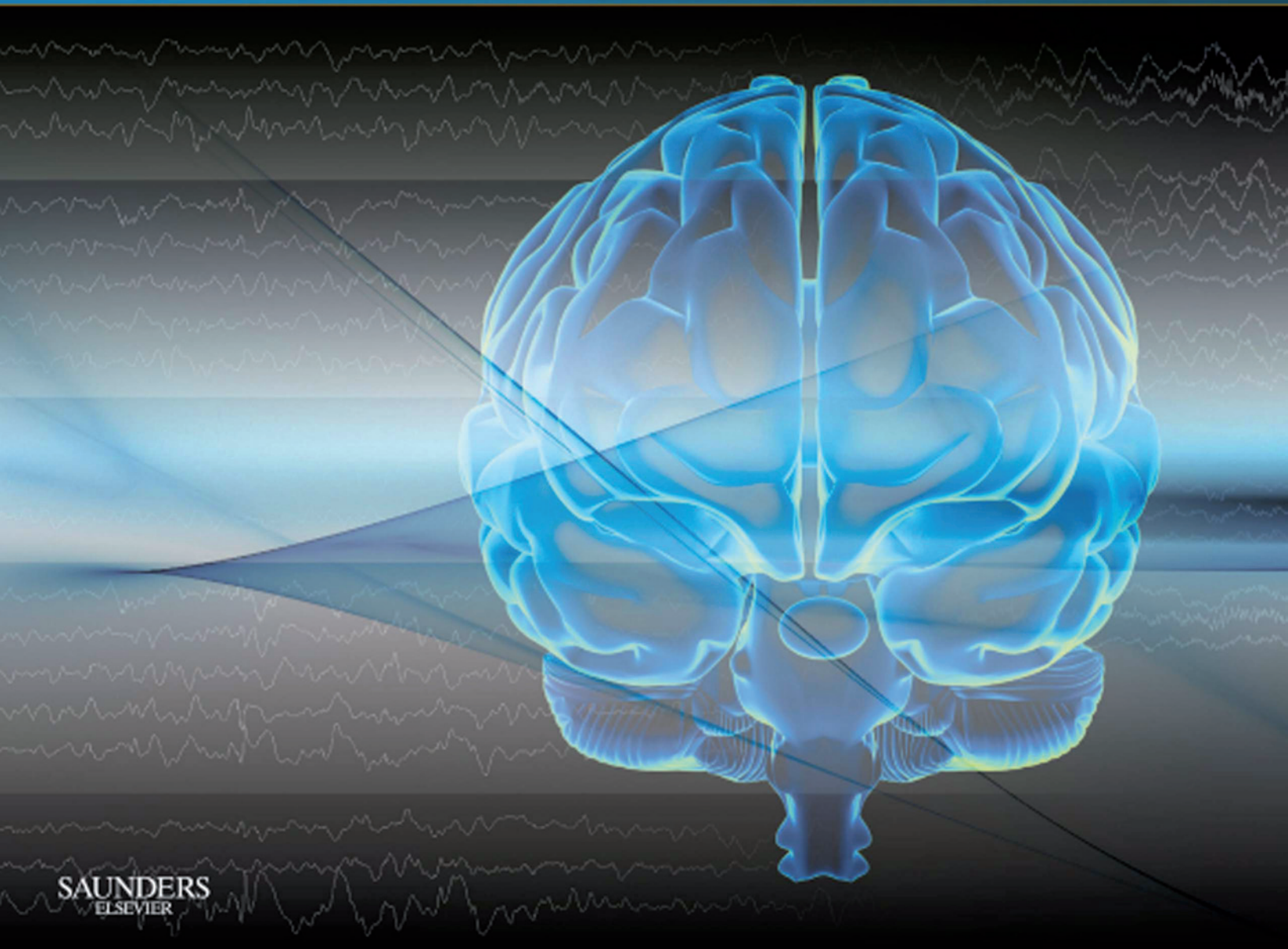
MARK LIBENSON

PRACTICAL APPROACH TO Electroencephalography



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PRACTICAL APPROACH TO ELECTROENCEPHALOGRAPHY

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PRACTICAL APPROACH TO ELECTROENCEPHALOGRAPHY ISBN: 978-0-7506-7478-2

Library of Congress Cataloging-in-Publication Data

Libenson, Mark H.
Practical approach to electroencephalography/Mark H. Libenson. — 1st ed.
p. ; cm.
Includes bibliographical references.
ISBN 978-0-7506-7478-2
1. Electroencephalography. I. Title.
[DNLM: 1. Electroencephalography. WL 150 L694p 2009]
RC386.6.E43L53 2009
616.8'047547—dc22

2009039416

Acquisitions Editor: Adrianne Brigido
Developmental Editor: Taylor Ball
Design Direction: Steven Stave

Printed in the United States of America

Last digit is the print number: 9 8 7 6 5 4 3 2 1

To my mother, **Pearl**; my wife, **Lisa**; and my children, **Sara** and **Andrew**, in thanks for their love and support

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Foreword

During my neurology training in the 1970s, I learned a great deal from my mentors about how to take care of patients with neurological disorders. In those pre–computed tomography (CT) and magnetic resonance imaging (MRI) days we depended a great deal on the history and neurological examination and the most valuable ancillary test available, the electroencephalogram (EEG). In addition to helping us determine whether the patient had epilepsy, the EEG provided us information regarding the presence or absence of structural lesions, the cause of the metabolic encephalopathy, or the type of encephalitis the patient had. With the advent of CT and MRI we are now able to image the brain in exquisite detail. The arrival of these neuroimaging techniques led some of my teachers to suggest that the EEG would be relegated to a secondary role in the evaluation of patients with neurological disorders. While my professors were correct about many things, in this case they were mistaken.

Even as embracing new imaging technologies is warranted, it is now clear that the EEG is becoming more important, not less, in the assessment of individuals with a wide variety of neurological disorders. Rather than looking at structure at a fixed moment in time, electroencephalography allows one to assess physiological function over time and across sleep and wake states. This ability to assess physiological function second by second over extended periods of time provides data that simply are not matched by those of other technologies.

Although the EEG was discovered in the 1920s by Hans Berger, the EEG has not remained static, but rather new technology has been added and it has matured into a time-tested diagnostic test. The digitalization of analogue signals has dramatically increased the information base and flexibility of EEG, greatly expanding the usefulness of the test. While collecting and displaying this enormously powerful data has become easier, the challenge for physicians is to correctly interpret the findings.

It is therefore timely that Mark Libenson has written *Practical Approach to Electroencephalography,* a book beautifully designed to teach learners to understand both the physiological and technological basis of the study and to interpret the findings. While there are other excellent authoritarian textbooks on electroencephalography available, this book fills an important gap by providing a practical approach to how to evaluate the EEG. For the individual new to the field, the book provides an excellent starting point, taking the reader from EEG basic principles to constructing a useful report. Clearly written and beautifully illustrated with more than 450 high-quality figures, the book is a pleasure to read and study.

While EEG can be useful in the evaluation of patients with a wide gamut of neurological disorders, it is most widely used in epilepsy. The EEG can provide information on seizure type, epileptic syndrome, and etiology and is widely used for localization of the seizure onset zone. Understanding the EEG signature of the epileptic syndromes is a critical step in the diagnosis and management of epilepsy. Knowing the importance of epileptic syndromes, Dr. Libenson has devoted an entire chapter to epilepsy, emphasizing the EEG findings of the epilepsy syndromes.

There is little doubt that this book will be widely studied. I plan to recommend it for our neurology residents and clinical neurophysiology fellows. However, this book is not just for trainees; seasoned veterans will learn a lot from Dr. Libenson. I know I did.

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Acknowledgments

Practical Approach to Electroencephalography is based on a personal experience of learning, practicing, and teaching Clinical Neurophysiology and Neurology; as such, many of the ideas presented here reflect concepts shared with me or taught to me by others. I consider myself fortunate to have learned the art and science of reading EEGs from a group of extraordinary neurophysiologists, including Drs. Gregory Holmes, Edward Bromfield, and Bruce Ehrenberg. I am indebted to many colleagues, present and past, at the Floating Hospital for Children at Tufts Medical Center and at Children's Hospital Boston, for what they have taught me. I am also grateful for the expertise of the EEG technologists of those institutions where many of the tracings used in this text were recorded. In particular, I am thankful for the inspiration and generosity of my chiefs, Drs. Paul Rosman, Joseph Volpe, Blaise Bourgeois, and Scott Pomeroy.

Dr. David Preston has provided guidance, assistance, and invaluable inspiration at all stages of this project, and I am deeply grateful for his helpful review of and suggestions for several of the chapters. I would also like to thank Jeffrey Marcus, PhD, for his advice and expertise in the fields of electronics and digital signal processing.

I wish to thank my editors, Ms. Susan Pioli, who helped conceive the form of this project with me, and Ms. Adrianne Brigido, Mr. Taylor Ball, and the staff members of Elsevier, who helped guide this project to completion. I am grateful for the efforts of Ms. Alison Clapp, librarian at Children's Hospital Boston, for her ability to track down obscure materials with impressive speed. The administrative support of Ms. Alison Armstrong, Ms. Jenna Koretz, and Ms. Jacqueline Powers was instrumental in allowing me to carry out both my clinical responsibilities and writing simultaneously. Special thanks are due to Ms. Tristan Aspri for her excellent administrative skills and for her assistance in the preparation of portions of the manuscript and help in organizing many of the figures in this text.

I would like to thank the many fellows, residents, and students whom I have taught and who have taught me, often by making insightful observations and posing questions that have helped me to think through some of the difficult concepts of Clinical Neurophysiology. Finally, I would like to thank my wife, Lisa Muto. Without her support—both emotional and editorial—this project would not have been possible.

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Chapter

1 Introduction

Electroencephalography (EEG) remains one of the principle tools in the practice of clinical neurology. Notwithstanding the development of increasingly sophisticated imaging techniques over the years, including computed tomography (CT) scanning, magnetic resonance imaging (MRI), nuclear medicine imaging, and newer functional MRI techniques, the use of EEG in the assessment of neurological disorders continues to increase. The information learned from EEG testing differs in a number of ways from what is learned from radiological images such as MRI scans. Whereas MRI primarily furnishes anatomical information as a snapshot in time, EEG captures electrical information over time. Just as an X-ray image of the heart does not tell us whether a patient's heart is beating quickly or slowly, information easily learned from an electrocardiogram, likewise CT and MRI scans of the brain do not give direct information regarding electrical irritability in the brain or even indicate whether the patient is awake or asleep. Thus radiological imaging studies and electroencephalography are complementary techniques.

Electroencephalography is used in a variety of clinical situations, but the large majority of EEGs are obtained as a part of the evaluation of seizures or epilepsy. Estimates of the population prevalence of epilepsy range between 0.5% and 1%, suggesting that at least 2 million people in North America have epilepsy. EEG is also useful in the evaluation of confusional states and coma, and it can play an important role in separating psychiatric illness from organic disease.

Electroencephalography is a relatively young science. One hundred years ago, it was not yet a settled fact that there was electrical activity in the human brain. In 1875, Richard Caton was the first to report an observation of electrical activity from the brains of monkeys and rabbits, though techniques available at the time did not allow him to record these waveforms for posterity. Caton made his observations using a device called Thomson's mirror galvanometer (Caton, 1877). Oscillations in the mirror affixed to a galvanometer caused movements of a beam of light the mirror reflected on the wall of his laboratory. His report that "Feeble currents of varying direction pass through the multiplier when the electrodes are placed on two points of the external surface, or one electrode on the grey matter, and one on the surface of the skull" is considered the first description of an EEG wave (Caton, 1875). Thereafter, successful recordings were made by Caton and others from the brains of dogs, monkeys, rabbits, and cats, although some still claimed that the recorded waves were related to the pulsations of cerebral blood vessels rather than to brain electrical activity.

Hans Berger, considered the father of modern Electroencephalography, was the first to record EEG in humans (see Figure 1-1) while working as a professor of psychiatry at the University of Jena in Germany. His previous work included precise measurements of cerebral pulsations in both animals and humans and, later, the measurement of brain temperature variations in animals to determine whether temperature fluctuated in different behavioral states. His first attempts at recording brain waves in 1924 were carried out using a string galvanometer designed to record electrocardiograms (see Figure 1-2). The initial recordings were made in subjects with areas of missing cranial bone, either from palliative trepanations (creation of a window in the skull bone) for relief of increased intracranial pressure from brain tumors or from skull defects related to injuries sustained during the First World War. Because of these patients' skull defects, the needle electrodes he used could be placed only a few millimeters away from the brain surface.

In his first report, titled "On the Electroencephalogram of Man" and published in 1929, Berger outlines the path toward his first successful observation of the EEG of man (which he did not, at the time, have the equipment to record). The observation was made in 1924 in a 17-year-old boy who had undergone palliative trepanation over the left cerebral hemisphere for a brain tumor. The first published recorded rhythm, shown in Figure 1-3, was obtained in a 40-year-old man who had had a large bone flap removed to relieve pressure from a brain tumor. The recording was made with needle electrodes placed subcutaneously which, in this patient, represented the epidural space. The patient died from his tumor a few weeks later.

Berger's initial work was met with considerable skepticism, in part because the action potential of single nerves was just then under study. It was difficult to reconcile the short duration of the action potential, approximately 1 millisecond, with the much longer duration of the waves that comprised the "Berger Rhythm," the wavelengths of which were closer to 100 milliseconds.

Figure 1-1 Hans Berger at age 52 (1925), one year after he began his work on the human electroencephalogram. *(Courtesy Mrs. Ursula Berger. With permission, from Berger H, Gloor P.* On the electroencephalogram of man; the fourteen original reports on the human electroencephalogram *[Gloor P, translator and editor], Amsterdam and New York, Elsevier, 1969.)*

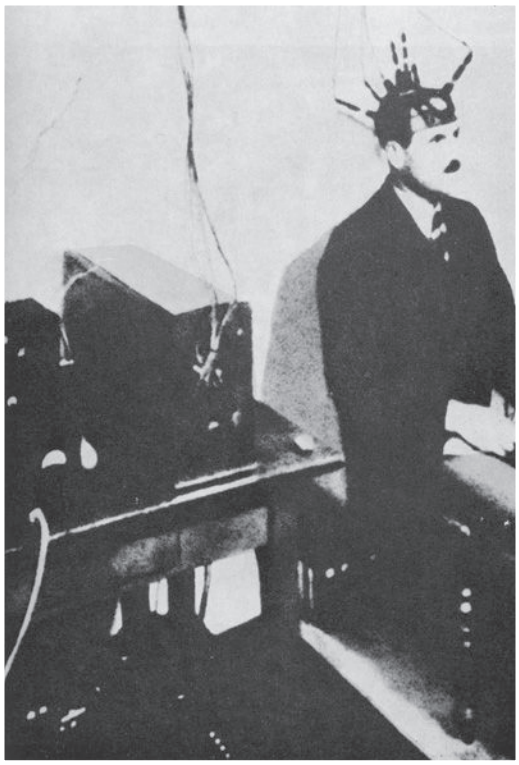

Figure 1-2 This photograph shows one of the first attempts at recording the electroencephalogram in humans. The patient had undergone a left-sided trepanation. The recording attempt was made with silver electrodes secured to the scalp with adhesive tape but was unsuccessful. This patient was recorded sitting up; later patients were studied lying on a couch with glass legs for electrical isolation. *(With permission, from Berger H, Gloor P.* On the electroencephalogram of man; the fourteen original reports on the human electroencephalogram *[Gloor P, translator and editor], Amsterdam and New York, Elsevier, 1969.)*

Berger also attempted recordings in subjects with intact skulls, starting as early as 1920 with unsuccessful attempts in a bald medical student "who put himself most obligingly" at Berger's disposal. The first series of successful recordings made in a subject with an intact skull were obtained from Berger's son, Klaus, when he was aged between 15 and 17 years (see Figure 1-4).

PLAN OF THE TEXT

The chapters in this text are ordered so that they can be read sequentially, although individual chapters may be referenced out of turn as necessary. Chapter 2, "Visual Analysis of the Electroencephalogram," begins with a brief overview of the appearance of the normal EEG during wakefulness, drowsiness, and sleep. After the basic visual features of the normal EEG are described, a discussion of EEG terminology follows. The vocabulary of EEG is intertwined with certain EEG concepts; a review of EEG terminology and several associated EEG concepts are discussed together in Chapter 3, "Introduction to Commonly Used Terms in Electroencephalography."

Chapter 4, "Electroencephalographic Localization," is the most important chapter in this text. Without excellent localization skills, it is impossible to become an excellent electroencephalographer. EEG localization techniques tell us much more than the location from which a wave originates. Localization techniques allow us to translate a group of waves on an EEG page into a three-dimensional map of charge on the scalp surface and to understand how the shapes on that map vary over time.

The ordering of the display of EEG channels generated from a specific electrode set is called the EEG montage. Montages are discussed in detail in Chapter 5,

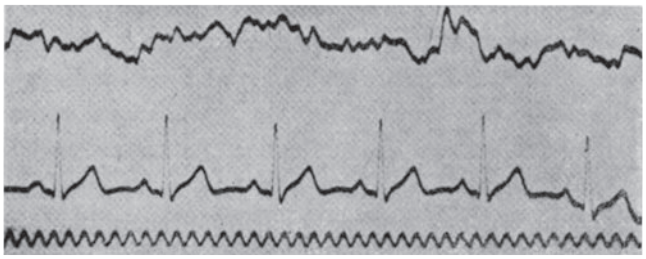

Figure 1-3 The first published recording of an electroencephalogram in a human. The top trace shows an electroencephalographic signal recorded from two needle electrodes in the area of a large bone defect. The middle trace shows the electrocardiogram and the bottom trace shows a 10-Hz calibration signal. *(With permission, from Berger H, Gloor P.* On the electroencephalogram of man; the fourteen original reports on the human electroencephalogram *[Gloor P, translator and editor], Amsterdam and New York, Elsevier, 1969.)*

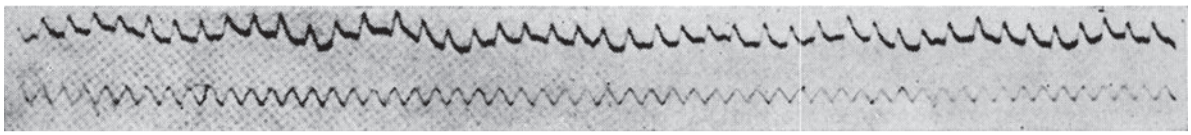

Figure 1-4 The first successful recordings of the EEG in man recorded from a patient with an intact skull were made in Hans Berger's son, Klaus, using needle electrodes. This particular example was obtained when Klaus was 16 years old. The top trace of this figure shows an EEG signal derived from a pair of electrodes "in the midline of the skull anteriorly within the hair line of the forehead and posteriorly two finger breadths above the external occipital protuberance." The bottom channel shows a 10 Hz calibration signal. *(With permission, from Berger H, Gloor P.* On the electroencephalogram of man; the fourteen original reports on the human electroencephalogram *[Gloor P, translator and editor], Amsterdam and New York, Elsevier, 1969.)*

"EEG Electrodes, Channels, and Montages and How They Are Chosen." Different montage strategies help the electroencephalographer to understand the distribution and polarity of different types of discharges. Visualizing these three-dimensional maps is not simply an academic exercise. The ability to classify specific EEG events as to whether they are normal or associated with disease depends significantly on the topography suggested by their polarities and localizations. The topography of an EEG event indicates whether it was generated in a specific part of the brain—or whether it originated from the brain at all. This latter possibility, that a wave on the EEG page has not come from the brain at all, brings us to a major topic in electroencephalography: the identification of EEG artifacts.

One of the main attractions of learning EEG interpretation is the prospect of understanding more about the phenomenology of the wave patterns that the brain generates. It may come as a surprise to the new student of electroencephalography how much time is spent analyzing and identifying waves in the EEG that are caused by the patient tossing and turning or blinking the eyes: so-called EEG artifacts, which is the topic of Chapter 6, "Artifacts." The considerable problem of identifying artifacts in the EEG stems from the high amplifier gains necessary to record the microvolt-level EEG signals that are the object of our interest. A by-product of the high levels of amplification necessary to produce the beautiful EEG traces to which we have become accustomed is that everything gets amplified, including noncerebral electrical events. Experienced electroencephalographers will often be heard to say that "half of EEG is correctly identifying the artifacts." Although many artifactual waves are obviously not of cerebral origin, some may closely mimic true brain wave activity. Artifact recognition involves both pattern recognition and careful localization and topographic description of the wave in question, a skill that is absolutely essential in EEG interpretation.

All EEG signals are passed through a set of filters before being displayed. As discussed in Chapter 7, "Filters in the Electroencephalogram," filters may have a significant impact on the appearance of EEG waves, with the potential to enhance, suppress, or distort EEG information. Although knowledge of filter circuit design is not required for fundamental EEG interpretation, the electrical and digital strategies used to create simple high and low filters are also reviewed. Those not inclined to technical discussions may decide to postpone review of this portion of Chapter 7 until they are more comfortable with general EEG interpretation.

Often the only representation to the outside world of the time and thought that the electroencephalographer has put into the interpretation of the EEG is the written EEG report. The EEG report must include a distillation of the technique used to record the EEG, what the EEG tracing looked like and, most importantly, the EEG findings and their clinical implications. Strategies for producing useful EEG reports are discussed in Chapter 8, "The Structure and Philosophy of the EEG Report."

Before the advent of modern neuroimaging, EEG was an important tool for localizing anatomical lesions such as brain tumors and strokes in addition to its role in diagnosing seizure disorders. Although EEG abnormalities can still be divided into epilepsy- and nonepilepsy-related groups, the evaluation of possible seizure disorders has become the most common reason for EEG testing. Chapter 9, "The Abnormal Electroencephalogram," discusses both epileptic and nonepileptic EEG abnormalities in the EEG and describes the types of clinical disorders associated with each. Chapter 10, "The Electroencephalogram in Epilepsy," examines these associations from the opposite point of view, reviewing selected epilepsy syndromes and describing the EEG findings associated with each.

Over the past 70 years, a variety of EEG findings that were initially felt to be abnormal because of their resemblance to epileptiform abnormalities have been discovered to occur with significant frequency in the normal population and are, therefore, classified as "normal variants." The clinical significance of some of these variants is still under debate. This interesting group of EEG findings is discussed in Chapter 11, "Normal Variants in the Electroencephalogram."

EEG is an important tool for assessing neurological prognosis in coma. Although EEG is not used as a sole criterion for the determination of brain death, it is sometimes used as a confirmatory study in patients in whom brain death is suspected. Chapter 12, "Electroencephalogram Patterns in Stupor and Coma," discusses how the EEG can be used to evaluate patients in coma as well as the technical requirements for performing recordings in the setting of suspected brain death.

The EEG begins to attain adult patterns in patients as young as 2 months of age. Before that point, however, the form of the EEG in babies is remarkably unlike

that of older patients. EEG patterns evolve with surprising rapidity from the earliest clinical recordings obtained at 23 to 24 weeks postconceptional age, to recordings performed in newborns at term (40 weeks postconceptional age), and finally when adult patterns appear. Chapter 13, "The Electroencephalogram of the Newborn," provides an introduction to the unique EEG patterns seen in newborns.

Much like driving a car, electroencephalography cannot be learned just by reading a textbook. While reading this text, the reader is encouraged to interpret as many EEG records as possible. Many students start to learn EEG record review by reading "over the shoulder" of a more experienced EEG reader. The real work of learning electroencephalography is done by "getting behind the wheel" and reading records alone, generating an opinion of the EEG's findings uninfluenced by the opinion of others, and then comparing the results to those of a more experienced reader.

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Chapter

2 Visual Analysis of the EEG: Wakefulness, Drowsiness, and Sleep

An orderly approach to visual analysis of the EEG is important, especially for those who are beginning to hone their EEG reading skills. Although not all EEG records necessarily lend themselves to a single reading approach, it is useful to start the process of record interpretation with a preplanned analysis strategy that is based on the findings of a typical EEG, such as the EEG of a normal adult or child. The approach can be modified from this starting point when more atypical tracings are encountered.

There are two fundamental strategies for EEG analysis and a good approach to reading includes a combination of both strategies. The first strategy consists of making a mental list of the EEG elements that one would expect to see in the EEG given the patient's age and sleep state and identifying and analyzing each of these elements in turn. The second strategy consists of examining the array of waveforms present on the page, identifying each, and classifying each as a normal element, an abnormal element, or an artifact. In summary, the first strategy consists of making a list of "what do I expect to see?" and attempting to find each element in the list of expected findings in the EEG record. The second step is to survey the landscape of the EEG and to attempt to identify each waveform that one sees. Of course, the two strategies are complementary and can be carried out in any order; combining the two strategies ensures that the reader will consider everything that does appear on the EEG page but will also notice what is absent from the EEG record but should be there.

The purpose of this chapter is to give a brief overview of a normal EEG tracing, including transitions from wakefulness to sleep and then back to wakefulness. Next, the elements involved in these transitions are examined more closely.

QUICK TOUR: Transition of the EEG from Wakefulness to Drowsiness, Sleep, and Arousal from Sleep

Wakefulness

Figure 2-1 shows the typical appearance of the EEG in a patient who is awake with eyes closed. The basic setup of the EEG page is summarized in the figure's caption. The most prominent rhythm on the page is denoted by the solid black arrows and is called the *posterior rhythm.* Note that this waveform is highly rhythmic and sinusoidal (i.e., shaped like a sine wave). It is of highest voltage in the posterior or occipital channels (black arrows) and becomes much less prominent in the anterior channels. The posterior rhythm is best seen when the patient is awake with eyes closed.

Table 2-1 Summary of Transition from Wakefulness to Sleep in the Routine EEG

Awake	Eyes closed: posterior rhythm present
	Eyes open: low-voltage, nondescript pattern seen posteriorly, posterior rhythm absent
Drowsy	Mild slowing of the posterior rhythm
	Slow roving lateral eye movements appear
	Disappearance of posterior rhythm in the occipital areas, replaced by low-voltage theta activity
	Diffuse increase in theta range activity, particularly at the vertex
Stage I Sleep	Vertex waves of sleep
Stage II Sleep	Sleep spindles
	K-complexes
Arousal	High-voltage hypersynchronous (rhythmic) slowing in some
	Return of posterior rhythm and typical waking patterns

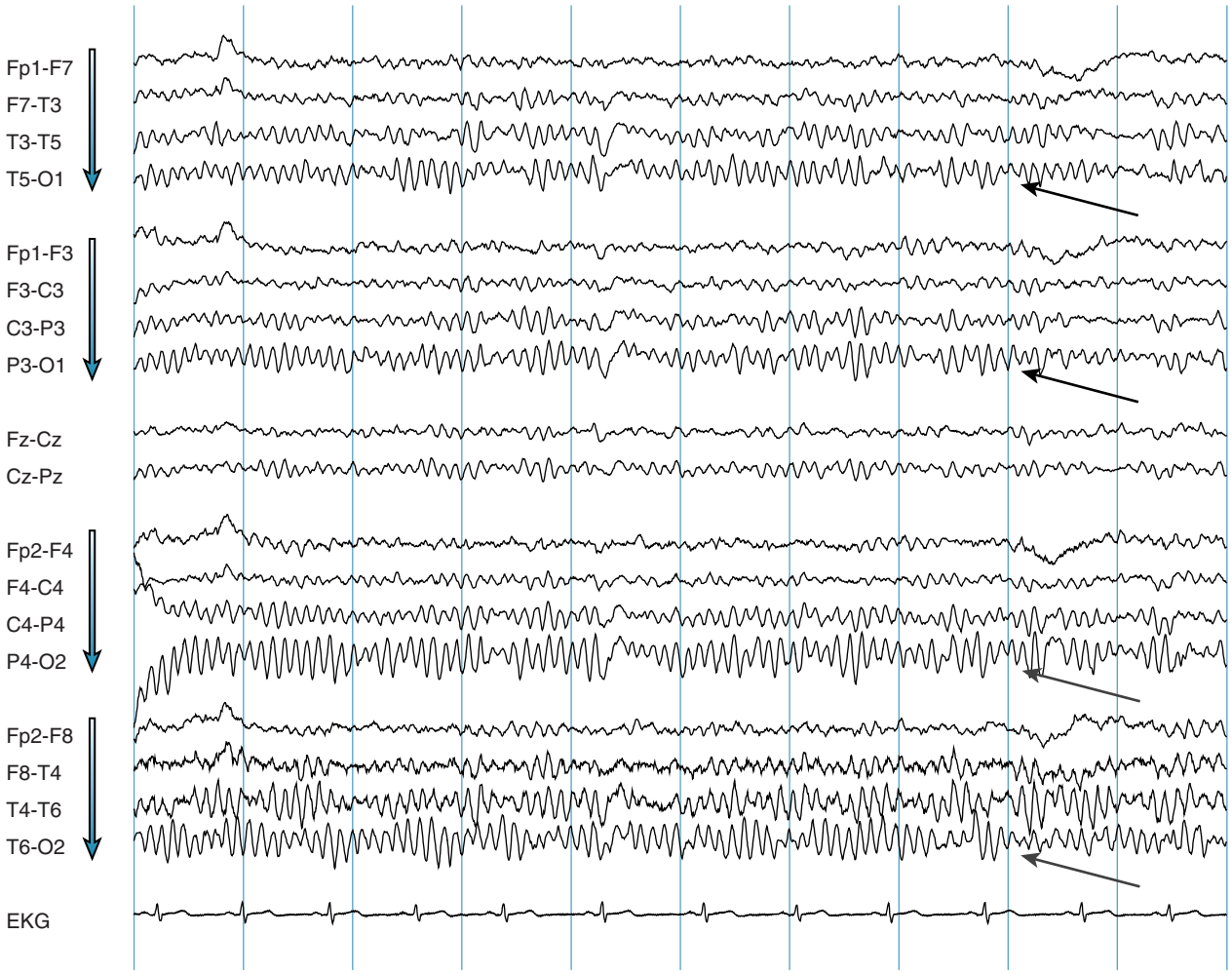

Figure 2-1 In this normal, awake electroencephalogram, the posterior rhythm is the most prominent waveform on the page. Each horizontal wave is generated by recording from the pair of electrodes denoted in the left margin. Each vertical division represents one second. Odd-numbered electrodes are placed on the left side of the scalp and even-numbered electrodes are on the right; those with z-subscripts (for "zero") are in the midline. The initial letters of the electrode names represent different brain regions: (Fp) frontopolar, (F) frontal, (T) temporal, (P) parietal, (C) central, and (O) occipital.

Each of the four major chains of electrodes can be examined from the anterior to posterior direction, as indicated by the four vertical arrows on the left side of the page. The posterior rhythm, a well-formed sinusoidal rhythm, becomes more prominent as each chain is scanned from front to back and is best defined in the channels that include the occipital electrodes, O1 and O2 (solid arrows). In this patient, as in most, the posterior rhythm is of slightly higher voltage over the right occipital area (bottom two solid arrows) compared with the left (top two solid arrows).

The normal awake EEG tracing often manifests two types of voltage and frequency transitions seen between anterior and posterior head regions, termed the *anteroposterior gradient* of the EEG: going from front to back, the *amplitude* of waves generally increases and the *frequency* of waves decreases, paralleling the shading of the arrows. Note that anteriorly in the brain (the top channels of each set of four), voltages are low, and more fast activity is seen. Posteriorly (bottom channels of each set of four, indicated by solid arrows), voltages are higher and waves are slower. In this example, posterior waves are higher because of the presence of the posterior rhythm, approximately 10 Hz in this sample.

Another typical feature of wakefulness is the presence of an *anteroposterior gradient* of voltage and frequency. Anteriorly, waves are generally of lower voltage and higher frequency. Posteriorly, waves are of higher voltage and lower frequency. Comparing the first line and the fourth line of this figure bears out these relationships. The top channel is relatively flat and has a *lower voltage, higher frequency (faster)* waveform. The fourth line has a *higher voltage, lower frequency (slower)* waveform. This is what is meant by the anteroposterior gradient: lower voltage, faster activity anteriorly and higher voltage, slower activity posteriorly. Additional examples of the anteroposterior gradient are given later in this chapter.

As seen in Figure 2-2, the posterior rhythm suppresses and often disappears completely with eye opening and fixation of gaze. When the eyes are closed, the rhythm returns. In summary, the posterior rhythm is a rhythm of wakefulness that is present when the eyes are closed. During wakefulness with the eyes open, the EEG shows a lower voltage, nondescript pattern in the occipital region, as is seen in Figure 2-2, between eye opening and closure.

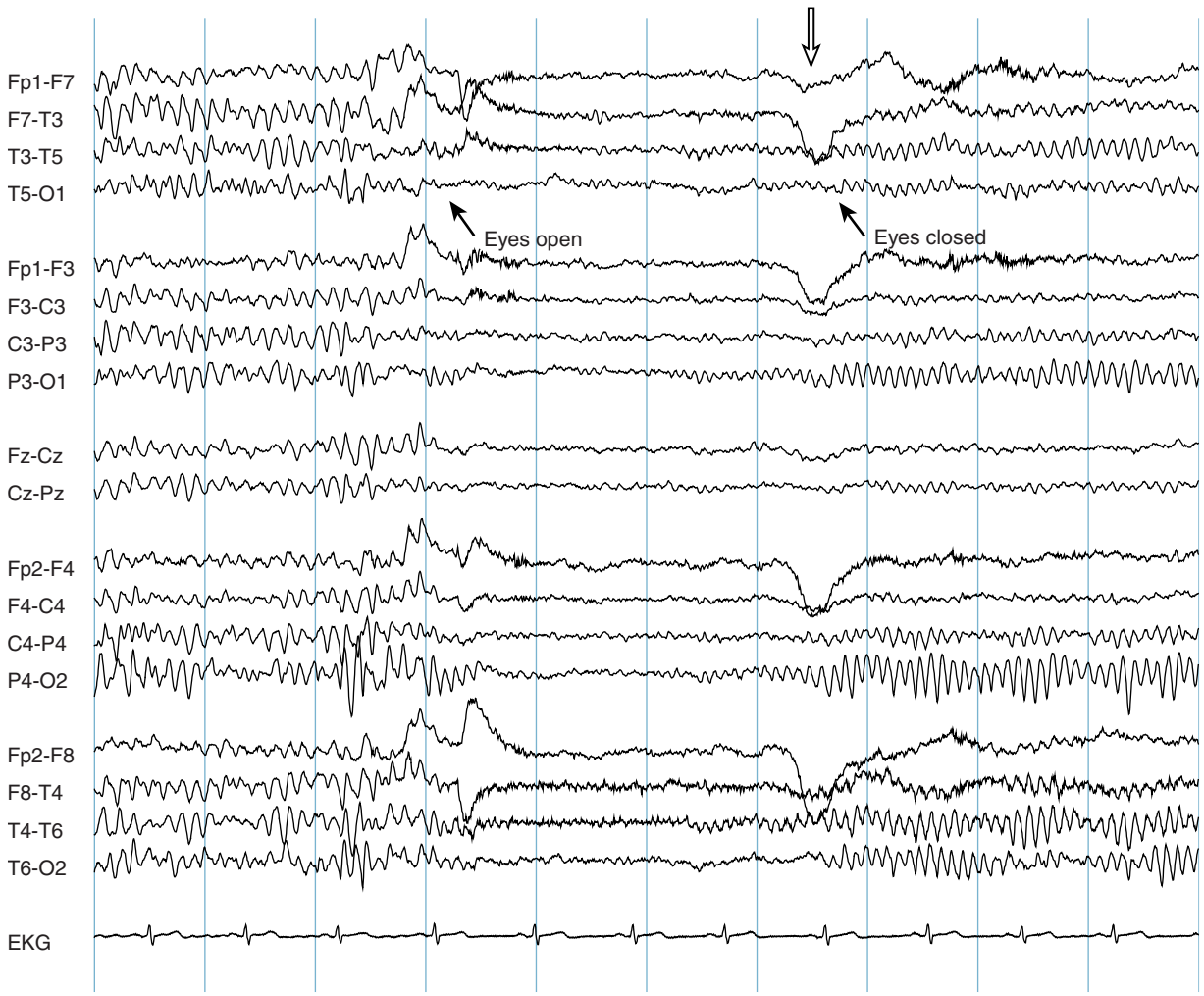

Figure 2-2 EEG of the same patient shown in Figure 2-1, awake, demonstrating the effect of spontaneous eye opening and closure on the posterior rhythm. The posterior rhythm suppresses dramatically with eye opening. Note also that the posterior rhythm actually begins to return 1.5 seconds before the eyes close, suggesting a period of relative visual inattention. The exact moment of eye closure is marked by the eye-closure artifact seen in the frontal leads (hollow arrow).

Drowsiness

One of the first EEG changes seen in drowsiness is a subtle slowing of the posterior rhythm. In Figure 2-3, the posterior rhythm is seen to slow over the course of the page from 10 Hz in the first second of the page to 8 Hz in the seventh second, an early indication of drowsiness in this patient. (This brief period of posterior rhythm slowing is not always identifiable; sometimes the posterior rhythm simply "drops out" without an observable period of slowing.) Another, more subtle finding is that of slow roving lateral eye movements of drowsiness, which are indicated by the shaded rectangles (see figure caption for further explanation). Such slow roving eye movements are commonly detected by the EEG but are not actually visible on casual observation of the patient because they are hidden by the patient's eyelids. Although the appearance of slow roving eye movements in the EEG technically represents an artifact (because they are not actual brain waves), they still provide useful information to the reader regarding onset of drowsiness. The EEG appearance of slow roving eye movements is discussed in more detail in Chapter 6.

The next EEG page (Figure 2-4) in this example shows two additional important changes that mark advancing drowsiness: first, the posterior rhythm has dropped out nearly completely. Second, there is an increase in theta-range (slow) activity throughout the tracing. Most characteristically, theta activity has appeared at the vertex, particularly in the midline central (Cz) electrode, although it may be seen in other locations as well.

On the following EEG page (Figure 2-5), the first true vertex waves of sleep are seen. These midline sharp waves mark the onset of Stage Ia sleep and may occur in dramatic bursts. After they are established, assuming no subsequent arousals, vertex-wave bursts continue in a repetitive fashion through Stage II sleep.

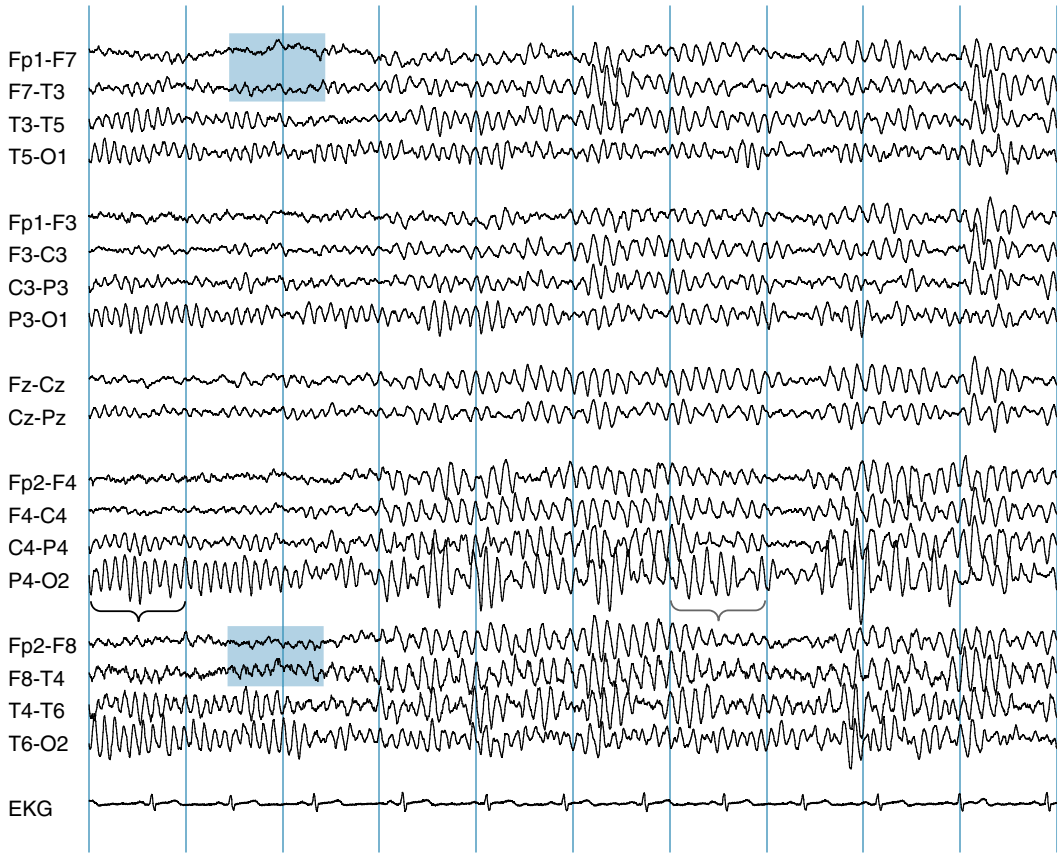

Figure 2-3 The initial transition to drowsiness is marked by a slowing of the posterior rhythm. A frequency of 10 Hz can be counted over the black brace in the first second of this page. The frequency falls to approximately 8 Hz by the seventh second over the blue brace. Artifact from slow roving eye movements can also be seen: note the subtle spreading apart of the waveforms of the two channels that include F7 (top blue rectangle) compared with a relative narrowing together of the two channels that include F8 (bottom blue rectangle). The appearance of this artifact is caused by a slow roving movement of the eyes to the left, a sign of early drowsiness, and is described in more detail in Chapter 6.

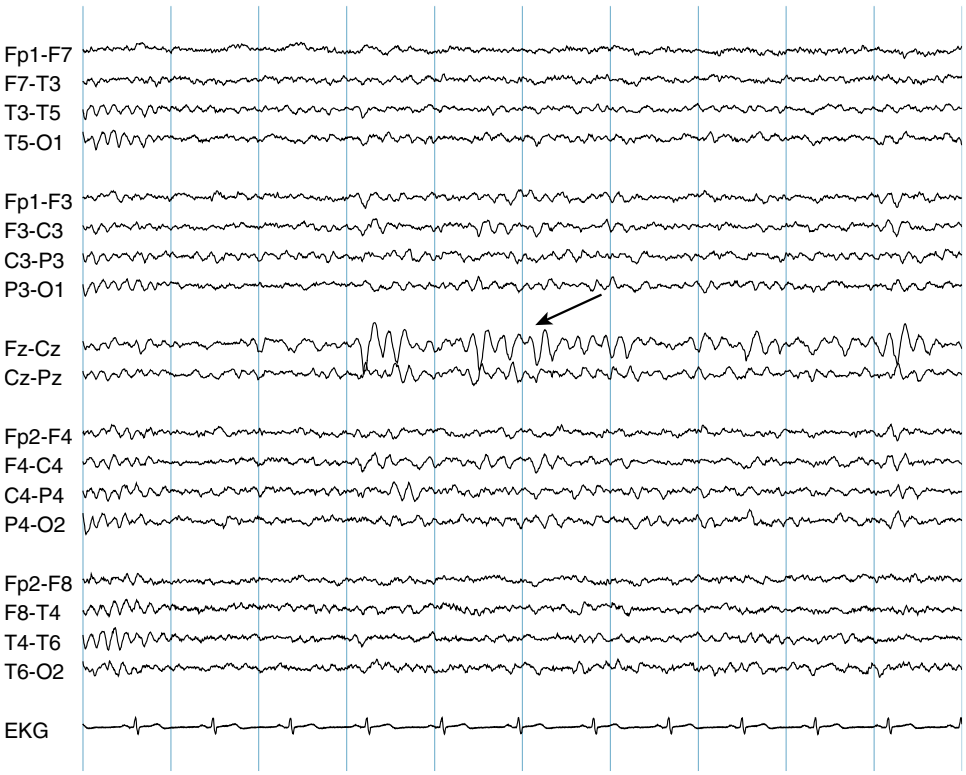

Figure 2-4 This page shows the transition from deepening drowsiness to light sleep. The posterior rhythm completely disappears after the first second and vertex activity increases (black arrow) in the form of theta waves. More low voltage theta range (slow) activity is seen in other brain areas as well.

Stage II Sleep

The onset of Stage II sleep is defined by the appearance of sleep spindles. The sleep spindles that occur early in Stage II sleep are usually of maximum voltage in the central electrodes (C3 and C4) and at the central vertex (Cz), as is seen in this example. They consist of lower voltage, regular 14-Hz waves lasting from 1 to a few seconds. In deeper Stage II sleep, the field of sleep spindles may include both the frontal and central areas. Figure 2-6 shows the appearance of the first, bicentral sleep spindles in this patient, intermixed with vertex waves. By the next page, the sleep spindles become more sharply defined (see Figure 2-7) and continue to be intermixed with repetitive vertex waves. The combination of repetitive vertex waves and spindles marks well-established Stage II sleep. An example of the fields of spindles and vertex waves is shown in Figures 2-8 and 2-9 and schematically in Figures 2-10 and 2-11.

Bursts of high voltage waves occurring across nearly all channels may be seen sporadically in sleep. These discharges, called *K-complexes,* can be dramatic and are sometimes mistaken for spike-wave discharges, an epileptiform abnormality. K-complexes may be set off by stimuli (such as a noise) in the environment of the sleeping patient that cause a mild *subarousal* (an increase in the level of arousal or a lightening of the sleep state that is not strong enough to awaken the patient fully). In fact, EEG technologists often demonstrate K-complexes in the EEG by tapping a pencil on the EEG instrument while the patient is in light sleep. The tapping sound may elicit a subarovsal and an associated K-complex. Most K-complexes, however, appear to occur spontaneously without an obvious trigger. The field of a K-complex differs from that of sleep spindles or sleep vertex waves and is shown in Figure 2-12. K-complexes may or may not be intermixed with a sleep spindle, as occurs in the example shown in Figure 2-13.

Arousal from Sleep

Arousal from sleep may occur uneventfully with a simple return of the posterior rhythm and other patterns of wakefulness described earlier. At other times, arousal from sleep may be marked by a dramatic run of diffuse, high-voltage rhythmic waves called an *arousal hypersynchrony.* Figure 2-14 shows a fairly simple arousal with a brief increase in rhythmic slowing followed by high-voltage motion artifact generated from the patient stirring in bed. This is followed by a return of the posterior rhythm.

The sequence of wakefulness to drowsiness to sleep is shown in a second patient in Figures 2-15 through 2-20, with fewer figure markings to help the render practice identification of normal sleep waveforms.

Natural Sleep

The foregoing example represents a quick tour through wakefulness, drowsiness, Stage Ia sleep, and Stage II sleep, followed by an arousal and return to wakefulness. The reader may ask why this sequence does not include examples of deeper sleep such as sleep Stages III and IV and REM sleep. In practice, these deeper sleep stages are not typically encountered during routine EEG recordings. The amount of sleep recorded during a routine EEG is usually less than 20 to 30 minutes and sometimes just a few minutes if much time has been spent getting the patient to fall asleep for the test. During routine EEG testing, few patients have the time to enter Stage III, IV, or REM sleep during routine EEG recordings. Examples of these patterns are seen in Figures 2-21 through 2-23.

During natural sleep, normal individuals sequentially cycle through Stages I through IV and then back up to Stage I, after which they may enter a brief REM stage after 1 to 2 hours. The first third of the night's sleep is dominated by slow-wave sleep, whereas REM sleep is most plentiful during the early morning hours before awakening, at which time the portions of the cycle devoted to REM sleep are lengthier.

VISUAL ANALYSIS OF THE EEG: Identification of Expected Elements

Wakefulness

The Posterior Rhythm

A good first step in the interpretation of the awake EEG is identification of the posterior rhythm, often the most distinctive and easily identifiable element of the waking EEG. The posterior rhythm is, as the name implies, best seen in the posterior head regions, although how far the field of the posterior rhythm spreads forward varies among patients. In some patients, the posterior rhythm is confined to the occipital areas, but, in many, the posterior rhythm spreads forward to include the whole of the posterior quadrants (see Figure 2-15), and at times the field may even reach the superior frontal electrodes (F3 and F4), as in Figure 2-1. The posterior rhythm should never be visible in the frontopolar electrodes, however.

In addition to its location on the head, two other distinctive qualities define the posterior rhythm. First, it is a rhythm of wakefulness. Indeed, the posterior rhythm is the hallmark of EEG wakefulness in those patients who manifest such a rhythm. Second, the posterior rhythm is predominantly seen when the subject's eyes are closed or when visual attention is lacking. The posterior rhythm may dramatically suppress when the eyes are opened, as was seen in Figure 2-2. The posterior rhythm suppresses equally well when patients open their eyes spontaneously as when they open their eyes in response to a request. Although visual attention and eye opening usually go together, during certain intervals, a patient may have the eyes open but may not be attending visually (fixating) on a target. Because it is specifically visual attention rather than eyelid opening that causes suppression of the posterior rhythm, the posterior rhythm is occasionally seen at times when the eyes are open but the subject is visually inattentive. Although the posterior rhythm can be identified in the great majority of patients, in a small number of normal individuals, no posterior rhythm is seen on

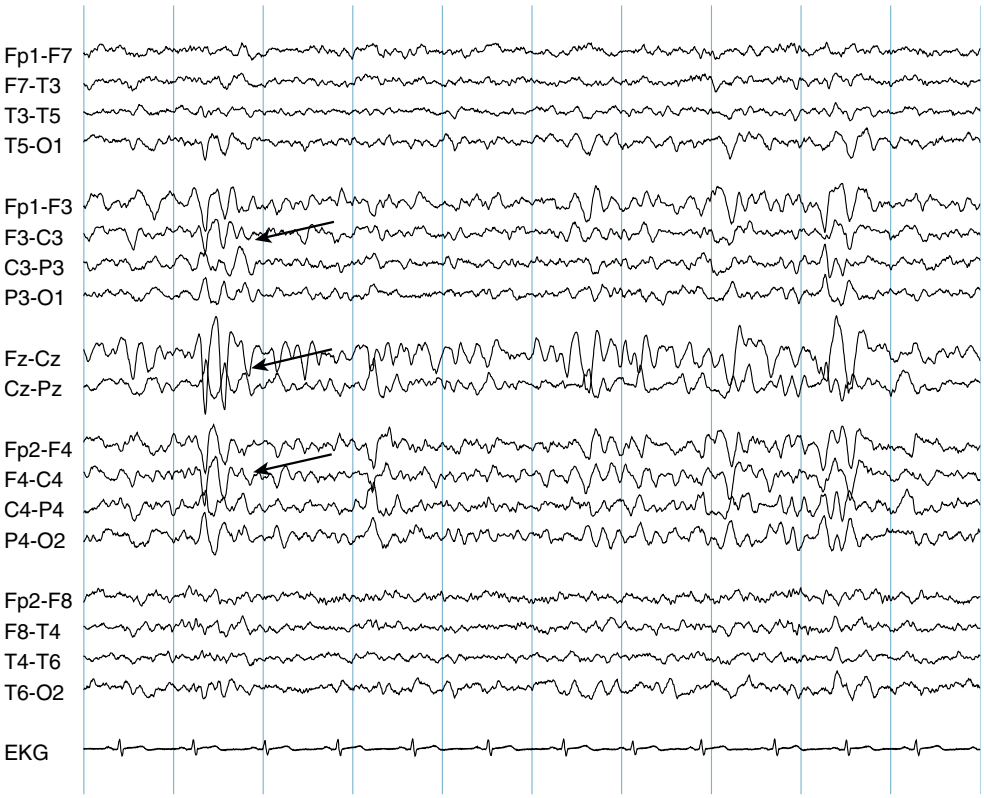

Figure 2-5 In this figure, vertex waves become well established. Note that the vertex wave voltage is highest in the C3, Cz, and C4 electrodes (as evidenced by phase reversals in those locations—see black arrows). A second set of vertex waves is seen near the end of the page. Rhythmic vertex theta can be seen between the two larger vertex-wave bursts. The appearance of vertex waves marks the onset of Stage Ib sleep.

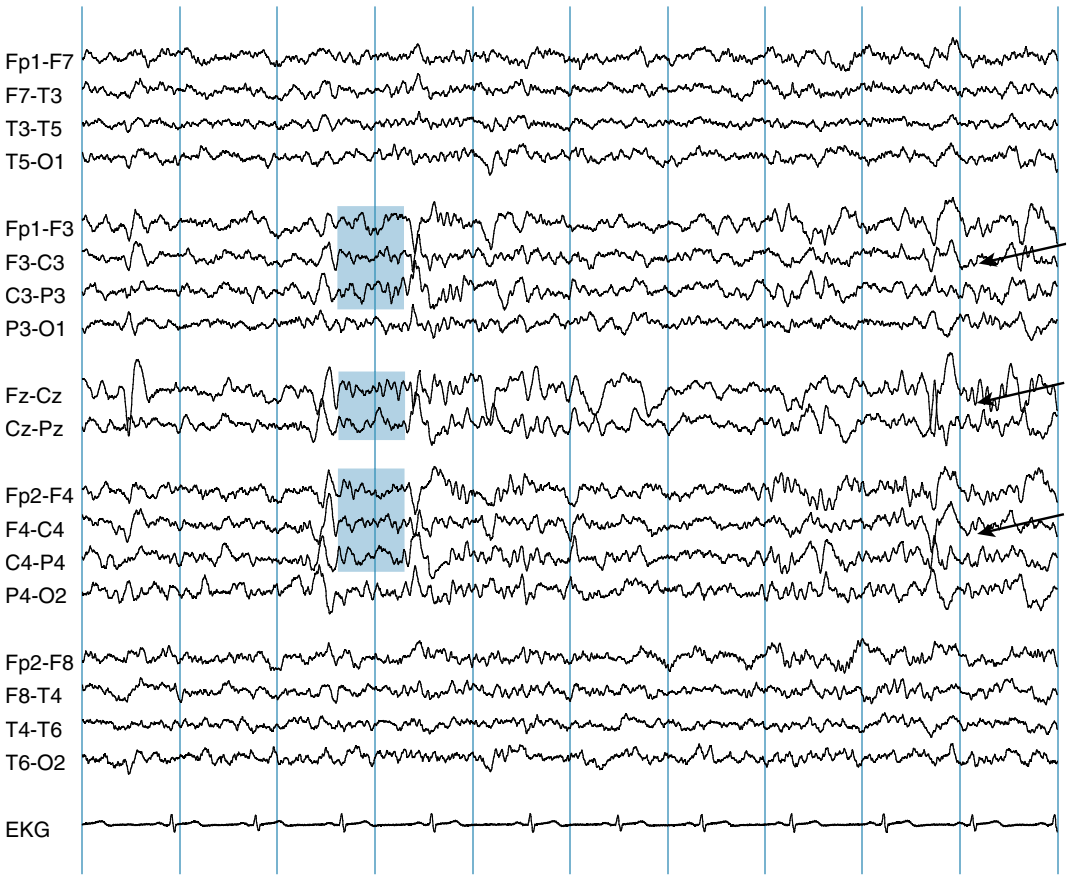

Figure 2-6 Transition from Stage Ia to Stage II sleep is marked by the appearance of sleep spindles. The sleep spindles are the lower voltage, sinusoidal 14-Hz waves that are following close on the heels of the vertex waves (a portion of the spindles is highlighted by the blue rectangles). Sleep spindles are also seen surrounding the vertex wave on the right side of the page (arrows). The spindles become better defined in the next examples.

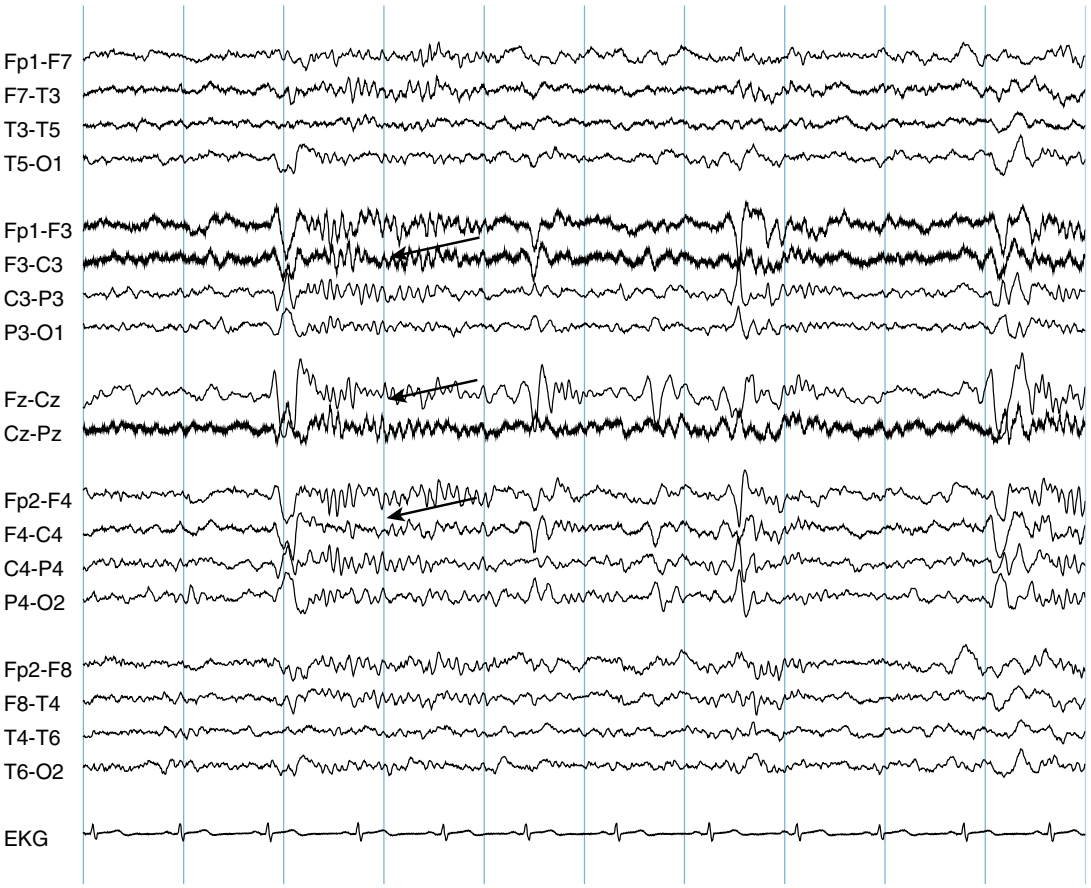

Figure 2-7 Well-established sleep spindles (arrows) following a vertex wave. These spindles are maximum in the frontocentral regions and in the midline. Repetitive vertex waves continue. Note that spindles often follow vertex waves, although the link between the two is not always consistent.

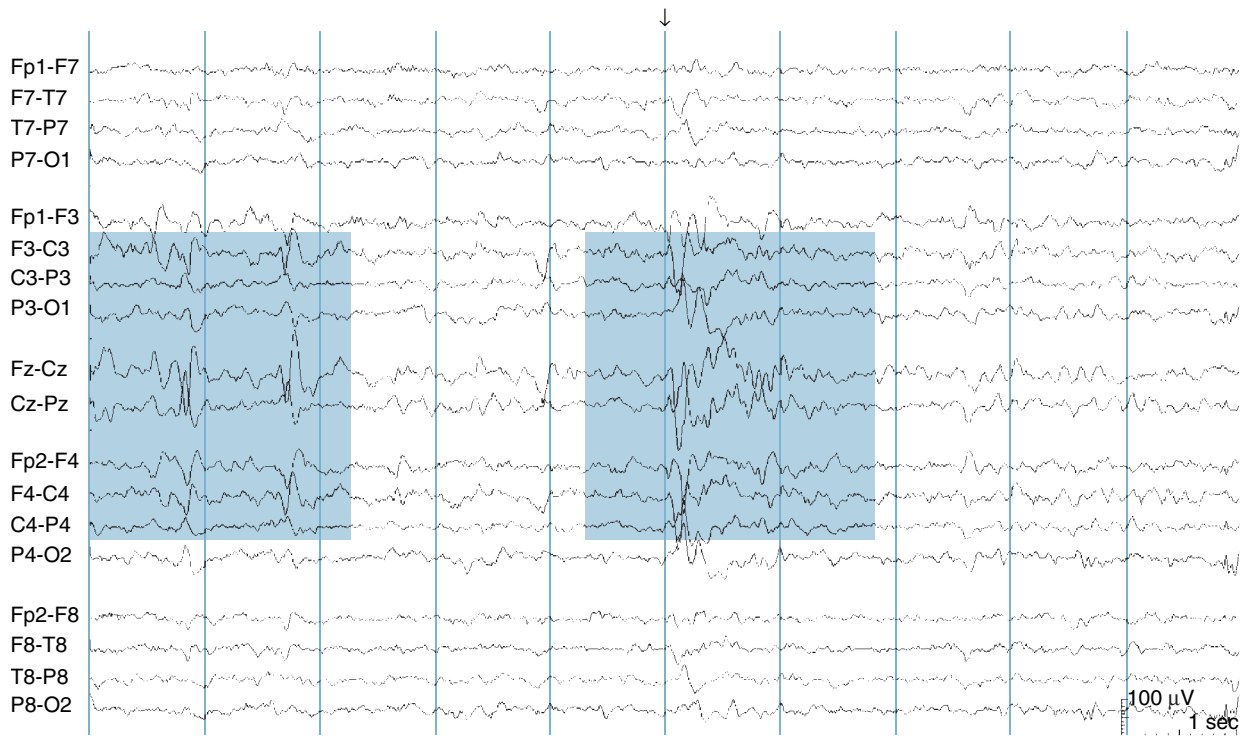

Figure 2-8 The blue rectangles highlight the brain areas in which vertex waves and spindles are seen most prominently, here displayed in a bipolar montage. Note that the temporal chains, represented by the four-channel groupings at the top and bottom of the page not included in the blue rectangles, are relatively uninvolved with the vertex wave and spindle waveforms.

scalp-recorded EEG. Absence of the posterior rhythm as an isolated finding is not necessarily considered an abnormality.

A Note on the Term "Alpha Rhythm"

Because in adults and older children the posterior rhythm's frequency is normally in the *alpha range* (8–13 Hz), the posterior rhythm is sometimes referred to as the "posterior alpha rhythm" or simply the "alpha rhythm." This terminology is not preferred for several reasons, in particular because the posterior rhythm may have a frequency below the alpha range in many clinical situations. A posterior rhythm below the alpha range is seen frequently in children, especially at the beginning of the first decade of life, an age at which a posterior rhythm under 8 Hz is considered a normal finding. Adults may also manifest a slowing of the posterior rhythm below 8 Hz, most often marking a pathological state or occasionally a pharmacological effect. Finally, rhythms in the alpha range (between 8 and 13 Hz) may be found elsewhere in the EEG and in different brain areas. These, too, could correctly be called "alpha rhythms," which could lead to some confusion. Use of the term *posterior rhythm* rather than *alpha rhythm* avoids the awkwardness of referring to an "alpha rhythm" that may be in the theta range. Therefore, the preferred term for the rhythm seen in posterior head regions during wakefulness that suppresses with eye opening is the *posterior rhythm.*

Most often, the shape of the posterior rhythm is sinusoidal, which is to say that the peaks and troughs of the waveform are similarly rounded and regular. In a minority of individuals, the posterior rhythm waveform is sharpened on one side (often the bottoms of the waves in bipolar montages) and rounded on the other. This is referred to as *spiky alpha variant* and is considered a normal variant (see Figure 2-24).

Amplitude Asymmetry of the Posterior Rhythm

The amplitude of the posterior rhythm usually differs between two hemispheres. Most often, the posterior rhythm is of lower voltage over the left hemisphere (the dominant hemisphere) compared with the right hemisphere. Although some amount of asymmetry of posterior rhythm amplitude is normal and, indeed, expected, a large asymmetry of the posterior rhythm may reflect an abnormality of one of the hemispheres; the posterior rhythm amplitude may be lower on one side because that hemisphere has been damaged or is abnormal in some way. Usually, the

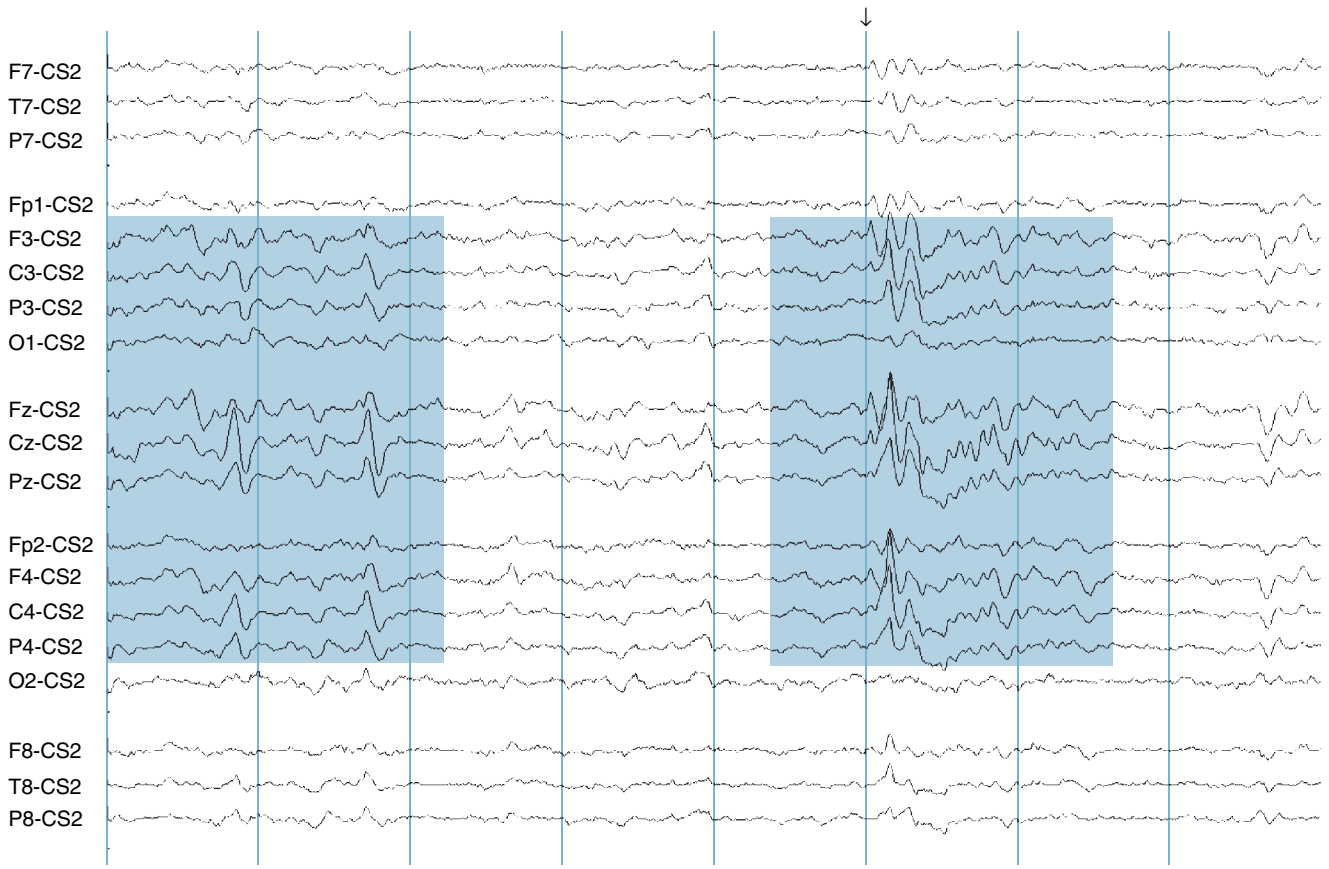

Figure 2-9 The same vertex waves and spindles displayed in a referential montage. Because referential montages dedicate one channel to each active electrode (compared to a pair of active electrodes per channel in bipolar montages), the individual electrodes that pick up the spindle and vertex-wave discharges are easier to discern. The blue rectangles highlight the field of these waves, which primarily include the central, frontal, and midline regions.

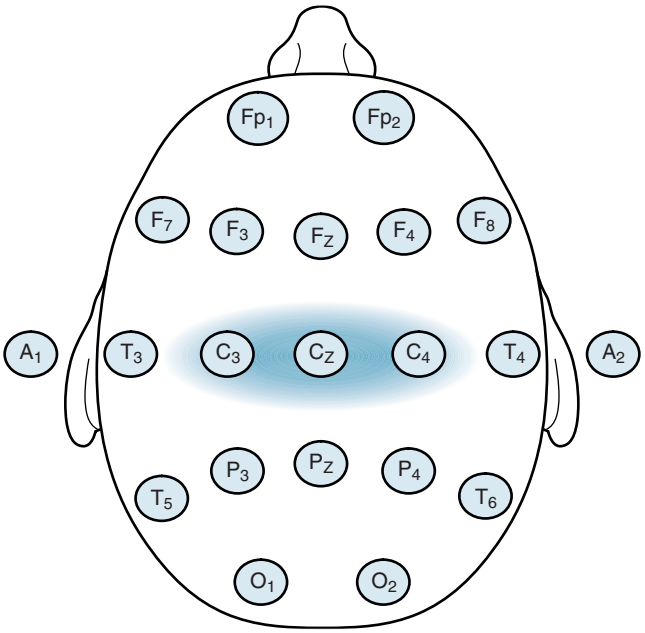

Figure 2-10 A schematic of the approximate field of vertex waves of sleep is shown. The maximum activity of vertex waves is at the Cz electrode with lesser voltages measured at the adjacent C3 and C4 electrodes.

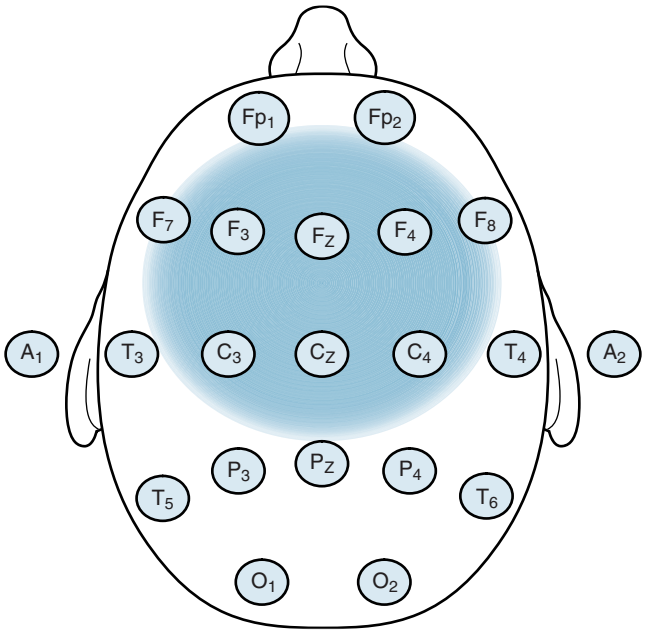

Figure 2-11 Classically, spindles are centered over the central areas, particularly the C3 and C4 electrodes. In many examples such as the EEG traces shown in Figures 2-8 and 2-9, spindles are also seen to spread frontally (F3 and F4 electrodes). The field of spindles only occasionally spreads laterally to the midtemporal electrodes where they should be of lower voltage compared with the central electrodes or not seen at all.

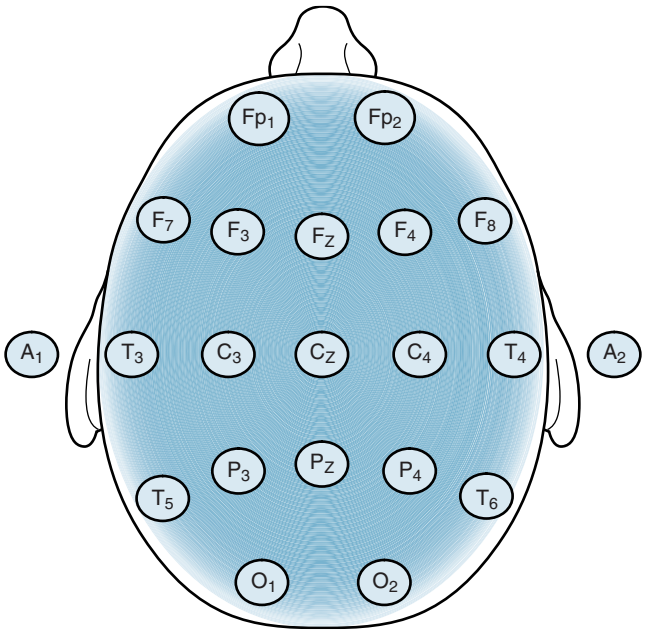

Figure 2-12 The field of a K-complex is diffuse and may include all brain areas. This helps differentiate it from simple spindles, which are maximum frontocentrally and concentrated in the midline and parasagittal areas as described in Figure 2-11. A sleep waveform of an intensity just as strong in the temporal areas as in the midline is not likely to represent a sleep spindle or vertex wave but may represent a K-complex.

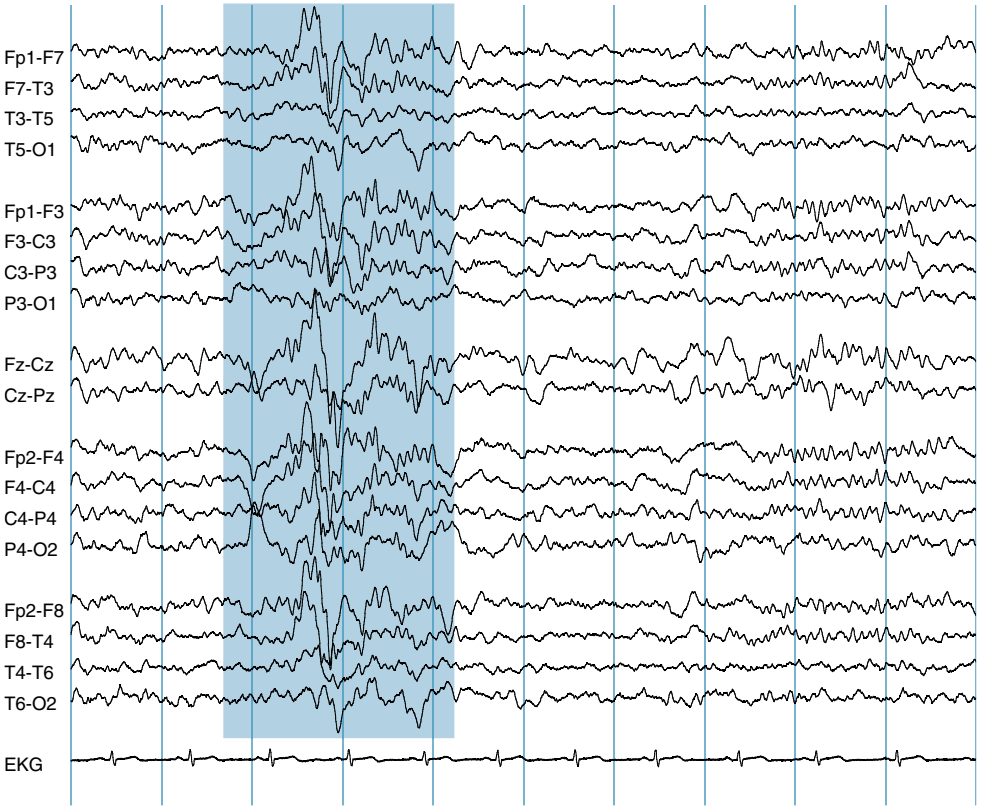

Figure 2-13 A K-complex is seen during Stage II sleep (blue rectangle). At first glance, the K-complex resembles a vertex wave followed by a sleep spindle. Note, however, that the high-voltage waves preceding the spindles in this example do not have the typical distribution of a vertex wave of sleep. Although vertex waves are maximum in C3, Cz, and C4, the example of this particular wave shows frontal voltages that are just as high in the temporal chains (Fp1-F7, F7-T3, Fp2-F8, F8-T4) as in the parasagittal chains. The broad field extending to the temporal chains of the high-voltage wave that precedes the spindles is the tip-off that this complex is not simply a combination of a vertex wave and sleep spindles but a K-complex.

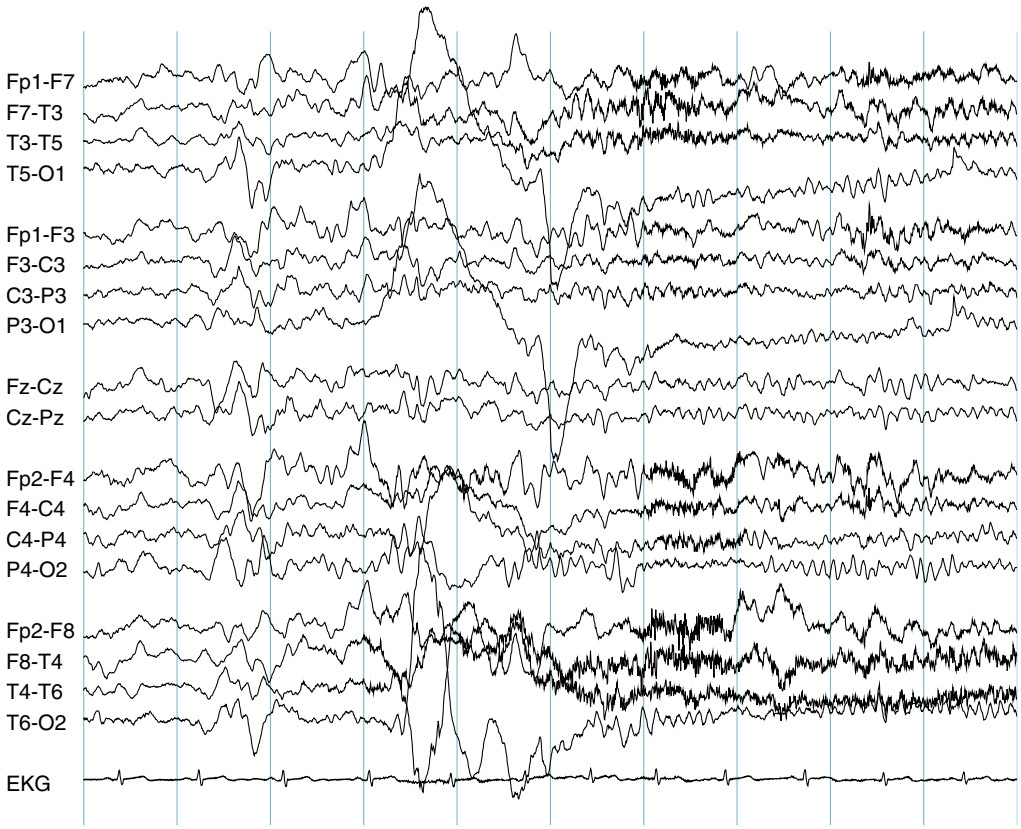

Figure 2-14 Several elements of this EEG page signal an arousal from sleep. Diffuse rhythmic slow waves, as seen in the second second of this tracing, are typical of arousal and sometimes much more dramatic than in this example. The very high-voltage deflections that cross into neighboring channels are large motion artifacts, often seen at the time of arousal from sleep, which is typically associated with body movements. The very fast waves that turn some channels dark black, most prominent in the temporal chains at the top and bottom of the page, represent muscle artifact also associated with movements related to arousal. Note the reappearance of the posterior rhythm in the second half of the page following the high-voltage motion artifact.

difference in the posterior rhythm amplitude between the two sides does not exceed 20 percent. Therefore it is useful to set a threshold for posterior rhythm asymmetry beyond which an amplitude difference is considered abnormal.

The following rule for determining when an asymmetry of the posterior rhythm is abnormal is somewhat arbitrary and is also fairly conservative: the dominant hemisphere is usually the left hemisphere. In the majority of patients, the left hemisphere usually has the lower voltage posterior rhythm. If the posterior rhythm happens to be of higher voltage on the left, it is only accepted as normal if it is up to twice the voltage of the right. In the more typical situation in which the posterior rhythm is of higher voltage on the right side, because it is already expected to be higher on that side, up to a threefold asymmetry of voltage is considered acceptable.

Stated more formally, when the posterior rhythm is of higher voltage over the nondominant hemisphere (the usual case), if the posterior rhythm's amplitude over the nondominant hemisphere is more than threefold greater than that of the dominant hemisphere, it is considered abnormal. When the posterior rhythm is of higher voltage over the dominant hemisphere (the less common case), if the posterior rhythm's amplitude on the dominant hemisphere's side is more than twofold greater than that of the nondominant side, it is considered abnormal.

One theory that has attempted to explain why the posterior rhythm is of higher voltage over the nondominant hemisphere is based on the observation that in several settings, EEG waves are of higher voltage at times when a brain area is not busy with mental processing. A simple example of this phenomenon is the posterior rhythm: when the occipital lobes are involved in visual processing, the posterior rhythm disappears and low voltages are seen in the occipital areas; when the occipital lobes are idle during periods of eye closure or visual inattention, occipital voltage increases in the form of the posterior rhythm. There is an analogous idling rhythm of the motor cortex called the *central mu rhythm* (see Chapter 11), which disappears when a motor movement is made or contemplated in the contralateral upper extremity and reappears when the limb is at rest. The observation of lower voltages during idle states can even be extended to sleep waves that are generally of higher amplitude than waking rhythms and presumably represent the higher voltage electrical activity of a more "idle" brain compared with the waves of the waking EEG. Returning to the phenomenon of the posterior rhythm being of lower voltage over the left hemisphere compared with the right, the theory holds that the dominant hemisphere tends to be more occupied with neural processing because of the nature of the tasks it handles by nature of its "dominance." In comparison, the nondominant hemisphere

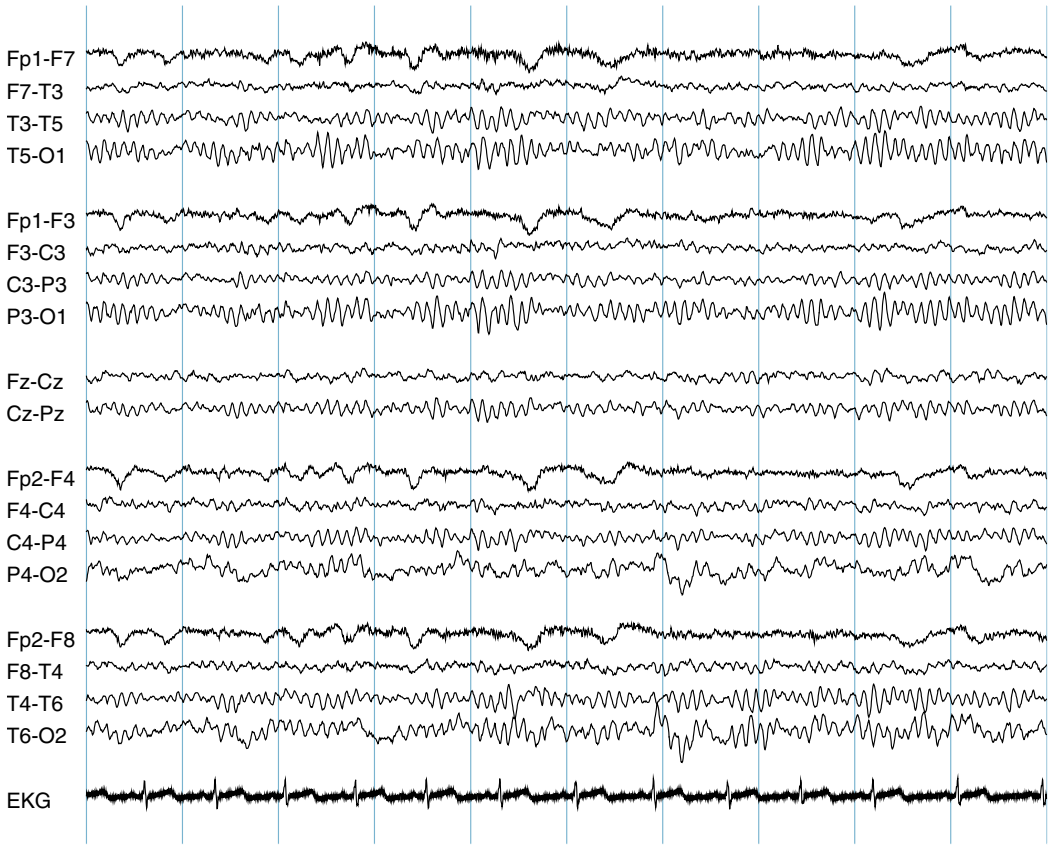

Figure 2-15 An example of a normal, awake patient. The posterior rhythm is seen well in the posterior channels: T5-O1, P3-O1, Cz-Pz, P4-O2, and T6-O2. The frontal channels in each chain of four—Fp1-F7, Fp1-F3, Fp2-F4, and Fp2-F8—are darker than others because of artifact from the frontalis muscle. Bobbing waves seen in those channels represent artifact related to vertical eye movements made under closed eyelids (given the presence of the posterior rhythm).

