

ARTICLE

EEG in psychiatric practice: to do or not to do?

Vellingiri Raja Badrakalimuthu, Radhika Swamiraju & Hugo de Waal

Vellingiri Raja Badrakalimuthu is Clinical Leadership Fellow and an honorary specialty registrar in old age liaison psychiatry at Beechcroft, Fulbourn Hospital, Cambridge. His special interests include neuroscience, psychopharmacology and electroconvulsive therapy. **Radhika Swamiraju** is an ST5 registrar in old age psychiatry and is based in Warrington. Her special interests include the interface between geriatric medicine and psychiatry. **Hugo de Waal** is consultant old age psychiatrist in Norwich. He is Head of the School of Psychiatry for the Eastern Deanery and a tutor at the University of East Anglia. His special interests include application of research outcomes in clinical practice, ethics and neuroscience. **Correspondence** Dr V. R. Badrakalimuthu, Beechcroft, Fulbourn Hospital, Cambridge CB21 5EF, UK. Email: raja.badrakalimuthu@cpft.nhs.uk

SUMMARY

Electroencephalography (EEG) is a non-invasive investigation that can aid the diagnosis of psychiatric and neuropsychiatric disorders. A good predictor of an abnormal EEG recording is the presence of an organic factor identified during the clinical assessment. The non-invasiveness and low cost of the procedure and its ability to measure spontaneous brain activity appear to attract clinicians to utilise this investigative tool. However, studies have reported that EEGs arising from psychiatric referrals have the lowest abnormality detection rate. The focus of this article is to improve this by highlighting the current pitfalls and providing recommendations for appropriate utilisation of EEG. We describe specific EEG changes associated with major psychiatric disorders. We conclude by offering pragmatic considerations when referring a patient for EEG, emphasising the fact that the information provided to the neurophysiologist plays a crucial role in interpreting the EEG recording in a diagnostically meaningful way.

DECLARATION OF INTEREST

None.

recordings with single aspects of temperament or personality; and the use of activation techniques to demonstrate hidden pathologies. There have been technological advances since his erudite article was published, but the fundamental problems remain.

In this article, we focus on the role of EEG in current psychiatric practice, EEG findings in specific psychiatric syndromes, clinical indications for requesting EEG, and interpretations of EEG recordings. We will also highlight measures to ensure appropriate and competent use of EEG in the routine clinical practice of psychiatrists.

EEG in current psychiatric practice

A retrospective review of EEG requests over a 12-month period found that 6.2% of referrals were made by psychiatrists but that psychiatric referrals had the lowest abnormality detection rate (O'Sullivan 2006). A history of epilepsy, being on clozapine and possible convulsive seizures were found to be the only significant predictors of an abnormal EEG recording. Another study reported the presence of an organic factor identified during clinical assessment to be significant predictor, but none of the abnormal recordings helped in discovering an aetiology that was not already suspected from assessment (Lam 1988). It also reported cases in which psychiatrists ignored abnormal recordings.

Of 187 requests for EEG on patients showing aggressive behaviour, 71% were made to ascertain evidence of epilepsy and 22% to determine the presence of organic brain dysfunction. Only one recording showed unequivocal evidence of an epileptic focus (Stone 2003). The low diagnostic yield reported in the Stone study is similar to the 0.5–2% found in EEG conducted to screen for epilepsy (Gregory 1993). Another issue regarding the diagnostic yield emerged from a study of psychiatric in-patients (Warner 1990): although a third of the EEG recordings were abnormal, this led to a change in diagnosis in only 1.7% of cases.

Limitations of EEG

Why is it that EEG produces findings that some consider to be clinically irrelevant? The most important limitation that one should take into

The first electroencephalography (EEG) on a human was reported by Hans Berger in 1920. The fundamental principle of EEG is to record the electrical activity produced by the brain, using electrodes. In considering the role of EEG in psychiatry almost 25 years later, Dennis Williams (1954) observed that studies of EEG recordings had offered the clinical psychiatrist little of positive value. He suggested several reasons for this: EEG does not give a true record of cerebral action potentials; attempts at correlating EEG recordings from millions of cells, the electrical activity of which was not understood, with patients' clinical picture proved meaningless; the recording techniques used were crude; and EEG records simple physiological changes in neuronal activity which are not reflected in the complex integration of the human brain. Nevertheless, Williams did not dismiss EEG out of hand. He outlined its usefulness in the exclusion of gross organic disorder; the study and modification of local cerebral disorders related to abnormal behaviour; the correlation of changes in EEG

account is that, by and large, EEG abnormalities are non-specific. With its poor spatial resolution, EEG cannot pinpoint the pathological process, nor can it aid the differentiation between functional diagnoses. In organic dysfunction, EEG would miss slow-growing, deep-seated tumours. Given that the epileptiform activity in epilepsy is not consistently present, one can expect a false-negative rate of around 50% on interictal EEG recordings (Hopkins 1988) – and a false-positive rate of 2% in the general population (Hughes 1999). Apart from such theoretical and clinical limitations, the cumbersome procedure in itself leads to artefacts that can be difficult to interpret. The comparative lack of psychiatric training among physiologists and the lack of training in observing and interpreting EEG among psychiatrists is another problem.

