TECHNIQUES IN CLINICAL SCIENCE

Modern electroencephalography

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Abstract Electroencephalography (EEG) has been in continuous development over at least 70 years and is firmly established as a tool in the management of epilepsy. For a while, the technique fell into disregard because of difficulties with interpretation, specificity and sensitivity. Whilst clinicians have to be aware of these problems, they have been largely addressed by recent computer digitization of signals, which permits longer standard recordings and monitoring linked to a simultaneous video. These techniques are not only an essential component of a specialist epilepsy service, where inpatient video-EEG telemetry is vital both for diagnosis and assessment before neurosurgical treatment, but also in general and acute medical settings, particularly for the management of status epilepticus. Further developments in computing will extend the use of EEG in all of these roles and long-term monitoring for diagnosis and management of coma will become more widely available.

Keywords EEG · Epilepsy · Status epilepticus · Surgery · Telemetry · Monitoring

The term electroencephalogram (EEG) was adopted from the first reports of attempts to record human cerebral electrical activity from the scalp by the German psychiatrist Hans Berger between 1928 and 1935 [1, 2]. The difficulty of recording miniscule voltage change generated in the cortex using electrodes applied to the scalp cannot be overestimated, and although Berger's initial attempts were

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John Radcliffe Hospital, Oxford University Hospitals NHS Trust, Oxford, UK e-mail: Robin.kennett@orh.nhs.uk not greatly successful, his ideas were taken on by clinicians who had a deep understanding of electronics, particularly Lord Adrian and Grey Walter [3, 4]. By the end of the 1930s recording devices, typically with three channels, were applied to patients with neurological disease, and the 'Harvard Studies' of Gibbs, Gibbs and Lennox (1937-1943) gave the first descriptions of EEG changes during epileptic seizures [5, 6]. Their observations using frequency analysis that Grand Mal epilepsy is associated with activity at 20-29/s, Petit Mal at 2-3/s and psychomotor seizures at 4-7/s, paved the way for the electroclinical syndromic diagnosis of epilepsy, which continues to be refined today. They concluded that the EEG is of 'great value in the diagnosis of epilepsy being abnormal in 48% of 'persons with a history of seizures', a statistic that is still frequently quoted. Around the same time, Grey Walter in Bristol, UK, applied statistical analysis to recordings of 4,000 patients. His salient observations were that 70% of patients with 'fits' could be diagnosed without EEG, and half of those without a diagnosis could be labelled after a recording. In other words, of every seven 'cases of fits' the EEG is indispensible for diagnosis in one, of value to supplement clinical method in three or four, and of no value in two or three. Moreover, he recognised that the EEG may be abnormal in patients without seizures, many of whom had brain tumours [4] (Table 1).

The 40 years following the Second World War saw little major progress in electroencephalography. Recordings continued to be made using electronic amplifiers and pen writers onto paper. The number of channels increased to 8 or 16, but the montage arrangement of these had to be predetermined and hard-wired, and great technical skill was needed to make useful recordings free of artefact. The knowledge of electro-clinical epilepsy syndromes slowly increased, but the role of EEG in management of patients

Table 1 A brief history of EEG

1928–1938	First recording of human EEG from the scalp by Berger, Adrian and Matthews	
1937–1945	Studies by Grey Walter, Gibbs, Gibbs and Lennox show changes related to epilepsy and when the investigation may be used clinically	
1950s-1900s	8 or 16 channel hard-wired recordings on to paper become incorporated into routine clinical practice	
1970s	First use of CCTV linked to analogue EEG	
1980s	Analogue 3 or 4 channel ambulatory EEG introduced for long-term monitoring	
1990s-2005	Digital EEG gradually replaces analogue recordings	
2005-current	Digital EEG with simultaneous video for both standard recording and telemetry, and ambulatory monitoring	