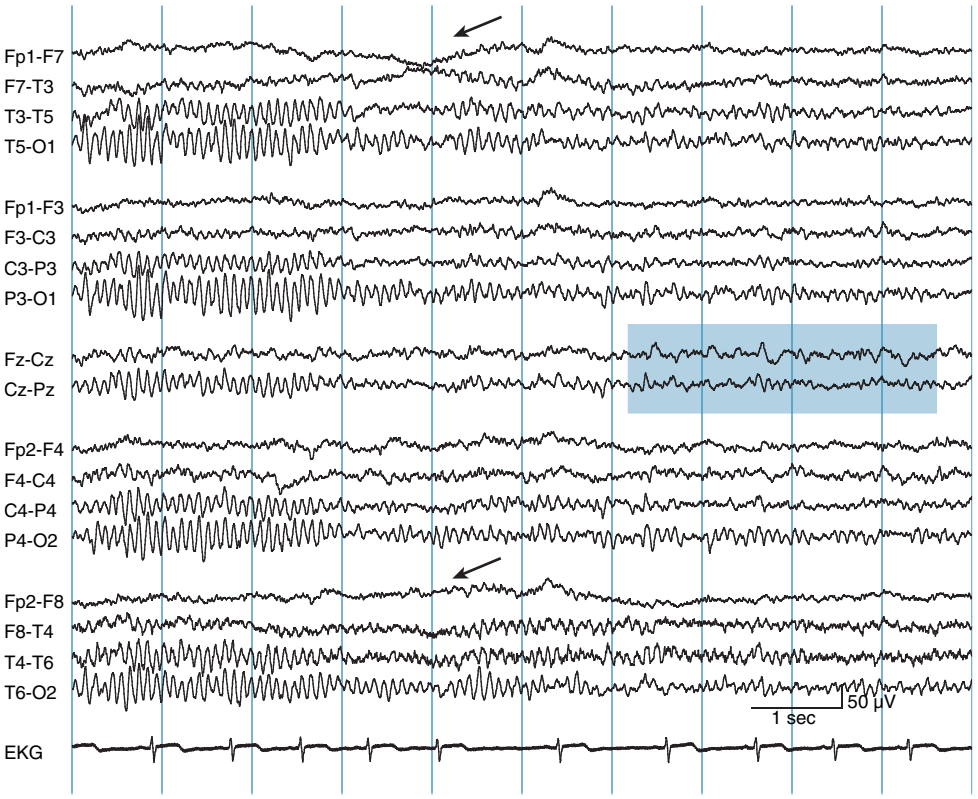

Figure 2-16 During the course of this page, the posterior rhythm drops in frequency by 1 Hz and becomes less prominent (seen best in T5-O1, P3-O1, P4-O2, and T6-O2). Slow roving lateral eye movements are seen as evidenced by approximation of the Fp1-F7 and F7-T3 channels at the same time as a mild "bulging apart" of the Fp2-F8 and F8-T4 channels (arrows). The reason that lateral eye movements create this appearance is explained in more detail in Chapter 6. An increase in theta activity, another sign of drowsiness, is seen at the vertex in the seventh and eighth seconds in the Fz-Cz channel (blue rectangle).

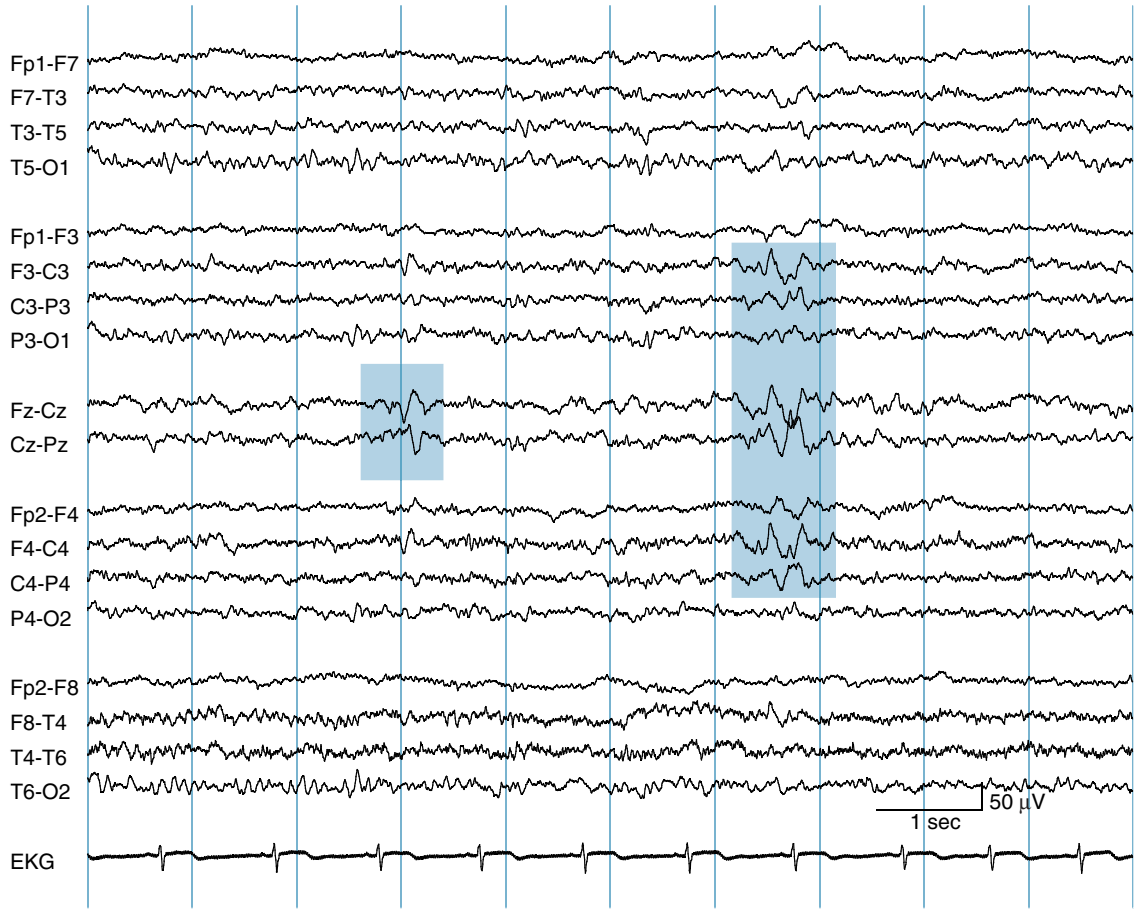

Figure 2-17 The first vertex waves appears at the beginning of the fourth second exclusively in Cz, visible in the Fz-Cz and Cz-Pz channels (small blue rectangle). The second vertex wave appears in the seventh second and now includes both Cz and the central electrodes, seen in the channels that include C3 and C4 (large blue rectangle).

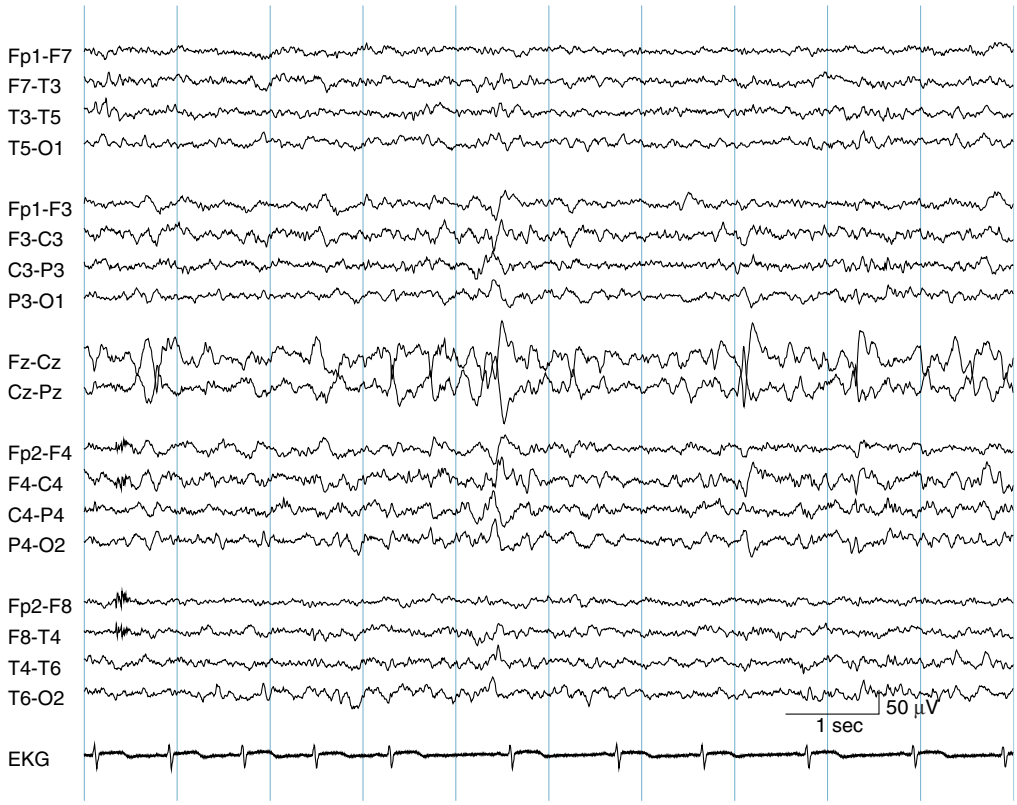

Figure 2-18 Cascades of vertex waves of sleep are seen regularly in the midline channels (Fz-Cz and Cz-Pz) as stage 1b sleep becomes well-established. Overall, the background shows an increased number of low-voltage slow waves.

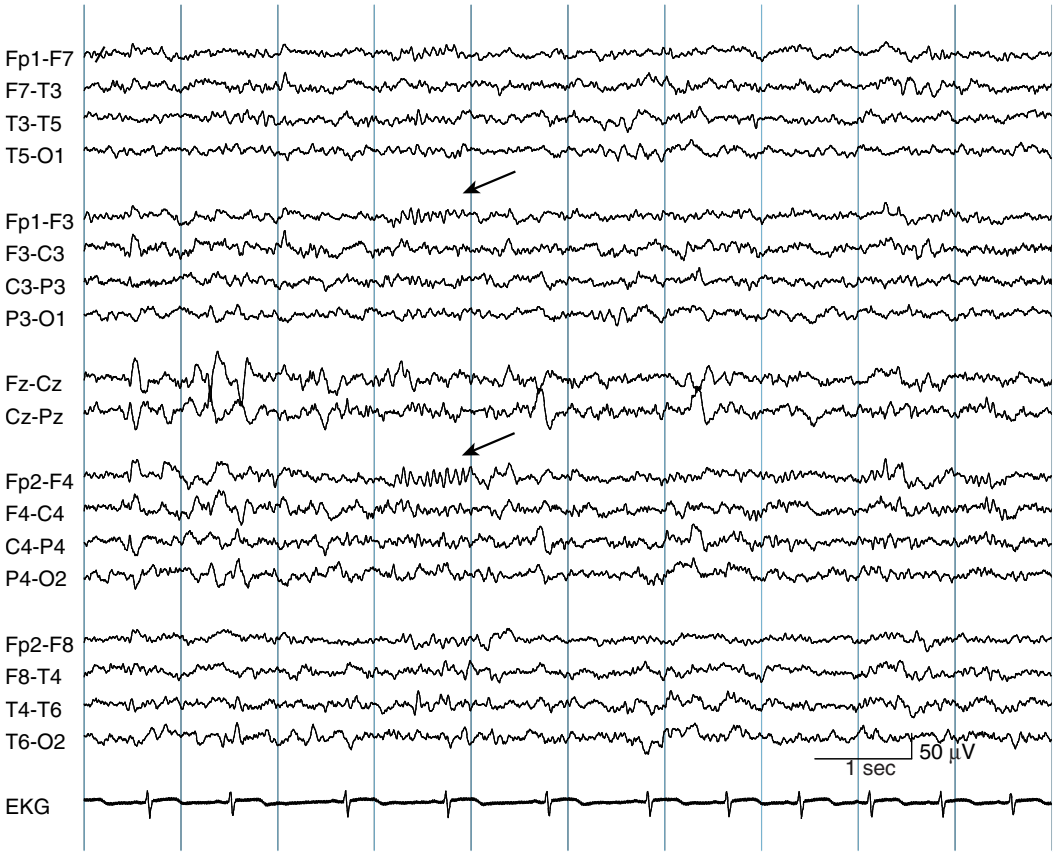

Figure 2-19 Vertex waves continue, and sleep spindles now make their appearance (arrows), marking the onset of Stage II sleep.

is considered to be more "idle" and therefore would be expected to generate a higher amplitude posterior rhythm. Although this theory may be useful in helping the electroencephalographer remember the side of the brain on which the posterior rhythm usually manifests higher voltage, the idea is unproven, and its greatest value may be as a mnemonic memory device.

Symmetry of Frequency of the Posterior Rhythm

The frequency of the posterior rhythm should be the same over each hemisphere. Although a mismatch in the frequency of the posterior rhythm between hemispheres should be considered an abnormality, true differences in the fundamental frequency of the posterior rhythm between hemispheres is uncommon. Most examples of apparent mismatches of the posterior rhythm frequency are actually caused by an intermixing of slow-wave activity into the posterior rhythm on one side. In these cases, the fundamental posterior rhythm frequency may be the same on both sides, but on one side the rhythm may be partly obliterated by a slow wave, creating the illusion of different posterior rhythm frequencies over each hemisphere.

The Posterior Rhythm in Infancy and Childhood

The posterior rhythm can be first recognized as early as 3 to 4 months of age in infants born at term, although in infancy, reactivity to eye closure is more difficult to demonstrate. At 3 to 4 months, a posterior rhythm frequency of 3 to 4 Hz is expected. Through the first decade, the posterior rhythm's frequency increases and reaches the adult range, which is between 8 and 13 Hz. Because a posterior rhythm below 8 Hz is considered abnormal after age 8 years, the rule of "eight by eight" can be applied, meaning that by 8 years a posterior rhythm frequency of 8 Hz is expected. Realistically, 8 years can be considered the outer age limit by which the posterior rhythm is expected to reach 8 Hz. In practice, many normal children will attain a posterior rhythm frequency of 8 Hz by as early as 2 years of age. This illustrates the distinction between knowing the *typical* and *limit of normal* posterior rhythm frequencies at different ages. The limit of normal of the posterior rhythm for a particular age dictates the cutoff used between normal and abnormal (an interpretation of abnormal implying an increased probability that a pathological state is present). By contrast, *typical* ranges

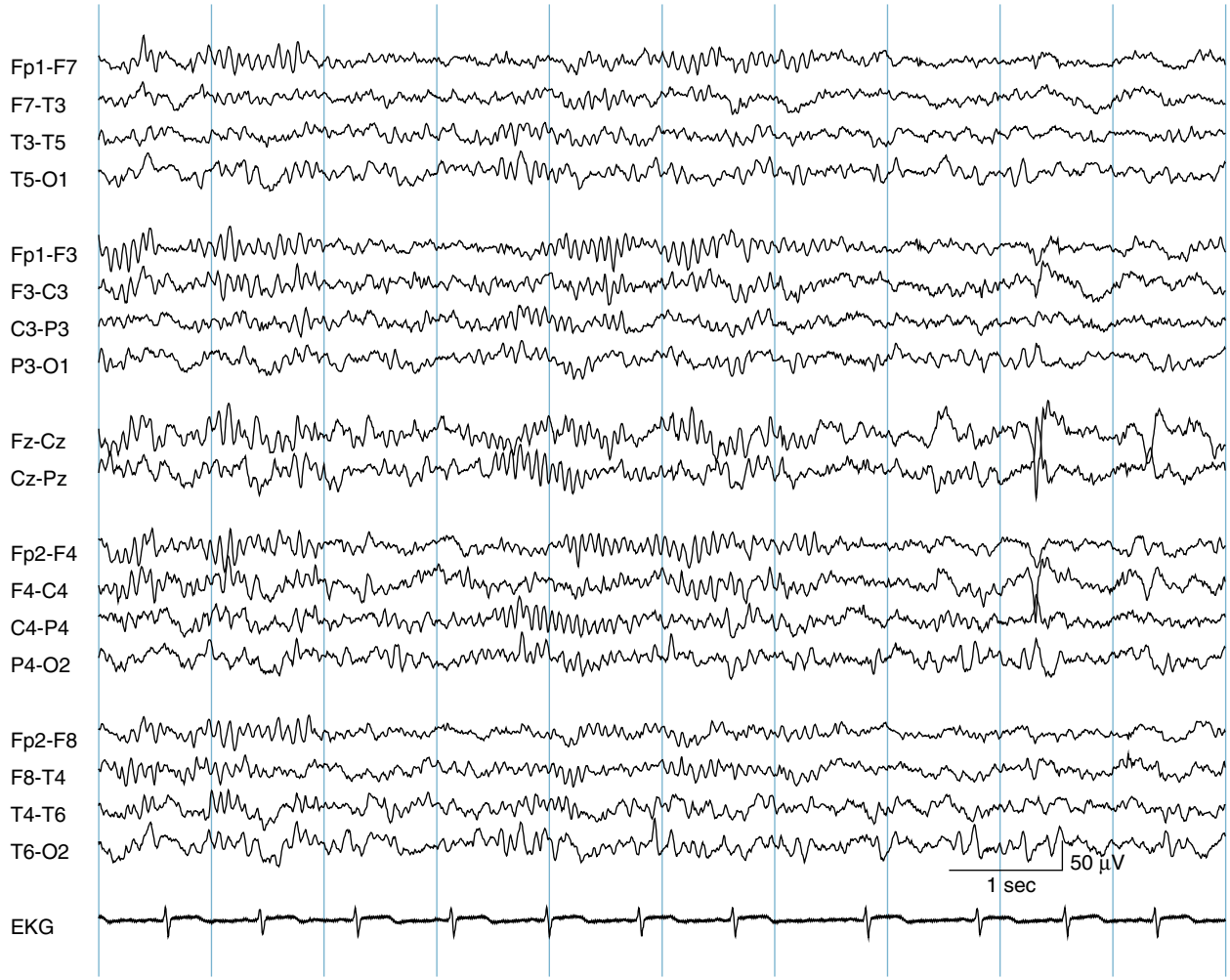

Figure 2-20 In deeper Stage II sleep, spindle activity becomes more prominent. Vertex waves also continue, seen in the last 2 seconds of the page.

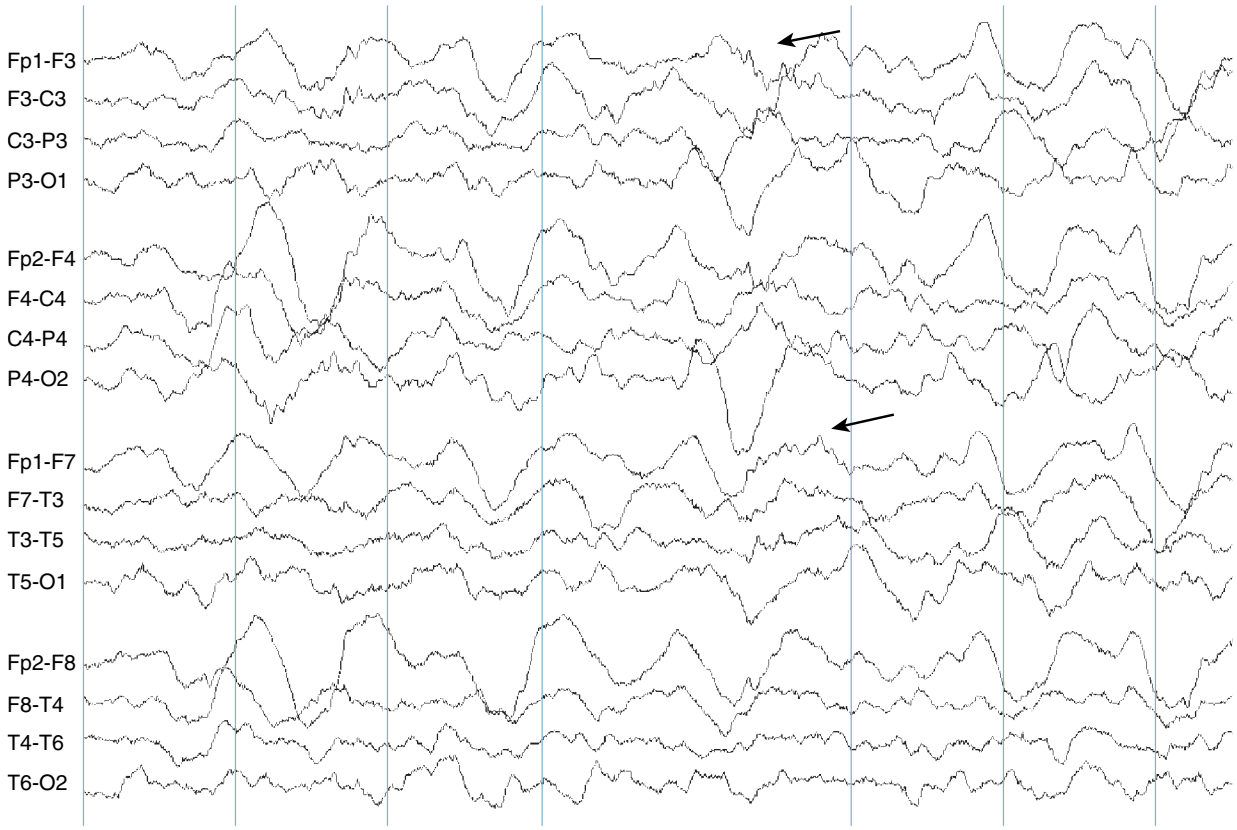

Figure 2-21 A sample of Stage III sleep is shown containing approximately 50% delta activity. Some sleep spindle activity persists (arrows) but is more difficult to appreciate against the backdrop of the slow wave activity.

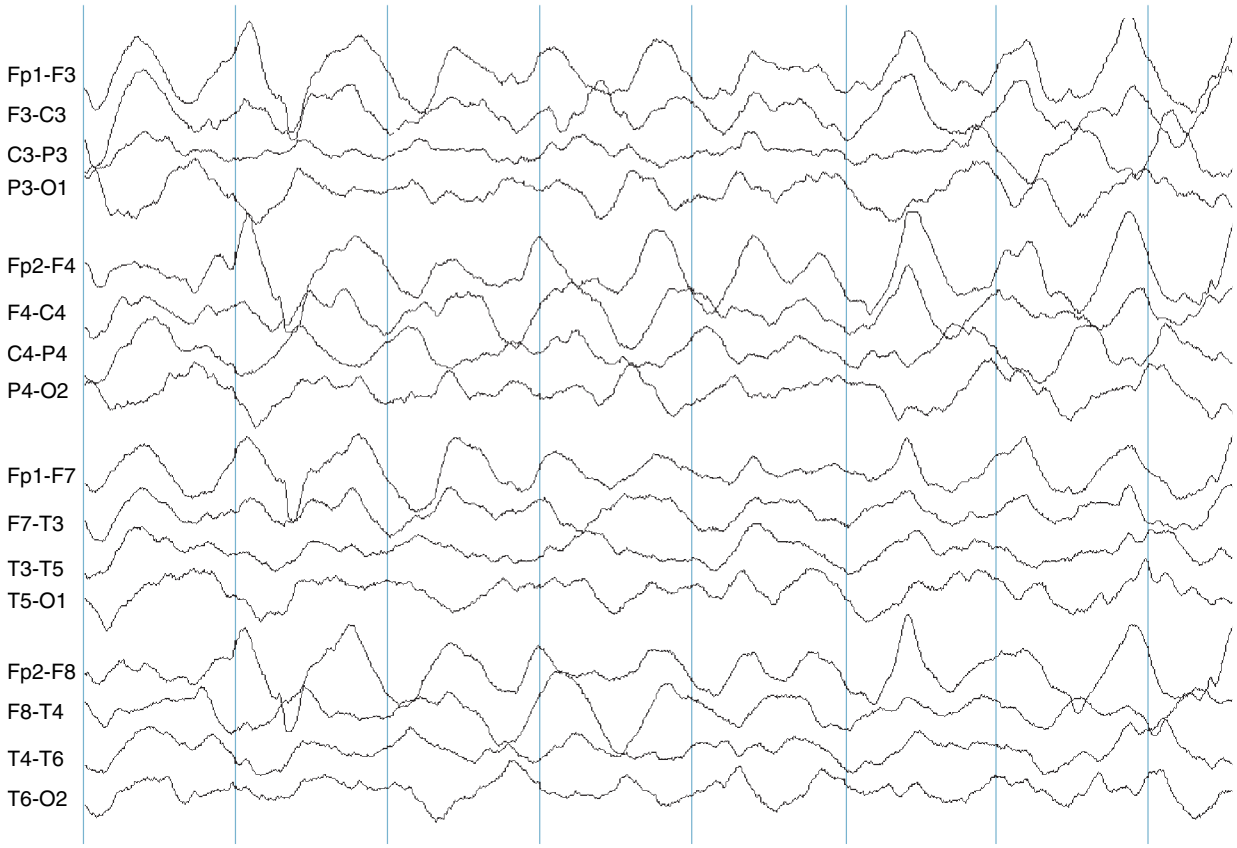

Figure 2-22 By definition, 50% or more of Stage IV sleep samples consist of delta activity, as is seen in this sample.

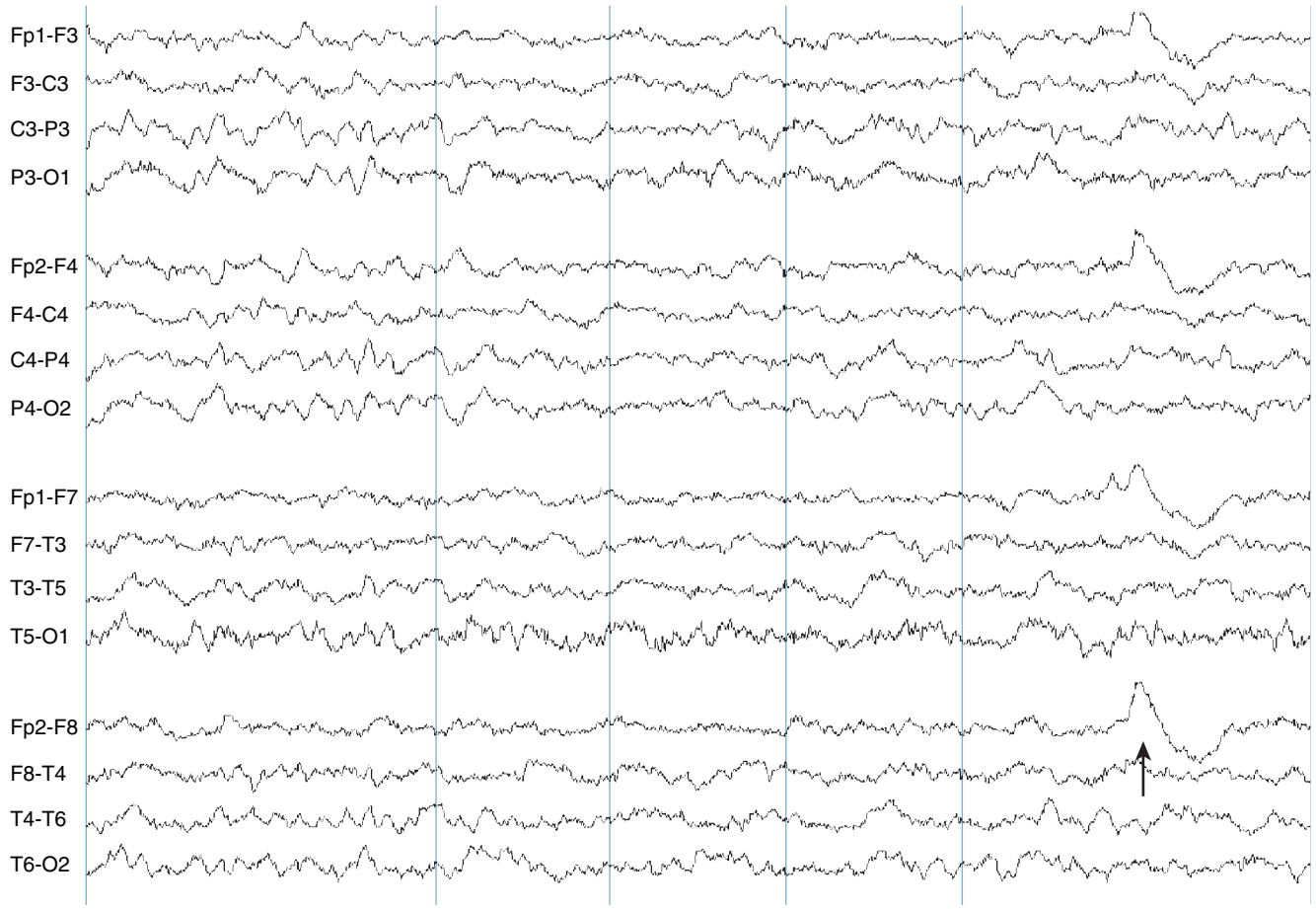

Figure 2-23 Rapid eye movement (REM) sleep, or dream sleep, is also referred to as *paradoxical sleep* because of the overall decrease in voltages that somewhat resembles an awake pattern. REMs are best seen in specialized channels designed to pick up eye movements but may also be detected in the frontal electrodes that are near the eyes, such as the large deflection seen in the four frontal channels in this example (arrow).

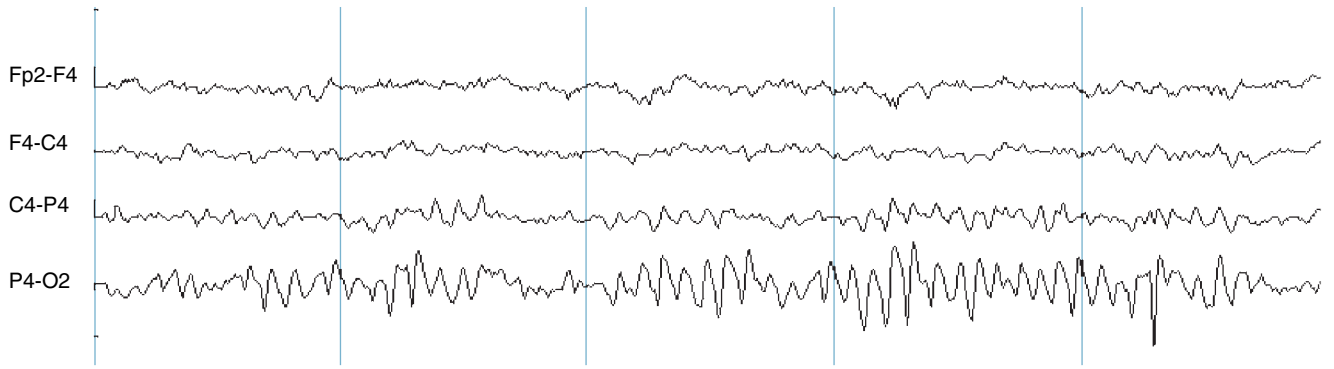

Figure 2-24 This example of the posterior rhythm is distinctive because portions are not sinusoidal (i.e., rounded on the tops and bottoms) but rather have a "spiky" appearance at the bottom of the waves. It is important not to mistake this pattern for true spikes in the EEG. The clue that these spiky waveforms do not represent spikes is that they clearly fit into the posterior rhythm; true spikes would appear to interrupt the rhythm.

for the posterior rhythm represent what the reader will commonly encounter during the interpretation of a large number of EEGs in different age groups; these typical values are generally higher than the cutoff values used for determining normal versus abnormal. The figures that represent the lower limit of normal are, perhaps, the most important to commit to memory because these figures will determine which tracings the reader will label normal or abnormal (see Table 2-2).

Beta (Fast) Activity

EEG fast activity is usually low-voltage activity. Because of its low amplitude, it is easy for fast activity to escape notice on first review unless the reader makes a point of looking for it. These lower voltage fast waves generally range from 13 to 30 Hz and sometimes look like nothing more than low-voltage "noise" or fuzziness that rides on the larger, slower waves (see Figure 2-25). Indeed, fast activity arising from the brain can appear fairly similar to the artifact from the electrical activity that muscles generate ("muscle artifact" or electromyogram artifact, usually called "EMG artifact"). Because electroencephalographers train themselves to filter out muscle artifact mentally to "see" the cerebral activity, it can be easy for fast activity's features to be missed during this visual filtering process. For this reason, fast activity in the EEG should be deliberately analyzed separately.

Although there is no official upper limit to the frequency of what can be called beta activity in the EEG, there are practical limits to what can be seen on the EEG recording. Because it is known that the majority of very fast activity in routine EEG recordings represents electrical noise rather than brain wave activity, the filter settings routinely used for most recordings are designed to screen out any very fast activity that might present, be it from the brain or from an artifact source. Other technical constraints of the recording instrument also limit the amount of very fast activity that can be seen. These constraints include the specific design of amplifier circuits and, in the case of digital EEG machines, the sampling rate at which EEG data is acquired. Currently, analysis of very fast activity (e.g., "fast ripples") is an area of ongoing research interest but does not yet play a part in conventional EEG interpretation.

Table 2-2 Typical Frequencies of the Posterior Rhythm and Lower Limits of Normal by Age

Age	Typical Posterior Rhythm (Hz)	Limit of Normal (Hz)
4 months	3–4	*
5 months	5	*
1 year	6	5
2 years	7	
3 years	8	6
6 years	9	7
8 years		8
13 years	10	

*Because a posterior rhythm may be difficult to discern in these age ranges, a lower limit of normal is not given.

The Anteroposterior Gradient

Fast activity is usually most prominent in the anterior brain regions and becomes less conspicuous in the posterior regions. At the same time, the overall voltage of all waves in the EEG during wakefulness generally increases going from anterior to posterior head regions. This gradual decrease of fast activity and gradual increase in amplitude going from front to back head regions during wakefulness is referred to as the *anteroposterior gradient* of the waking EEG. This gradient is shown in a referential montage that uses the nose as the reference electrode in Figure 2-26 and also in a bipolar montage in Figure 2-27. This gradient represents the structure of the typical waking EEG, but whether absence of the usual gradient represents an abnormality is dependent on the specific clinical context.

Fast activity may appear to be absent from the EEG for technical reasons related to EEG instrument settings. When a particular EEG tracing has intrinsically high background voltage, amplifier gains are usually set lower so that high-amplitude channels do not cross other channels and obstruct each other. Because fast activity is usually low-voltage activity (and an increase in the voltage of the background is usually not associated with an increase in the voltage of the fast activity), the low-amplifier gains used to display high-voltage EEGs disadvantage the display of any fast activity, which may become visually lost. When this occurs, high-voltage slow waves attain a "smooth" appearance. Resetting the amplifiers to more standard settings allows the lower voltage fast activity to reappear, although the remainder of the tracing may be rendered more difficult to interpret (see Figure 2-28). When a tracing appears to be devoid of fast activity, the possibility that amplifier gains are set low should be considered. Alternatively, use of more aggressive low-filter settings is a more advanced technique that can be used to display fast activity in a high-voltage record, discussed in Chapter 7.

DROWSINESS

Theta Activity

Theta activity refers to activity between 4 and 8 Hz. Waves in the theta range are generally considered abnormal in the adult EEG during wakefulness; however, the appearance of theta waves is one of the hallmarks of the onset of drowsiness. Theta waves are usually of low voltage and can appear in any brain area; in some, theta waves are most prominent occipitally in early drowsiness (see Figure 2-29). Theta waves may also be seen in the temporal regions during drowsiness,

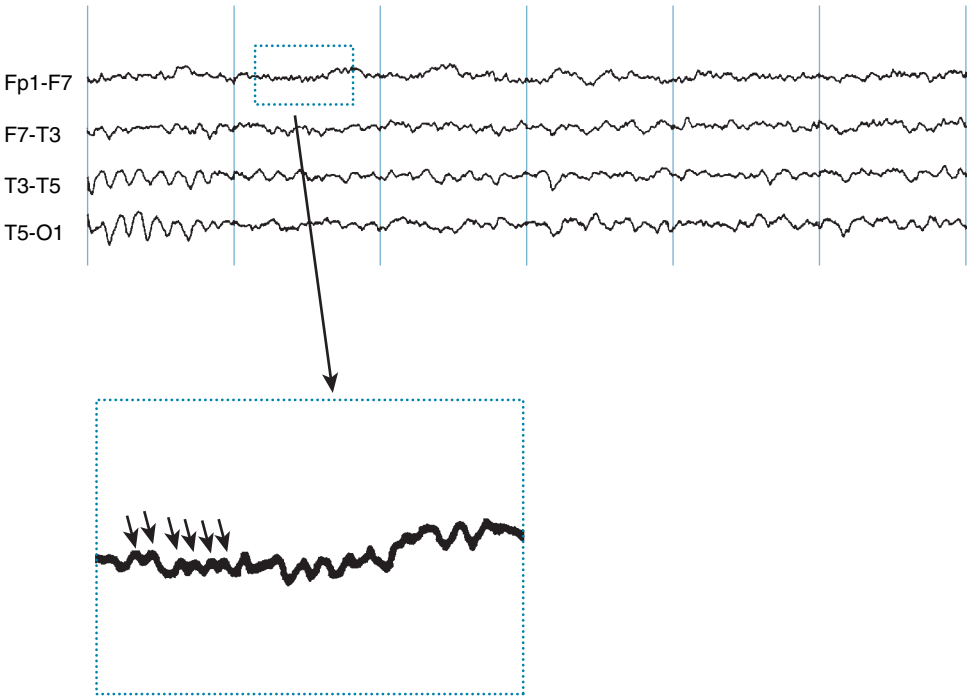

Figure 2-25 This figure shows a close-up of the beta activity present in the top channel of a bipolar montage. The small arrows indicate each beta wave. Note that in the unmagnified version, it would be easy to skip past this low-voltage fast activity without noticing it.

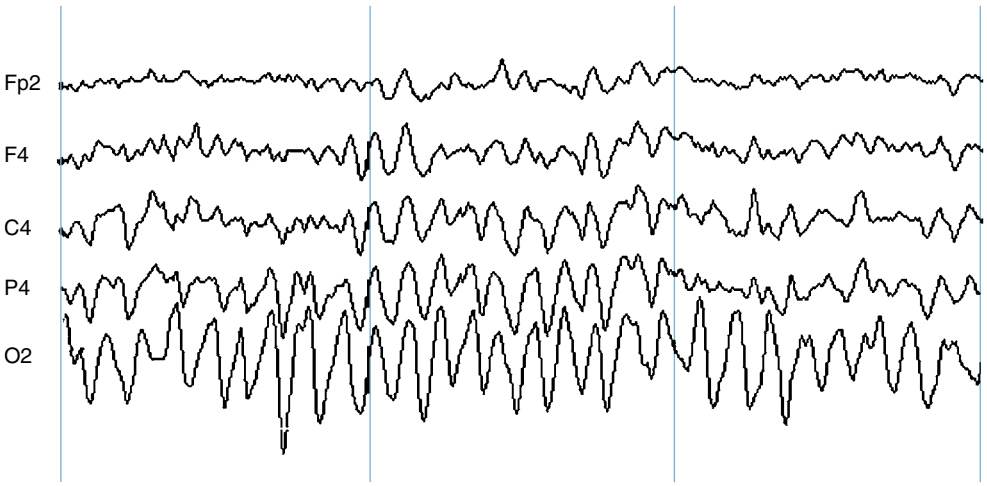

Figure 2-26 This referential recording of an awake patient shows the anteroposterior gradient of fast activity and an opposite gradient of slower activity. Low-voltage fast activity is most prominent in Fp2 and diminishes in the posterior channels. Slower activity, in the form of the posterior rhythm, diminishes in amplitude in the more anterior leads.

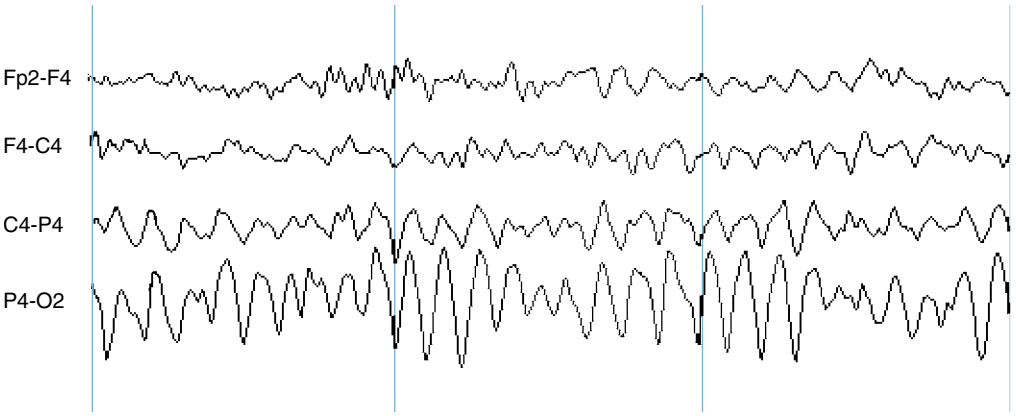

Figure 2-27 This bipolar recording illustrates the same effect as was seen in the previous example, with more plentiful faster rhythms seen frontally and higher voltage slower rhythms seen posteriorly.

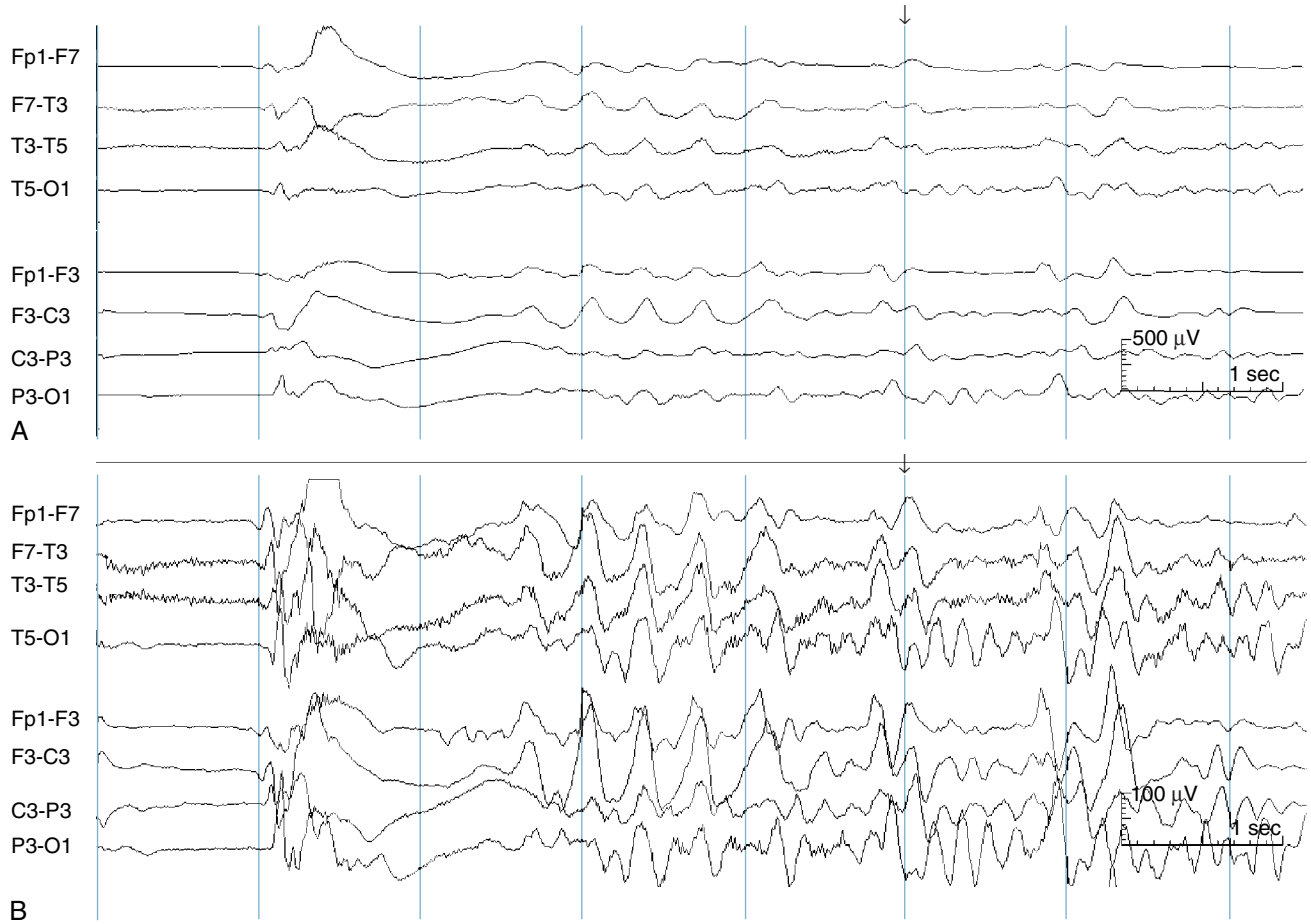

Figure 2-28 **(A)** The tracing shown in this portion of the figure appears "smooth" and relatively devoid of fast activity. In this example, the apparent lack of fast activity is, however, a consequence of the low amplifier gains used (note the calibration bar in the lower right-hand corner). Because some of the waves are of 400 to 500 μV, amplifier gains were set low, which has resulted in the lower voltage fast activity being lost. **(B)** The exact same tracing is shown here with amplifier gains at settings that are fivefold higher. Plentiful fast activity can now be appreciated. When a tracing appears to lack fast activity, the possibility that amplifier gains are set low should be considered.

and in others, they may be seen diffusely. Occasionally, theta waves (or slower delta waves) can be seen in drowsiness in rhythmic, hypersynchronous runs (see Figures 2-30 and 2-31). These rhythmic slow-wave runs, referred to as *hypnagogic hypersynchronies,* can be quite dramatic at times. Similar runs can also be seen on arousal, *hypnopompic hypersynchronies,* which can also be quite dramatic and lengthy (see Figures 2-32 and 2-33). Occasionally, these types of sleep-related hypersynchronies may be difficult to distinguish from seizure activity, which can also express as a high-voltage, rhythmic discharge. Usually, hypnagogic and hypnopompic hypersynchronies are not difficult to distinguish from epileptic seizure activity in that the hypersynchronies maintain a consistent frequency throughout their duration (rather than speeding up or slowing down as electrographic seizures typically do), and they lack clear sharp components.

Fast Activity

Although fast activity is detectable in most waking EEGs, a relatively sudden, diffuse increase in beta activity can mark onset of early drowsiness (see Figure 2-34). In some, this increase persists into light sleep, but any increased beta activity usually becomes less noticeable in Stage II sleep and beyond. Increases in beta activity with drowsiness are not seen, however, in all patients.

Posterior Rhythm

One of the earliest signs of drowsiness is a subtle decrease in the frequency of the posterior rhythm, although this slowing is usually seen only briefly before the posterior rhythm disappears entirely. In general, the posterior rhythm frequency only slows by approximately 1 to 2 Hz before it disappears, and such a deceleration is not seen in all patients. Before it finally disappears, theta activity may intermix with the posterior rhythm. When the posterior rhythm disappears completely, low-voltage, irregular theta activity can be seen in its place.

When the posterior rhythm disappears, there are, then, two main possible explanations: either the patient's eyes have opened or the patient has become drowsy. These two possibilities can be distinguished by close assessment of the EEG. When the posterior rhythm suppresses because of eye opening, it is usually

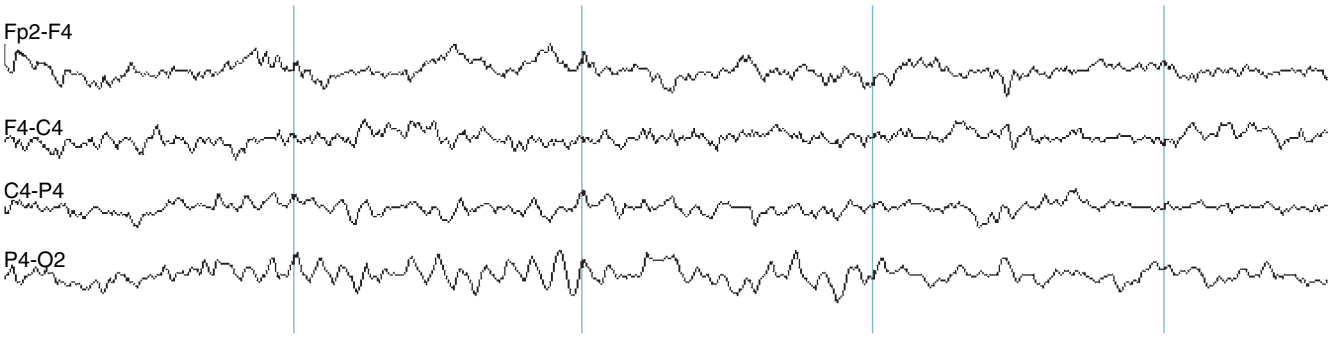

Figure 2-29 The posterior rhythm, seen in the P4-O2 channel, is gradually obliterated by theta activity as the patient passes from wakefulness to drowsiness. Theta waves are seen in other channels as well. The persistence of theta waves in the location previously occupied by the posterior rhythm is confirmation of a transition to drowsiness (see Figure 2-35 for comparison).

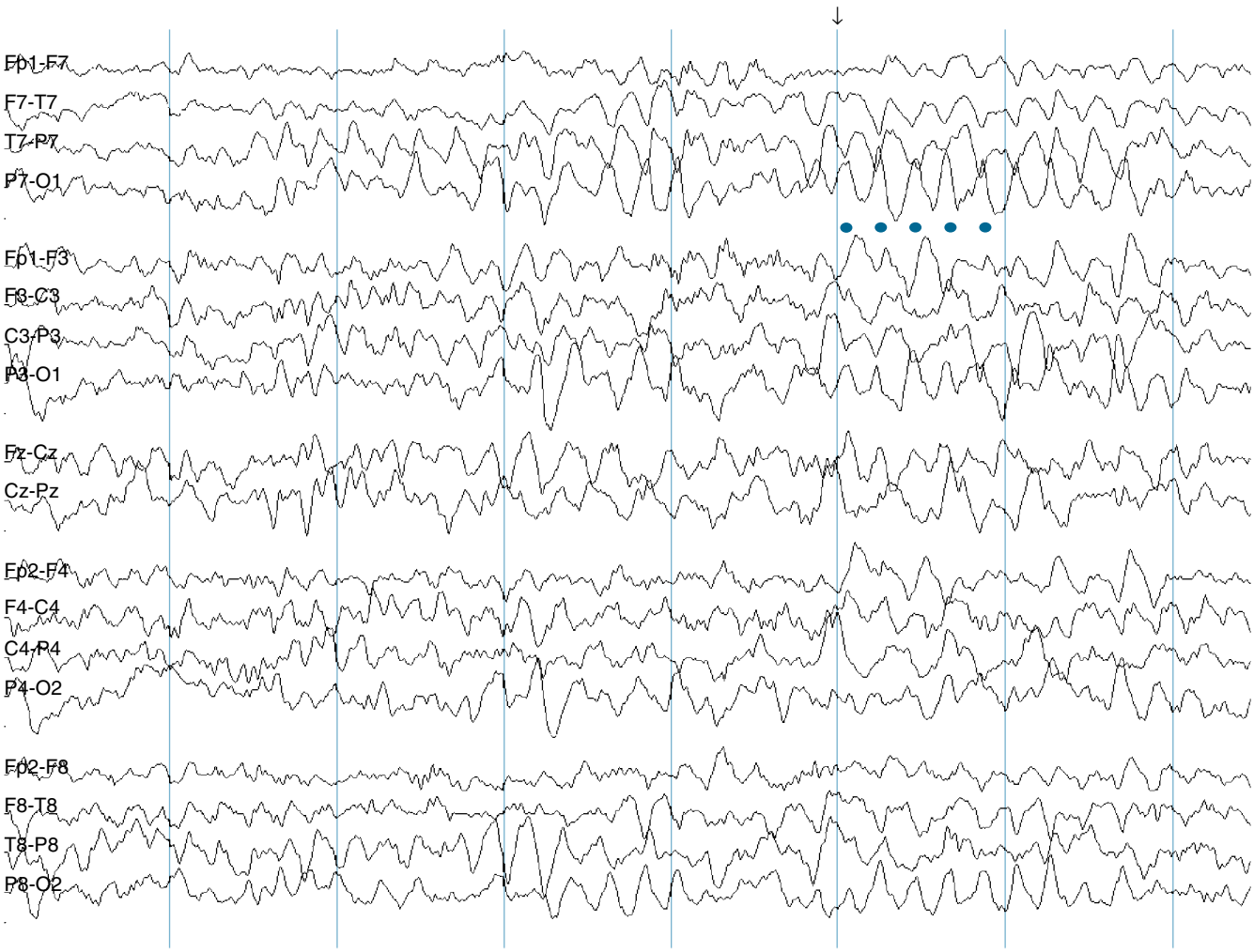

Figure 2-30 Hypersynchronous slowing with drowsiness. As this patient transitions from wakefulness to drowsiness, a fairly dramatic 5-Hz rhythmic slow wave is seen in multiple locations on the page, but most prominently in the posterior regions in this patient. A few of the hypersynchronous waves from the left occipital area are highlighted by the blue dots. This pattern may be seen during evolution to Stage II sleep.

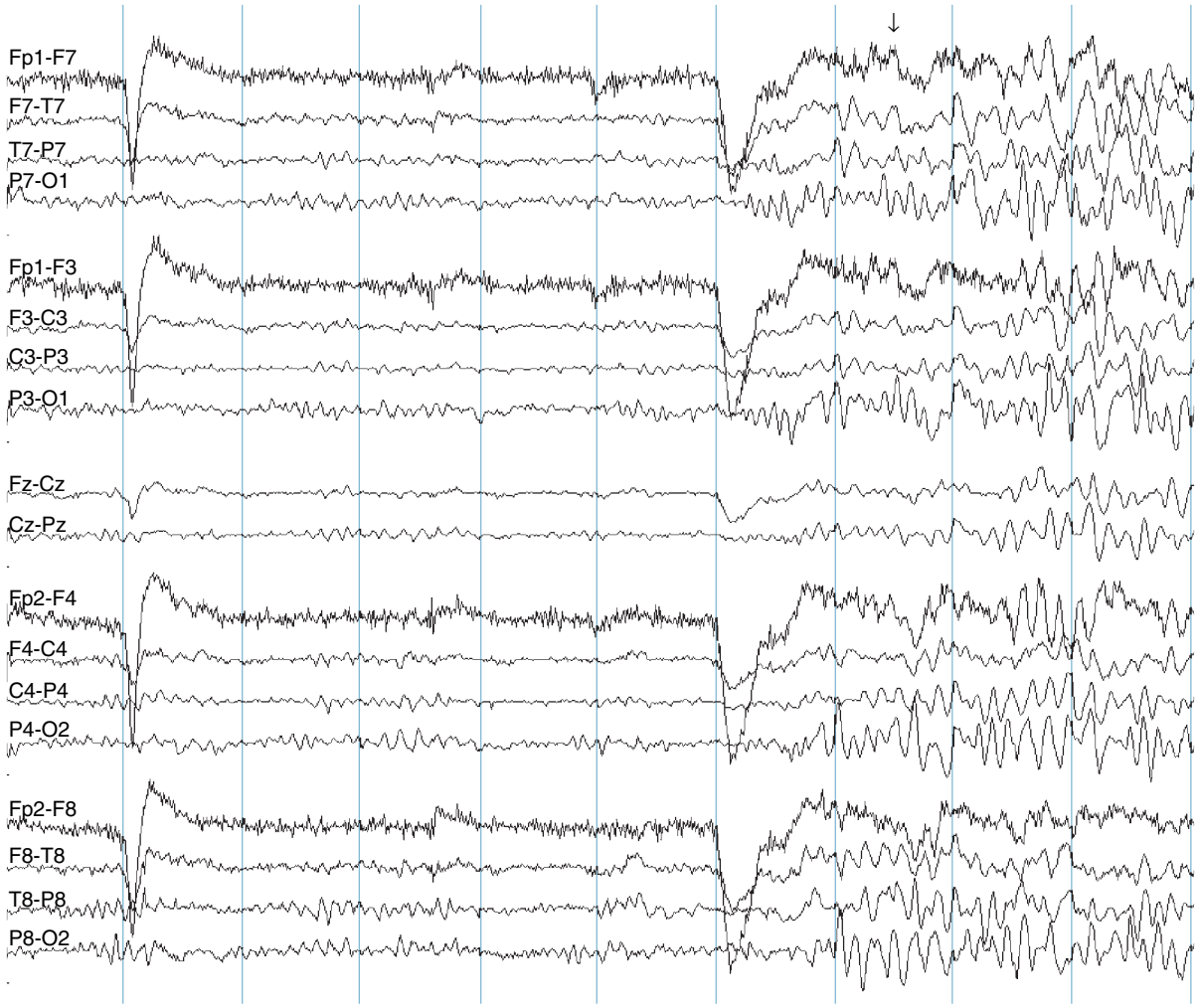

Figure 2-31 This figure shows a more abrupt transition from wakefulness to drowsiness. Eyeblinks and the posterior rhythm are visible on the first half of the page, followed by diffuse, rhythmic theta heralding the drowsy state during the last three seconds.

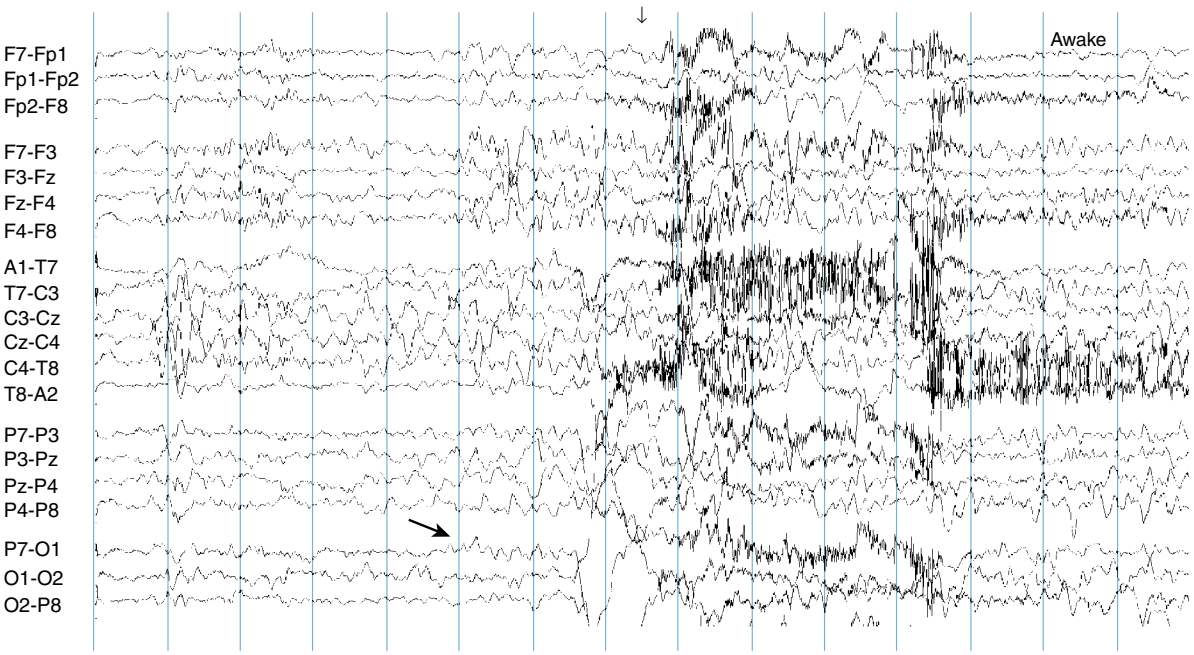

Figure 2-32 A typical appearance of an arousal from stage II sleep. This EEG, displayed in a transverse montage, shows a vertex wave and spindles in the first 3 seconds, followed by 4 seconds of rhythmic, hypersynchronous activity that represents a hypnopompic (arousal) hypersynchrony. The appearance of the posterior rhythm (arrow) and muscle artifact herald wakefulness.

replaced by a flat, nondescript tracing as in Figure 2-35. When the posterior rhythm drops out because of drowsiness, it is usually replaced by mixed theta frequencies as was seen in Figure 2-29. If disappearance of the posterior rhythm is related to drowsiness, the EEG record may show other evidence of drowsiness, such as slow roving eye movements (discussed next), an increase in beta activity, or increased theta activity in other locations.

Slow Roving Eye Movements of Drowsiness

Eye movement artifacts seen in the EEG do not represent electrocerebral activity, but they may provide valuable information to the electroencephalographer regarding sleep state. In drowsiness, after the patient's eyes are closed, slow lateral eye movements can be appreciated by virtue of the artifacts they produce in the anterior leads of the EEG (see Figure 2-36). Although not all patients manifest such eye movements, they occasionally represent the only identifiable sign of drowsiness.

Knowing whether a patient is drowsy can be important in a variety of reading situations. For instance, if theta waves are present in the tracing of an alert adult patient, their presence could constitute an abnormality. If, however, the electroencephalographer notes that slow roving eye movements accompany the theta waves, then it can be surmised that the segment in question represents early drowsiness, and the theta waves may be considered normal "drowsy waves."

IDENTIFICATION OF EXPECTED ELEMENTS: Sleep

Vertex Sharp Waves of Sleep

Vertex sharp wave transients appear in Stage I sleep and continue into Stage II sleep. These waves are often of high amplitude, especially in children, and have a voltage maximum in the central electrodes: Cz, C3, and C4 (see Figure 2-5 earlier in the chapter). These waves can be multiphasic, meaning that each wave may include more than one up-and-down deflection. In some patients, they are blunted in appearance, but in others they can be quite sharp. The polarity of vertex sharp waves of sleep may be either positive or negative, but they are more often negative (wave polarity is discussed in more detail in Chapter 4, "Localization"). When light sleep is well established, vertex waves appear every several seconds in a more or less rhythmic or semiperiodic fashion. Indeed, when the rhythmic appearance of vertex waves is established and then halts, this is often a clue that, rather than continuing into deeper sleep, there has been a subarousal, perhaps caused by a noise in the environment or an internal stimulus. Sleep apnea may also be a cause of arousals or subarousals.

Sleep Spindles

By definition, the appearance of sleep spindles marks the onset of Stage II. The term *spindle* refers to the fusiform, or sausage-shape, of the spindle waveform as it evolves over one to several seconds, resembling the shape that thread winds onto a wooden spindle. Despite their name, spindles often do not manifest a spindle shape; many spindles maintain a relatively constant amplitude throughout their evolution (as was seen in Figure 2-7). Spindles are easily recognized by their characteristic frequency and location. The most typical spindle frequency is 14 Hz, although slower frequencies at the bottom of the 12- to 14-Hz range can be seen. Likewise, the most typical spindle location is at the vertex and central areas bilaterally (Cz, C3, and C4 electrodes), similar to the field of vertex waves as described in the previous paragraph. Especially in children, spindles may appear to migrate anteriorly with deepening Stage II sleep, at which time they may become most prominent in the frontal regions. Especially when they appear more frontally, spindle frequencies may fall as low as 12 Hz (see Figure 2-37).

Spindle duration varies to some extent according to age, with longer spindles seen in infancy and early childhood. In infants, spindles of 3 to 4 seconds are common, whereas in adults, spindles of 1 to 2 seconds are typical. Spindle interhemispheric synchrony (the tendency for spindles to occur in each hemisphere at the same time) also occurs as a function of age. Particularly in infants aged under 12 months, the initial spindles seen at onset of Stage II sleep are often asynchronous, occurring first on one side of the brain and then on the other (see Figure 2-38). As Stage II sleep deepens, even in infants, there is a tendency for spindles to become more synchronous. After 24 months of age, spindles are expected to occur bisynchronously, even at Stage II onset.

Asynchrony of sleep spindles should be distinguished from asymmetry of sleep spindles. In the case of *asynchrony*, spindles may occur over one hemisphere followed by their occurrence over the opposite hemisphere; over time there should be a tendency for the spindles to "even out" right versus left. When spindles show a persistent *asymmetry*, either in their amplitude or in the number that appear over each hemisphere, an abnormality should be suspected.

K-Complexes

K-complexes are complex, polymorphic, and often dramatic bursts that occur during sleep. There is some disagreement as to the precise meaning of the term *K-complex*. Some have used the term synonymously with vertex waves of sleep. Others have used the term K-complex to refer to a combination of a sleep spindle superimposed on a vertex wave. The best use of the term probably remains the original definition of the K-complex: a complex of waveforms generated during a sound stimulus occurring during sleep (Loomis 1936; see Daly and Pedley p. 183). These waves were recognized as the burst of activity seen when a noise occurred, planned or unplanned, in the recording environment while the patient was in Stage II sleep. Although such

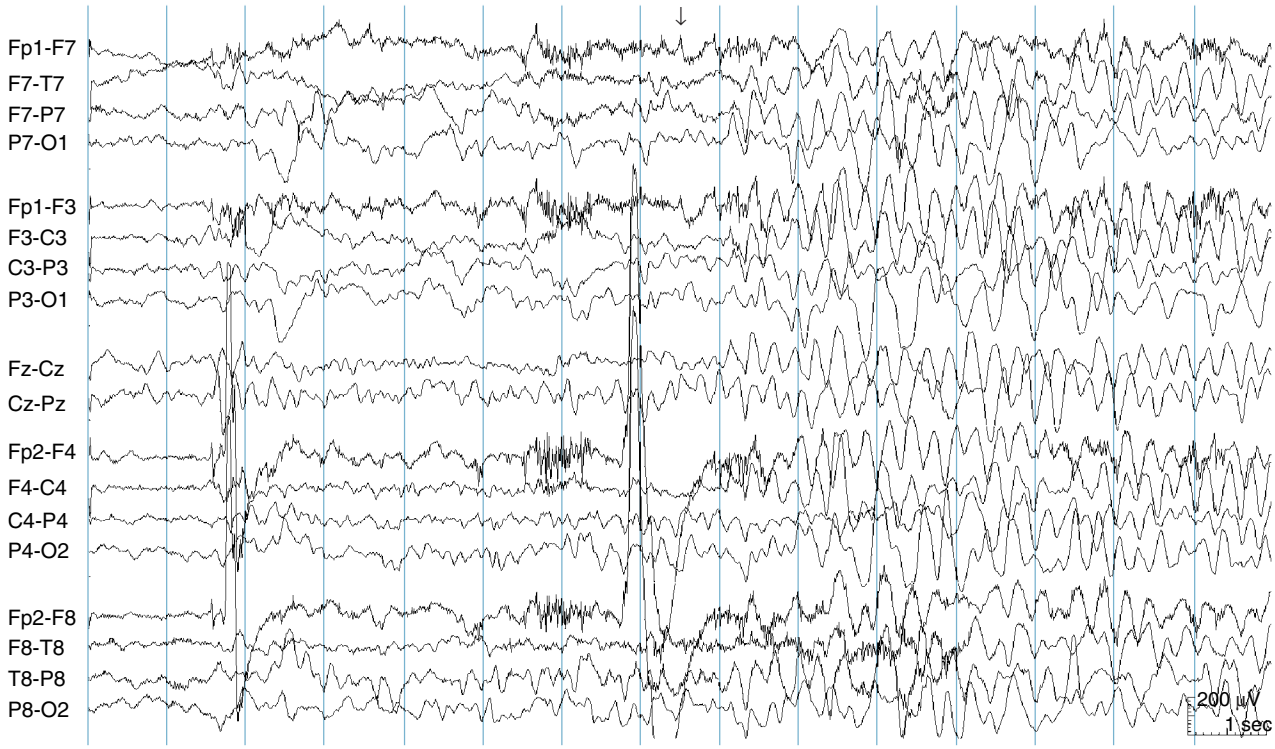

Figure 2-33 High-voltage rhythmic waves dominate the second half of this page and represent an arousal hypersynchrony. The darkened channels and high-voltage deflections seen particularly on the first half of the page represent muscle and motion artifact related to the arousal.

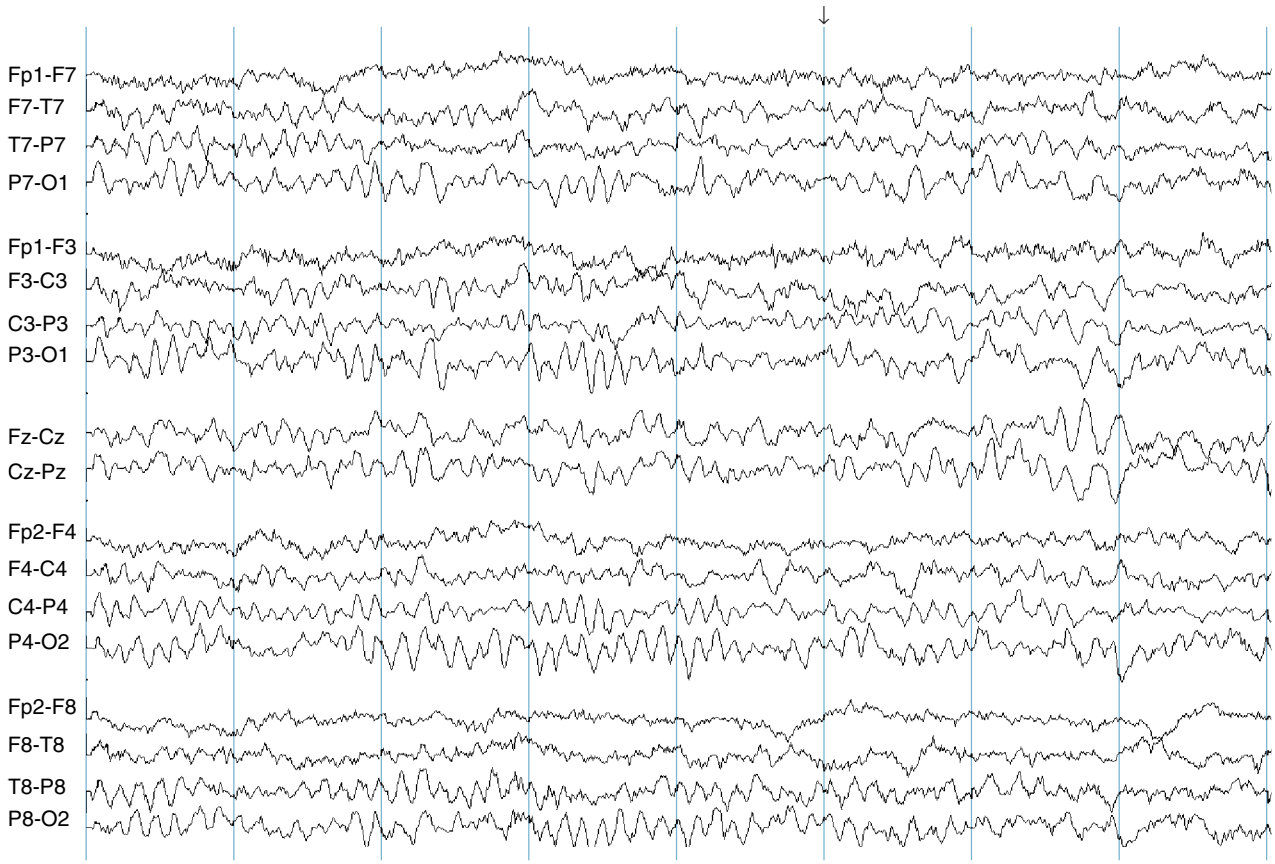

Figure 2-34 Increased fast activity associated with onset of drowsiness. Note the increased amount of very low-voltage fast activity riding the larger waves in nearly every channel. Although most obvious in the frontal channels, the increased fast activity can even be seen superimposed on the posterior rhythm. Increased fast activity is not consistently seen at the onset of drowsiness.

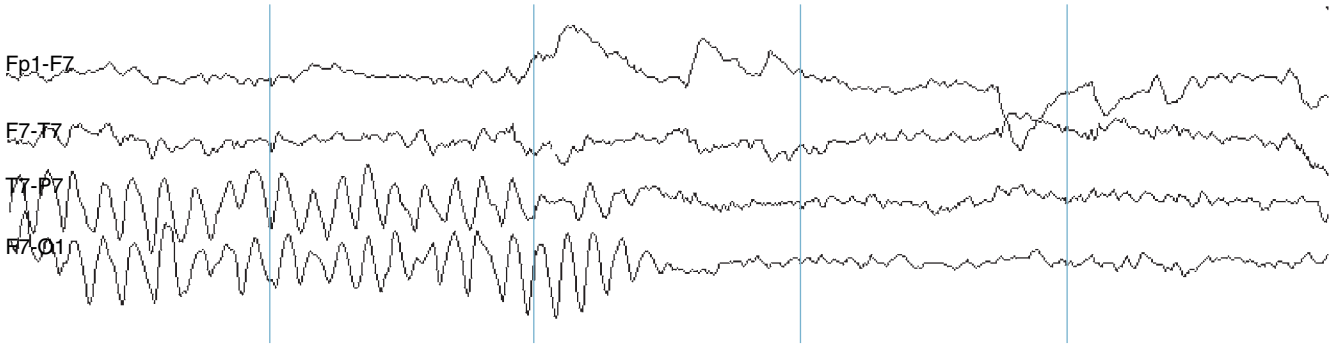

Figure 2-35 In contrast to Figure 2-25, in this example the posterior rhythm transitions to a nearly flat pattern in the bottom two channels suggesting suppression of the posterior rhythm from visual attention or eye opening rather than from onset of drowsiness. This idea is backed up by the presence of the jagged eye movement artifact seen in the top channel (Fp1-F7), which also suggests that the patient is awake and alert.

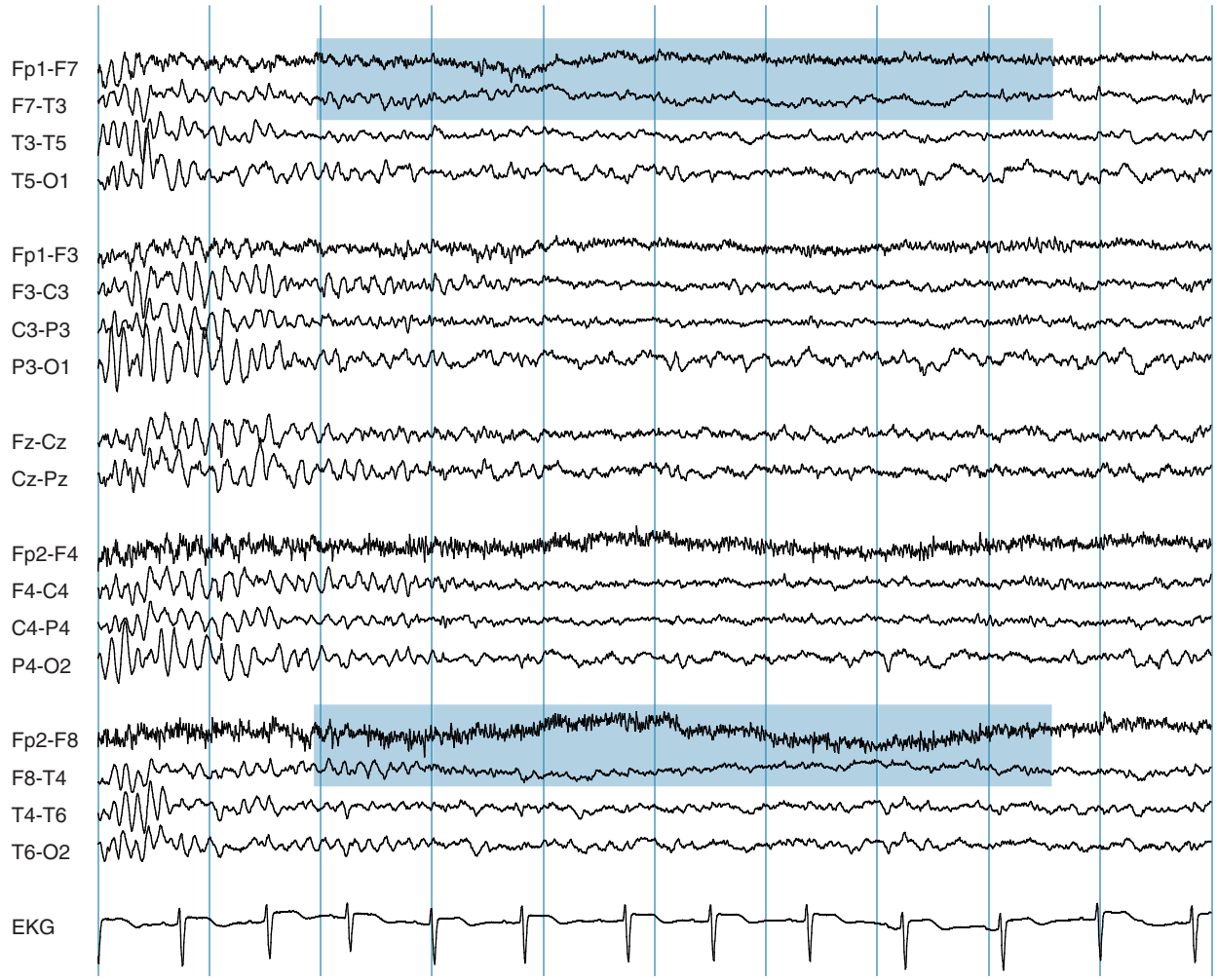

Figure 2-36 Slow roving eye movements are noted in the anterior temporal channels in the form of slow, phase-reversing waves, marking drowsiness. Note that the channels that include the F7 electrode (top blue rectangle) become closer to each other at the same time that the channels that include the F8 electrode (bottom blue rectangle) become farther apart. This distinctive pattern is consistent with lateral eye movement artifact, discussed in more detail in Chapter 6.

complexes often do contain a mixture of spindles and vertex-wave activity, it is not required that these elements be identifiable within a K-complex. The broader definition is important because K-complexes elicited by sound often manifest an electrical field that is much more widespread than those of spindles and vertex waves. Unlike spindles and vertex waves, which are particularly concentrated in the central areas (Cz, C3, and C4) and much less-so in the temporal areas, K-complexes often involve all brain areas at once. The difference in the field distribution of sleep spindles and K-complexes can be seen when comparing Figures 2-6 and Figure 2-13 and is schematized in Figures 2-11 and 2-12.

Because some K-complexes resemble spike-wave discharges, it is important to be able to distinguish between this normal sleep finding and abnormal epileptiform activity. Although K-complexes may occur with auditory stimuli, for several reasons, it is not necessary to prove that a sound has elicited the waveform to identify a K-complex. First, one may not know whether a sound has occurred in the patient's environment because this fact is not always noted on the EEG. Second, K-complexes may be associated with subarousals, which may also be caused by nonauditory stimuli, even internal stimuli, which could be impossible for an outside observer to identify.

Slow Wave Activity: Theta and Delta Activity

Theta activity, and especially delta activity, are hallmarks of slow wave sleep. In addition to the appearance of K-complexes, vertex waves, and sleep spindles, Stage II sleep may contain up to 20% delta activity. By definition, Stage III sleep epochs contain 20% to 50% delta activity. Sleep spindles may persist into Stage III sleep and are seen intermixed with the slow-wave activity. By definition, Stage IV sleep contains more than 50% delta activity. With the appearance of Stage IV sleep, spindles all but disappear.

Identification of Expected Elements: Arousal

Although in some cases, arousal from sleep is marked by an unceremonious transition from a sleep pattern to an awake pattern, in many a hypersynchronous run of slowing accompanies the transition. In addition, because most patients stir somewhat during an arousal, it is typical to see a combination of muscle and motion artifact at the time that patients awaken (see Figure 2-32).

VISUAL ANALYSIS OF THE EEG: Identification of Abnormal Elements

The second step of the process of visual analysis is identification of abnormal findings in the EEG. Although it may be useful to ask the question, "do I see spikes here?" epileptiform activity will often come to the EEG reader's notice without the need to go through a formal inventory of potential abnormalities. Other types of abnormalities, such as voltage asymmetries, are more subtle, and it is worthwhile to run through a list of the various frequency bands as such asymmetries may not be immediately obvious to the eye without specifically searching them out.

Asymmetries of voltage are best consciously inventoried according to frequency band, sequentially asking whether delta, theta, alpha, and beta activity are similar on both sides. Asymmetries of specific elements may also be seen, such as asymmetric sleep spindles or asymmetry of the posterior rhythm.

Additional abnormalities to look for include the broad category of "epileptiform abnormalities," including spikes, sharp waves, spike-wave complexes, and certain repetitive patterns. Continuous slow waves and intermittent slow waves may also represent abnormalities, depending on the context. These and other types of EEG abnormalities are discussed in more detail in Chapter 9.

SUMMARY OF ELEMENTS OF WAKEFULNESS AND SLEEP

- Wakefulness:
  - Posterior rhythm is seen when eyes are closed.
  - Anteroposterior gradient of fast activity and voltage:
    - Beta activity is seen most plentifully anteriorly and diminishes posteriorly.
    - Amplitudes are lower anteriorly and increase posteriorly.
- Drowsiness:
  - Posterior rhythm slows and "drops out," replaced by theta frequencies.
  - Rhythmic theta (4–8 Hz) runs may be seen.
  - Diffuse increase in fast activity may be seen.
  - Artifacts of "slow roving eye movements of drowsiness" may be identified.
- Sleep:
  - Stage I
    - Vertex sharp waves appear
    - K-complexes
  - Stage II
    - Sleep spindles appear
  - Stage III
    - 20%-50% delta activity.
  - Stage IV
    - >50% delta activity
  - REM
    - Flattening to a low-voltage pattern with rapid eye movements and sawtooth waves

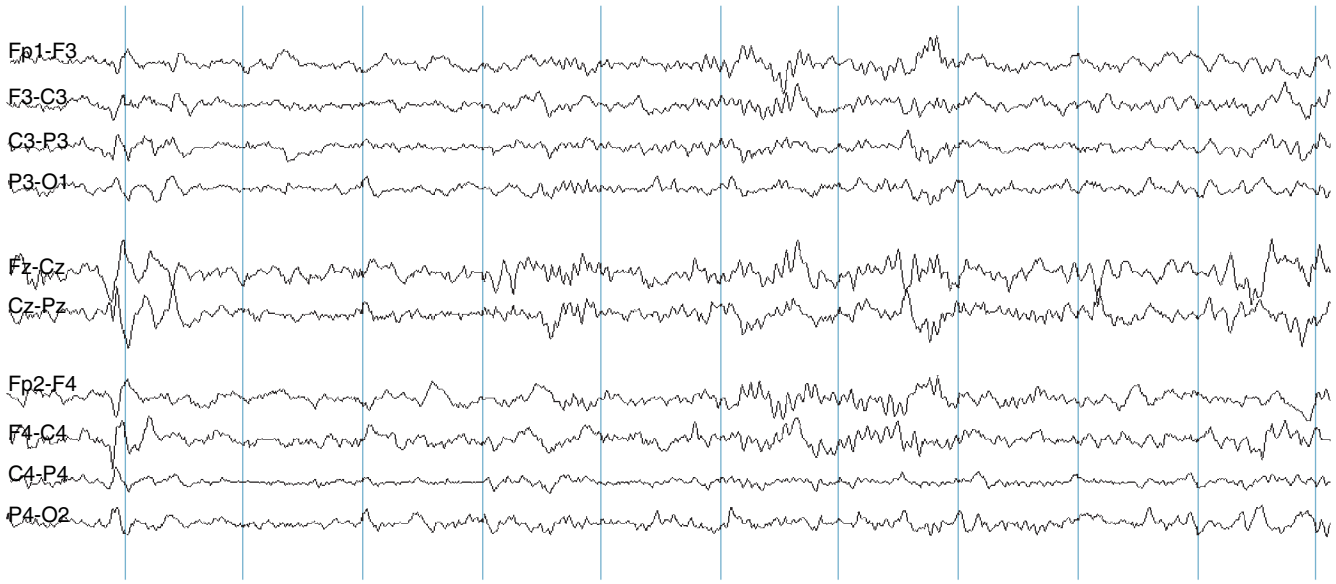

Figure 2-37 In some subjects, especially in deeper Stage II sleep, the sleep spindle field becomes more anterior (frontal and central) rather than purely central. Note that in this example, the spindles are most visible in the Fp1-F3, F3-C3, Fz-Cz, Cz-Pz, Fp2-F4, and F4-C4 channels.

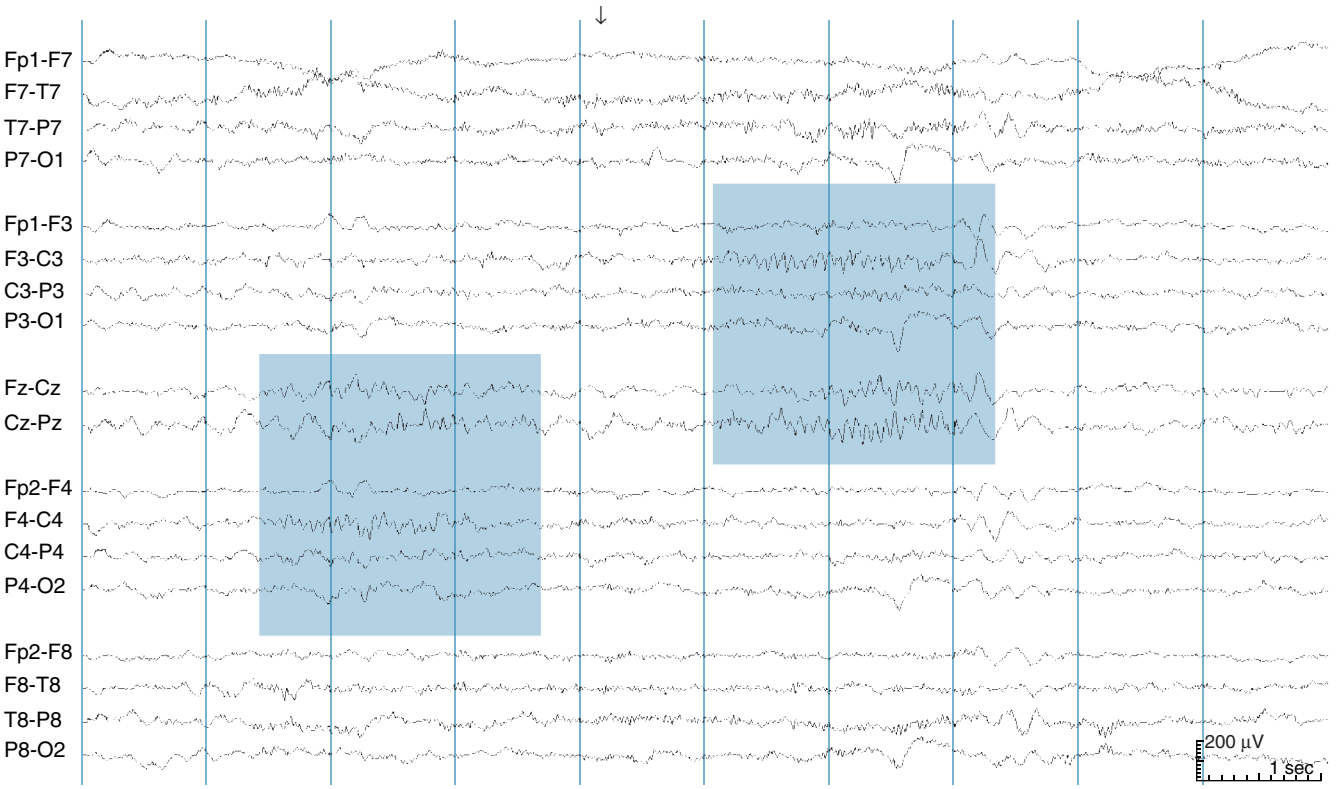

Figure 2-38 Asynchronous spindle activity in an infant. Unlike older children and adults in whom spindle activity generally occurs synchronously over both hemispheres, in infants spindles may alternate over the hemispheres in early Stage II sleep. In deeper Stage II sleep, even in infants, spindles often become bilaterally synchronous.