Despite these shortcomings, EEG is attractive for clinicians: the procedure is non-invasive, can measure spontaneous brain activity, requires a lower level of patient cooperation than functional magnetic resonance imaging, and is relatively inexpensive and easily available.

The EEG procedure

An EEG wave is the representation of the differences in electrical potential between an electrode placed on the scalp and a reference electrode placed elsewhere on the head. The transient nature of this electrical difference leads to oscillation, which produces the EEG pattern or trace.

Standard scalp EEG, which uses 21 electrodes, can take up to 40 minutes. In addition to the external placing used in standard EEG, the electrodes can be placed directly inside the skull (intracranial EEG), under the skin of the scalp (subdural) or on the external brain surface and dura mater (electrocorticography).

If abnormality is strongly suspected but the resting EEG is normal, activated EEG is used to increase the probability of recording abnormal patterns. Strenuous hyperventilation and photic stimulation (stimulation of the visual cortex using light) are the most common activation procedures.

In EEG-video monitoring, a standard EEG is conducted with simultaneous video recording of the patient. The advantage of EEG-video monitoring lies in the observation of the temporal relationship between the behaviour under investigation and the EEG recording. Serial and continuous EEG recordings can help to increase the diagnostic yield.

Quantitative EEG (qEEG) utilises analytical algorithms that automatically assess resting and evoked activity of the brain (Box 1). This method

BOX 1 Quantitative EEG

Frequency analysis is the most powerful analytical tool used in qEEG to limit artefacts. It acts by reducing EEG data to a manageable number of coefficients. The fast Fourier transformation, based on Fourier analysis, simplifies computation of spectral coefficients. As EEG traces contain regular patterns, they may be better understood by their spectral (frequency) content. In a spectral analysis, voltage amplitudes are translated into frequencies because mental activities are better reflected in periodicities.

The power spectral density (power spectrum) approach has always been a popular method for classifying EEG signals. The power spectral density (PSD) describes how the power (or variance) of a time series

is distributed with frequency. The first step in EEG classification is to determine whether the signals have distinguishable features in their power spectrum, where power is a state parameter that can be used instead of the magnitude of the frequency of the wave. Power is sometimes represented as log power (base 10 logarithm).

Frequency or spectral analysis involves selection of elementary shapes or frequencies (waveforms) which are added together like weights on a scale until their total matches the pattern under investigation. The height or intensity of a waveform – its amplitude – is computed in microvolts for each frequency.

For further information see Kaiser (2005).

involves comparing data from a specific individual with those of database of a large population or of a defined population with a specific disorder.

In qEEG, event-related (evoked) potentials – neuronal electrical responses to stimuli – are extracted from EEG recordings by time domain analysis (analysis of the EEG trace with respect to time). Event-related oscillations are changes in the power spectrum frequency of the ongoing EEG, which can be directly or loosely time-related. They are complementary to the routine EEG as they provide an understanding of higher brain functions, including cognition.

Physiological factors that influence EEG

EEG and age

Electroencephalogram patterns (Table 1) are influenced by age. Infant EEG recording shows slow-wave and high-amplitude rhythms. The asynchronous, more mature rhythms develop between 2 and 6 years of age. Generalised low-amplitude beta activity starts to appear in puberty, and adult EEG shows posterior alpha and anterior beta activity. In old age, there is a slowing of the alpha frequency accompanied by a decrease in delta activity.

EEG and state (awake v. sleep pattern)

Electroencephalograms are also affected by the level of alertness or consciousness. A normal 'eyes-closed' wake EEG trace is characterised by highly rhythmic alpha waves (Table 1). Beta waves are not uncommon, especially over the frontocentral regions in a normal adult wake EEG trace. Theta

TABLE 1 EEG rhythms

Wave type	Frequency, Hz	Feature
Delta	<4	Frontal in adults and posterior in children High-amplitude waves Seen in slow-wave sleep of adults
Theta	4–7	Can be found in young children Transient theta waves can be seen in up to 15% of the normal population
Alpha	8–12	Bilateral and occipital Higher amplitude on the dominant side Seen in relaxed state with the eyes closed Attenuated with attention
Beta	12–30	Bilateral, frontal Low-amplitude waves Seen in alert stage
Mu	7–11	Associated with motor activity and seen in precentral areas Attenuated with contralateral limb movement
Lambda		Occipital Single sharp wave Associated with ocular movement
Gamma	34–100	Associated with cognitive and motor functions

activity is very limited on wake EEG traces and there should be no delta activity. Alpha coma is a diffuse alpha wave occurring in a comatose state. Table 2 shows the stages of sleep and related EEG patterns.