Table 2 Current role of EEG inclinical practice

Indication	Technique
Diagnosis of epilepsy and syndromic classification in children and adults	Standard EEG, ambulatory monitoring and video- EEG telemetry
Management of coma or impaired cognitive state, including status epilepticus and on neonatal, paediatric and adult intensive care units	Standard EEG, continuous EEG and cerebral function monitoring
Evaluation for surgical treatment of epilepsy	Standard EEG, video-EEG telemetry, telemetry with depth or subdural electrodes, cortical function mapping

with epilepsy fell into disrepute, both because of the low sensitivity of recordings, but also because over-interpretation of abnormalities was blamed for the mis-diagnosis of epilepsy [7]. The EEG for structural brain disease was made obsolete by brain imaging techniques.

The EEG can hardly be described as a new technique, so what has happened since the 1990s? The first major change was the advent of computerization: scalp recordings of electrical signals could now be digitized, allowing the information to be reformatted or subjected to mathematical analysis. Reformatting allows the same signal to be reviewed in a variety of different bipolar or referential montages, leading to more secure localisation of abnormalities and importantly the correct identification of artefact. The second consequence of signal digitization is that much longer recording became feasible than in the analogue era, when the EEG typically lasted 20-30 min. The third development was the incorporation of video recording of patients simultaneously with the EEG, both leading to better identification of artefact, but also allowing repeated review of subtle clinical change.

These developments have refined the way in which the EEG should be used in the modern era (Table 2). The investigation is now primarily used for the management of epilepsy and related disorders. Although technology has advanced considerably, the difficulty of recording from the scalp remains, and we are dependent on highly skilled scientific staff to prepare patients and optimally apply electrodes. Most standard recordings now use the 10–20 International System of electrode positions [8], using silver/ silver chloride electrodes, which have been found to be the

most reliable and give the least electrode artefact, attached using a sticky conductive paste (Fig. 1). Patients have to be put at ease and the EEG performed in a relaxed environment—measures essential to help keep technical and biological artefact to a minimum. The mainstay of EEG is the standard outpatient recording but now with a simultaneous video to observe any clinical change or event and prolonged enough to enable drowsiness or light sleep—states known to enhance the detection of interictal epileptiform discharges. The well-known activating procedures of hyperventilation and intermittent photic stimulation should be used as routine [9]. Prolonged standard recordings of 1–2 h may be long enough to include some of the patient's typical symptoms if they occur often enough, and it may be possible to reproduce situations that provoke the patient's attacks.

Attention to all these details gives the best chance of recording interictal epileptiform abnormalities that are strongly correlated with the presence of epilepsy, such as 3/s generalised high-amplitude spike-slow wave complexes, photosensitivity and focal spikes or sharp waves (Fig. 2). Both electrophysiologist and primary clinician, whether paediatrician or neurologist, need to be aware of the difficulties of diagnosis from both clinical and electrophysiological perspectives, and it is valuable for them to meet together to look through EEGs and videos, especially when there is doubt about the significance of an EEG finding. Particularly, the clinician needs to be aware of the high rate of false-positive findings in patients with structural cortical disease, the problem of interpreting unusual rare EEG variants that are benign but have epileptiform appearance [10], and the low sensitivity of short duration



Fig. 1 Normal EEG recording from an adult using a longitudinal temporal and transverse bipolar montage



Fig. 2 Scalp EEG recording of ictal onset in a patient with absence epilepsy using a longitudinal bipolar montage

EEG recordings. Electrophysiologists can learn from clinicians the sequence of events and patterns of epileptic seizures and the differential diagnoses of events that are very similar to epilepsy, such as non epileptic blank spells or breath-holding attacks in children. Differentiating between frontal lobe epilepsy and parasomnias and some forms of non-epileptic attack disorder can be difficult for both clinician and electrophysiologist.