SUGGESTED READINGS

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Chapter

3 Introduction to Commonly Used Terms in Electroencephalography

In this chapter, we review the basic terms used in electroencephalography and strategies for communicating EEG findings to others. The creation of a concise description of an EEG tracing is a key part of the art and science of EEG interpretation. The ideal EEG description allows its reader, on the basis of the report alone, to visualize the appearance of the EEG even in the absence of the tracing. The report also includes a section that describes the technique used to record the EEG and a separate clinical interpretation of the results that discusses the potential implications that the findings might have for the patient. The specifics of EEG reporting are discussed in more detail in chapter 8, the structure and philosophy of the EEG report.

Careful use and understanding of EEG terminology brings specific advantages to both the writers and readers of EEG reports. The most obvious benefit is that strict use of EEG terminology facilitates nonambiguous communication of EEG findings to others. The use of idiosyncratic terms or a personal EEG vocabulary should be avoided because they may not be fully understandable to others. Standard definitions for EEG terms were published in 1974 and more recently in 1999 (see Suggest Readings). Another reason for using a common vocabulary is to aid in the matching of EEG findings (e.g., "spikes" or "intermittent rhythmic slowing") to the clinical syndromes that may be associated with those findings. This is important in both clinical diagnosis and the conduct of research involving EEG. Finally, using the most precise term possible in a description helps discipline the EEG reader during both the interpretation and report preparation process.

DESCRIPTION OF EEG WAVES

As mentioned, an ideal description of an EEG waveform would allow the reader to visualize or perhaps even produce an accurate drawing of the wave on the basis of the written description alone. If a wave can be drawn two or more fundamentally different ways from a particular written description, the ambiguity in the description provides a clue as to how it could be improved. In general, a complete description of an EEG wave or event includes the following features: location, voltage (amplitude), shape (morphology), frequency, rhythmicity, continuity, and the amount of the wave seen and in which particular clinical states (awake, asleep) it occurs in the record (Table 3-1). For instance, a left temporal theta wave could be described as follows: "Occasional examples of intermittent, medium voltage, sinusoidal 6–7 Hz waves are seen in the left temporal area during drowsiness."

FREQUENCY

Frequencies are quoted either in cycles per second (cps) or in Hertz (Hz). The terms *cps* and *Hz* are synonymous. For historical reasons, each frequency band in the EEG is named using one of four Greek letters. By convention, the names of the letters are written out: delta, theta, alpha, and beta; the symbols for these Greek letters are not used. The terms delta, theta, alpha, and beta are used as a shorthand to refer to rhythms in specific frequency bands and are defined as follows:

delta	0 to <4 Hz
theta	4 to <8 Hz
alpha	8 to 13 Hz
beta	>13 Hz

These frequency ranges are straightforward, but a few observations are worth noting. Note that according to the formal definition, the alpha range is the only "all-inclusive" range, and includes both boundary frequencies (8 and 13 Hz). The other frequencies include the boundary frequency on one side of the range but not the boundary frequency on the other side of the range. For example, the theta range, for which the boundary frequencies are 4 and 8 Hz, includes 4 Hz but does not include 8 Hz (which belongs to the alpha range), although it does include 7.9 Hz.

Very Fast and Very Slow Activity

Although the list above implies that beta-range frequencies are unbounded on the high side, in reality EEG activity recorded at the scalp with routine EEG instruments rarely exceeds 30 to 40 Hz, and even

Table 3-1 Some Wave Parameters with Specific Descriptive Examples of Each

Descriptive Parameter	Example
frequency	given in cycles per second (cps) or Hertz (Hz) or by frequency range: delta, theta, alpha, or beta
location	occipital, frontopolar, generalized, multifocal
morphology	spike, sinusoidal waveform
rhythmicity	rhythmic delta, irregular theta, semirhythmic slow waves
amplitude	high voltage slowing, a 75 μV sharp wave
continuity	continuous slowing, intermittent theta, periodic sharp wave

signals from cortically implanted electrodes rarely exceed 50 Hz. High-frequency filtering techniques and other inherent equipment limitations make it difficult to see faster frequencies in routine recordings, even should they be there to record. The assumption behind using high filters is that little or no cerebral activity exceeds 30 to 40 Hz so that any signals above this range likely represent electrical noise or "artifact" from muscle, electrical interference, or other sources—hence, the strategy to filter out most activity above this range. In the early days of EEG, frequencies above 30 Hz were said to be in the *gamma range,* but the term (and the concept) was later discouraged. More recently, there has been renewed research interest in "gamma activity," although such very fast activity has yet to establish its place in the mainstream of conventional EEG interpretation.

Similarly, although the lower bound of the delta frequency range is "zero Hz," it was initially felt that there was little cerebral activity below 0.5 Hz. Newer techniques have successfully recorded very slow activity, which are termed *DC potentials* because they resemble shifts of the baseline (direct currents) rather than oscillations. Again, because standard recording techniques usually exclude most such very slow activity, frequencies much below 0.5 Hz are usually not reliably observed in conventional EEG recordings. Despite the possible existence of some very slow potentials in the EEG, in some situations, the great majority of activity below 0.5 Hz usually represents motion or electrical artifact rather than electrocerebral activity. Conventional filtering techniques attempt to remove these large baseline shifts from the recording because they usually represent artifact and may render the EEG tracing unreadable (see Chapter 7, Filters).

The term *slow activity* refers to any waves for which the frequency falls below the alpha range (i.e., delta and theta activity—activity below 8 Hz). Likewise, the term *fast activity* refers to activity for which the frequency lies above the alpha range (i.e., beta activity–activity above 13 Hz).

Frequency, Wavelength, and the Relationship of Frequency to Wavelength

The simplest way to assess the frequency of a wave is to count how many times it cycles within 1 second. If a wave cycles four times (i.e., manifests four "peaks" and four "troughs") in 1 second, then it is said to be a 4 cps or 4 Hz wave. At times, however, a wave cannot be counted for a full second. Luckily, it is possible to determine the frequency of a wave not just by counting how many times it cycles within a second but simply by measuring its duration or *wavelength* (measured from peak to peak or from trough to trough). The result of a straightforward calculation shows that if a wave cycles five times per second then each wave's duration must be one fifth of a second or 0.2 seconds. Similarly, if a wave cycles eight times per second, each wave will last one eighth of a second or 0.125 seconds. This yields the easy to remember relationship below, where λ gives the wavelength and f is the wave's frequency:

$$\lambda = \frac{1}{f}$$

The rearrangement of the above relationship is also useful and allows estimation of a wave's frequency by measuring its duration (wavelength) on the page, even if only a single wave is available for analysis:

$$f = \frac{1}{\lambda}$$

Both of these simple relationships always hold true. This allows the EEG reader to measure the duration (or wavelength) of any particular wave seen in the EEG (in seconds) and, by taking the reciprocal, to determine its frequency (in cps or Hz). Because standard EEG recordings show pages with vertical divisions of 1 second, the wavelength of a given wave can usually be easily measured or visually estimated by determining what fraction of a second it occupies. On some occasions, it is necessary to measure the wave directly, either with a ruler specifically designed for the purpose or with a digital time cursor in the case of digital recordings. The reciprocal of the wavelength measurement is calculated, yielding the frequency.

A Note on Units

The foregoing examples assume that wavelengths are stated in seconds, but it is common practice to quote wave durations in milliseconds (msec). Rather than stating that a wave lasts 0.2 seconds, it is often said that the wavelength is 200 msec. Using milliseconds is perfectly satisfactory, except when it comes to using the formula cited earlier to calculate frequency. The reciprocal of 200 is 0.005, which clearly is not the correct frequency of a wave of 200 msec duration. This is because inserting a wavelength in milliseconds into the formula yields the result in the undesirable unit of "cycles per millisecond." To yield the frequency in cycles per *second,* or Hertz, the wavelength used in the formula must always be stated in *seconds* (1 cycle/0.2 sec = 5 cps). Examples of how the

frequency of alpha, delta, theta, and beta activity are assessed are shown in Figures 3-1 through 3-4.

Waves of Mixed Frequency and the Fourier Theorem

The foregoing discussion deals with descriptions of simple waves of a single frequency. However, after a brief look at actual EEG recordings, it is clear that few, if any, of the waveforms seen on the EEG page resemble the pure sine waves seen in math textbooks. Indeed, as a rule, EEG waves represent a mixture of waves of different frequencies and amplitudes. Part of the reason for this mixture of frequencies is that the waveform recorded at the scalp often consists of a mixture of the products of different wave generators from various locations in the brain. In addition, some wave-generating circuits in the brain produce complex, nonsinusoidal waveforms.

One important skill in EEG interpretation is the ability to "deconstruct" EEG waves visually into their component frequencies. The idea that a repetitive complex wave can always be broken down into simpler, fundamental waves was proved by Joseph Fourier in a mathematical theorem called the Fourier theorem.

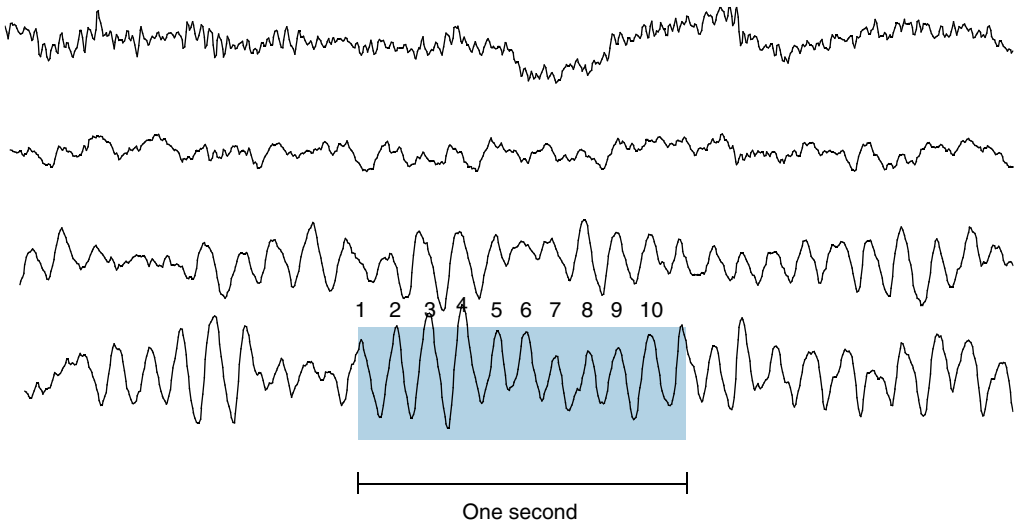

Figure 3-1 The frequency of the wave in the blue rectangle is most easily determined by counting the number of wave peaks or troughs seen in 1 second. These waves are rhythmic, fairly sinusoidal in shape, and vary in amplitude. In this example, 10 wave peaks are counted in a 1-second time period, indicating that this is an example of fairly regular 10-Hz alpha activity. This particular tracing represents an example of the posterior rhythm of a 7-year-old boy.

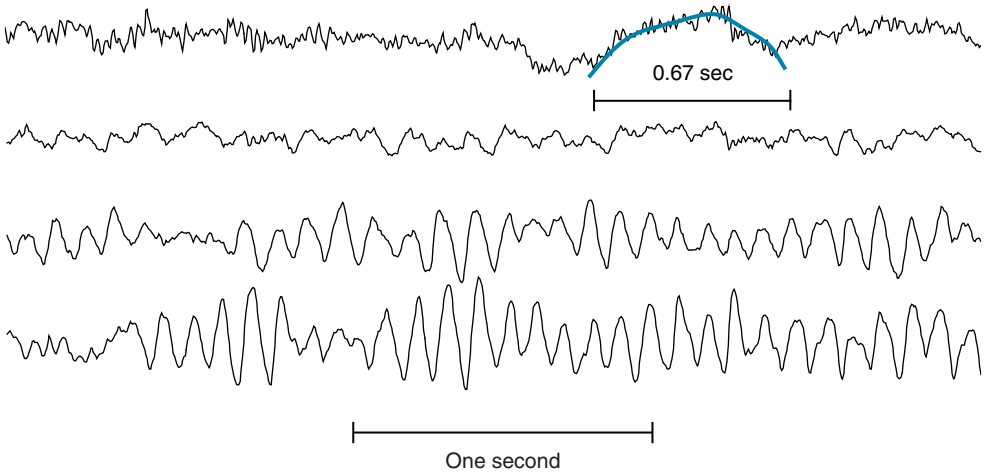

Figure 3-2 The frequency of the nonrepeating slow wave denoted by the penstroke in the top channel is most accurately assessed by measuring its wavelength. The wave in this example is 0.67 seconds in duration, measured from wave trough to wave trough. The simple relationship of wavelength to frequency described in the text in which frequency is the reciprocal of wavelength (1 cycle / 0.67 seconds = 1.5 cycles per second) tells us that this is a single 1.5-Hz delta wave. Note that the wave's frequency can be determined even though it stands alone and does not repeat, and also that the shape of this slow wave can be seen despite the superimposed fast activity. If a wave of this size did repeat, it can be visually estimated that 1.5 such waves would fit into 1 second.

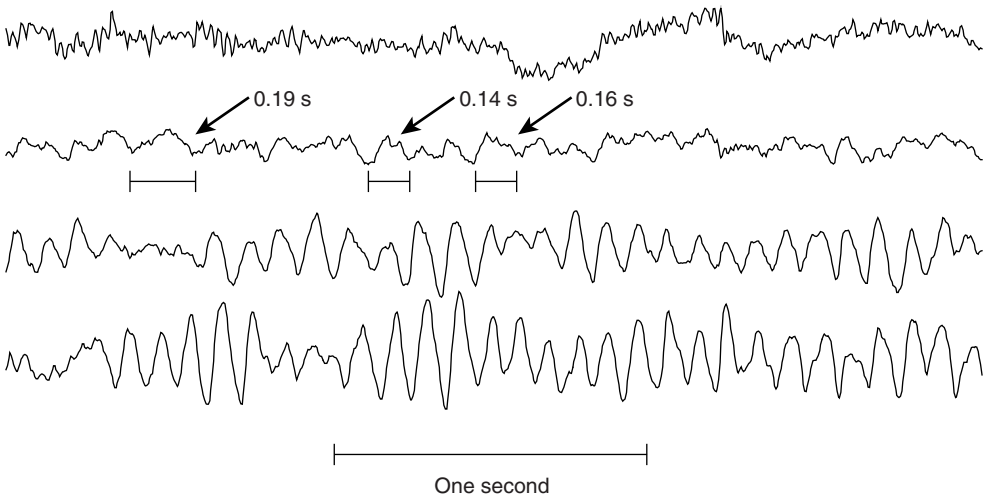

Figure 3-3 The frequency of irregular theta waves can be assessed using the same technique of measuring wavelength that was used for the previous figure. In the second channel of this figure, several semirhythmic theta waves can be seen with the durations shown in the figure. Although the waves indicated by the arrows do suggest some rhythmicity, each varies to some extent in wavelength and cadence. By calculating their reciprocals, the measured wavelengths of 0.19, 0.14, and 0.16 seconds correspond to frequencies of 5.3, 7.1, and 6.3 Hz, respectively, all of which are in the theta range (4–8 Hz).

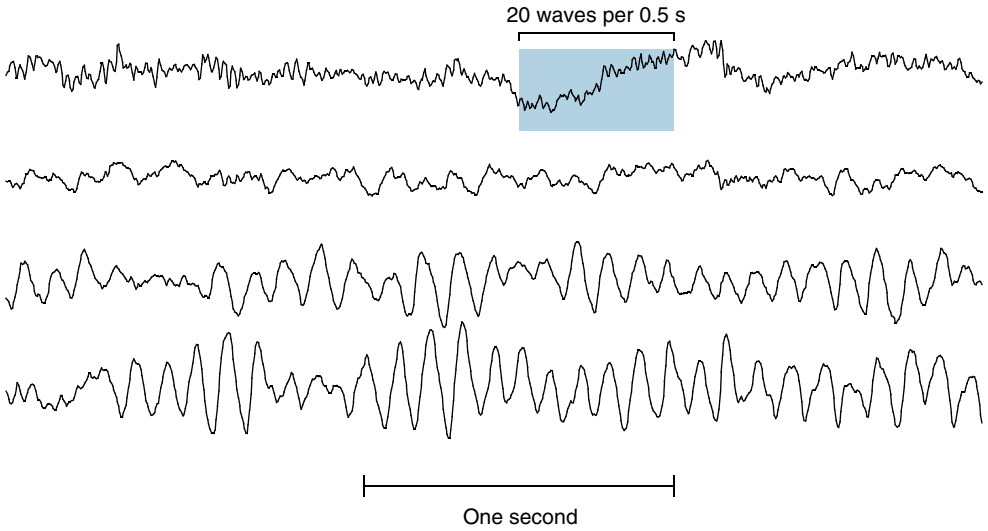

Figure 3-4 The fast activity seen in the shaded area of the first trace is too fast to make an accurate measurement of wavelength and would be cumbersome to count for a full second. Here, in the half-second interval denoted by the shaded rectangle, 20 waves are counted, implying a frequency of 40 Hz. Given the high frequency of these waves and the fact that they were recorded over the frontalis muscle, these may represent an example of electrical muscle artifact.

This theorem states, in simplified terms, that any waveform, no matter how simple or complex, can be exactly described by the sum of a sufficient number of simple sine waves. Although a comprehensive discussion of the Fourier theorem is beyond the scope of this text and not necessary to EEG interpretation, it reminds us that even the most complex EEG waves may be thought of as the sum of some number of fundamental sine waves. For example, the apparent complex wave shown in Figure 3-5 actually represents the combination of a 1-Hz wave, a 2.4-Hz wave, an 11.7-Hz wave, and a 34-Hz wave, each of a different amplitude. It is not necessarily the electroencephalographer's goal to identify *all* of the component parts of any particular EEG wave, but it is useful to be able to identify the fundamental wave and the main superimposed waves of an EEG signal. Figures 3-6 through 3-9 illustrate the component waves that make up the complex wave of this example.

For those interested in the basic mathematics that describe these waves, a review of the formula for a simple sine wave function helps us to recall some of the features of a wave that can be described thus:

$$f(x) = A \sin (bx + \phi).$$

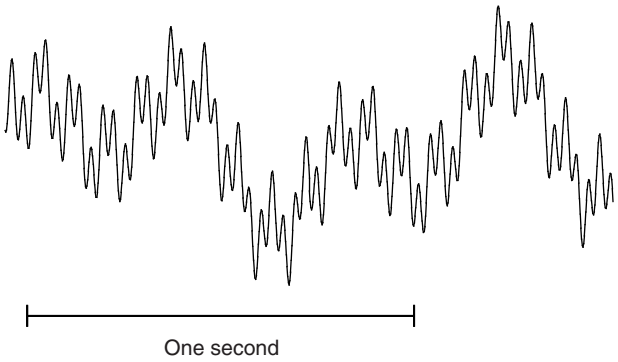

Figure 3-5 The complex waveform shown in this figure represents the summation of four separate sine waves with frequencies of 1 Hz, 2.4 Hz, 11.7 Hz, and 34 Hz wave, each of a different amplitude. The four individual contributing components of the wave are shown in the following figures. A 1-second scale is shown.

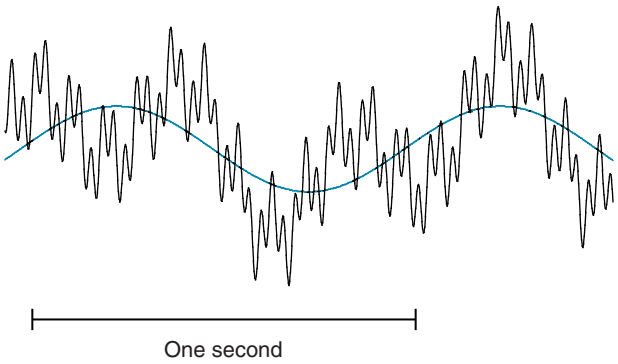

Figure 3-6 The original 1-Hz component of the mixed wave is superimposed on it. Some of the gradual "up and down" shape of the complex wave can be seen to be explained by this underlying 1-Hz slow-wave component, but the contribution of this slow component is difficult to visualize.

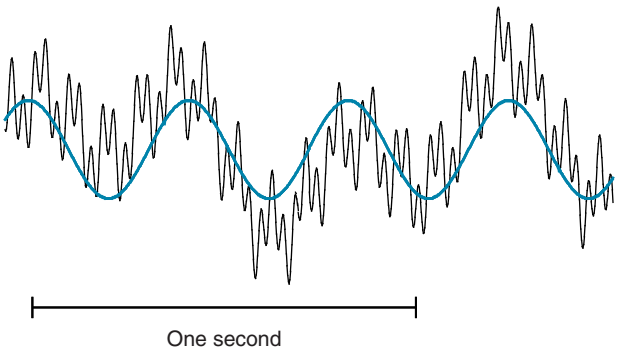

Figure 3-7 The same wave as Figure 3-6 with the 2.4-Hz component isolated and superimposed. Of all the component waves (with the possible exception of the fastest 34-Hz wave), this 2.4-Hz component frequency can be most easily identified in the mixed wave. Along with the 1-Hz wave, the 2.4-Hz wave lends some of the "roller-coaster" shape to the final wave.

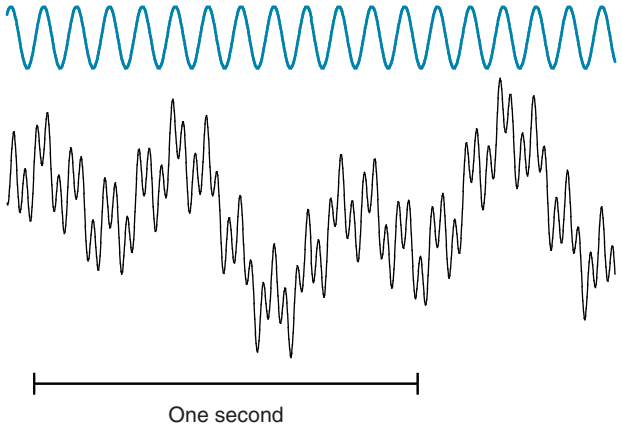

Figure 3-8 The 11.7-Hz wave component is shown above the mixed wave for the sake of clarity. This specific component is somewhat difficult for the eye to sort out from the mixture of frequencies in the complex wave, but it can be identified.

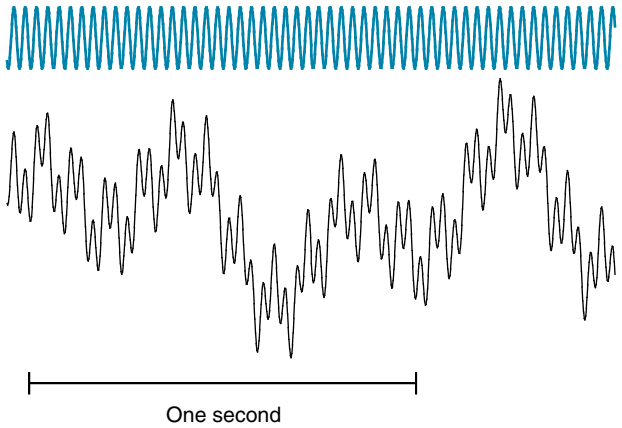

Figure 3-9 The 34-Hz component of the initial wave is probably the easiest to identify. It is seen to "ride" atop the lower frequency waves.

In this formula, varying the coefficient A higher and lower will change the height or amplitude of the resulting wave. Varying b will change the frequency of the wave. Finally, changing the value of ϕ will shift the wave to the left or right on the x axis, which is also referred to as shifting the phase of the wave. It is not necessary to know this formula to interpret EEGs, but it is useful to keep the formula and its coefficients in mind when learning to separate out and describe the component parts of wave mixtures.

Although addition of one sine wave to another can be described with mathematical formulas, it is more useful to become accustomed to the appearance of how sine waves add *visually* as opposed to mathematically.

Figure 3-10 shows the result of superimposing or adding a lower voltage 10-Hz wave onto a higher voltage 1-Hz wave. Note that the lower voltage fast activity appears to "ride" the hills and valleys of the slower wave. The key is that both individual waves can still be visually appreciated even though they are mixed together.

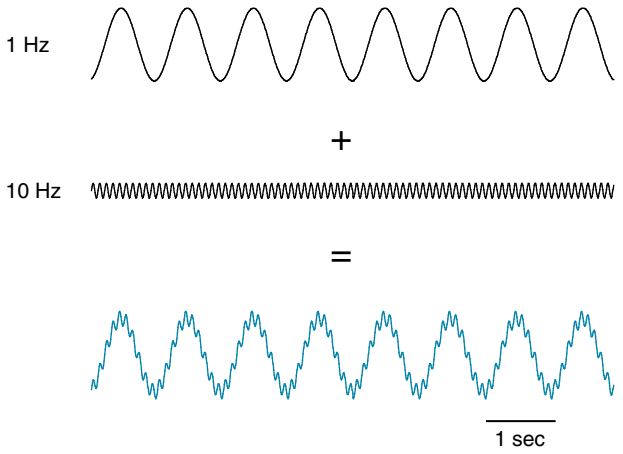

Figure 3-10 The result of the addition of a 1-Hz wave to a lower voltage 10-Hz wave is shown. The top trace shows a 1-Hz sine wave at an amplitude of 100 μV, and the middle trace shows a 10-Hz wave at 20 μV. When the 1-Hz and 10-Hz waves are added together, the bottom wave results, showing the 10-Hz wave "riding" on the fundamental 1-Hz frequency.

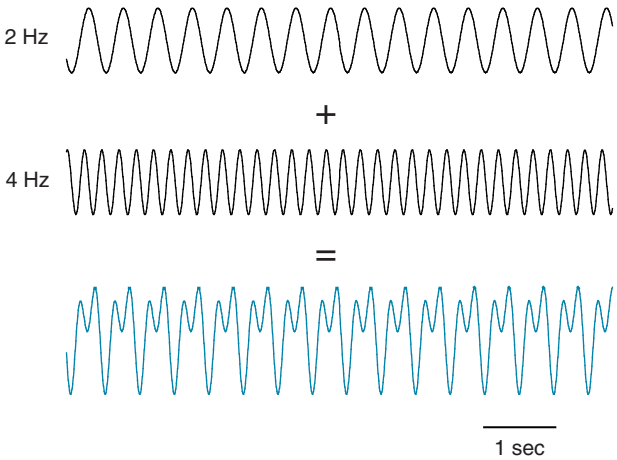

Figure 3-11 This figure illustrates the addition of a fundamental wave to its harmonic. The top trace shows the fundamental 2-Hz wave at 25 μV, and the middle trace shows a 4-Hz sine wave of the same amplitude. When a wave is added to another wave that is a multiple of the first wave's frequency (i.e., a *harmonic* frequency), the distinctive tracing seen in the bottom trace results. Note the regular "notching" pattern of the smaller wavelet riding on the larger wave. Because the two waves partially reinforce, the summation wave has an amplitude of 40 μV in this example. The degree to which the two waves reinforce and the particular shape that results is partly a function of how the two waves are shifted in the left–right axis with regard to one another (phase shift).

When two waves are mixed and one has a frequency that is a multiple of the other, the higher frequency multiple is called a *harmonic,* and the lower frequency wave may be called the *fundamental* frequency. (Occasionally, the lower frequency wave may be called the *subharmonic.*) For instance, if there is a 5-Hz fundamental frequency, it is not uncommon to see a superimposed 10-Hz harmonic. When a wave is mixed together with its harmonic, the resulting wave often has a particular appearance of regular notching.

Figure 3-11 shows an example of adding a 2-Hz wave to its 4-Hz harmonic. Note how the shoulder of the slower wave is regularly notched by the faster wave in this idealized example. The notching may appear on the upslope, downslope, peak, or trough of the slower wave, depending on how the phase of one wave is lined up with the phase of the other when the two are added together. This pattern is important to recognize because the notching patterns created by these harmonics can occasionally be mistaken for spike-wave discharges.

Figure 3-12 shows how the appearance of the superimposition of the same 2-Hz and 4-Hz waveforms can differ depending on how the phase of one is shifted to the right or left in comparison to the other.

Figure 3-13 shows the result of adding a fundamental 2-Hz wave (top trace) to a 7-Hz wave of slightly varying amplitude (middle trace). In reality, the important skill is not necessarily to be able to predict what the result will be of adding two particular waves together but, rather, the reverse procedure: to be able to look at the bottom trace in Figure 3-13 and to be able to visually reconstruct the distinct 2-Hz and 7-Hz rhythms contained within the more complex waveform. Although the examples shown in these figures are of idealized sine waves, the same visual skills apply to the "deconstruction" of actual EEG waveforms. Of course, the idealized waves seen in these examples do not occur in such pure forms in complex biological systems such as the brain, which are more complex and subject to more variation.

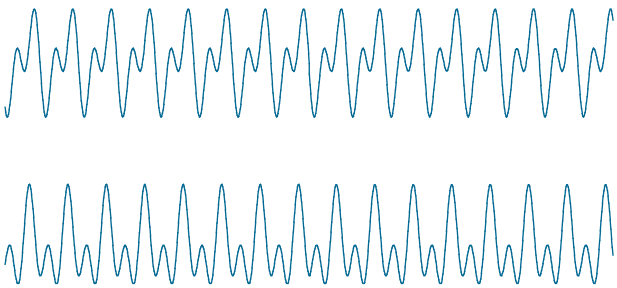

Figure 3-12 The two waves shown here represent the summation of the exact same two component waves (top and middle waves) from the previous figure, except that the phase of the second wave is shifted to the left or right a slightly different amount compared with the first wave before the two are added together. Such combinations of fundamental waves with their harmonics do occur in cortical circuits and should not be mistaken for "spike wave."

Figures 3-14 and 3-15 show examples of other ways that pure sine waves can vary, such as in frequency and amplitude, and represent somewhat more realistic approximations of real-life EEG waves.

Figure 3-16 shows a close-up of a recorded EEG wave that contains mixed frequencies.

LOCATION

The location of an EEG event is usually best described in terms of the electrode(s) it involves. The proper placement of the EEG electrodes is the job of the EEG technologist who produces the recordings. The schema used for electrode naming and placement is called the International 10-20 system, formalized by Jasper in

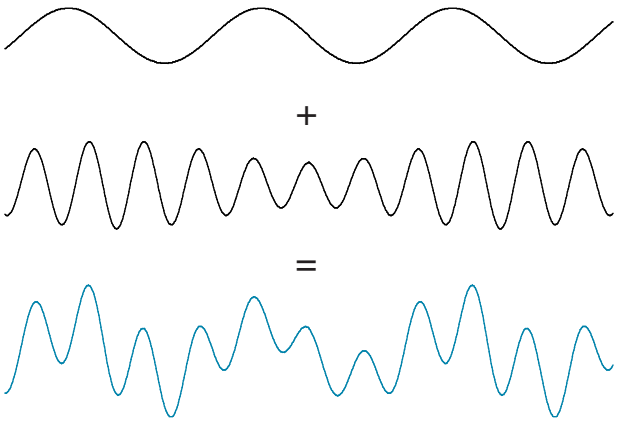

Figure 3-13 The top trace shows a 2-Hz wave of constant amplitude, and the middle trace shows a 7-Hz wave of slightly varying amplitude. The bottom wave shows the result of adding the top wave to the middle wave. In this example, the 7-Hz activity is most readily appreciated in the combined wave, whereas the 2-Hz component is less obvious, although it makes a definite contribution to the final wave's shape.

1958. Two 10-20 electrode systems are in use today, the system using the original electrode nomenclature and a modified 10-20 system in which some of the electrode positions have been renamed. Luckily, the great majority of the electrodes have the same names in both systems. Unfortunately, to date, neither system has established primacy over the other, and use of both systems is still widespread. At the risk of some confusion, examples of both systems are used in this text so that readers will gain some familiarity with each. Some feel the newer system is more logical, whereas others do not feel it offers enough advantages to overturn decades of tradition in nomenclature. Because no single electrode name is used to indicate different positions in the two systems, the two conventions can coexist unambiguously side by side. Although it may be tempting for new electroencephalographers to memorize only the newer system because it may seem to represent the "wave of the future," it is probably best first to memorize the system in use in one's own laboratory but to have at least a passing acquaintance with the other if called on to read records or reports that come from other laboratories.

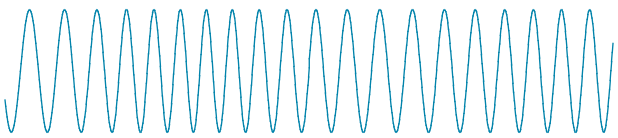

Figure 3-14 The sine wave shown has a constant amplitude (height) but a 15% variation in frequency. Note that the width of each wave increases and decreases perceptibly. Such slight variations in frequency are common in physiologic EEG waves.

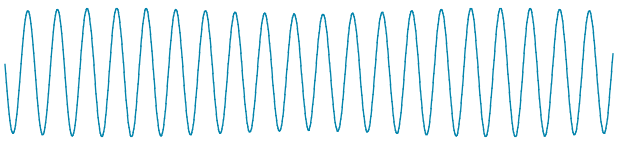

Figure 3-15 This sine wave has the same average frequency as the previous wave, but in this example, the frequency is held constant, and there is a 10% variation in the wave's amplitude.

Figure 3-16 An EEG signal acquired from a patient shows a complex, "real-world" mixture of delta, theta, and faster rhythms. Although some sinusoidal elements can be appreciated, many of the waveforms are irregular.

The term *10-20* is based on the general strategy of measuring the distance between two fixed anatomical points, such as the nasion (the point where the bridge of the nose meets the forehead) and the inion (the prominent point on the occiput), and then placing electrodes at 10% or 20% intervals along that line. This 10-20 system represented an improvement over previous electrode placement systems, some of which relied on absolute measurements (instead of percentages) but failed miserably when heads of different sizes were studied—imagine the effect of using the same system of absolute measurements on the heads of adults or patients with hydrocephalus and the smaller heads of newborns or even premature infants. The general plan for the placement of the 21 primary electrodes of the original 10-20 system is shown in Figure 3-17. The electrode nomenclature for the modified 10-20 system are shown in Figure 3-18. The four electrode positions for which names have changed are shown shaded.

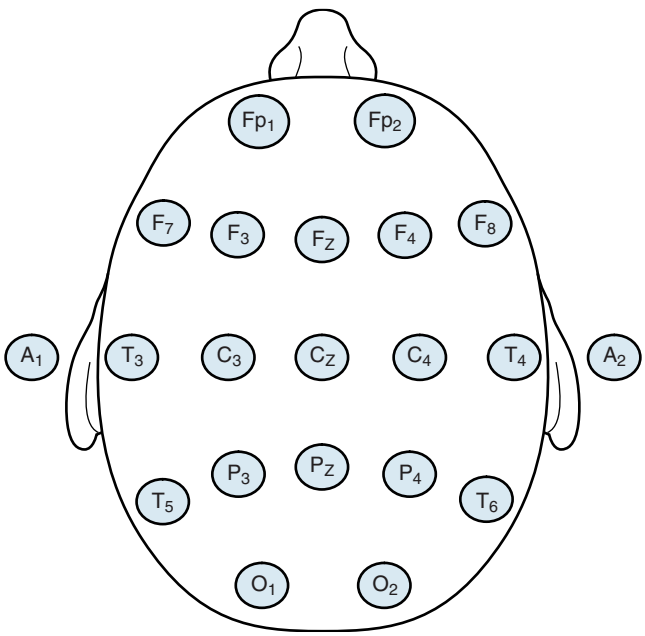

Figure 3-17 The electrode nomenclature for the original 10-20 electrode system is shown. This original naming system is still in use in many EEG laboratories.

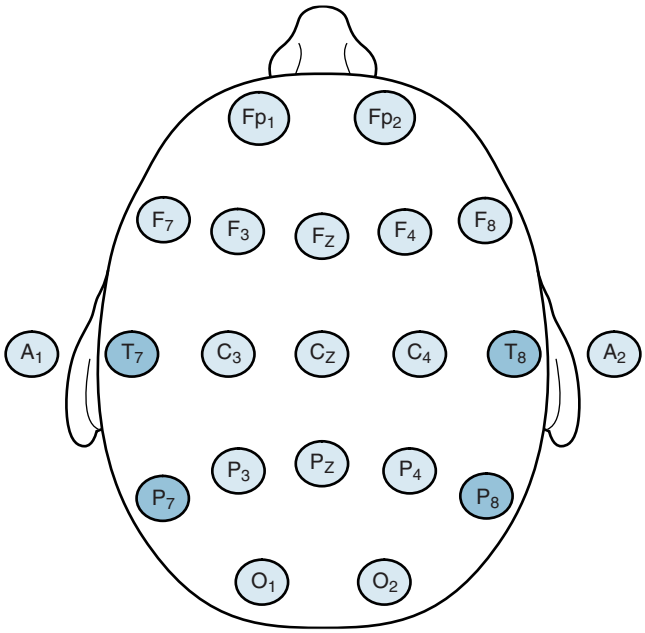

Figure 3-18 Primary electrode names for the newer "modified" 10-20 electrode system are shown. Note that the majority of electrode names are unchanged; the shaded electrodes represent the name modifications proposed by this new system. In addition to those electrodes shown, this system also establishes names for electrodes in intermediate positions that are not routinely used in standard EEG recordings (see Figure 3-21). This modified 10-20 system has not yet been universally adopted, however.

Comparison of the Original and Modified 10-20 Systems

The electrode nomenclature used in the original International 10-20 system has different strengths and weaknesses. Most of the electrodes are named using a letter to denote the area of the brain they overlie: *F* for frontal, *Fp* for frontopolar, *T* for temporal, *C* for central, *P* for parietal, and *O* for occipital. The earlobe electrode is denoted by an *A*. Electrodes over the left hemisphere are subscripted with odd numbers and those over the right hemisphere are subscripted with even numbers. Electrodes in the midline are denoted with a *z*-subscript, standing for "zero." The electrode nomenclature is generally based on longitudinal and transverse arrays (see Figures 3-19 and 3-20). Although electrode names may be written with subscript nomenclature such as F_7, many EEG instruments (as well as this text) use an unsubscripted format ("F7") to increase readability.

Certain idiosyncrasies common to both systems should be noted. Although the F7 and F8 electrodes are, indeed, placed over a portion of the frontal lobes as their names imply, these electrodes best reflect activity from the anterior temporal lobes, and, for this reason, activity recorded from F7 and F8 is often described as anterior temporal lobe activity, despite the fact that they carry an *F* in their names. The C3 and C4 electrodes are referred to as the central electrodes even though there is no central lobe of the brain. These electrodes derive their names from the region of the primary sensorimotor area, the precentral and postcentral gyri, and the central sulcus, which divides the two.

Certain features of the old system that were deemed undesirable have been addressed by the new system. In general, it was considered preferable that the electrodes running in each sagittal chain (the chains that run from the front of the head to the back of the head) use the same numeric designations when possible, although in the old 10-20 system the electrode names for the temporal sagittal chain (F7, T3, and T5) each have different numeric designation. Second, in the original system, some of the letter designations appear in more than one coronal plane (the electrode chains that run transversely, across the head), such as T3 and T5, which are in the central an parietal transverse chains, respectively.

The modified system of EEG electrode placement proposed by the American Electroencephalographic Society addressed these problems making only minimal modifications (see Figure 3-18). In the modified system, all of the electrodes in the sagittal temporal chains carry a label that includes the number *7* or *8* (e.g., F7, T7, and P7), thus conforming to the idea of using as few numbers as possible in each sagittal plane. Individual letter designations now appear in only one coronal plane (the rule that electrode names T3 and T5 broke in the original system). Finally, new, intermediate electrode positions are given standardized names—there are now 75 electrode positions, which include electrode placements lying in between the standard positions described by the original International 10-20 system. These include electrode names such as F_5 (for the position between F_3 and F_7), FC_3 for the position between F_3 and C_3, and so on. This more comprehensive system of electrode naming is also called the *10-10 System* or *10-10 terminology* (see Figure 3-21). These additional electrodes are not typically used in routine recordings but may be used when special circumstances require them. Because this system modification required the renaming of four of the twenty-one standard electrode positions, many laboratories felt the introduction of the new system would be confusing and have not elected to adopt the modifications, preferring to remain with traditional system instead (Table 3-2).

Focal, Lateralized, Multifocal, and Generalized

These localization terms are essentially self-explanatory. A *focal* discharge or wave is confined to a single brain area, usually to a tightly clustered group of electrodes but occasionally to a single electrode. A *lateralized* event is localized to a single hemisphere, and most or all of the hemisphere is involved with the activity in question. A *multifocal* phenomenon involves three or more brain areas; for example, a patient with spikes in Fp1, O2, and T7 can be said to have multifocal spikes. A *generalized*

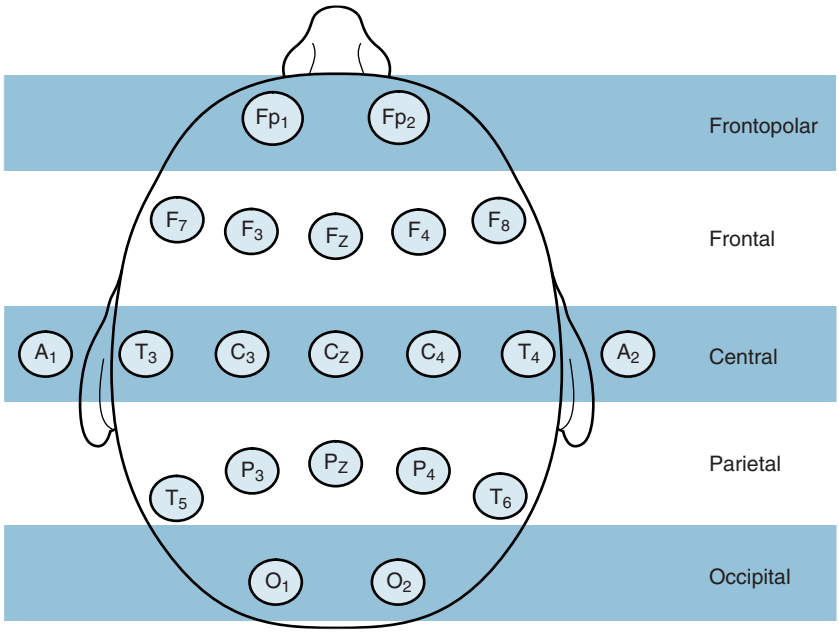

Figure 3-19 The international 10-20 systems group electrodes into five primary transverse planes as shown. One of the goals of the modified 10-20 system was to increase consistency of electrode names in these planes by allowing all of the positions in the parietal plane to begin with the letter 'P' (see Figure 3-19).

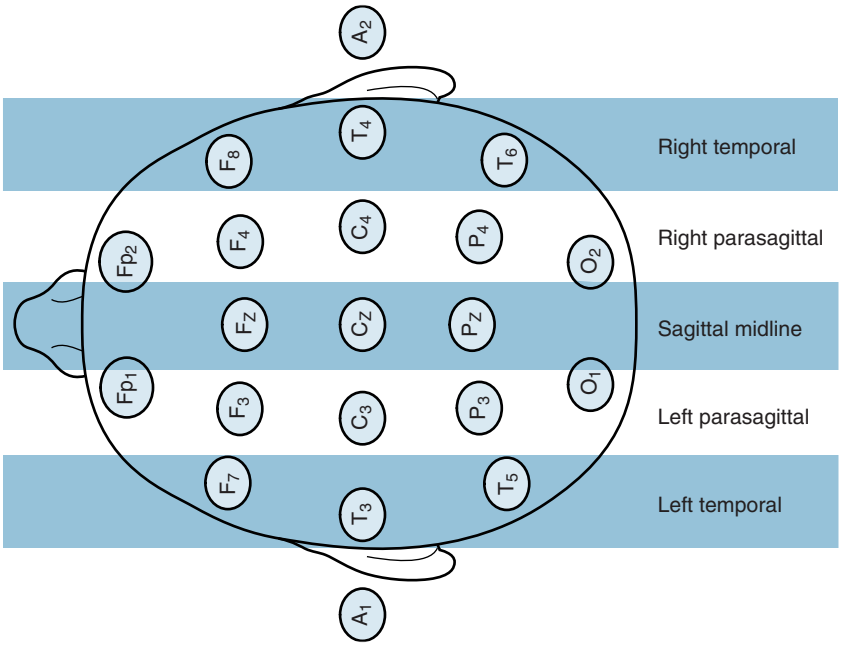

Figure 3-20 The numbering systems for the international 10-20 systems is based on five primary sagittal planes as shown. One goal of the modified 10-20 system was to increase the consistency of the electrode names' number subscripts, allowing the names of all the electrodes in the lateral (temporal) chains to end in 7 or 8 (see Figure 3-18).

discharge involves all scalp areas or almost all scalp areas at the same time, as in the example of generalized spike-wave discharges.

VOLTAGE

Because the EEG waves recorded in any given channel essentially consist of continuous voltage readouts between two given points, the voltage of an EEG wave is simply reflected by its amplitude or height on the page. Indeed, measuring the voltage of a wave is as simple as measuring its height, as long as the extent to which it has been amplified is factored in. Therefore, even though a wave may appear to be large on the tracing, it is not necessarily a high-voltage wave; it may simply be that the amplifier gain has been turned up high. Likewise, waves that appear small on the EEG page may actually be of much higher voltage than the visual impression they make if the amplifier gain has been turned down. In fact, without knowing how the amplifier gain has been set, it is impossible to know a wave's true voltage, whether or not it looks big or small on the page.

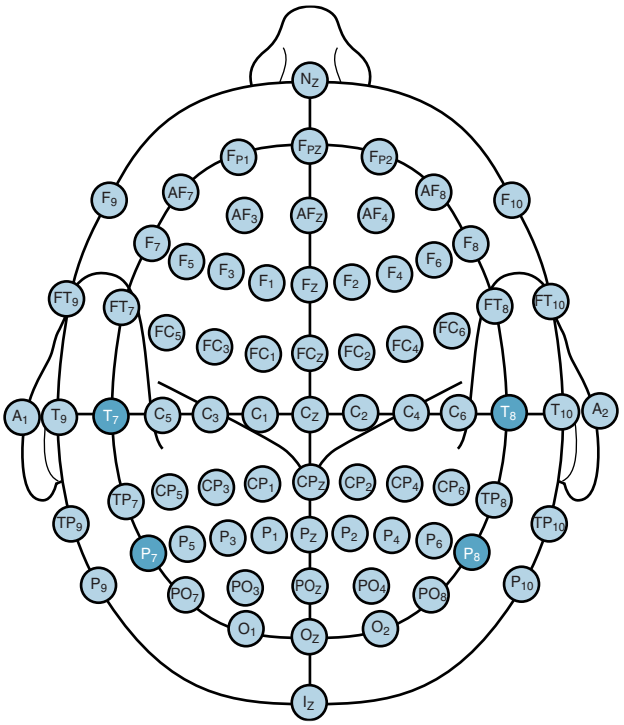

Figure 3-21 The full 10-10 combinatorial system is shown. Note that intermediate electrode positions are given official names and measured positions. Only the electrode positions shown in black have had their names changed in this modified version of the basic 10-20 system. *(From American Clinical Neurophysiology Society Guideline 5: Guidelines for Standard Electrode Position Nomenclature.* J Clin Neurophysiol *2:107–110, 2006.)*

Table 3-2 Frequently Used Names for Each Electrode Position

Electrode	Common Name
Fp1 and Fp2	frontopolar or frontal polar
F7 and F8	anterior temporal
F3 and F4	superior frontal
Fz	frontal midline
T7 and T8 (T3 and T4)	midtemporal
C3 and C4	central
Cz	vertex or central midline
P7 and P8 (T5 and T6)	posterior temporal
P3 and P4	parietal
Pz	parietal midline
O1 and O2	occipital

While there is some variation in the common English names given to the different electrode positions, the table above gives names that are most frequently used for each electrode position. The original 10-20 nomenclature appears in parentheses.

Gain Versus Sensitivity

The job of the amplifier is to create readable pen deflections from what are, in absolute terms, very low voltage electrical events on the scalp (measured on the microvolt scale). One reasonable way to describe the strength (or gain) of an EEG amplifier would be to quote how many millimeters it can make an EEG pen deflect for each microvolt of input it receives from the scalp. Amplifier gain can, therefore, be measured in millimeters per microvolt (mm/μV)—the stronger the amplifier, the more millimeters it can make the pen deflect per microvolt of input. Looked at in this way, as the amplifier gain increases, the resulting wave looks bigger and bigger on the page, which makes good intuitive sense. As it happens, however, EEG amplifier settings are not usually described in gain, but in *sensitivity*.

Amplifier Sensitivity

When the question "how *sensitive* is this amplifier?" is asked, this is similar to asking "how many microvolts of input are necessary to make this amplifier deflect a standard of one millimeter?" If the amplifier is "strong," then only a few microvolts would be required to generate the 1-mm deflection. If the amplifier is weak, it takes a large input in microvolts to generate the 1-mm deflection. It follows that the unit of sensitivity is microvolts per millimeter (μV/mm)—this is the standard way in which amplifier strength is described in clinical electroencephalography. Because of the situation in which a stronger amplifier setting requires *fewer* microvolts to deflect a millimeter, the stronger the amplifier gain, the lower the number to describe the sensitivity. Given the same EEG scalp activity, the printout shows waves of greater heights with settings of 2 μV/mm and waves of much lower heights when the setting is 20 μV/mm (by a factor of 10). Because the unit of sensitivity (μV/mm) is the reciprocal of the unit of gain (mm/μV), this leads to a somewhat nonintuitive situation that can take some getting used to: as an amplifier's strength or gain goes up its sensitivity goes down:

$$gain = \frac{millimeters}{microvolt}\left(= \frac{mm}{\mu V}\right)$$

whereas

$$sensitivity = \frac{microvolts}{millimeter}\left(= \frac{\mu V}{mm}\right)$$

The units for amplifier gain are discussed here only for explanatory purposes. It is the sensitivity expression that is used in clinical electroencephalography. When a sensitivity of 5 μV/mm is used, it can be useful to understand this sensitivity with the mental construct: "It takes 5 μV of scalp EEG activity to make every millimeter of wave." If the sensitivity is then changed to 10 μV/mm, the thought would be: "Now it takes 10 μV of scalp activity to make a millimeter of wave—the amplifier setting is now weaker, and the displayed waves should look smaller."

Luckily, figuring the actual voltage of an EEG wave is easy after the sensitivity is known (and this is the figure that is always available to the reader). This is accomplished by simply measuring the height of the wave in millimeters and multiplying by the sensitivity. The product is the wave voltage. For instance, a wave that measures 5 mm in height when the amplifier

sensitivity is 7 μV/mm is a 35-μV wave. This follows mathematically because the millimeter unit cancels out in the expression:

$$5\ mm\ x\ \frac{7\ \mu V}{mm} = 35\ \mu V$$

Measurements of wave heights can be taken with either a millimeter ruler on a paper EEG page or with a digital calibration marker on digital instruments.

How high should the amplifiers be set at any given time? On paper EEG machines, the technologist makes this decision on the basis of the overall voltage of the patient's EEG. The technologist should also make the same decision when using digital EEG machines, but the amplifier sensitivity can be further adjusted after the recording has been completed at the time of interpretation. In general, the amplifier should be set so that EEG waves are large enough to appreciate, but not so large that they become distorted or overlap with the channel above or below. The amplifier sensitivities used can have striking and sometimes unexpected effects on the apparent shape of EEG waves. Figure 3-22 shows a typical EEG tracing during wakefulness recorded at a reasonable sensitivity. In Figure 3-23, the sensitivity is high, and some wave detail is lost. In Figure 3-24, the sensitivity is set too low, resulting in channels crossing each other, and the general impression that all of the waves are spiky in appearance. This same phenomenon is evident again in Figure 3-25 in which the same posterior rhythm segment is displayed at three sensitivities, giving three different appearances. A similar effect is noted in the examples of a Stage II sleep recording shown in Figures 3-26 through 3-28. The lesson of these figures is that the reader must

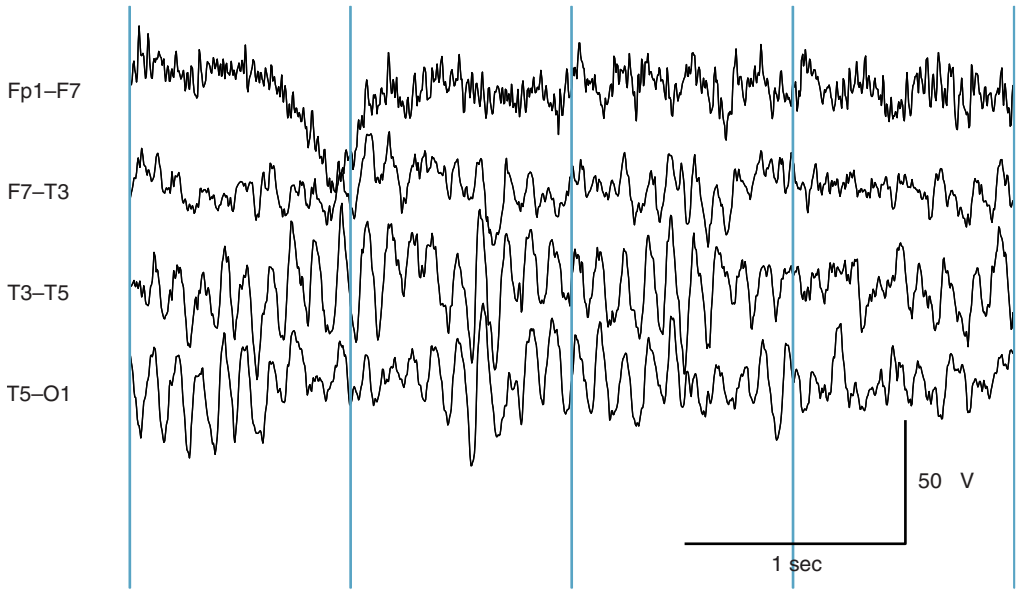

Figure 3-22 Several seconds of wakefulness are recorded. The posterior rhythm is best seen in the bottom two channels, T3–T5 and T5–O1. The amplifier sensitivity is set at 10 μV/mm. Wave morphology is easy to appreciate at these settings, although some of the channels do occasionally cross over into others.

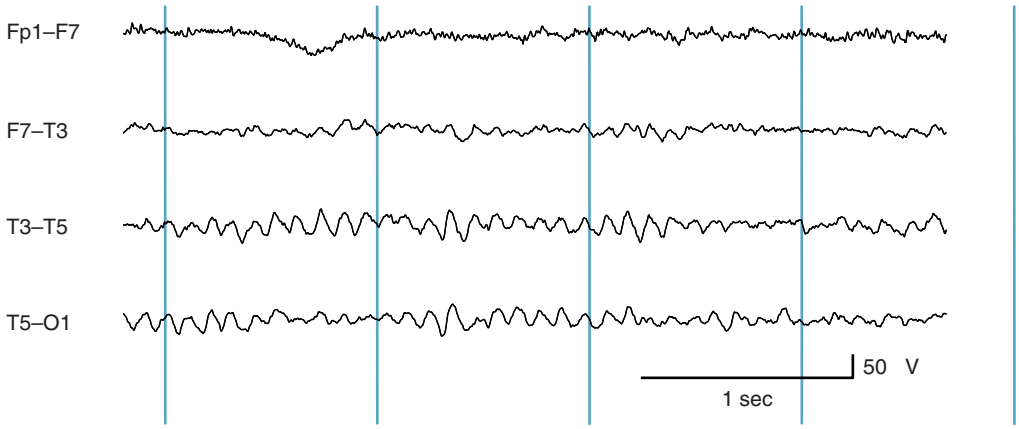

Figure 3-23 The same EEG segment is shown as in the previous example, but this time displayed at 50 μV/mm. The posterior rhythm is still easy to identify and count, but there is a dramatic loss of detail in the lower voltage, fast activity.

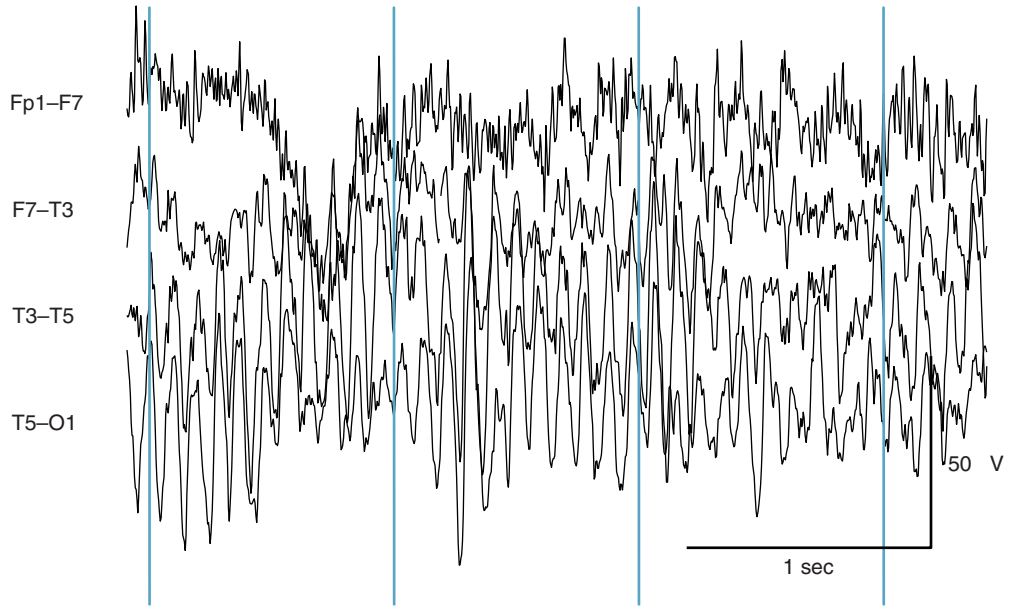

Figure 3-24 The same EEG segment shown in the previous two figures is displayed at a sensitivity of 5 μV/mm. Channels cross frequently making it difficult to discern some of the waves. The posterior rhythm takes on a spiky appearance (compare to the sinusoidal appearance of the posterior rhythm in the previous figure). If only the display of this EEG segment were considered, the reader might question whether the waves of the bottom two channels represent a succession of rapid spikes rather than the posterior rhythm.

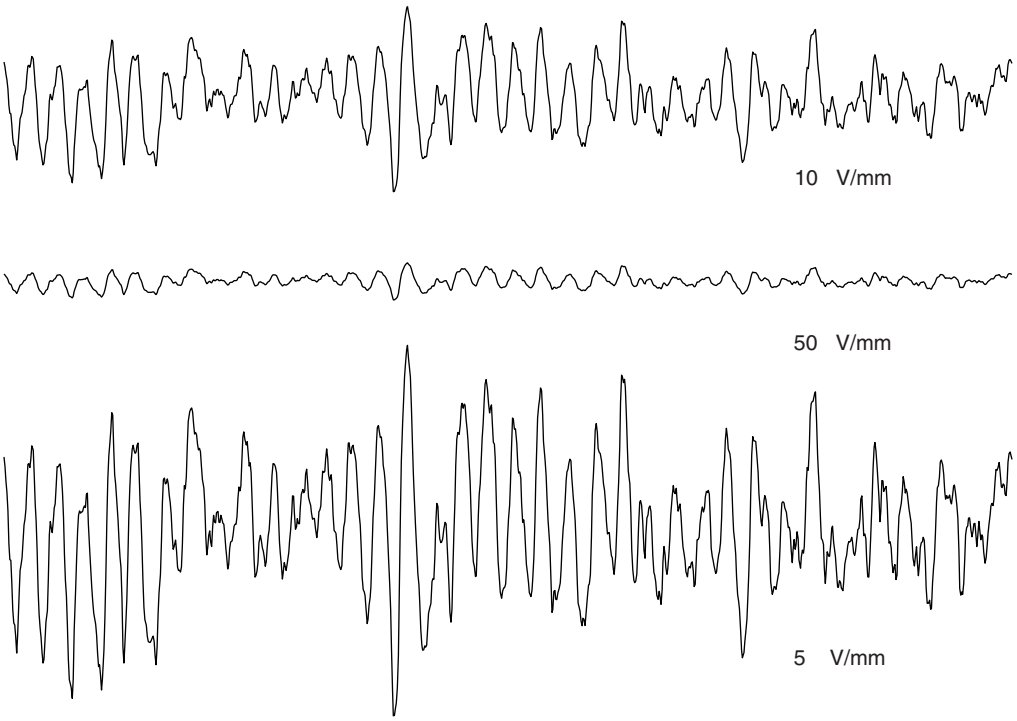

Figure 3-25 A highlight of the posterior rhythm from the previous examples is shown. Note that the peaks and troughs of the posterior rhythm become sharpened at high amplifier settings. Although there is little question that this is an "innocent" and typical sinusoidal posterior rhythm when displayed at 10 and 50 μV/mm, the appearance at 5 μV/mm might suggest the possibility of epileptiform activity to some. Keeping in mind that all three sweeps are representations of the exact same brain wave activity, the importance of the choice of display settings is clear.

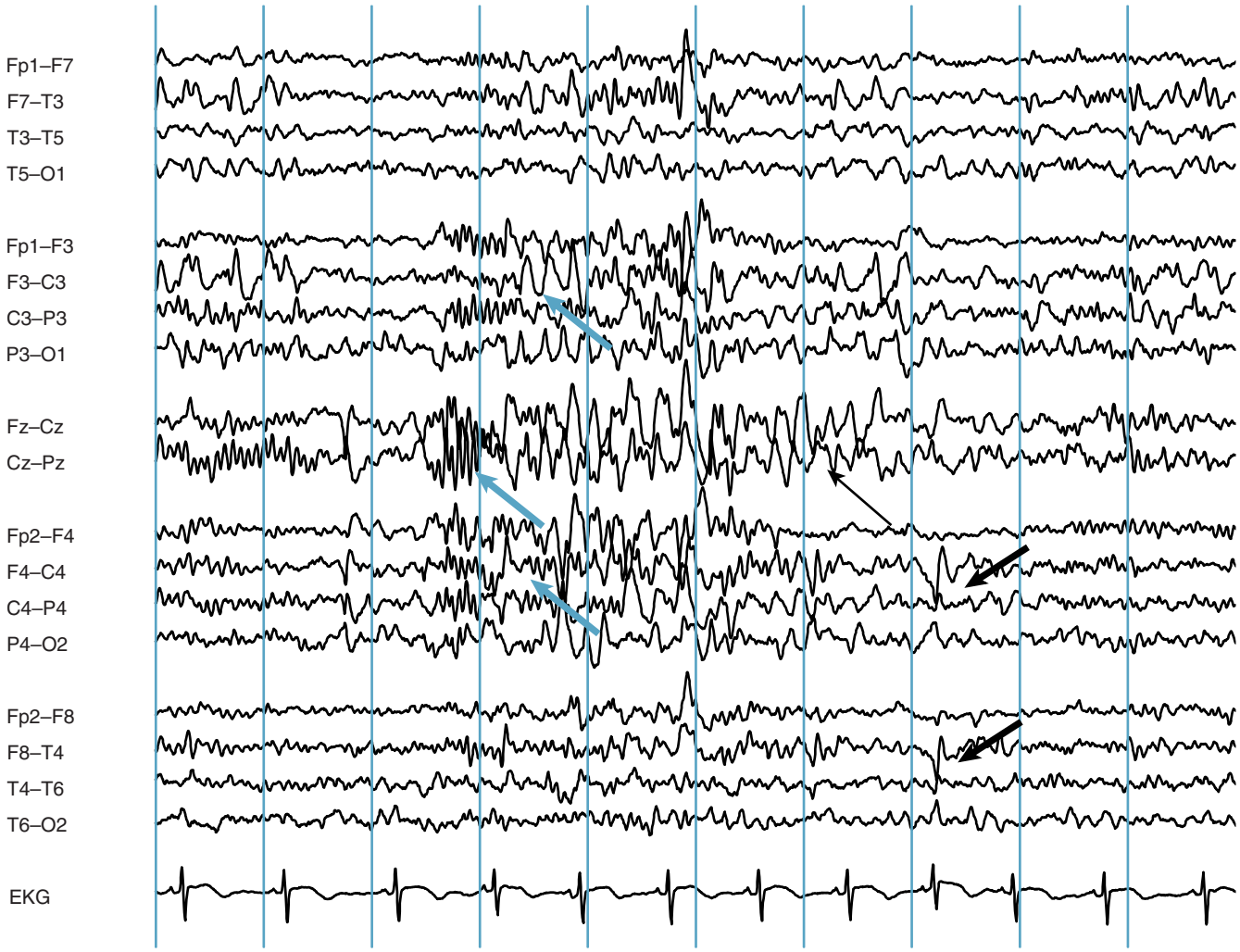

Figure 3-26 A 10-second segment of Stage II sleep is displayed at 20 μV/mm. Sleep spindles are seen at the vertex and in each central area (three blue arrows). A cascade of vertex waves of sleep is seen at the vertex (thin black arrow). Compare the appearance of these and other waves to the following two figures. This patient also happens to have right centrotemporal sharp waves (two heavy black arrows).

always be alert to the amplifier settings and whether those settings bias toward underemphasis or overemphasis of the sharpness of waves.

Synchrony and Desynchronization

At first glance, one would expect that higher voltage EEG waves imply more neuronal activity under the recording electrode and that flatter EEG waves imply less activity. This is not, however, always the case. EEG waves will only achieve a significant voltage when the neurons below the measuring electrode are acting in unison. Rather than measuring the activity of a single neuron, a scalp EEG electrode measures the summation of the activity of millions of neurons below its location on the scalp. When the neurons under a single electrode are manifesting the same type of charge shift at the same time (e.g., from negative to positive), the measuring electrode will "see" a net voltage change toward positive. However, if half of the neurons below the electrode make a net shift to positive at the same time as the other half of the neurons make a net shift to negative, the positive-shifting population may cancel out the negative-shifting population, and no net change will be "seen" by the scalp electrode.

For this reason, EEG waves are of highest voltage when the neurons in the measured area are acting in unison, or in *synchrony* with one another. In contrast, another group of neurons under another electrode may be shifting charge just as actively, but if each is behaving in a fashion different from that of its neighbor the summed activity will be cancelled out, and the measuring EEG electrode detects little net change in voltage when summing up the activity of all of the neurons in its recording area. This phenomenon of EEG flattening when neurons in an area do not shift charge in unison is called *desynchronization*. Figure 3-29 shows a schematic of synchronized neurons under a measuring

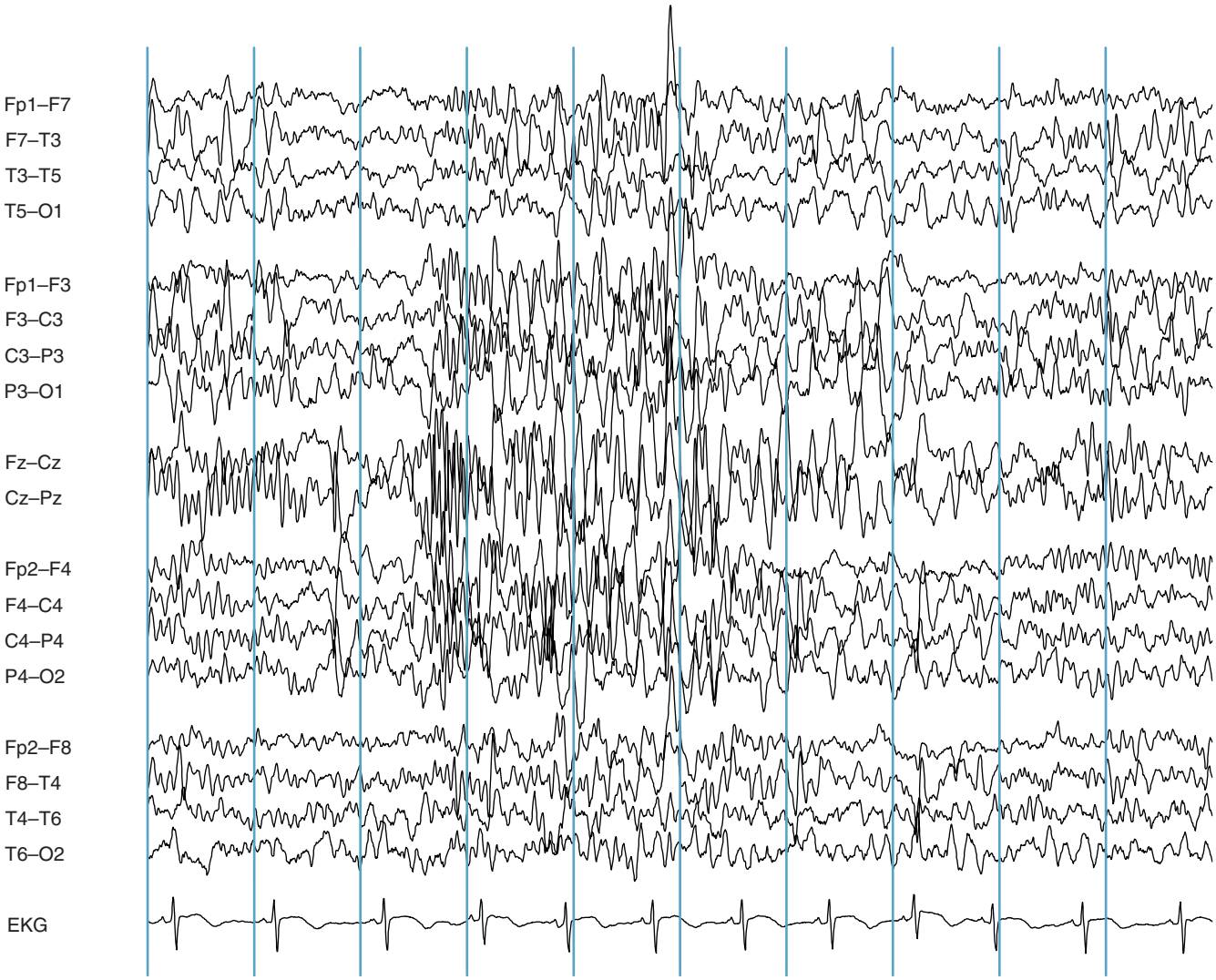

Figure 3-27 The same 10-second segment of Stage II sleep from the previous figure is displayed at 10 μV/mm. The sleep spindles, which are generally of low voltage, take on a misleading, dramatic appearance. The shapes of the vertex waves become difficult to discern, the spindles appear sharp, and the right centrotemporal sharp waves are harder to pick out from the surrounding rhythms, making a 10-μV/mm sensitivity for display of this segment a poor choice.

electrode producing an EEG wave. In Figure 3-30, the neurons are equally active in terms of changing charge but are not acting in unison. The result of this lack of synchrony is a flattening or "desynchronization" of the EEG wave. In electroencephalography, saying that a channel has become desynchronized is synonymous with saying that it has flattened.

For the reasons described earlier, it is important to keep in mind that the flattening of the EEG in a particular brain region does not necessarily indicate that that brain area is less active or "resting." Indeed, the opposite is often the case. Several types of EEG waveforms manifest lower voltage waves when the area is carrying out a more complex task but show larger waves when comparatively idle. The posterior rhythm is a good example of this phenomenon. The occipital region is primarily responsible for interpreting visual information. The posterior rhythm is a well-formed sinusoidal rhythm appearing in posterior areas when the subject is awake but with eyes closed. When the subject's eyes open and visual activities begin to take place in the occipital lobes, the posterior rhythm desynchronizes, or flattens. During visual processing, it can be imagined that each neuron gets a different task to carry out. When the occipital lobes are idle (eyes closed), it can be imagined that individual occipital neurons do not each have separate tasks and that they can be more easily "influenced by others," for example, by neural generators from deeper centers. Thus it is theorized that pacemakers from deeper centers can more easily recruit idle neurons to become synchronized to form a discernible wave. When the occipital lobes are occupied with visual processing, each occipital neuron gets a different task to do as part of its job of visual interpretation. The neurons shift charge in a nonsynchronous fashion, the activity of some groups cancels out the activity of other groups, and, when averaged by the recording electrode, the posterior rhythm

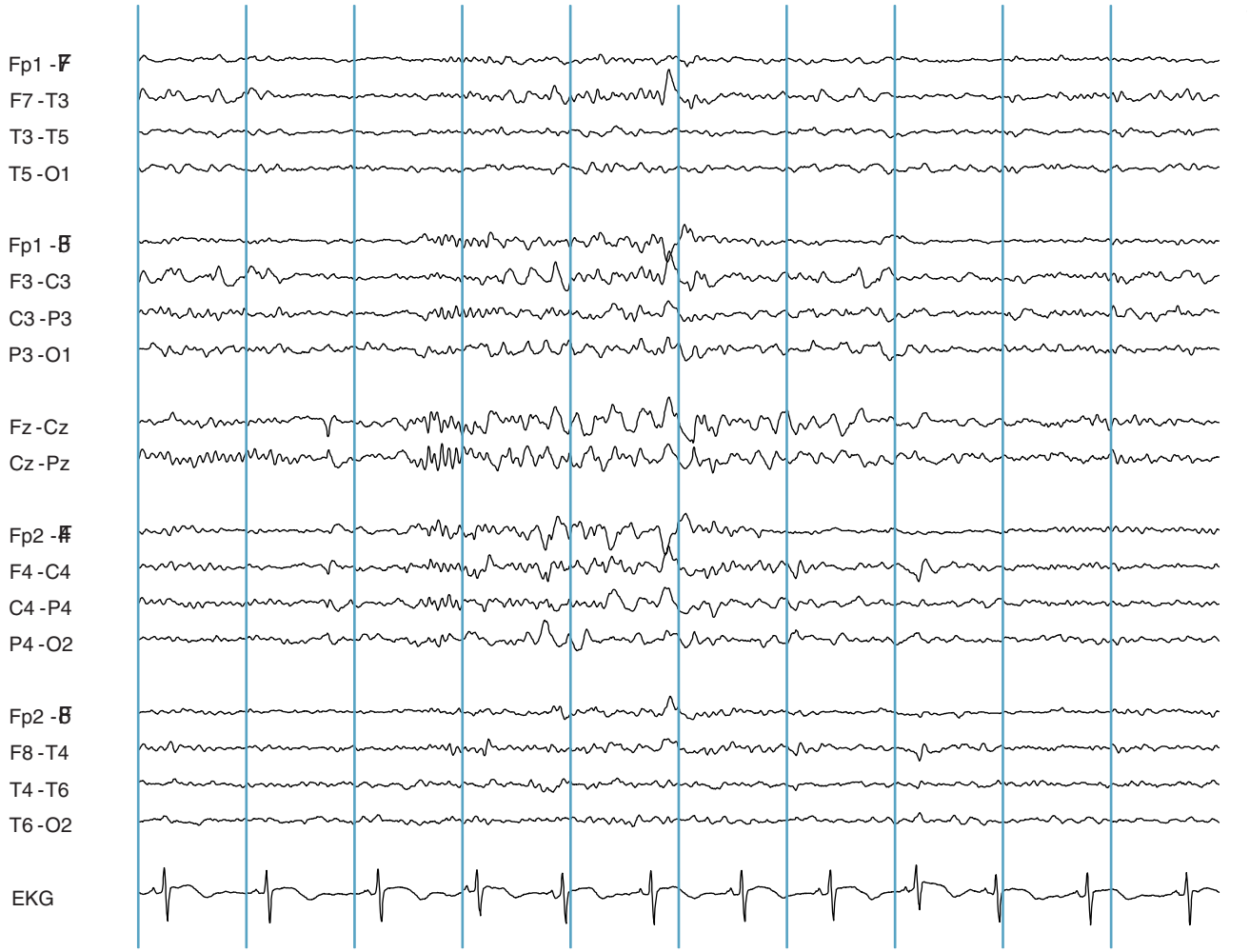

Figure 3-28 The same 10-second segment as shown in the previous two figures is displayed at 50 μV/mm. Although the vertex waves and spindles can still be appreciated, the detail of the fast activity is lost to a great extent, giving the tracing a "smoother" appearance. The right centrotemporal sharp waves have lost some of their sharp characteristic and could be missed with these display settings.

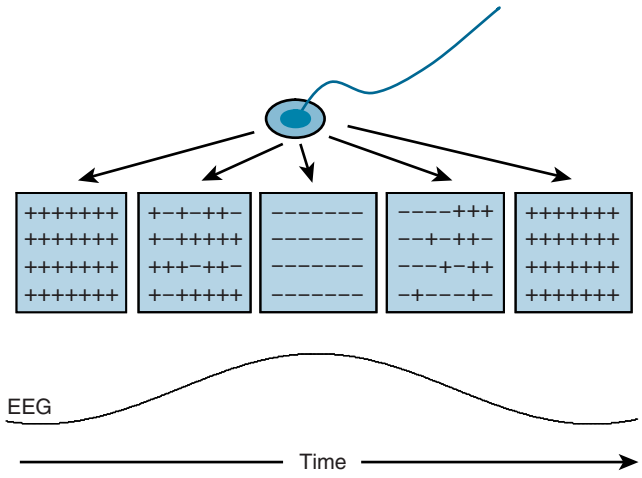

Figure 3-29 This schematic "imagines" a single electrode's view of 28 separate neurons whose polarities shift between positive and negative over time. At the beginning of the time period (first square), all of the neurons have positive polarity. By the middle of the time period, the successive squares show the population of neurons has shifted toward all negative (middle square), and by the end they have returned to all positive (final square). The recording electrode senses the "net" or "average" change in polarity of the population. Because on average, the neurons are changing in unison (in a *synchronized* fashion), the recording electrode registers a net change in charge and generates the EEG oscillation shown.

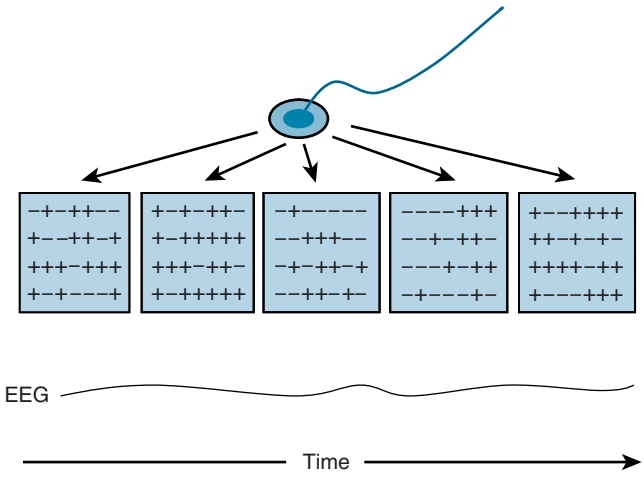

Figure 3-30 Although the *individual* neurons in this figure shift charge between positive and negative more often than in the previous figure, the changes are not occurring in synchrony. The "net" or "average" change in charge over the recording electrode is small between each square. Each neuron's behavior is not synchronized to that of its neighbor, and despite the fact there is more overall neuronal activity in this example, because the activity does not occur in unison a relatively flatter, *desynchronized* EEG trace results.

flattens. Similarly, in normal subjects, the EEG attains its highest voltage during sleep, in particular, during slow-wave sleep, a period of relatively decreased cognitive activity. Of course, EEG flatness does not always reflect the activities of a busy brain. In the examples of large strokes or deep coma, EEG flatness may also represent a simple absence of neuronal activity. The distinction between the two is usually easily made from the context in which the activity appears.

RHYTHMICITY AND CONTINUITY

In reality, most EEG waves recorded in clinical practice do not really resemble the perfect sine waves shown in some of the figures in this chapter, many of which would be more at home on the pages of a mathematics textbook than on the pages of an electroencephalogram. But in what ways do typical EEG waves tend to differ from idealized sine waves? We have already discussed variations in amplitude and frequency that can occur over time, in addition to the fact that many EEG waves consist of a mixture of different sine waves. Beyond voltage and frequency parameters, EEG waves may also vary according to their rhythmicity and continuity.

Rhythmicity

A wave's rhythmicity is a reflection of its regularity and, therefore, its predictability. If an EEG wave is highly rhythmic, it should be easy to predict when the next wave in a series will appear. A continuous sine wave is an example of an ideally rhythmic wave. Actual EEG waves never manifest this level of perfect rhythmicity, although some highly rhythmic waves come close, such as those shown in Figure 3-31. In some cases, a wave is less regular, but there is still some predictability to its pattern. Such a wave can be described as *semirhythmic* (see Figure 3-32). Finally, a wave may appear with no predictability of cadence at all, in which case it can be described as *irregular* as in Figure 3-33. A less commonly seen pattern is a *periodic* pattern. A periodic pattern consists of a wave or complex of waves that appears every 1 to 4 seconds. A periodic pattern following a seizure is shown in Figure 3-34.

Continuity

A waveform or complex may appear continuously or discontinuously (in an on-and-off pattern). In the case of slow waves, continuous slowing in an area may have a different clinical implication than intermittent (discontinuous) slowing. For instance, a patient may have a persistent theta wave in the left temporal area (continuous theta). Alternatively, a pattern may be seen in which single theta waves appear every now and then (intermittent theta). A schematic of a frankly discontinuous waveform is shown in Figure 3-35 in which periods of relative flatness interrupt the higher voltage activity.

In the case of neonatal EEG, the concept of continuity is of central importance. In the newborn, different stages of wakefulness and sleep are distinguished, in part, by the continuity or discontinuity of EEG background activity according to gestational age. In general, after the neonatal period, almost all normal EEG background activity is continuous, which is to say that start-and-stop or on-and-off background patterns are not expected. In contrast, in newborn EEGs, many normal background patterns manifest discontiunous patterns. Figure 3-36 shows a normal discontinuous pattern in a newborn at 35 weeks gestational age. Figure 3-37 shows the same baby's EEG pattern while awake showing continuous activity.

Synchrony Versus Independence

The term *synchrony* can be used to describe the timing of how electrical events occur in different brain areas relative to one another. (This use of the term is not related to the concepts of neuronal synchronization

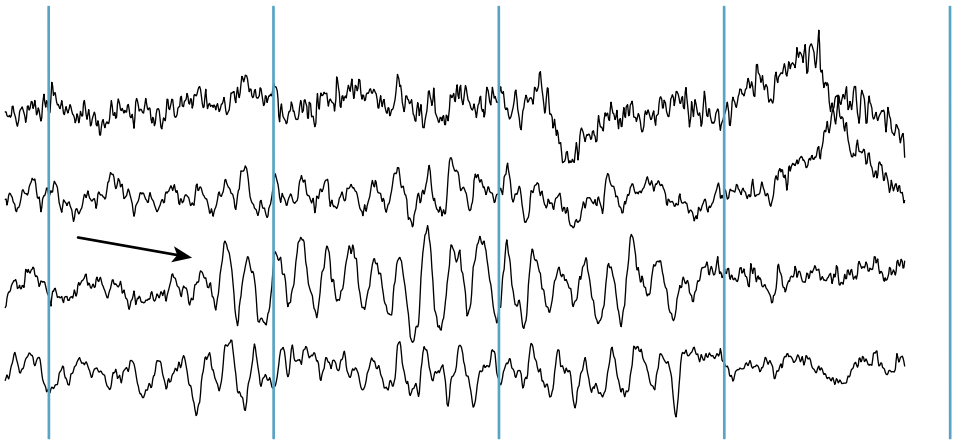

Figure 3-31 The waves in the third channel (arrow) represent an example of the posterior rhythm. Over the 2 seconds followed by the arrow, the peaks and troughs of the waves appear in a predictable fashion and can be described as highly rhythmic.

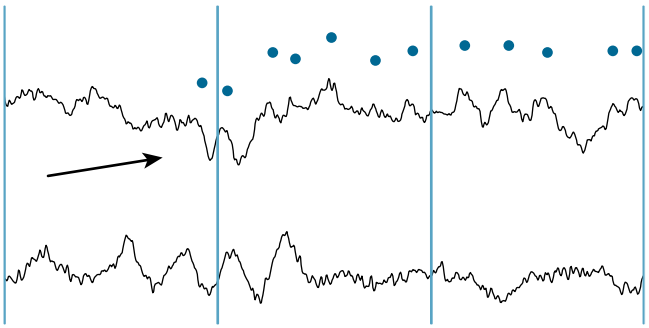

Figure 3-32 The theta waves shown in this example do not appear in a strictly rhythmic pattern, but there is still some predictability to the cadence of their appearance, which could be described as semirhythmic. The black dots are placed above the wave peaks to highlight the fact that the wave's rhythm is far from random.

Figure 3-33 This wave is a mixture of irregular frequencies. Looking at any particular portion of the wave, it would be difficult to predict what comes next.

and desynchronization discussed earlier.) For instance, if we are told that a young woman has sharp waves in both the left and the right central areas, this does not tell the whole story. Do these bilateral central sharp waves appear at the same time, *synchronously*, or do they occur at some times on one side and at other times on the other side, *independently*. When an event occurs at the same time on both sides, it can be said to be interhemispherically or bilaterally *synchronous*. The term *bisynchronous* can be used as shorthand for bilaterally synchronous to describe activity that occurs synchronously between the two hemispheres. The example of the newborn EEG shown in Figure 3-36 shows examples of both a bisynchronous burst and independent bursts.