EEG in major psychiatric disorders in working-age and older adults

Many associations between specific EEG findings and psychiatric disorders are marred by conflicting reports. In this section we list the robust EEG findings in relation to specific psychiatric disorders and drugs.

Delirium

Electroencephalography can be a valuable tool of investigation in delirium. The EEG trace in delirium characteristically shows slowing or drop-out of the posterior dominant rhythm (earliest), generalised theta or delta slow-wave activity, a

poorly organised background rhythm, and no reaction to opening and closing of the eyes. Serum anticholinergic activity has been suggested as a biomarker for delirium and such activity has been correlated with the following EEG findings: occipital slowing and qEEG with delta and theta increase as well as alpha decrease, as a result of which there is a lower slow-wave ratio compared with traces obtained in dementia (Thomas 2008a). On qEEG, theta activity and relative power in the delta band collectively distinguish delirium from dementia (Jacobson 1993). Prolonged-activation EEG can be used to diagnose delirium in dementia with 67% sensitivity and 91% specificity based on variations in alpha and delta power density (Thomas 2008b).

Dementia

In dementia, qEEG abnormalities include higher frontocentral and parieto-occipital theta values, and lower parieto-occipital beta values. Lower peak frequency has been significantly associated with decline in cognitive function in patients with probable early-stage Alzheimer's dementia (Clauss 1998). There is evidence that visual and quantitative analyses of EEG traces can be used to differentiate between Alzheimer's and vascular dementia (Gawel 2009). Quantitative EEG findings indicate that increased EEG slowing is associated with a poorer cognitive performance in patients with vascular dementia (Muresanu 2008).

Electroencephalography can help in discriminating between Alzheimer's disease and Lewy body dementia at the earliest stages of illness. In a study (Bonanni 2008) that recruited 50 patients with a diagnosis of Alzheimer's dementia, Lewy body dementia or Parkinson's disease dementia, dominant wave frequencies were 8.3 Hz (s.d. = 0.6) for the Alzheimer's disease group and 7.4 Hz (s.d. = 1) for the Lewy body dementia group. Less than half (46%) of the patients with Parkinson's disease dementia exhibited the EEG abnormalities seen in those with Lewy body dementia.

In a smaller study ($n=19$), qEEG results for patients with frontotemporal dementia were marked by no increase in slow activities but by a decrease in fast activities (Lindau 2003). The relative power from the temporal region in the beta-2 band and from the parietal region in the theta, alpha and beta-2 bands are informative qEEG variables in distinguishing between frontotemporal dementia and Alzheimer's disease, with a diagnostic accuracy of 84.6% (Yener 1996).

Despite such observations, a meta-analysis has showed that evidence of diagnostic utility of EEG

TABLE 2 Sleep and EEG patterns

Stage	Feature	EEG pattern
1	Transitional stage between sleep and wakefulness	Waves with a frequency 3–7 Hz
2	Fragmentation of thought processes	K-complex and sleep spindles
3 and 4	Slow-wave sleep	Delta waves of frequency 0.5–1 Hz Stage 4: delta waves more than 50% of the record
REM	Occurs every 90 minutes	Mixed frequency Saw-tooth waves Low-voltage waves

REM, rapid eye movement.

in dementia and mild cognitive impairment is still insufficient (Jelic 2009).

Psychotic disorders

One of the difficulties in interpreting EEG findings that show increased frontal delta activity in people with schizophrenia is that such individuals may be more prone to involuntary saccadic eye movements (Matsue 1986). Antipsychotic medications increase the amount of alpha activity, adding another confounding factor to the EEG recording.

A study of 19 medication-free patients with chronic schizophrenia found greater delta and left-sided beta activity than in the 21 controls (Karson 1988). In the very small number of patients who had reduced alpha frequency, there was an association with larger mean ventricular size. This reduction in alpha frequency has been confirmed in a study of 20 never-treated schizophrenia patients compared with age-matched controls (Omori 1995).

Most research involving qEEG activity power spectra has shown that people with schizophrenia exhibit increased beta and slow-frequency powers and reduced main alpha power (John 1994). One study (Gross 2006), involving 16 patients with treatment-resistant chronic schizophrenia experiencing a relapse, used qEEG to investigate the correlation between the power of different frequency bands over the frontocentral scalp area and Liddle's factors and negative subscale scores on the Positive and Negative Syndrome Scale (PANSS). The only significant finding was a positive correlation between beta power and psychomotor poverty. Similar studies have shown that negative symptoms are correlated with an increase in beta- and delta-band activity (Williamson 1989), whereas positive symptoms are correlated with increased theta and delta activity (Fehr 2001). Interestingly, a study of qEEG findings in late-onset schizophrenia ($n = 10$) showed no significant abnormality, thus raising the possibility of a biological difference in the disorder based on the age at onset (Reeves 2003).

