG report is normally tailored to answer the clinical question posed, thus the usefulness of the report is largely dependent on the quality of the clinical information supplied. BJHM

Conflict of interest: none.

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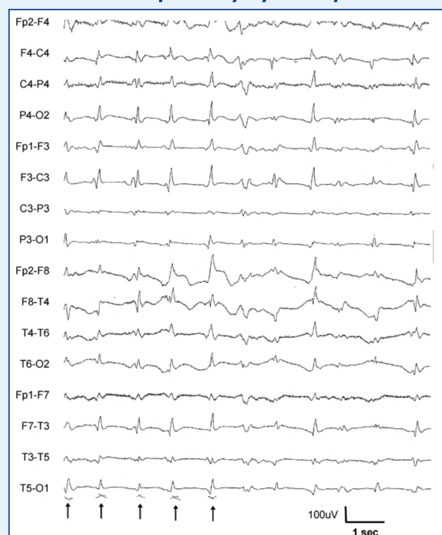
Figure 5. Generalized, one per second, periodic complexes in a patient with sporadic Creutzfeldt–Jakob disease. The arrows mark the discharges which were accompanied by myoclonic jerks.



KEY POINTS

- In patients with impaired consciousness, the electroencephalogram (EEG) is helpful in identifying patients in non-convulsive status epilepticus, and those with an encephalopathy.
- The EEG provides information on the degree and extent of cerebral dysfunction, but is not helpful in identifying the aetiology.
- The accuracy of the interpretation of the EEG findings is highly dependent on the quality of the clinical information supplied.
- The EEG, in isolation, can neither confirm nor exclude the diagnosis of epilepsy.
- In patients with recurrent seizures, the EEG may help with syndromic diagnosis.