PAPER SPEED

Only a short time ago, almost all EEG studies were directly recorded onto paper for review. Today the majority of laboratories have adopted digital EEG equipment. In digital EEG laboratories, almost all studies are viewed on a computer monitor and are rarely printed on paper. Even though paper is a thing of the past for many laboratories, the issue of "paper speed" is still an important consideration in EEG interpretation.

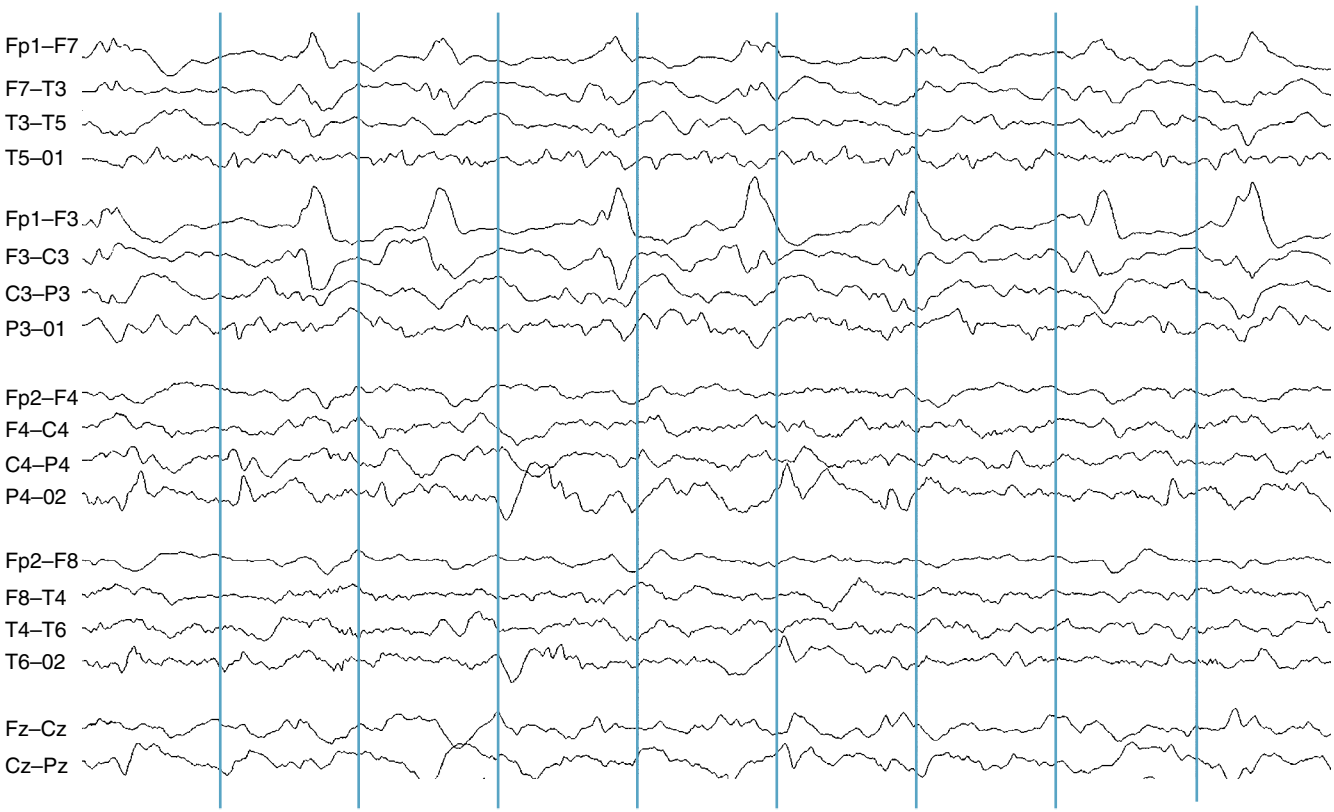

Figure 3-34 Wave complexes that appear only once every 1 to 4 seconds may be termed periodic. Here, a periodic waveform is seen in the left frontal area firing at at a rate of just less than one per second following a seizure.

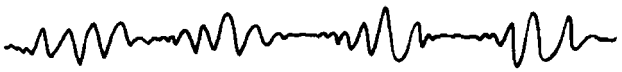

Figure 3-35 This schematic of a discontinuous waveform shows an on-and-off pattern of EEG wave activity separated by brief periods of relative quiet.

On paper systems, the display of an EEG wave could either be "pulled apart" or "squeezed together" like an accordion by varying the speed at which the paper was pulled under the writing pens. The correlate to paper speed on digital systems is the number of seconds displayed on each page (essentially, on each screen) at a time. Displaying many seconds on a screen compresses the appearance of the waves and is similar to a slow paper speed. Displaying only a few seconds on the monitor screen has the same impact as a fast paper speed. On many digital instruments, despite the complete absence of paper in the recording process, the term "paper speed" is still used and described in millimeters per second. Some instruments use the related units of seconds per page.

The paper speed chosen for the display (or seconds of EEG displayed per screen) actually has a large impact on the shape and appearance of EEG waves. The apparent sharpness of a wave is strongly affected by how the method of display "stretches out" or "squeezes together" a waveform. Slower paper speeds (i.e., higher numbers of seconds per page) can sharpen the appearance of any waveform considerably. Figures 3-38 and 3-39 show how wave shapes can dramatically change according to the paper speeds at which they are displayed. The same spike-wave complexes are shown displayed at three paper speeds in Figure 3-40.

The standard paper speed used for recording routine EEGs is 30 mm/sec. Note that this unit has lost exactness in the computer era because the number of horizontal millimeters that corresponds to 1 second is

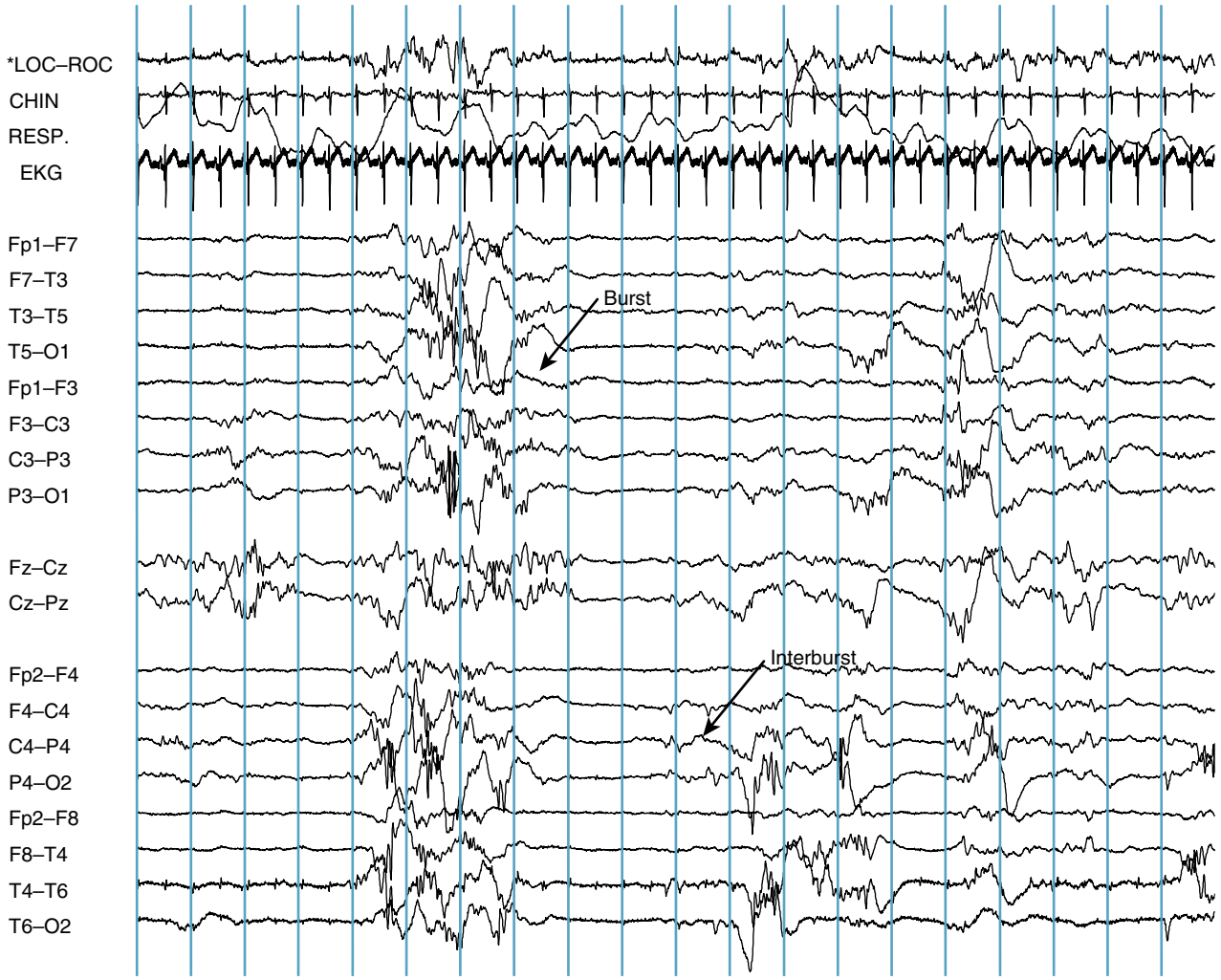

Figure 3-36 This pattern was recorded in a normal newborn of 35 weeks gestational age and is discontinuous. There are bursts of EEG activity lasting 3 to 4 seconds, surrounded by quieter periods (interbursts). Compare this discontinuous pattern to what is seen in the next figure, which was acquired in the same baby during wakefulness. Note that the periods of discontinuity in this figure differ somewhat between hemispheres. The first burst is essentially bisynchronous (appearing in both hemispheres at about the same time). The next burst occurs first over the right hemisphere and then over the left hemisphere (asynchronously or independently).

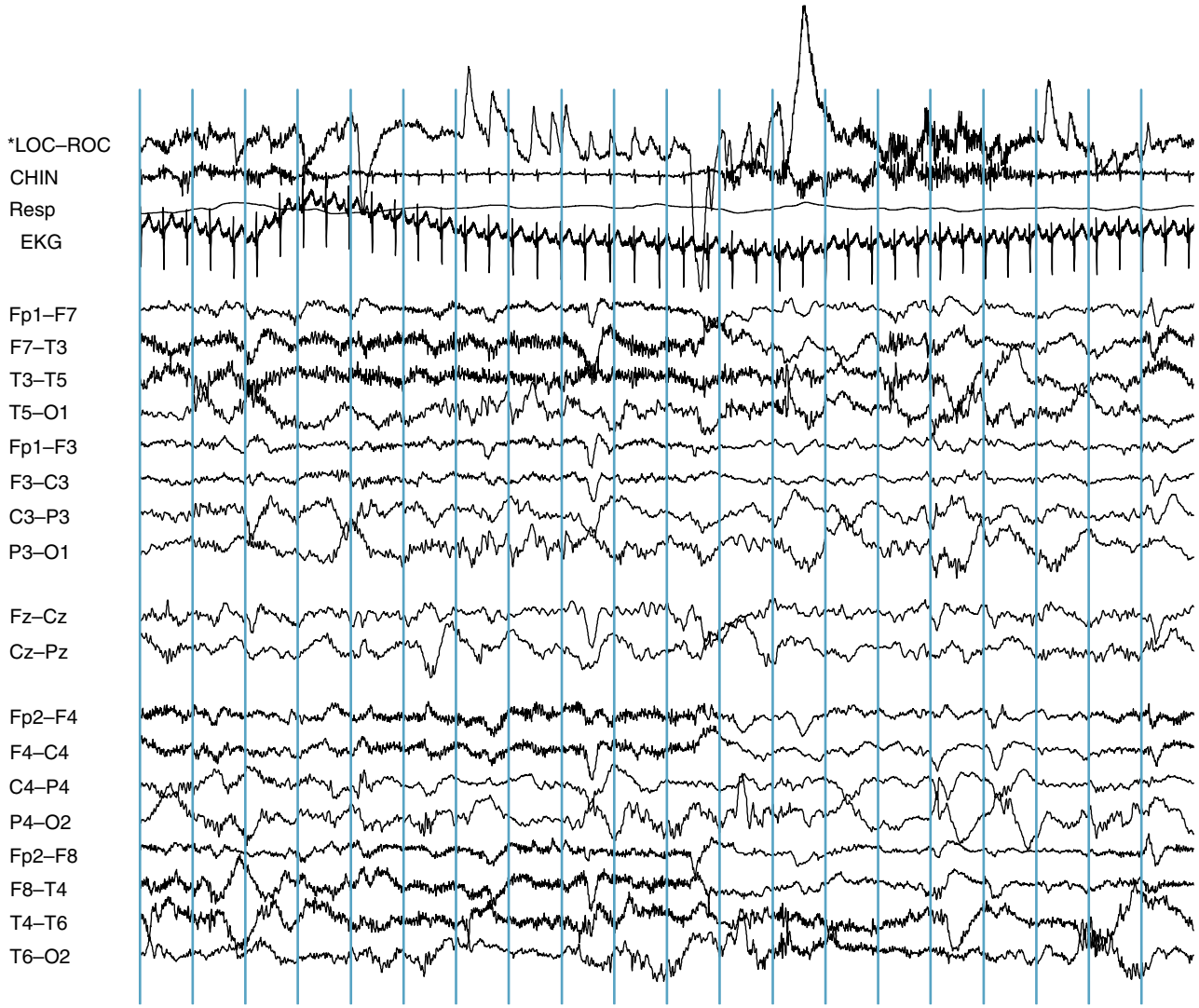

Figure 3-37 This recording was obtained in the same baby while awake. Note that the EEG activity is fairly irregular, but it is continuous. There is no sense of an "on-and-off" pattern as was seen in the previous figure. Some of the channels appear "dark" with muscle and motion artifact as a consequence of the baby's movements.

completely dependent on the size of the monitor used for the display; the software used to display the EEG is usually not aware of the dimensions of the monitor in use. Consider what happens to the meaning of the terms 30 mm/sec or 7 μV/mm when an EEG is displayed on a big screen in an auditorium. For this reason, on digital equipment it is best to make use of the major vertical divisions on the page (each of which equals 1 second) or the calibration bar when measuring wave durations despite the holdover of the millimeter unit for expressing paper speed or sensitivity.

Epileptiform Activity

The term *epileptiform activity* refers to EEG events or waves that stand out from the underlying rhythm and usually have a peaked or sharp appearance. It is best to think of the term epileptiform as meaning "in the shape (or form) of an epileptic discharge." The presence of epileptiform discharges does not guarantee a diagnosis of epilepsy. Rather, the presence of epileptiform activity suggests an *increased risk* of seizures from that area but does not definitely establish whether the patient has had or will ever have epileptic seizures. A careful distinction is made between ictal (seizure) and interictal (between seizures) activity. Strictly speaking, the term epileptiform activity should not be used for ictal activity (EEG seizure activity). Epileptiform activity refers specifically to interictal activity.

It is fortuitous that the EEGs of seizure patients manifest abnormal discharges between seizures; if it were only possible to make the diagnosis of seizures by having the luck of recording actual seizure activity in the EEG laboratory, then EEG would only rarely be a useful diagnostic tool in epilepsy. Except in the small minority of patients who have very frequent seizures, it

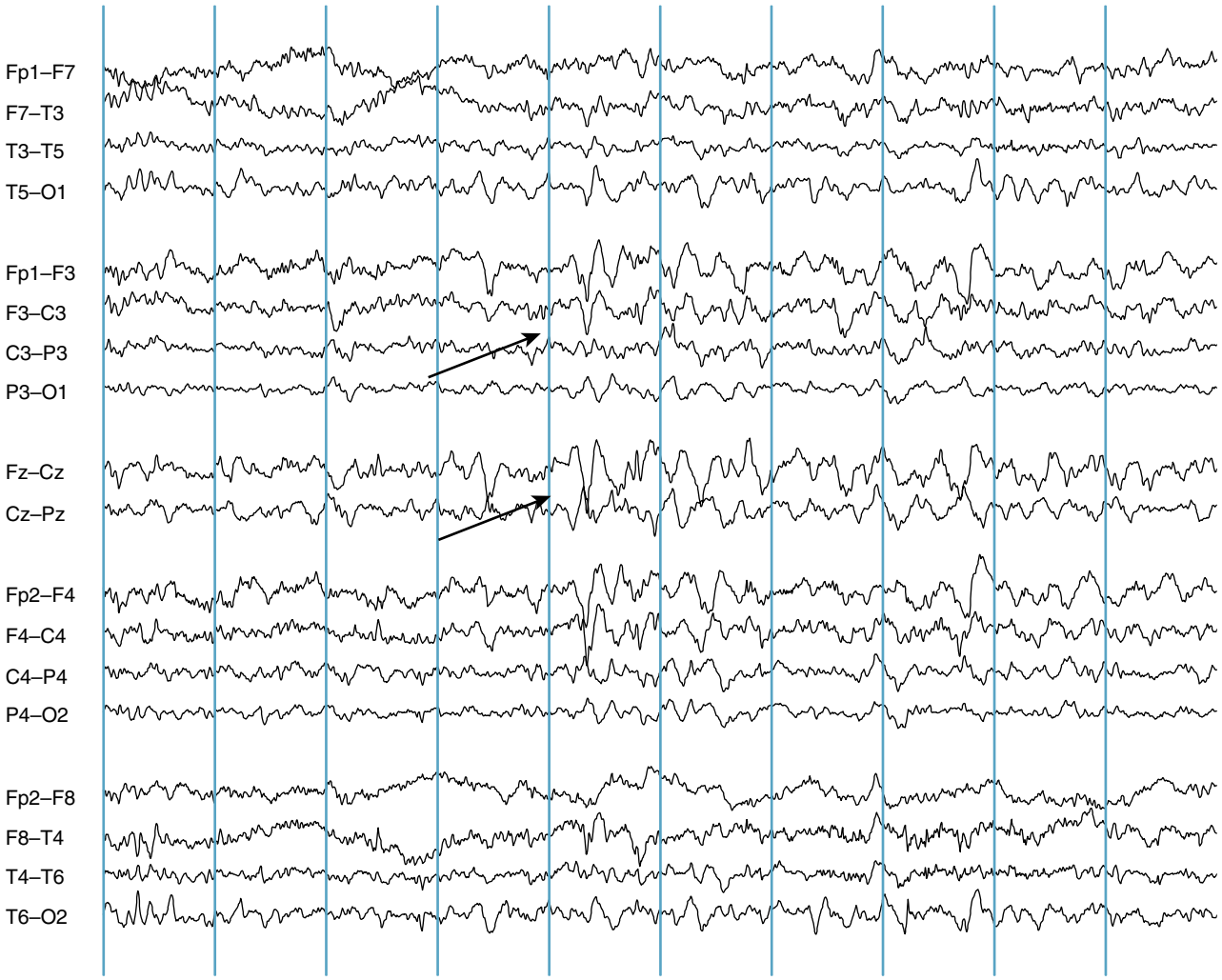

Figure 3-38 This tracing is displayed at 10 seconds per page. The arrows show theta waves with slightly rounded peaks.

is relatively uncommon to record an actual seizure during a standard EEG recording. It is these interictal "footprints" that make the EEG such an effective tool in the diagnosis of seizures. Epileptiform activity is felt to represent increased cortical excitability or irritability. Apart from epileptiform activity, the EEG may also provide indirect clues that may aid in the diagnosis of epilepsy, such as regions of abnormal slow-wave activity or other findings.

The difference between interictal activity and electrographic seizure activity is usually straightforward. For example, the sporadic individual anterior temporal spikes seen in patients with temporal lobe epilepsy would not be mistaken for actual seizure activity. The patient looks and feels perfectly well while these spikes are firing. It is important to communicate this distinction to patients and their families (and also to referring physicians) who may incorrectly assume that epileptiform activity in the EEG implies that the patient is experiencing ongoing seizure activity that had not been recognized. The patient should be informed that the abnormal discharges seen are a sign of electrical irritability in a particular location and may mark a decreased threshold for seizure in that area (a potentially epileptogenic area), but they do not mean that a seizure has or is occurring in that area, nor do they guarantee that a seizure will occur in that area in the future.

A Note on Clinical Seizures, Electrographic Seizures, and Interictal Epileptiform Activity

A clinical epileptic seizure can be defined as a change in neurologic function associated with and caused by an abnormal, hypersynchronous discharge in the brain (see Figure 3-41). Usually, but not always, that discharge can be recorded by routine EEG techniques using scalp electrodes. Even if not recordable at the scalp, this definition implies that during true epileptic seizures, there would always exist some theoretical electrode placement (perhaps even within the substance of the brain itself) that could record the abnormal discharge.

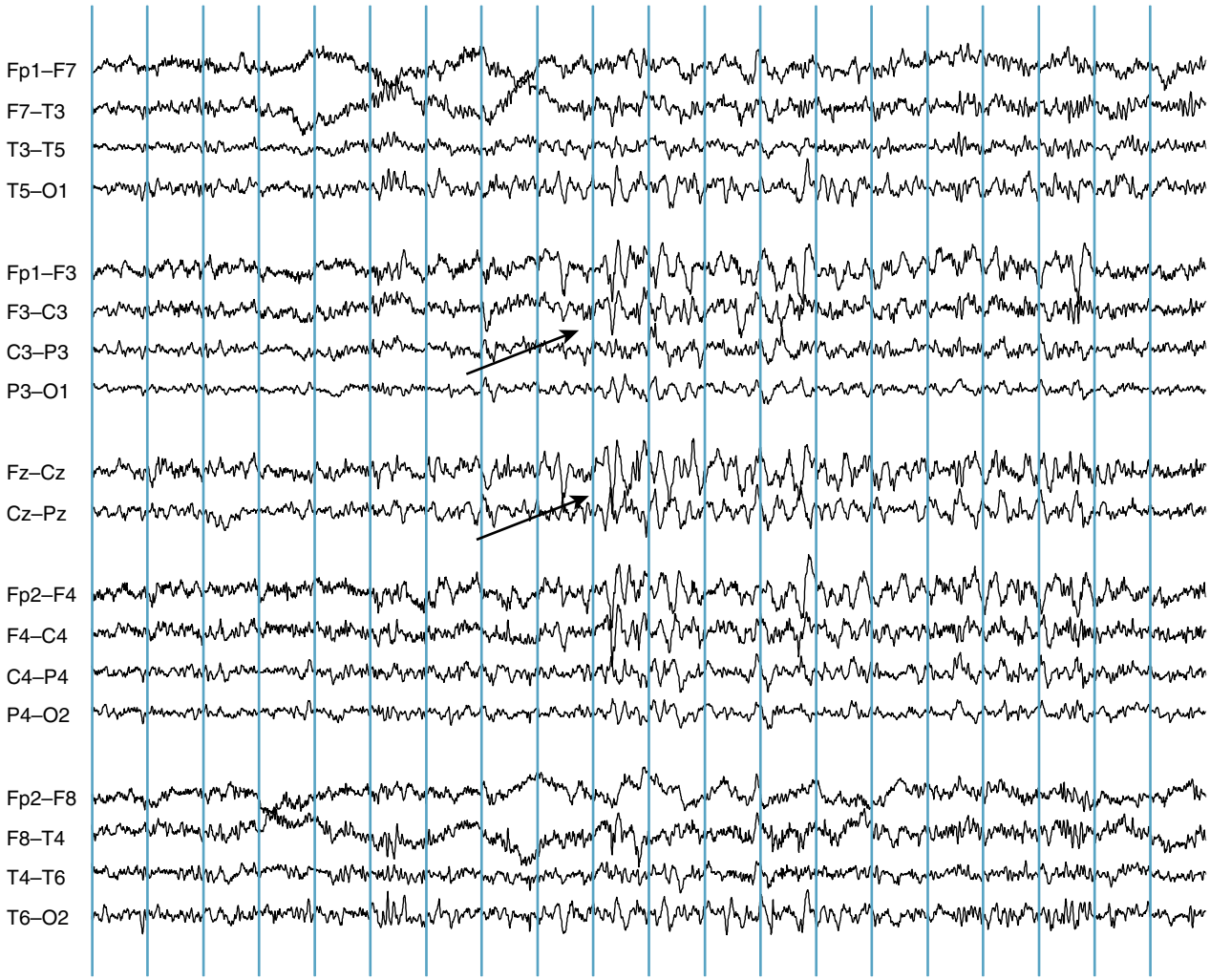

Figure 3-39 The figure shows the same theta waves from the previous figure (arrows) but displayed at 20 seconds per page. Note that the apparent sharpness of the waves is significantly accentuated at this display setting. Slower paper speeds compress the horizontal aspect of the display increasing apparent sharpness.

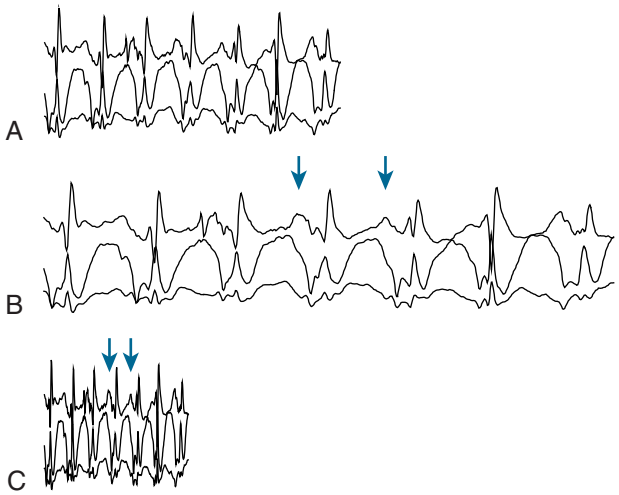

Figure 3-40 The paper speed chosen for display can have a large impact on the appearance of wave shape. Here, the same spike-wave complexes are displayed at three paper speeds: **(A)** a typical paper speed, **(B)** a fast paper speed, and **(C)** a slow paper speed. Note that the same spikes that appear quite sharp in panel A acquire a more rounded appearance in panel B. In panel C, even the aftercoming slow waves begin to acquire a sharpened appearance: compare the large and round appearance of the aftercoming slow waves displayed with fast paper speed in panel B to the same slow waves, now sharp-appearing, recorded at slow paper speed in panel C (arrows).

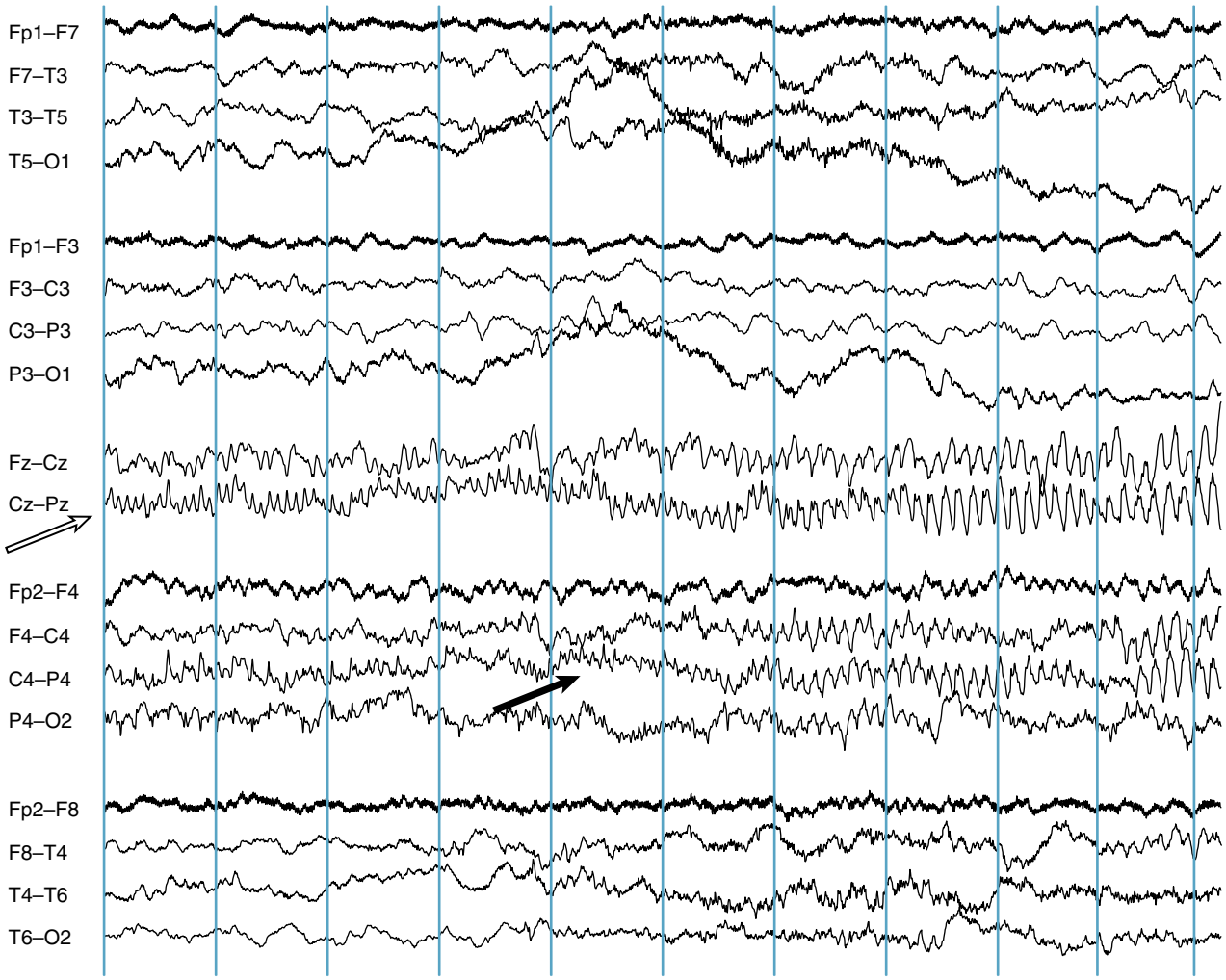

Figure 3-41 This seizure discharge is seen active at the vertex electrode (Cz) at the beginning of the segment (hollow arrow) and spreads to involve the right central area several seconds later (solid arrow).

An *electrographic seizure* refers to a seizure discharge seen in the EEG in the absence of any associated demonstrable clinical change in the patient. The change in neurologic function experienced during a clinical seizure can range from complete loss of consciousness with dramatic motor symptoms to more subtle changes such as confusional states, psychic symptoms, or visual or sensory phenomena. The definition of an epileptic seizure implies that a change in function, potentially evident to an outside observer or reportable by the patients themselves, has occurred during the episode. In some cases, however, an electrical seizure pattern is recorded on the EEG without any evidence of clinical change. For such "subclinical" electrical events, the terms *electrographic seizure* or *electrographic seizure pattern* are used. These terms are best used when there has been no clinical change associated with the discharge or when the electroencephalographer does not or cannot know whether there has been a clinical or neurologic change. Whether such electrographic seizures should be treated in the same way as electroclinical seizures is an important question in epileptology.

Types of Epileptiform Activity

The most common epileptiform discharges in the EEG are spikes, sharp waves, polyspikes, and spike and slow-wave complexes (also referred to as spike-wave complexes in this text). *Spike* is a descriptive term for an EEG wave that stands out from the background, has a sharp peak, and whose duration (base) is 70 msec or less (see Figure 3-42). *Sharp waves* are EEG waves with a sharp peak that also stand out from the background whose duration (base) is between 70 and 200 msec (see Figure 3-43). Although this definition seems precise, the duration cutoff between a spike discharge and a sharp-wave discharge is somewhat arbitrary and in most cases is not clinically important. In fact, in some patients the same discharge may vary during a recording so that some examples would be considered spikes and others sharp waves, even though they all clearly represent the same electrical phenomenon. There are few situations in which deciding whether a discharge of borderline duration is a spike as opposed to a sharp wave changes the clinical implications of the wave. Nevertheless, the terms are descriptively useful in that each

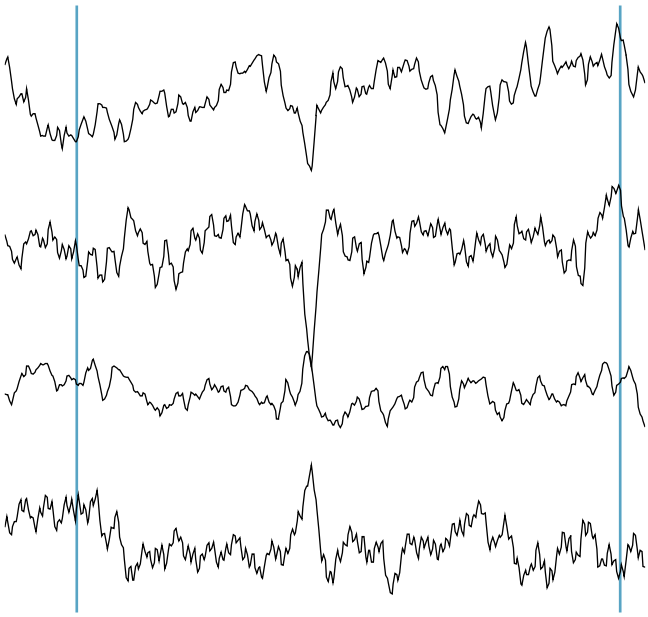

Figure 3-42 The appearance of a spike across a bipolar chain. Each vertical division represents 1 second (or 1,000 msec). Because the bases of these waves are of less than 70 msec in duration, they are classified as spikes rather than as sharp waves.

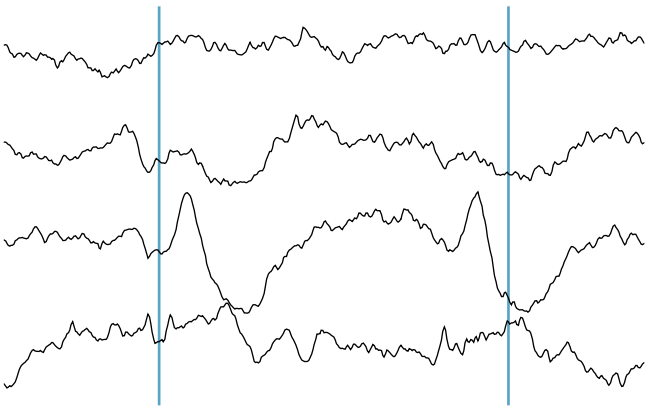

Figure 3-43 Two sharp waves are seen in the third channel. Each vertical division represents 1 second (or 1,000 msec). Because the bases of these waves are of approximately 150 milliseconds in duration, they are classified as sharp waves rather than spikes.

conveys a specific image to the reader and certain EEG phenomena do tend to manifest either as spikes or as sharp waves.

Sharpness

The sharpness of a wave is difficult to quantify and is somewhat subjective. Interestingly, the quantitative difference between sharp waves and spikes is based on the base of the wave rather than the sharpness of the peak. Furthermore, as examples in this chapter have shown, waves can appear sharper visually when displayed at higher amplitudes or slower paper speeds. The difficulty of defining sharpness is reflected by some of the amusing ways it has been defined: "if you touch it, it would feel sharp" or "if you sat on it, it would hurt!"

The term *spike-wave complex* refers to a pattern in which a spike is immediately followed by a slow wave (see Figure 3-44). Occasionally the initial sharp component of the complex is more than 70 msec in duration (i.e., a sharp wave) and the term *sharp-and-slow-wave complex* can be used. Even in the case of discharges that have been called simple spikes or sharp waves, careful examination can reveal a sometimes subtle aftercoming slow-wave component. *Polyspike* or *polyspike and slow wave discharges* are similar to spike-wave complexes except that there are multiple phases (up-and-down sweeps) to the spike component (see Figure 3-45). An alternative term for polyspike is *multispike*.

With only a few exceptions, the use of the terms *spike* and *sharp* wave connotes abnormal EEG activity. One

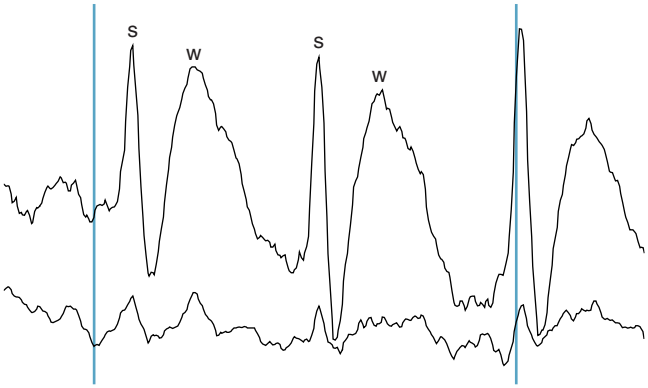

Figure 3-44 Three spike-wave discharges demonstrating classic spike-wave morphology are shown. Each vertical division represents 1 second. An "s" denotes the spike component, and the "w" denotes the aftercoming slow wave.

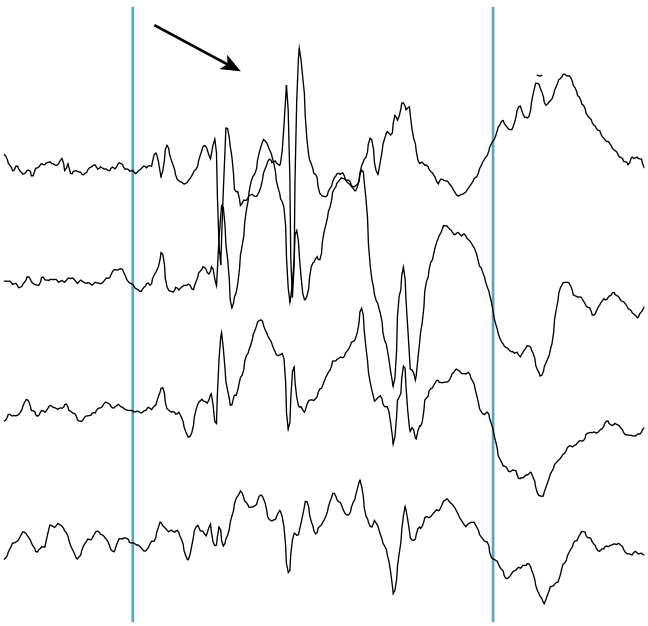

Figure 3-45 Polyspike-wave discharges are displayed in a bipolar chain. Each vertical division represents 1 second. The discharge is classified as a polyspike and slow wave because the initial spike component has multiple (up and down) phases (arrow) before the following slow wave.

common exception is vertex sharp waves of sleep. When spikes or sharps are known to be benign from their context, the electroencephalographer has the option of using the essentially synonymous but more neutral-sounding terms *transient* or *sharp transient* to avoid the connotation of abnormality that the terms sharp and spike suggest to some. The term *transient* is a generic term that refers to any very fast (short duration) electrical event. Most spikes and sharp waves may also be considered transients, and there is, therefore, considerable overlap in the terms. It has, however, become customary to reserve the term transient for sharp activity that is felt to be benign, such as POSTS (positive occipital sharp transients of sleep) and various normal sharp activity seen in the newborn EEG (e.g., frontal sharp transients).

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Chapter

4 Electroencephalographic Localization

It is easy to underestimate the importance of learning the skills of precise EEG localization compared with the bigger picture of EEG interpretation. On the face of it, the purpose of localization is to identify the location in the brain at which a recorded EEG event has occurred. Indeed, if this were the only benefit of learning accurate localization, it would still be a key skill in EEG interpretation. However, as we shall see in this chapter and elsewhere in this text, localization can help answer not only where an EEG event has occurred but also what an EEG event is. Understanding the topography and polarity of a discharge is the first step in deciding whether an EEG event is truly of cerebral origin or is instead an electrical artifact. Consider for a moment the potential negative impact on patient care of reporting an electrical artifact as an abnormal cerebral discharge. As we shall see, the shape or distribution of certain EEG discharges may not be consistent with cerebral activity because the localization does not make either topographical or biological sense. The skill of accurate EEG localization includes the skill of determine whether the shape or distribution of a discharge is or is not consistent with an event that originates from the brain. In addition, certain discharge topographies can suggest a specific type of discharge and the clinical syndrome to which the discharge might belong; in certain cases, the location of the discharge can reveal what it is.

Complete localization of an event includes pinning down both the event's location and its polarity. For instance, an accurate localization would include not only the fact that an event occurs in the left anterior temporal electrode but also whether it is negative or positive at the scalp surface. Almost all types of EEG events can be localized, whether they be spikes, sharp waves, slow waves, or even voltage asymmetries. In the following discussion, the examples used are spike-like discharges, although the principles discussed here apply equally well to almost all types of EEG events (e.g., sharp waves, slow waves, etc.).

FOCAL EVENTS, ELECTRIC FIELDS, AND GENERALIZED EVENTS

Focal Events

A focal EEG event is one that occurs on a limited part of the brain surface rather than over the whole brain surface. Occasionally, an event may be so focal that it occurs in only one electrode. The large majority of discharges will also be detectable, though perhaps more weakly, in adjacent electrodes. The electrode position that picks up the highest voltage, be it positive or negative, is referred to as the discharge's maximum. Although the discharge may be best seen at the maximum, adjacent electrodes often pick up varying amounts of the discharge. The hypothetical discharge shown in Figure 4-1 shows a discharge maximum in the right parietal electrode (P4) with a field that includes the right posterior temporal electrode (T6) and, to a lesser extent, the right occipital electrode (O2). A focal discharge having a broader field, such as the one shown in Figure 4-2, can have a maximum at one electrode (C4 in this example) but involve a substantial part of one hemisphere.

Rarely, an EEG event may occur so focally that its activity is only recorded in a single electrode, such as the example of the highly focal right parietal discharge shown in Figure 4-3. According to the schematic, the discharge would only be detected by the single electrode (P4), and adjacent electrodes would be electrically quiet. In practice, such highly focal discharges are uncommon and represent the exception rather than the rule (although this phenomenon of highly focal discharges is more commonly seen in newborns). The first two examples given above in which a discharge is detected by a group of electrodes is the more commonly encountered situation. The pattern of how strongly the discharge is picked up in various electrodes helps define the shape of the discharge's electric field as discussed next.

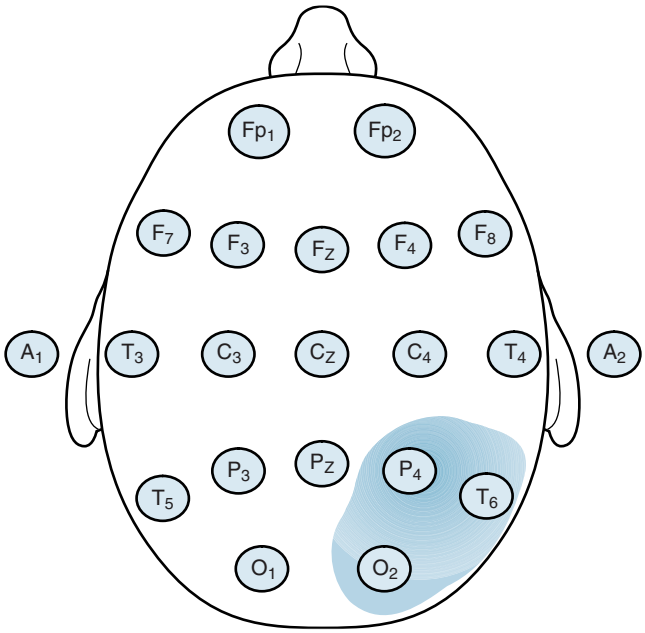


Figure 4-1 This focal discharge is clearly maximum in P4, but the electric field of the discharge extends to include T4 and O2. Rather than seeing a discharge occur at a single electrode position, this is the more common situation in which the maximum of a focal discharge is picked up in one location (P4 in this example) but the discharge can be detected to a lesser extent in adjacent electrodes (T6 and O2). In this case, the discharge can be said to be maximum in P4 with a field that extends to T6 and O2.

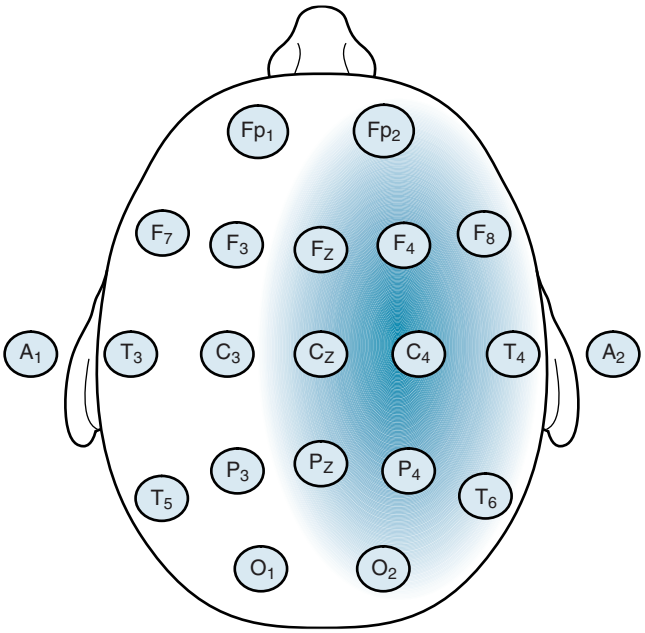


Figure 4-2 In the case of a lateralized discharge, the field of the discharge involves either a whole or nearly whole hemisphere. In this example, the discharge can be said to be lateralized to the right hemisphere.

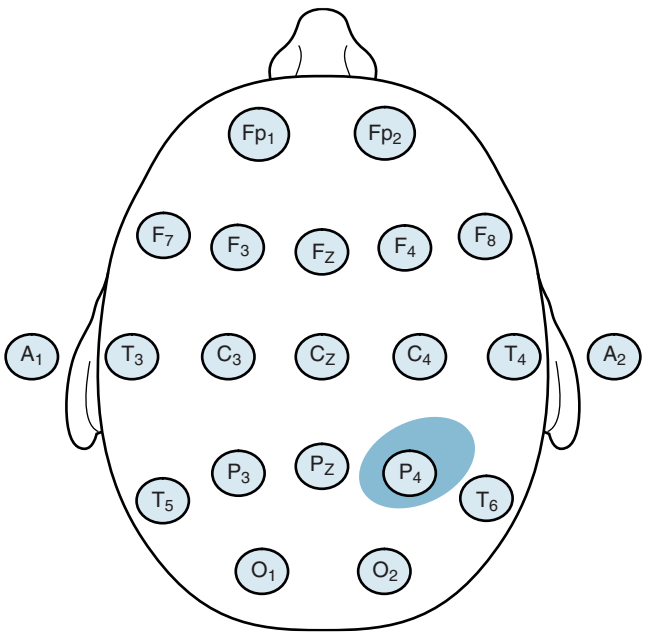


Figure 4-3 The discharge depicted here is highly focal and is only detected by a single electrode (P4). The adjacent electrodes are electrically "quiet." This highly focal pattern is relatively uncommon; most focal discharges affect multiple electrodes at once. When an event is limited to a single electrode, the possibility of an electrode artifact should be considered.

Electric Fields

Several analogies have been used to describe the shape of the electric field of a typical discharge on the scalp surface and how its intensity drops off with distance from the maximum. The analogy of a pebble dropped into a quiet pool of water can be used to describe the way a simple field's strength dissipates as it becomes more distant from the point of highest intensity (where the pebble hit the water). The wave that is formed is strongest at the point of impact but diminishes with increasing distance from the central maximum. This example is useful because many electric fields measured on the scalp do show this radially symmetric shape but, in practice, many electric fields dissipate with varying shapes.

Rather than showing a smooth and steady decrease in voltage in every direction from the central maximum point, it is possible for fields to dissipate gently in one direction and abruptly in another. A better analogy for the shape of electric fields is the visualization of mountain peaks. Mountain peaks give a more realistic picture of EEG fields because they need not be so perfectly symmetrical in shape as the circular waves caused by a pebble hitting water. Imagining the terrain around a 5,000-foot mountain peak, we might expect that the height of land surrounding the peak will fall off with varying steepness in each direction. Likewise, electric fields may manifest a steeper slope of voltage decrease in one direction and a more gentle slope in another direction. An abrupt and immediate falloff in voltage from a central point in all directions as shown

in Figure 4-4 would correspond to a thin needle of land 5,000 feet high with nothing surrounding it, an uncommon finding both in geography and in electroencephalography but akin to the discharge in Figure 4-3.

The mountain peak analogy reminds us that an electric field can be visualized as a surface in three dimensions. Just as slope refers to the steepness of a curve imagined in two dimensions at a given point, the steepness of a surface imagined in three dimensions at a given point is referred to as the *gradient* at that point. (The *slope* of a curve at a given point is the slope of a line tangent to that curve at a given point. Similarly, the *gradient* of a surface at a given point is defined by the slope of an imaginary plane tangent to the surface at that given point.) The rate at which the terrain that surrounds the summit of a mountain falls off is the *steepness* of the terrain. The rate at which an electric field changes intensity at a particular point on a surface is called the electrical *gradient;* the two properties are analogous. The exercise of visualizing the shapes of electric fields is similar to visualizing the contours and steepness of a region of mountain terrain. Just as we would never confuse the area of maximum altitude of a mountain with the point of maximum steepness of a mountain (which may or may not be at the same point), so we will take care not to confuse the point of maximum voltage of an EEG event with the point of the maximum gradient of the field surrounding the maximum.

The illustration in Figure 4-4 depicts a highly focal discharge, similar to that shown in Figure 4-3. The plane of the rectangular grid is a representation of the voltages measured on the scalp surface. In this figure, areas close to the discharge's peak are not involved, and relatively nearby electrodes would not "perceive" any change in voltage. Such highly restricted or "punched-out" discharges are relatively uncommon (just as 5,000-foot mountains in the shape of a needle are uncommon). The field shown in Figure 4-5 is somewhat more realistic, with a peak or maximum in the same position, but a more gradual falloff in voltage as distance increases from the maximum point. In this example, adjacent electrodes would pick up increasingly weaker voltages with increasing distance from the point of maximum. Figure 4-6 shows a discharge with a broad field, and, although the middle electrode would pick up the highest voltage, the adjacent electrodes would detect a voltage intensity of more than half that detected by the middle electrode. Figure 4-7 reminds us that, quite often, the electric field can slope off asymmetrically from its peak.

Generalized Events

In contrast to the focal events described earlier, some events occur in all brain areas at once and are said to occur in a *generalized* distribution. Figure 4-8 depicts a discharge with a perfectly even electric field. With cerebral discharges, even in the case of generalized discharges, there is almost always some unevenness to the field, and an area of maximum intensity can still be identified. In Figure 4-9, the discharge affects all brain areas and is, therefore, generalized, but the intensity is highest in the F3, Fz, and F4 electrodes.

The purpose of this chapter is to help the reader become adept at translating the patterns of pen deflections recorded on the EEG page into the particular localizations, polarities, and the shapes of the gradients that those patterns imply. In short, we examine the patterns that EEG pens will draw when they encounter

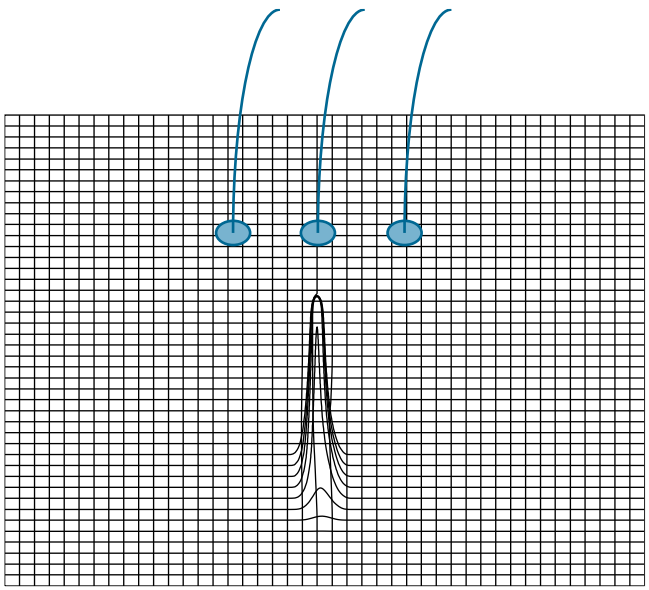


Figure 4-4 This schematic illustrates three electrodes recording over a highly focal discharge. Note that because of the pinpoint location of the discharge, only the middle electrode detects a change in voltage.

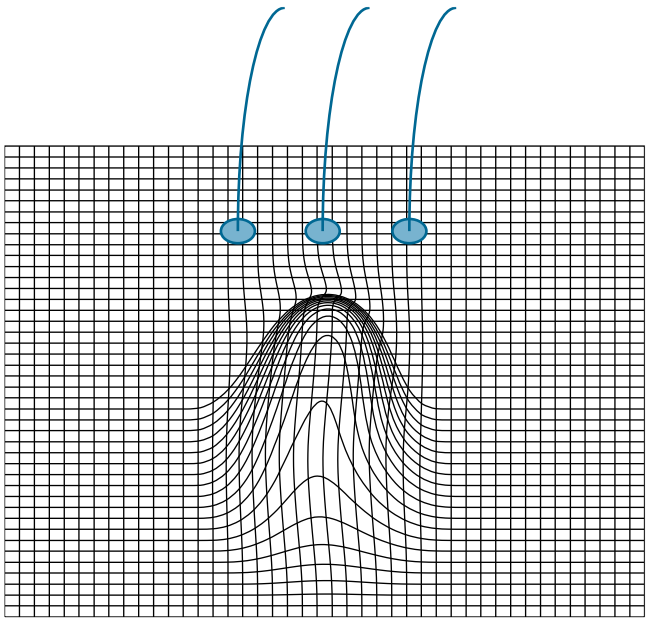


Figure 4-5 The discharge depicted here has a more broadly sloping electric field, and although the middle electrode picks up the biggest voltage change, adjacent electrodes pick up lower voltage changes on the "shoulders" of the field.

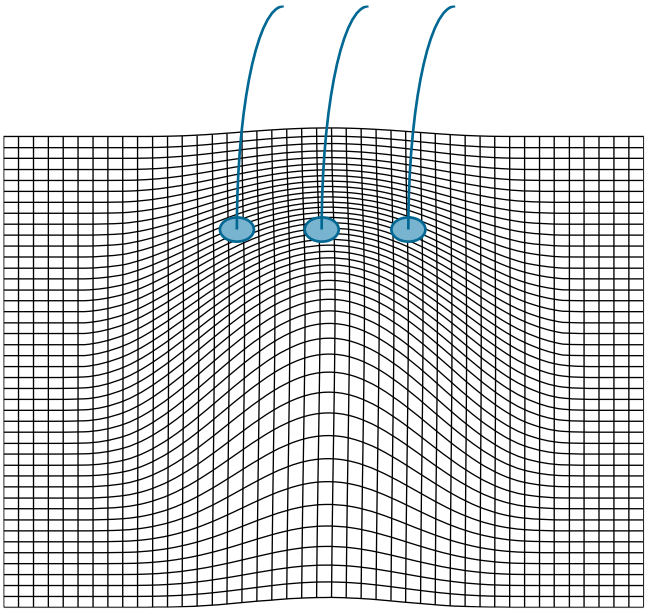


Figure 4-6 Again, the middle electrode picks up the maximum voltage change, but the adjacent electrodes also pick up a significant portion of the discharge on the slopes of its electric field.

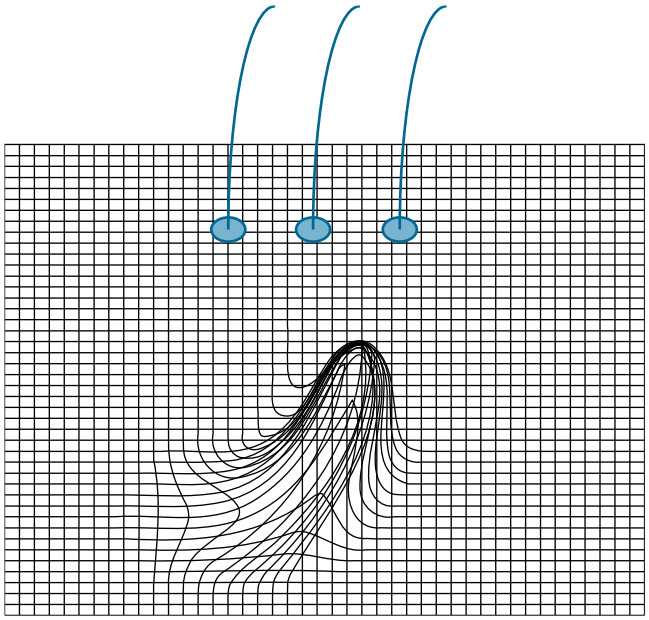


Figure 4-7 The intensity of the field of this focal discharge slopes off more steeply in some directions and more gradually in others. Such asymmetric gradients are common.

electrical fields of various shapes, including the types depicted in the figures discussed earlier. Ideally, after analyzing an EEG discharge on an EEG page, the reader will be able to imagine a "mountain range" configuration that accurately depicts the shape and the gradients of the discharge's electric field.

EEG WAVES AND EEG POLARITY

Pen Up or Pen Down?

An understanding of what causes the EEG instrument's pen to go up or down is the foundation concept of EEG localization and is one of the central skills to master in this chapter. Luckily, the convention is easy to understand. The challenge is to remember to apply it consistently when analyzing EEG patterns.

EEG Channels Are Fancy Voltmeters

A voltmeter measures the potential difference or "voltage drop" between two points. Because voltages are always differences, whenever a voltage measurement is made, the reader should be aware which two identifiable points are being compared. Even when it appears that the voltage at a single point is being described, it is implicit that the point is being compared with some standard (perhaps a ground or other "neutral" point). Likewise, even though there may be a temptation to think of the output of an EEG channel as representing the activity at some single point on the scalp, every channel deflection we see on the EEG is, in reality, a *comparison of two different points* on the body (or occasionally a combination of more than two points, as we shall see in later chapters). Any wave seen on the EEG is, therefore, a comparison of the electrical potential at two locations rather than an absolute measurement made at a single location. The waves that we are analyzing are, indeed, the outputs of constantly fluctuating voltmeters comparing two inputs (see Figure 4-10).

Inherent to the concept of the voltmeter is the idea of subtraction. If one probe of a voltmeter contacts a point at 105 μV and the second probe contacts a point that is at 100 μV, the voltmeter will read out 5 μV. Likewise, if the pair of points recorded is −112 and −117 or even 3 and −2, the voltmeter will read out the same result: the difference of 5 μV. The result of 5 μV that the voltmeter yields gives no clue as to which of these pairings generated it: 105 and 100, −112 and −117, or 3 and −2. The fact that an EEG channel only shows us the difference between two points rather than absolute values has both advantages and limitations. Because each wave displayed on the EEG is a continuous voltmeter output, the reader should always have in mind the following question: *which two points are contributing to this channel's appearance?*

The reader may be wondering which would be more logical, for the pen to go up or for the pen to go down, in the example in the preceding paragraph in which the difference is positive 5 μV. The answer to this question cannot be derived using mathematical or physical principles. Rather, it is an arbitrary convention that was decided by EEG machine manufacturers in the early days of EEG. The answer for the earlier example is that the pen goes down. The convention assumes that there are two input terminals to the amplifier, Grid 1 and Grid 2, which can also be called Input 1 and Input 2. The convention is: "First input more negative: pen goes up. First input more positive: pen goes down." Some

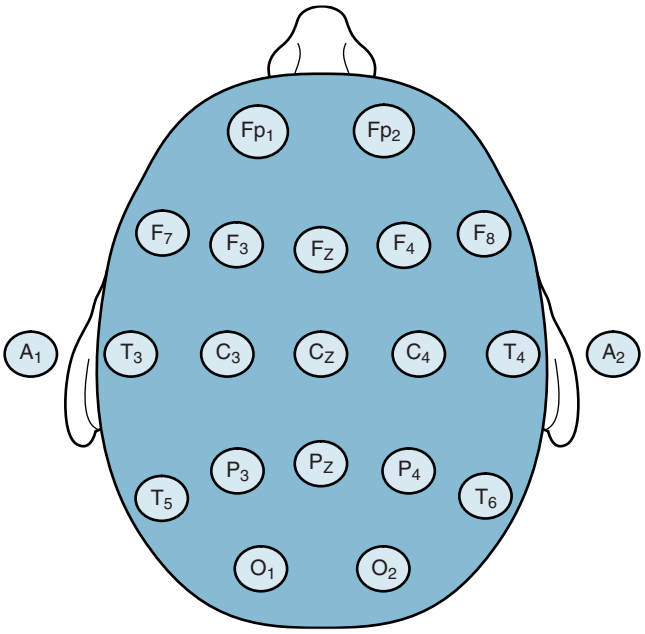


Figure 4-8 This is a depiction of an idealized generalized discharge. The shading of this figure implies that the event is distributed in a perfectly even fashion, although in actual recordings, some amount of unevenness is usually seen even in the fields of generalized discharges.

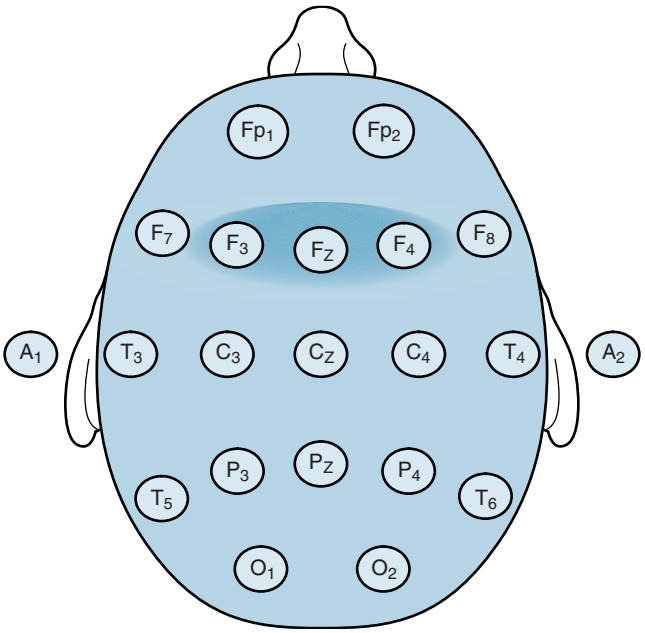


Figure 4-9 This generalized discharge has a clear maximum in the superior frontal and midline frontal electrodes, F3, F4, and Fz. This is a common localization pattern for the "classic generalized spike-wave" discharge.

may find this convention counterintuitive because in most areas of mathematics and science, values are graphed above the x axis when they are positive and below the x axis when they are negative. Alas, in electroencephalography, the opposite is true; this "upside-down"

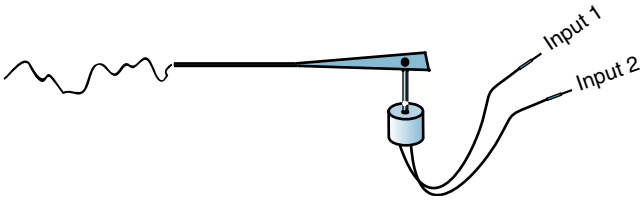


Figure 4-10 An EEG channel can be thought of as the pen output of a continuously recording voltmeter with the two inputs specified by the recording montage in use.

convention is here to stay and can take some getting used to. It may be helpful to use the counterintuitive nature of the convention to help remember it.

Here is how the convention works in more detail: as noted, every EEG amplifier has two principle inputs, which we will refer to as *INPUT 1* and *INPUT 2*. The EEG technologist decides which electrodes are plugged into INPUT 1 and INPUT2 for any given channel according to which EEG montage is chosen. The situation of INPUT 1 being more negative than INPUT 2 can be stated in a number of ways, such as: "the difference between INPUT 1 and INPUT 2 is negative" or as the mathematical expression: "INPUT 1 − INPUT 2 $<$ 0." In such cases the amplifier's pen deflects upward. Of course, the opposite holds true for the reverse situation: when INPUT 1 is more positive than INPUT 2, the pen deflects downward as in the example above (see Figure 4-11).

When INPUT 1 is **more positive** than INPUT 2, the pen goes **down.**
When INPUT 1 is **more negative** than INPUT 2, the pen goes **up.**

Of all the ways to state this rule, the subtraction expression "if INPUT 1 − INPUT 2 $>$ 0 …" is most correct because it deals with all the possible combinations of each of the two inputs being positive, negative, or zero. One could quibble with the idea of saying that −3 is "more positive" than −5 because neither is positive. In this text, we use this simpler shorthand, stating that one electrode is "more positive" or "more negative" than another without regard to the sign of the electrodes' absolute voltages for simplicity's sake. Figure 4-12 shows another representation of this convention.

EEG Amplifiers

To understand the meaning of pen deflections, it is useful to consider the nature of the amplifiers used in the electroencephalograph. Perhaps the simplest conceivable amplifier design would be the *single-end input amplifier*. This type of amplifier would take a single signal as its input and furnish an amplified version of that signal as its output (see Figures 4-13 and 4-14). This is not, however, the type of amplifier used in EEG machines, and for good reason. A single-end amplifier is essentially using the building's electrical ground as

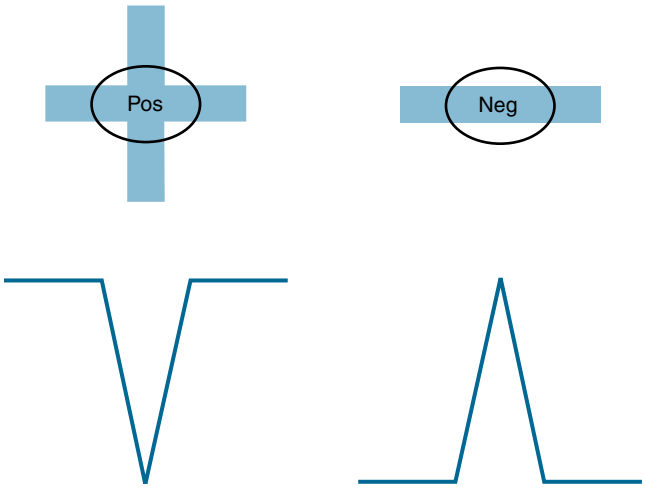


Figure 4-11 The convention for pen movement is best memorized on the basis of the INPUT 1 electrode or "Electrode 1." When Electrode 1 is more positive than Electrode 2, the pen goes down. When Electrode 1 is more negative than Electrode 2, the pen goes up. For this reason, in referential montages in which the electrode of interest is attached to INPUT 1 and a "neutral" reference electrode is attached to INPUT 2, negative events on the scalp appear as upgoing deflections.

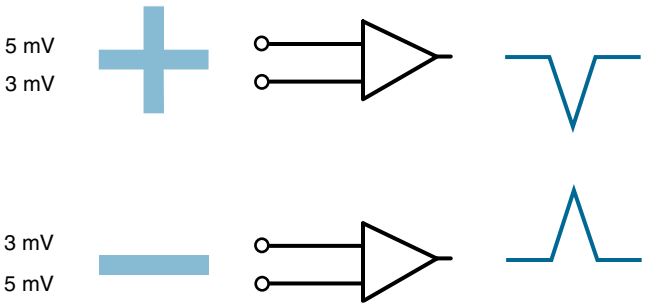


Figure 4-12 When the common mode rejection amplifiers used in EEG recording encounter a "more positive" voltage in INPUT 1 compared with INPUT 2, the pen goes down as is seen in the top example. Likewise, when the voltage in INPUT 1 is "more negative" compared with INPUT 2, the pen goes up, as is seen in the bottom example. The top example shows a 5 μV wave presented to INPUT 1 and a 3 μV wave presented to INPUT 2. The difference is positive, and, by convention, the pen goes down. The bottom example shows the result of switching the input voltages. Now the difference between INPUT 1 and INPUT 2 is negative, and the pen goes up.

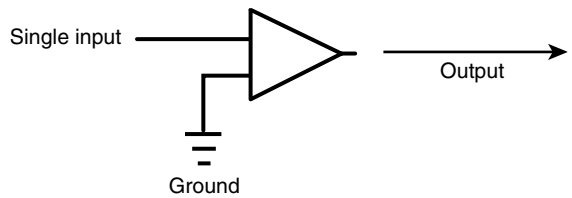


Figure 4-13 A single-end amplifier takes a single active input and compares it to electrical ground, theoretically an electrically neutral position, and amplifies the difference. In practice, building grounds are often contaminated with significant amounts of electrical noise from other devices attached to the ground at other locations. When compared with and subtracted from the weak EEG signals in the single active input, the noisiness of the ground could swamp the low-voltage brain wave signal.

the comparison input; however, such grounds are usually much too electrically contaminated or "dirty" to be useful for this purpose.

Actual EEG amplifiers, like the voltmeters described earlier, use two inputs (see Figure 4-15). The signal from INPUT 2 is subtracted from the INPUT 1 signal; the result is then amplified and serves as the output. This type of amplifier is also called a *common mode rejection* (CMR) amplifier. As we shall see, the strategy of subtracting one input signal from the other has several important advantages. Figure 4-16 shows how a CMR amplifier behaves with two sample electrical inputs. Note that the component of each signal that is common to both inputs is cancelled out, or "rejected." Only the difference between the two signals appears in the output. Why is elimination of the part of the signal that is common to both electrodes an advantage? This technique of amplification is especially useful in the field of electroencephalography in which the signals of interest—brain waves—are of very low voltage compared with the ambient electrical noise from external sources that runs through the patient's body. Because the pattern of this external noise signal tends to be similar in the various scalp electrodes, CMR amplifiers cancel out much or all of the external noise component of the signal, ideally leaving only the cerebral component for interpretation.

Event Localization Using a Bipolar Montage

The examples that follow consider a theoretical spike discharge on the scalp and how it would appear on the EEG record. The first example we examine is a hypothetical spike in the left central region of the brain (the area under the C3 electrode). A spike is, by definition, a quick event, and in this example, it will have a negative polarity (the scalp region where the spike

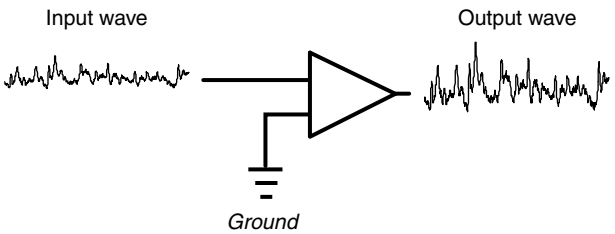


Figure 4-14 This idealized single-end amplifier takes a single signal as input and outputs an amplified version of the same signal. This is not the type of amplifier setup used in EEG machines.

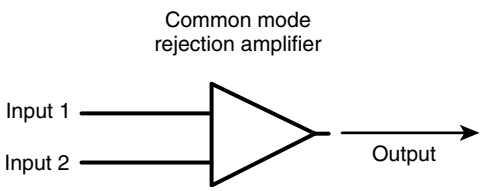


Figure 4-15 The common mode rejection amplifier accepts two inputs, each usually from a single electrode, and yields a single amplified output.

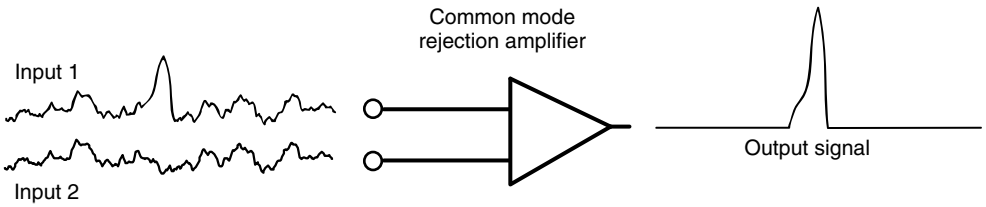


Figure 4-16 An idealized view of a CMR amplifier processing two signals. Note that there is a single difference between the INPUT 1 and INPUT 2 signals, a single upward deflection. The complex underlying waveform is common to both inputs and, when the two signals are subtracted, is cancelled out or "rejected." The "difference signal," consisting of the upward deflection, is amplified and represents the output. In this example, one can imagine that the waveform recorded from INPUT 2 is obtained from a location such as the earlobe and consists solely of electrical noise. The signal recorded from INPUT 1, which has been placed on the nearby scalp, is contaminated by the same electrical noise as the earlobe but also contains a single "brain wave," the upward deflection. The result of the CMR amplifier's action is to subtract out the noise signal and display an amplified brainwave.

occurs will be momentarily negative compared with the surrounding scalp, which, for the purposes of this example, is neutral). The electrode set we use to record the spike is a single chain of standard electrodes that goes along the scalp from front to back just to the left of the midline: Fp1, F3, C3, P3, and O1 (the left frontopolar, superior frontal, central, parietal, and occipital electrodes, respectively). The technique used for the following examples is the typical strategy of creating a *bipolar chain* with these electrodes by looking at a succession of pairings of adjacent electrodes from front to back: Fp1 to F3, F3 to C3, C3 to P3, and P3 to O1. The term *bipolar* is used because each EEG channel generated represents a comparison of two cerebral locations. Each of the two electrode locations can be said to be "of interest" because both reflect brain activity, and it is possible that a clinically important event could arise from under any of the five electrodes. (In the contrasting situation of referential montages described in more detail later, INPUT 1 is connected to an electrode over the brain, and INPUT 2 is connected to some other reference point that is presumably not "of interest" but is used for the purpose of subtracting noise.) Note that there are five electrodes in this chain but there are necessarily only four consecutive pairings. Thus, a chain of five consecutive electrodes will produce four *channels* of EEG in a bipolar chain.

"Negative Phase Reversal"

Figure 4-17 depicts the field of a 50-μV spike occurring at C3 with no activity at all in the surrounding electrodes. On the right, the four channels representing the output of the bipolar chain are depicted. It is worthwhile to go through each line of the output to understand exactly why voltage measurement shown on the left side of the figure is associated with the EEG trace on the right side of the figure.

The first channel, Fp1-F3 is measuring the difference between 0 μV at Fp1 and 0 μV at F3. Because there is no difference, the Fp1-F3 channel remains flat.

The second channel, F3-C3, is comparing 0 μV in INPUT 1 (F3) and −50 μV in INPUT 2 (C3). Because the convention is that if the first electrode is "more positive" than the second, the pen goes down, a downward pen deflection is produced.

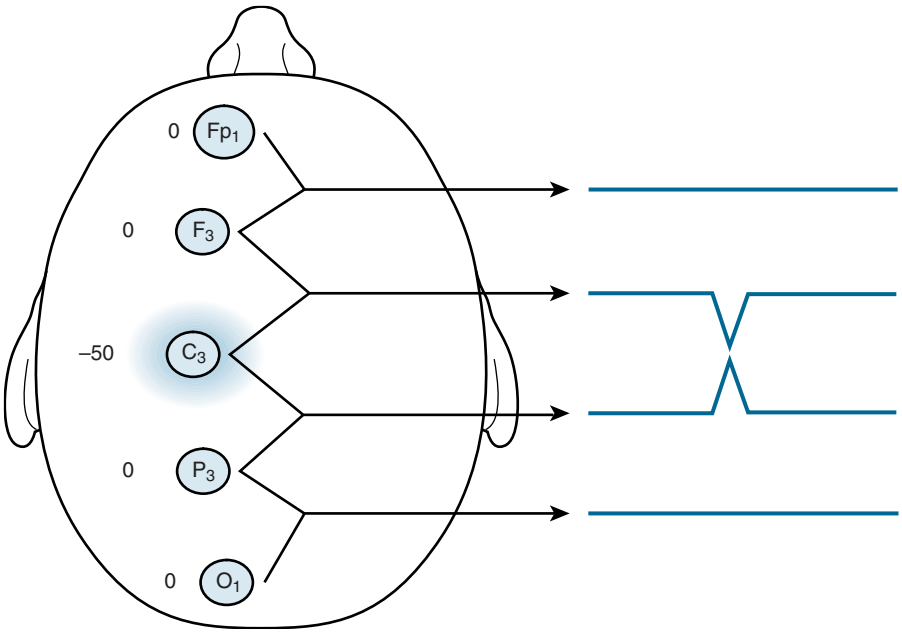


Figure 4-17 A *negative phase reversal* showing a focal negatively charged spike occurring only in the C3 electrode—none of the negativity of the spike is picked up in adjacent electrodes. The numerical voltages that each electrode detects are shown. The dark shading denotes the region of the scalp that becomes negatively charged during the spike. The resultant EEG trace seen in a bipolar chain (Fp1-F3, F3-C3, C3-P3, and P3-O1) is shown to the right and the reasons for each specific pen deflection are described in the text.

The third channel, C3-P3, compares −50 μV in INPUT 1 to 0 μV in INPUT 2. Now Electrode 1 is more negative than Electrode 2 (the opposite of what occurred in the second channel), so the convention tells us that the pen goes up.

Finally, in the fourth channel, there is no voltage difference measured between P3 and O1 (both 0 μV), so the channel remains flat.

The resulting trace shows one of the classic patterns of EEG waves, the *phase reversal*, which is expanded on below. For the time being, note that, in this example, where the pen deflection reversed direction, or *phase* (in this case from down in the second channel to up in the third channel), the electrode in common to the channels where the phase reversed from down to up (C3 since the phase reversed between F3-**C3** and **C3**-P3), points to the location of the discharge's maximum.

As discussed earlier, focal EEG events on the brain surface rarely occur solely in an isolated pinpoint area of the scalp as in the example in Figure 4-17. Instead, there is typically a point of voltage maximum around which lower voltages can be detected. The example shown in Figure 4-18 is more similar to real-world events. Although the C3 electrode is the point of maximum intensity of this event, the adjacent areas measured by F3 and P3 are also affected, but not as strongly. This gradual drop-off in intensity of voltage shows that the event has a somewhat broader field, even though we are still dealing with a −50-μV spike in C3. A good description of this spike would be that the spike has a maximum negativity at C3 but a field that also includes F3 and P3.

Now let's return to Figure 4-18, which shows a more realistic gradient around a spike focus at C3. The resulting EEG trace is similar to the one we saw earlier but with some notable differences:

The first channel, Fp1-F3, is measuring the difference between 0 and −30 μV. Because Electrode 1 is "more positive" than Electrode 2, the pen deflects downward an amount corresponding to the difference of 30 μV.

The second channel, F3-C3, compares −30 μV in Electrode 1 (F3) and −50 μV in Electrode 2 (C3). Because the first electrode is again "more positive" than the second, a downward pen deflection is produced in this channel as well, corresponding to the smaller difference of 20 μV.

The third channel, C3-P3, compares −50 μV in Electrode 1 to −30 μV in Electrode 2. For the first time, Electrode 1 is more negative than Electrode 2 (the opposite of the case in the second channel), so the convention tells us that the pen now goes up.

Finally, in the fourth channel, Electrode 1 measures −30 μV, and Electrode 2 measures 0 μV. The pen again deflects upward, corresponding to the fact that Electrode 1 is more negative than Electrode 2 by a 30-μV difference.

Comparing the pattern of EEG waves in Figure 4-18 to the previous figure, we see that there are now deflections in all four channels. This reflects the fact that the field of this spike involves both adjacent electrodes, F3 and P3, and that the gradient of the field stretches out more broadly than in the first example. Stated another way, there is now an electrical gradient (difference) between all four channel pairs. The fact that there is a deflection in each channel reflects the fact that no two adjacent electrodes measure the exact same voltage and that there was an increase or decrease in voltage in every comparison. Because in channels 1 and 2 the pen goes down and in channels 3 and 4 the pen goes up, we say there is a *phase reversal* occurring between channels 2 and 3. A phase reversal is the point along a bipolar electrode chain at which the direction of the pen deflection changes (from down to up or from up to down). Because in this example C3 is the electrode common to channels 2 and 3, the two channels between which the phase "reversed," this is the point of maximum of the discharge.

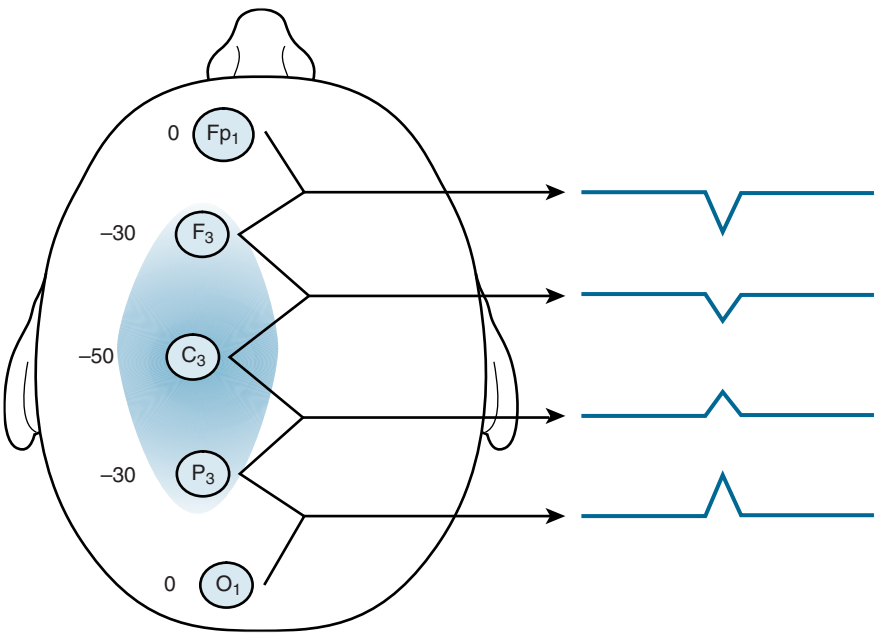


Figure 4-18 A –50-μV spike in C3 with a field extending to include F3 and P3 viewed in a bipolar montage. The F3-C3 and C3-P3 channel deflections reflect the 20-μV difference between those electrode pairs. The higher deflections seen in the top and bottom channels, Fp1-F3 and P3-O1, reflect the 30-μV gradient between those electrode pairs.

A second interesting observation can be made about this recording. Even though the biggest pen deflections can be seen in channels 1 and 4, the true *maximum* of the discharge lies between channels 2 and 3. This apparently paradoxical result occurs because, in this example, the electrical gradient was steeper at some distance from the maximum (nearer the forehead and occiput), similar to the example of a mountain peak in which the terrain could be steeper around the base of the mountain and less steep as it reaches its peak. Still, the "peak" is at C3, even though the deflections in Fp1-F3 and P3-O1 are larger. Clearly, in bipolar montages the maximum of a discharge cannot be located simply by finding the biggest waves—a pitfall to be avoided. This is because the measuring stick used to determine wave heights in bipolar montage tracings is the differences between pairs of points rather than the absolute value of the voltage at any given point. *In bipolar montages, rather than using wave heights, the maximum is located by finding the phase reversal.*

Each EEG channel in a bipolar chain passing from front to back along the head is, in reality, answering the question in its march along the brain: "is the next electrode getting more negative or more positive?" The following analogy illustrates this effect another way: a farmer has a large field and would like to build his house at the highest point in the field to have the best view of his farm from his house. The farmer does not have a standard instrument to measure height (an altimeter), and he does not trust himself to find the highest point using standard visual inspection. Rather, the only tool he can use to locate the highest point in the field is a crude instrument that only reports the difference in height of his current position compared with his last position as he takes steps across the field. It can inform him that the field has gotten higher compared with his last location (in which case its pen goes down) or that the field has gotten lower compared with his last location (in which case its pen goes up). He takes readings every time he walks 10 feet forward. Even without having the benefit of an altimeter that could report absolute numerical altitudes, as he walks forward through his field, it is easy to imagine a strategy he can use to locate the highest point along any track he walks.

Imagine that the farmer turns on the instrument (which senses his starting position) and then walks forward the first 10 feet. The instrument, comparing the starting position to his present position, reports "it's getting higher" (the pen goes down). After the next 10 feet, it reports that it is getting higher again (the pen goes down again). After the next 10 feet, it reports "it's getting lower" (the pen now goes up). This is the point of the "phase reversal" when the pen direction has changed. The farmer does not know the absolute altitude at the previous point, but he does know that the area between the points at which his instrument's pen flipped from down to up must mark the highest point on the track he has walked so far. After the pen readings shift from downward to upward, he knows he has located a height maximum along the line he is exploring. This is the equivalent of a phase reversal in a bipolar chain on the EEG. Before making a final decision, he will probably want to get to the end of the field to make sure that he found the highest point on the whole path. If he wanted to make a topographic map of the whole field, he could perform the same top to bottom walk along the field in several parallel lines. To be even more complete, he might repeat the measurements walking on parallel tracks from right to left (at right angles to the original paths). Using this strategy and recording the amount his pen deflects on every measurement, he would eventually be able to draw a relative topographic map of the whole field, something like the grid shown in Figure 4-6.

In these examples, greater altitude in the farmer's field is analogous to EEG voltage becoming more negative. The EEG reader can imagine "walking down" or scanning a bipolar chain along the scalp such as from Fp1 all the way to O1 and, like the farmer, feel as the pens go down that "it's getting more negative" and then as the pens finally reverse direction that "it's getting more positive." When the pens switch direction, this implies that the point of maximum negativity was passed, identifying the position of the maximum voltage of the spike. Figure 4-19 shows the deflections that the farmer's instrument might generate while walking along a single path, or likewise what an EEG machine might detect when exploring the parasagittal chain when there is a spike maximum at C3.

This example of measuring the altitude in a field with a device that only reports the change in altitude compared with the previous point is directly analogous to the process of examining successive electrodes (each channel representing a pairing of electrodes in an electric field) in a bipolar chain. Although the example assumed a field with a particular surface and predicted which type of pen deflections that surface would generate when explored with a chain of electrodes, practical EEG interpretation involves the reverse process: examining the channel outputs and ascertaining the shape of the underlying field.