If clinical doubt continues to exist further EEG studies can help define the diagnosis. The previous advice was to repeat the standard recording after a period of sleep deprivation [11]. Whilst this can increase the detection of interictal epileptiform activity, it is becoming more common practice to proceed to prolonged EEG monitoring. Ambulatory EEG with a full set of scalp electrodes can be performed over 2 or 3 days. Although this investigation requires more staff time for data analysis, it has the advantage of greater likelihood of recording interictal epileptiform discharges, especially during natural sleep, and if the patient is having daily attacks, they should occur during the monitoring. The finding of ictal electrographic rhythms temporally related to the symptoms confirms epilepsy and helps define the seizure type. Equally, if no EEG change occurs during episodes the diagnosis of nonepileptic attack disorder becomes more probable, especially if the clinical features described by the patient could only occur in association with EEG change, such as prolonged absence or unconsciousness. The diagnosis of epilepsy is usually clarified by this procedure but where doubt persists, in-patient video-EEG teleImetry monitoring of seizures becomes the definitive investigation. As with all EEG, this investigation gives the greatest reward when reviewed together by an electrophysiologist and a clinician familiar with the differential diagnosis of attack disorders. This is especially true in paediatric and neonatal practice.

Video/EEG telemetry is one of the central components of assessment of patients before surgical treatment of intractable epilepsy [12] (Table 3). The first step is to ensure that the patient's intractable attacks are epileptic seizures. This requires a period of video-EEG telemetry to record a sample of the habitual episodes. This investigation also serves the purpose of making sure that the patient has only one clinical and electrographic seizure type, and that this is consistent with any pathological process identified by MR imaging. Several seizures have to be recorded to satisfactorily address these issues, and a statistical approach indicates that five consecutive seizures of the same type give a 95% probability that 9/10 of all attacks are of this same nature [10]. The aim of pre-surgical videotelemetry assessment is thus to record five seizures, which may be easily achieved in patients with very frequent attacks. Often seizures do not occur regularly enough and anticonvulsant medication may have to be reduced before starting telemetry and the period of recording extended to 7 days. With the inherent risk of generalised convulsions after withdrawal of medication it is essential that pre-surgical telemetry is carried out in a dedicated environment with close clinical supervision by staff experienced in the protocols for managing seizures or status epilepticus. The preliminary telemetry monitoring results are assessed in relation to other baseline investigations, such as MR imaging and neuropsychological testing, at an epilepsy surgery multidisciplinary team meeting where a decision is made whether to offer the patient surgery, to proceed to further investigation or abandon the surgical option. Mesial temporal epilepsy due to hippocampal sclerosis can generally be surgically treated after preliminary investigations unless there is doubt about the side of seizure onset, when intracranial recording of seizure is needed (Fig. 3). Neocortical epilepsies usually require further evaluation, including intracranial EEG recording with depth or subdural electrodes and mapping of eloquent cortex by direct brain stimulation to enable a safe cortical resection. Intracranial EEG techniques are most useful for lesional cortical epilepsy, especially if the pathology identified by imaging is in the region of the primary motor or sensory cortex or in a region likely to contain language function. Non-lesional cortical epilepsy presents a challenge because of the large area of cortex that potentially has to be explored to find the ictal onset, and ideally

Technique	Rationale
Standard EEG and video-EEG telemetry with scalp electrodes	To confirm that the attacks: Are epileptic seizures
	They are a single seizure type
	They are clinically and electrographically appropriate for the known brain pathology
	They are lateralized
Telemetry with depth or subdural intracranial electrodes	In mesial temporal epilepsy to confirm side of ictal onset when doubt persists after extracranial recording
	To locate the site of ictal onset from the neocortex in lesional and non-lesional cortical epilepsy
Cortical function mapping	To determine the location of ictal onset in relation to eloquent areas of cortex (language area, primary motor or sensory cortex), reducing the risk of permanent neurological deficit after cortical resection

Table 3 Use of EEG in pre-surgical evaluation of epilepsy



Fig. 3 Recording of ictal onset in a patient with right mesial temporal epilepsy due to hippocampal sclerosis using subdural electrodes inserted to cover the inferior surface of the temporal lobe cortex. Each strip of electrodes has four contacts, with no. 1 being most medial

a target area should be identified by PET or SPECT scanning before embarking on intracranial EEG recordings.