P300 and P50 waves

The finding of reduction in auditorily evoked P300 wave activity and increased P300 latency among patients with schizophrenia is robust, unlike visually evoked P300 potentials (Box 2). As the visual P300 potentials are influenced by clinical variables, one hypothesis is that auditory P300 is a trait marker of schizophrenia, whereas visual P300 is more of a state marker (Ford 1999). Auditory P300 abnormalities have been detected irrespective of the stage of illness (including presentations in the prodromal stage, in individuals who are not

actively manifesting symptoms and are considered to be in remission, and in individuals with a family history of schizophrenia) and also in people with schizotypal personality (van der Stelt 2005). These groups also show a prolonged auditory P300 latency. Sagittal reductions in P300 cognitive event-related potentials and reduced left temporal voltage related to asymmetry in the posterior superior temporal gyrus have been reported in patients with schizophrenia. A note of caution is that although the reduction in left temporal voltage has been replicated, many studies have failed to establish it robustly, possibly because of patient selection and response mode.

Abnormal P50 sensory gating correlates due to defects in attention have been found not only in people with schizophrenia but also in their relatives (Clementz 1998).

In a meta-analysis looking at the usefulness of spectral EEG as a diagnostic tool for schizophrenia, Boutros *et al* (2008) draw the conclusion that most of the studies they looked at confirmed the increased preponderance of slow rhythms in people with schizophrenia, irrespective of their treatment status. However, they point to the lack of robust data on the sensitivity and specificity of the findings and the lack of multicentre studies using standardised criteria. Shagass *et al* (1984) reported a sensitivity of 50% and a specificity of 90% in a comparison of individuals with schizophrenia and those with depression using slow-wave rhythms as a diagnostic marker.

Affective disorders

Mania

A review by Shelley *et al* (2008) reports that EEG abnormalities such as small sharp spikes, 6/s spike and positive spikes are more common in patients

BOX 2 Event-related potentials

Event-related potentials (ERPs) are recorded while the individual is performing a sensory or cognitive task. They reflect the summated activity of network ensembles active during the task and are characterised by a specific pattern called the waveform, which is composed of negative and positive deflections (waves). For example, a target stimulus detected amid a series of other non-target stimuli produces a positive wave around 300 ms (latency) after the stimulus. This is known as the P300 response.

Sensory gating refers to the pre-attentional habituation of responses to repeated exposure to the same sensory stimulus. P50 is an electroencephalogram (EEG) event-related potential waveform used to assess sensory gating.

with mania than in those with depression, in female than in male patients with bipolar disorder, and in non-familial cases of late-age onset mood disorder. Findings such as small sharp spikes, 6/s spike and wave complexes and positive spikes in suicidal patients are of ambiguous clinical significance. There is some evidence to show that interhemispheric asymmetry and decreased coherence of the EEG trace in bipolar disorder can be used to differentiate it from schizophrenia. Anteroposterior P300 topography also differentiates schizophrenia from bipolar disorder with psychotic mania. One study ($n = 35$ with schizophrenia, $n = 20$ with psychotic mania) found posterior P300 reductions in the schizophrenia group and anterior reductions in the bipolar group (Salisbury 1999).

Depression

Depression is the most common mental disorder experienced by people with epilepsy (Kanner 2003). Consequently, even if epilepsy has not been diagnosed, EEG findings for people with depression need to be interpreted with caution as they may indicate underlying epileptic disorder rather than being related to depression.

Resting EEG shows greater alpha-wave activity with eyes closed in patients with depression, which has been interpreted as indicating a reduction in cortical activity in depression. Similarly, increased alpha and beta activity has been reported in patients with psychotic depression, but this should be interpreted with caution because such changes can be brought on by metabolic alterations due to poor nutrition (Margerison 1962) or by alterations in adrenaline levels (Gjessing 1967).

Visual P300 (P300 evoked using a visual stimulus) has been found to be abnormal in patients with major depressive disorder and schizophrenia (Blackwood 1987).

Hypnograms showing the pattern of sleep stages through the night reveal irregular transitions between the different sleep states and frequent awakening in people with depression. There is a reduction in deep-sleep stages associated with long periods of rapid eye movement (REM) sleep (Benca 1992). Antidepressants can resolve such irregular transitions and return the EEG pattern during sleep to normal (Figuroa Helland 2008).

Resting EEG has also been used to show that patients with depression who respond to fluoxetine (Bruder 2008), imipramine (Knott 1996), amitriptyline alone and with an adjunctive anticonvulsant or lithium (Ulrich 1986) have a greater alpha power, particularly in the occipital area, than non-responders and healthy controls. Bruder *et al* (2008) confirmed greater alpha waves

(suggestive of lower neuronal activity) over the right than over the left hemisphere, with a high test–retest correlation. Increased pre-treatment activity in the anterior cingulate cortex has been recorded in patients who responded to citalopram and reboxetine when compared with non-responders (Mulert 2007). Deldin & Chiu (2005) report from their EEG study of cognitive restructuring that patients with depression who responded to cognitive treatment exhibited greater overall cortical activity, and cortical asymmetry of greater right relative to left activity in frontal areas, than non-responders.