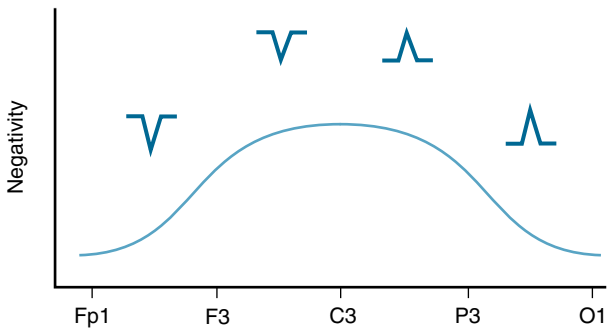


Figure 4-19 The figure shows an imaginary "walk" down a bipolar chain extending from the frontopolar area (Fp1) to the occipital area (O1). (Note that the y-axis shows degree of negativity so that more negative voltages cause the curve to go up.) There is a maximum negativity at the C3 electrode. As the curve goes up, the successive measurements (Fp1 to F3, F3 to C3) show progressively increasing negativity causing downward pen deflections. After the comparisons pass over the "voltage hump" of the C3 electrode (C3 to P3 and P3 to O1), the first electrode in the comparison pair is now more negative than the second electrode, and the pen points up. When the maximum point (C3) is passed, the pen direction reverses phase (from down to up), and this phase reversal marks the maximum. (The pen deflections shown above the graph are similar to the readings that could have been obtained by the farmer in the farmer-in-the-field analogy described in the text.)

The "Positive Phase Reversal"

Next, consider the situation of a positive spike occurring on the scalp, again at C3, as shown in Figures 4-20 and 4-21:

The first channel, Fp1-F3, reflects the difference between 0 and 20 μV. Because Electrode 1 is "more negative" than Electrode 2, the pen deflects upward an amount corresponding to 20 μV.

The second channel, F3-C3, compares 20 μV in Electrode 1 (F3) and 50 μV in Electrode 2 (C3). Because the first electrode is again "more negative" than the second, another upward pen deflection is produced, this time corresponding to the difference of 30 μV.

The third channel, C3-P3, compares 50 μV in Electrode 1 to 20 μV in Electrode 2. Now Electrode 1 is more positive than Electrode 2 (the opposite of the case in the second channel), so the convention tells us that the pen goes down.

Finally, in the fourth channel, Electrode 1 measures 20 μV and Electrode 2 measures 0 μV. The pen again deflects downward, corresponding to the fact that Electrode 1 is more negative than Electrode 2 by 20 μV.

In contrast to the type of phase reversal illustrated in Figure 4-18, Figure 4-20 shows an example of a second type of phase reversal, the *positive phase reversal*. This type differs from the first in that the first represented a transition of pens going down to pens going up (as the eye proceeds down the page or pen deflections facing toward one another). In this second type of phase reversal, there is a transition of pens going up to pens going down, or pen deflections facing away from one another. The phase reversal with the pens-down-to-pens-up transition marks an area of maximum negativity, and the second type of phase reversal with the pens-up-to-pens-down transition marks an area of maximum positivity. As shorthand, the first type of phase reversal with the pens pointing toward each other can be called a *negative phase reversal*, and the second type with the pens pointing away from each other a *positive phase reversal* (see Figure 4-22). Working through these examples step by step as we did earlier, it becomes clear that in a bipolar montage:

When the phase-reversing waves point toward each other, the point of maximum intensity is negative. When the phase-reversing waves point away from each other, the point of maximum intensity is positive.

This generalization always holds true because of the basic convention of direction of pen deflection and polarity. In the case of the negative phase reversal, the transition from pens going down to pens going up is the same as passing from "getting more negative" to "getting more positive comparisons." The opposite story holds true for the positive phase reversal with deflections that face away from each other. As you look downward through a chain with upgoing waves, the upgoing pens imply that the field is getting progressively more positive. After the waves flip to downward, the opposite is true: the field is now becoming progressively more negative, implying that a peak positivity has been passed.

Although it is possible to derive the fact that phase reversing waves that point toward each other represent negativities and phase reversing waves that point away from each other represent positivities from the basic convention of "negative-up," this simple pair of facts (as depicted in Figure 4-22) is worth memorizing to speed the process of EEG interpretation once you have convinced yourself that it always holds true.

The "Isoelectric" Phase Reversal

So far, we have examined examples of a discharge's maximum intensity when that maximum occurs at the very point at which an electrode has been placed.

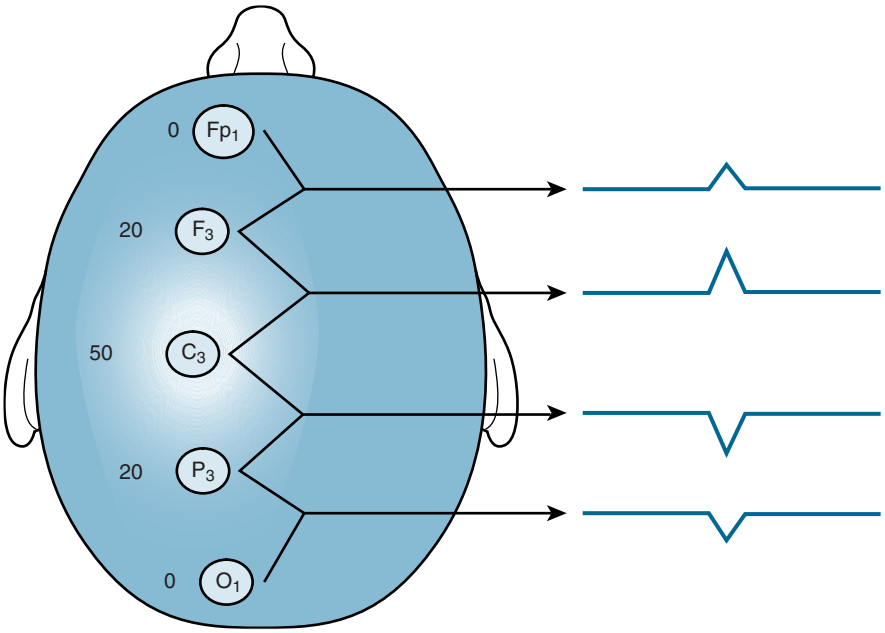


Figure 4-20 The diagram shows an example of a *positive phase reversal* with pens pointing away from each other and a maximum in C3. As in previous diagrams, dark areas are more negative than light areas. In this figure, there is a maximum positivity of 50 μV in the C3 electrode, the field of which drops off with increasing distance from the maximum point. The upgoing deflections in the first two channels indicate that the field is becoming more strongly positive traveling from Fp1 toward C3. The downgoing deflections in the third and fourth channels indicate that the field becomes less positive after traveling past C3. This combination of events marks C3, the point of phase reversal, as the point of maximum positivity in this bipolar chain.

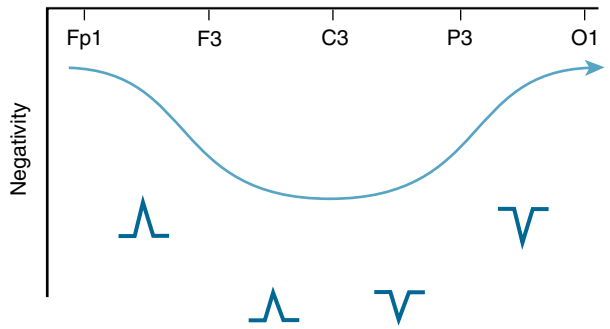


Figure 4-21 This figure shows a two-dimensional representation of a positive event with a maximum at C3 (compare to Figure 4-19). The upward deflection of the pens in the first two channels reflects the fact that, in the comparisons of Electrode 1 to Electrode 2 (Fp1 to F3 and F3 to C3), Electrode 2 is more positive, recalling that Electrode 1 being more negative causes the pen to go up. In the third and fourth channels, the comparison shows that Electrode 1 is more positive than Electrode 2, and so the pens go down.

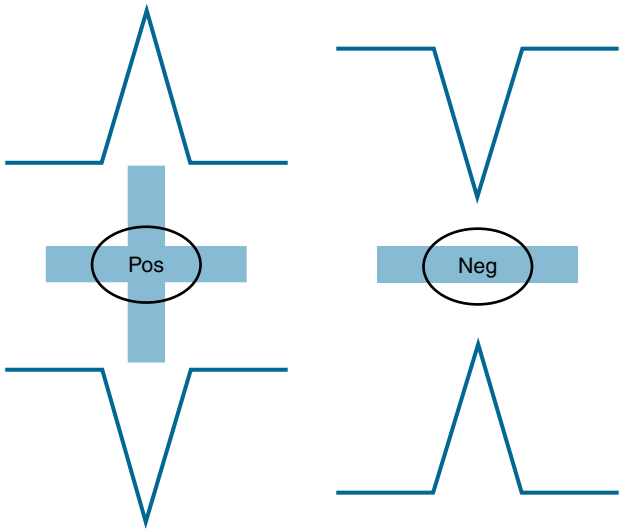


Figure 4-22 This diagram serves as a reminder that in bipolar montages, in the case of a phase reversal in which the pens point away from each other, there is an area of maximum positivity at the common electrode. Conversely, when the pens point toward each other, the phase reversal denotes the location of maximum negativity. The fact that there is only enough space to fit a minus sign between the points of the negative phase reversal but that there is enough space to fit a plus sign between the points of a positive phase reversal can serve as a memory device as to which configuration corresponds to which polarity.

Sometimes, however, a voltage maximum may occur between two electrode positions, and it is worthwhile to consider how this can change the appearance of a phase reversal. Figure 4-23 shows just such an example. The maximum negativity of −50 occurs between the F3 and C3 electrodes. At the F3 and C3 electrodes, voltages of −40 are measured. In the resulting bipolar recording, the expected negative phase reversal is seen, this time between the first and third channels, with the spikes pointing toward each other in the case of a negativity, as expected. This time, however, because F3 and C3 have the same voltage, the second, intervening channel is flat. This occurs because F3 and C3 are isoelectric to one another (they have the same voltage), and because a comparison of electrodes measuring the same voltage would generate no pen deflection, it is no surprise that this intervening channel is flat. This example illustrates the general concept that a phase reversal is still "valid" even with an intervening flat channel.

In such examples of a phase reversal with an intervening isoelectric (flat) channel, it is tempting to conclude that the maximum always lies in between the two electrodes of the isoelectric channel, as depicted in Figure 4-24. Although this is often the case, other field topographies are possible. For instance, given these pen deflections, it could be that −40 μV is, indeed, the maximum and is shared by F3 and C3—the field may be "flat" between these two points as shown in Figure 4-25. In the case of this isoelectric type of phase reversal, as long as the two electrodes in question are fairly close to one another (such as F3 and C3), we generally assume that the true maximum is somewhere between F3 and C3, inclusive. In reality, we cannot know the true contour of the field between F3 and C3 based on the tracing at hand; if it were necessary to know more, we would have to place additional intervening electrodes. An example of an isoelectric phase reversal recorded from the brain in an actual EEG is shown in Figure 4-26.

Event Localization Using a Referential Montage

Referential montages compare an electrode placed over a brain area "of interest" to a reference point elsewhere on the head or body. The "electrode of interest" is almost always over a brain region and may also be referred to as the *active electrode*. The reference electrode, attached to INPUT 2 of the amplifier, is located at some other point (or points) on the body, sometimes near the brain but often at a distance from the brain. Examples of points chosen for the reference electrode include the earlobes, the skin over the mastoid processes (behind the ears), the nose, the chin, the Cz electrode, and the base of the neck. Another strategy for creating a reference electrode is to use the average of some or all of the brain or other electrodes as a virtual reference electrode. The comparative advantages and disadvantages of different reference electrode strategies is discussed in detail in the chapter on montages.

The *active electrode* is customarily attached to INPUT 1 of the amplifier, and the reference electrode is attached to INPUT 2. The strategy of this type of montage, in the best of all possible worlds, is to find an electrode pair in which INPUT 1 contains the brain activity of interest (plus, perhaps, some unavoidable amount of contaminating electrical noise), and INPUT 2 contains only the electrical noise that is contaminating INPUT 1 but none of its brain activity. With this type of pairing, the result of subtracting INPUT 2 from INPUT 1 is a cancellation of the electrical noise, leaving a pure trace

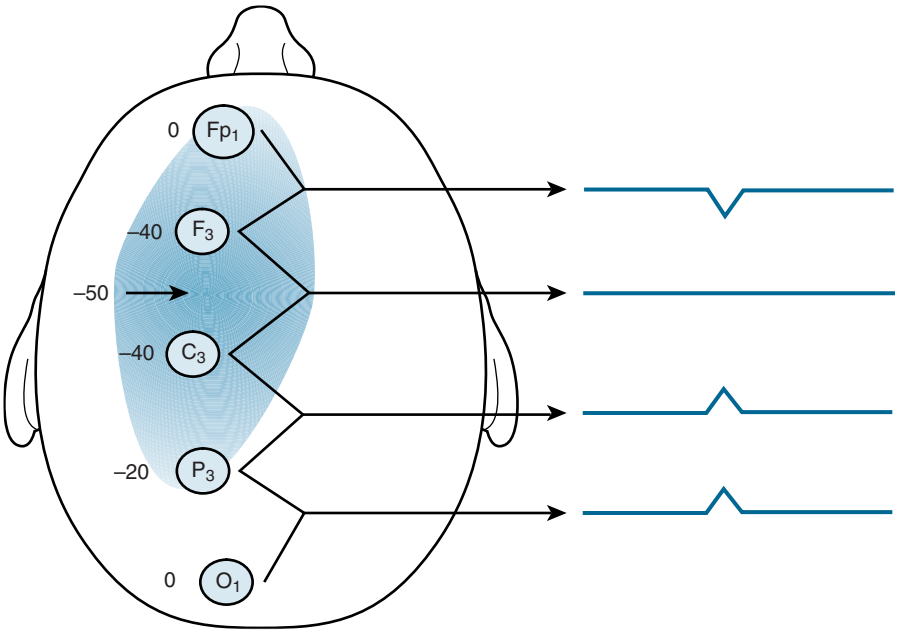


Figure 4-23 An *isoelectric phase reversal* is shown. In this example, the point of the *measured* maximum negativity of −40 μV is shared by the F3 and C3 electrodes. This diagram implies that the true maximum negativity of −50 μV lies between the F3 and C3 electrodes and is not directly measured since there is no electrode between F3 and C3. The resulting trace on the right side of the figure shows an example of an "isoelectric phase reversal." Here, the location of the maximum is actually marked by a flat line in the second channel (F3-C3) because those two electrodes are measuring the same voltage (−40 μV) and are said to be isoelectric. The nature of the phase reversal can be appreciated by the phase change between the first and the third channels.

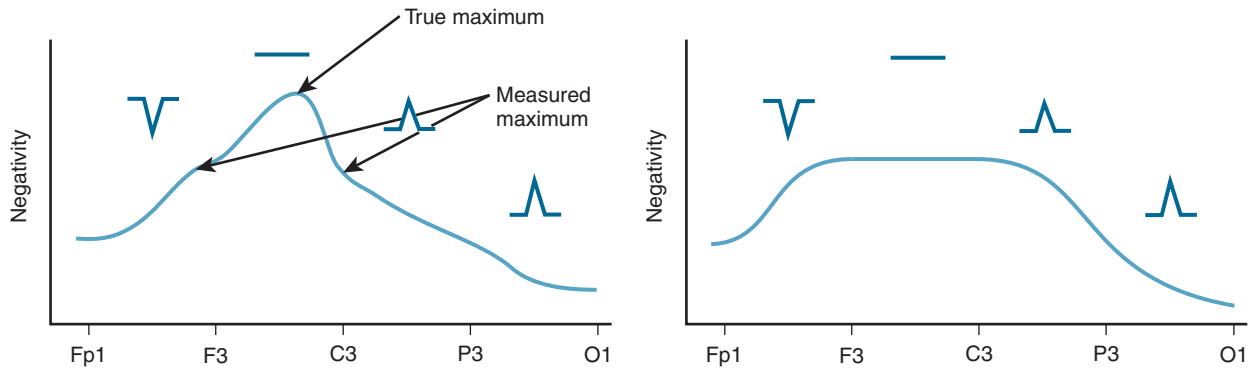


Figure 4-24 Two of the many possible shapes of a voltage gradient that could account for the type of isoelectric phase reversal shown on the right side of Figure 4-23. Panel A shows a gradient in which the true voltage maximum is actually located in between the isoelectric electrodes (F3 and C3). Panel B shows another possibility: that there is a plateau of maximum voltage between the two electrodes. If it were necessary to distinguish between these and other possibilities, additional electrodes would have to be placed between F3 and C3.

of the brain activity picked up by the active electrode that is attached to INPUT 1. This is an example of the ideal active electrode–reference electrode pair that is strived for in a good-quality referential recording. For the purposes of the idealized examples in this chapter, we assume ideal, noise-free active and reference electrodes that yield electrically noise-free recordings.

In some ways, the setup of referential montages makes them easier to interpret. Despite their many advantages, however, referential montages are often not the easiest montages to read. It is useful to compare the relative advantages and disadvantages of the referential technique to its cousin bipolar technique as we discuss them.

We now reconsider some of the same examples used earlier, starting with the simplest case of the highly focal negative spike in C3. Figure 4-27 shows the result of comparing each electrode in the parasagittal chain to a reference: Fp1-ref, F3-ref, C3-ref, P3-ref, and O1-ref. In these examples, the "ref" electrode is a hypothetical neutral reference electrode with a voltage of zero. Because in this example the spike's field does not extend to the adjacent electrodes, there is no voltage difference between those electrodes surrounding C3 (Fp1, F3, P3, and O1) and the reference. C3, in contrast, is more negative than the reference, causing the pen to deflect upward (first electrode more negative—pen up!). This results in an EEG with four flat lines representing the four surrounding neutral electrodes and one visible spike generated by the C3-ref channel. There is a convenient simplicity to the result displayed in a referential montage: the pen only moves in a

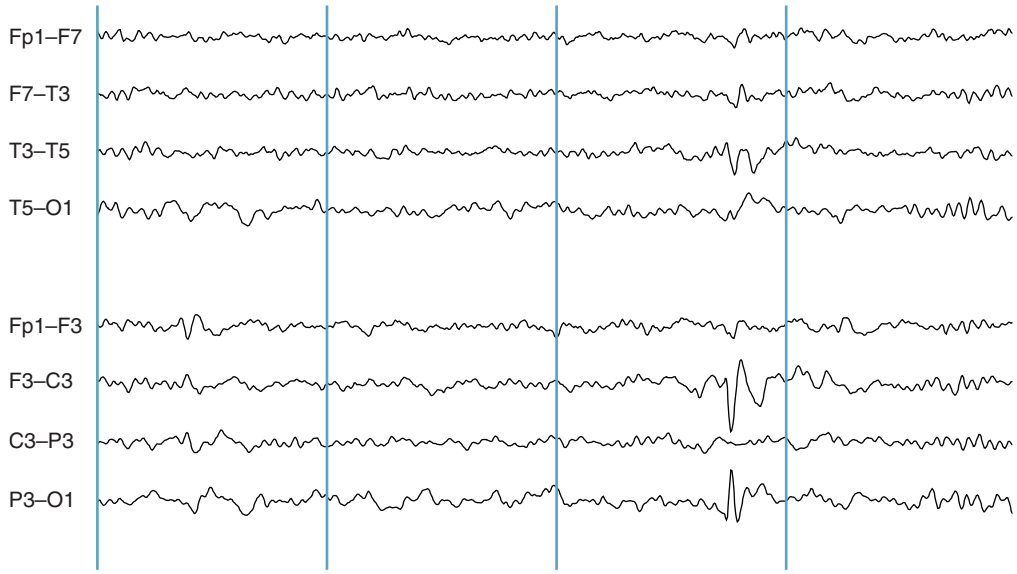


Figure 4-25 A nearly isoelectric phase reversal is seen surrounding the C3-P3 channel, which is nearly flat. This nearly flat channel suggests that C3 and P3 are of nearly the same voltage. The downward deflection in F3-C3 indicates that C3 is more negative than F3. The upward deflection in P3-O1 indicates that P3 is more negative than O1. All of these relationships can be seen when examining Figure 4-26, which shows the same spike displayed in a referential montage.

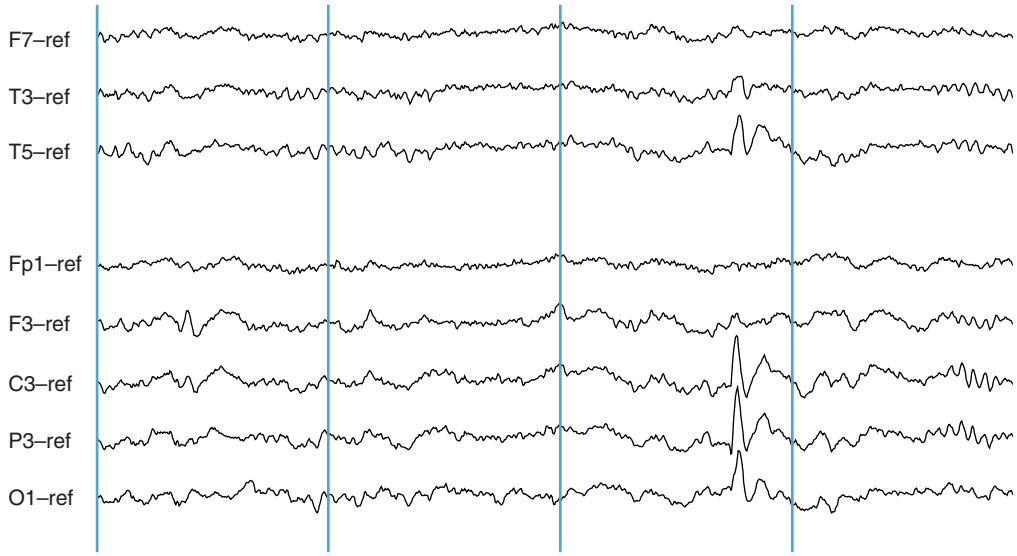


Figure 4-26 The same spike as was shown in Figure 4-25 is now displayed in a referential montage. As could be predicted by its appearance in the bipolar montage, C3 and P3 are the areas of maximum negativity and are of nearly the same voltage (F3-ref and P3-ref are of the same height). Fp1 and F3 are neutral and the field of the discharge's negativity spreads a small amount to O1.

channel that corresponds to an electrically active electrode. This is different from the same example we saw in the bipolar chain illustrated in Figure 4-17 in which a single electrode's activity caused the pens to move in two channels.

Next we examine a −50-μV spike in C3, the field of which spreads to the adjacent electrodes, F3 and P3. Figure 4-28 shows the output of a referential montage for a spike with the same field as was illustrated in Figure 4-18 in a bipolar montage. Once again, the output seems relatively simple, with a −50-μV deflection seen in the C3-ref channel and lower amplitude deflections seen in the flanking channels, F3-ref and P3-ref, reflecting the fact that the spike's field also involves those electrodes, although to a lesser extent. All of the deflections are upgoing because all of the active electrodes are relatively negative when compared with the neutral reference. The height of the deflection is proportional to the strength of the field in microvolts at each point.

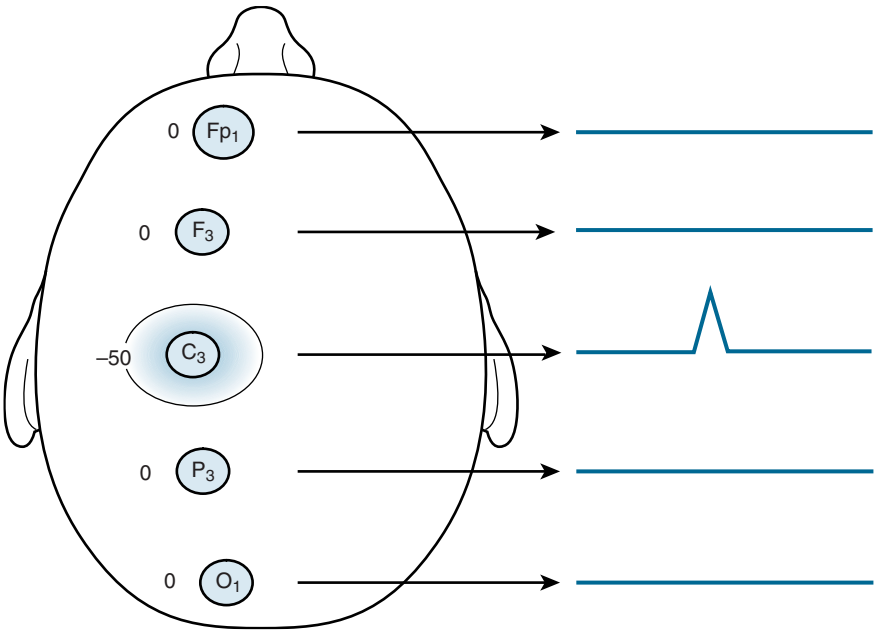


Figure 4-27 A highly focal −50–μV spike limited to the C3 electrode is displayed with a referential montage. The five channels correspond to Fp1-ref, F3-ref, C3-ref, P3-ref, and O1-ref, where *ref* represents an idealized, electrically neutral reference electrode. Because C3 is more negative than the reference electrode, the pen deflects upward. The other pens are silent because the surrounding electrodes are neutral.

Finally, we look at the example of a 50-μV "positive spike" in C3 with a field that includes F3 and P3. Figure 4-29 shows the EEG corresponding to such a spike using a referential montage. The main difference between this example and the previous one is that the pens go down, because the active electrodes are now more positive than the neutral reference electrode. Again, the relative heights of the spike in each channel correspond to the relative voltages at each electrode in the example. In a referential montage, knowing the height of a wave in millimeters and the sensitivity setting of the amplifier makes measurement of a wave's absolute voltage a matter of simple multiplication (see Figure 4-30).

Comparison of the Bipolar and Referential Recording Techniques

According to the descriptions given earlier, the technique of the referential montage has multiple advantages over the bipolar montage technique: each channel includes only one "electrode of interest" or active electrode. Compare this to the more complex situation with bipolar montages in which a deflection in

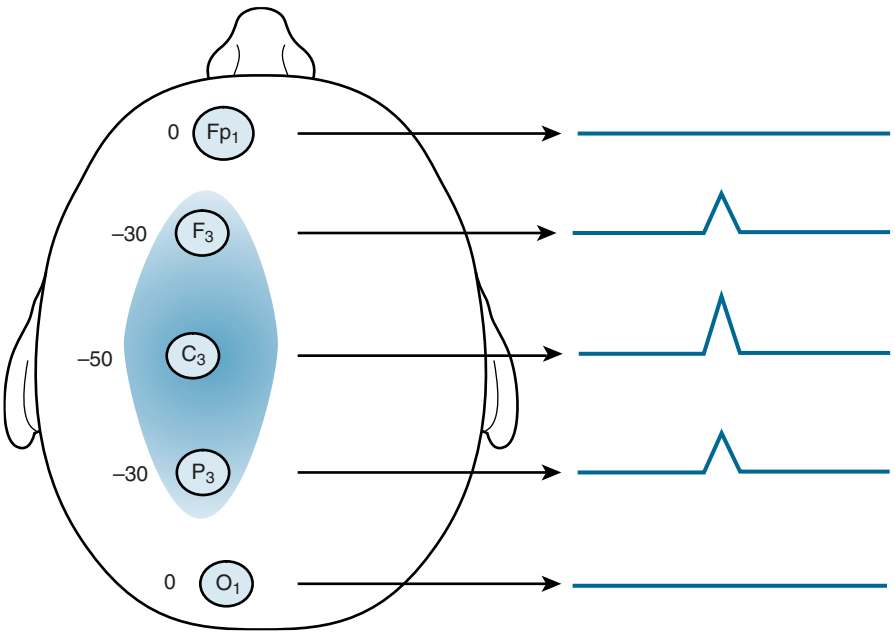


Figure 4-28 A −50-μV spike in C3 with a field extending to F3 and P3 viewed in a referential montage. The C3-ref channel deflects an amount corresponding to −50 μV, just as it did in Figure 4-27. Because the field involves F3 and P3 in this example, deflections corresponding to −30 μV are seen in F3-ref and P3-ref. Note that the spike amplitude in these channels is only 60% that of the C3-ref channel, reflecting the diminishing field in F3 and P3.

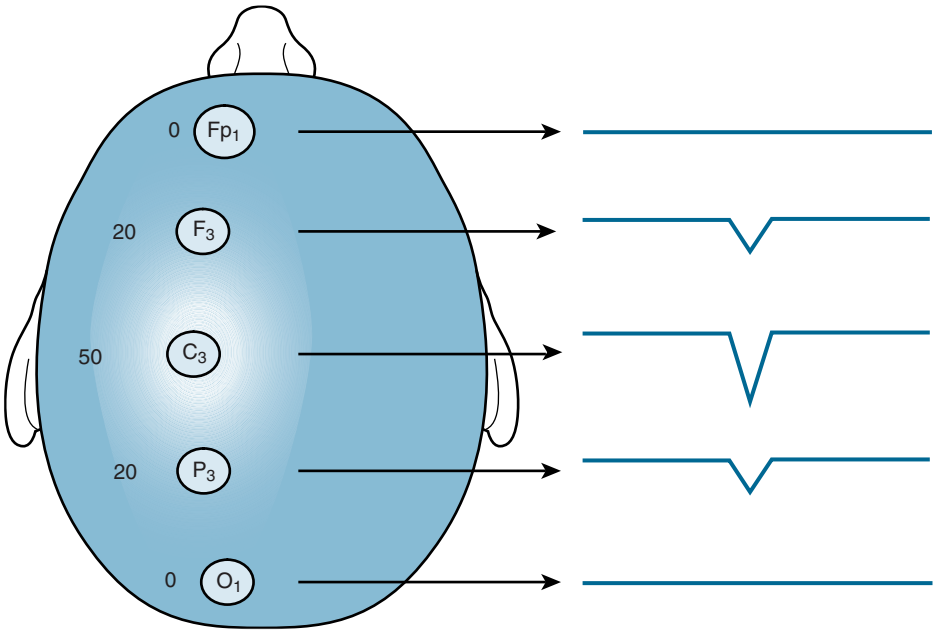


Figure 4-29 A 50-μV "positive spike" in C3 with field extending to F3 and P3. Because the active scalp electrodes are more positive than the neutral reference, the pens go down. The biggest pen deflection marks the maximum of the spike's field at C3.

the Fp1-F3 channel could indicate an event involving only Fp1, only F3, or both Fp1 and F3, but to different extents. The voltage of the event in a referential channel is directly proportional to the height of the wave. This is not necessarily true in bipolar montages, in which the point of maximum voltage can even be a flat channel, as we have seen in the example of the isoelectric phase reversal shown in Figure 4-23.

Why, then, does it often seem easier to read bipolar montages? The answer is related to the unpredictability of the assumption of the "clean" reference used in these examples. Ideally, we would like the reference to include only the exact same noise that is present in the active electrode. In practice, references often have noise of their own, sometimes to the point that they obliterate important signal information in the active electrode rendering the recording unreadable. This is not to say that, sometimes, a near-ideal reference cannot be found. With different patients during different parts of a given EEG study, depending on the patient's behavior and other factors, the ability to make a clean referential recording varies greatly. Different reference electrode locations can yield large differences in the quality of the results. Each recording technique has its own advantages and disadvantages (Table 4-1).

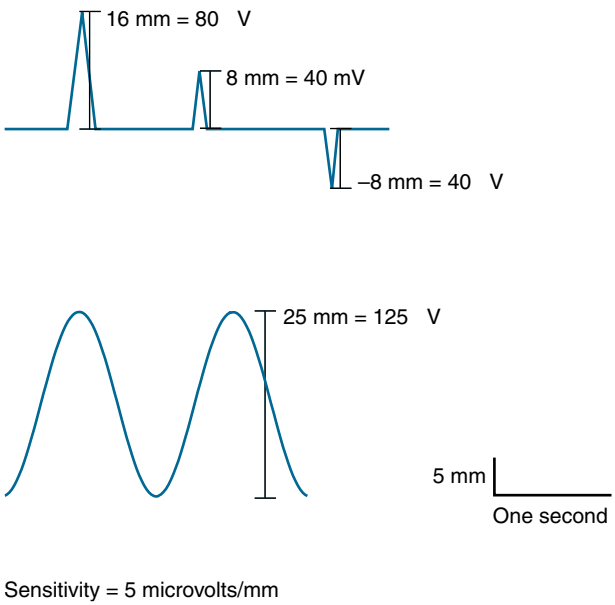


Figure 4-30 The first step in ascertaining the voltage of a wave in an EEG trace is knowing the recording sensitivity used, stated in microvolts per millimeter. The sensitivity tells the reader how many microvolts of voltage each millimeter of pen deflection represents. In this example, the sensitivity is 5 μV/mm. In the top trace, the measured heights from the baseline of the spikes are 16 mm, 8 mm, and −8 mm, respectively. The relationship of millimeters to voltage is found by a simple multiplication 16 mm × (5 μV/mm) = 80 μV. Voltage measurements of sinusoidal waves as shown in the lower trace are made by measuring the peak-to-trough height of the waves, as shown.

Predicting the Appearance of the Bipolar Montage on the Basis of the Referential Montage

The following section consists of a set of exercises designed to aid the reader in visualizing how various EEG waves might appear differently depending on whether they are displayed in a bipolar or a referential montage. The ability to complete these exercises correctly implies a good understanding of the differences between the two montage types and how each type works. The reader may choose to solve the 14 montage problems, each shown on the following odd-numbered pages. The solutions are shown on the overleaf of each page.

We will start by looking at events in a referential montage and determine what their appearance would be in a bipolar montage. The examples used focus on a single chain of five electrodes, extending from the left frontopolar region to the left occipital region: Fp1, F3, C3, P3, and O1. This chain of electrodes is referred to as the left parasagittal chain. Again, to keep things simple, a single spike is used for these examples. In reality, the same line of reasoning applied to this spike can also be applied to any type of wave, such as sharp waves or slow waves. You may wish to look at the figures whose captions start with "Question" first (starting with figure 4-31) to attempt to solve the conversion problems before reading the narrative that follows which includes an explanation of the answers.

Figure 4-31 shows a simple event localized to a single electrode, C3. The appearance of this event in the bipolar montage can be predicted by imagining a series of subtractions. First, it should be noted that in a five-electrode chain, the referential montage displays five channels (Fp1-ref, F3-ref, C3-ref, P3-ref, O1-ref), but the bipolar montage solution will only include four channels, each channel representing a pairing of electrodes (Fp1-F3, F3-C3, C3-P3, P3-O1). For the purpose of solving these problems, the height of each wave can be measured in any unit (inches, millimeters, etc.)—the graphical answers are independent of the unit of measure used.

To determine what the appearance of the discharge in Figure 4-31 will look like in a bipolar montage, the value of the first bipolar channel is determined by finding the difference between the magnitudes of the discharge in Fp1 and F3. Because the value of each is zero, the difference between the two channels is also zero, and the resulting Fp1-F3 channel is flat (see figure 4-34). The situation is different when determining the appearance of the F3-C3 channel. In this example, on the basis of an examination of the referential montage, F3 is zero, but C3 is upgoing by some amount (implying that C3 is negative by some amount). Because in the F3-C3 channel Electrode 1 is zero and Electrode 2 is negative, the pen goes down. Why is this? Because, as discussed earlier, according to the convention, when the first input (F3) is "more positive" than the second input in the pair (C3), the pen goes down. Another way to determine the pen direction is that when a subtraction of *Electrode 1* − *Electrode 2* is performed, the result will be a positive value, implying that the pen goes down. The most succinct way of stating this relationship is "F3 is 'more positive' than C3, so the pen goes down." The words "more positive" are in quotes because F3 is not, strictly speaking, positive.

Moving to the next electrode pair, C3-P3, now the pertinent comparison is a negative voltage in C3 minus a potential of zero in P3. C3 is more negative than P3, so the pen deflects upward. The final comparison is between P3 and O1, both of which have a zero value, so the comparison yields a flat line, reflecting a difference of zero. This succession of comparisons yields the appearance of Figure 4-34, the solution to the problem. This figure shows a typical-appearing phase reversal for a localized, negative surface event.

Table 4-1 The Comparative Advantages and Disadvantages of Bipolar and Referential Recording Techniques

	Bipolar Montages	Referential Montage
Locating the electrical maximum	Maximum point is found by determining the point of phase reversal—the electrode in common between the two phase reversing channels is the point of maximum.	Maximum point is found by locating the channel with the largest deflection. The active electrode in that channel is the point of maximum. Phase reversal has no meaning.
Measuring wave voltage	Absolute voltage cannot be accurately determined by measuring wave amplitudes; only relative voltages among electrodes can be determined.	Absolute voltage of a wave can be determined by measuring the wave's height, assuming neutral reference.
Number of channels in EEG output	There is one fewer channel than the number of electrodes in a chain.	There is the same number of channels as the number of electrodes in a chain.
Appearance of negative and positive polarities	Negativity is marked by phase reversal with deflections pointing toward each other. Positivity is marked by phase reversal with deflections pointing away from each other.	Assuming a neutral reference, negativity is marked by upgoing deflection. Positivity is marked by downgoing deflection.
Drawbacks of technique	Absolute voltages are difficult to ascertain. Highest deflections do not necessarily mark point of maximum voltage.	A noisy reference adds noise to all channels.

The next example, shown in Figure 4-32 shows a discharge that resembles the previous example, a negative event with maximum in C3, but this time there is a field surrounding the C3 electrode. Examination of the figure reveals that the intensity of the field in C3 is exactly twice the intensity measured in the F3 and P3 electrodes. As a result, when going down through the electrode pairs, if F3 and P3 measure 5 "units" in height and C3 measures 10 "units" in height, the Fp1-F3 will reflect a 5-unit difference (and display a wave that is 5 units in height going downward, because Electrode 1 is more positive than Electrode 2). Likewise, F3-C3 will deflect the exact same amount in the same direction, because the difference between that electrode pair is the same, again with the first electrode being more positive than the second. For that reason, the first two channels of the bipolar solution are identical downward deflections, the height of which is exactly the same as

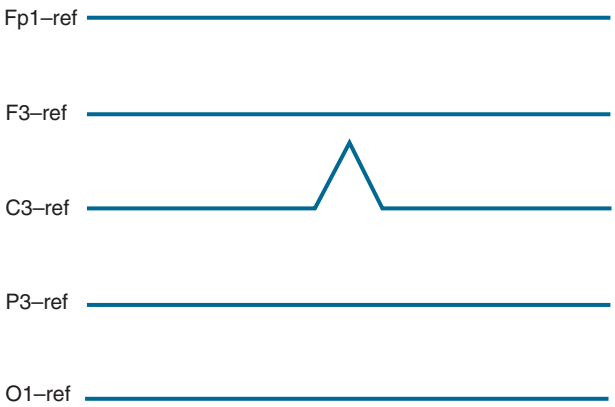


Figure 4-31 Question 1. Predict how this discharge, displayed in a referential montage, will appear in a bipolar montage (answers to all questions appear on the subsequent page). The discharge consists of a simple, highly focal spike with a pure negativity in C3. The adjacent electrodes are inactive, implying that there is no gradient or gradual "falloff" in voltage in the surrounding electrodes.

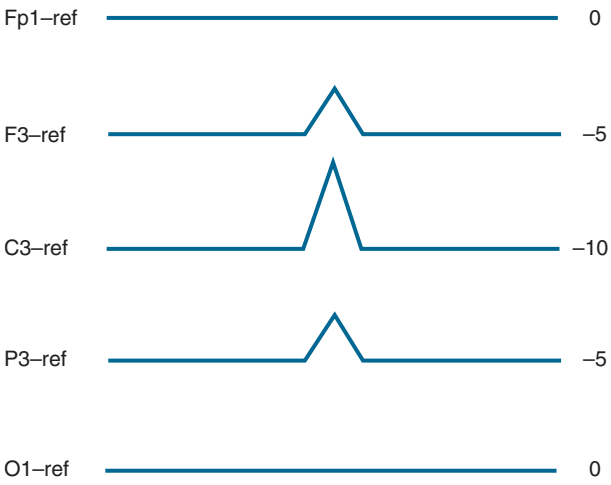


Figure 4-32 Question 2. Predict how this discharge, displayed in a referential montage, will appear in a bipolar montage. Like the previous example, this tracing shows a focal spike in C3, but this time with a larger field surrounding C3 as evidenced by the smaller upward deflections in F3 and P3. Note that the field dissipates in an even fashion with distance from C3. Absolute voltage measurements are given on the right side of the figure.

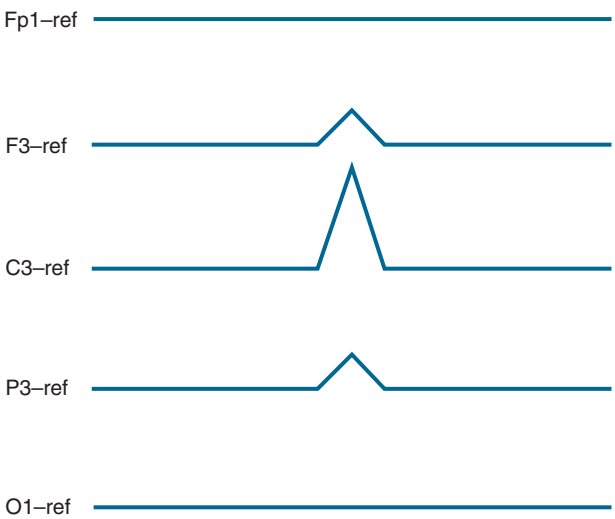


Figure 4-33 Question 3. Predict how this discharge displayed in a referential montage will appear in a bipolar montage. This discharge is similar to the previous example except for the fact that voltage falls off more abruptly between C3 and its adjacent electrodes, F3 and P3. This steep gradient around C3 should be reflected in the appearance of the corresponding bipolar recording (shown on the next page).

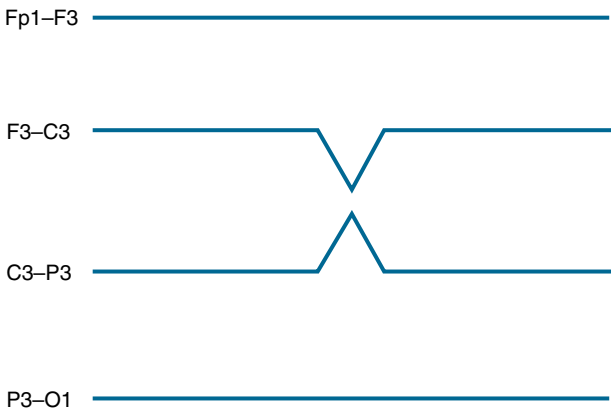


Figure 4-34 Answer 1. The appearance of the same simple C3 spike shown on the previous page displayed in a bipolar montage. The flat lines in Fp1-F3 and P3-O1 reflect the fact that there is no electrical gradient between the outermost channels (Fp1 and F3, P3 and O1). Although such highly focal discharges involving only a single electrode may, indeed, be of cerebral origin, an electrode artifact may also produce this highly focal appearance.

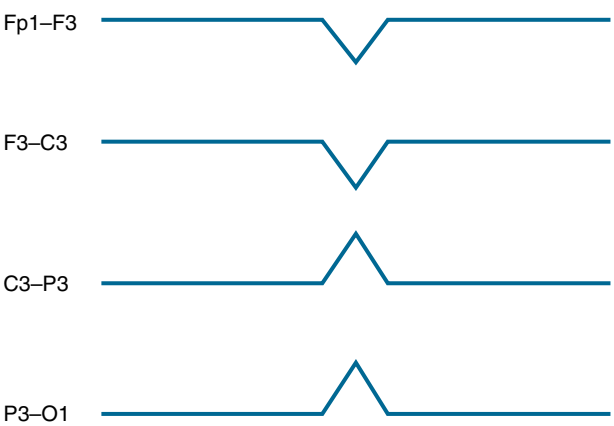


Figure 4-35 Answer 2. In the referential montage shown in Figure 4-32 the height of the wave at C3 is evidence that the maximum negativity is located in that electrode. In the bipolar montage, C3's maximum negativity manifests as a phase reversal at C3 in this figure. The similar deflections seen in the outer channels, Fp1-F3 and P3-O1, reflect the fact that there is a gentle and steady gradient of decreasing negativity with increasing distance from the C3 electrode. This is one of the most common configurations seen for focal negative events arising from the brain. Deflections in all four channels in this figure confirm the presence of a field extending away from the discharge's maximum.

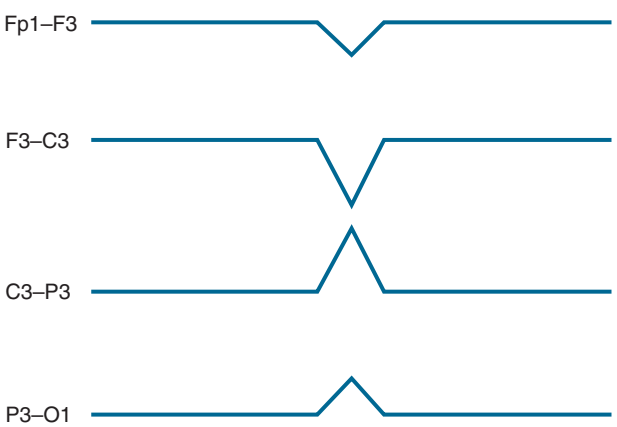


Figure 4-36 Answer 3. A representation of the discharge seen in Figure 4-33 is shown in a bipolar montage. The steep drop-off in voltage between C3 and its neighboring electrodes is manifested by higher deflections in F3-C3 and C3-P3 as compared to the previous example. The phase reversal seen in C3 still denotes this location as the point of maximum negativity.

the difference between the adjacent electrodes' voltage. The third bipolar channel, the C3-P3 comparison, has the same magnitude of difference as the previous comparison, but now because C3 is more negative than P3, the pen goes up the same number of units. This flipping of the pen direction (phase reversal) now that the voltage trend has changed (from successive electrodes becoming more negative to successive electrodes becoming more positive) defines the point of maximum negative voltage. This change in voltage trend causing the wave to flip its phase is the essence of the phase reversal. The phase "reverses" because, as the parasagittal chain is explored from front to back, the trend of successive voltage comparisons is no longer one of increasingly negative measurements but, instead, one of increasingly positive measurements. Finally, the last two channels, P3 and O1, have the same five unit difference with P3 more negative than O1, yielding the final 5-unit upgoing deflection. Figure 4-35 shows the expected phase reversal at C3.

The discharge shown in Figure 4-33 is similar to the preceding example, with a negative discharge seen in C3 and a diminishing field seen in F3 and P3. The example differs from the previous one in that the intensity of the

discharge drops off more abruptly from C3 to F3 and P3 (the gradient is steeper between C3 and F3 and between C3 and P3). When this type of field is shown in the bipolar montage, the location of the phase reversal at C3 has not changed (see Figure 4-36). The fact that the heights of the discharge in the second and third channels (F3-C3 and C3-P3) of Figure 4-36 are much higher than those in the outer channels (Fp1-F3 and P3-O1) reflects the fact that the gradient of the voltage drop-off immediately around C3 is steeper in this than in the previous example. This is analogous to the example of a mountain peak that is quite steep near its summit and less steep near its base.

Figure 4-37 shows what is, again, a very similar discharge with maximum negativity at C3. This time, the field is still strong in the adjacent electrodes, F3 and P3. The steep drop-off in voltage only occurs farther away from the C3 maximum of the discharge, between F3 and Fp1 and P3 and O1. When this discharge is displayed in a bipolar montage, the phase reversal again remains at C3, just as expected because this is still the point of maximum but second and third channels show low amplitudes, while the first and fourth channels show high amplitudes (see figure 4-40). This example provides an excellent demonstration of one of the relative weaknesses of the bipolar recording technique. A survey of the discharge in Figure 4-40 shows the highest (and most eye-catching) wave amplitudes near Fp1 and O1, the outer ends of the chain. The eye is not particularly drawn to the middle two channels with their lower amplitude deflections, yet these low-amplitude deflections mark the true maximum of the discharge. Why does this occur? As the reader well understands by now, the height of a wave in the bipolar montage does not represent the absolute voltage, but rather the rate of falloff of the voltage or the *voltage gradient* between two electrodes. The point of maximum is marked by the position of the phase reversal, even if the deflections in the bipolar montage do not have particularly high amplitudes in that location. This observation helps emphasize the concept that, in bipolar montages, even when of very low voltage, phase reversals mark the point of maximum, whereas in referential montages, it is the greatest wave height that marks the point of maximum.

Figure 4-38 shows a discharge with a maximum shared between the F3 and C3 electrodes. Note that, on the basis of this configuration, it is possible that the discharge is "flat" in the region between the two electrodes, or, alternatively, there may be an even higher maximum point somewhere between the F3 and C3 electrodes (a schematic of these possibilities was shown in Figure 4-24). The two possibilities cannot necessarily be distinguished from the information provided by the tracing, and it is not necessary to know which of the two possibilities is the case to solve this problem. A discharge with equal maximum points appears as a particular type of phase reversal in a bipolar montage, as is shown in Figure 4-41. This pattern may be called an *isoelectric phase reversal* because the phases reverse around a flat, isoelectric channel (F3-C3). Here again, the interpreter must be aware that, despite the fact that this channel is flat, because the phase of the discharges reverses in the surrounding channels, this flat line marks the region of maximum voltage.

The wave pattern illustrated in Figure 4-39 represents a commonly encountered type of electrical event seen in referential montages. This example consists of a sharp wave with the exact same intensity across the whole parasagittal electrode chain (and perhaps across the whole scalp). Because there is no potential difference between any of the electrodes in this chain, the resulting bipolar tracing (see Figure 4-42) shows flat lines in each of its four channels—the apparent sharp wave cancels out completely in each of the four comparisons. Although it is not impossible for a discharge to be of similar voltage across a wide region of the head, a discharge *that arises from the brain* with exactly equal voltage across all scalp electrodes is rarely, if ever, encountered. The most common explanation for this type of pattern is electrical noise from an external source. Because of the noise's external origin, it is possible for the noise signal to be of equal voltage all across the scalp. By contrast, discharges of cerebral origin almost always vary in voltage across the scalp, and therefore are almost always discernible on bipolar montages.

This tendency to cancel out common noisy activity is what gives bipolar montages their "cleaner" appearance and makes them appear to be easier to read. The efficiency with which bipolar montages can cancel out external noise is one of the major advantages of the bipolar recording technique. Less commonly, this type of cancellation of electrical events that are widely represented across the head can be a disadvantage. Occasionally, genuine cerebral activity (e.g., sharp waves or slow waves) can have a wide field across the scalp. Displaying such waves in a bipolar montage can result in a large amount of cancellation of such potentials, leading the reader to underestimate the voltage of the events. More often, however, the reader is happy that external noise sources are cancelled with the bipolar technique because this renders the underlying true electrocerebral activity easier to appreciate.

Figure 4-43 shows a downgoing spike, implying that the spike has positive rather than negative polarity. There is a surrounding field consisting of smaller positivities in F3 and C3. The appearance in the bipolar montage seen in Figure 4-46 is that of the classic "positive phase reversal" in which the phase-reversing waves point away from each other. Although epileptiform discharges more commonly show a negative charge at the scalp, occasionally they will show positive polarity, as in this example. Many nonepileptiform EEG waves may also manifest positive polarity, such as vertex waves of sleep, which can have both positive and negative components.

Figure 4-44 shows another frequently encountered waveform. In this example, there is a strong positivity in Fp1. The field of the positivity spills into F3, but with less intensity. The remaining three electrodes (C3, P3, and O1) do not detect the discharge. The same tracing in the bipolar montage shows one of the most commonly seen deflections in the EEG of awake individuals. The Fp1-F3 channel goes down, reflecting the fact that Fp1 is "more positive" than F3. Likewise, F3-C3 shows a

downgoing wave because F3 is more positive than C3, although the difference is not as great. The final two channels (C3-P3 and P3-O1) are flat because C3, P3, and O1 are all neutral.

The wave illustrated in this figure is consistent with an *eyeblink artifact,* which is common in the awake EEG (described in more detail in Chapter 6). Its appearance in the bipolar montage is illustrated in Figure 4-47. Because there is a net positive charge on the front of the globe of the eye, when the eyes are closed, the eyes bob upward (the so-called Bell's phenomenon), causing a momentary positivity in the most anterior electrodes.

The pattern seen in Figure 4-45 suggests the familiar appearance of the posterior rhythm seen in the occipital lobes. The visual subtraction of sinusoidal curves such as those seen in this example is somewhat more challenging than subtracting the simple spikes we have been looking at so far, but the principles are the same. As we shall see, this example gives a surprising result based on the fact that, in this patient, the posterior rhythm is completely synchronized in the P3 and O1 electrodes. Figure 4-48 shows the expected flat channel in Fp1-F3 as both of these electrodes are inactive. A comparison of the flat F3 electrode to the lower voltage sinusoidal wave in C3 shows a mirror-image reflection of that wave, with the peaks and troughs flipped. The subtraction of P3 from C3 yields a similar result. As expected, at least mathematically, P3 and O1 cancel completely, and the P3-O1 channel is flat. This yields the paradoxical result of a posterior rhythm, strongest in the P3 and O1 channels, that is not seen at all in the most posterior channel (P3-01) of the bipolar recording. In practice, this situation can occur in individuals in whom the posterior rhythm is highly synchronized and "in phase" in the posterior channels (the peaks and troughs of the waves in P3-ref and O1-ref line up perfectly with one another). In most patients, however, the posterior rhythm representations in P3 and O1 are somewhat out of phase with each other, and the subtraction of one from the other does yield a recognizable sinusoidal wave. Imagine that the wave in O1-ref were shifted one half wavelength to the right with respect to the wave in P3-ref. In that case, the rhythm seen in the P3-O1 channel of the bipolar montage would, indeed, have very high voltage.

Predicting the Appearance of the Referential Montage Based on the Bipolar Montage

The reverse of the problems we have been working thus far, predicting how a page will appear in the referential montage on the basis of its appearance in the bipolar montage, is associated with some unexpected challenges. The first such problem we will consider is shown in Figure 4-49, which shows a simple negative phase reversal in a bipolar montage. What will this look like in a referential montage? Going through each of the four channels, we develop a set of constraints: Analysis of the bipolar tracing tells us that because Fp1-F3 is flat, Fp1 and F3 must be of the same voltage. By the same line of reasoning, P3 and O1 must be of the same voltage. Further, because C3 is more negative than F3 by the same amount that C3 is more negative than P3 (because the height of F3-C3 is the same as, but opposite in polarity to, the height of C3-P3), the voltage of Fp1, F3, P3, and O1 must all be the same. C3 must be more negative than those four electrodes by an amount equivalent to the heights seen in F3-C3 and C3-P3. Any solution that meets these constraints is potentially valid. Figure 4-50 shows three possible solutions to this problem.

Note that all three proposed solutions are equally correct from a "mathematical" point of view fitting the constraints implied by the bipolar montage: Fp1 = F3 = P3 = O1, and C3 has a "more negative" voltage than those four electrodes. However, certain of these solutions are more likely to be found in clinical EEG than others. Indeed, the top trace showing the pure negative discharge in C3 is the most likely to occur in actual EEG recordings. The middle trace, in which there is a negativity seen across all electrodes but strongest in C3, remains a possible solution. It is less attractive from the physiological point of view because it requires Fp1, F3, P3, and O1 all to be of the *exact same voltage*, but still within the field of the negative discharge—a relatively unlikely occurrence. The bottom trace of figure 4-50 in which the surrounding electrodes are mildly positive but equipotential and C3 is mildly negative is also a mathematically sound solution, but even less biologically plausible for similar reasons. It would require a central negativity surrounded by a positivity of precisely constant intensity (no gradient).

Differences in solving the conversion problem in the bipolar to referential direction include the fact that the bipolar trace has four channels but the solution, the corresponding referential trace, needs to have five channels. More important, when converting referential traces to bipolar traces, there was only a single correct solution. As was seen with the previous example, any given trace in a bipolar montage can correspond to an infinite number of possible solutions in the referential montage. The reason for this is akin to the fact that there is only a single solution to the problem: "5 − 3 = ?"—the answer must be 2. If we ask the "reverse" version of this question, "subtraction of what two numbers gives 2 as a result?" the answer is an infinite number of pairs, such as 5 and 3, 101 and 99, −6 and −8, and so on. This analogy holds up well because bipolar traces are analogous to a display of differences and referential traces to a display of "absolute values."

Figure 4-51 shows a similar negative discharge maximum in C3, but this time with a more gradual gradient of decreasing voltage surrounding it, as evidenced by the deflections in the outer channels (Fp1-F3 and P3-O1). The possible solutions shown in Figure 4-54 are all potentially correct because they all show the most negative voltage in C3 with voltages that are gradually decreasingly negative in the surrounding electrodes. The top and bottom traces, however, are the most biologically plausible with a negative event maximum in C3 and a gradient of decreasing negativity with increasing distance from C3. The middle trace is mathematically correct but biologically much less likely. Although in the middle trace the "maximum negativity" among

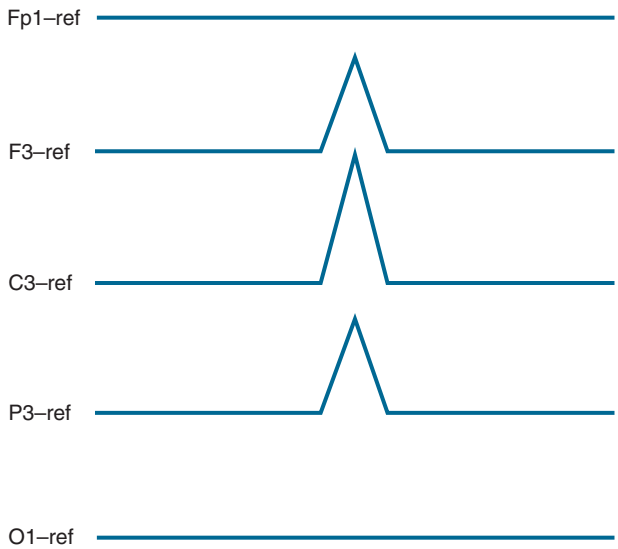


Figure 4-37 Question 4. Predict how this discharge, displayed in a referential montage, will appear in a bipolar montage. Again, the highest voltage is seen at C3, but now there is only a small drop-off in voltage in the adjacent electrodes, F3 and C3, and then a more abrupt drop-off of voltage in the outermost electrodes, Fp1 and O1.

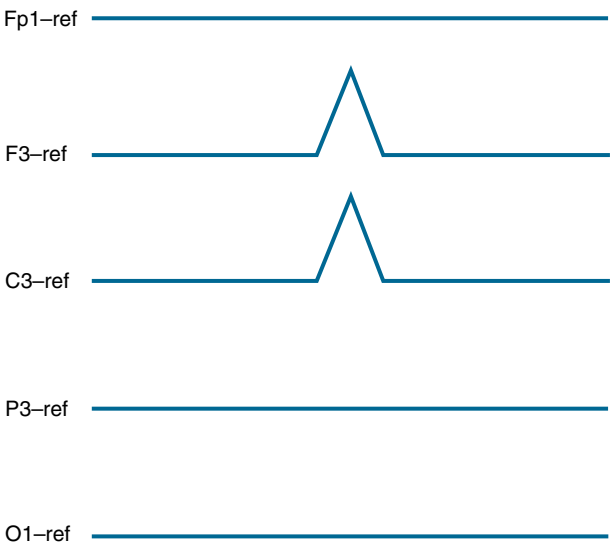


Figure 4-38 Question 5. Predict how this discharge, of equal voltage in F3 and C3 with surrounding electrodes quiet and displayed in a referential montage, will appear in a bipolar montage.

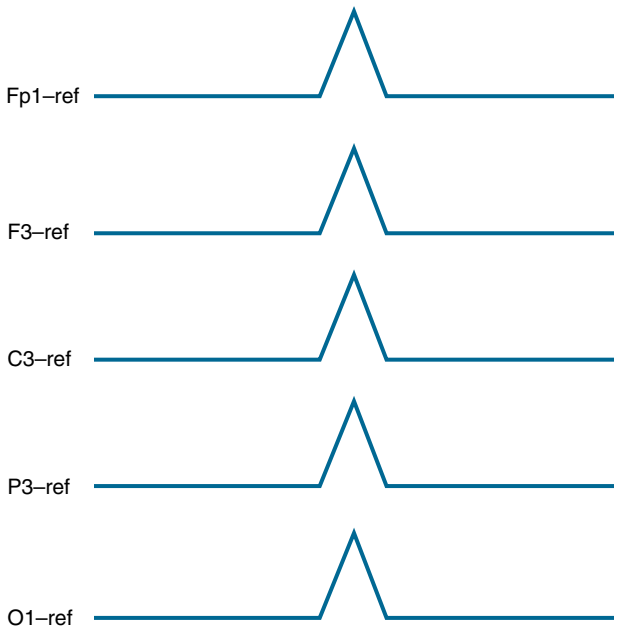


Figure 4-39 Question 6. Predict how this discharge, a negatively charged event that is spread evenly across the whole electrode chain, will appear in a bipolar montage.

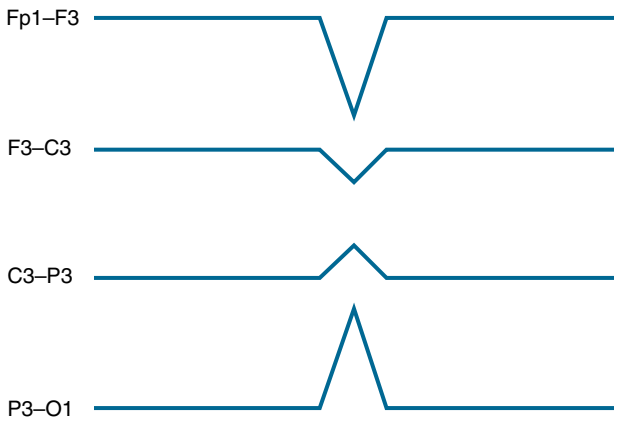


Figure 4-40 Answer 4. Note that in this display of the discharge in a bipolar montage, the waves with the greatest deflections, which happen to be in the outer channels, do not denote the discharge's maximum. Rather, these waves are tallest because the voltage drop-off happens to be steepest in these areas. As expected, the phase reversal in C3 still denotes the location of maximum negativity, even though the waves in these channels happen to be of lower amplitude than their neighbors.

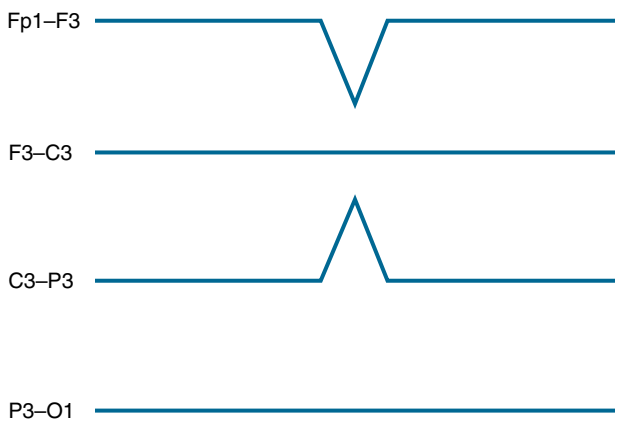


Figure 4-41 Answer 5. The discharge shown on the previous page manifests as an *isoelectric phase reversal* when displayed here in a bipolar montage. A flat channel is seen between the two phase-reversing channels. Because there is no voltage difference between the F3 and C3 electrodes (which share the maximum), there is an apparently paradoxical appearance where the point of maximum voltage is marked by a flat channel.

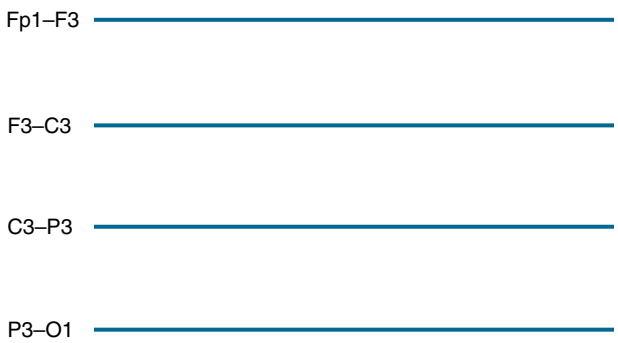


Figure 4-42 Answer 6. Because there is no gradient across any of the electrodes in this chain, all channels are flat in the bipolar montage. Although this result may appear to represent a loss of information, (after all, a spike did seem to be present in all the channels of the referential montage) the bulk of scalp events for which the voltage is the same across wide areas often reflect electrical noise from a distant source. Electrical activity of cerebral origin usually manifests a gradient (varying voltages) across the scalp. Thus the cancellation of such activity is one of the main advantages of bipolar recording techniques.

the electrodes remains in C3, this solution implies two concomitant positive events at each pole of the brain (frontal pole and occipital pole) having the exact same magnitude and gradient and a completely neutral area in the C3 region, a relatively unlikely occurrence.

The discharge in Figure 4-52 again shows a spike with maximum negativity in C3. In this example, however, the higher amplitude deflections in the two center channels, F3-C3 and C3-P3, imply a steeper gradient of voltage change with increasing distance from the C3 electrode. Figure 4-55 shows results that are similar to the previous solutions seen in Figure 4-54. Again, the top and bottom traces are the most likely correct solutions for the same reasons given for the previous example—they are more biologically plausible. Note that the relative heights of the waves in F3 and P3 are much less than the height of C3, reflecting the more abrupt drop-off in voltage with distance from the point of maximum seen in this example. The top and bottom traces are commonly encountered patterns for EEG events recorded on the scalp.

Figure 4-53 shows an example of a particular type of phase reversal, the *isoelectric phase reversal*, that was discussed earlier. In this type of phase reversal, waves reverse phase (in this case, changing from downgoing to upgoing), but there is an intervening flat channel. Figure 4-56 shows three mathematically correct solutions, although the top and middle traces are the most plausible of the three. The bottom trace is a possible solution, but it requires a positive event to occur in C3, P3, and O1 at the exact same voltage across a relatively large area. The lack of a change in voltage or gradient

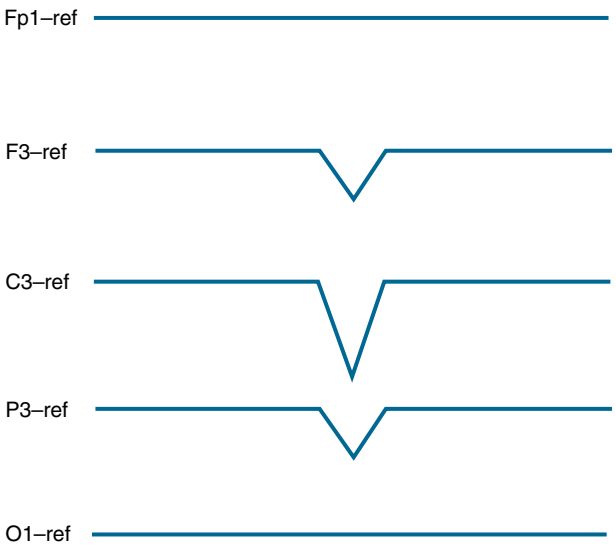


Figure 4-43 Question 7. Predict how this discharge, displayed in a referential montage, will appear in a bipolar montage. The figure shows a *positive* spike with maximum at C3 and with smoothly diminishing voltages at F3 and P3.

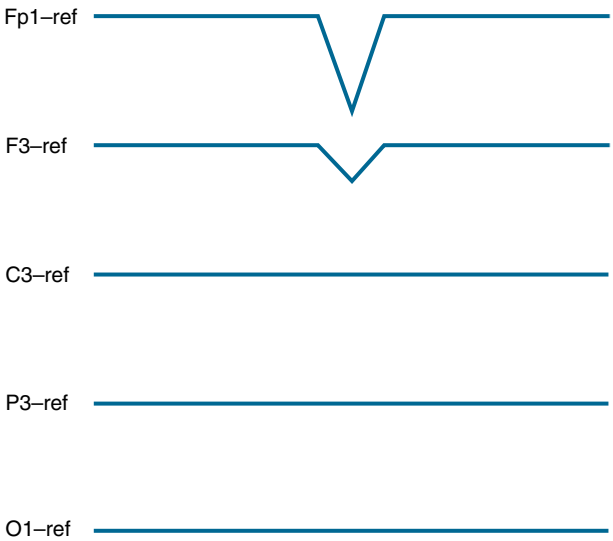


Figure 4-44 Question 8. This pattern, displayed in a referential montage, suggests a strong positivity in Fp1 with diminishing strength in F3 and no field in C3, P3, and O1. Predict its appearance in the bipolar montage.

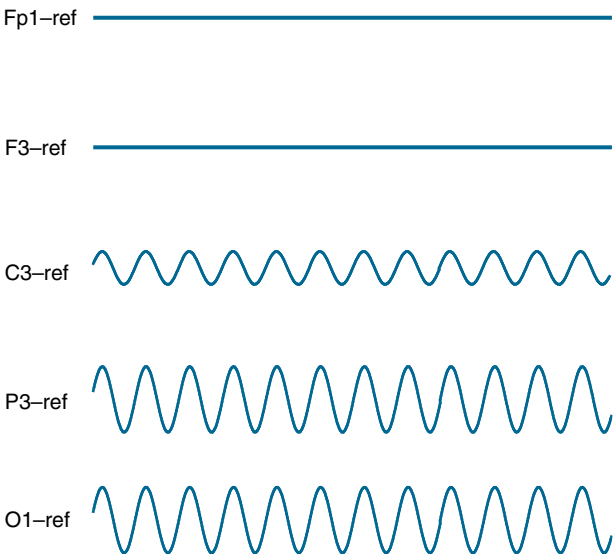


Figure 4-45 Question 9. Predict how this discharge, displayed in a referential montage, will appear in a bipolar montage. Sinusoidal waves are present in this recording, most prominently in the posterior two electrodes and at half the voltage in the C3 electrode. This pattern is similar to what might be seen as the posterior (occipital) rhythm in some patients. In this example, the P3 and O1 signals are perfectly in phase, which is not always the case in actual patient recordings.

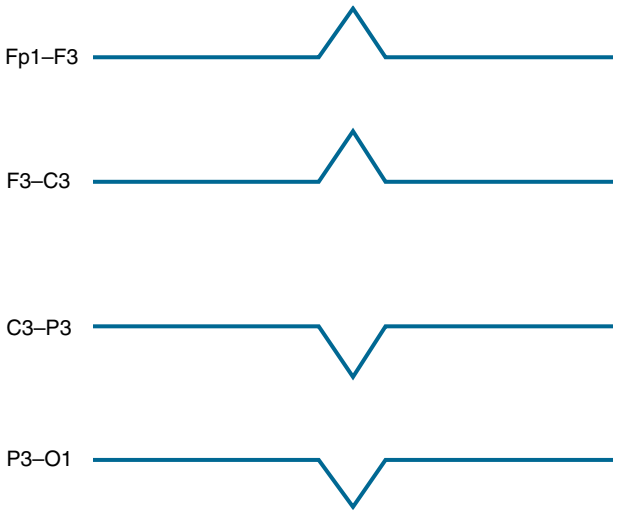


Figure 4-46 Answer 7. This figure shows the corresponding "positive phase reversal" with waves that point away from each other. Because the phases reverse at C3, this is the location of maximum positivity. The discharge should be compared to the discharge shown with the same field and the same intensity but opposite (negative) polarity in Figures 4-32 and 4-35.

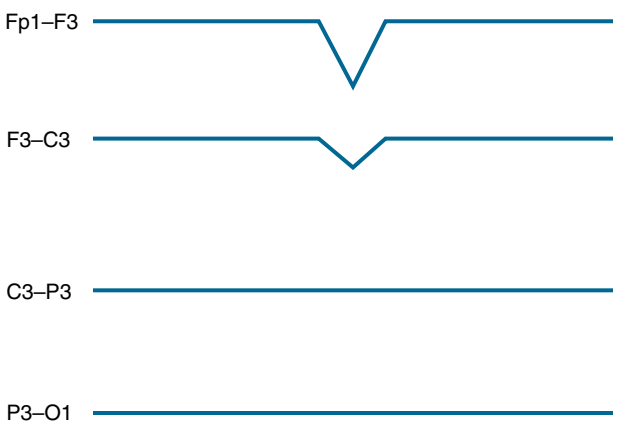


Figure 4-47 Answer 8. The downward deflections in the first two channels reflect diminishing positivity across the first three electrodes in the chain (Fp1, F3, and C3). This pattern is consistent with eyeblink artifact, caused by a sudden positive charge near the frontopolar area related to upturning of the globe of the eye with eye closure.

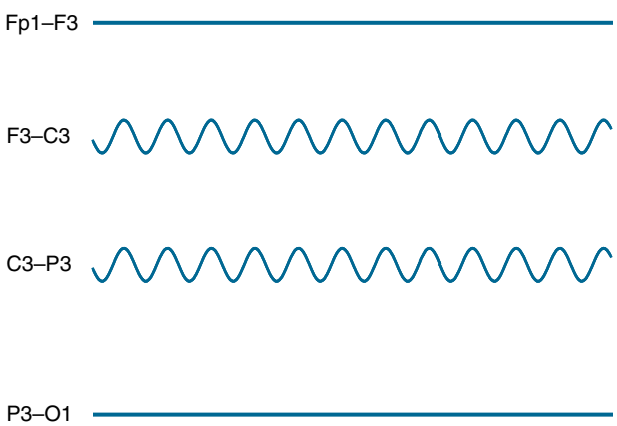


Figure 4-48 Answer 9. The bipolar display of these sinusoidal waves shows a paradoxical effect in which the posterior rhythm is not evident in the most posterior electrode pairing (P3-O1) because of complete cancellation. This is a result of posterior rhythm waves in P3 and O1 being perfectly in phase, which is not always the case in actual clinical recordings (see text).

across C3, P3, and O1 make this a relatively unlikely explanation for the discharge shown in Figure 4-53.

The relatively simple tracing shown in Figure 4-57 initially appears straightforward but actually brings up particular difficulties in interpretation. A sole deflection is present in F3-C3, which implies that all the electrodes in the channel above it (Fp1 and F3) are at the exact same voltage, and all the electrodes in the channels below it (C3, P3, and O1) are also at their own exact same, but higher voltage (i.e., Fp1=F3 < C3=P3=O1). The pattern suggests a plane of lower voltage anteriorly, a quick step-up between F3 and C3, and then a higher voltage plateau in the posterior electrodes. Though such escarpments may be seen in the world of geography, it is very unlikely that the brain would produce two such large perfectly equipotential zones immediately adjacent to one another. Figure 4-60 shows some of the possible mathematical solutions to this problem, but, in reality, none of the solutions is particularly attractive for the reasons described earlier. Indeed, when a lone deflection such as this one is encountered in the middle of a chain in a bipolar recording, it is probable that it represents an electrical artifact in the F3-C3 channel amplifier rather than a true cerebral event.

Note the distinction between a single channel/amplifier artifact and a single electrode artifact. If there is an artifact in a single electrode (perhaps because the electrode is touched, is loose, or "pops"), a deflection will be seen in all the channels that include that electrode. For instance, if the deflection in Figure 4-57 were due to electrode artifact in F3, why is it not also seen in the Fp1-F3 channel? Likewise, if it were due to an artifact in the C3 electrode, why is it not seen in the C3-P3 channel? Such single-channel artifacts are less common than single-electrode artifacts.

Figure 4-58 shows a pattern that can be interpreted either as increasing negativity going down the chain caused by a negative event in the posterior region, or increasing positivity going up the chain (through C3, F3, and Fp1) caused by a positive event anteriorly. If the former is the case with the tracing the result of a negative event in the posterior half of the brain, then there is again the problem that this negative event would have to be exactly equipotential in the bottom three electrodes: C3, P3, and O1. Because a negative event with the *exact* same voltage in C3, P3, and O1 does not seem plausible, this solution is much less likely. Indeed, note that all of the possible solutions suggested in Figure 4-61 must, and do, show the same voltage in those three electrodes. A simpler and more likely explanation is that this represents a positive event in the front of the head, as is seen in the top trace of Figure 4-61. The configuration of this discharge is again consistent with an eyeblink artifact as was described in Figures 4-44 and 4-47.

Figure 4-59 show the simplest possible bipolar tracing with four flat channels. This exercise serves as a reminder of what might be hiding behind any bipolar recording, whether or not it consists of flat channels. The top trace of Figure 4-62 reminds us that this bipolar tracing may truly reflect electrical silence. Because like activity cancels across bipolar chains, the middle trace shows a single spike present in all channels that has been "hidden" by the bipolar recording technique. Such cancellation is a frequent occurrence, especially if the spike represents noise from an external source. For instance, electrocardiographic (EKG) activity is almost always present throughout all head areas but is often not evident in bipolar displays. This is explained by the fact that the EKG potential may be of the same shape and amplitude across the regions in question and will cancel in the bipolar recording (it will also cancel in a referential recording if the chosen reference happens to contain the exact same representation of the EKG signal). Finally, the bottom trace of Figure 4-60 may be the most realistic of the group, because large amounts of electrical noise in the head can be cancelled out by the bipolar recording technique. It is always a good idea to keep in mind all the possibilities of what may be going on "behind the scenes" in a low-voltage bipolar tracing. Most often, the suppression of this common activity is seen as one of the principal advantages of the bipolar recording technique rather than as a loss of useful information.

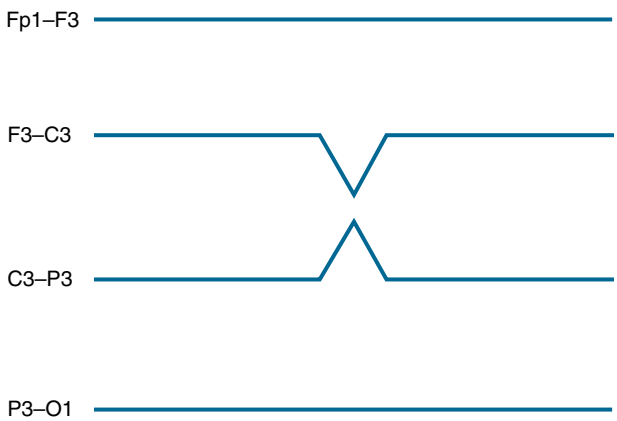


Figure 4-49 Question 10. Predict how this discharge, displayed in a bipolar montage, might appear in a referential montage. The figure depicts a typical discharge with negative polarity and maximum in C3.

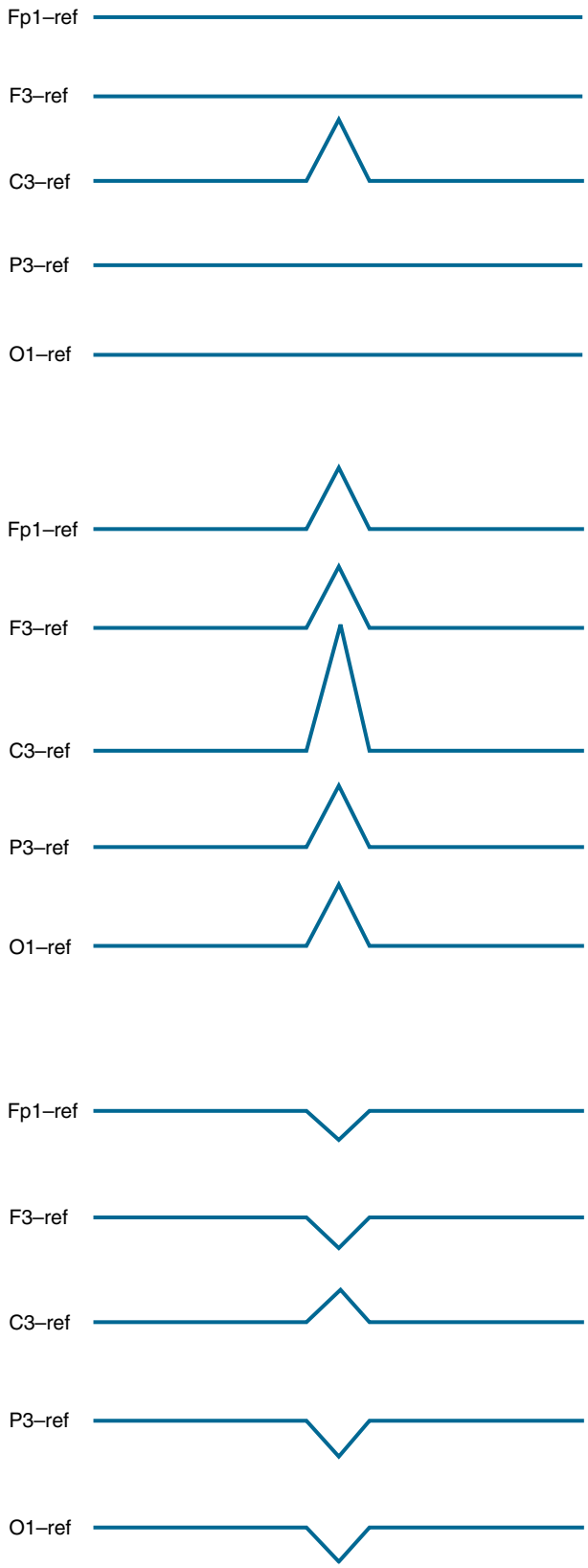


Figure 4-50 Answer 10. Three of many possible representations in the referential montage of the discharge shown on the previous page. All three of the solutions shown in this figure are mathematically possible, but the top solution is most likely (see text). They all have in common the fact that the outside four channels are of the same voltage and the middle channel is slightly more negative in comparison.

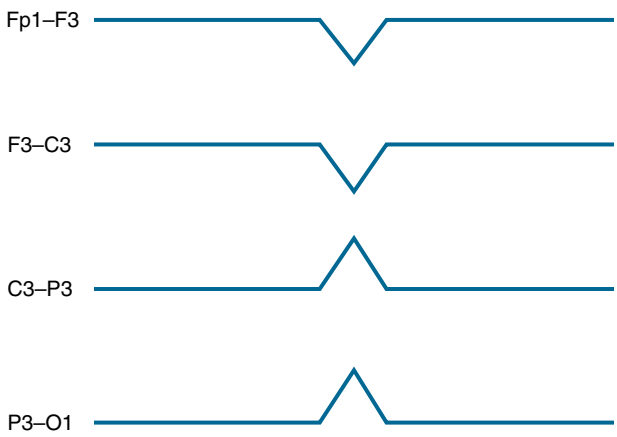


Figure 4-51 Question 11. Again, this discharge shows a maximum with negative polarity (phase reversal with waves pointing toward each other) in C3. There is a smooth gradient surrounding the maximum.

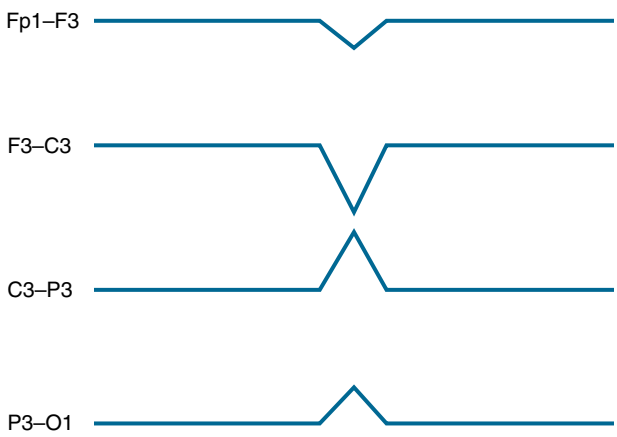


Figure 4-52 Question 12. Predict how this discharge, displayed in a bipolar montage, might appear in a referential montage. The figure shows another type of discharge with maximum negativity at C3; however, the second and third channels show a steeper drop-off of voltage than the outer channels.

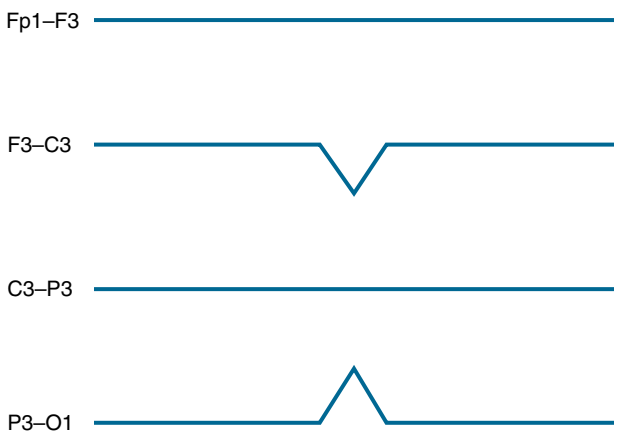


Figure 4-53 Question 13. Predict how this discharge, displayed in a bipolar montage, might appear in a referential montage. This figure depicts a phase reversal with an intervening flat channel. This type of pattern is also referred to as an *isoelectric phase reversal*.

