Chapter 14
Role of Electroencephalography for Cerebral Functions in Neuroanesthesia

Taketoshi Maehara

Abstract Electroencephalography (EEG) is the recording of electrical activity along the scalp. EEG is derived from a summation of postsynaptic potentials in the apical dendrites of the pyramidal neurons of the cerebral cortex. While modern neuroimaging techniques are capable of disclosing subtle brain lesions, EEG is still an important tool for examining measures of consciousness, the sleep cycle, the effects of hypoxia on the human brain, and epileptic activity. In this chapter, we describe basic and general information about EEG, the clinical usefulness of EEG, and characteristic EEG readings. We also briefly review how anesthetic agents affect EEG readings.

Keywords EEG • Anesthesia • ECoG • Sleep cycle • Epilepsy • Consciousness disturbance • Anesthetic agents

14.1 Introduction

Electroencephalography (EEG) is the recording of electrical activity along the scalp, first described by Hans Berger (1873–1941) in a report published in 1929 [1]. Electrical activity was recorded on photographic paper continuously over several minutes. Berger's 1929 report even described the alpha rhythm and alpha blocking response. Throughout the next decade, the 1930s, extensive studies on Berger's reports on human EEG led to fruitful results in the measurement of consciousness, the sleep cycle, the effects of hypoxia on the human brain, and epileptic activity [2]. Electroencephalography used to be the most important method for the diagnosis of cerebral disease, before the advent of computed tomography (CT) and magnetic resonance imaging (MRI). While modern neuroimaging techniques are far superior in depicting subtle brain lesions, EEG is still an important tool for examining cerebral dysfunction.

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H. Uchino et al. (eds.), *Neuroanesthesia and Cerebrospinal Protection*,
DOI 10.1007/978-4-431-54490-6_14

Digital EEG, a modality that brings more useful EEG findings, is now available at most institutes. Digitally obtained EEG signals can be formatted more flexibly and measured more precisely than traditional analog EEG on paper [3]. Electroencephalography also has the potential to allow assessment of brain function, even in patients under anesthesia. Hence, basic and clinical knowledge of EEG is of great help to anesthesiologists in managing patients during surgery.

14.2 Mechanism of Electroencephalography

The electrical charges of the brain are maintained by billions of neurons, which constantly exchange ions with the extracellular environment. Ions of the same charge repel each other. The phenomenon of volume conduction occurs when large numbers of these ions are pushed out from the neuron population at the same time in a wave. Because this wave of ions reaches the electrodes of the scalp from different angles, each electrode registers a different voltage. Electroencephalography measures differences in the recorded voltages between pairs of electrodes over time.

The electrical potentials of single neurons are so small that it takes millions of neurons with similar spatial orientation acting in sync to product a readable EEG signal. Neurons with different spatial orientations cannot easily produce the volume conduction effect. Most EEG signals are thought to be produced by the pyramidal neurons of the cortex, as these neurons are well aligned and fire together. Electroencephalography signals are derived from the summation of postsynaptic potentials in the apical dendrites of the pyramidal neurons of the cerebral cortex [4].

14.3 Normal EEG

In conventional scalp EEG, electrode locations and names are specified by the International 10–20 system. The EEG voltage signal at each channel reflects a difference between voltages at two electrodes. Montages are representations of EEG channels in series used for examinations (Fig. 14.1). In a referential montage, each channel represents the difference between two reference electrodes (usually at the earlobes: A1, left ear; A2, right ear). In a bipolar montage, each channel represents the difference between two electrodes positioned next to each other. In an average reference montage, the outputs of all of the amplifiers are summed and averaged for use as a common reference [4].

An EEG is composed of frequencies and amplitudes. In the normal adult, the slow range (0.3–7 Hz) and the very fast range (>30 Hz) are little represented. Medium (8–13 Hz) and fast (14–30 Hz) are predominant. These frequencies are called delta wave (0.1–3.5 Hz), theta wave (4–7.5 Hz), alpha wave (8–13 Hz), beta wave (14–30 Hz), and gamma wave (>30 Hz) [4, 5].