Other than in the diagnosis and syndromic classification of epilepsy, the EEG has a more limited role in aiding clinical decisions. After a single attack an abnormal EEG, particularly with specific epileptiform discharges, is of predictive value for further seizures [13, 14]. Although anticonvulsant medication is usually reserved for recurrent overt seizures, an abnormal EEG may define the epilepsy type and thus direct the most appropriate medication should further attacks occur. Once the diagnosis of epilepsy has been made and treatment started, the EEG is of little value in monitoring drug therapy. This is because most anticonvulsants do not modify the interictal epileptiform discharges that are recorded during standard EEGs. The exceptions are patients with photosensitive epilepsy in whom a reduction of the photoparoxysmal response equates to better seizure control [15], and children with subtle absences that may be unrecognised but nonetheless impair cognition [16]. In these children spike-wave bursts may show marked diurnal variation, which can be quantified by ambulatory EEG to assess the effect of treatment. If patients are seizure free on medication the EEG has been shown to be of some predictive value for seizure recurrence [17], but the literature is difficult to interpret because in many studies different seizure types are grouped. The epilepsy syndromes with the greatest inherent risk of relapse, such as juvenile myoclonic and photosensitive epilepsy or symptomatic seizures, are those showing the most frequent interictal epileptiform activity, and the EEG is therefore not an independent variable when assessing risk of relapse off therapy.

The EEG plays a vital role in the management of status epilepticus and it is a pity that the investigation is not widely available in UK district general hospitals where this neurological emergency is often encountered. An urgent recording when the patient presents leads to the correct diagnosis [18]. It is a sobering fact that 28% of patients with non-epileptic seizures have been admitted to ITU [19], whereas an EEG could prevent escalation of in appropriate therapy [20]. Once diagnosed serial EEG recording can be used to ensure that seizure activity has been adequately treated as continuing attacks are known to be associated with a poorer prognosis [21]. This is particularly important if the patient has been given neuromuscular blockade, when clinical signs are lost, or in non-convulsive status epilepticus or status with minimal clinical manifestation [22]. It can also monitor the depth of drug-induced narcosis to maintain optimal levels of treatment. Whilst this monitoring on ITU can be performed with standard EEG, this has the disadvantage of producing a large volume of data that require expert evaluation, often done at an interval after the recordings have been made. To overcome these difficulties computerised quantitative EEG analysis has been developed for ITU use [23]. This is typically based on

a fast Fourier transformation (FFT) that defines a continuous EEG signal according to its sine wave components. This analysis is repeated at intervals and the simplest display is to sequentially stack one transform onto the next. Alternatively the amplitude of the transform can be multiplied by frequency for a predetermined range of frequencies to give a 'power'. The powers for each chosen frequency range can then be represented on a colour scale and plotted against time to give a colour spectral display. Both of these displays shows characteristic changes during seizures that can easily be recognised by ITU medical and nursing staff, allowing immediate intervention. Most seizures are associated with an increase in power of the slow end of the frequency spectrum but some have reduced power at this end and increase power at higher frequencies. Both produce a clear change in the display during seizures. Whilst not a substitute for full EEG analysis, these mathematical transformations are being used increasingly in the ITU to give instantaneous recognition of changes in cerebral activity.

The role of EEG in the management of encephalopathy, encephalitis and dementia has not changed greatly in recent years. The EEG remains a cheap, readily available and instantaneous measure of cerebral cortical function. The presence of slow wave abnormalities differentiates delirium from psychosis [24], and it can be used to grade the severity of encephalopathy, giving a broad indication of prognosis [25]. Herpes simplex encephalitis and Creutzfeld-Jakob disease are associated with characteristic EEG findings, but more often changes are not specific for the underlying cerebral disease, limiting its value. It must be remembered that the EEG only records from the cerebral cortex and may therefore be normal in conditions causing severe cognitive impairment such as limbic encephalitis.

