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suicidal behaviour in schizophrenia. J Psychopharmacol. 2010;24(5):677. Demers CH, Bogdan R, Agrawal A. The genetics, neurogenetics and pharmacogenetics of addiction. Curr Behav Neurosci Rep. 2014;1–12. Farmer A, Elkin A, McGuffin P. The genetics of bipolar affective disorder. Curr Opin Psychiatry. 2007;20:8. Fears SC, Mathews CA, Freimer NB. Genetic linkage analysis of psychiatric disorders. In: Sadock BJ, Sadock, VA, Ruiz P, eds. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 320. Gianakopoulos PJ, Zhang Y, Pencea N, Orlic-Milacic M, Mittal K, Windpassinger C, White SJ, Kroisel PM, Chow EW, Saunders CJ, Minassian BA, Vincent JB. Mutations in MECP2 exon 1 in classical Rett patients disrupt MECP2_e1 transcription, but not transcription of MECP2_e2. Am J Med Genet B Neuropsychiatr Genet. 2012;159B(2):210. Guerrini R, Parrini E. Neuronal migration disorders. Neurobiol Dis. 2010; 38(2):154. Kumar KR, Djarmati-Westenberger A, Grünewald A. Genetics of Parkinson's disease. Semin Neurol. 2011;31(5):433. Novarino G, El-Fishawy P, Kayserili H, Meguid NA, Scott EM, Schroth J, Silhavy JL, Kara M, Khalil RO, Ben-Omran T, Ercan-Sencicek AG, Hashish AF, Sanders SJ, Gupta AR, Hashem HS, Matern D, Gabriel S, Sweetman L, Rahimi Y, Harris RA, State MW, Gleeson JG. Mutations in BCKD-kinase lead to a potentially treatable form of autism with epilepsy. Science. 2012;338(6105):394. Perisco AM, Bourgeron T. Searching for ways out of the autism maze: Genetic, epigenetic and environmental clues. Trends Neurosci. 2006;29:349. Spors H, Albeanu DF, Murthy VN, Rinberg D, Uchida N, Wachowiak M, Friedrich RW. Illuminating vertebrate olfactory processing. J Neurosci. 2012;32(41):14102. 1.8 Applied Electrophysiology Electroencephalography (EEG) is the recording of the electrical activity of the brain. It is used in clinical psychiatry principally to evaluate the presence of seizures, particularly temporal lobe, frontal lobe, and petit mal seizures (absence seizures), which can produce complex behaviors. EEG is also used during electroconvulsive therapy (ECT) to monitor the success of the stimulus in producing seizure activity, and as a key component of polysomnography used in the evaluation of sleep disorders. Quantitative electroencephalography (QEEG) and cerebral evoked potentials (EPs) represent newer EEG-based methods that provide improved research and clinical insights into brain functioning. ELECTROENCEPHALOGRAPHY A brain wave is the transient difference in electrical potential (greatly amplified) between any two points on the scalp or between some electrode placed on the scalp and

a reference electrode located elsewhere on the head (i.e., ear lobe or nose). The difference in electrical potential measured between any two EEG electrodes fluctuates or oscillates rapidly, usually many times per second. It is this oscillation that produces the characteristic “squiggly line” that is recognized as the appearance of “brain waves.” Brain waves reflect change by becoming faster or slower in frequency or lower or higher in voltage, or perhaps some combination of these two responses. A normal EEG can never constitute positive proof of absence of brain dysfunction. Even in diseases

with established brain pathophysiology, such as multiple sclerosis, deep subcortical neoplasm, some seizure disorders, and Parkinson’s disease and other movement disorders, a substantial incidence of patients with normal EEG studies may be encountered. Nonetheless, a normal EEG can often provide convincing evidence for excluding certain types of brain pathology that may present with behavioral or psychiatric symptoms. More often, information from the patient’s symptoms, clinical course and history, and other laboratory results identifies a probable cause for the EEG findings. EEG studies are often ordered when a pathophysiological process is already suspected or a patient experiences a sudden, unexplained change in mental status. Electrode Placement The electrodes normally used to record the EEG are attached to the scalp with a conductive paste. A standard array consists of 21 electrodes. Placement of the electrodes is based on the 10/20 International System of Electrode Placement (Fig. 1.8-1). This system measures the distance between readily identifiable landmarks on the head and then locates electrode positions at 10 percent or 20 percent of that distance in an anterior–posterior or transverse direction. Electrodes are then designated by an uppercase letter denoting the brain region beneath that electrode and a number, with odd numbers used for the left hemisphere and with even numbers signifying the right hemisphere (the subscript Z denotes midline electrodes). Thus, the O2 electrode is placed over the right occipital region, and the P3 lead is found over the left parietal area (Fig. 1.8-2). FIGURE 1.8-1