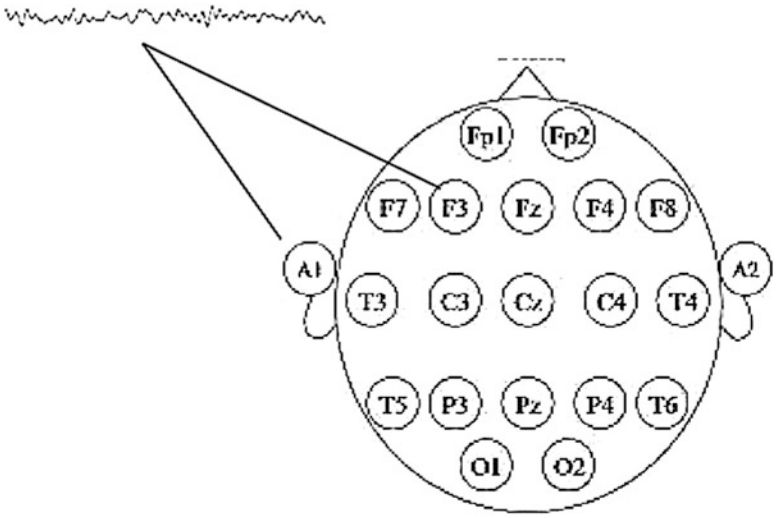

Fig. 14.1 EEG between F3 and A1 in the International 10–20 system

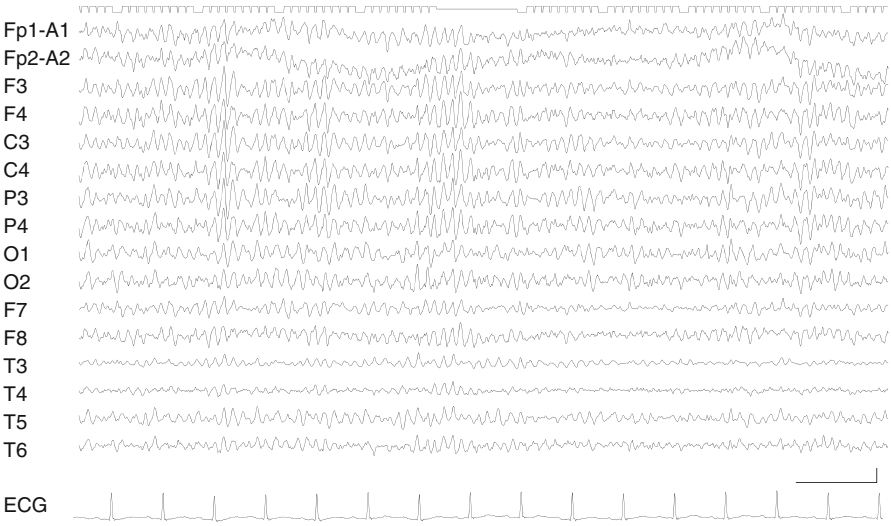

Fig. 14.2 Normal EEG of a waking adult with eyes closed (referential montage). Alpha rhythm is observed over the posterior part of the head

The EEG amplitudes are measured from peak to peak. The amplitudes of the scalp EEG usually lie in a range from 10 to 50 μV in adults. In a normal adult, alpha rhythm is observed over the posterior part of the head when the individual is awake with his or her eyes closed (Fig. 14.2).

14.4 Scalp EEG and Electrocorticography (ECoG)

EEG signals recorded on the cerebral cortex (ECoG) have much larger amplitudes and are sharply demarcated. As higher-frequency activity is attenuated to a greater extent than slower frequencies during transmission to the scalp, most rhythms tend to have a much sharper appearance in ECoG than in EEG. The ECoG contains more prominent and abundant beta potentials than EEG. The amplitudes of ECoG discharges range from 0.5 to 1.5 mV, that is, 5–10 times higher than the amplitudes of EEG [6].

14.5 Sleep Cycle

To avoid misdiagnosis, a physician should understand the characteristic EEG patterns and waves during sleep cycles before attempting to detect EEG abnormalities. Under the classification first applied after the discovery of REM sleep in 1953 [7], sleep stages were divided into non-REM sleep (slow-wave sleep) and REM sleep (paradoxical sleep of fast sleep). Under the present classification, sleep stages and their characteristic waves are designated as follows: Stage 1 (drowsiness), from alpha dropout to vertex waves; Stage 2 (light sleep), spindles, vertex waves, and K complexes (Fig. 14.3); Stage 3 (intermediate sleep), much slowing, K complexes, and some spindles; Stage 4 (deep sleep), much slowing and some K complexes; Stage REM (REM sleep), desynchronization with faster frequencies [5].