Pre-treatment alpha measures thus seem to be a predictor of response to antidepressants, but the studies need to be replicated with larger samples to confirm the clinical relevance of these findings.

Anxiety disorders

Although resting EEG recordings from people with anxiety disorders do not show typical deviations from normality, under experimentally induced anxiety, recordings show hypersynchronised alpha activity (Herrmann 1996).

Interestingly, a small study (Arriaga 1991) reported that EEG sleep recordings of participants with primary dysthymia showed reduced slow-wave sleep and absence of REM sleep disturbances. These indicate that biologically, primary dysthymia may be closer to generalised anxiety disorder than to affective disorder.

People with panic attacks accompanied by agoraphobic disorders show significant decreases in the power density of the alpha rhythm and increases in the power density of the beta rhythm in the right hemisphere (Gordeev 2008). This reflects a significant activation of the ascending mesencephalic reticular formation. The most characteristic feature in people with panic attacks without agoraphobia is a significant increase in the power density of the theta rhythm in temporal areas of the right hemisphere, reflecting increased activity in temporolimbic structures.

People with obsessive–compulsive disorder (OCD) have increased current density for delta waves in the insula and for beta waves in the frontal, parietal and limbic lobes. Prichep *et al* (1993) showed that a group of patients who had a relative excess of alpha waves responded well to serotonin reuptake inhibitors for their OCD.

Studies (Jokic-Begic 2003; Chae 2004) show that people with post-traumatic stress disorder (PTSD) have globally reduced complexity in their resting EEG wave forms. However, contrary to almost all hypotheses, Shankman *et al* (2008) found that individuals with PTSD did not differ from controls on resting EEG asymmetry.

Personality disorders

Enhanced cortical slow activity in the delta range has been consistently reported in violent criminal offenders. Increased slow-wave activity in adolescence has been shown to predict emergence of antisocial behaviour later in life (Raine 1990). Attenuated alpha rhythm and increase in theta and delta activity in the frontal lobe has been found in violent offenders with antisocial personality disorder (Reyes 2009). Diminished P300 activity across varied activity has been reported in people with aggression and impulsivity (Gerstle 1998) and in individuals diagnosed with antisocial personality disorder (Bauer 1994).

Substance misuse and medications

Alcohol, illicit drugs and prescribed medications (discussed under particular psychiatric disorders) significantly affect EEG recordings. As expected, this influence is greatest on alpha-wave patterns, the dominant pattern seen in the wake state, with increased activity seen in cannabis and cocaine withdrawal and decreased activity in opioid misuse.

In alcohol withdrawal, EEG traces may show attenuation of voltage and prominence of beta activity. The findings of a study (Moeller 1993) that compared the sleep patterns of people with primary major depression and with major depression secondary to alcohol dependence suggest an additive effect of alcohol dependence and comorbid depression on sleep, with changes to delta sleep, REM and non-REM sleep patterns.

Electroconvulsive therapy

During electroconvulsive therapy (ECT), EEG recordings show a sequence of high-voltage sharp waves and spikes. This is followed by rhythmic slow waves with a definite endpoint. Wahlund *et al* (2009) examined frequency distributions of ictal EEG after ECT stimulation in diagnostic subgroups of depression. Psychotic depression has a high occurrence of delta and theta waves, unipolar depression has a high occurrence of delta, theta and gamma waves, and bipolar depression has a high occurrence of gamma waves (accuracy 94%).

The EEG request

When to ask for an EEG

It is common sense to consider EEG under certain specific circumstances, such as those listed in Box 3. This list is neither prescriptive nor exhaustive, but it implies that clinicians should be highly vigilant for possible organic factors underlying any psychiatric presentation.

BOX 3 Some situations in which an EEG might be requested

An EEG might be requested:

- to interpret paroxysmal activity
- to analyse sleep disorders
- to establish the cause of cognitive decline when there is a suspicion of Huntington's disease, Creutzfeldt–Jakob disease, chronic delirium, etc.

A serial, ambulatory or video EEG may be useful in:

- interpreting periodic behavioural dysfunction
- distinguishing certain types of seizures
- and, to certain extent, differentiating between true and pseudo-seizures

What information to provide?

The physiologist or physician who reports on an EEG must have access to all relevant clinical information pertaining to the case being investigated. Box 4 lists the most commonly useful information.

Training issues

The most important problem highlighted in studies of current practice is the gap in knowledge among psychiatrists, physicians, physiologists and EEG technicians about utilising and interpreting EEG in patients with psychiatric disorders. Although it is probably practically unfeasible and clinically unnecessary to have an EEG laboratory physically attached to a psychiatric unit, having a laboratory in close proximity to the unit can facilitate collaborative work between psychiatrists and EEG technicians, resulting in a more integrated and appropriate use of EEG (Pogarell 2005).