International 10–20 Electrode Placement System. (Courtesy of Grass, Astro-Med, Inc. Product Group.) FIGURE 1.8-2 A left-lateral diagram of the head showing the locations of the routine 10–20 electrodes (left-side electrode locations F7 and T3 and the new electrode placement [T1]) in relation to the temporal pole. (Modification of figure reprinted courtesy of Grass, AstroMed, Inc. Product Group.) In special circumstances, other electrodes may be used. Nasopharyngeal (NP) electrodes can be inserted into the NP space through the nostrils and can be closer to the temporal lobe than scalp electrodes. No actual penetration of tissue occurs. These electrodes may be contraindicated with many psychiatric patients displaying behaviors, such as confusion, agitation, or belligerence, which could pull the leads out, possibly lacerating the nasal passage. Sphenoidal electrodes use a hollow needle through which a fine electrode that is insulated, except at the tip, is inserted between the zygoma and the sigmoid notch in the mandible, until it is in contact with the base of the skull lateral to the foramen ovale. Activated EEG Certain activating procedures are used to increase the probability that abnormal discharges, particularly spike or spike-wave seizure discharges, will occur. Strenuous hyperventilation is one of the most frequently used activation procedures. While

remaining reclined with the eyes closed, the patient is asked to overbreathe through the open mouth with deep breaths for 1 to 4 minutes, depending on the laboratory (3 minutes is common). In general, hyperventilation is one of the safest EEG-activating procedures, and, for most of the population, it presents no physical risk. It can pose a risk for patients with cardiopulmonary disease

or risk factors for cerebral vascular pathophysiology, however. Photic stimulation (PS) generally involves placing an intense strobe light approximately 12 inches in front of the subject's closed eyes and flashing at frequencies that can range from 1 to 50 Hz, depending on how the procedure is carried out. Retinal damage does not occur, because each strobe flash, although intense, is extremely brief in duration. When the resting EEG is normal, and a seizure disorder or behavior that is suspected to be a manifestation of a paroxysmal EEG dysrhythmia is suspected, PS can be a valuable activation method to use. EEG recording during sleep, natural, or sedated, is now widely accepted as an essential technique for eliciting a variety of paroxysmal discharges, when the wake tracing is normal, or for increasing the number of abnormal discharges to permit a more definitive interpretation. It has been shown that the central nervous system (CNS) stress produced by 24 hours of sleep deprivation alone can lead to the activation of paroxysmal EEG discharges in some cases. NORMAL EEG TRACING The normal EEG tracing (Fig. 1.8-3) is composed of a complex mixture of many different frequencies. Discrete frequency bands within the broad EEG frequency spectrum are designated with Greek letters. FIGURE 1.8-3 Normal electroencephalogram (EEG) tracings in an awake 28-year-old man. (Reprinted from Emerson RG, Walesak TS, Turner CA. EEG and evoked potentials. In: Rowland LP,

ed. Merritt's Textbook of Neurology. 9th ed. Baltimore: Lippincott Williams & Wilkins; 1995:68, with permission.) Awake EEG The four basic wave forms are alpha, beta, delta, and theta. Highly rhythmic alpha waves with a frequency range of 8 to 13 Hz constitute the dominant brain wave frequency of the normal eyes-closed awake EEG. Alpha frequency can be increased or decreased by a wide variety of pharmacological, metabolic, or endocrine variables. Frequencies that are faster than the upper 13 Hz limit of the alpha rhythm are termed beta waves, and they are not uncommon in normal adult waking EEG studies, particularly over the frontal– central regions. Delta waves (≤3.5 Hz) are not present in the normal waking EEG, but are a prominent feature of deeper stages of sleep. The presence of significant generalized or focal delta waves in the awake EEG is strongly indicative of a pathophysiological process. Waves with a frequency of 4.0 to 7.5 Hz are collectively referred to as theta waves. A small amount of sporadic, arrhythmic, and isolated theta activity can be seen in many normal waking EEG studies, particularly in frontal– temporal regions. Although theta activity is limited in the waking EEG, it is a prominent feature of the drowsy and sleep tracing. Excessive theta in awake EEG, generalized or focal in nature, suggests the operation of a pathological process. With maturation, EEG activity gradually goes from a preponderance of irregular medium- to high-voltage delta activity in the tracing of the infant, to greater frequency and more rhythmic pattern. Rhythmic activity in the upper theta–lower alpha range (7 to 8 Hz) can be seen in posterior areas by early childhood, and, by mid-adolescence, the EEG essentially has the appearance of an adult tracing. Sleep EEG The EEG patterns that characterize drowsy and sleep states are different from the patterns seen during the awake state. The rhythmic posterior alpha activity of the waking state subsides during drowsiness and is replaced by irregular low-voltage theta activity. As drowsiness deepens, slower frequencies emerge, and sporadic vertex sharp waves may appear at central electrode sites, particularly among younger persons. Finally, the progression into sleep is marked by the appearance of 14-Hz sleep spindles (also called sigma waves), which, in turn, gradually become replaced by high-voltage delta waves as deep sleep stages are reached. EEG ABNORMALITIES Apart from some of the obvious indications for an EEG study (i.e., suspected seizures), EEG studies are not routinely performed as part of a diagnostic work-up in psychiatry. EEG, however, is a valuable assessment tool in clinical situations in which the initial presentation or the clinical course appear to be unusual or atypical (Table 1.8-1). Table

1.8-2 summarizes some common types of EEG abnormalities.