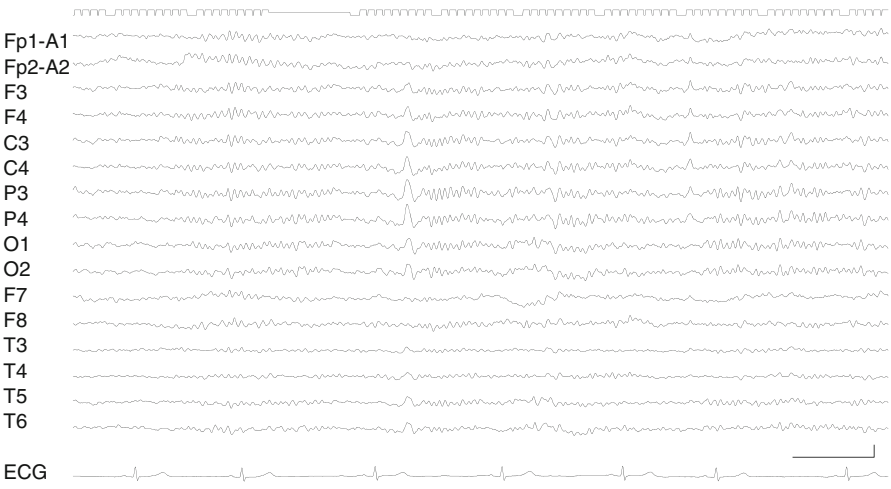

Fig. 14.3 Non-REM (Stage 2) EEG in a normal adult (referential montage). Vertex wave and spindles are observed

14.6 Clinical Use

EEG is an important tool for diagnosing various cerebral conditions such as epilepsy or consciousness disturbances such as encephalopathy, drug intoxication, or delirium. Cerebral ischemic change, severe head trauma, and increased ICP all cause neuronal damage. Decreased EEG frequency, a consequence of attenuated alpha rhythm and enhanced delta and theta waves, therefore serves as a marker of cerebral dysfunction. EEG is used as an adjunct test of brain death [4]. These applications are described in detail in other chapters of this book.

14.6.1 Diagnosis of Epilepsy

EEG is typically described in terms of basic rhythm and transient discharges. Abnormal EEG activity is broadly divided into epileptiform and non-epileptiform activity. It can also be classified as focal and diffuse abnormality. Most patients with temporal lobe epilepsy, for example, show interictal epileptic discharges during Stage 1 or 2 (Fig. 14.4). Activations, such as eye closure, photic stimulation, and hyperventilation, are also used to detect epileptic abnormality. Ictal EEG is an important technique for detecting epileptogenic areas of epileptic brains (Fig. 14.5).

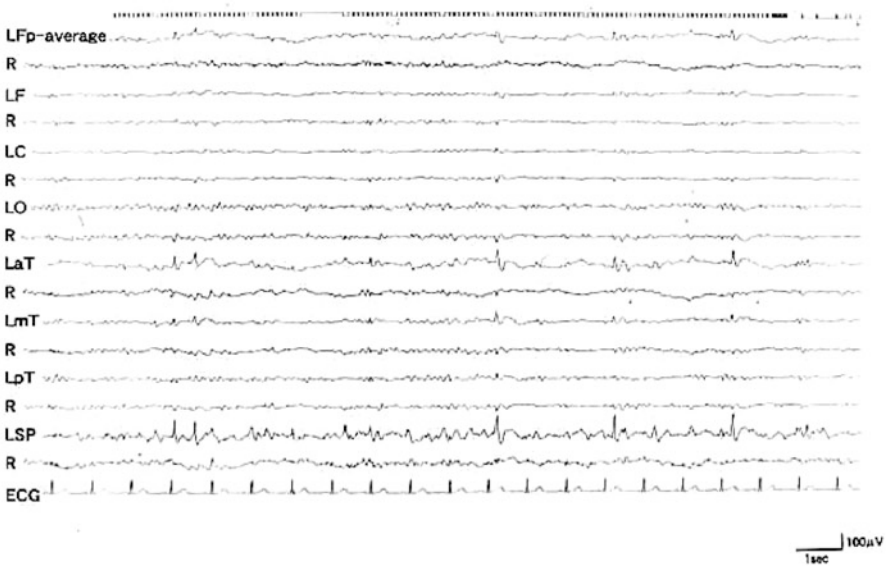

Fig. 14.4 Interictal EEG of a patient with right temporal lobe epilepsy (average reference montage). Spikes are frequently recorded in the right anterior temporal area, especially in the right sphenoidal (SP) electrode