BOX 4 Information to submit with the EEG request

- Description of symptoms, including onset, frequency and duration
- Triggers for those symptoms (e.g. hyperventilation)
- Relevant medical history
- Relevant psychiatric history
- Family history of neurological or neuropsychiatric disorders
- Reports and traces from previous EEGs
- Medications, particularly those known to cause EEG abnormalities
- Results from other investigations
- Differential diagnoses under consideration

MCQ answers

1 a 2 c 3 d 4 b 5 b

Psychiatrists require robust training in the diagnostic usefulness of EEG and interpretation of results, and physicians, physiologists and technicians should have a working knowledge of relevant psychiatric disorders. Psychiatric trainees should be encouraged to participate in case discussions with colleagues regarding EEG. The Royal College of Psychiatrists' curriculum should continue to demand that budding psychiatrists have a good knowledge of this investigation and its clinical relevance. A CPD module on EEG would be of much benefit to both psychiatric trainees and mature clinicians, for whom it might serve as a refresher course.

Conclusions

The application of EEG in psychiatry is in understanding the aetiology and pathological mechanisms of psychiatric disorders, thus aiding clinical diagnosis and guiding appropriate treatment. It can be useful in clinical scenarios where there is a suspected interplay between functional symptoms and organic aetiology. Psychiatrists should base their use of EEG on robust clinical assessment and should be aware of the limitations inherent in the procedure.

References

- Arriaga F, Paiva T (1991) Clinical and EEG sleep changes in primary dysthymia and generalised anxiety: a comparison with normal controls. *Neuropsychobiology* **24**: 109–14.
- Bauer LO, O'Connor S, Hesselbrock VM (1994) Frontal P300 decrements in antisocial personality disorder. *Alcohol Clinical and Experimental Research* **18**: 300–5.
- Benca RM, Obermeyer WH, Thisted RA, et al (1992) Sleep and psychiatric disorders. *Archives of General Psychiatry* **49**: 651–8.
- Blackwood DH, Whalley LJ, Christie JE, et al (1987) Changes in auditory P3 event-related potential in schizophrenia and depression. *British Journal of Psychiatry* **150**: 154–60.
- Bonanni L, Thomas A, Tiraboschi P, et al (2008) EEG comparisons in early Alzheimer's disease, dementia with Lewy bodies and Parkinson's disease with dementia patients with 2-year follow-up. *Brain* **131**: 690–705.
- Boutros NN, Arfken C, Galderisi S, et al (2008) The status of spectral EEG abnormality as a diagnostic test for schizophrenia. *Schizophrenia Research* **99**: 225–37.
- Bruder GE, Sedoruk JP, Stewart JW, et al (2008) EEG alpha measures predict therapeutic response to an SSRI antidepressant: pre- and post-treatment findings. *Biological Psychiatry* **63**: 1171–7.
- Chae JH, Jeong J, Paterson BS, et al (2004) Dimensional complexity of the EEG in patients with post traumatic stress disorder. *Psychiatric Research* **131**: 79–89.
- Clauss JJ, Kwa VI, Teunisse S, et al (1998) Slowing on quantitative spectral EEG is a marker for rate of subsequent cognitive and functional decline in early Alzheimer's disease. *Alzheimer's Disease and Associated Disorders* **12**: 167–74.
- Clementz BA, Geyer MA, Braff DL (1998) Poor P50 suppression among schizophrenia patients and their first-degree biological relatives. *American Journal of Psychiatry* **155**: 1691–4.
- Deldin PJ, Chiu P (2005) Cognitive restructuring and EEG in major depression. *Biological Psychology* **70**: 141–51.