Table 1.8-1 Warning Signs of the Presence of Covert Medical or Organic Factors Causing or Contributing to Psychiatric Presentation Table 1.8-2 Common Electroencephalography (EEG) Abnormalities Some psychotropic medications and recreational or abused drugs produce EEG changes, yet, with the exception of the benzodiazepines and some compounds with a

propensity to induce paroxysmal EEG discharges, little, if any, clinically relevant effect is noted when the medication is not causing toxicity. Benzodiazepines, which always generate a significant amount of diffuse beta activity, have EEG-protective effects, so that they can mask alterations caused by concomitant medications (Table 1.8-3). Table 1.8-3 Electroencephalography (EEG) Alterations Associated with Medication and Drugs Medical and neurological conditions produce a wide range of abnormal EEG findings. EEG studies, thus, can contribute to the detection of unsuspected organic pathophysiology influencing a psychiatric presentation (Fig. 1.8-4). Table 1.8-4 lists EEG alterations in medical disorders and Table 1.8-5 lists EEG alterations associated with psychiatric disorders.

FIGURE 1.8-4 Diffuse slowing in a 67-year-old patient with dementia. Six- to seven cycles per second (cps) activity predominates over the parieto-occipital regions. Although reactive to eye closure, the frequency of this rhythm is abnormally slow. (Reprinted from Emerson RG, Walesak TS, Turner CA. EEG and evoked potentials. In: Rowland LP, ed. Merritt’s Textbook of Neurology. 9th ed. Baltimore: Lippincott Williams & Wilkins; 1995:68, with permission.) Table 1.8-4 Electroencephalography (EEG) Alterations Associated with Medical Disorders

Table 1.8-5 Electroencephalography (EEG) Alterations Associated with Psychiatric Disorders

TOPOGRAPHIC QUANTITATIVE ELECTROENCEPHALOGRAPHY (QEEG) Unlike standard EEG interpretation, which relies on waveform recognition, QEEG involves a computer analysis of data extracted from the EEG. Findings are compared with a large population database of subjects without any known neurological or psychiatric disorder as well as QEEG profiles that may be characteristic of some defined diagnostic group. In QEEG, the analog-based electrical signals are processed digitally and converted to graphic, colored topographical displays. These images are sometimes called “brain maps.” Color Plate 1.8-5 illustrates topographic QEEG images of a patient with a closed head injury. QEEG remains primarily a research method, but it holds considerable clinical potential for psychiatry, mainly in establishing neurophysiological subtypes of specific disorders and for identifying electrophysiological predictors of response. Examples of some of the more promising results of QEEG research include the identification of subtypes of cocaine dependence and the subtype most likely to be associated with sustained abstinence; identification of subtypes of obsessive–compulsive disorder (OCD) that predict clinical responsiveness or lack of responsiveness to selective serotonin reuptake inhibitors (SSRIs); and the differentiation between normals, attention–deficit disorder and attentiondeficit/hyperactivity disorder (ADHD), and learning disability subpopulations. QEEG findings in ADHD show that increased theta abnormality frontally may be a strong predictor of response to methylphenidate and other psychostimulants and that favorable clinical responses may be associated with a normalization of the EEG abnormality. CEREBRAL EVOKED POTENTIALS Cerebral EPs are a series of surface (scalp) recordable waves that result from brain visual, auditory, somatosensory, and cognitive stimulation. They have been shown to be abnormal in many psychiatric conditions, including schizophrenia and Alzheimer’s

disease, thus creating difficulty in using cerebral EPs for differential diagnosis purposes. REFERENCES Alhaj H, Wisniewski G, McAllister-Williams RH. The use of the EEG in measuring therapeutic drug action: Focus on depression and antidepressants. J Psychopharmacol. 2011;25:1175. André VM, Cepeda C, Fisher YE, Huynh MY, Bardakjian N, Singh S, Yang XW, Levine MS. Differential electrophysiological changes in striatal output neurons in Huntington's disease. J Neurosci. 2011;31:1170. Boutros NN, Arfken CL. A four-step approach to developing diagnostic testing in psychiatry. Clin EEG Neurosci. 2007;38:62. Boutros NN, Galderisi S, Pogarell O, Riggio S, eds. Standard Electroencephalography in Clinical Psychiatry: A Practical Handbook. Hoboken, NJ: Wiley-Blackwell; 2011. Boutros NN, Iacono WG, Galderisi S. Applied electrophysiology. In: Sadock BJ, Sadock VA, Ruiz P, eds. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2009:211. Gosselin N, Bottari C, Chen JK, Petrides M, Tinawi S, de Guise E, Ptito A. Electrophysiology and functional MRI in postacute mild traumatic brain injury. J Neurotrauma. 2011;28:329. Horan WP, Wynn JK, Kring AM, Simons RF, Green MF. Electrophysiological correlates of emotional responding in schizophrenia. J Abnorm Psychol. 2010;119:18. Hunter AM, Cook IA, Leuchter AF. The promise of the quantitative electroencephalogram as a predictor of antidepressant

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