The immediate future of EEG will be incremental change that is already under way, especially with greater application of longer recordings and monitoring. I have described how this is of value in epilepsy, but another area where long-term EEG is likely to be employed more in the future is with the emerging specialty of sleep medicine. Over night polysomnography allows sleep staging, deriving a 'hypnogram' that helps define the sleep pattern. Combined with simultaneous video this readily detects disturbances such as parasomnia, including REM sleep behaviour disorder, and periodic leg movements. A major burden of sleep medicine is excessive day time sleepiness, which often requires investigation with a combination of multiple sleep latency (MSLT) and polysomnography testing.

It is disappointing that EEG has not received the attention given by physicists and mathematicians to other investigation modalities, but future advancement of computer and signal analysis will benefit assessment of long-term EEG recordings. Spike and seizure detection software is currently available, but the nature of EEG signals is such that there is a high false-positive detection rate necessitating detailed technical review. Improvement in software will speed this arduous process of data reduction. Another problem encountered in scalp monitoring of seizures is artefact obscuring ictal rhythms. With computer-based analysis it is possible to subtract muscle and eve-movement potentials from cerebral rhythms, but this requires considerable computer power and is not yet commercially available. Magneto-encephalography (MEG), like EEG, has good temporal resolution of cerebral events, but is less affected by distortion from the skull and scalp. Thus, theoretically MEG has an advantage over EEG, but it has not found a niche in clinical practice, mainly because of the cost and inflexibility of the recording system. Specialist epilepsy surgery units can co-register the MEG signal to MRI images, but the drawbacks of MEG mean that it is unlikely to replace EEG as a clinical tool. Another technique that like MEG has a history but limited present or future clinical role is EEG brain mapping. The complexity of EEG signal generators and the very great mathematical problem of the inversesolution (deriving three-dimensional coordinates of an electrical signal from two-dimensional scalp recording) is likely to hamper future development.

An aspiration of neurophysiology is to be able to use EEG for treatment as well as for diagnosis, and there are three areas under development. Computer-brain interfacing (CBI) would be of obvious benefit to paralysed patients. It remains to be seen whether individuals are able to consciously modify their cerebral electrical rhythms (unlike eye movements that produce an easily detectable scalp potential) and whether this can be used to drive computer software. The second area is applying EEG evaluation to predict when seizures are about to occur with enough warning to allow therapeutic intervention to abort them. Despite computer-driven analysis this goal has yet to be realised from scalp recordings, but there is a prospect of being able to record from indwelling cerebral electrodes, which may be more successful. As with many aspects of EEG, the generators of cerebral potentials are complex and any continuous monitoring to detect consistent subtle changes requires huge computing power.

Electroencephalography is often considered to be an old-fashioned investigation of limited clinical value, but technology has changed in recent years, and in this article I have outlined areas of clinical practice in which it remains an invaluable tool. It is essential for the syndromic diagnosis of epilepsy in children and adults, in the evaluation of patients for surgical treatment of epilepsy and in the management of status epilepticus. It also provides a readily accessible means for assessing cerebral function and should be used routinely for patients in coma or on neurological intensive care units. It has not yet become established in this setting, mainly because of the need to develop computer signal analysis, but in the future such advances will undoubtedly assist in data reduction, enabling longterm recordings and ensuring that electroencephalography will be playing a part in clinical medicine for years to come.