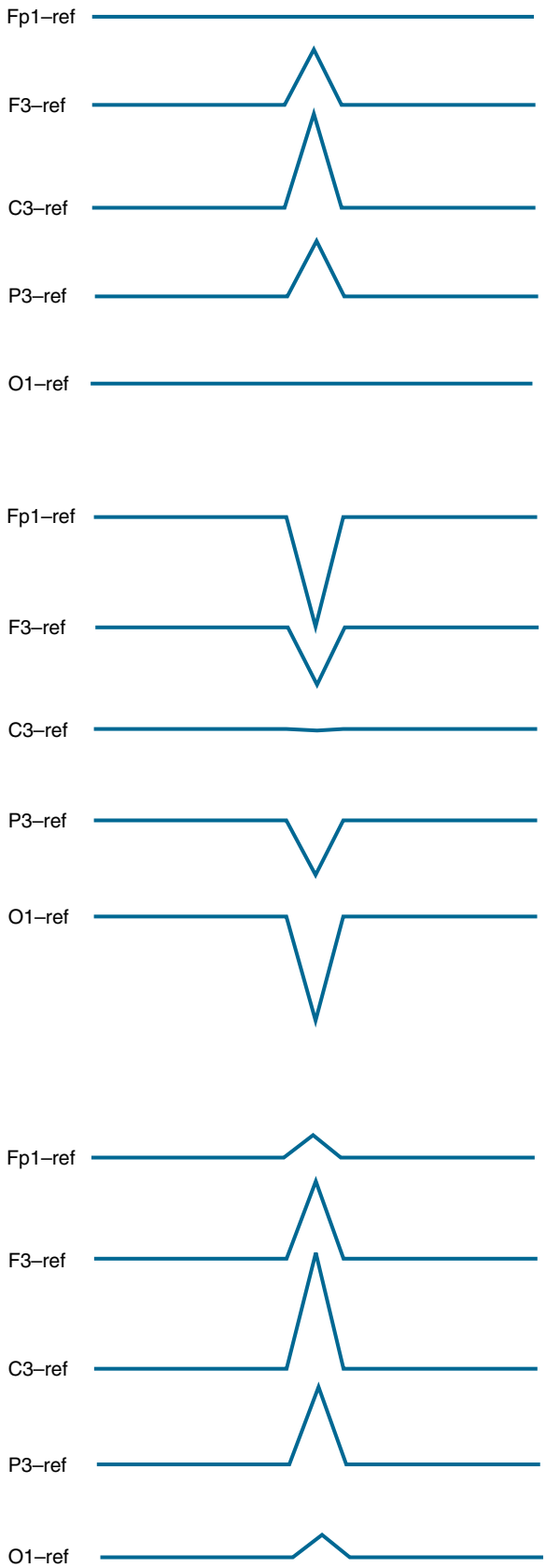


Figure 4-54 Answer 11. Possible representations in the referential montage of the discharge shown on the previous page. The top and bottom traces depict the discharge as having a net negative charge, with decreasingly negative voltage as distance from C3 increases. The middle trace shows an alternative solution. This is a mathematically correct solution that shows C3 as neutral, surrounded by electrodes that become increasingly positive with distance, however this solution is less plausible for reasons discussed in the text.

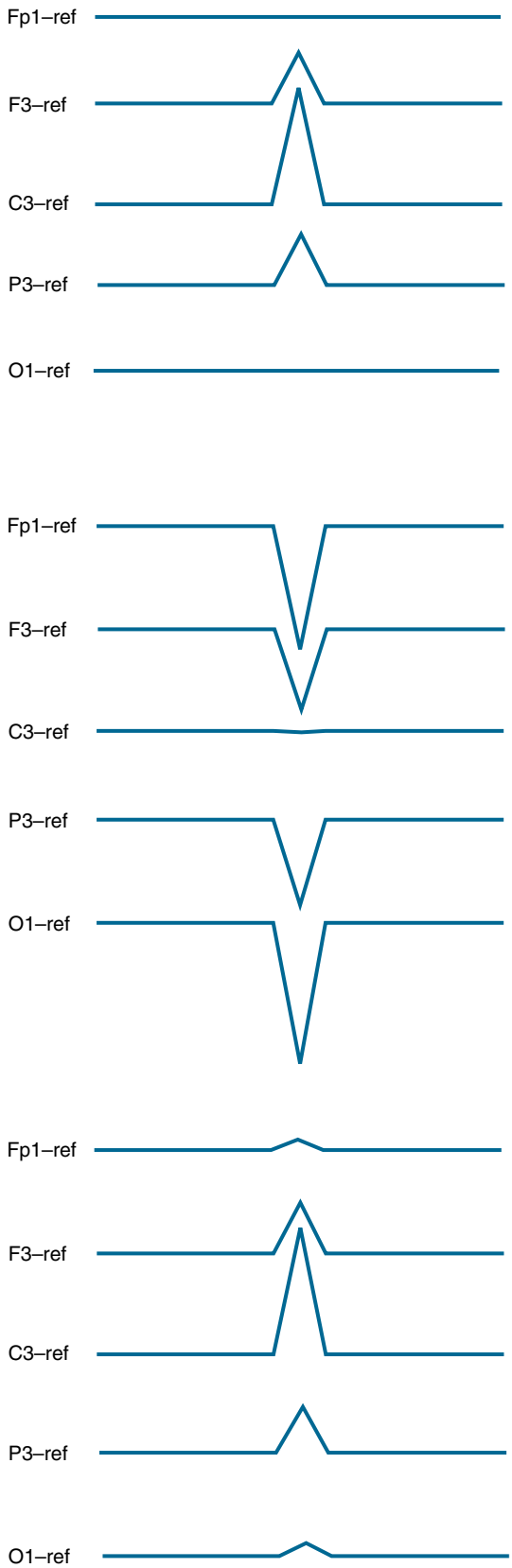


Figure 4-55 Answer 12. Possible representations in the referential montage of the discharge shown on the previous page. The solutions are similar to those shown for the previous figure; however, the field in the immediately surrounding electrodes, F3 and P3, is weaker. Again, the top and bottom traces are most biologically plausible.

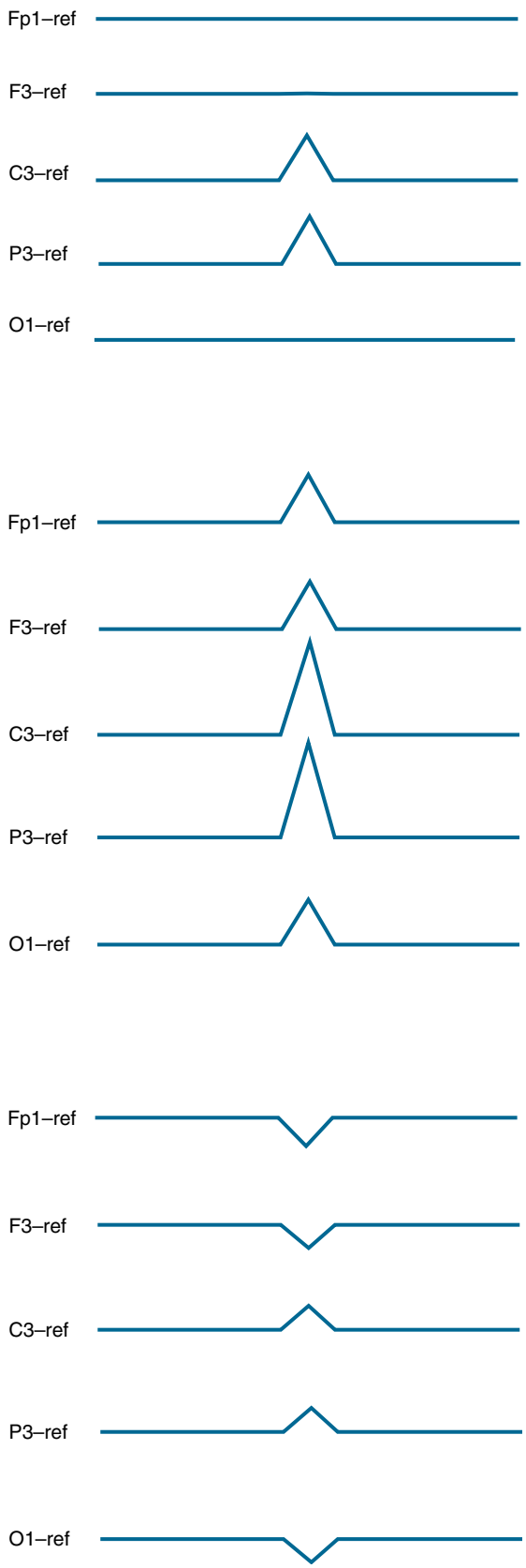


Figure 4-56 Answer 13. Three possible representations of the discharge shown on the previous page are shown in the referential montage. The top tracing is the most plausible solution to this problem, showing a discharge with a shared negative maximum in C3 and P3. Although the middle and bottom tracings are mathematically correct, they are less biologically plausible (see text). All of the solutions have in common the fact that C3 and P3 are of the same voltage and more negative than the surrounding electrodes, which are, in turn, of an equal but more positive voltage.

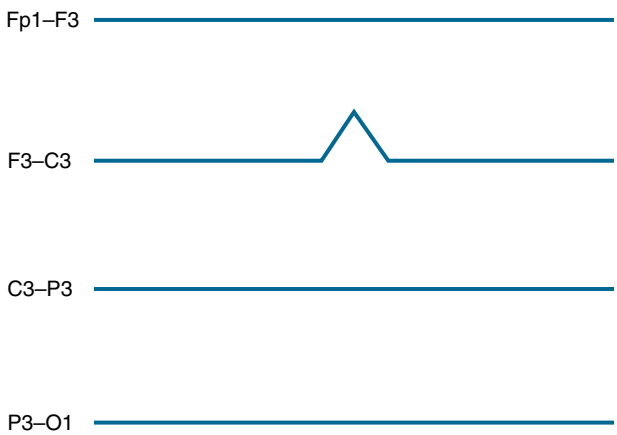


Figure 4-57 Question 14. Predict how this pattern, a bipolar tracing showing a single channel with an upward deflection in the middle of a chain, might appear in a referential montage.

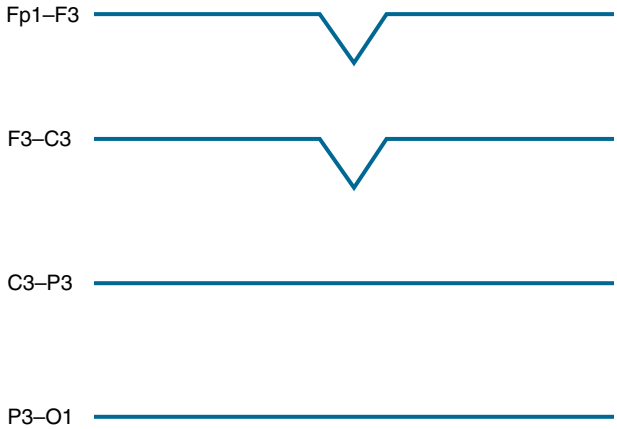


Figure 4-58 Question 15. Predict how this discharge, displayed in a bipolar montage, might appear in a referential montage. What type of commonly encountered EEG event does this pattern depict?

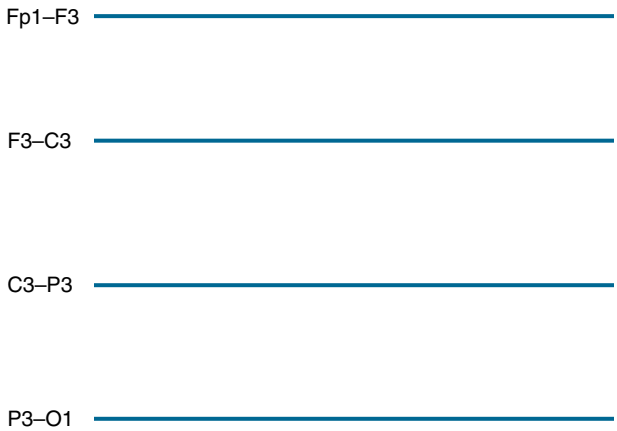


Figure 4-59 Question 16. Predict how this pattern, displayed in a bipolar montage, might appear in a referential montage. This example, showing four flat channels, is the simplest possible tracing but still has multiple possible solutions.

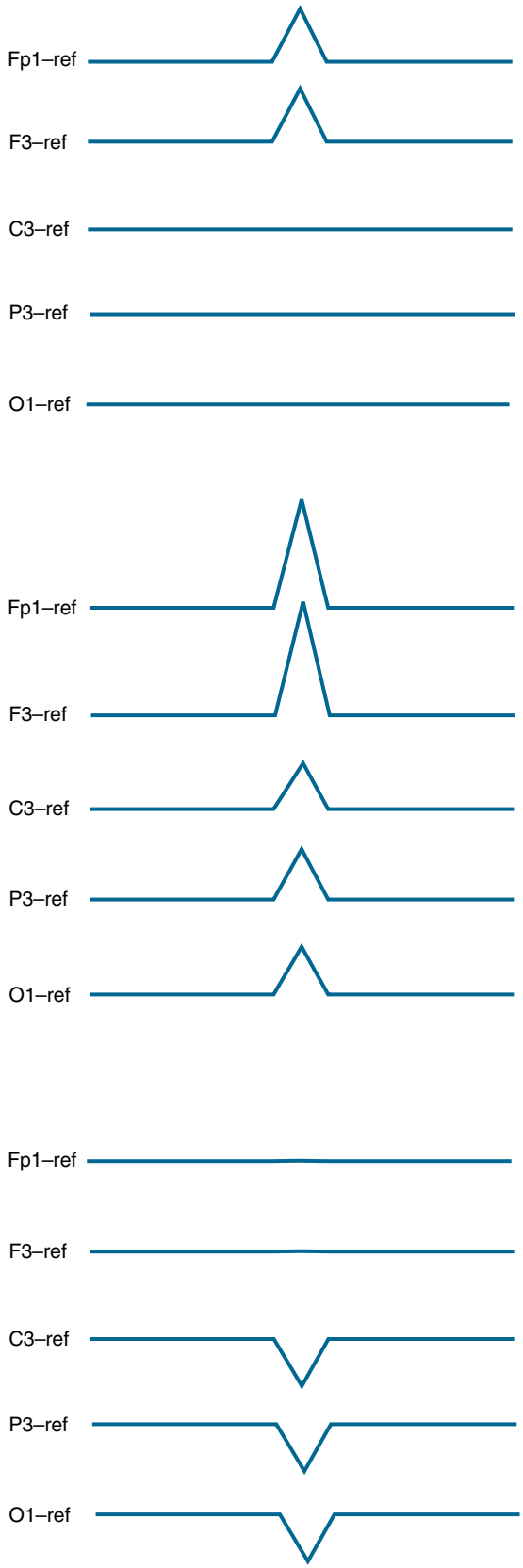


Figure 4-60 Answer 14. Possible representations of the discharge shown on the previous page are shown in the referential montage. The flat channels in the bipolar montage tell us that voltages at Fp1 = F3, and that C3 = P3 = O1, but at a more positive voltage. Although any solution that fits these constraints is mathematically valid, none is particularly plausible in terms of cerebral activity, and such patterns often represent an amplifier or channel artifact (see text).

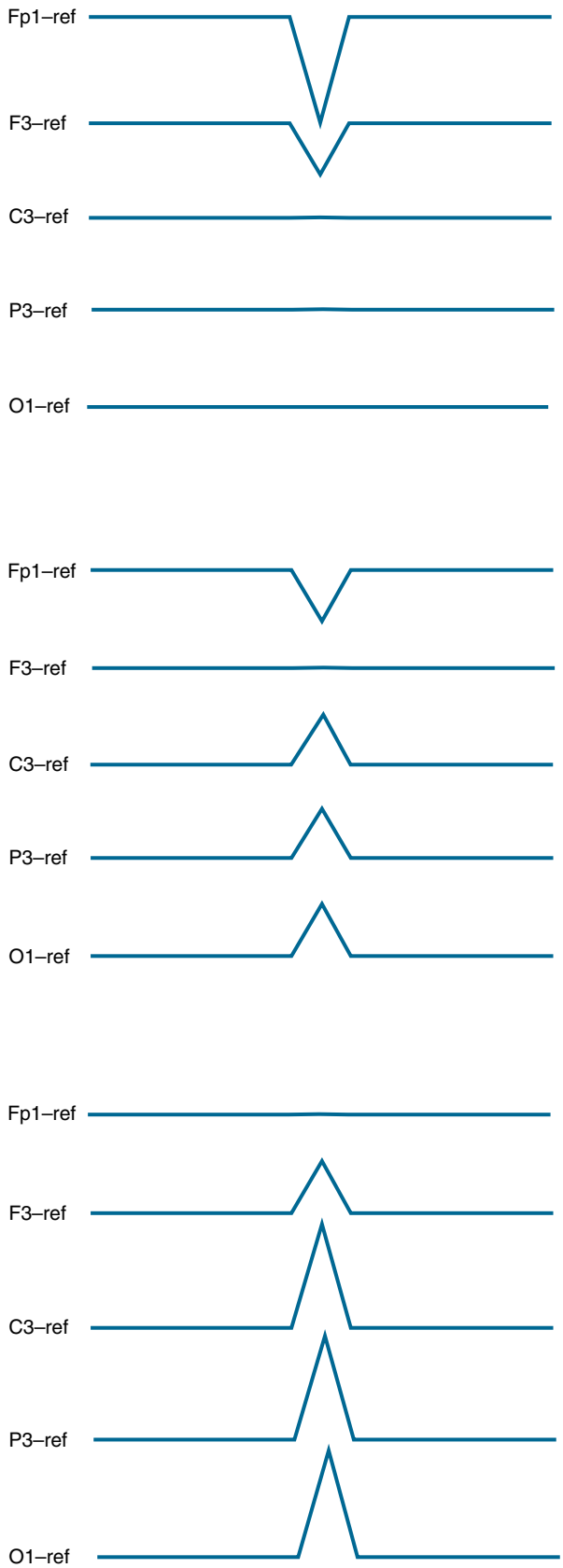


Figure 4-61 Answer 15. Possible representations in the referential montage of the discharge shown in Figure 4-58. The top tracing is the most likely solution, representing a positive event that is strongest anteriorly. The bottom tracing suggests a negative event posteriorly but is less plausible because C3, P3, and O1 would have to be negative but of exactly equal non-zero voltage (see text). This pattern of a large anterior positivity is highly suggestive of eyeblink artifact.

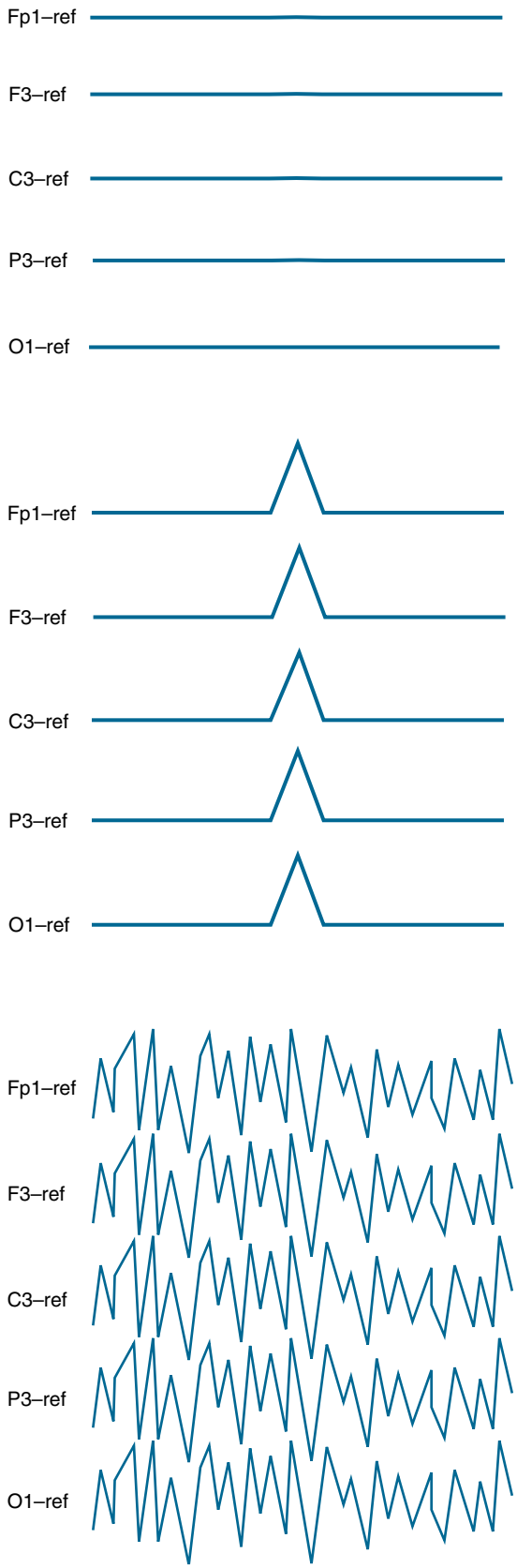


Figure 4-62 Answer 16. Possible representations in the referential montage of the discharge shown on the previous page. The top tracing depicts electrocerebral silence. The middle and bottom traces are also valid, representing electrical noise in all electrodes that canceled in the original bipolar tracing.

Chapter

5 Electroencephalographic Electrodes, Channels, and Montages and How They Are Chosen

In electroencephalography, the term *montage* refers to the order and choice of channels displayed on the EEG page. Several decisions go into the design of a good display montage: which electrodes to use, how the electrodes should be paired to comprise each channel, and the ordering of the channels in a way that will render the tracing easy to interpret visually. Decisions of secondary importance include choice of the color in which to display each channel and placement of gaps or white space between groups of channels to allow easier visualization of groups.

Although a large number of montages is theoretically possible, a given laboratory traditionally uses a relatively small set of montages. Smaller montage sets have the advantage of allowing the readers in a given laboratory to become more quickly familiar with each montage, offering more efficient and faster scanning of tracings. Therefore, one should hesitate before adding a montage to a laboratory's set that already includes the same electrode pairings that are present in another member of the set; such a montage is redundant and may not really add a "new view" of the EEG.

In addition to scalp electrodes placed to record brain electrical activity, additional "monitoring" electrodes may be placed on nonscalp areas to record other physiologic activities including heartbeat, respirations, eye movements, contraction of certain muscles, including the respiratory muscles, and limb movements. The schema for the placement and naming of the EEG electrodes according to the international 10-20 system was described in Chapter 3. While considering the strategies used in different montage designs, the reader should consider how montage choice affects the reading process and also how new montages might be designed in special situations. Such situations could include a patient in whom part of the head is not accessible for one reason or another (e.g., a surgical bandage) or the occasional patient in whom accurate localization requires the placement of intervening electrodes.

CATEGORIES OF MONTAGES: REFERENTIAL AND BIPOLAR

Montages are generally divided into two large groups, *referential* and *bipolar,* denoting the technique by which EEG data are displayed. Because there are significant differences in the strategies used by these two montage families, each is discussed in its own section. Localization techniques for each of these montage types were discussed Chapter 4.

Recommended Montage Conventions

Although electrode channels can be presented in any order when creating a montage, certain conventions are encouraged (see the American Clinical Neurophysiology Society [ACNS] Guidelines in References): channels should be placed in a "left over right" configuration (electrode chains from the left side of the head are placed nearer the top of the page, and chains from the right side of the head are placed nearer the bottom of the page). More anterior electrodes should be placed before more posterior electrodes (front-to-back ordering of channels going down the page is encouraged). Interelectrode distances should be consistent (electrode positions should not be unpredictably skipped in electrode chains). Finally, the design should favor the simplest arrangement possible (see Figure 5-1).

In the days of paper EEG recording, it was up to the EEG technologist to choose the montage that would best demonstrate the patient's findings at any particular point in the tracing. If an EEG event happened to be recorded in a montage that was disadvantageous for interpretation, the ink had already dried on the page, and it was not possible to change settings retrospectively. The newer digital EEG technology presents a new set of problems. Although the same EEG page can be viewed in a variety of montages during the course of interpretation, some readers may find that it takes less

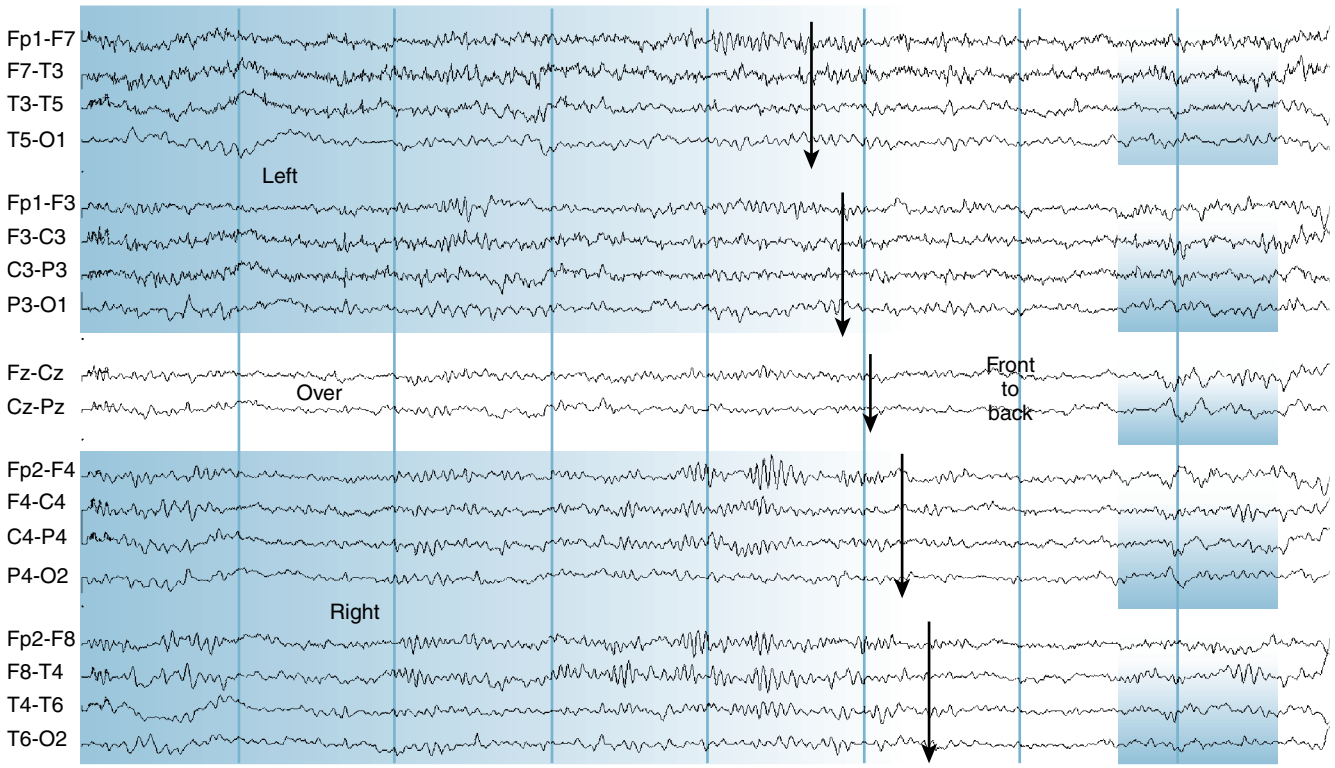


Figure 5-1 This page of EEG is shaded in a way that highlights the groupings and structure of a standard AP bipolar montage. The top two sets of four channels with darker shading (labeled *LEFT*) represent the left hemisphere and the bottom two sets of four channels with a lighter shading (labeled *RIGHT*) represent the right hemisphere. The middle, unshaded channels represent the midline. This montage therefore conforms to the general convention of placing left-sided channels over right-sided channels. Within each grouping of four channels, each chain runs from the front of the head to the back of the head (depicted by the gradient shadings on the right side of the figure). The downward-pointing arrows show how the eye scans from front to back through each four-channel (or two-channel) grouping. After this scheme is understood, it is possible to predict how each channel on the page corresponds to a location on the head based simply on its position in the groupings without needing to consult the channel labels.

energy to leave the display in one montage for the whole tracing, usually choosing the montage with which they are most comfortable. Although scanning a whole EEG study in a single montage may allow for faster reading, occasionally an EEG finding may be seen well in one montage but only poorly seen, or perhaps not seen at all, in another. It is therefore best practice for the EEG technologist or the reader to cycle through a minimum set of montages during the course of each study. This will usually include some combination of longitudinal bipolar, transverse bipolar, and referential montages.

MONTAGE DESIGN

Referential Versus Bipolar Montages

Referential and bipolar recording techniques appear to use two distinct strategies for recording, but, as we shall see, there is considerable overlap between the two. A referential montage compares each "point of interest" on the head (which can be referred to as "the electrode of interest") to a reference point somewhere else on the body, perhaps still on the scalp, which it is hoped will be neutral or "indifferent." One of the main drawbacks of referential montage recordings is that the reference point often cannot be completely neutral. Here is an example of the left parasagittal chain of five electrodes as it might be displayed in a referential montage:

Fp1–ref
F3–ref
C3–ref
P3–ref
O1–ref

The term *ref* as used here denotes a reference electrode such as the nose, the chin, the back of the neck, or the earlobes, another electrode on the scalp, or sometimes a combination of scalp electrodes. The specific choice used for the reference electrode is further discussed later in this chapter.

Considering the technique used to display the same chain of five parasagittal electrodes in a bipolar montage, the following channels would be used:

Fp1–F3
F3–C3
C3–P3
P3–O1

The term bipolar derives from the fact that, as can be seen from this chain, each channel represents the voltage difference between two "poles" on the scalp. Bipolar montages are occasionally referred to as *differential* montages because they display the difference between adjacent electrodes. Although the bipolar technique is a powerful recording technique and a favorite among many readers, bipolar montages also have their drawbacks, as discussed later. Figures 5-2 and 5-3 show a schematic of the difference between the referential and bipolar recording strategies.

Note that, even the so-called referential montage is, in reality, "bipolar." Every channel must represent a comparison of two electrode positions—there are always two "poles" being compared. This is one of the criticisms of the term bipolar montage; it seems to imply that the counterpart term, referential montage, is not bipolar in some way. Indeed, there is no such thing as a "monopolar" montage, and technically all montages are, at their root, "bipolar" because every channel displayed on the EEG page, be it part of a bipolar or referential montage, is a voltage comparison between at least two points.

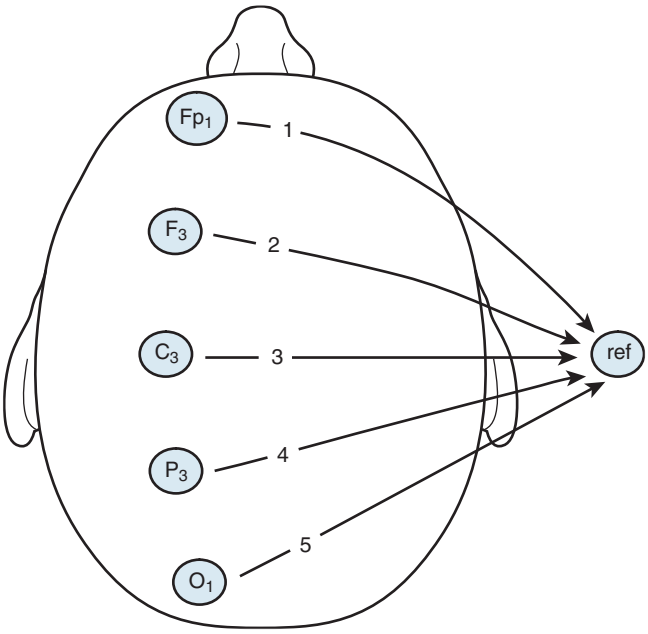


Figure 5-2 Referential montages are designed to compare each "electrode of interest" to a separate reference position that may be either close to or distant from the scalp electrodes depending on the montage design. Each arrow leads from the Input 1 electrode to the Input 2 electrode and creates a channel derivation. This example shows a setup for part of a referential montage that would include the five electrodes in the left parasagittal chain: Fp1-ref, F3-ref, C3-ref, P3-ref, and 01-ref. Note that a referential montage designed to show readouts for five separate electrodes would generate five separate channels, labeled 1 through 5 in this illustration.

Theoretical Strategies: How Can EEG Activity Best Be Recorded From a Single Point?

It is useful to consider the theoretical pros and cons of each recording technique starting by asking the elemental question: what would be the best way to demonstrate the electrocerebral activity at any given point on the head? Consider the example of the left frontopolar area, where the Fp1 electrode records at a point on the forehead above the left eye. If we were assigned the task of deciding how best to record the electrical activity occurring under the Fp1 electrode, momentarily leaving aside what is going on elsewhere in the brain, what would be the best way to do this?

Recalling that our basic tool for creating an EEG channel is to print the amplified output of a voltmeter for which the needle sweeps one way or the other depending on the voltage difference between two points, there must always be two separate amplifier inputs, Input 1 and Input 2, to make a comparison. We decide to attach the "electrode of interest," Fp1, to one pole of our voltmeter (Input 1), but what would be the most advantageous choice of electrode(s) to attach to Input 2, the comparison point? This comparison electrode is called the *reference electrode,* and serves as the reference point against which the electrode of interest is measured. As described in Chapter 4, the voltage measured from the reference will be subtracted from the voltage measured at Fp1, and the resulting difference is the output for the channel that we will label as Fp1-ref.

It is clear that the choice of the reference electrode could have as big an impact on the appearance of the resulting output trace as the Fp1 electrode. At first glance, the channel label "Fp1-ref" may seem to imply that its output represents the voltage at Fp1. Note,

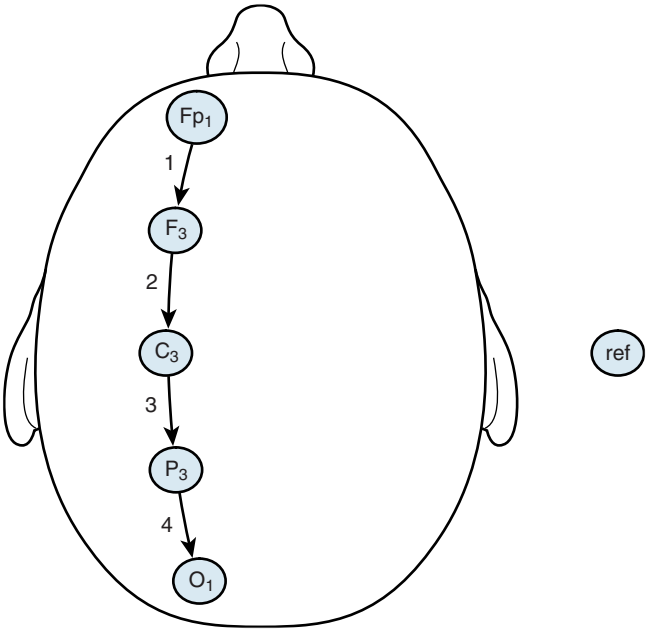


Figure 5-3 In this illustration, the same five parasagittal electrodes used in Figure 5-2 are connected to each other sequentially in a standard "bipolar chain." Again, each arrow points from an Input 1 electrode to an Input 2 electrode creating the following montage: Fp1-F3, F3-F3, C3-P3, and P3-01. The reference position is not used. Each of the four channels generated with this technique represents the subtraction of an electrode from the previous electrode in the chain. Because the electrodes are paired to form each channel, five electrodes generate four channels of output.

however, that the "active electrode," Fp1, does not enjoy any privileged position by being attached to Input 1 as opposed to Input 2; the two voltages will simply be subtracted one from the other and displayed. For that reason, if the chosen reference electrode were the earlobe and there happened to be more electrical activity present in the earlobe than in Fp1, the output of an Fp1-earlobe channel would be dominated by the electrical activity in the earlobe—not the desired result. Clearly, the choice of the reference electrode in referential montages is important.

Choice of a Reference Electrode

Considering different possible locations to place the reference electrode, because we are looking for a location with a "neutral" voltage for comparison, one possible choice would be an electrical ground. Possible grounds include the grounding prong from a three-prong wall outlet or a metallic cold water pipe that runs into the ground. In reality, the choice of attaching Input 2 to an electrical ground is not feasible. The resulting tracing would be full of noise, and we would have little success in our goal of displaying the very low-voltage EEG activity coming from the Fp1 electrode for two reasons.

First, electrical grounds, particularly those in large buildings such as hospitals, are not really electrically neutral. A variety of electrical devices throughout the building are attached to the same ground, and these devices spill electrical noise into the ground. Therefore, an Fp1-ground channel could include a terrific amount of noise from all of these distant sources and the signal of interest, the microvolt-level electrical activity emanating from the brain from the region of the Fp1 electrode, could be overwhelmed by all of this electrical noise.

The second and perhaps less intuitive problem is that the Fp1 electrode itself has a large array of electrical activity in it, but not all of that activity is brain wave activity. In addition to the very low-voltage activity that emanates from the left frontal pole of the brain (the activity that we really want to see), there is also a large amount of ambient electrical noise at that scalp location. Some of this noise comes from the human body, including electrical activity given off by the frontalis muscle of the forehead, electrical activity from the heart (the electrocardiogram), electrical noise from movement of the eyes, and even noise from swallowing movements. Even more troublesome, however, is the fact that, especially indoors, the environments we live in are full of electrical activity. Relatively large amounts of electrical noise flow through our bodies at all times, especially in electrically active environments such as hospital rooms or EEG laboratories where EEG recordings are usually performed. As previously described, contamination of our bodies with 60-Hz signals from electrical outlets is especially common. Comparison of Fp1 to a completely indifferent electrode (such as an ideally quiet ground) would do nothing to attenuate (or "subtract out") the large amount of electrical noise in the vicinity of the Fp1 electrode in favor of the true brain wave signal in that area. Therefore, paradoxically, we do want some noise in the reference electrode. Ideally, we would like the reference electrode to have the exact noise that is present in the Fp1 electrode so that our differential amplifiers will subtract it out.

Considering these goals, choice of an ideal reference electrode becomes an interesting challenge. Instead of using electrical ground as the reference electrode, consider the possibility of using an electrode placed on the foot. The choice of the foot electrode would be a vast improvement over using the ground because a recording electrode on the foot would detect a lot of the same electrical "body noise" as an electrode on the head (and also would lack the ground noise). Because the signals from Fp1 and the foot would be subtracted from one another, electrical noise in the body common to both locations would cancel out and only the difference would appear in the resulting tracing. The chance that the brain wave signal we seek under Fp1 would rise above the fray and be recognizable to us is now much higher.

Next, consider the idea of using an electrode position adjacent to Fp1 (such as Fp2, the electrode over the right frontal pole) as the reference electrode. Subtracting Fp2 from Fp1 yields a signal that appears much cleaner and will be of lower voltage compared with the Fp1-foot pairing. At first glance, this Fp1-Fp2 pairing would seem to be the most satisfactory because the amount of noise in the channel is minimized—any electrical noise in Fp1 is probably also present in Fp2 and that common noise should cancel out. However, there is also a significant potential disadvantage that may not be obvious at first glance. If there is brain wave activity common to both the Fp1 and Fp2 electrodes, the subtraction of the Fp2 signal from Fp1 may actually cancel out some of that common brain wave activity, the activity we actually seek to record. The only signal displayed will be the *difference* between the two locations that may only represent a portion of the total brain wave activity at Fp1, the display of which was the original goal of picking an ideal reference. The idea that there could be a lot of brain wave activity in common between Fp1 and Fp2 is plausible because there could be, for example, a frontal slow wave shared more or less equally by Fp1 and Fp2. The common mode rejection amplifiers used in our EEG instruments would cancel some or all of this common brain wave activity when the Fp2 activity is subtracted from Fp1. We would then have a tracing that appears to be "electrically clean" at the expense of losing some of the EEG activity we are looking for.

Finally, consider the extreme example of moving the reference electrode so close to Fp1 that it is nearly in the same position as Fp1. In such an example, the signal in Fp1 and the reference are so similar that the difference output will become nearly flat. Some generalizations can then be made regarding the distance between the electrodes attached to Input 1 and Input 2 of a channel's amplifier. When two electrodes are moved closer to one another, there is greater commonality

between what each detects in terms of both the noise signal and the brain wave signal. Therefore, as two electrodes become closer to one another, a channel comparing them will flatten. Conversely, as interelectrode distances increase, there is a bigger difference in both the noise signal and the brain wave signal and the channel's output signal tend to increase.

All things being equal, larger interelectrode distances are associated with higher voltages, and smaller interelectrode distances are associated with lower voltages. This effect explains the recommendation that electrodes should not be skipped in bipolar montage chains (or stated another way, that interelectrode distances should be held constant). A channel pair with an inconsistently large interelectrode distance will tend to produce a higher voltage channel, possibly giving the erroneous impression that there is more electrical activity at that location than at adjacent locations. Variation in interelectrode distances may occur in two ways: a montage design that skips an electrode or a significant mismeasurement of electrode position during electrode application (see Figure 5-4).

What would the ideal reference electrode location be?

The ideal reference electrode includes all of the electrical noise in the "electrode of interest" but none of the electrocerebral activity.

It will come as no surprise that no such ideal reference electrode exists. The goal is to find some compromise location for the reference electrode that will tend to cancel out noise fairly well but will not cancel out too much brain wave activity. The choice of a good reference electrode position represents a balance between wanting the electrode to be close to the "electrode of interest" so that it will share as much noise signal with it as possible and wanting it to be distant enough so that there is not too much cerebral activity in common between the two electrodes (to avoid cancelling out EEG information). Commonly used reference points include the earlobes or mastoid areas, the nose, the CS2 location representing the skin overlying the spinous process of the seventh cervical vertebra (*vertebra prominens* or the most prominent bony bump at the base of the neck), or more rarely the Cz electrode. More complex reference electrodes may be used as well, including techniques in which the arithmetic mean of some or all of the scalp electrodes is used as a "virtual" reference electrode (discussed later in more detail). Each of these techniques is associated with its own advantages and disadvantages, which will also vary according to clinical circumstance.

Each potential reference location is prone to certain problems and advantages—no single reference electrode position is ideal for all patients at all times. For instance, the earlobes often share a portion of the electrocerebral activity present in the adjacent

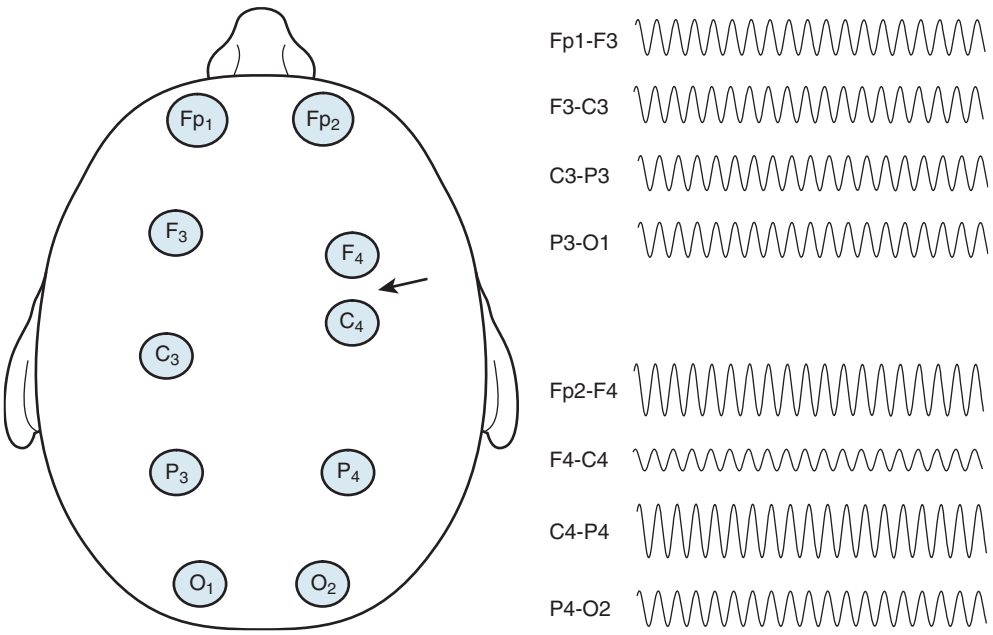


Figure 5-4 This figure illustrates the consequences of mismeasurement of electrode positions. Note that the electrodes of the left parasagittal chain, starting with Fp1, are measured in the usual way with constant interelectrode distances. The electrode positions of the right parasagittal chain, however, have been mismeasured so that the F4 and C4 electrodes have been placed too close together, resulting in an inadvertent increase in the interelectrode distance in the Fp2-F4 and C4-P4 electrode pairs, while the F4-C4 interelectrode distance is too small (arrow). The left parasagittal chain, the output of which is represented by the top four channels on the right side of the page, correctly displays equal voltages in each channel. As a consequence of the mismeasurement in the right parasagittal chain, the channels for which interelectrode distances are too large, Fp2-F4 and C4-P4, show exaggerated, higher voltages, and the channel with the decreased interelectrode distance, F4-C4, shows a misleadingly decreased voltage. Note that if each of these chains had been displayed using a referential montage, the error in measurement in the right parasagittal chain would not necessarily be evident.

midtemporal areas, causing a cancellation effect. This makes an earlobe reference a poor choice for a patient with a high voltage midtemporal discharge. Also, the left earlobe in particular is often contaminated with EKG artifact because of the left-sided position of the heart. In some, the nose reference may be contaminated by a surprising amount of muscle artifact, whereas in others it can be fairly clean. The cervical area is contaminated by muscle artifact or EKG signal in some patients and may also be disturbed by movement when patients are lying on their backs. The midline vertex electrode, Cz, is usually free of muscle artifact because of its location at the vertex of the scalp but contains a large amount of electrocerebral activity, especially during sleep. In different individuals, any of these reference locations could generate satisfactory or unsatisfactory recordings at different times.

In the case of designing a referential montage, after the reference electrode has been chosen, there is little more to decide than the order to arrange the channels on the page. There are two general strategies for channel arrangement. One is to arrange the channels in left–right pairs so that each position on the brain can be compared with its homologous counterpart in the opposite hemisphere. This approach leads to a "left–right–left–right…" arrangement of channels (e.g. Fp1 followed by Fp2, F7 followed by F8, F3 followed by F4, and so on). The alternative strategy for ordering the channels conforms to the "left-over-right" and "front-to-back" conventions mentioned above in which channels are "clumped" according to their location. Although each of the two systems offers specific advantages, neither is clearly superior to the other as each EEG electrode's channel will appear somewhere on the page with either system, only in a different order.

The main difference between the two systems, one of which alternates left- and right-sided electrodes and the other of which groups left-sided electrodes in one area and right-sided electrodes in another, is the comparative ease of visual scanning for asymmetries. Choice of one of these arrangements over the other is essentially a matter of preference. Figures 5-5 and 5-6 show examples of dramatic left-sided slowing displayed with each of these two common strategies. The latter system, which groups electrodes together by location, is seen to have certain advantages. Asymmetries that occur between hemispheres are not usually localized to a single electrode but to a regional group of electrodes. For instance, if a temporal slow wave is present, it may be recorded in multiple electrodes such as F7, T7, and P7. When an abnormality such as a spike or a slow wave is

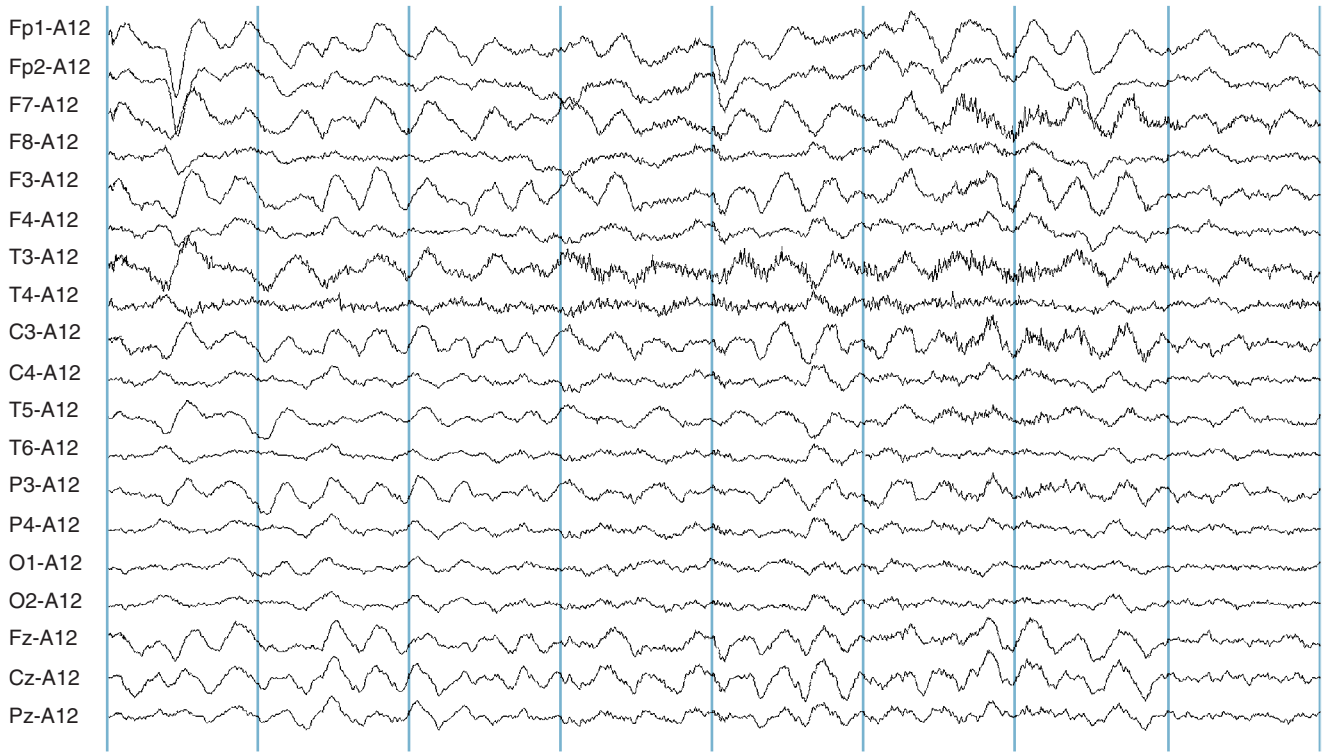


Figure 5-5 This EEG page shows an example of left hemispheric delta activity shown in a referential montage in which the channels are ordered on an alternating left–right basis. This commonly used arrangement has the advantage of allowing the reader to compare homologous areas of the brain in adjacent channels. For instance, the top two lines show channels comparing the left frontopolar area to the right frontopolar area, and so on. In this case, the reference used is an average of the A1 and A2 electrodes (placed on the left and right earlobes, respectively). Close observation reveals, at least on the top half of the page (and therefore over the anterior portion of the brain), that every other channel shows a more prominent delta wave, indicating increased slow wave activity over the left anterior hemisphere.

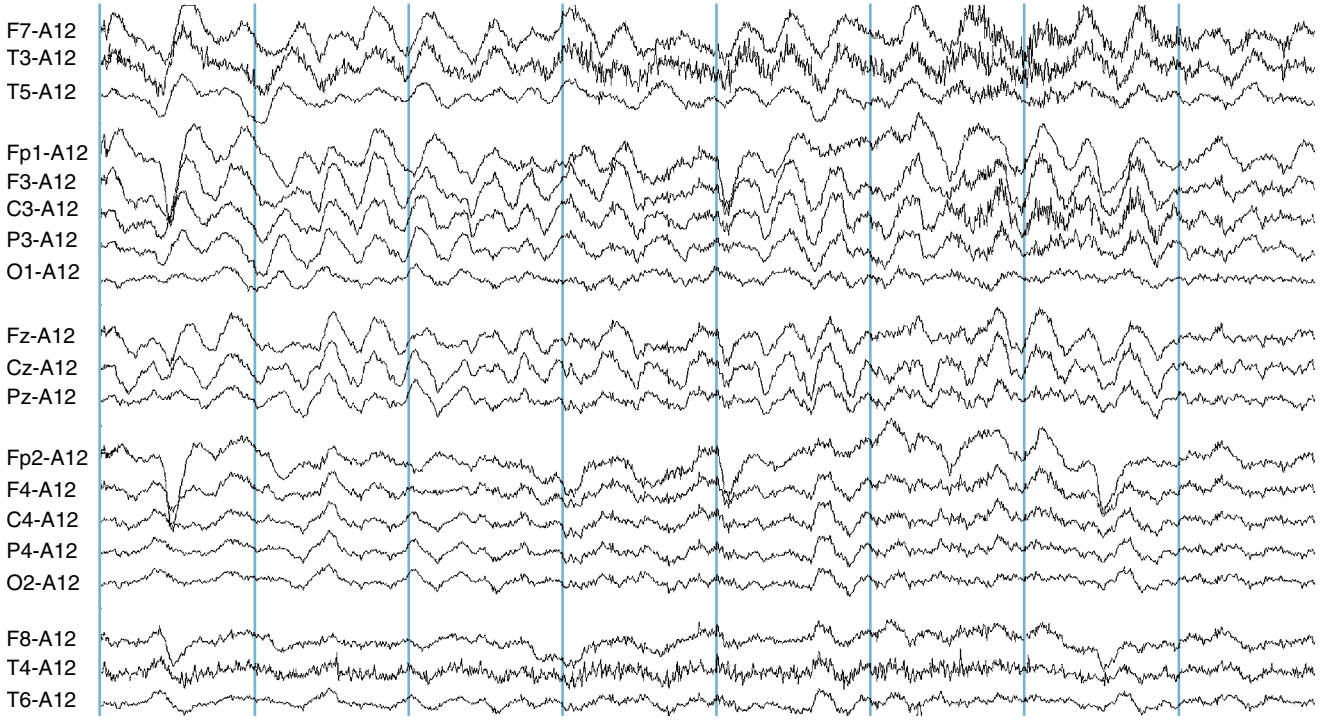


Figure 5-6 This EEG page shows the exact same left-sided slowing as was seen in the previous figure, however, the channels are ordered in chains so that the left hemisphere electrodes are shown on the top half of the page and the right hemisphere electrodes on the bottom. Starting at the top, channels are grouped on the page as the left temporal, left parasagittal, midline, right parasagittal, and right temporal chains (see channel labels). The same reference is used as in the previous figure, an average of the A1 and A2 electrodes. Note that, with nearby electrodes grouped together, it is visually much easier to appreciate the localization of the slowing over the left side of the brain (top half of the page). For this reason, this arrangement is favored by the author.

present in multiple *adjacent* channels on a page, it tends to be easier to identify visually when the spatial clustering system is used. In the alternating left–right setup, the abnormality will only be seen in alternating channels. Another advantage of the clustering system is the ease of visualizing the topography of the discharge on the scalp; with this system a region of the page corresponds to a region of the brain starting with the fact that the left hemisphere is represented on the top of the page and the right hemisphere is on the bottom. Grouping the channels by region, especially when gaps are used between electrode groups as in the example illustrated here, makes it simple to know where a given channel is on the head at a glance, even without reading the channel labels. In contrast, when reading in a montage in which the left and right channels are alternated, it can be difficult to know the location of each electrode without consulting the channel labels. Proponents of the left–right alternating system like the fact that homologous channels are adjacent to one another, making individual interchannel comparisons easier. Considering these reasons together, the author prefers the clustering system (left over right and front to back), which should allow for easier and more accurate scanning and spatial visualization for most readers. However, the choice of either of these two arrangements is really a matter of laboratory or reader preference.

Bipolar Montages

In practice, most electroencephalographers do the bulk of their EEG interpretation using bipolar montages rather than referential montages, despite the potential disadvantages of the former. Bipolar montages are generally preferred because they produce "cleaner" tracings due to the proximity of the electrode pairs, which leads to more efficient noise cancellation. The clean appearance of the tracings and ease of reading does come at a potential price, however: the risk that the proximity of the electrode pairs has led to cancellation or an understated appearance of some of the brain wave activity, perhaps unbeknownst to the reader. When recording in a referential montage, if noise is present in the reference electrode, that noise may potentially obliterate all the channels at once. Given the different strengths and weaknesses of each technique, it is best practice to interpret any given EEG study using some combination of the two techniques.

Categories of Bipolar Montages

There are two principal categories of bipolar montages, the anteroposterior (AP) bipolar montage and the transverse bipolar montage. The two types differ in that, with AP bipolar montages, electrode chains run from front to back (anteroposteriorly) down the head,

and in transverse bipolar montages, the chains run from left to right (transversely) across the head. The fact that the reader has both of these montage families available for use raises the question of whether any particular discharge might only be visible in the AP bipolar but not in the transverse bipolar montage, or vice versa. Is it really necessary to use both AP and transverse bipolar montages for EEG interpretation, and, if so, what would be lost by not doing so?

It is true that the large majority of discharges seen in an AP bipolar montage will also be visible in a transverse bipolar montage; however, there are exceptions. Certain discharges may show a steep gradient in the AP direction and a shallow gradient in the transverse direction (or vice versa). Although such discharge topographies are relatively infrequent, examples of scalp events that are only evident in one type of bipolar montage but not the other do occur.

Using a combination of these two montage types may also aid significantly in localization. The AP bipolar montage will indicate the maximum of the discharge in the front-to-back direction, usually by the location of a phase reversal. Likewise, the transverse montage will localize the maximum of the discharge in the left-to-right direction. Combining the two techniques allows localization of the maximum on a two-dimensional model of the scalp surface.

Circumferential Montages

Some laboratories also use a third ordering for bipolar electrode chains, one that makes a complete circumference around the head (see Figure 5-7). This style of montage has been given a variety of names, such as a *halo, circumferential, or hatband* montage. Close examination of the electrode pairs used in the circumferential montages shows that almost every pairing is also present in the standard AP bipolar montage. The only electrode pairings that are unique to circumferential compared with AP bipolar montages are the Fp1-Fp2 and O1-O2 pairs. Because these two pairs are included in the transverse bipolar montages, circumferential montages are not a mandatory member of a laboratory montage set, although the decision to use such a montage is at the discretion of the electroencephalographer. For this reason, the use of a minimum of three montages—AP bipolar, transverse bipolar, and referential—is usually adequate to cover all necessary electrode pairings in standard EEG recording. Circumferential montages, when used, lend themselves to a particular technique for visual scanning. A visual axis is chosen and surrounding channel "layers" are compared (see Figures 5-8 and 5-9).

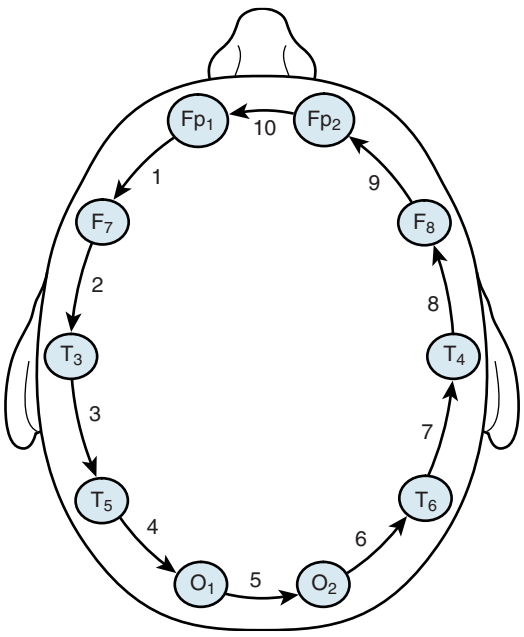


Figure 5-7 This arrangement of a bipolar chain that encircles the head is called a *circumferential, halo*, or *hatband* montage in different laboratories. In the arrangement shown in this figure, the electrode chain starts with Fp1 and proceeds in a counterclockwise direction. In some versions of this montage, the circumferential chain can start with one of the occipital electrodes.

Disadvantages of the Bipolar (Differential) Technique

We have already alluded to certain disadvantages of the bipolar montage strategy. These stem from the fact that we are looking at the display of the differences between electrodes that are quite near and, typically, adjacent to one another. Clearly, a significant amount of information can be lost using this technique. An analogy for how information is lost with the bipolar technique is illustrated by the following example: imagine that we are told about the weather during a 5-day period in the following fashion: "Between Monday and Tuesday, it got five degrees warmer, and on Wednesday it got another three degrees warmer. By Thursday the temperature dropped seven degrees, and by Friday the temperature dropped another two degrees." Although there may be a lot of accurate information in these two sentences, the reader still cannot tell whether it is hot or cold outside. All of the information is given as subtractions, the difference between one day's temperature and the next. We don't know whether the temperature was below zero throughout this time period or whether these facts describe the temperature changes during a very hot week in July. All we know are the relative ups and downs. Likewise, in a bipolar montage, when we note pen deflections telling us that voltages are getting more negative or more positive as we travel along a chain, we do not get *absolute* voltage information.

At first glance, if the bipolar montage recording system were as poor as the temperature reporting system given in this example in which we could not tell a hot day from a cold day, how is it that bipolar montages are useful at all? The answer is that, when we read an EEG page, we make an implicit assumption that the average baseline of everything that we see printed out is near neutral voltage (or "zero"). Indeed, because of the laws of electricity and charge, it is quite unlikely that *all* scalp areas will be either strongly negative or strongly positive for a prolonged period of time. Therefore, as we read, we are making a subconscious assumption that

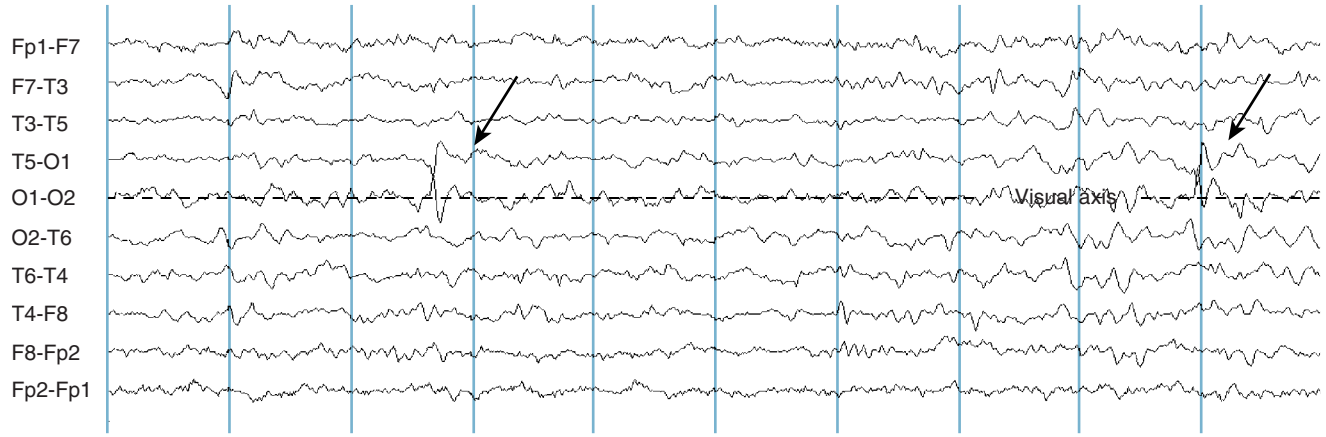


Figure 5-8 A circumferential bipolar chain is shown. Note the spike-wave discharges (arrows) phase-reversing in the O1 electrode. It is clear that the discharge represents an asymmetrical event because it is confined to one side of the visual axis, which is represented by a dashed line.

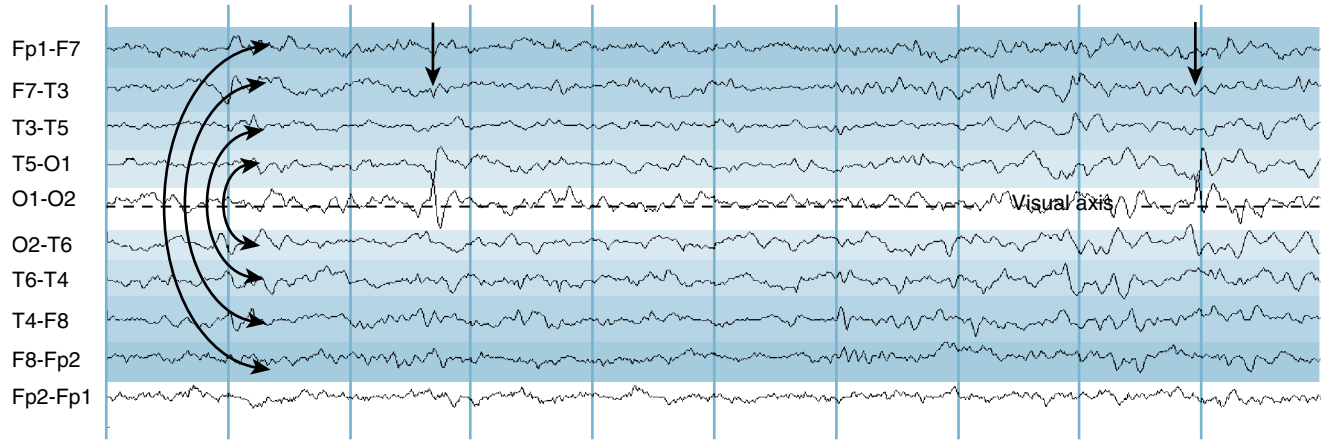


Figure 5-9 The same page of EEG material from the previous figure is shown with arrows and shading that suggest a method for visual scanning of a circumferential montage. Comparisons are made about the inner (white) channel with the dashed line, which represents the "visual axis." Like-shaded areas represent homologous areas of the brain. For instance, higher voltage slowing in one dark blue band compared with its counterpart on the opposite side would suggest an asymmetry of that slow activity between the hemispheres.

the average of everything that we see on the page is near zero. Nevertheless, we do lose absolute voltage information. We cannot know whether certain events may be slightly positive or slightly negative just by looking at the bipolar montage; we can only assert that an event is, for instance, "more negative" than the areas around it. For this reason, absolute measurements of voltage cannot be made as easily as they can be in a referential montage or, if they are made, they mean something different. When a reference electrode is used, we are making the implicit assumption for the purposes of measurement that the voltage at that electrode is neutral or zero (although we might shy away from making this assumption at a time that there is clearly a lot of noise in the reference electrode). This assumption is shakier when made in a bipolar montage, a system in which we only see voltage differences between nearby points on the scalp rather than absolute voltage measurements.

An extension of the problem of not knowing the absolute magnitude of the voltage at any given point when reading in a bipolar montage is the tendency for cancellation of like activity in adjacent electrodes. Here, again, we can imagine a case in which there is a posterior slow wave more or less equally represented in the three posterior electrodes: C3, P3, and O1. In such an example, when recording with a bipolar montage, this posterior slow wave could cancel itself out to a large extent in the areas where it is at highest voltage, simply because it happens to be equally represented between the pairs of electrodes being compared (e.g., C3-P3 and P3-O1). This effect is illustrated in Figure 5-10. This effect does not occur in referential montages.

This is one of the main disadvantages of bipolar recordings—that adjacent, in-phase activity tends to cancel out. This can cause a paradoxical effect: disappearance of a wave in an area where its voltage is highest and appearance of that wave only when a chain

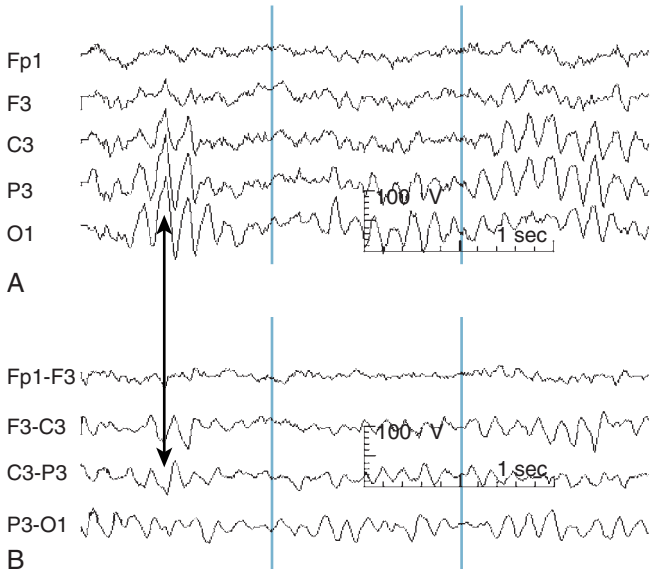


Figure 5-10 **(A)** EEG activity displayed in a referential montage and **(B)** shows the exact same activity displayed in a bipolar montage. Panel A shows fairly prominent alpha activity, especially in the three posterior channels (C3, P3, and O1) during the first (arrow) and third seconds. Note that this posterior-predominant alpha activity is relatively difficult to see in Panel B when the subtractions for the bipolar montage take place. The mathematical reason for this becomes evident when analyzing the alpha waves shown at the upper point of the double-headed arrow. Because the phase and shape of these waves are seen to be quite similar in the C3, P3, and O1 channels in the referential montage (Panel A) there is significant cancellation in the bipolar display shown (Panel B). In fact, these waves can barely be recognized in the P3-O1 channel, even though they are strongly present in both P3 and O1. Because of effects such as these, it is preferable to confirm subtle voltage asymmetries seen in bipolar montages by checking a referential montage.

moves into an area where it begins to disappear. It is in the comparison of the area where the wave is present to the area where it disappears that the "difference" is picked up by the bipolar recording technique, not necessarily the area in which the voltage is highest. Some of these effects are also illustrated in the exercises in Chapter 4 on localization. Fortunately, in practice this effect is not as big a problem as it might seem to be, because like activity in adjacent electrodes is often out of phase (not perfectly lined up with the wave in the adjacent electrode) and therefore does appear in the "correct" location in bipolar montages after subtraction. Still, it is wise to confirm a mild voltage asymmetry noted in a bipolar montage by reexamining it in a referential montage to exclude this potential shortcoming of bipolar recordings.

Techniques for Visual Scanning of Montages

AP Bipolar Montages

After you are familiar with the montage set used by your laboratory, it is useful to develop a visual scanning strategy for each montage in the set, a visual method or thought process for looking at each page of EEG that allows quick determination of interhemispheric or anterior versus posterior asymmetries. The first example we will look at is the standard AP bipolar montage used most frequently in the examples in this book. This AP bipolar montage conforms to the conventions suggested by the American Clinical Neurophysiology Society (ACNS) in that it uses the left-over-right and front-to-back conventions. Figure 5-11 uses shading to demonstrate the technique for detecting asymmetries. An axis should be imagined through the two midline channels, which have been left unshaded and represent the cranial midline. Next, the two inner (parasagittal) chains, which are lightly shaded in blue, should be compared. Is there more slow activity or fast activity in one light blue area compared with the other light blue area? If so, a parasagittal asymmetry is likely. Similarly, is there an asymmetry of activity between the more darkly shaded outer (temporal) chains? If so, a temporal asymmetry may be present.

Other laboratories may use the left parasagittal/right parasagittal/left temporal/right temporal ordering for the AP bipolar electrode chains. If so, the scanning strategy is adjusted accordingly. Figures 5-12 and 5-13 show a comparison of the two most common arrangements for the AP bipolar montage. Neither is technically superior to the other because both include the exact same set of channels, simply presented in a different order, and each will be commonly encountered in different laboratories.

Transverse Bipolar Montages

The transverse bipolar montage can be scanned in a fashion that is somewhat similar. Again, note the shading of each group shown in Figure 5-14. In this text, most illustrations of the transverse bipolar montage include a white space (a "gap") inserted between each chain, making it visually simpler to scan, although the practice of inserting these gaps is not common to all laboratories. Gaps make it easier to see the groupings of individual electrode chains, but they sacrifice space on the page and therefore diminish the amount of line height that can be assigned to display each channel. The dashed lines in the figure suggests the axis around which each electrode chain should be visually scanned. If channels look different in the area above the axis compared with the area below it, this suggests a difference in that chain between the left and the right sides of the brain. The reader should practice visualizing the superimposition of this shading on the page to help quickly determine which part of the page corresponds to which part of the brain.

Referential Montages

Scanning referential montages that use the alternative method of left–right channel pairings is fairly straightforward in that odd channels are left-sided and even channels are right-sided, but there is no simple method for picking a line on the page and quickly ascertaining the area that that channel corresponds to on the brain surface without referring to the channel labels. This is the advantage of the second system of electrode ordering for referential montages, which mimics the

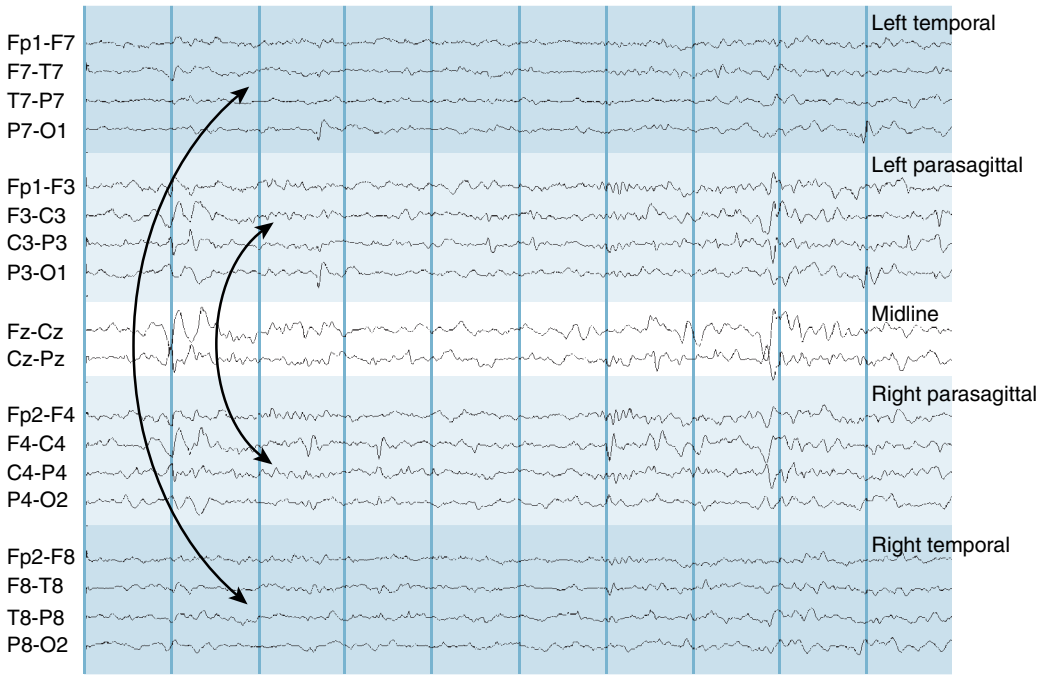


Figure 5-11 This schematic shows the visual scanning strategy used to compare homologous areas of the brain for the standard anteroposterior bipolar montage most commonly used in this text. The anteroposterior chain groupings progress from left to right as the eye scans down the page. The outer channel groups, shaded darker blue, represent the temporal areas and the inner channel groups, shaded lighter gray, represent the parasagittal areas. The unshaded strip of two channels in the middle of the page corresponds to the midline of the head. Note that the sleep vertex waves in the second and eighth seconds manifest as bursts of sharp waves seen fairly symmetrically in both parasagittal areas and in the midline but do not involve the temporal chains, as expected for vertex waves of sleep. By contrast, nonsymmetrical scattered spikes are seen in other locations. Identify the low voltage spikes at C3, O1, and C4.

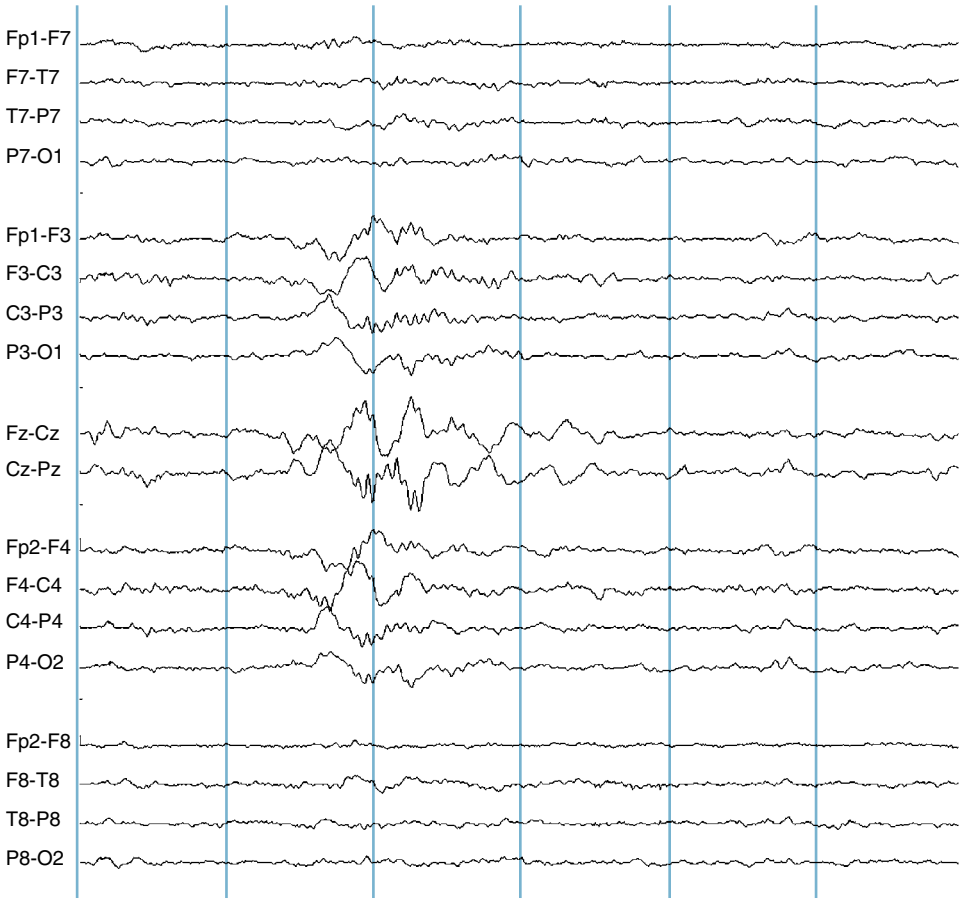


Figure 5-12 An example of vertex waves of sleep and sleep spindles is shown in the standard anteroposterior bipolar montage most commonly used in this text. The reader should appreciate that the topography of the page going from top to bottom corresponds to a left-to-right sweep across the head, as if from left ear to right ear. A main advantage of this system is that chains that are adjacent on the head are adjacent on the page.

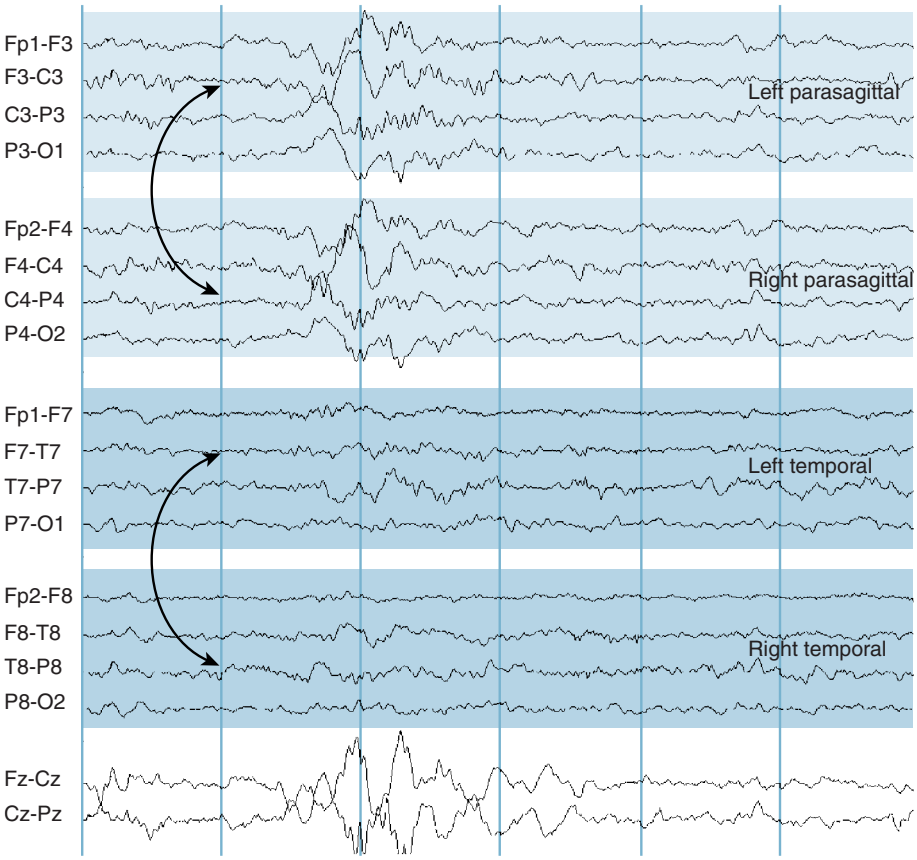


Figure 5-13 The same page of EEG as in the previous figure is shown in an anteroposterior bipolar montage using another common ordering of the chains. The shading and arrows imply visual comparisons of homologous areas. The top two sets of four channels correspond to the left and right parasagittal areas, and the bottom two sets of channels, shaded darker blue, correspond to the left and right temporal chains. In this setup, the midline channels are at the bottom of the page. This system may facilitate comparison of like areas but requires the eye to jump around the page more to localize events because topographically adjacent areas are no longer adjacent on the page.

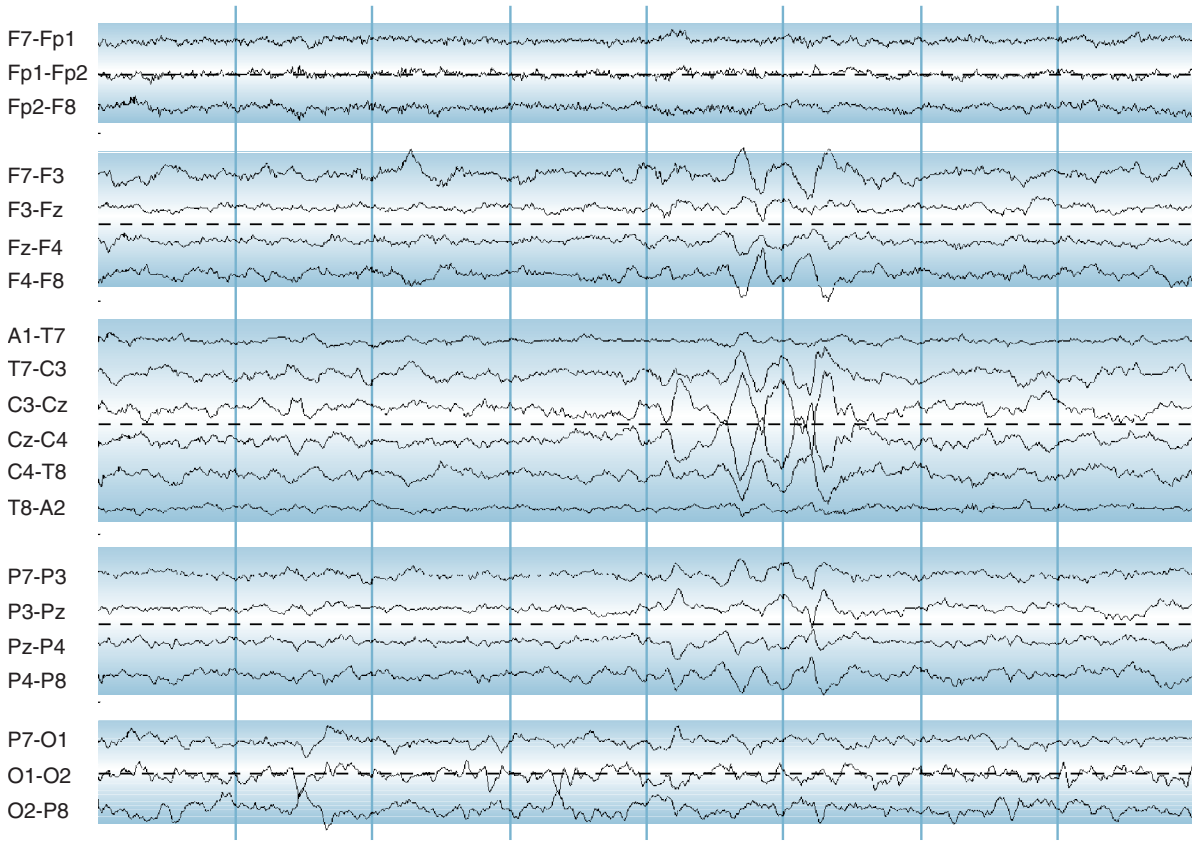


Figure 5-14 The figure shows an example of Stage II sleep displayed in a transverse bipolar montage. Each of the six chains is shown with gradient shading denoting the distance of each channel from the central visual axes, which are indicated by the dashed lines. In each grouping of channels, areas of like shading are compared with one another to establish symmetry. An event occurring above the visual axis but not below the axis in a group, be it a slow wave or a sharp discharge, suggests an asymmetry of that event between the hemispheres.

ordering used for AP bipolar montages: the brain location of any channel can be easily ascertained based on its position in the clusters of channels on the page.

Montages for Special Situations

The montage setups just described represent strategies useful for standard situations. When a patient has a surgical incision or some other obstruction to electrode placement, the technologist must often decide how to rework a montage "on the fly" to make a useful display of the data despite the unavailability of certain electrodes. Standard montages must also be modified when additional electrodes are used. When doing so, keeping interelectrode distances consistent, if possible, can eliminate the effect of a channel manifesting a higher voltage simply because of increased interelectrode distance. In some situations, though, variation of interelectrode distances may be unavoidable. The question of whether a voltage change is the result of a variation in interelectrode distance can usually be resolved by analyzing referential montages which are not sensitive to variations in interelectrode distance.

Regarding the use of additional electrodes, the most common "extra" locations added are the T1 and T2 electrode positions, which are designed to record from the anterior temporal lobe. The T1 and T2 names represent old nomenclature and correspond approximately to the FT9 and FT10 electrode positions of the modified 10-10 electrode system. A variety of methods can be used to incorporate these additional electrodes into a display montage. An example montage is shown in Figure 5-15. Use of these special temporal electrodes has to a great extent supplanted the use of more invasive electrodes, such as sphenoidal and pharyngeal electrodes.

The Choice of the Reference Electrode (Revisited): "More Important Than It Seems"

As described earlier, the choice of the reference electrode can have a large impact on the appearance of the resulting EEG tracing. Recall that, when looking at a channel labeled Fp1–reference (abbreviated as *Fp1–ref* in this text), there is an implicit suggestion that the signal we are seeing represents activity from Fp1. In the best of all possible worlds, this is true (because in the best of all possible worlds, the reference electrode is completely indifferent or neutral). It is worth keeping in mind, however, that the "electrode of interest," Fp1, and the reference electrode have the same potential to contribute to the appearance of the final output; neither has "mathematical" primacy over the other. The Fp1–ref channel only successfully represents Fp1 activity when the reference is quiet. Under what circumstances is the reference not quiet, and how might this affect our choice of reference for any given patient at any given time?