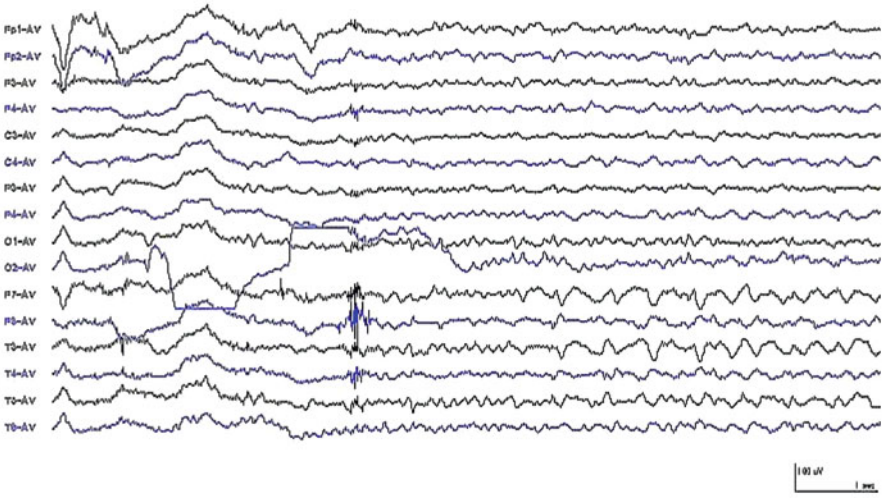

Fig. 14.5 Ictal EEG of a patient with left temporal lobe epilepsy (average reference montage). A rhythmic delta wave is observed in the left temporal area

Intraoperative ECoG is used to define and delineate epileptogenic areas during epilepsy surgery. Volatile anesthetics (desflurane, sevoflurane, halothane, enflurane, and isoflurane) have different pro- and anticonvulsant effects (described later in this chapter). Sevoflurane at 1.5 MAC is effective in enhancing the spike activity of epileptogenic areas during epilepsy surgery [8].

Status epilepticus is caused by various conditions such as encephalopathy, metabolic disease, or acute AED withdrawal during epilepsy treatment. In patients suffering from status epilepticus, rapid seizure cessation is crucial for the prevention of brain damage. Under the recent criteria, a seizure lasting more than 5 min should be regarded as status epilepticus. When intravenous phenobarbital or phenytoin is ineffective, general anesthesia is employed in intensive care units under EEG monitoring setup for burst-suppression activity [9].

14.6.2 Consciousness Disturbance

EEG is an important examination in some patients with consciousness disturbances, even in those without structural brain lesions.

Nonconvulsive status epilepticus (NCSE) is one of the major diseases treated in the neuro ICU. EEG is mandatory for the accurate diagnosis and treatment of NCSE, as it clearly shows ictal epileptic discharges (Fig. 14.6). Consciousness disturbances of various degrees can also result from hepatic encephalopathy caused by liver dysfunction, a disorder of the metabolic central nervous system. Patients with hepatic encephalopathy typically manifest massive EEG slowing, with or

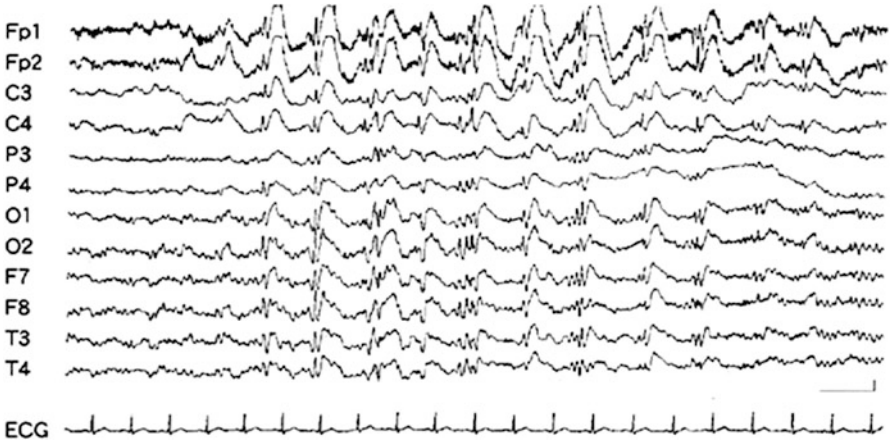

Fig. 14.6 EEG of nonconvulsive status epilepticus in a patient with metabolic disorder (referential montage). Bilateral synchronized spike and waves are continuously recorded

without triphasic waves (Fig. 14.7). Benzodiazepines, a class of agents commonly used as short- and long-term sedatives, are also used for seizure control. The beta activation following administration of benzodiazepines is similar to that seen with barbiturate. This beta activation should not be interpreted as an arousal reaction [10] (Fig. 14.8). Delirium is characterized by an acute change of mental status and consciousness. It is an especially perilous condition in elderly patients during hospital stays or after operations. Slow waves and alpha waves are both observed in EEG. Lastly, EEG is extremely useful for distinguishing delirium from NCSE or psychiatric disorders [11] (Fig. 14.9).