- Fehr T, Kissler J, Moratti S, et al (2001) Source distribution of neuromagnetic slow waves and MEG-delta activity in schizophrenic patients. *Biological Psychiatry* **50**: 108–16.
- Figuroa Helland VC, Postnova S, Schwarz U, et al (2008) Comparison of the different methods for the evaluation of treatment effects from the sleep EEG of patients with major depression. *Journal of Biological Physics* **34**: 393–404.
- Ford JM (1999) Schizophrenia: the broken P300 and beyond. *Psychophysiology* **36**: 667–82.
- Gawel M, Zalewska E, Szmids-Salkowska E, et al (2009) The value of quantitative EEG in differential diagnosis of Alzheimer's disease and subcortical vascular dementia. *Journal of Neurological Sciences* **283**: 127–33.
- Gerstle JE, Mathias CW, Stanford MS (1998) Auditory P300 and self-reported impulsive aggression. *Progress in Neuropsychopharmacology of Biological Psychiatry* **22**: 575–83.
- Gjessing LR, Harding GFA, Jenner FA, et al (1967) The EEG in three cases of periodic catatonia. *British Journal of Psychiatry* **113**: 1271–82.
- Gordeev SA (2008) Clinical-psychophysiological studies of patients with panic attacks with and without agoraphobic disorders. *Neuroscience and Behavioural Physiology* **38**: 633–7.
- Gregory RP, Oates T, Merry RT (1993) Electroencephalogram epileptiform abnormalities in candidates for aircrew training. *Electromyography and Clinical Neurophysiology* **86**: 75–7.
- Gross A, Joutsiniemi SL, Rimon R, et al (2006) Correlation of symptom clusters of schizophrenia with absolute powers of main frequency bands in quantitative EEG. *Behavioral and Brain Functions* **2**: 23.
- Herrmann WM, Winterer G (1996) Electroencephalography in psychiatry – current status and outlook [in German]. *Der Nervenarzt* **67**: 348–59.
- Hopkins A, Garman A, Clarke C (1988) The first seizure in adult life. Value of clinical features, electroencephalography, and computerised tomographic scanning in prediction of seizure recurrence. *Lancet* **1**: 721–6.
- Hughes JR, John ER (1999) Conventional and quantitative electroencephalography in psychiatry. *Journal of Neuropsychiatry and Clinical Neuroscience* **11**: 190–208.
- Jacobson SA, Leuchter AF, Walter DO (1993) Conventional and quantitative EEG in the diagnosis of delirium among elderly. *Journal of Neurology, Neurosurgery and Psychiatry* **56**: 153–8.
- Jelic V, Kowalski J (2009) Evidence based evaluation of diagnostic of resting EEG in dementia and mild cognitive impairment. *Clinical EEG and Neuroscience* **40**: 129–42.
- John ER, Pritchard LS, Alper KR, et al (1994) Quantitative electrophysiological characteristics and typing of schizophrenia. *Biological Psychiatry* **36**: 801–26.
- Jokic-Begic N, Begic D (2003) Quantitative electroencephalogram (qEEG) in combat veterans with post-traumatic stress disorder. *Nordic Journal of Psychiatry* **57**: 351–5.
- Kaiser DA (2005) Basic Principles of QEEG. *Journal of Adult Development* **12**: 99–104.
- Kanner AM (2003) Depression in epilepsy: prevalence, clinical semiology, pathogenic mechanisms and treatment. *Biological Psychiatry* **54**: 388–98.
- Karson CN, Coppola R, Daniel DG, et al (1988) Computerised EEG in schizophrenia. *Schizophrenia Bulletin* **2**: 193–9.
- Knott VJ, Telner JI, Lapierre YD, et al (1996) Quantitative EEG in the prediction of antidepressant response to imipramine. *Journal of Affect Disorders* **39**: 175–84.
- Lam RW, Hurwitz TA, Wada JA (1988) The clinical use of EEG in a general psychiatric setting. *Hospital and Community Psychiatry* **39**: 533–6.
- Lindau M, Jelic V, Johansson SE, et al (2003) Quantitative EEG abnormalities and cognitive dysfunctions in frontotemporal dementia and Alzheimer's disease. *Dementia and Geriatric Cognitive Disorders* **15**: 106–14.
- Margerison JH, Anderson WM, Dawson J, et al (1962) The relationship between sodium metabolism, verbal output and the EEG in 21 depressives. *Electroencephalography and Clinical Neurophysiology* **14**: 853–7.