The importance of being aware of what reference electrode is in use at any given time when interpreting a referential montage cannot be overstated. There are several reasons for this, but two are particularly important. First, if there is noise in the reference electrode, that noise will appear in every channel in which that reference electrode has been used. Remembering that the reference electrode has as much potential to affect the tracing as the "electrode of interest," whatever noise appears in the reference will likely appear in the output signal. Because different reference electrodes are more likely to generate specific types of artifact, knowing which reference electrode is in use helps the reader correctly reject certain waveforms as artifact. Therefore, if a wave is seen in a channel, the reader must ask whether it comes from the electrode of interest or from the reference electrode. This thought process must be used for all waves of significance identified in the EEG when referential montages are used.

The second, more complex type of problem arises when the reference electrode itself is picking up brain wave activity. A commonly used reference, the earlobe, is well known to pick up a portion of temporal lobe activity in many patients. What impact might this fact have on the display of a temporal lobe discharge when an earlobe reference is used? As you may have guessed, when the reference electrode is "active" with a discharge, it will tend to cancel that discharge in the temporal lobe channels when the subtractions take place; the resulting display will appear to understate the voltage of the discharge. In the most extreme example, if the discharge is picked up equally well by the earlobe reference and T7, the discharge could conceivably be completely cancelled out in a T7-earlobe channel.

There are three scenarios to consider in the undesirable situation in which the reference electrode picks up an active discharge. In the first, the "electrode of interest" is recording a discharge and is being compared with a reference electrode that is also recording the discharge (at least partially). In the second, the "electrode of interest" is in an electrically quiet area (such as the right occipital area in the above example), but the reference electrode (the earlobe) is picking up a portion of the discharge. In the third there is some other type of electrical noise contaminating the reference electrode. Each of these possibilities is discussed below.

Figure 5-16 shows examples of the first two situations and illustrates the effects of increasing spillover or contamination of the reference electrode (in this case, the left earlobe) of a discharge picked up by T7 (the left midtemporal electrode). It also shows the potential pitfalls of using the earlobe reference to display the activity at a distant, inactive location such as O2 (the right occipital electrode) when there is contamination of the reference. Because the discharge does not involve O2, the reader would expect that the O2 channel will be flat, but this is not the case when an earlobe reference (A1) that is contaminated with an active discharge is used in a channel (O2-A1) to display O2 activity. This is a realistic example because the ipsilateral earlobe often does have brain wave activity in common with the left midtemporal area. An example of a third type of situation in which unrelated noise contaminates the reference electrode is shown in Figure 5-17.

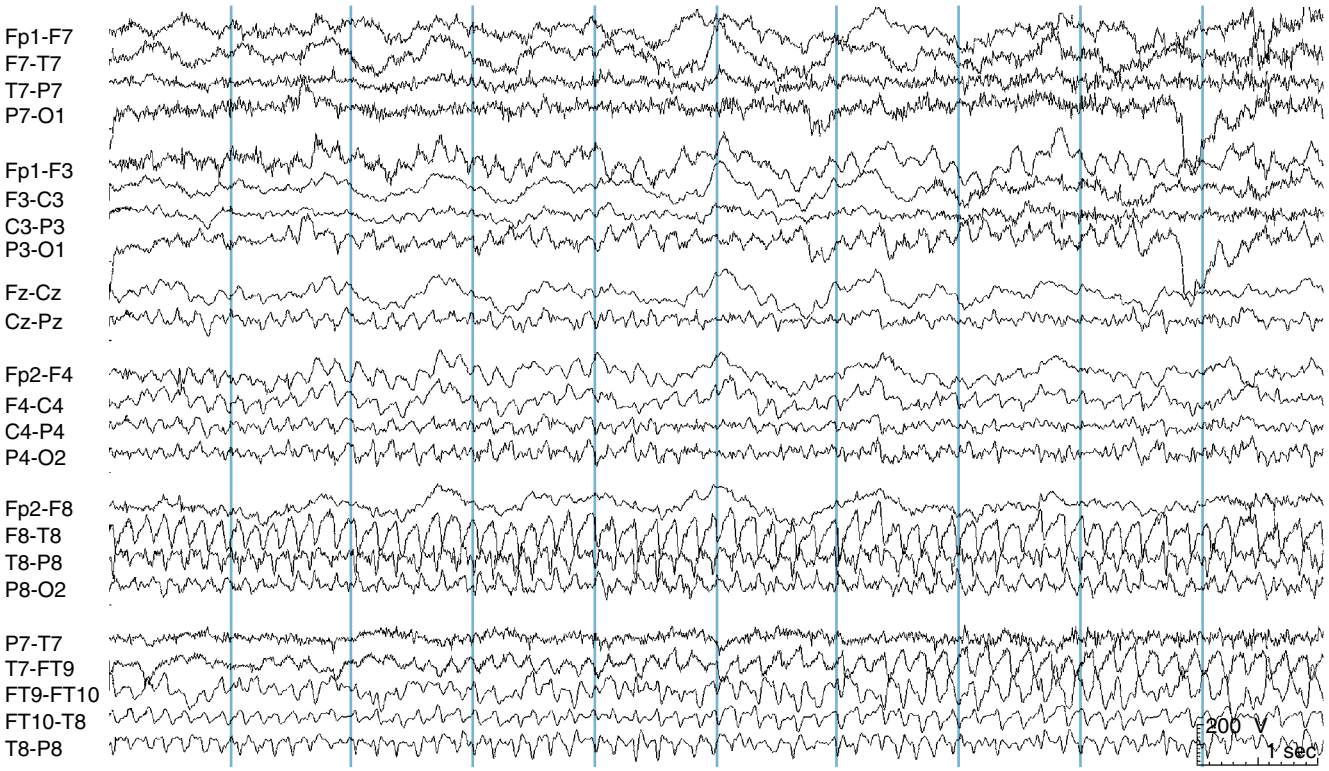


Figure 5-15 This EEG page shows an actual electrographic seizure discharge recorded with additional FT9 and FT10 electrodes (similar in position to the T1 and T2 electrodes used in other laboratories). The top 18 channels are identical to the standard anteroposterior bipolar montage used elsewhere in this text. The bottom five channels consist of a loop that includes the additional temporal electrodes. This seizure discharge is initially picked up well by electrodes at T8, P8, and FT10, as well as other nearby electrodes. The loop of five electrodes at the bottom of the page can be visually scanned imagining a visual axis through the middle FT9-FT10 channel. Note in this case that the seizure discharge begins below the axis defined by the FT9-FT10 channel (beginning of the page) implying that it has started on the right side of the brain. By the end of the page, the discharge is seen both above and below the axis, implying that it has become bilateral.

The Average Reference: Strengths and Weaknesses

The "average reference" montage is a favorite among some electroencephalographers but disliked by others. In practice, montages that use the average reference are neither "good" nor "bad"; in some circumstances they are very useful, whereas in others their disadvantages are so substantial that they can lead to serious reading errors and should not be used. The average reference is a simple arithmetic average of all the electrodes on the scalp chosen to be in the average. As expected, it can be calculated by summing the voltages measured by all of the electrodes used and dividing by the number of electrodes. Although the average reference can be defined to use all of the scalp electrodes, sometimes certain noisy electrodes may be excluded from the average, such as Fp1 and Fp2, which are often contaminated by high voltage eyeblink artifact.

When considering the possible value of the average reference, the first impression may be that the average reference represents brain activity and that perhaps it is a bad idea to subtract brain activity away from the signal being displayed. The counterargument to this is that the brain activity in all scalp electrodes, when averaged, should tend to self-cancel leaving a relatively "clean" average that is near zero. This condition does hold much of the time but, as we shall see, not all of the time. Another potential strength of the average reference is seen when an identical noise signal is present in all electrodes, a situation that is not uncommon. In such cases, that signal will remain represented in the average reference and be available to cancel out that same noise signal in the "electrode of interest." For example, a 20-µV "spike" of noise contaminates every scalp electrode; it will then average to 20 µV in the average reference and is subtracted from every electrode of interest in an average reference montage effectively removing the spike. When will each of these conditions hold true, and when will they not?

When reading an EEG tracing in an average reference montage, a good strategy is to estimate what the average reference signal should look like at any moment. Considering all the channels together, the average reference can be visually estimated by mentally summing up the voltages of all the channels, noting the relative strength of upgoing waves compared with downgoing waves and predicting the average. It is clear that when the various channels are of low voltage and there is not much activity, the average reference, too, will be of low voltage and relatively neutral. At such times, the average reference generally gives a very clean and useful comparison. What happens, though, when there is a lot of activity in the EEG? In the case of a very focal spike, when it is averaged among the large number of inactive electrodes, that spike's voltage becomes diluted

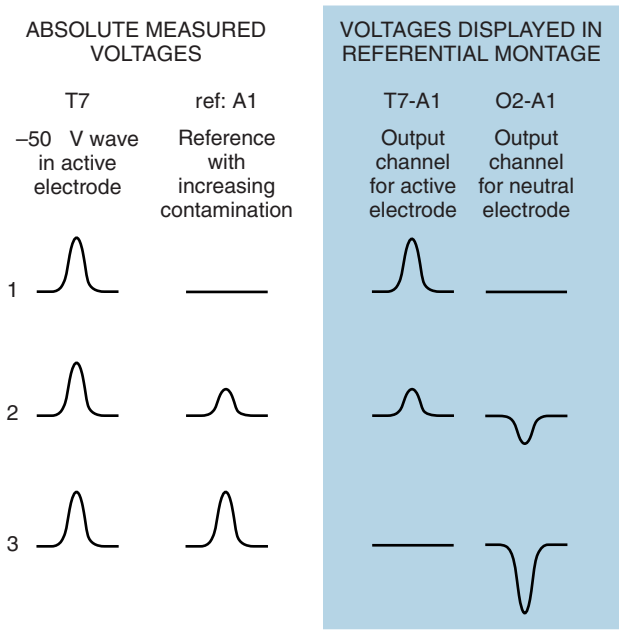


Figure 5-16 This figure illustrates how increasing amounts of contamination of the reference electrode (A1, or the left earlobe, in this example) affect the appearance of the output channels for both an electrode that is "active" with a discharge (T7 or the left midtemporal electrode in this example) and for a theoretically neutral electrode (O2 or the right occipital electrode in this example). The examples shown on rows 1, 2, and 3 all assume a sharp wave discharge of the same strength in T7 (depicted in the first column) however, the amount of the discharge picked up by the A1 reference electrode (the amount of "contamination" of A1) increases in each row (depicted in the second column). By the bottom row, the amount of the T7 discharge that spills over into A1 begins to match the voltage picked up by the active electrode, T7. The first two (unshaded) columns show the "true" amount of voltage change present in the T7 and A1 electrodes; the O2 electrode is assumed to be neutral. The second two (shaded) columns show the appearance of the output channels for T7-A1 and for the distant and inactive right occipital area, O2-A1 as they would appear on the EEG screen. Each channel output is based on the subtraction of the voltage of the second electrode in the pair from the first, as usual.

Row 1 shows the simple case in which T7 is active and the A1 reference is neutral or "clean." In this case, the T7-A1 channel shows an accurate representation of the activity at T7. There is no problem in the O2-A1 channel; O2 is quiet and the A1 reference is clean, so the O2-A1 channel accurately portrays the lack of activity at O2. In Row 2, the activity at T7 is unchanged, but now the A1 reference picks up some 50% of the T7 discharge. Now the T7-A1 channel will display the discharge, but only at one half its actual height. Worse, however, is the fact that a "flipped" version of the discharge is seen at O2-A1, even though O2 is an area that we know to be electrically inactive. Finally, we consider the example of Row 3 in which the A1 reference electrode picks up the discharge just as well as the "active" T7 electrode. The channel outputs are now quite misleading; the discharge is absent from the T7-A1 channel (even though we know this to be a T7 discharge), and now the O2-A1 channel, which is presumably representing the activity in the quiet right occipital area, has a large (but "flipped") discharge because of the contaminated A1 reference. A combination of a careful eye and review of other montages (including bipolar montages) may be necessary in order not to be led astray when real-life EEG discharges resemble the examples shown in Rows 2 and 3.

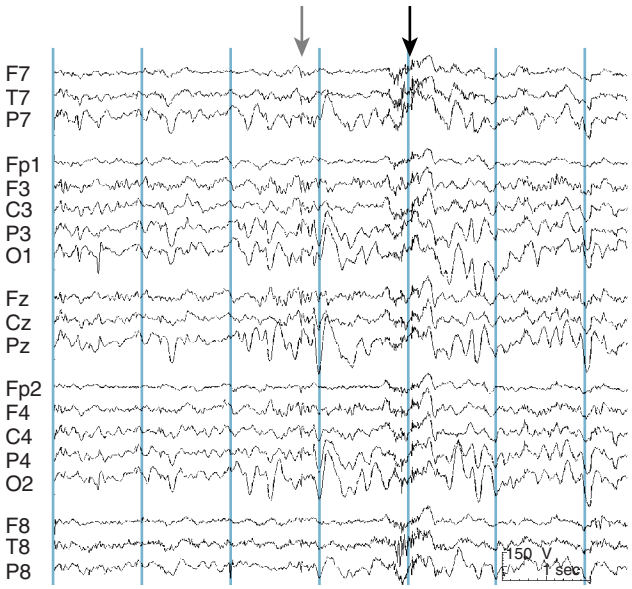


Figure 5-17 This EEG page is displayed in a referential montage with the nose used as the reference. When there is electrical noise in the reference, it will appear in every channel in a more or less identical fashion. The light gray arrow shows a single, low-voltage transient, the origin of which is likely the nose. Note that this transient manifests the same shape and has similar polarity and voltage in all channels. Likewise, a burst of muscle artifact is seen in all channels one second later (dark arrow), also likely of nose origin because of similar morphology in all channels. The higher voltage of muscle artifact in the T8 channel probably represents a mixture of muscle activity in the nose and additional muscle activity from the T8 (right midtemporal) area.

significantly when averaged with the quiet electrodes and may have little visible effect on the average. What if the spike is present in several electrodes, or what if the spike is of particularly high voltage? As an electrical scalp event rises in voltage or is present in an increasing number of electrodes, it will clearly affect the average reference to an increasing degree. When discharges on the scalp make the average reference "active," it is important to remember that this activity will now be subtracted from every "electrode of interest" in the montage.

Consider the simple example of a 20-electrode set, and, at one point, the reader notes that two of the channels measure −100 μV and the other 18 channels are flat (0 μV). The value of the average reference electrode at that time will be (−100 + −100) / 20 or −10 μV. The two electrodes that are active with the −100 μV spike will be compared with the −10 μV reference (−100 μV minus −10 μV = −90 μV) and display a −90 μV (upward) deflection, which is a satisfactory approximation of the true −100 μV spike. The same subtraction also takes place, though, for each of the inactive electrodes so that any inactive electrode's channel when compared with this average reference will show a small positive (downward) deflection of 10 μV, even though these electrodes are, in reality, neutral (Input 1 electrode equals zero and Input 2 electrode equals −10; Input 1 − Input 2 = +10 μV). This small amount of deflection may be tolerable, especially if it is expected by the alert reader who predicts that the average reference will be mildly negative at that moment on the basis of observation of all of the channels. A reader who does not take this effect into account could erroneously conclude that there is a low-voltage concurrent positive spike occurring broadly across the brain, which is not the case.

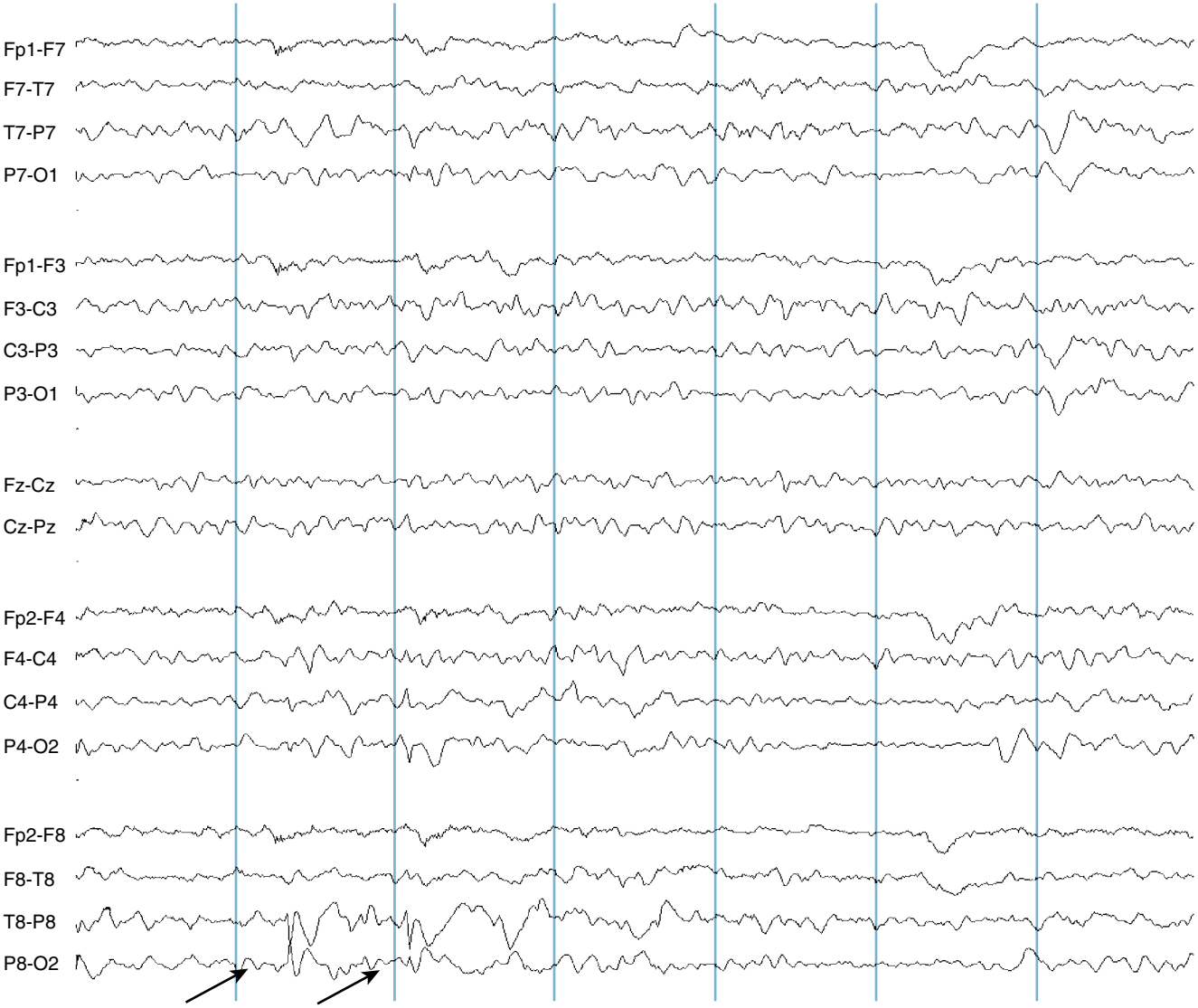


Figure 5-18 Two spike-wave discharges are seen phase-reversing in the bottom two channels (arrows). The technique of identifying the location of the phase reversal efficiently identifies the point of maximum of this discharge as P8, the electrode shared by the bottom two channels (T8-P8 and P8-O2). Because the waves' phases point toward each other, the spikes' phase reversal indicates a *negative* spike. A comparison to the appearance of the same discharge displayed in a referential montage is shown in the next figure.

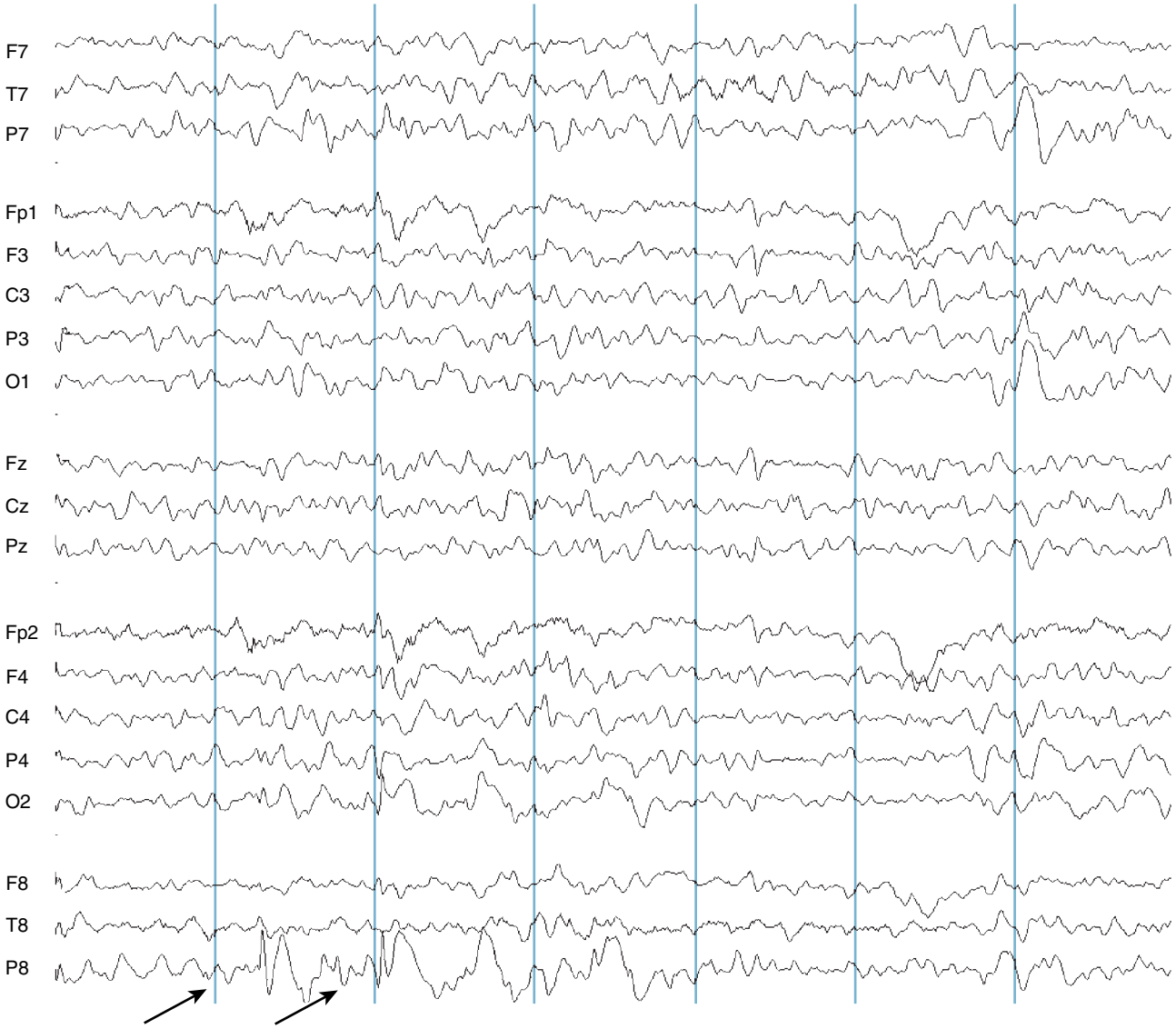


Figure 5-19 The same P8 discharges (arrows) from the previous figure are shown, this time in a referential montage in which the average reference is used. The localization technique for referential montages is used in which the maximum wave height, and therefore the discharge's maximum, is identified at P8, the bottom channel. Because the spikes are upgoing we can conclude that the spike is negative with respect to the reference. Because the discharge is so focal and not of particularly high voltage, it does not significantly affect the average of all the electrodes. Therefore, in this case, the average reference is a satisfactory choice of reference to display this discharge.

Figures 5-18 and 5-19 show an example of an effective use of the average reference. In this example, a spike in P8 is so focal that it does not contaminate the average reference to any significant extent and the average reference montage displays an excellent representation of the field of the spike. Figure 5-20 shows an example of a seizure discharge with a broad field across the right hemisphere that is clearly restricted to the right side of the brain, displayed in a bipolar montage. Considering what this discharge might look like if displayed in an average reference montage, the alert reader will note that the discharge involves many electrodes and would thus be expected to be visible in the average reference. Figure 5-21 shows the same page displayed in an average reference montage. Because the average reference is contaminated with the discharge, as expected, this montage gives the false impression that the seizure discharge is present on both sides of the brain.

A schematic of an idealized posterior-maximum spike is shown along with numerical voltages in Figure 5-22 to illustrate the mathematics behind contamination of the average reference. The top half of the figure shows the "true" discharge, and the bottom half shows what the display will look like if an average reference montage is used. A reader who only sees the bottom display and who does not understand average reference montages may not appreciate that the discharge represents a simple negative spike involving only the posterior half of the brain.

The average reference can be contaminated by nearly any type of EEG activity, not just by abnormal discharges. In some patients, normal activity such as vertex waves of sleep and sleep spindles are of high

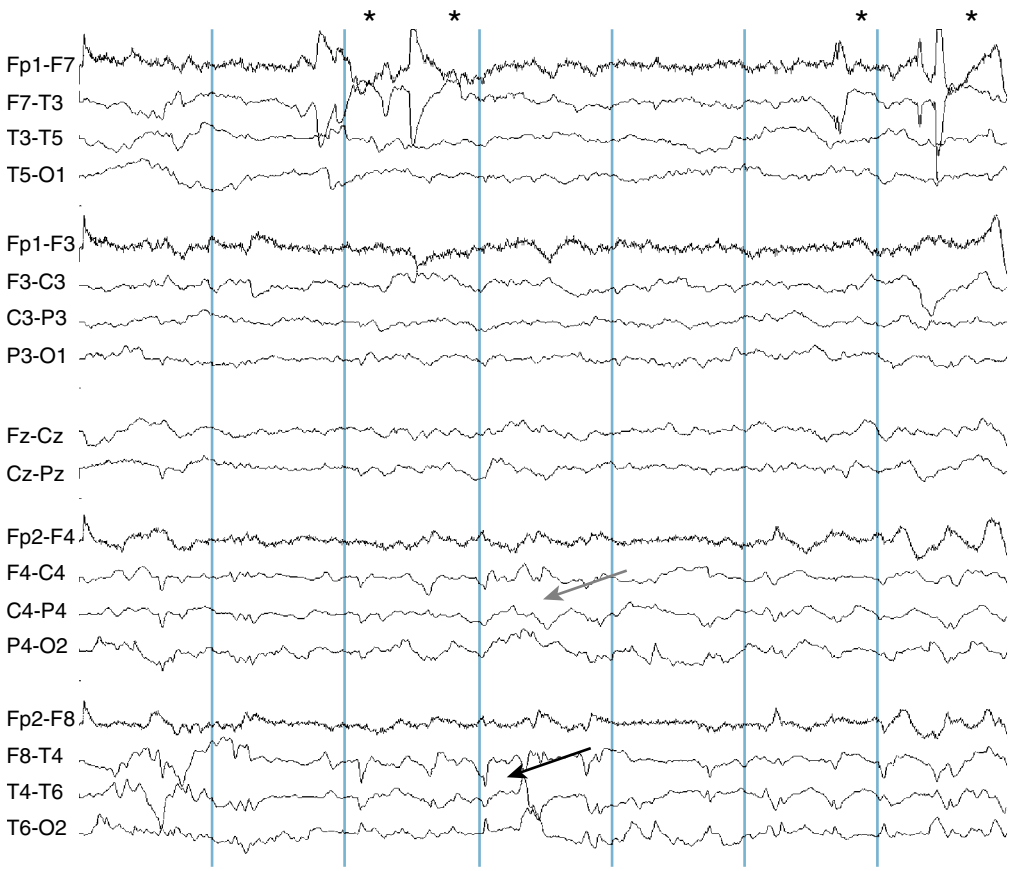


Figure 5-20 This figure shows a low-voltage, repetitive seizure discharge with maximum in the right mid- to posterior temporal area (black arrow). The field of the discharge spreads to include the right parasagittal area (gray arrow). Note that the discharge is not present in the left hemisphere (the top eight channels), however. Some high-voltage artifact can be seen arising from the F7 electrode (asterisks) related to a poor contact, seen in the top two channels and unrelated to the seizure discharge.

enough voltage and have a broad enough field they will affect all channels of an average reference montage, clearly an undesirable effect. Figures 5-23 and 5-24 show how even normal sleep activity can make a montage that uses the average reference confusing to interpret. Figure 5-25 shows the same page of EEG in an AP bipolar montage for comparison. The montage that uses the nose reference clearly shows the vertex waves and spindles in their proper locations, the midline and parasagittal areas; these waves are much less evident in the temporal chains. Because the spindles and vertex waves are widespread enough to contaminate the average reference, the average reference montage gives the mistaken impression that the field of the spindles and vertex waves extends strongly into the temporal areas, which it does not.

Detecting Contamination in the Average Reference Electrode

Because an average reference montage typically uses the average as the reference for every channel, it can be challenging to identify whether there is "contamination" in the average reference and what that contamination consists of. There are two simple visual techniques to identify contamination of a reference electrode.

Consider what you would expect to see if a cerebral discharge were to contaminate the average reference. The average reference is customarily attached to Input 2 of the amplifier. If the average reference happened to consist of an upgoing deflection (and keeping in mind that the CMR amplifiers used on EEG machines will subtract Input 2 from Input 1), this will result in a tendency manifest a downgoing deflection in the resulting output channel. In an example where many scalp electrodes detect a true upgoing (negative) brain wave, the average reference will become contaminated with that upgoing wave. When subtracted from the electrodes that are truly inactive, a "flipped" or downgoing version of the wave will appear in the inactive electrodes' channels.

Taking this phenomenon into account provides an effective technique by which to detect whether and in what way the average reference electrode is contaminated. The reader can seek out an electrode position that is assumed to be neutral and examine the channel from the presumed inactive location. For example, if there is strong suggestion that a discharge is coming from the left frontal area, the reader may wish to examine the channel representing the distant right occipital region and assume that it is neutral. If the channel that compares that neutral electrode to the average reference shows the discharge, but with its phase reversed or "flipped," this strongly suggests that the reference is

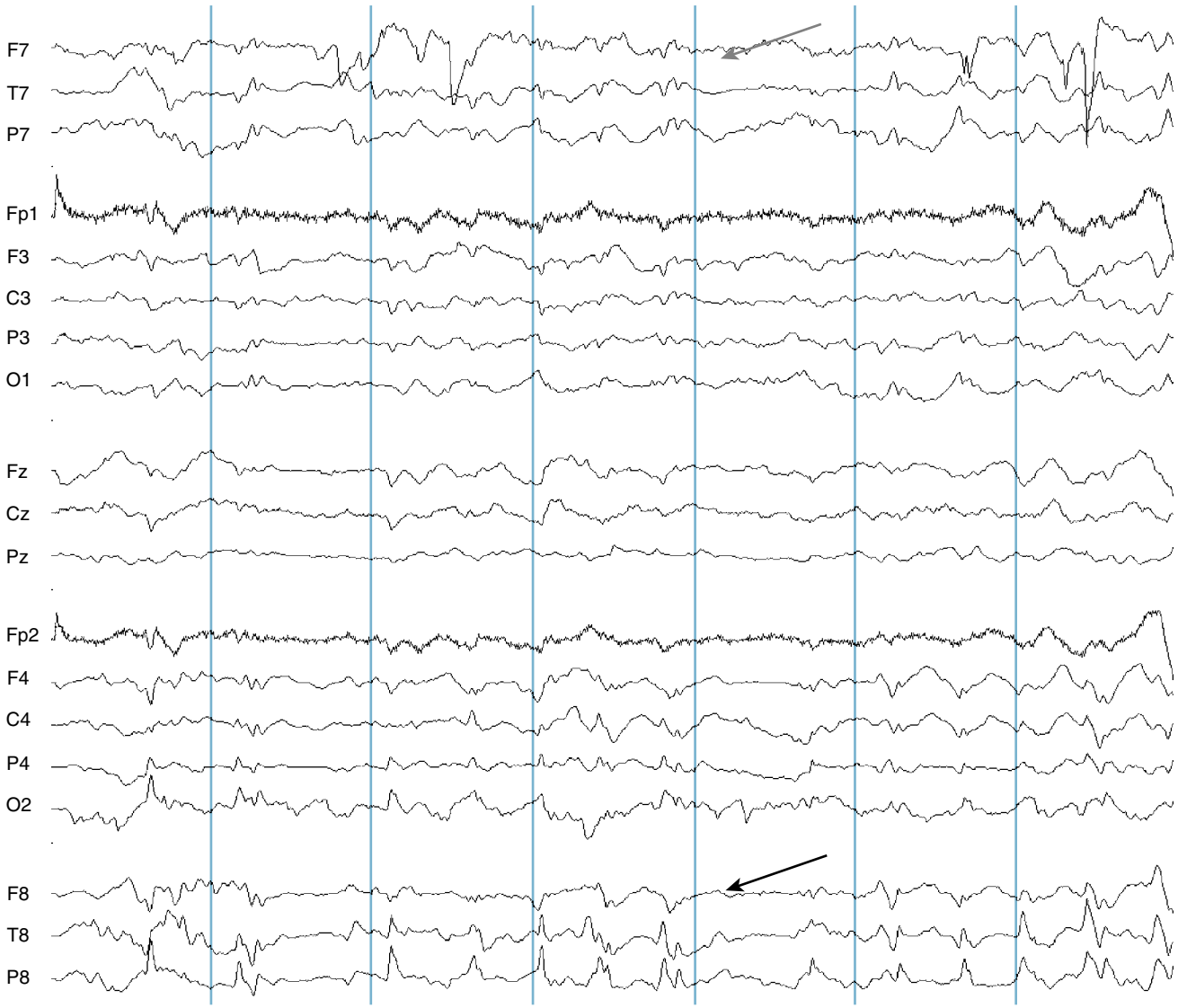
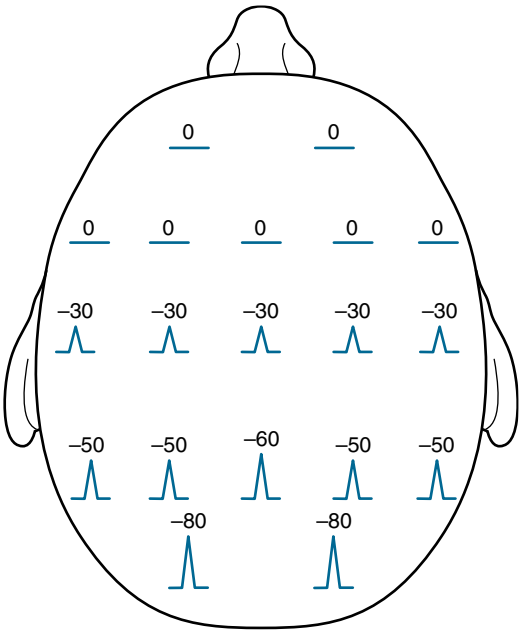


Figure 5-21 The same right posterior quadrant seizure discharge as shown in the previous figure is now displayed in a referential montage using the average reference. Because the discharge involves several channels, when all scalp electrodes are averaged, the discharge is actually noticeable in the average reference itself. Note that, even though the discharge is well seen in the channels in which we know it to be maximal, T8 and P8 (black arrow), and also to a lesser extent in the right parasagittal chain, the discharge now falsely localizes to areas in the opposite hemisphere, even the left temporal chain where we know it not to be present (gray arrow). This occurs because the contaminated average reference is being subtracted from the electrodes in the inactive left temporal chain. A clue to the presence of this phenomenon is the "flipped" phase of the apparent seizure discharge in the left hemisphere. A reader who does not realize that the average reference is in use and contaminated by the discharge could erroneously report that this seizure discharge involves both hemispheres of the brain.

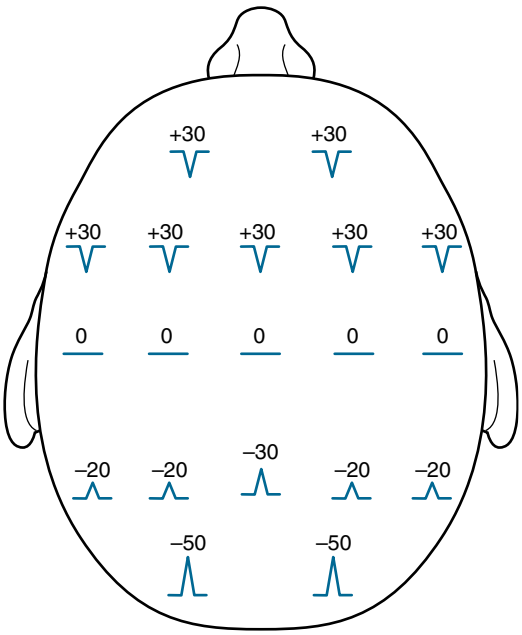
contaminated, as was shown in the bottom half of Figure 5-22. Therefore, when using an average reference montage, just because a deflection is seen across all channels, the reader should not simply conclude that the discharge is present in all those channels; rather, the possibility that the reference is contaminated must be considered, especially when the discharge's polarity appears to be reversed in several channels.

This line of reasoning can also be extended to the use of a standard (nonaveraged) reference electrode and is useful when the reference is close to the vicinity of the discharge and actually records a portion of that discharge. In such cases, the discharge should be seen in the channels that include active electrodes, but it will be somewhat attenuated; some of the discharge will be "subtracted away" because the reference electrode is also active. Again, and similar to what was described in the previous paragraph for the average reference, when the activity in the reference is subtracted from channels with neutral electrodes, a "flipped" version of the discharge appears in those locations in what can initially be a misleading, but ultimately informative fashion. Note that the effect of the "flipped" discharge also served as a clue to contamination of the average reference in Figure 5-21.

A related and dramatic version of the problem of the contaminated reference is seen in referential montages when the Cz electrode is chosen as the reference. In good electroencephalography laboratories, Cz is



A True field of a posterior-maximum spike displayed with an ideal, "neutral" reference



B The same posterior-maximum spike displayed with an average reference

Figure 5-22 The top panel **(A)** illustrates the true field of a spike discharge that is maximal in the occipital area with a field spreading forward to involve the posterior half of the head. The discharge does not, however, involve the anterior two rows of electrodes, which are shown in the diagram to measure zero voltage. The voltage measured at each electrode is given in microvolts. The bottom panel **(B)** shows the exact same spike, now displayed using an average reference. The resulting display can be predicted first by calculating the mean of the 19 recorded scalp electrodes, which comes to −30 μV (add all of the voltages in Panel A together and divide by 19), giving the voltage of the average reference. Because the average reference is attached to Input 2 of the CMR amplifier, its value will be subtracted from that of each Input 1 electrode. Arithmetically, the *subtraction* of −30 μV from each measurement is the equivalent of adding 30 μV ($x - -30\,\mu V$ is the same as $x + 30\,\mu V$). Because of the convention of "pen-down = positive," this subtraction results in each waveform being "lowered" by a height of 30 μV. Therefore, use of the average reference gives the unwanted effect of a broad apparent positivity in the neutral anterior electrodes ($0\,\mu V - -30\,\mu V = +30\,\mu V$). It attenuates the apparent amplitude of the middle row of electrodes so that they appear neutral as shown and also attenuates the values of the more strongly negative electrodes in the posterior two rows. The alert reader must notice the unexpected "flipping" of the phase of the waveform between the front and the back of the head and consider the possibility that this atypical appearance may be related to a contaminated average reference rather than a discharge that is positive anteriorly and negative posteriorly.

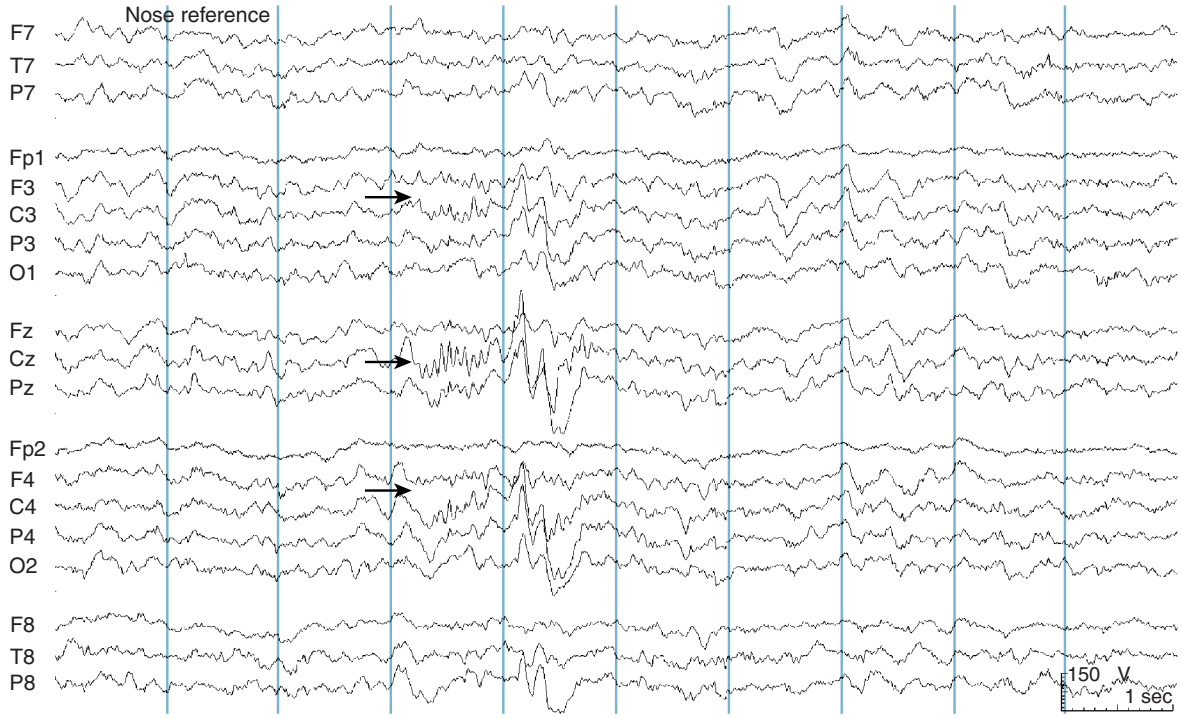


Figure 5-23 A page of EEG is displayed in a referential montage using the nose as the reference electrode. Stage II sleep is seen with vertex waves and spindles. Because the comparison electrode, the nose, represents a distant and indifferent reference point, this page gives an excellent representation of the field of both the spindles and vertex waves. As expected, the spindles are maximum in the central midline electrode, Cz, and also in the adjacent central C3 and C4 electrodes (arrows). The spindle field also extends forward to include F3 and F4. Likewise, the vertex waves seen in the following second have a similar field, maximum in the midline and in the parasagittal chains, but their field does not extend to the temporal chains to any great extent. Compare to the next figure.

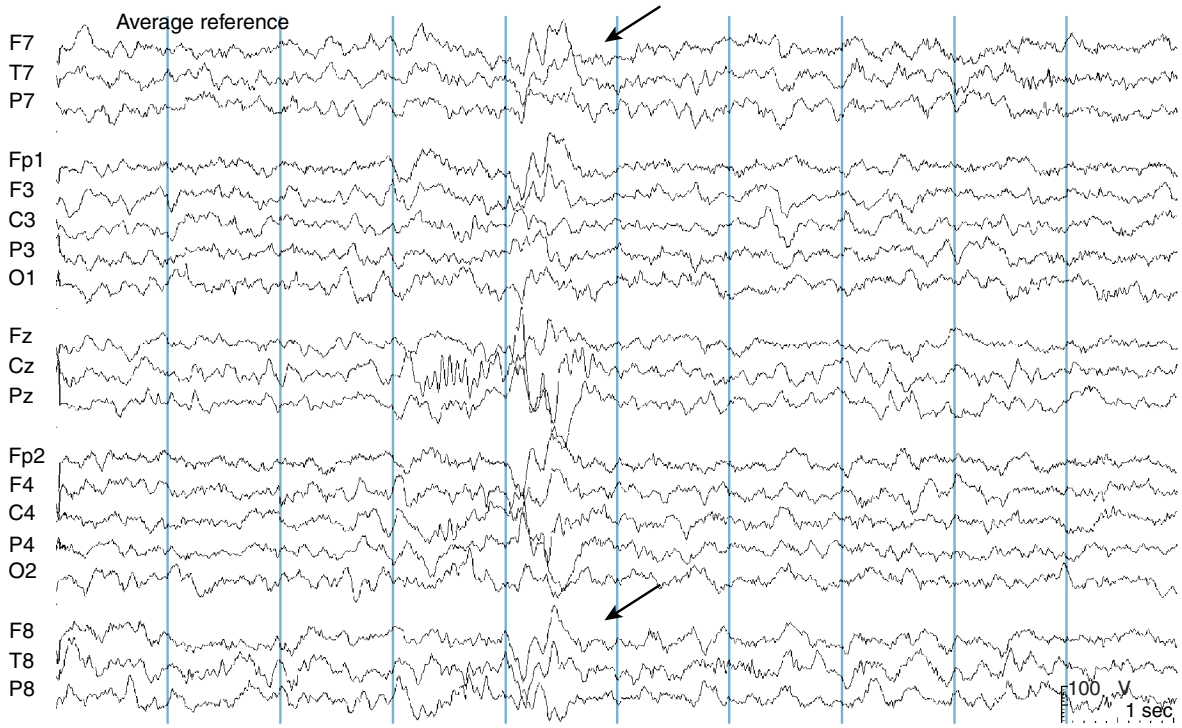


Figure 5-24 The same page of the EEG as shown in the previous figure is now displayed using an average reference montage. Although the spindles are still seen to be maximum in Cz, they now appear to have a much broader field and are even seen in the temporal chains. The reason for this misleading appearance is that the average reference is "contaminated" with the spindles. Even more misleading, the field of the vertex waves now appears to extend past the parasagittal areas to include the temporal areas (arrows). The apparent presence of the vertex waves and spindles in the left and right temporal chains represents a false localization, which is caused by contamination of the average reference with the vertex wave and spindle activity.

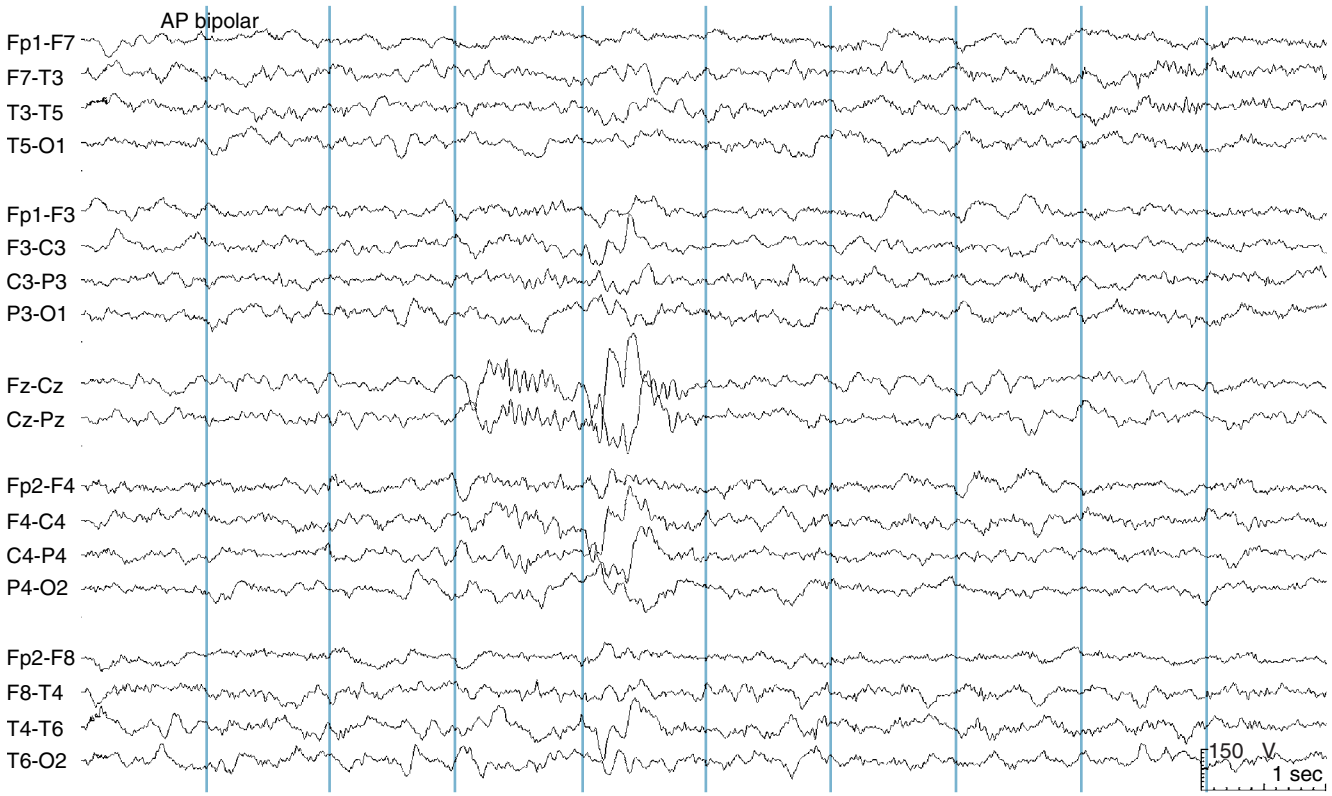


Figure 5-25 The same page of EEG as shown in the previous two figures is displayed this time with an anteroposterior (AP) bipolar montage. Note that on this page, the AP bipolar montage gives a fairly accurate representation of the fields of the vertex waves and spindles. The field of neither wave extends to the temporal chains (top four and bottom four channels).

almost never chosen as the reference electrode for the referential montage. Very occasionally, however, Cz is the only available choice in a very active patient because there is no scalp muscle to generate muscle artifact under this midline electrode. Using a Cz reference can help avoid contamination with muscle artifact, but what happens when the patient falls asleep? Sleep vertex wave activity is maximally represented in the Cz electrode, and spindle activity is also plentiful there. For this reason, the sleeping patient in whom a Cz reference is used will manifest vertex waves and spindles in every channel (see Figure 5-26). Such a tracing can be difficult to interpret and is essentially unsatisfactory for most purposes.

The second simple technique that can allow quick visual exclusion of a wave that arises from a contaminated reference is the phenomenon of a wave or signal that shows the exact same morphology in every channel. Higher voltage contamination will tend to have an identical appearance in all channels because that signal has been subtracted from each channel's output. Figure 5-27 shows an example of such easily identifiable contamination.

The Laplacian Reference

The Laplacian reference is based on mathematical expressions that attempt to model the spherical surface of the scalp. This type of reference is occasionally used in routine EEG recordings but has a number of disadvantages. The Laplacian reference is a type of average reference in which the weight of each electrode in the average is affected by its distance from the electrode of interest. Electrodes that are closer to the electrode of interest are given more weight in the average, and more distant electrodes are given proportionally less weight. Because the more distant electrodes may only have a small impact on this type of weighted average and also because the mathematical expression for the full Laplacian reference is relatively cumbersome (and differs for each electrode on the scalp), some labs use a simplified version of the Laplacian reference. In this abbreviated version, only the immediately surrounding electrodes (a maximum of four) are used in the average; the more distant electrodes only make small contributions to the Laplacian because of their reduced weighting, and thus their values are discarded. Even this simplified technique has its weaknesses, such as the problem of how to handle electrodes that are on the edge of the electrode array such as O1 or Fp2 (typically only three electrodes are averaged in such instances). Likewise, noise in just one of the surrounding electrodes can have a fairly significant impact on this type of reference.

While use of the Laplacian reference can produce some satisfactory recordings, there is a fundamental disadvantage to this technique. As stated earlier, a basic tenet of careful interpretation of a referential montage

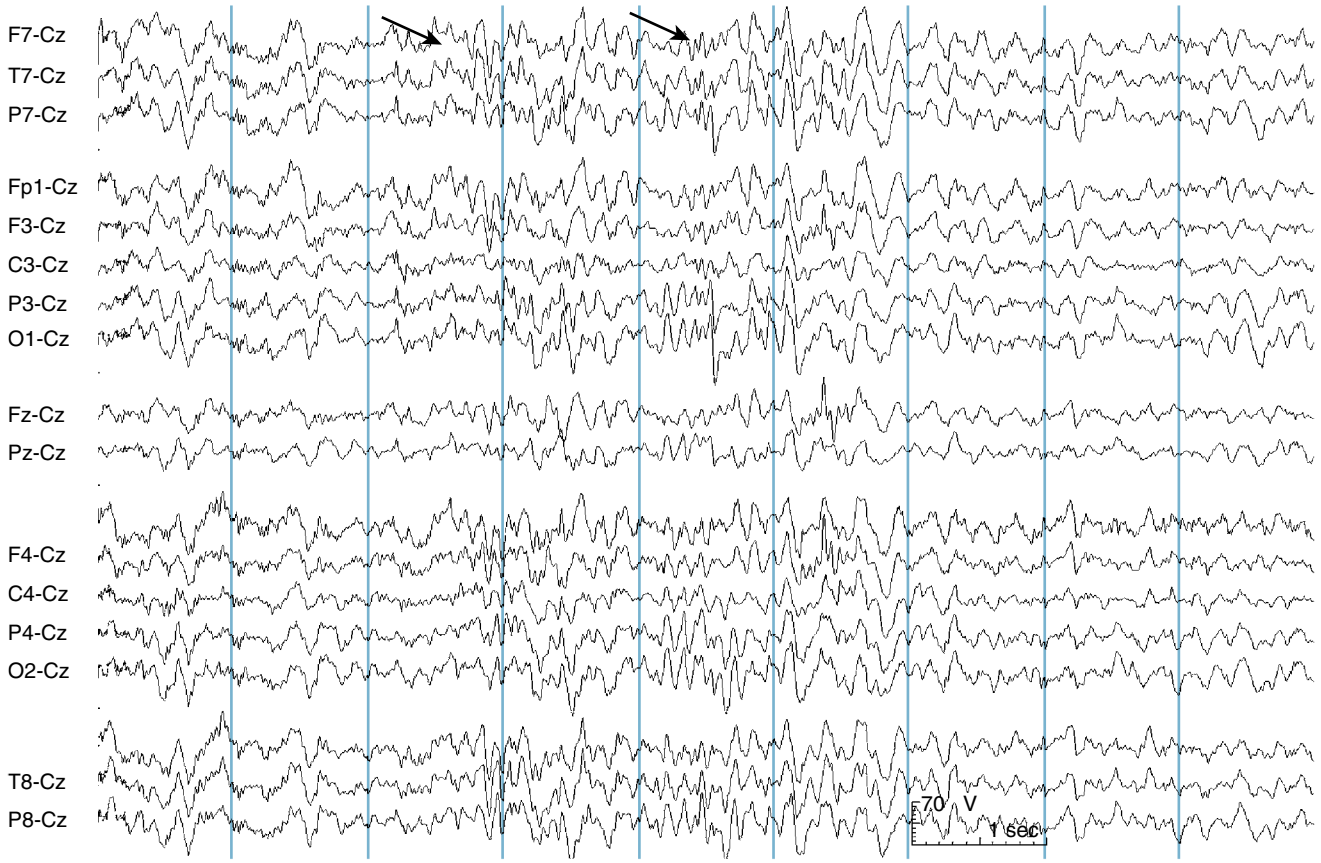


Figure 5-26 A page of EEG sleep is displayed using a Cz reference. Because the Cz electrode is active with spindle and vertex-wave activity during sleep, this electrode position makes a poor choice for the reference electrode. Note that spindles and vertex waves appear to involve all channels on the page (arrows). Ironically, because of cancellation effects, the spindles are least well seen in the channels where their fields are strongest, C3 and C4.

is the importance of always knowing the exact reference in use. The problem with the Laplacian reference is that the question of "what is the reference?" is a constantly moving target because for every scalp electrode the reference is different. The fact that each electrode has a different reference may either slow down the analytic process of EEG interpretation or give the reader the incentive not to consider what the true reference is for every given channel, possibly leading to sloppy interpretation of complex electrical events. For these reasons, some feel that the Laplacian reference contributes little in the way of advantages over the average reference but presents a specific disadvantage to a reader who appropriately needs to know the composition of the reference for all channels at all times.

Using Colors in Montages

Digital computer systems offer laboratories the option of displaying different channels in different colors. For instance, some laboratories may choose to display all channels related to the left hemisphere in brown and all channels related to the right hemisphere in green. Although this may seem a like a good idea at first, this information should already be readily available based on channel position on the page. Because the eye is being trained to look for differences, the use of different colors may actually become a minor stumbling block given that the different colors create the appearance of a difference at the outset. To some, a darker color may look bolder during visual scanning, and therefore "bigger" than a lighter-colored channel, introducing an immediate bias toward one hemisphere. The author prefers montage setups in which a channel's page position rather than its color is the indicator of brain position. Channel colors are most useful in the case of montages that alternate right- and left-sided channels. In general, when colors are used in montages, like colors are preferable for like brain regions.

The Appearance of Standard EEG Features in Different Montages

Each standard EEG feature has a characteristic appearance in the different EEG montages. The following figures review the appearance of eyeblink artifact, lateral eye movement artifact, the posterior rhythm, vertex waves of sleep, sleep spindles, and focal spikes in a variety of montages.

Figure 5-28 shows the typical appearance of eyeblink artifact in the AP bipolar montage. Because eyeblink

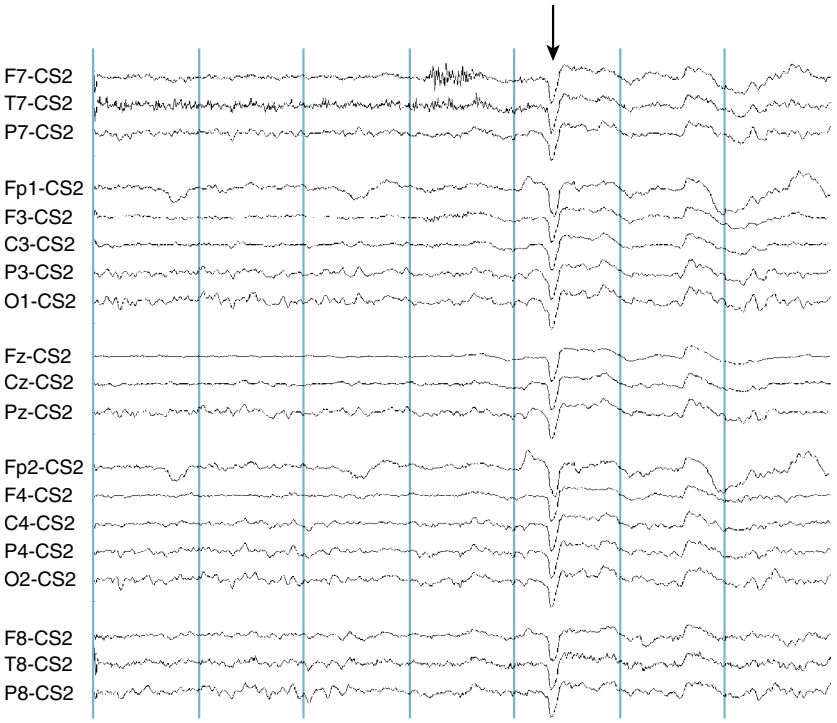


Figure 5-27 An identical downward deflection is noted in all channels in the fifth second of the tracing (arrow). When a waveform of identical shape and amplitude is seen in a montage in which all channels are compared to a single reference, the simplest explanation is that the deflection arises from the reference.

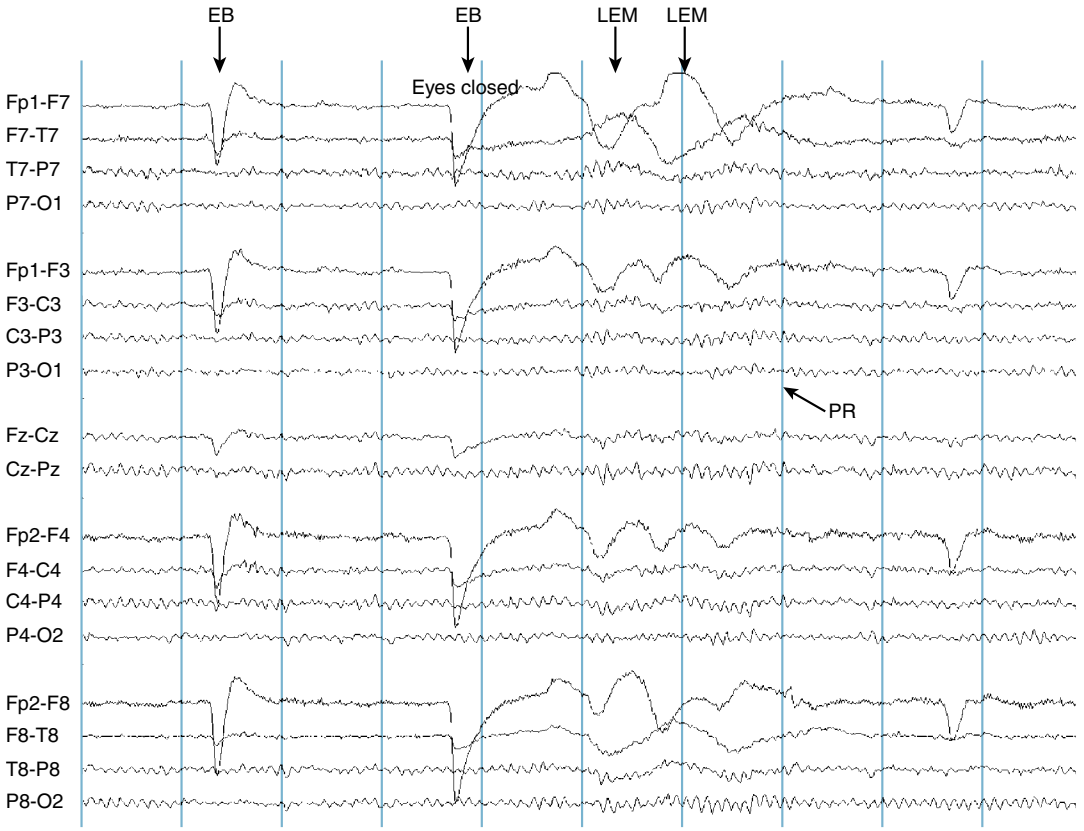


Figure 5-28 A page of wakefulness is shown in the **anteroposterior (AP) bipolar montage.** Eyeblink (EB) artifacts are the most prominent waveforms in this awake tracing, seen as a dramatic downward deflection most prominent in the top channel of each grouping of four channels in the AP bipolar montage. A smaller but similar deflection is also seen in the first channel of the midline electrodes (Fz-Cz). Eyeblink artifact is caused by an upward bobbing of the eyes that reflexively occurs during eye closure (Bell's phenomenon). Because the anterior aspect of the eye is positively charged with respect to the posterior pole, eyeblinks result in a net positivity being detected by the frontal electrodes. Because the gradient (voltage difference) is typically greatest for eyeblink artifact between the most anterior electrodes (Fp1 and Fp2) and those directly posterior to them (F7, F3, F4, and F8), the eyeblink artifact waveform is almost always of highest amplitude in the most anterior channels (e.g., Fp1-F3 and Fp2-F4), as seen in this figure. Typically, this artifact is also visible, but to a lesser extent, in the next more posterior channels (e.g., F3-C3 and F4-C4). Because of the anterior source of the eyeblink potential, it is unusual to see a deflection posterior to these channels. Indeed, as is seen in this figure, the eyeblink deflection is not picked up in channels such as C3-P3 or T8-P8. At the end of the sixth second, high-voltage waveforms show opposing phases on the left compared with the right (lateral eye movement [LEM]). This type of artifact is caused by lateral eye movements and is discussed in more detail in Chapter 6. PR = posterior rhythm.

artifact is caused by a large transient positivity in Fp1 and Fp2, the abrupt, downward-sloping waveforms seen in the four most anterior channels are characteristic for eyeblink artifact. The opposing phases seen in the anterior channels after the second eyeblink artifact are typical for lateral eye movements (see the figure caption for further explanation). In this figure, the posterior rhythm is seen in the posterior channels and even reaches as for forward as the F3-C3 and F4-C4 channels. Does this imply that the posterior rhythm is actually present in C3 and C4? The answer is no—the posterior rhythm is seen in F3-C3 because it disappears between F3 and C3. Because the two channels differ in that C3 picks up the posterior rhythm and F3 does not, the subtraction of the two channels still displays the posterior rhythm's waveform.

This same EEG page is shown in a variety of montages in Figures 5-29 through 5-35. It is worthwhile to take the time to identify each of the main elements of the awake EEG in each of these montages.

Figures 5-36 through 5-42 show the appearance of vertex waves, sleep spindles, and focal spikes from the same page of EEG recorded during sleep in the various montages (see figure captions for additional description). The top half of the figures show the EEG without markings to allow the reader to practice identifying key features. The bottom panel shows the same page with an "answer key" superimposed.

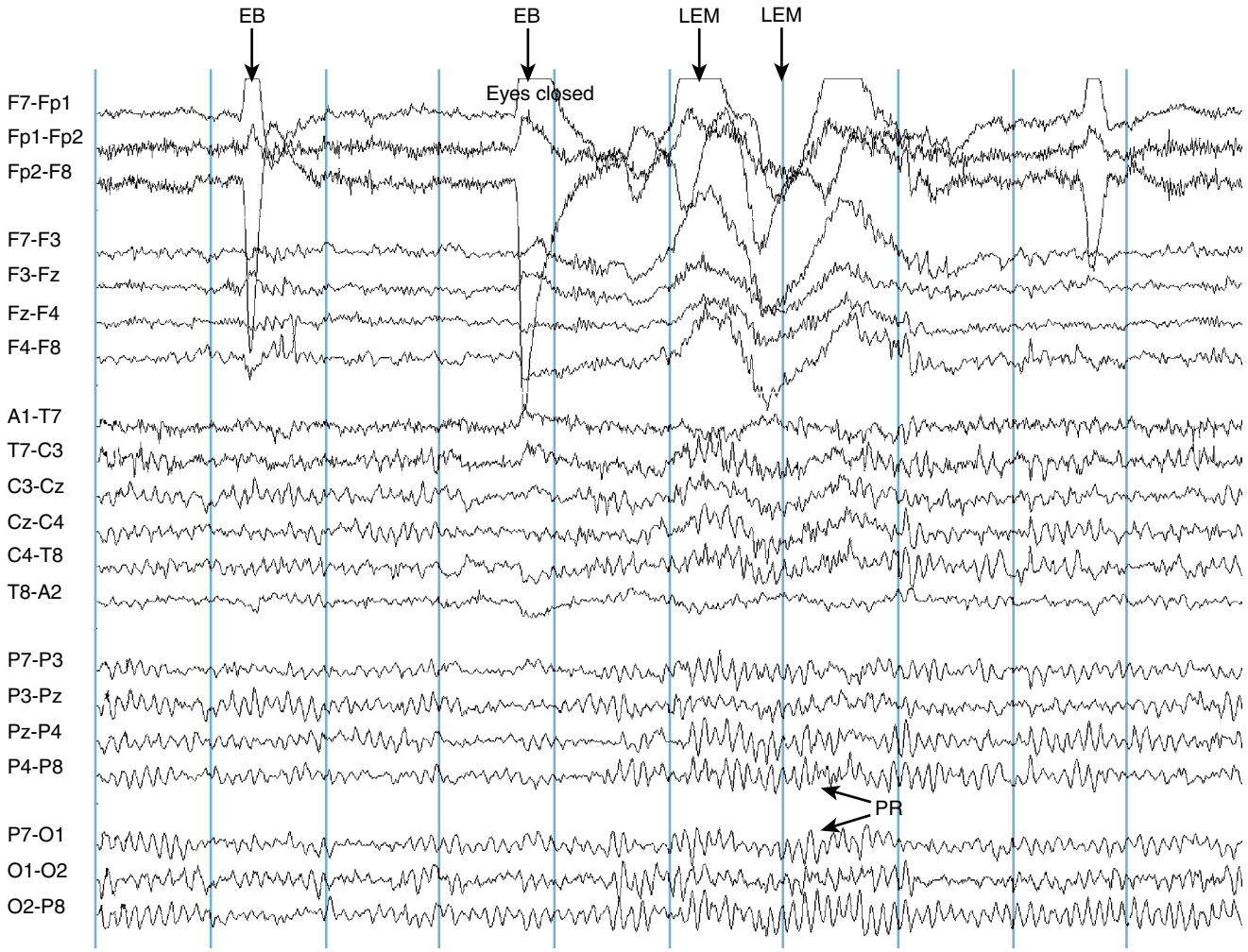


Figure 5-29 The same page of wakefulness as in Figure 5-28 is shown in the **transverse bipolar montage.** The eyeblink (EB) artifact is now essentially confined to the top three channels. Because the upward movement of the globes, which represents a net positive charge, is detected equally in Fp1 and Fp2, the amount of deflection in the Fp1-Fp2 channel is small. The Fp1 and Fp2 electrodes are, however, closer to the positivity of the eyeball as it bobs upward than the F7 and F8 electrodes. For this reason, the net positivity picked up by Fp1 being greater than at F7, the first channel, F7-Fp1, deflects upward. Likewise, because Fp2 detects more positivity than the F8 electrode, the third, Fp2-F8 channel, deflects downward. The posterior rhythm (PR) is well seen in the bottom three channels as expected, and also in the previous four channels, which link the left and right posterior temporal areas together, and even in the chain that links the left earlobe to the right earlobe (middle group of six channels). Only occasional fragments of the posterior rhythm are picked up in the chain that links the anterior temporal electrodes, F7 and F8 (the fourth through seventh channels nearer the top of the page). LEM = lateral eye movement.

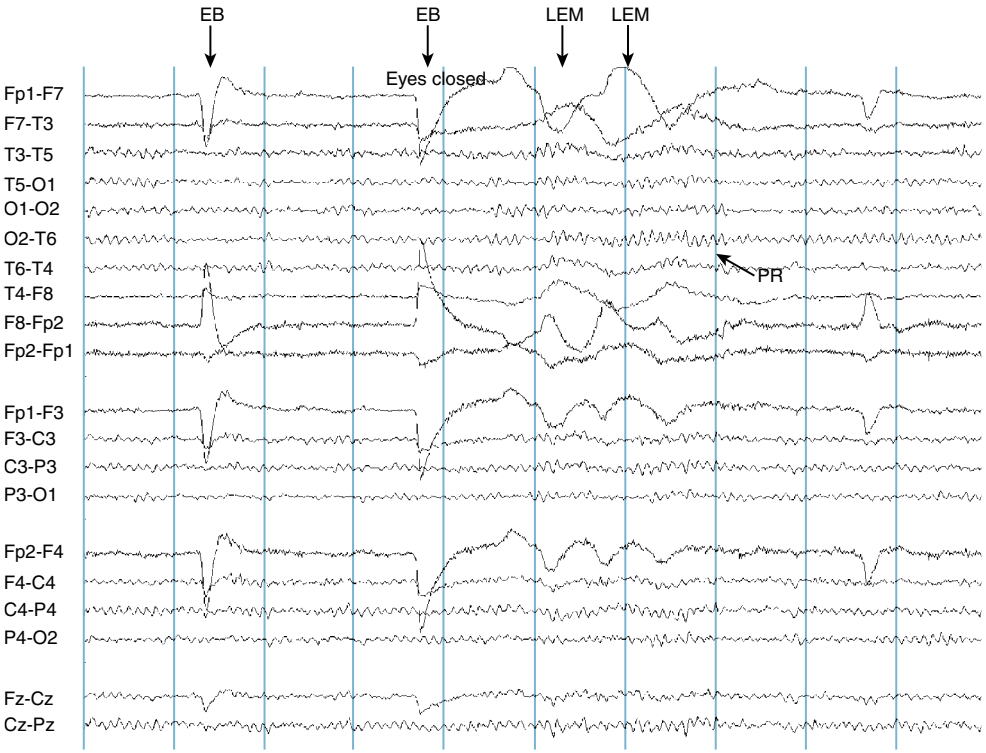


Figure 5-30 A page of wakefulness is shown in the **circumferential or "hatband" montage.** The top 10 channels represent the circumferential portion of the display. The posterior rhythm (PR) is best seen in the middle channels of the circumferential portion, because these pass through the occipital regions. This montage is most easily scanned for symmetry through the midline visual axis formed by the O1-O2 channel. Eyeblink (EB) artifact is seen in the expected positions at the ends of the circumferential chain, because these represent the frontal region. LEM = lateral eye movement.

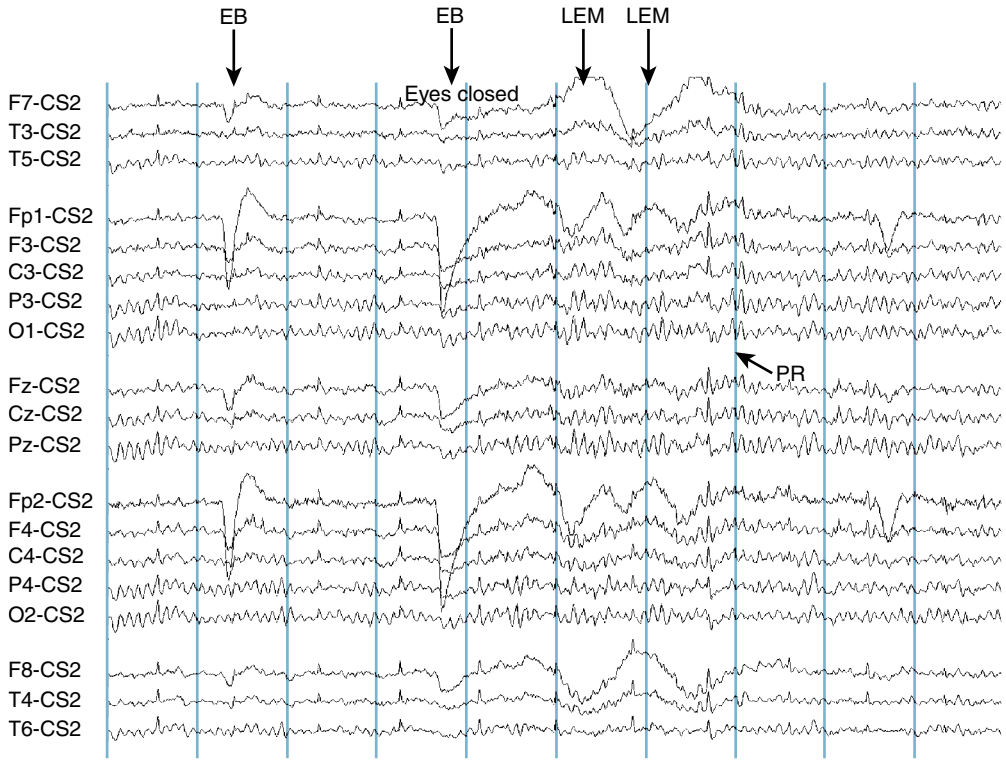


Figure 5-31 A page of wakefulness is shown in the **anteroposterior referential montage using a CS2 reference** (an electrode placed on the back of the neck). Recalling that in some patients electrocardiographic (EKG) artifact is present in the neck reference, rhythmic EKG artifact can be seen in all channels on this page. The fact that these "little spikes" appear with the same amplitude and morphology in every channel is a clue that they originate in the reference electrode rather than from the brain. In fact, on the basis of this artifact, the patient's EKG rhythm can be counted out at 66 beats per minute. The field of the posterior rhythm is well defined, appearing, for the most part, in the posterior channels. Careful observation, however, shows that a small amount of the posterior rhythm (PR) is visible even in the most anterior channels. The astute reader will conclude that this is not because the posterior rhythm is actually present in the frontal areas but rather that the CS2 electrode is actually picking up the posterior rhythm to a small extent. This is not completely unexpected because the posterior rhythm's field is maximum in the occipital regions, fairly close to the position of this neck electrode. The limitations of the use of the CS2 electrode evident in this page do not apply to all patients; in some patients, EKG artifact and posterior rhythm contamination are not seen in the CS2 electrode. EB = eyeblink; LEM = lateral eye movement.

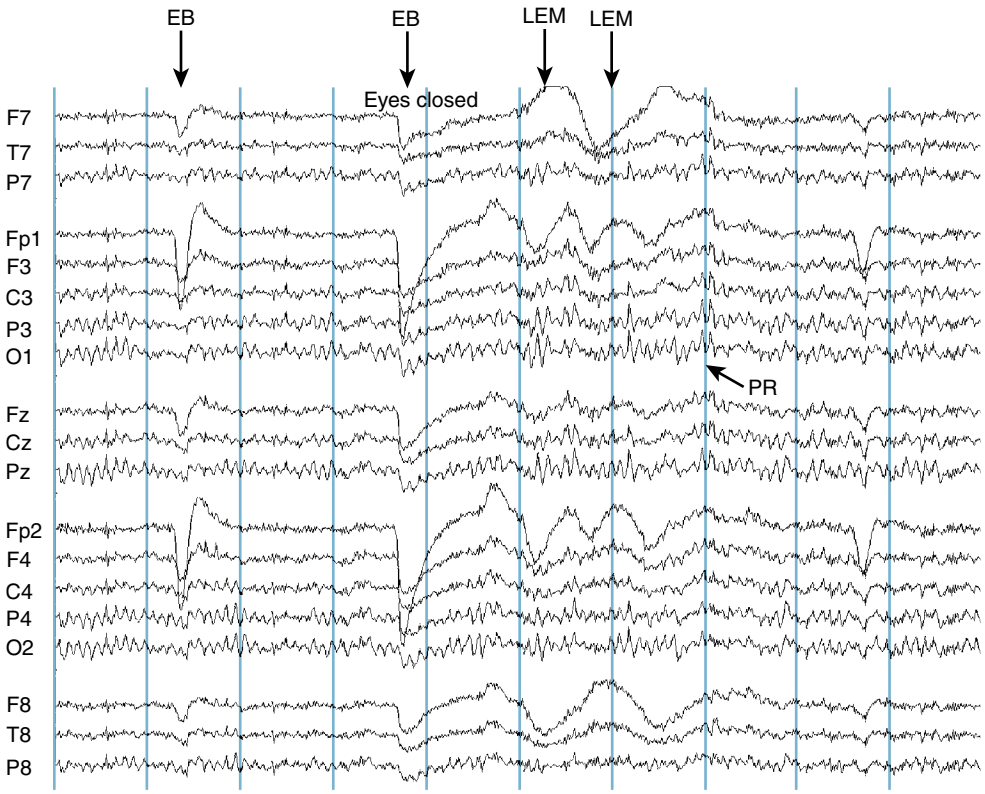


Figure 5-32 The same page of wakefulness is shown in the **AP referential montage using the chin as reference.** Because in this patient, the chin does not pick up electrocardiographic (EKG) artifact, we do not see the EKG spikes as in the previous figure. Instead, in this patient, there is a small amount of muscle artifact in the chin, which causes this artifact to appear in every channel of this tracing. Note also that the posterior rhythm (PR) is appropriately completely absent from the Fp1 and Fp2 electrodes channels in this page (compare with the contamination phenomenon seen in the previous figure). EB = eyeblink; LEM = lateral eye movement; PR = posterior rhythm.

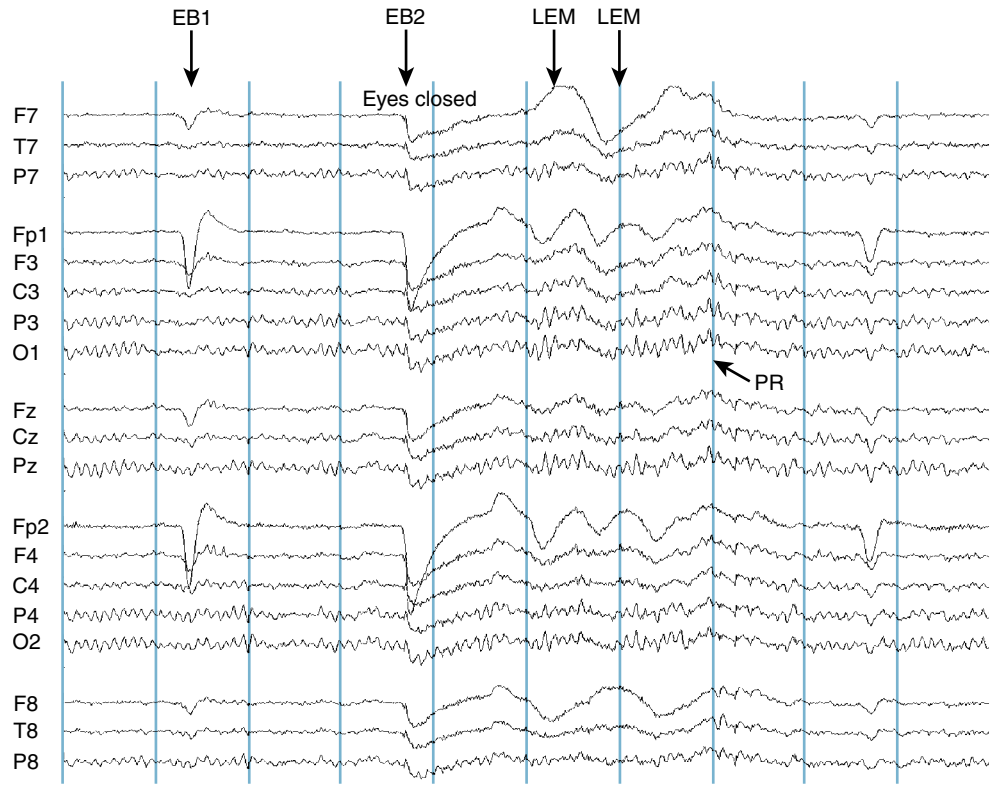


Figure 5-33 A page of wakefulness is shown in the **anteroposterior referential montage using the nose as the reference.** In this patient, the nose reference happens to be quiet, yielding an excellent quality tracing. Note the small spike-like artifact seen in the fourth second (under EB2 arrow). Because this small transient is similar in all channels, it can be identified as an artifact that originates in the nose reference. Again, eyeblink (EB) artifacts are seen confined to the anterior (Fp1, Fp2, F7, F3, F4, and F8) channels. The field of the posterior rhythm (PR) in this patient extends up to the C3 and C4 electrodes. Only occasionally are fragments of this rhythm seen as far forward as F3 and F4. As expected, the posterior rhythm cannot be identified in the frontopolar leads, Fp1 and Fp2. LEM = lateral eye movement.

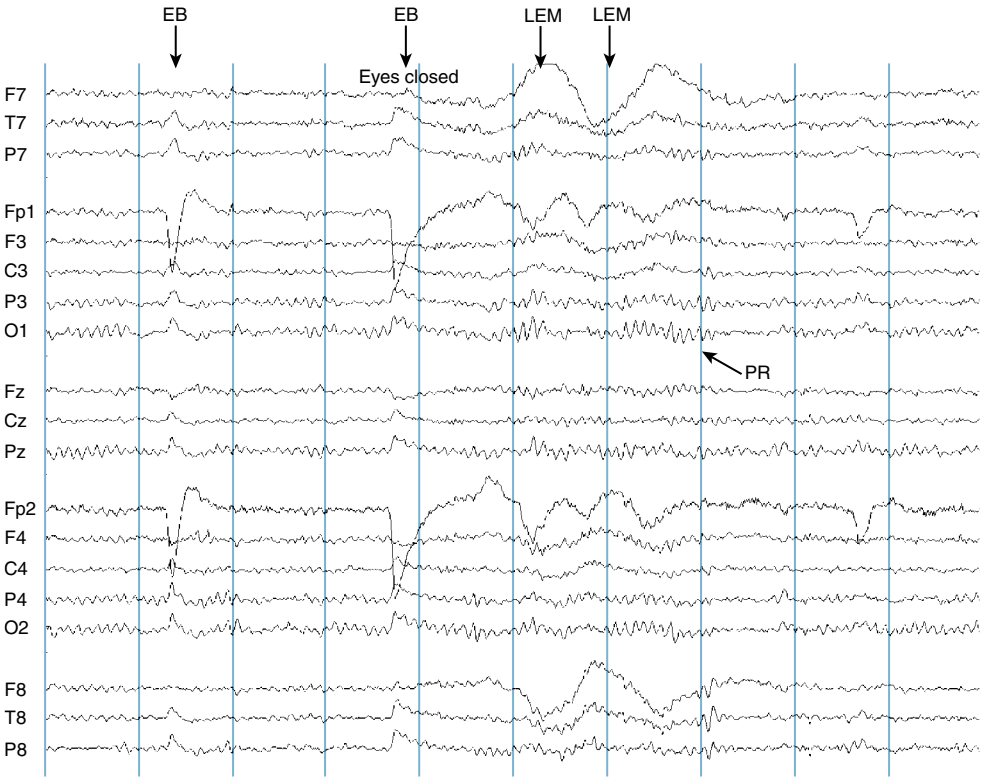


Figure 5-34 A page of wakefulness is shown in the **anteroposterior referential montage using the average reference.** Some of the advantages and disadvantages of the average reference are evident in this page. The average reference yields a clean display from the point of view of being free of muscle artifact. However, note that the eyeblink (EB) artifacts seen in the anterior channels are being "answered" with upgoing waves in the posterior channels. This is an artifact of contamination of the average reference by the high-voltage eyeblink deflections. Likewise, the posterior rhythm (PR) can be seen in all channels, again an artifact of the effect of contamination of the average reference by the posterior rhythm (see further description of these problems in the paragraphs on the average reference). LEM = lateral eye movement.