14.7 Effect of Anesthetic Drugs

Brain electrical activity is directly influenced by anesthetic drugs such as anesthetic gases, hypnotics, opioids, sedatives, and muscle relaxants. The effects of these drugs on CBF, CBV, and ICP may also be linked with indirect neuronal effects that alter EEG findings. Most anesthetic agents elicit changes in both the frequencies and amplitudes of EEG signals, usually in a dose-effective manner [10].

14.7.1 General EEG Changes

EEG findings change according to the depth of anesthesia. Desynchronization first appears in the excitatory phase, and then synchronization follows in the early stages of anesthesia. Further slowing and increased suppression follow in the surgical

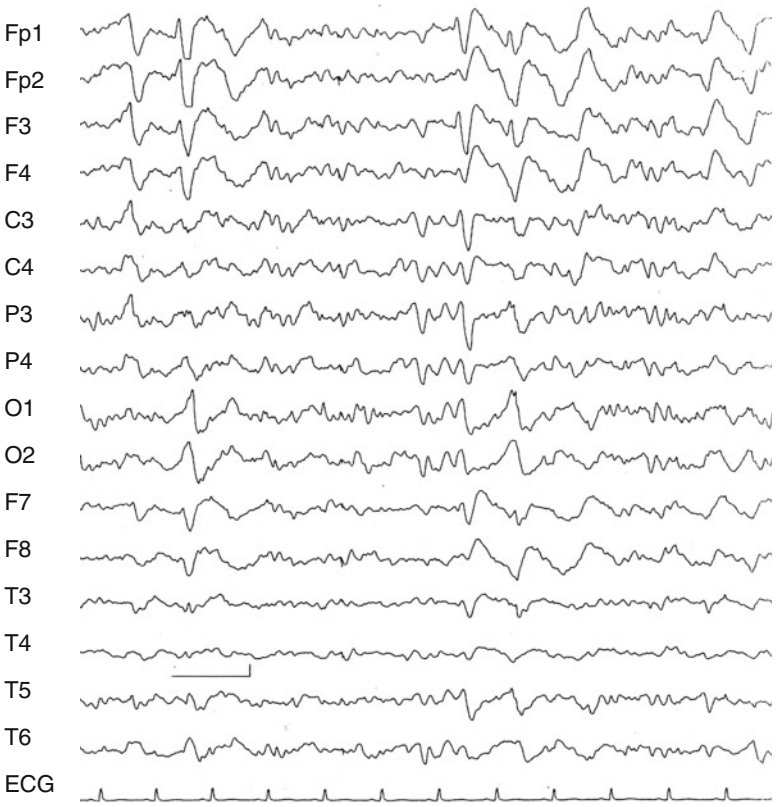

Fig. 14.7 EEG of a patient with hepatic encephalopathy (referential montage). Triphasic waves are observed in the anterior half of the brain

tolerance stage. Burst suppression manifests in an early overdose stage, and general suppression results if the dose reaches a toxic level. As to frequency changes, a loss of alpha and induction of beta component are recorded in the desynchronization state. Synchronization with higher amplitude alpha and theta component follows.

Several drugs elicit exceptional EEG activity at the initial stage. Ketamine elicits a high-amplitude theta activity superimposed with low-amplitude beta components. Propofol induces an initial increase in beta activity followed by increases of theta and delta waves after the onset of sleep. Barbiturates induce an initial increase in fast activity with spindles on EEG.

The volatile anesthetics desflurane, sevoflurane, halothane, enflurane, and isoflurane produce fast wave EEG activity at subanesthetic concentrations followed by slow components with the onset of unconsciousness. Patients given nitrous oxide (NO) may manifest a loss of alpha frequencies at NO concentrations of 30–40 %, followed by increased beta activity at concentrations around 50 % [12]. Later, at NO concentrations of 75–80 %, predominant theta components gradually appear [13].