- Matsue Y, Okuma T, Hidemitsu S, et al (1986) Saccadic eye-movement in tracking, fixation and rest in schizophrenic and normal subjects. *Biological Psychiatry* 21: 382–9.
- Moeller FG, Gillin JC, Irwin M (1993) A comparison of sleep EEGs in patients with primary major depression and major depression secondary to alcoholism. *Journal of Affective Disorders* 27: 39–42.
- Mulert C, Juckel G, Brunnermeier M, et al (2007) Prediction of treatment response in major depression: integration of concepts. *Journal of Affective Disorders* 98: 215–25.
- Muresanu DF, Alvarez XA, Moessler H, et al (2008) A pilot study to evaluate the effects of cerebrolysin on cognition and qEEG in vascular dementia: Cognitive improvement correlates with qEEG acceleration. *Journal of Neurological Sciences* 267: 112–9.
- Omori M, Koshino Y, Murata T, et al (1995) Quantitative EEG in never treated schizophrenic patients. *Biological Psychiatry* 38: 305–9.
- O'Sullivan SS, Mullins GM, Cassidy EM, et al (2006) The role of standard EEG in clinical psychiatry. *Human Psychopharmacology: Clinical and Experimental* 21: 265–71.
- Pogarell O, Hegerl U, Boutros N (2005) Clinical neurophysiology services in psychiatry departments. *Psychiatric Services* 56: 871.
- Prichip LS, Mas F, Hollander E, et al (1993) Quantitative electroencephalographic subtyping of obsessive compulsive disorder. *Psychiatric Research in Neuroimaging* 50: 25–32.
- Raine A, Venables PH, Williams M (1990) Relationships between N1, P300 and CNV recorded at age 15 and criminal behaviour at age 24. *Psychophysiology* 27: 567–75.
- Reeves RR, Struve FA (2003) Quantitative electroencephalography in patients with late-onset schizophrenia. *International Psychogeriatrics* 15: 273–8.
- Reyes AC, Amador AA (2009) Qualitative and quantitative EEG abnormalities in violent offenders with antisocial personality disorder. *Journal of Forensic and Legal Medicine* 16: 59–63.
- Salisbury DF, Shenton ME, McCarley RW (1999) P300 topography differs in schizophrenia and manic psychosis. *Biological Psychiatry* 45: 98–106.
- Shagass C, Roemer RA, Straumanis JJ, et al (1984) Psychiatric diagnostic discriminations with combinations of quantitative EEG variables. *British Journal of Psychiatry* 144: 581–92.
- Shankman SA, Silverstein SM, Williams LM, et al (2008) Resting electroencephalogram asymmetry and posttraumatic stress disorder. *Journal of Trauma and Stress* 21: 190–8.
- Shelley BP, Trimble MR, Boutros NN (2008) Electroencephalographic cerebral dysrhythmic abnormalities in the trinity of nonepileptic general population, neuropsychiatric, and neurobehavioral disorders. *Journal of Neuropsychiatry and Clinical Neuroscience* 20: 7–22.
- Stone J, Moran G (2003) The utility of EEG in psychiatry and aggression. *Psychiatric Bulletin* 27: 171–2.
- Thomas C, Hestermann U, Kopitz J, et al (2008a) Serum anticholinergic activity and cerebral cholinergic dysfunction: an EEG study in frail elderly with and without delirium. *BMC Neuroscience* 9: 86–97.
- Thomas C, Hestermann U, Walther S, et al (2008b) Prolonged activation EEG differentiates dementia with and without delirium in frail elderly patients. *Journal of Neurology, Neurosurgery and Psychiatry* 79: 119–25.
- Ulrich G, Renfordt E, Frick K (1986) The topographical distribution of alpha-activity in the resting EEG of endogenous-depressive patients with and without clinical response to pharmacotherapy. *Pharmacopsychiatry* 19: 272–3.
- van der Stelt O, Lieberman JA, Belger A (2005) Auditory P300 in high-risk, recent-onset and chronic schizophrenia. *Schizophrenia Research* 77: 309–20.
- Wahlund B, Piazza P, von Rosen D, et al (2009) Serial (ictal) -EEG characteristics in subgroups of depressive disorder in patients receiving electroconvulsive therapy (ECT): a preliminary study and multivariate approach. *Computational Intelligence and Neuroscience* (June 15): Epub: 965209.
- Warner MD, Boutros NN, Peabody CA (1990) Usefulness of EEGs in a psychiatric inpatient population. *Journal of Clinical Psychiatry* 51: 363–4.
- Williams D (1954) The electroencephalogram in affective disorders. *Proceedings of the Royal Society of Medicine* 47: 779–82.
- Williamson PC, Kutcher SP, Cooper PW, et al (1989) Psychological, topographical EEG and CT scan correlates of frontal lobe function in schizophrenia. *Psychiatry Research* 29: 137–49.
- Yener GG, Leuchter AF, Jenden D, et al (1996) Quantitative EEG in frontotemporal dementia. *Clinical Electroencephalography* 27: 61–8.

MCQs

Select the single best option for each question stem

1 Research has shown that:

- a clozapine therapy is a significant predictor of abnormal EEG recordings
- b the false-positive rate for an epileptiform pattern on EEG traces in general population is less than 1%
- c a third of abnormal EEG traces lead to change in diagnosis
- d psychiatric referrals have a high abnormality detection rate because they are extremely selective
- e the most common request for EEG is to determine the presence of organic brain dysfunction.

2 In EEG:

- a auditory stimulation is the most common procedure used in activated EEG
- b the frequency range of recorded waves is between 1 and 20 Hz
- c in old age the trace shows a decrease in delta activity
- d the normal eyes-closed wake EEG trace is characterised by highly rhythmic delta waves
- e standard recording uses 14 electrodes.

3 The earliest change in EEG due to acute confusional state is:

- a the appearance of delta waves
- b generalised theta waves
- c intermittent delta activity
- d slowing of the alpha rhythm frequency
- e triphasic waves.

4 During sleep, the onset of K-complex and spindles is seen in:

- a stage 1 non-REM
- b stage 2 non-REM
- c stage 3 non-REM
- d stage 4 non-REM
- e REM stage.

5 In EEG of people with schizophrenia:

- a increased beta activity is common among patients treated with antipsychotics
- b increased delta activity in the left temporal area is associated with negative symptoms
- c antipsychotic medications decrease alpha activity
- d relatively high mean alpha is a specific finding
- e 6/s spike and wave complex are common in patients with command hallucinations.