References

- 1. Berger H (1929) Uber das Elektrenkephalogramm des Menschen. Arch Psychiat Nervenkr 87:527–570
- Berger H (1938) Uber das Elektrenkephalogramm des Menschen (14th report). Arch Psychiat Nervenkr 108:407–431
- 3. Adrian ED, Matthews BHC (1934) The Berger rhythm, potential changes from the occipital lobe in man. Brain 57:345–359
- Grey Walter W (1950) Epilepsy. In: Hill JDN, Parr G (eds) Electroencephalography. Macdonald, London, pp 228–272
- 5. Gibbs FA, Gibbs EL, Lennox WG (1937) Epilepsy, a paroxysmal cerebral dysrhythmia. Brain 60:377–388
- Gibbs FA, Gibbs EL, Lennox WG (1943) Electroencephalographic classification of epileptic patients and control subjects. Arch Neurol (Chicago) 50:111–128
- Benbardis SR, Tatum WO (2003) Overinterpretation of EEGs and misdiagnosis of epilepsy. J Clin Neurophysiol 20:42–44
- Sharbrough F, Chatrian G-E, Lesser RP, Lüders H, Nuwer M, Picton TW (1991) American Electroencephalographic Society guidelines for standard electrode position nomenclature. J Clin Neurophysiol 8:200–202
- 9. Flink R, Pedersen B, Guekht AB et al (2002) (ILAE Commission Report). Guidelines for the use of EEG methodology in the diagnosis of epilepsy. Acta Neurol Scand 106:1–7
- Kennett RP (2000) Neurophysiologic investigation of adults. In: Oxbury JM, Polkey CE, Duchowny M (eds) Focal Intractable Epilepsy. WB Saunders, London, pp 333–362
- Halasz P, Filakovszky J, Vargha A, Bagdy G (2002) Effect of sleep deprivation on spike-wave discharges in idiopathic generalised epilepsy: a 4 x 24 hour continuous long term EEG monitoring study. Epilepsy Res 51:123–132

- Luders HO, Comair YG (eds) (2001) Epilepsy Surgery, 2nd edn. Lippincott, Williams and Wilkins, Philadephia
- Berg AT, Shinnar S (1991) The risk of seizure recurrence following a first unprovoked seizure: a quantitative review. Neurology 41:965–972
- Kim LG, Johnson TL, Marson AG, Chadwick DW (2006) Prediction of risk of seizure recurrence after a single seizure and early epilepsy: further results from the MESS trial. Lancet Neurol 5:317–322
- Harding GF, Herrick CE, Jeavons PM (1978) A controlled study of the effect of sodium valproate on photosensitive epilepsy and its prognosis. Epilepsia 19:555–565
- Binnie CD (2001) Cognitive performance, subtle seizures, and the EEG. Epilepsia 42(suppl 1):16–18
- Berg AT, Shinnar S (1994) Relapse following discontinuation of antiepileptic drugs: a meta-analysis. Neurology 44:601–608
- Kaplan PW (2006) The EEG of status epilepticus. J Clin Neurophysiol 23:221–229
- Reuber M, Pukrop R, Bauer J, Helmstaedter C, Tessendorf N, Elger CE (2003) Outcome in psychogenic nonepileptic seizures: 1- to 10-year follow up in 164 patients. Ann Neurol 53:305–311
- Boesebeck F, Freemann S, Kellinghaus C, Evers S (2009) Misdiagnosis of epileptic and non-epileptic seizures in a neurological intensive care unit. Acta Neurol Scand 122:189–195
- 21. Nei M, Lee JM, Shanker VL, Sperling MR (1999) The EEG and prognosis in status epilepticus. Epilepsia 40:157–163
- De Lorenzo RJ, Waterhouse EJ, Towne AR et al (1998) Persistent nonconvulsive status epilepticus after the control of convulsive status epilepticus. Epilepsia 39:833–840
- Rijsdijk M, Leijten FSS, Slooter AJC (2008) Continuous EEG monitoring in the intensive care unit. Neth J Crit Care 12:157–162
- Sidhu KS, Balon R, Ajluni V, Boutros NN (2009) Standard EEG and the difficult-to-assess mental status. Ann Clin Psychiatry 21:103–108
- 25. Yamashita S, Morinaga T, Ohgo S, Sakamoto T, Kaku N, Sugimoto S, Matsukura S (1995) Prognostic value of electroencephalogram (EEG) in anoxic encephalopathy after cardiopulmonary resuscitation: relationship amoung anoxic period, EEG grading and outcome. Intern Med 34:71–76