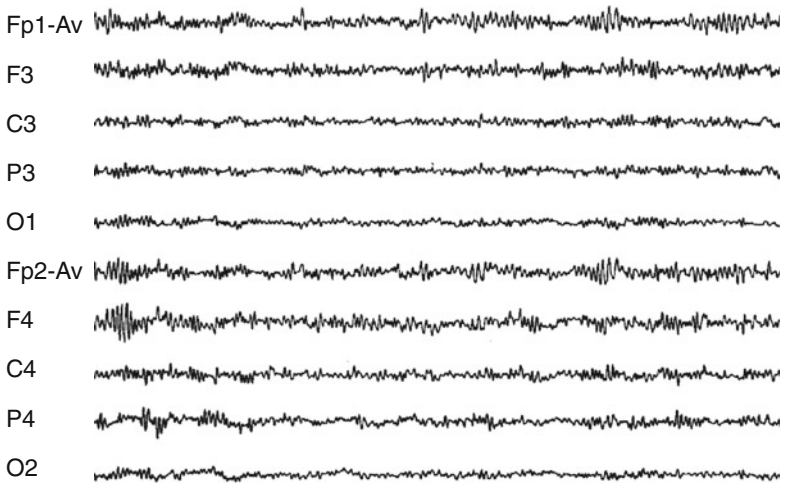

Fig. 14.8 EEG of a patient with benzodiazepine intoxication (average reference montage). Beta waves are diffusely observed

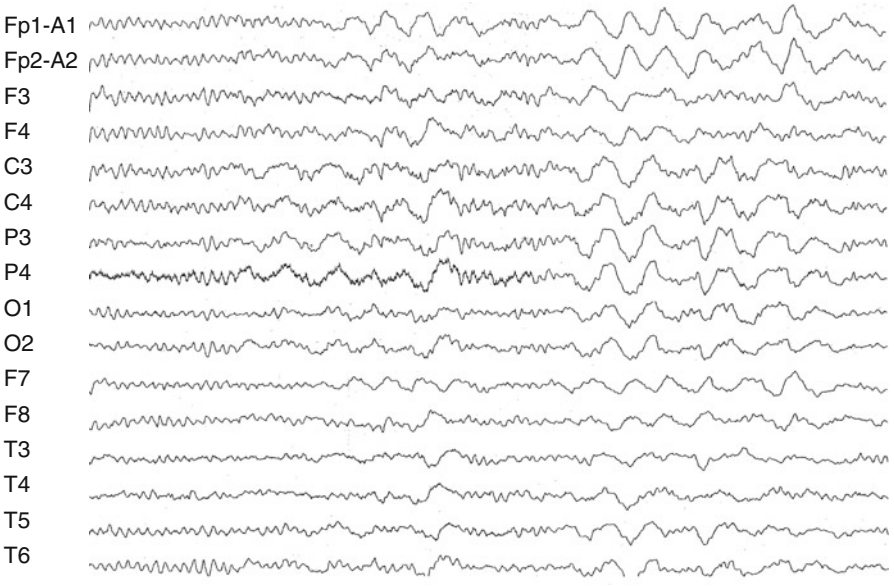

Fig. 14.9 EEG of a patient with delirium (referential montage). Both slow waves and alpha waves are observed in EEG

The EEG activity returns to baseline within 1 h following cessation of NO use. Halothane leads to a progressive slowing of EEG frequencies as the drug concentration rises [14]. At subanesthetic concentrations (<1.2 %) of isoflurane, the EEG frequency is increased, and voltage is slightly decreased.

Opioids cause a dose-dependent slowing of the EEG without initial excitation [15]. Diazepam reduces activity in the theta band and increases activity in the beta band, but the beta activation is lost in the presence of NO, opioids, or volatile anesthetics. Muscle relaxants seem to have no effect on the EEG signals.

14.7.2 Pro- and Anticonvulsive Properties

Halothane elicits no apparent enhancement of epileptiform activity [16]. Isoflurane is also thought to lack seizure-inducing effects. Propofol has anticonvulsant actions [17] and is successfully used as a continuous infusion for the treatment of refractory status epilepticus. Muscle relaxants seem to lack both pro- and anticonvulsant properties.

Unlike the agents just mentioned, there are other anesthetic drugs that may be capable of inducing seizures. Enflurane may cause generalized seizures with characteristic tonic-clonic activity and high-voltage EEG activity in humans, even at concentrations below those necessary for anesthesia [18]. Higher doses (above 4–5 mg/kg) of ketamine are known to lower the epileptic threshold. Sevoflurane has epileptogenic properties that serve well during epilepsy surgery [8].

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