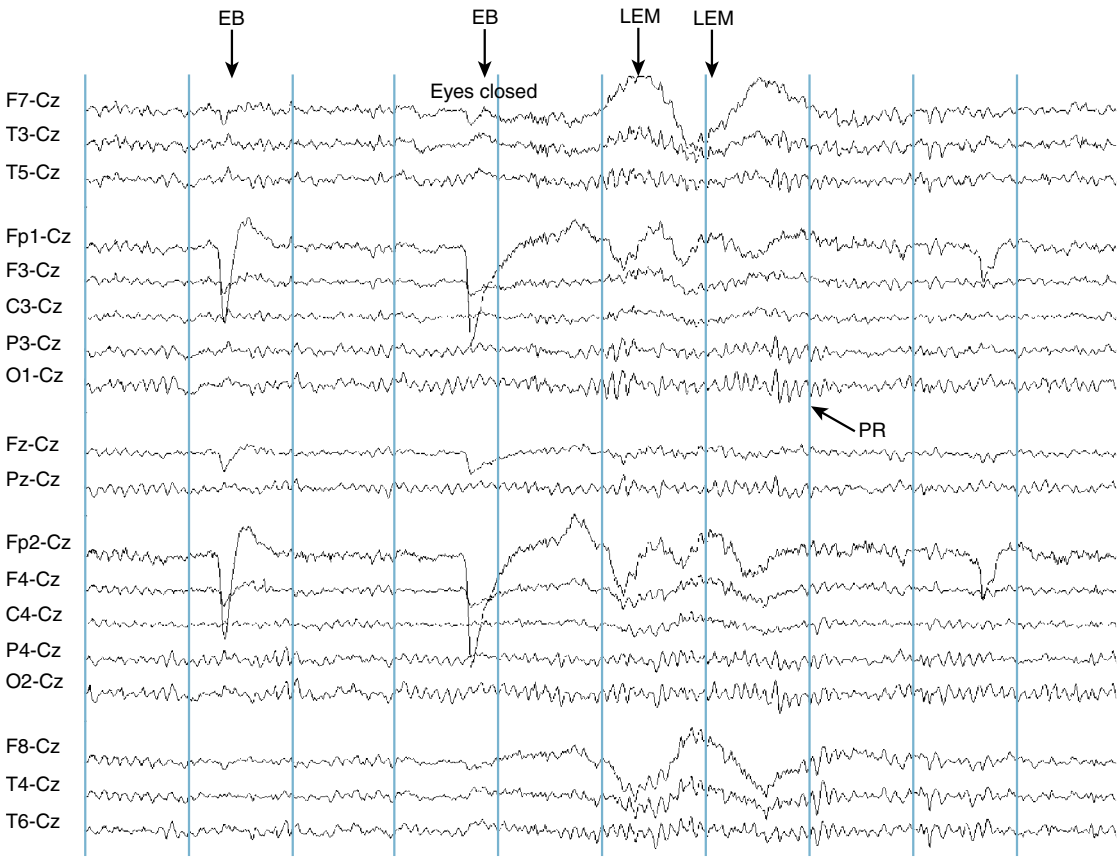


Figure 5-35 A page of wakefulness is shown in the **anteroposterior referential montage using Cz as the reference.** In this patient at this time in the tracing, the Cz electrode is relatively quiet. For this reason, this page appears to be of generally good quality, though note that the posterior rhythm can often be seen in all channels on the page, reflecting the fact that Cz is close enough to the back of the head that it intermittently picks up the posterior rhythm. This can be confirmed by looking at Figure 5-33 in which the posterior rhythm can often be discerned in the Cz-nose channel. Because all channels on this page are being compared to Cz, any activity that occurs in Cz has the potential to appear on every channel of the page. EB = eyeblink, LEM = lateral eye movement, PR = posterior rhythm.

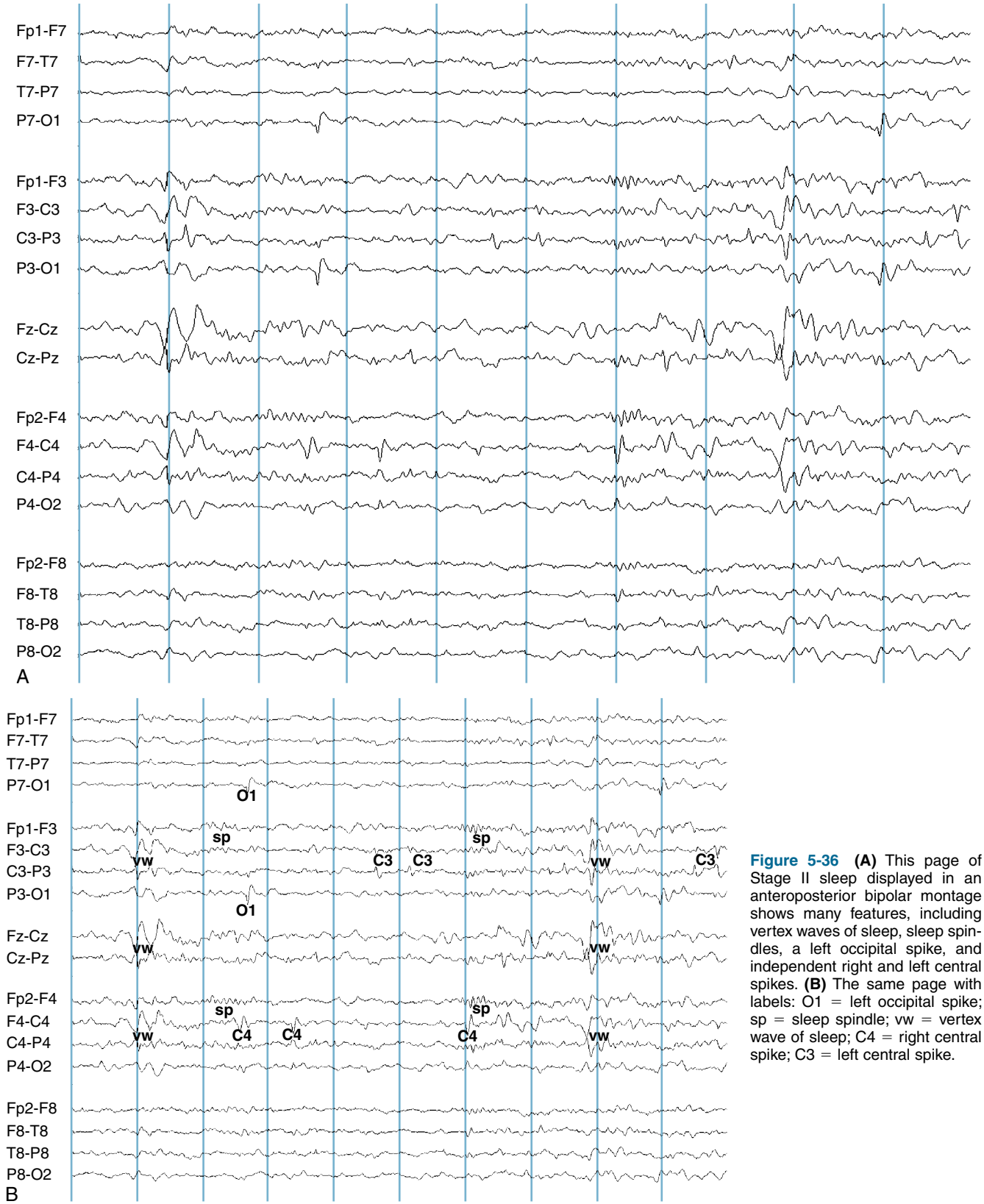


Figure 5-36 **(A)** This page of Stage II sleep displayed in an anteroposterior bipolar montage shows many features, including vertex waves of sleep, sleep spindles, a left occipital spike, and independent right and left central spikes. **(B)** The same page with labels: O1 = left occipital spike; sp = sleep spindle; vw = vertex wave of sleep; C4 = right central spike; C3 = left central spike.

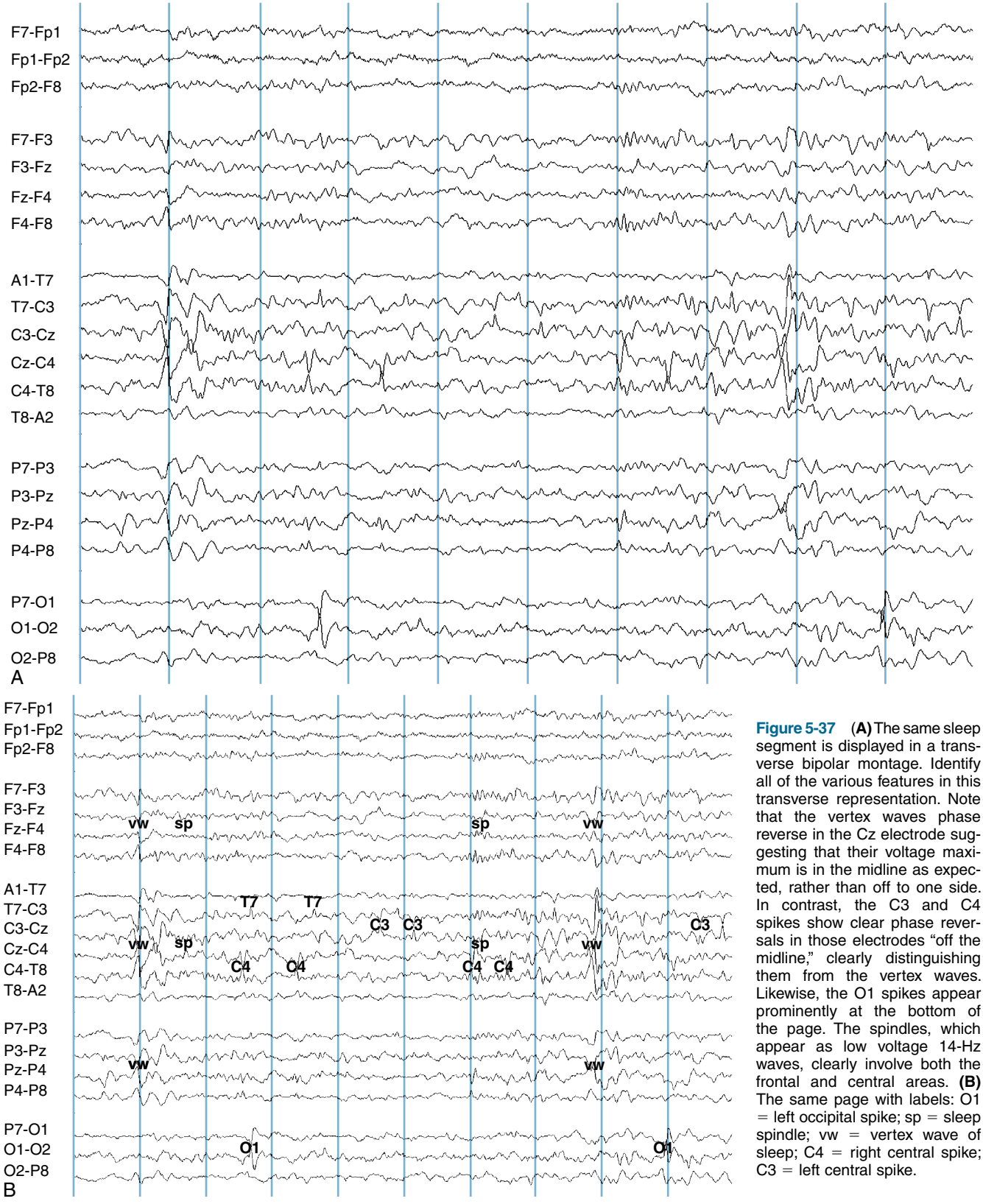


Figure 5-37 **(A)** The same sleep segment is displayed in a transverse bipolar montage. Identify all of the various features in this transverse representation. Note that the vertex waves phase reverse in the Cz electrode suggesting that their voltage maximum is in the midline as expected, rather than off to one side. In contrast, the C3 and C4 spikes show clear phase reversals in those electrodes "off the midline," clearly distinguishing them from the vertex waves. Likewise, the O1 spikes appear prominently at the bottom of the page. The spindles, which appear as low voltage 14-Hz waves, clearly involve both the frontal and central areas. **(B)** The same page with labels: O1 = left occipital spike; sp = sleep spindle; vw = vertex wave of sleep; C4 = right central spike; C3 = left central spike.

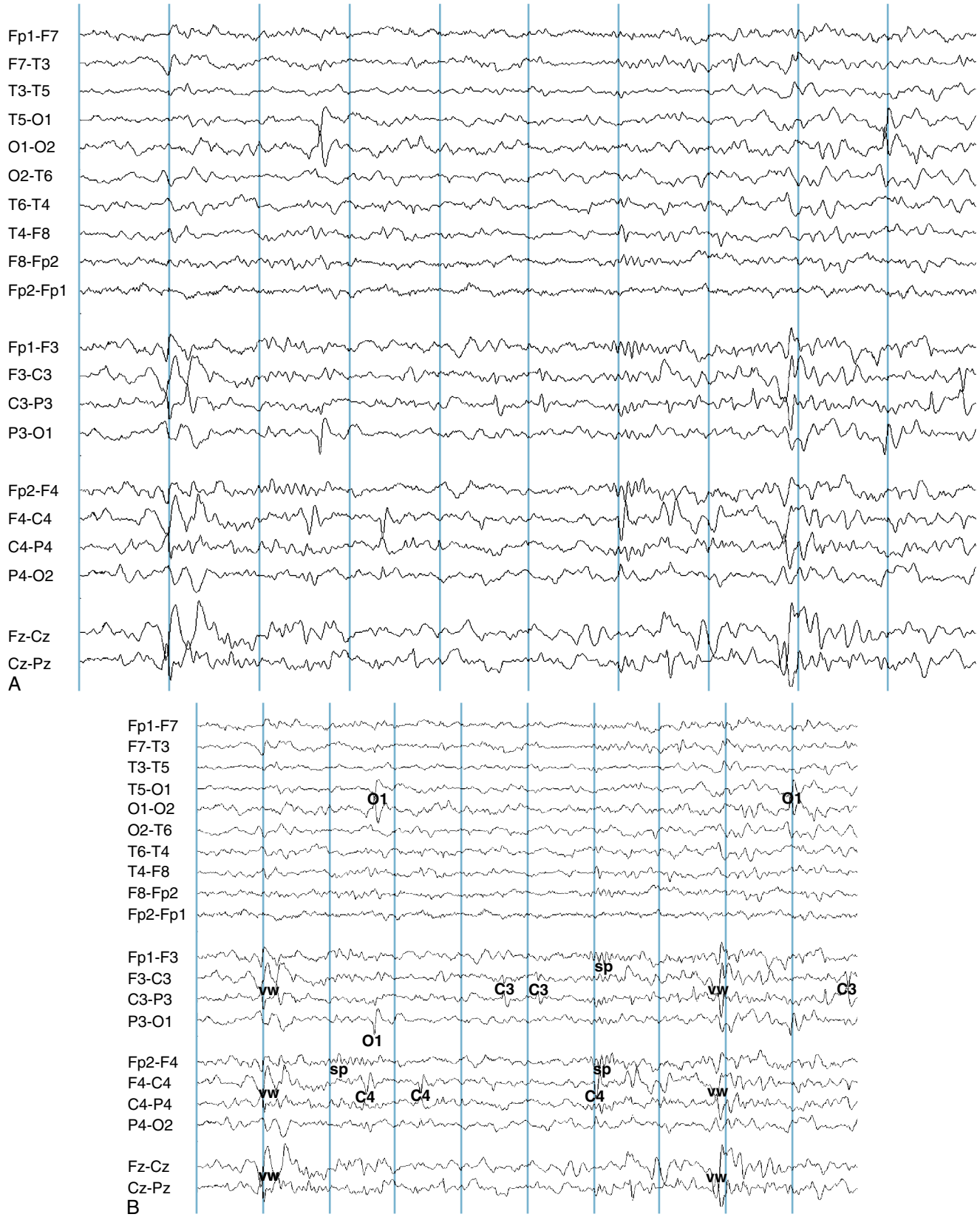


Figure 5-38 **(A)** The same page of sleep is displayed in a circumferential bipolar montage. The vertex waves and spindles are best seen on the bottom half of the page, which includes the parasagittal chains and the midline. The O1 spike appears prominently in the circumferential channels on the top half of the page. The C3 and C4 spikes are also evident. **(B)** The same page with labels: O1 = left occipital spike; sp = sleep spindle; vw = vertex wave of sleep; C4 = right central spike; C3 = left central spike.

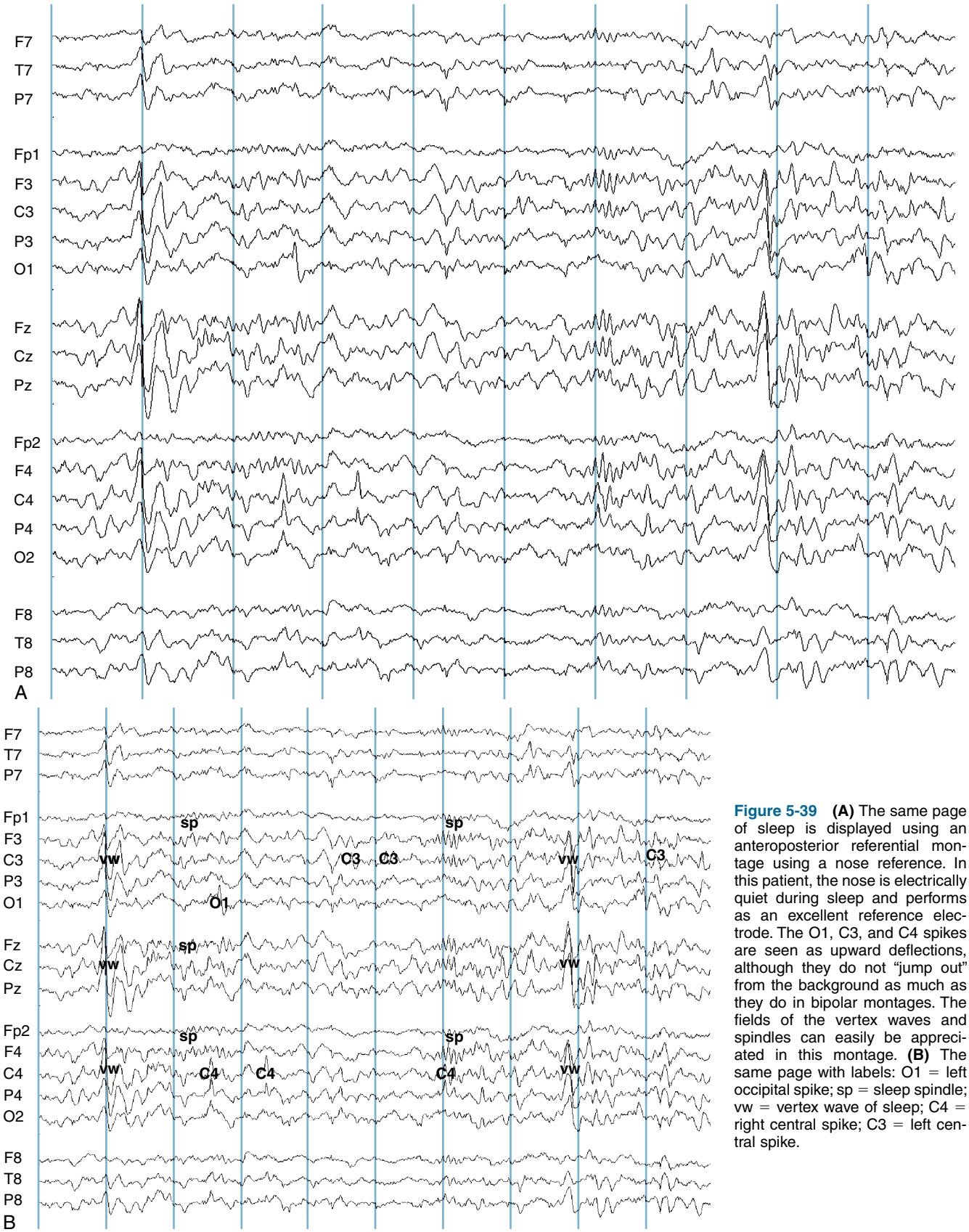


Figure 5-39 **(A)** The same page of sleep is displayed using an anteroposterior referential montage using a nose reference. In this patient, the nose is electrically quiet during sleep and performs as an excellent reference electrode. The O1, C3, and C4 spikes are seen as upward deflections, although they do not "jump out" from the background as much as they do in bipolar montages. The fields of the vertex waves and spindles can easily be appreciated in this montage. **(B)** The same page with labels: O1 = left occipital spike; sp = sleep spindle; vw = vertex wave of sleep; C4 = right central spike; C3 = left central spike.

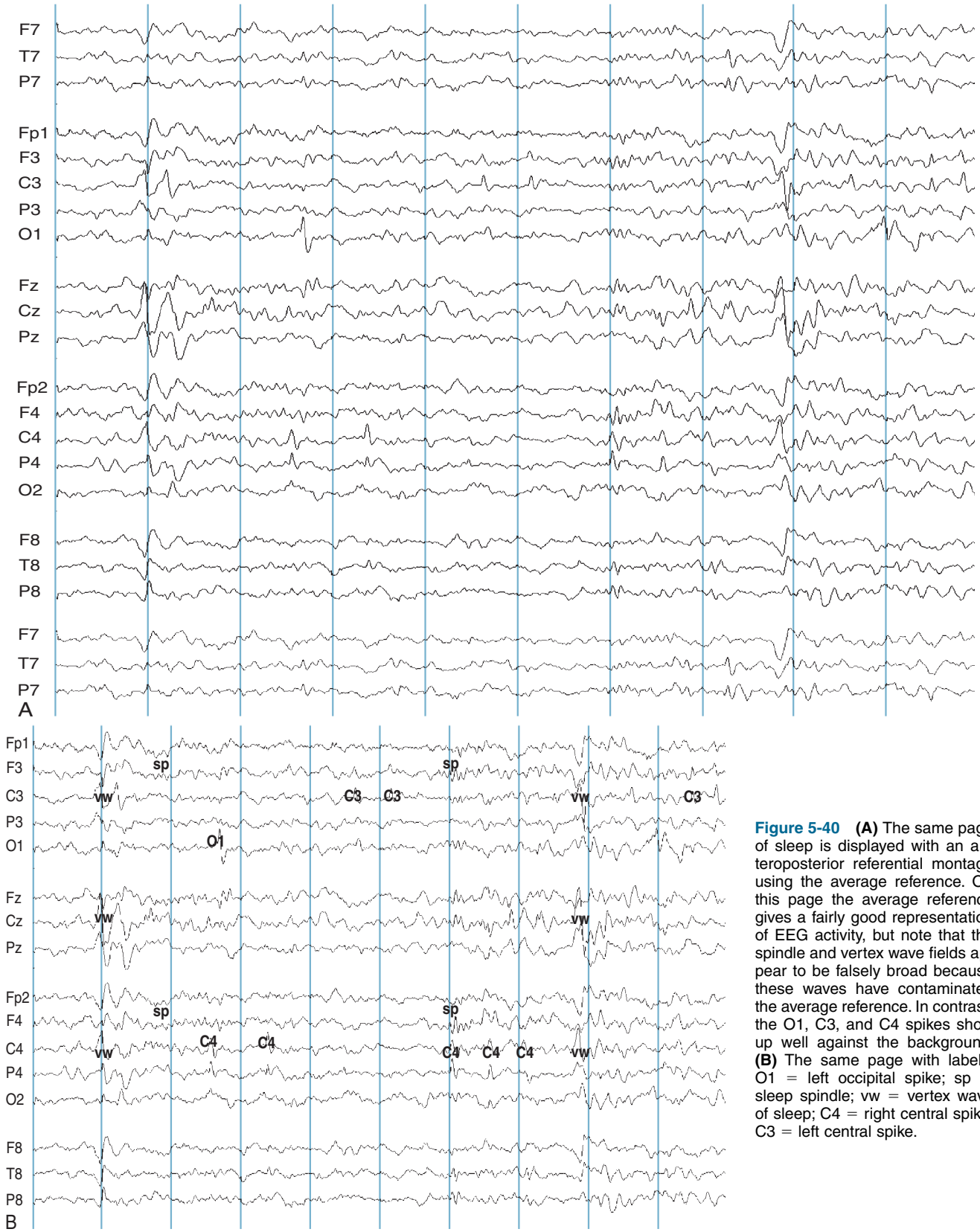


Figure 5-40 **(A)** The same page of sleep is displayed with an anteroposterior referential montage using the average reference. On this page the average reference gives a fairly good representation of EEG activity, but note that the spindle and vertex wave fields appear to be falsely broad because these waves have contaminated the average reference. In contrast, the O1, C3, and C4 spikes show up well against the background. **(B)** The same page with labels: O1 = left occipital spike; sp = sleep spindle; vw = vertex wave of sleep; C4 = right central spike; C3 = left central spike.

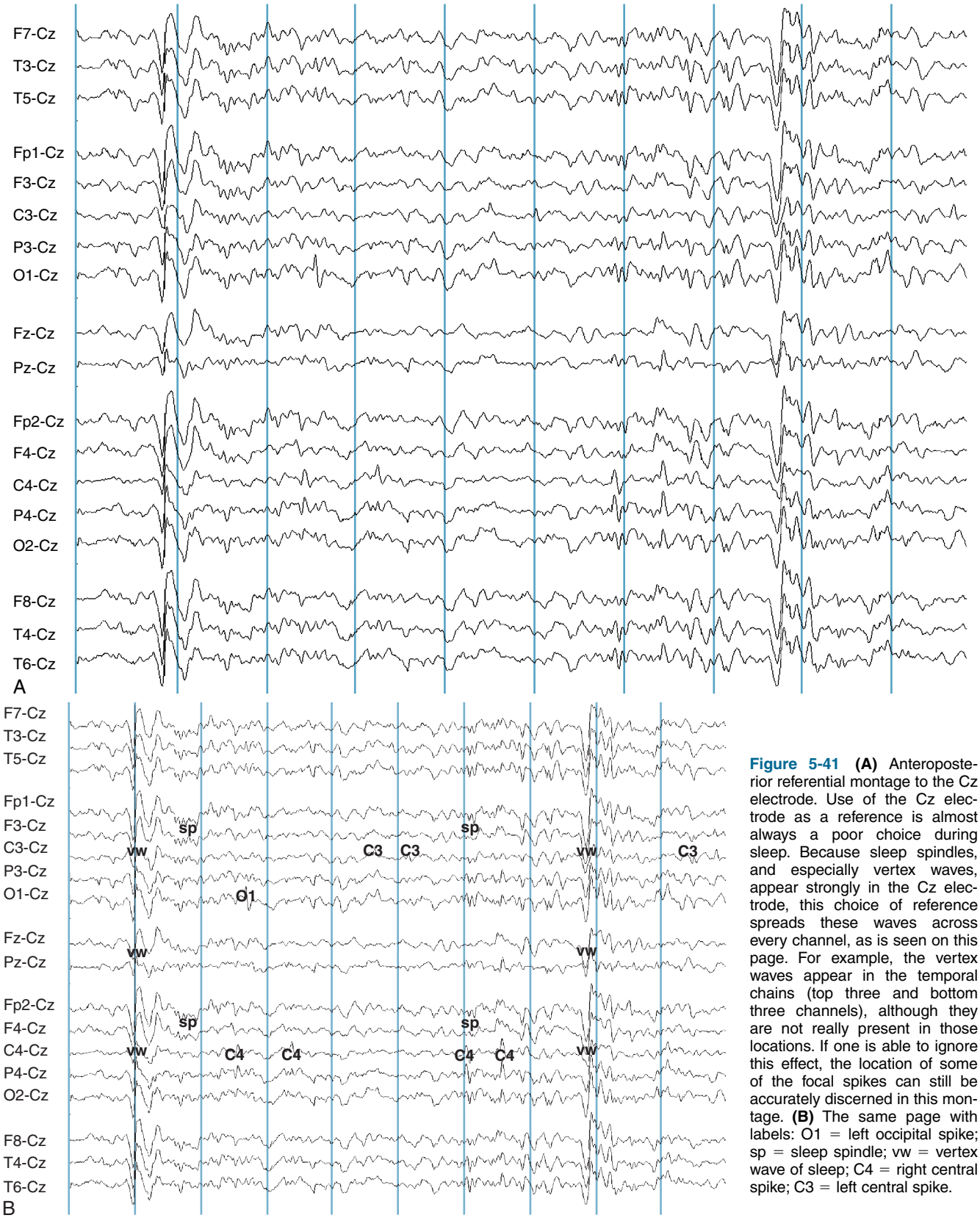


Figure 5-41 **(A)** Anteroposterior referential montage to the Cz electrode. Use of the Cz electrode as a reference is almost always a poor choice during sleep. Because sleep spindles, and especially vertex waves, appear strongly in the Cz electrode, this choice of reference spreads these waves across every channel, as is seen on this page. For example, the vertex waves appear in the temporal chains (top three and bottom three channels), although they are not really present in those locations. If one is able to ignore this effect, the location of some of the focal spikes can still be accurately discerned in this montage. **(B)** The same page with labels: O1 = left occipital spike; sp = sleep spindle; vw = vertex wave of sleep; C4 = right central spike; C3 = left central spike.

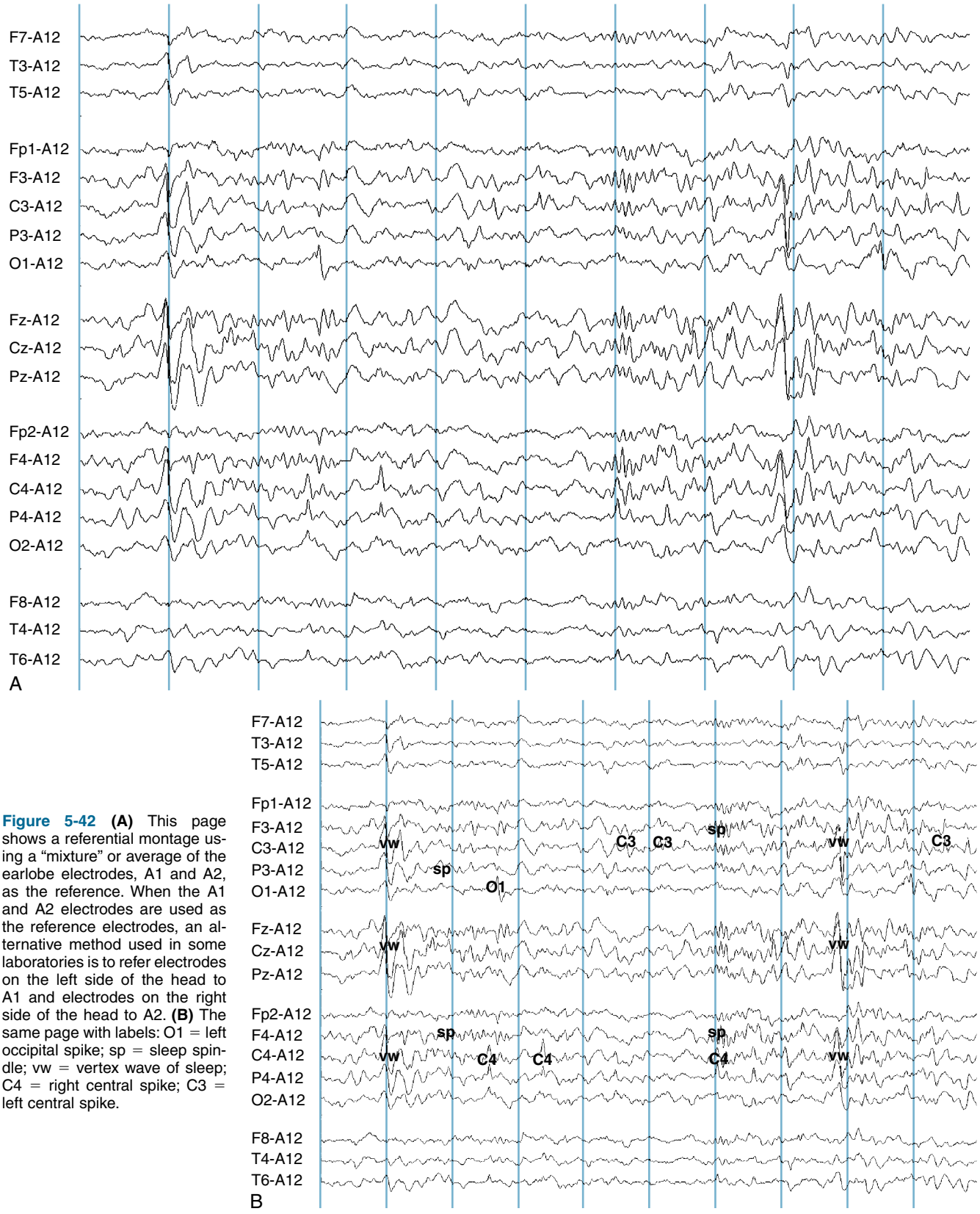


Figure 5-42 **(A)** This page shows a referential montage using a "mixture" or average of the earlobe electrodes, A1 and A2, as the reference. When the A1 and A2 electrodes are used as the reference electrodes, an alternative method used in some laboratories is to refer electrodes on the left side of the head to A1 and electrodes on the right side of the head to A2. **(B)** The same page with labels: O1 = left occipital spike; sp = sleep spindle; vw = vertex wave of sleep; C4 = right central spike; C3 = left central spike.

REFERENCE

Guideline 6: A proposal for standard montages to be used in clinical EEG. ACNS Guidelines. *J Clin Neurophysiol* 23:111–117, 2006.

Chapter

6 Electroencephalographic Artifacts

An artifact is a waveform in the EEG that is not of cerebral origin. Those new to the world of EEG interpretation are often surprised to learn that at least half the challenge of EEG reading consists of identifying artifacts correctly so as not to mistake them for true EEG (cerebral) activity. This is quite different from other types of interpretation in clinical electrophysiology such as electrocardiogram (EKG) reading in which the problem of distinguishing electrical artifact from actual EKG waves is only occasionally a problem.

The mistake of interpreting an electrical artifact as a true EEG wave can have important negative consequences. Reporting an innocent "electrode pop" artifact as an epileptiform spike is an error that can have serious clinical repercussions. Unfortunately, mistaking an artifact for an EEG wave is not an uncommon type of error among inexperienced readers—even experienced readers can find the distinction between electrocerebral activity and artifact difficult at times. For these reasons, new readers should devote considerable energy to mastering the thought process used to distinguish artifacts from true EEG activity.

Some of the most common EEG artifacts are so distinctive in appearance that the experienced reader rapidly screens them out without the need for close analysis. A good example of this is muscle artifact. Muscle artifact from the temporal, frontal, and occipital areas is so frequently encountered that, for some portions of the tracing, the challenge of interpretation is to "read through" these artifacts to see the true EEG activity.

Artifacts are not always a hindrance to EEG interpretation. Sometimes they can yield valuable information. The presence of the same muscle artifact that obliterates the EEG from the temporal areas may also serve as a useful indicator that the patient is moving and awake rather than asleep. Likewise, the presence of eyeblink artifact would not be expected in the sleeping patient and therefore helps to exclude the possibility that the patient is asleep. Other types of eye movement artifact can help identify sleep stages. The presence of slow roving lateral eye movement artifact is often a useful sign of drowsiness. The presence of REM (rapid eye movement) sleep is confirmed, as its name suggests, by the presence of REM artifact. Other types of artifact, however, can be a real hindrance to EEG interpretation. A patient who is constantly moving may generate a record so dominated by motion artifact as to render it uninterpretable. In some patients, the motor activity associated with a seizure can generate so much muscle and motion artifact that the underlying EEG seizure discharge cannot be identified.

STRATEGIES OF ARTIFACT RECOGNITION

There are two main principles that are critical to the process of distinguishing what is "real" brain wave activity from artifact in the EEG record. The first is wave morphology, or using the shape of a wave to determine its nature; certain wave shapes and patterns are highly characteristic of certain types of artifacts. The second is the principle of "biologic plausibility"; electrical events of cerebral origin should show a distribution of intensity on the scalp surface that is plausibly consistent with brain wave activity. Artifacts may show a topography of voltages that is bizarre and unpredictable indicating a biologically implausible event. When an event has a biologically implausible topography, the EEG reader is more likely to interpret it as electrical or motion artifact. We expand on these concepts in the examples that follow.

Wave Morphology

Certain artifacts in the EEG have characteristic shapes, such as eyeblink artifact, muscle artifact, and some electrode pops. Some of these artifacts have such a specific appearance that their identification essentially consists of a "sight diagnosis" or pattern recognition. The conclusion that some waves represent artifact may require a combination of both techniques, wave pattern recognition and analysis of wave topography. Examples of various specific types of artifact are shown later in this chapter.

"Biological Plausibility" Versus "Biological Implausibility"

Although some artifacts can be recognized on the basis of the shapes of their waves, the most powerful tool in distinguishing a true brain wave from an artifact is to establish its specific topography. Here, the major operating principle is that biological events recorded on the scalp tend to have a point of maximum voltage with the surrounding measured voltages diminishing with varying steepness from the point of maximum (see Figure 6-1).

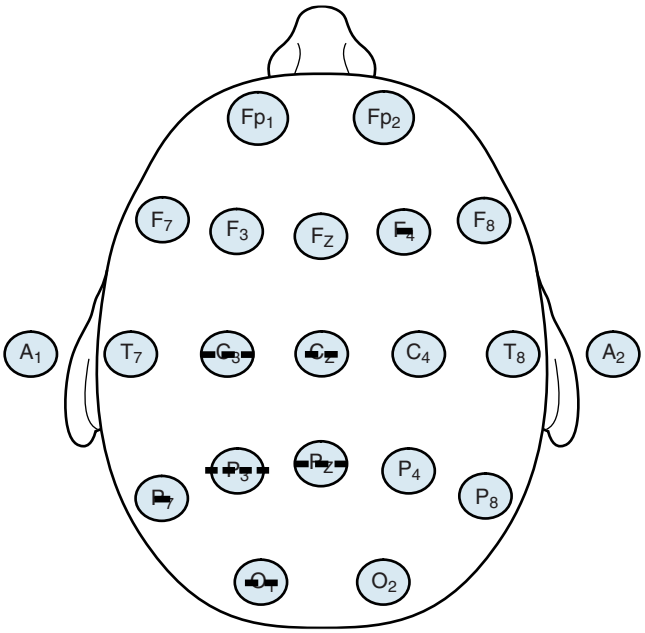


Figure 6-1 A schematic depiction of a pure negativity occurring on the scalp, maximum in the left parietal electrode (P3). Note that the intensity of the negative fields, denoted by the number of minus signs, dissipates gradually away from the maximum, but with varying "steepness" in different directions.

The voltage gradients seen with electrocerebral events are expected to have some degree of "smoothness." In contrast, the electric fields of artifacts may have a patchy and unpredictable contour (compare to Figure 6-2).

A second principle is that the polarity of most discharges tends to be consistent. Most discharges, if negative at their maximum point, remain negative wherever they are detected on the scalp as shown in Figure 6-1. In general, negative events tend to be negative everywhere, and positive events tend to be positive everywhere. Exceptions to this rule exist, including the occasional examples of discharges that manifest a so-called horizontal dipole (the example of the "horizontal" or tangential dipole is discussed in detail in Chapter 10, "The EEG in Epilepsy"). Even in the small minority of epileptiform events that manifest a simultaneous combined positivity and negativity, those areas of opposite charge are usually segregated in a simple and orderly fashion rather than showing a pattern of several separated regions of positivity and negativity (see Figure 6-3).

Patterns with significant charge inconsistencies are also not expected from biological systems. The example of an apparent discharge with strong negativities in the left posterior quadrant and the right anterior quadrant but no measured voltage change in the intervening areas as depicted in Figure 6-4 is not likely of cerebral origin. Because this is not a "biological" pattern, there would be a strong suspicion of motion or some other type of artifact having affected two distant electrodes. This pattern and the pattern that was seen in Figure 6-2 may be produced by the haphazard jostling of electrodes during patient movement.

Whenever analyzing any wave on the EEG page, the reader should visualize the voltage topography of the discharge to confirm a distribution of charge that is potentially consistent with cerebral activity. Usually with

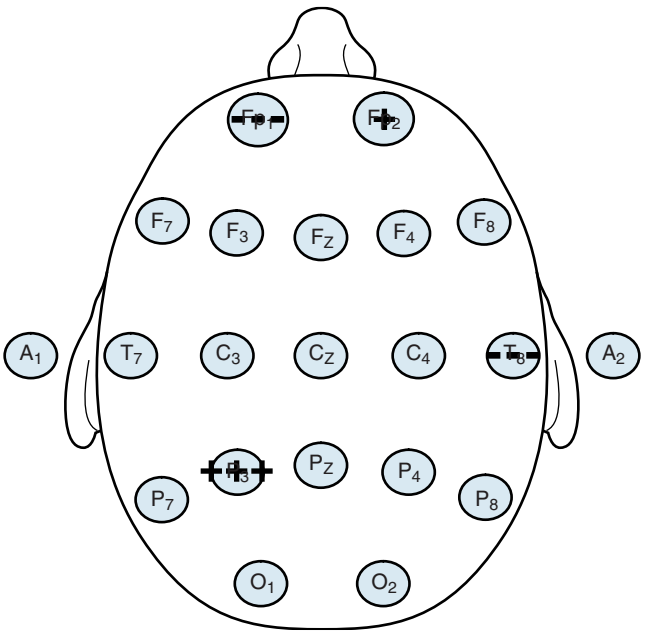


Figure 6-2 In contrast to the previous figure, the field of this discharge is scattered and patchy, showing different polarities in different locations. The pattern of this scalp electrical event does not seem "plausible" for electrocerebral activity. Patterns such as these are most likely caused by electrical or motion artifact.

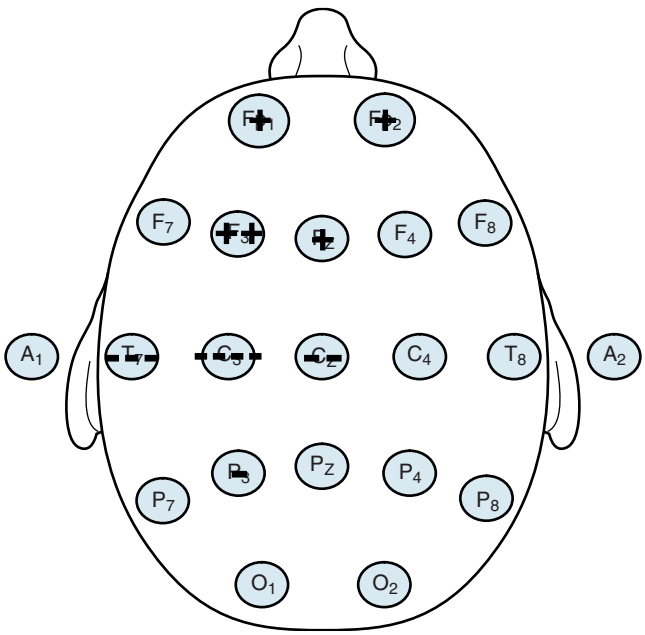


Figure 6-3 A schematic of a "horizontal dipole" is shown, a relatively uncommon finding outside of benign rolandic epilepsy and related syndromes. In contrast to most discharges that show a single polarity on the scalp at any one time (usually negative), in this example, a negative charge and a positive charge are recorded on the scalp simultaneously. Even though both polarities are present, the transition between the two is smooth. The configuration shown here of a negativity in the centrotemporal area and a positivity in the anterior head regions is characteristic of the so-called rolandic spike, discussed in more detail in Chapter 10, "The EEG in Epilepsy."

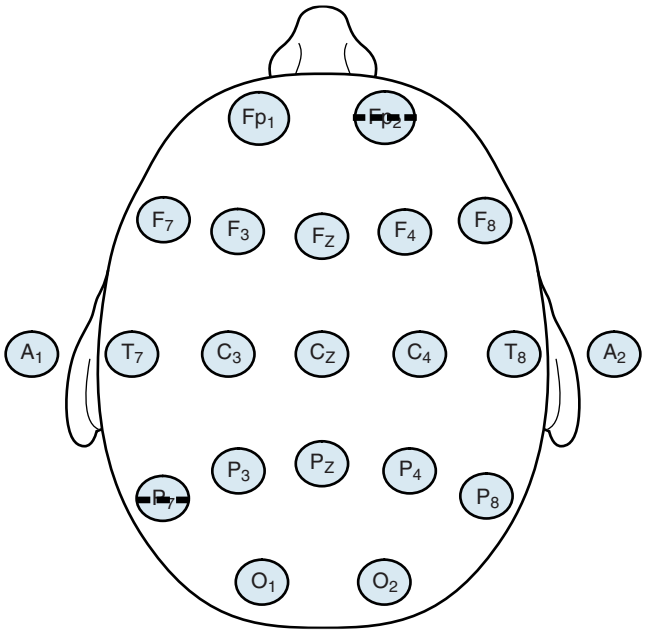


Figure 6-4 A localized negativity is seen in the left posterior temporal area simultaneously with a strong negativity in the right frontopolar area. The intervening areas are electrically neutral. This field does not seem consistent with the biology of the brain. Rather, this pattern, which lacks a gradient and only involves two individual but distant electrodes, is much more suggestive of electrode or motion artifact than a true electrocerebral discharge.

a bit of practice this can be done quickly. Discharges with bizarre or unpredictable electrical fields likely represent artifact. The importance of localization skills cannot be overemphasized and it is for this reason that the techniques of localization discussed in Chapter 4, "Electroencephalographic Localization," should be mastered before moving on.

How do such "biologically implausible" artifacts arise in the first place? Most such artifacts are related to head motion. When the patient's head moves, some combination of electrode wires is tugged on. The pattern of wires that is pulled with a head movement is, for the most part, unpredictable and may not follow any particular spatial pattern on the scalp. Disparate and distant deflections on the scalp, especially with varying polarities, are much more suggestive of motion artifact than of electrocerebral activity.

A type of motion artifact that might reliably follow a spatial pattern is head rolling or rocking. In such instances, the electrodes lying against the bed are most susceptible to motion artifact. For example, if the occipital portion of the patient's head is in contact with the bed, head motion artifact will tend to be most dramatic in the occipital electrodes. Likewise, if the patient is lying on the left side of the head, the left temporal electrodes will be most prone to motion artifact. Artifact may appear in these or other electrodes with voluntary head movements, or even with the low-amplitude head movements associated with the patient's respirations.

Some significant EEG diagnostic challenges occur when patients are referred for the question of whether sudden movements are epileptic. The diagnostic problem is made more difficult in that each movement may generate a motion artifact causing the appearance that each is correlated with an EEG burst. There would be no difficulty if the movement in question were, for instance, a nonepileptic twitching of the hand because a hand movement would not cause motion artifact in distant scalp electrodes. The hand movement would occur during the study, and there would be no EEG change, strongly suggesting that the hand movements were nonepileptic. If the movement involves the head, however, there may be a simultaneous burst in the EEG, creating some confusion as to whether the movement caused the wave (and is an artifact) or the wave caused the movement (and is an epileptiform discharge). The approach to this problem takes into account that when the head moves, is turned on the pillow, or is subject to some other type of external movement, the pattern of electrodes that are disturbed tends to be more random. Artifacts caused by electrode motion or head motion tend to show a nonregular distribution of inconsistent voltages like those shown in Figures 6-2 and 6-4. In comparison, the patterns associated with electrocerebral activity are expected to show more consistent polarities and a voltage gradient that tapers with distance from the point of maximum.

A second approach to this type of problem is to analyze the way in which the EEG discharge is time-locked to the movement. When a movement is caused by an EEG burst (e.g., epileptic myoclonus), there is always a slight time lag between the EEG burst and the movement because it takes some amount of time for the cortical signal to propagate through the nervous system to the muscles. This latency between the burst and the movement may be confirmed either by a synchronized video recording of the patient or an electrode placed on an involved limb. With use of the latter, the EEG burst appears first, followed by the muscle burst in the limb electrode. If an apparent EEG burst is really an example of motion artifact, the two are generally seen to occur on the video simultaneously, or the body movement may even start before the EEG deflection.

SPECIFIC TYPES OF EEG ARTIFACT

Artifacts Associated With Eye Movements

Eyeblink Artifact

Electroencephalographers know that when individuals close their eyes, the globes of the eyes deviate upward. This comes as a surprise to some because when we watch a person casually blink or close the eyes, this upward movement of the globes is hidden from view by the closed eyelids. It is only in special situations that this reflex upward eye deviation with eye closure, termed Bell's phenomenon, is readily observable. Bell's phenomenon becomes strikingly evident in the case of individuals who suffer from Bell's palsy. In Bell's palsy, there is a paralysis of the facial nerve (cranial nerve

VII), one of whose functions is to close the eyes. The nerves that move the globe of the eye (cranial nerves III, IV, and VI) are unaffected in patients with Bell's palsy. Therefore, Bell's palsy patients have normal eye movement but cannot close the affected eyelid. Thus, the facial nerve paralysis caused by Bell's palsy literally uncovers Bell's phenomenon—we can see what the globe of the eye is doing during intended eyelid closure. Bell's palsy patients have normal eye closure on the unaffected side, but when blinking or closing the eye on the paralyzed side, the globe of the eye can be seen to deviate upward, unhidden by the eyelid. Upward deviation of the globes may also be seen when a person's eyelids are forcibly pried open during attempted voluntary eye closure. Finally, in some individuals who are in the process of falling asleep, especially babies, the eyelids may lower to a "half-mast" position but do not completely close. Because of a partial Bell's phenomenon associated with the partial eye closure, only the whites of the eyes can be seen in the half-open palpebral fissures.

The type of artifact that an upward movement of the globes might cause during eye blinking or eye closure is not intuitively obvious without knowing that the globe of the eye has a particular distribution of charge. As it happens, the cornea (the front of the globe of the eye) carries a net positive charge and the posterior pole of the globe carries a net negative charge, forming a dipole. Because of the presence of this positive charge on the cornea, the bobbing upward of the eye with eye closure is easily detected by EEG electrodes. Figure 6-5 illustrates how the EEG electrodes closest to the eye "perceive" an eyeblink. With upward eye deviation, the electrodes closest to the globes, Fp1 and Fp2, perceive the strong positivity of the corneal surfaces. The F3 and F4 electrodes, which are immediately posterior to the Fp1 and Fp2 electrodes, also pick up some of this positivity, but to a much lesser degree than the Fp1 and Fp2 electrodes. With Fp1 being more positive than F3 and Fp2 being more positive than F4, the Fp1-F3 and Fp2-F4 channels show the characteristic sharp, downward-sweeping waveforms of eyeblink artifact (see Figure 6-6). A similar type of artifact is seen with eye fluttering (see Figure 6-7).

A useful general rule regarding eyeblink artifact is that it should not be detectable in the electrodes of the posterior half of the head; movements of the globes of the eye should not be perceived by the parietal or occipital electrodes because they are too distant. Eyeblink artifact is only occasionally picked up by the central electrodes, C3 and C4, and when it is it should be of low voltage (see Figure 6-8).

Lateral Eye Movement Artifact

An extension of this same phenomenon involving the motion of the positive charge on the anterior aspect of the globe is used to understand the appearance of lateral eye movement artifact. In the case of lateral eye movements, the anterior temporal electrodes, F7 and F8, are the electrode contacts best placed to perceive the positivity of the cornea as the eyes move from side to side in the horizontal plane. For example, when a patient gazes to the left, the F7 electrode picks up a net positivity. Simultaneously, as the eyes gaze to the left, the F8 electrode picks up a net negativity from the

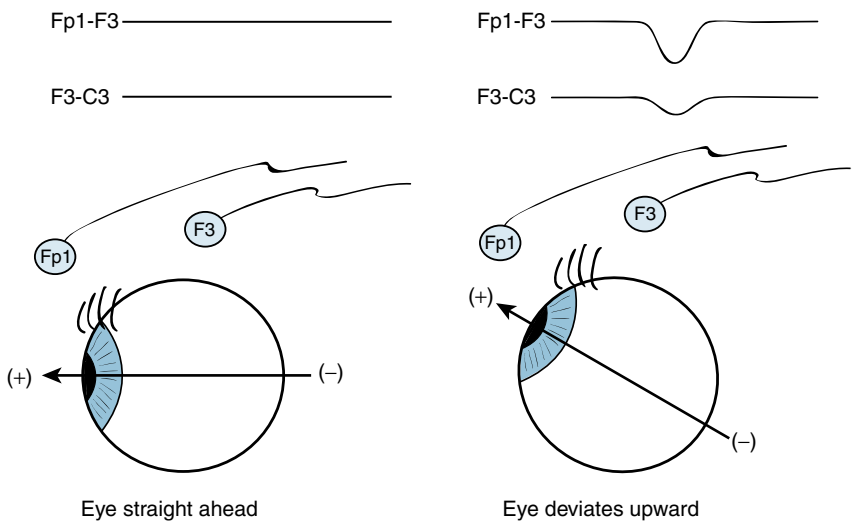


Figure 6-5 This figure shows how the dipole across the globe of the eye leads to the appearance of eyeblink artifact on the EEG. The cornea of the eye carries a net positive charge relative to the posterior pole of the eye, which carries a net negative charge. When the eyes are blinked or closed, the globes of the eye deviate upward under the closed eyelids (Bell's phenomenon). This results in the exposure of the Fp1 electrode in the diagram to the net positivity of the cornea. The F3 electrode, which is more distant from the cornea, also picks up some of the positivity, but to a lesser extent. Thus, when the eyes bob upward as shown in the right panel, the Fp1-F3 channel deviates downward (Fp1 is more positive than F3). When the F3 electrode is compared with the C3 electrode (not shown), the smaller amount of positivity picked up by the F3 electrode is compared to the neutral C3 electrode (C3 is too distant from the cornea to detect this event), and the F3-C3 channel shows another, smaller downgoing wave. More posterior channels in AP bipolar chains would not be expected to show deflections from eyeblink artifact.

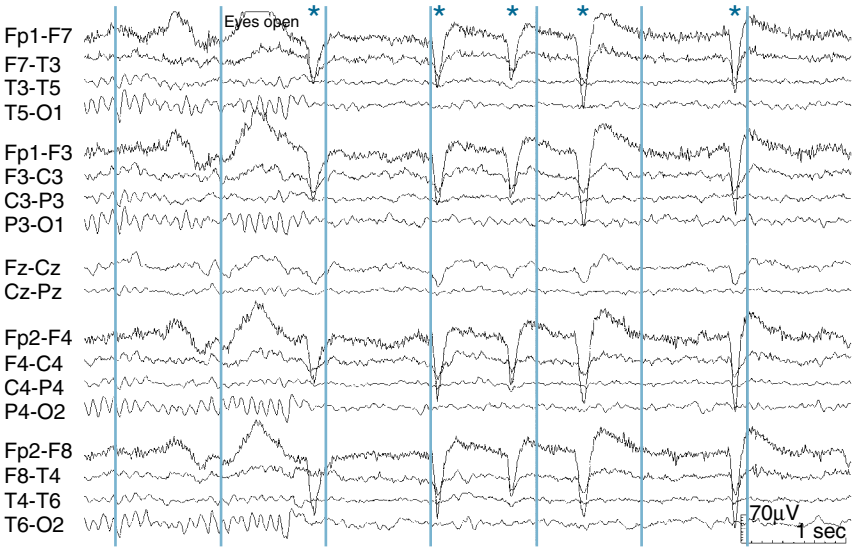


Figure 6-6 The typical appearance of eyeblink artifact showing high voltage downward deflections in the top two channels of each four-channel chain of the standard anteroposterior bipolar montage. Asterisks at the top of the page denote the timing of each eyeblink artifact. Note that, in each set of four channels, the deepest deflection is seen in the top channel. A less intense deflection is seen in the channel just below the top channel in each channel. Eyeblink deflections are not seen in the more posterior channels. The presence of repetitive eyeblink artifact usually indicates that the eyes are open. This is consistent with the observation in this figure that the posterior rhythm disappears at the time of the eyeblink artifact. The technologist has made a notation confirming this.

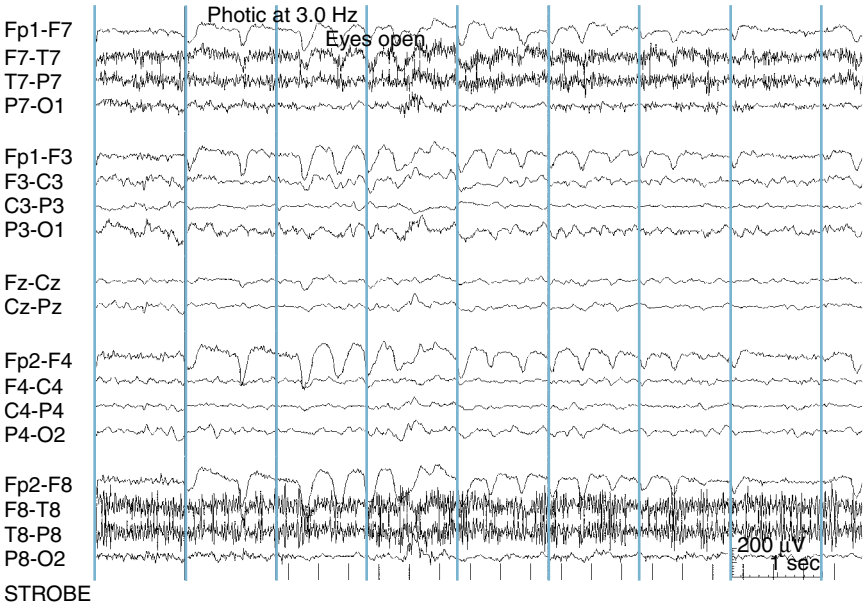


Figure 6-7 An artifact similar to eyeblink artifact is seen during eye fluttering. In this example, the eye fluttering is caused by the repetitive flashes of the strobe light during photic stimulation (note strobe flashes in the bottom channel). Occasionally, eye fluttering or eye bobbing artifact can be difficult to distinguish from frontal slow-wave activity. In such cases, special electrodes placed below the eyes can help distinguish between the two possibilities. Muscle artifact is seen in each temporal area.

posterior pole of the globe (see Figure 6-9). This results in a characteristic pattern seen with a simultaneous "positive phase reversal" in the anterior temporal electrode on the side to which the eyes have turned and a "negative phase reversal" in the anterior temporal electrode on the side away from which the eyes have turned. In the case of more rapid voluntary saccades or REM sleep, these lateral eye movement artifacts can have a squared-off appearance at wave onset. During drowsiness, slow roving lateral eye movements may be seen in which the artifact shows a more rounded appearance (see Figure 6-10). Therefore, identification of both eyeblink and lateral eye movement artifact requires a combination of wave pattern recognition and localization of the electrical events to their characteristic locations.

Slow Roving Eye Movements of Drowsiness and Sleep

Slow roving eye movements of drowsiness mentioned earlier represent an important example of lateral eye movement artifact. This type of eye movement shows an electrical pattern that is similar to other lateral eye movements, with a "positive phase reversal" at the anterior temporal electrode on the side to which the eyes are moving and a "negative phase reversal" at the opposite anterior temporal electrode. The slow roving version of lateral eye movements has a distinctively rounded, more prolonged shape (see Figure 6-10). In contrast, lateral eye movements occurring during wakefulness or during REM sleep tend to be quicker and have a more squared-off onset. Correct identification of

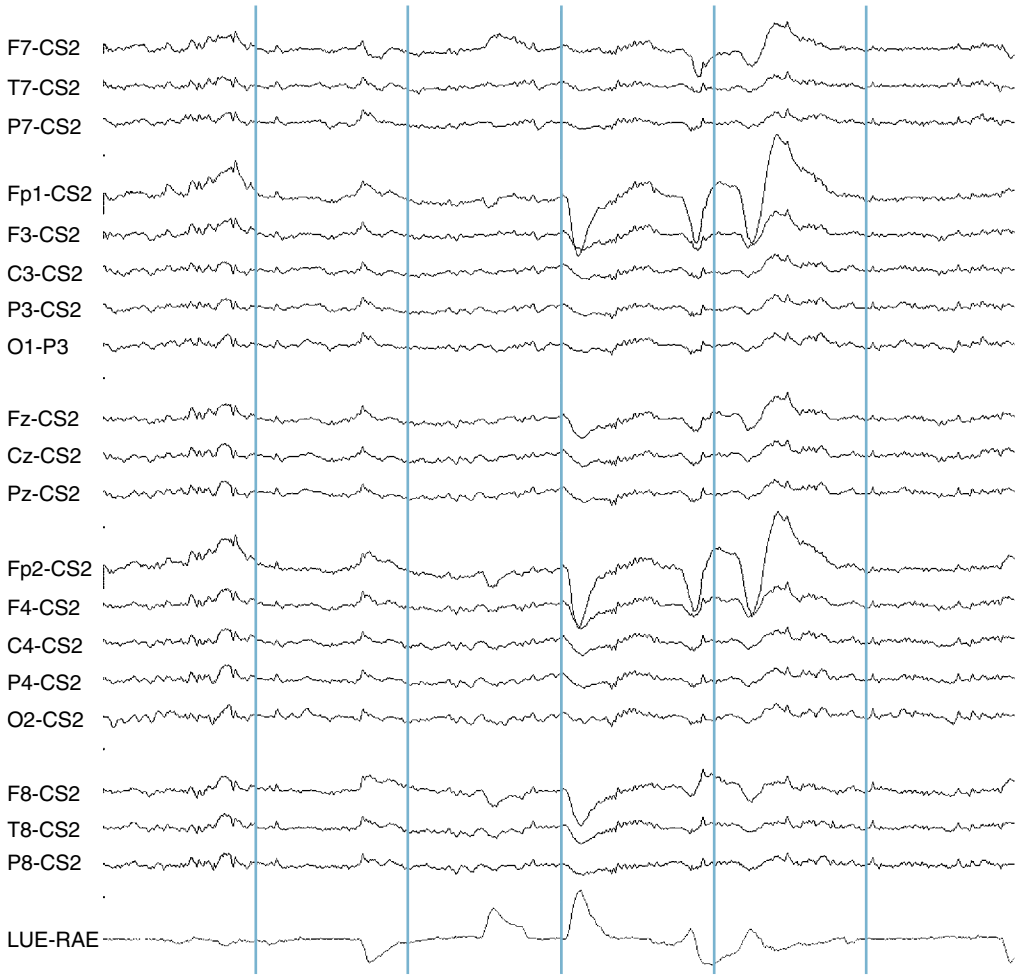


Figure 6-8 Three eyeblink artifacts are shown in a referential montage, with each active electrode compared to a CS2 reference, a location on the back of the neck distant from the eyes. The artifact is of high voltage in the Fp1 and Fp2 channels and is picked up to a lesser extent in F7 and F8. More posterior channels are neutral. Low-voltage EKG artifact can be seen in all channels indicating that it is being picked up by the reference electrode.

eyeblink artifact and the various lateral eye movement artifacts can be helpful in determining sleep stage.

Nystagmus Artifact

Nystagmus artifact is a special example of lateral eye movement artifact, identifiable by its rhythmicity. It is easy to distinguish from repetitive waveforms of cerebral origin based on its characteristic polarity in the anterior temporal electrodes (F7 and F8). Figure 6-11 shows a repetitive, rhythmic waveform for which the polarity of the phase reversal is opposite on the left and right sides at any given moment, indicating a repetitive lateral eye movement consistent with nystagmus.

Lateral Rectus Spikes

In addition to the types of lateral eye movement artifacts described earlier, lateral eye movements may also cause an artifact that appears as a sharp transient in the anterior temporal area with each lateral eye movement (see Figures 6-12 and 6-13). This type of artifact is important to recognize so as not to mistake these sharp transients for true epileptic spikes. It is hypothesized that these sharp transients represent electromyogram (EMG or muscle) artifact from activation of the lateral rectus muscle during lateral eye movements.

Electrode Pops and Other Single Electrode Artifacts

Electrode Pops

Electrode pops may occur because of a buildup of static charge in an individual electrode followed by a quick release of that charge. Sometimes an electrode pops because it has been poorly applied, but frequently there is no clear explanation for electrode popping. Electrode pops often have a distinctive shape (see Figure 6-14), although the morphology of electrode pops may vary.

The key attribute of the electrode pop is the strict localization of the event to a single electrode. Because, by its nature, an electrode pop is confined to a single

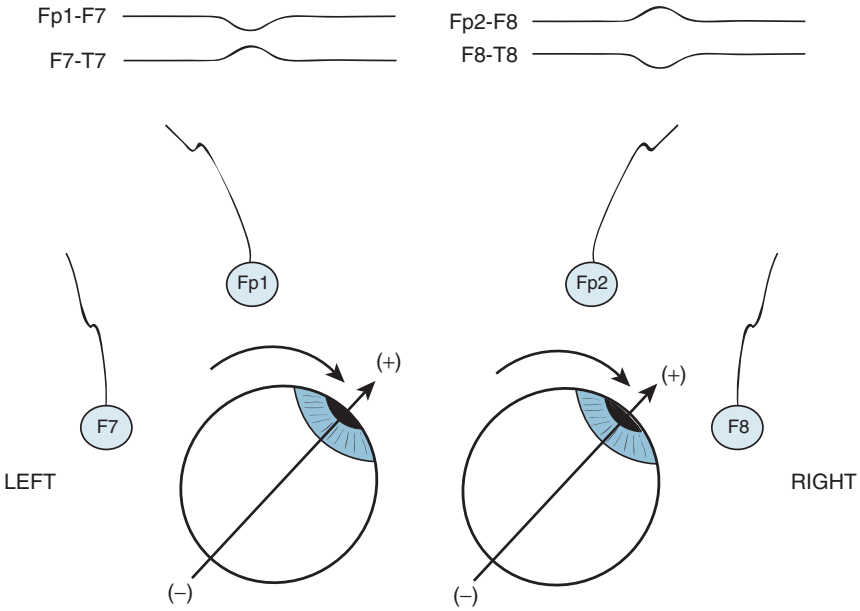


Figure 6-9 Lateral eye movements create a specific type of artifact in the EEG. The diagram here shows the example of both eyes gazing to the right, as seen from above. Because the cornea holds a net positive charge and the posterior pole of the eye a net negative charge, a lateral eye movement creates opposing situations for each pair of frontopolar and anterior temporal electrodes (Fp1 and F7, Fp2 and F8). When the eyes move to the right, the right anterior temporal electrode (F8) is exposed to the positive charge of the cornea of the right eye. With F8 being more positive than Fp2, the "positive phase reversal" depicted above the right eye results. At the same time, the left anterior temporal electrode is exposed to a net negative charge by the medial movement of the left eye, as shown in the figure. This results in a simultaneous "negative phase reversal" appearing at the left anterior temporal electrode (F7). Lateral eye movements are therefore identifiable by the very characteristic simultaneous appearance of a "negative phase reversal" in one anterior temporal electrode and a "positive phase reversal" in the opposite anterior temporal electrode.

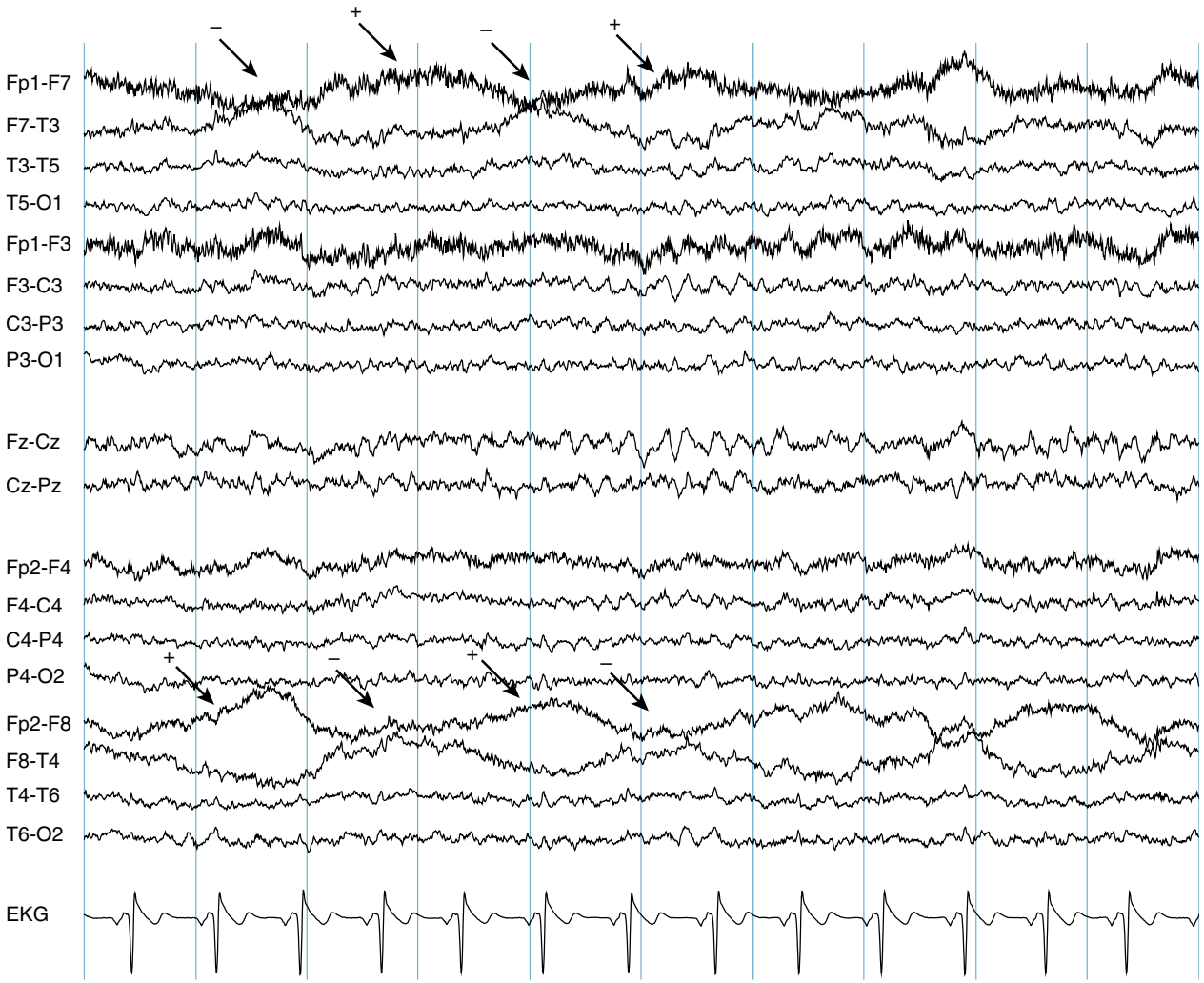


Figure 6-10 Slow roving lateral eye movements of drowsiness have a characteristic appearance based on the polarities seen with lateral eye movement artifact, as described in the previous figure. In this example, "negative phase reversals" are seen in the F7 electrode at the same time as "positive phase reversals" are seen in the F8 electrode (arrows). Each electrode experiences positivities and negativities of more or less equal duration, reflecting sequential slow shifting of the eyes to the left and then to the right under closed eyelids. This differs from voluntary saccadic movements, which manifest sharper waveforms and also do not typically give equal time to the leftward and rightward phases of the movements.

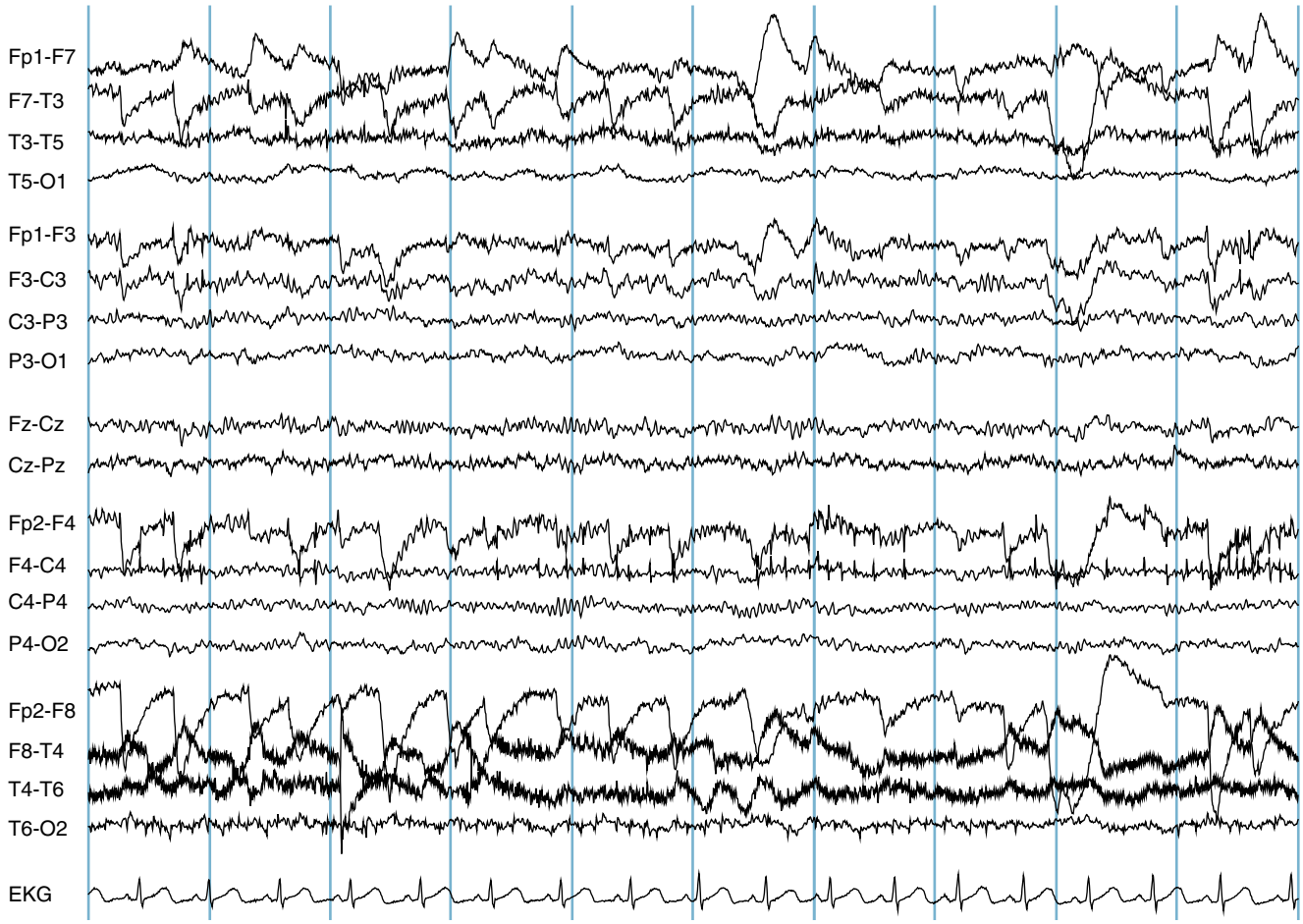


Figure 6-11 The characteristic appearance of nystagmus is seen in the form of repetitive lateral eye movement artifact with sharp contours maximum in the anterior temporal electrodes, F7 and F8. Note the opposite polarities at any given time in the F7 and F8 electrodes and compare the more jagged contours of the eye movement artifact seen here with those of the slow roving lateral eye movements in Figure 6-10.

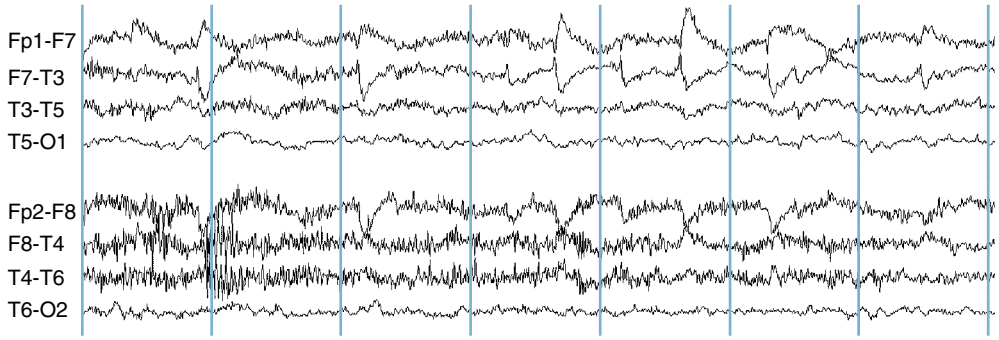


Figure 6-12 Lateral rectus spikes are seen in the left anterior temporal electrode, F7. The timing of the lateral eye movements can be discerned by the presence of a "positive phase reversal" seen at the same time in F7 as a "negative phase reversal" is seen in F8. A sharp transient is seen at the onset of each positivity in F7. Although this transient does resemble an epileptic spike, the fact that it occurs at the onset of each lateral eye movement helps to identify it as a lateral rectus spike artifact on gaze to the left.

electrode, it is said to "have no field." This is an important concept in artifact identification. A "biological" (brain wave) event usually manifests some amount of an electric field in at least some of the surrounding electrodes—the voltage of the event tapers away from the point of maximum in a more or less gradual fashion. However, if a wave is caused by a problem in a single electrode, be it a "pop" or a problem with the contact, the field of the event will be restricted to a single electrode; the adjacent electrodes perceive nothing at the moment of the "pop."

Figure 6-15 shows an example of an electrical event completely restricted to a single electrode, T3. Careful scrutiny of surrounding electrodes reveals no evidence

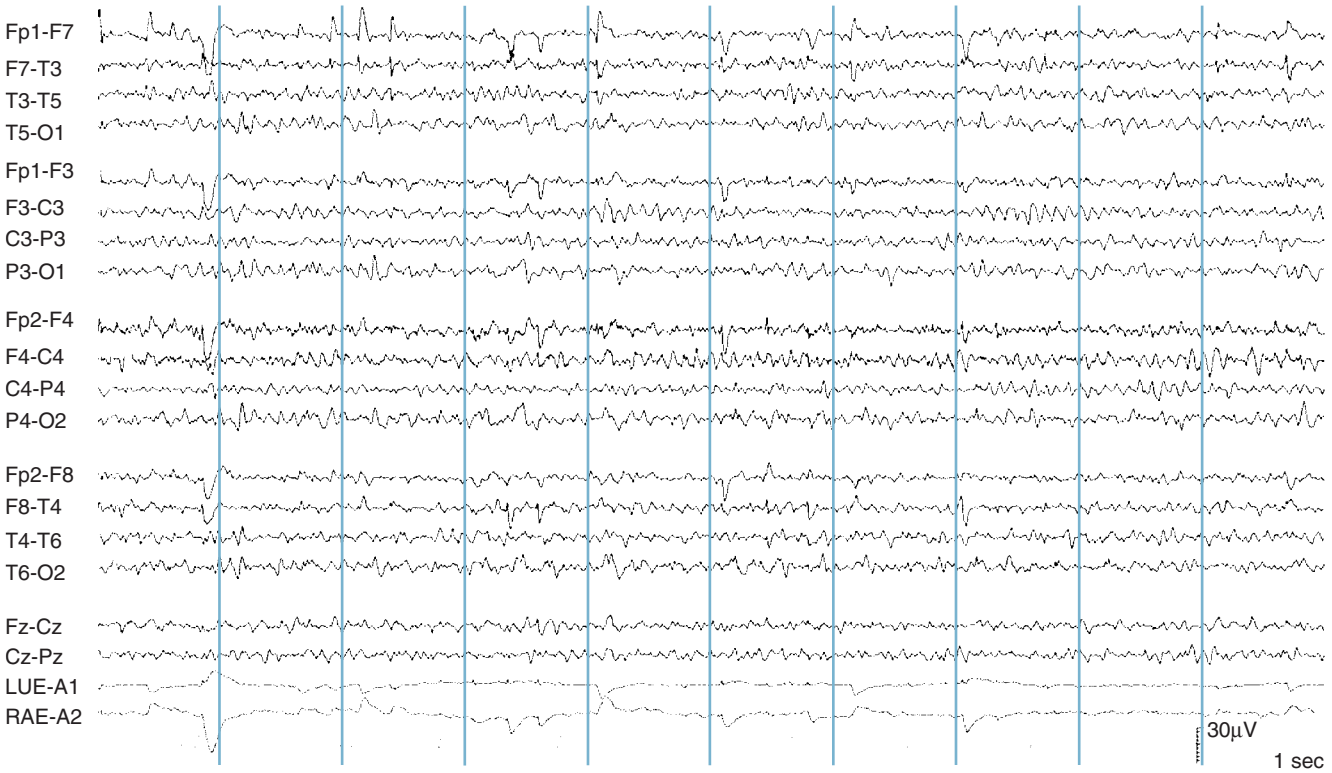


Figure 6-13 The spikes shown on this page are not as easy to identify as lateral rectus spikes compared to the examples shown in the previous figure. The two eye-monitoring channels, LUE-A1 ("left under eye"–left earlobe) and RAE-A2 ("right above eye"–right earlobe), clearly establish that the apparent spikes seen in the F7 electrode correspond to lateral eye movements.

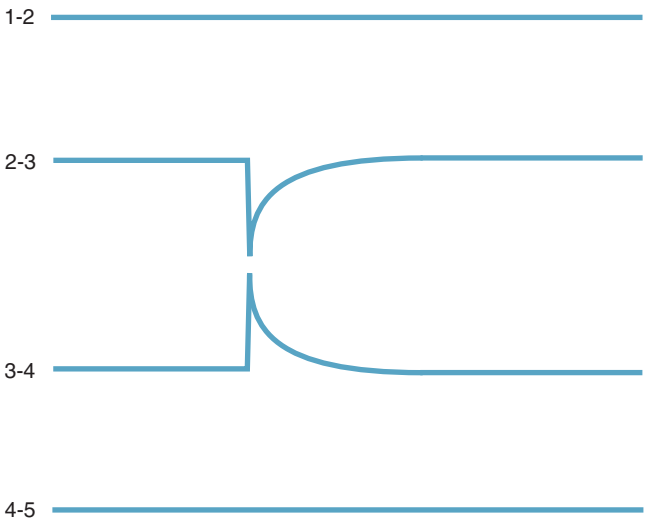


Figure 6-14 A schematic representation of an electrode pop shown in a hypothetical bipolar chain formed from five adjacent electrodes. The two most characteristic features of an electrode pop artifact are evident in the shape of the discharge and the fact that it is isolated to a single electrode. The shape shown in this figure with a fast upswing and a slow decay is highly suggestive of electrode popping, although electrode pops may assume other shapes as well. The high voltage event in "Electrode 3" with no voltage change whatsoever in the adjacent electrodes is characteristic of this type of artifact.

of an electric field or gradient in any of the adjacent electrodes (F7, T5, or C3). Another way of proving the isolation of this event is ask whether, if the two channels that include the T3 electrode were to be erased from the EEG, would it still be possible to identify the moment of the would-be spike? In this example, not even the smallest deflection in the adjacent channels can be seen, establishing this as a single electrode event representing and an electrical artifact rather than an electrical event of cerebral origin.

Although electrode popping often occurs as an isolated event, sometimes highly repetitive popping can occur which can resemble seizure activity. Figure 6-16 shows an example of repetitive electrode popping in the P4 electrode. Note that the adjacent electrodes, O2, C4, and T6, show no evidence of the deflection. The

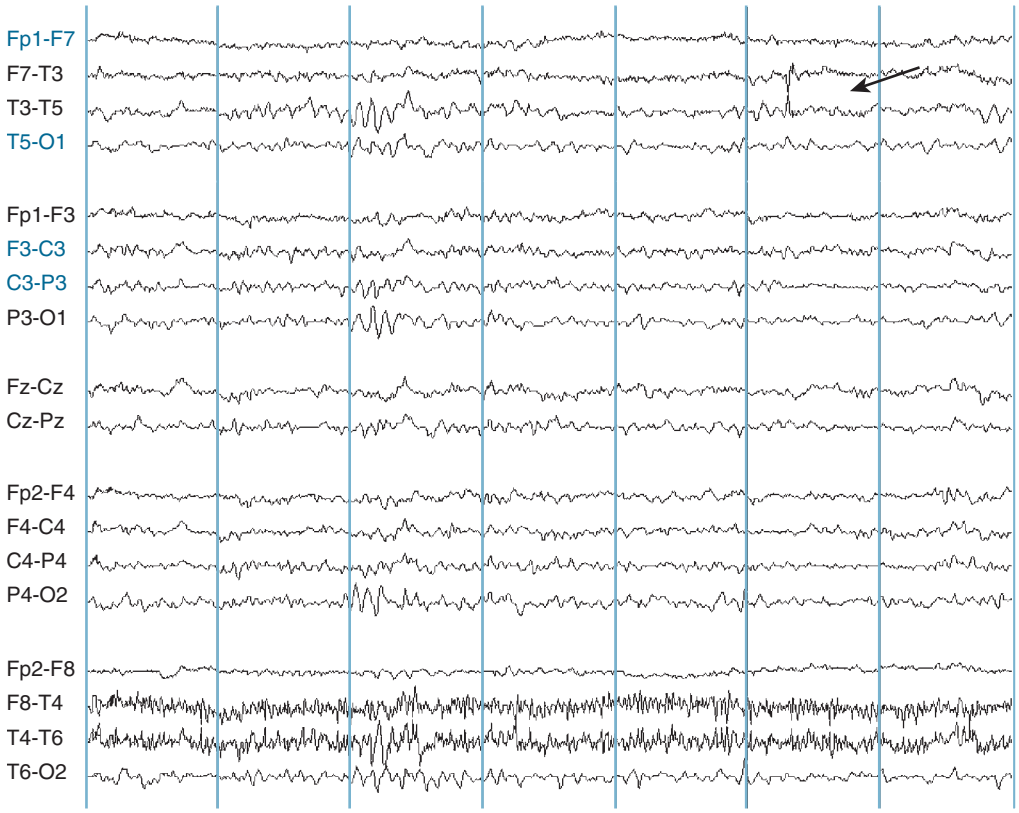


Figure 6-15 A single electrode artifact is noted in the T3 electrode (gray arrow). Morphologically, this sharp transient could be difficult to distinguish from a true epileptic spike. The main clue to its noncerebral origin is the complete lack of an electric field in the surrounding electrodes (F7, F3, C3, P3, and T5). This is confirmed by the absence of any simultaneous deflection in the channels that include these electrodes: Fp1-F7, T5-01, F3-C3, and C3-P3 (highlighted in blue).

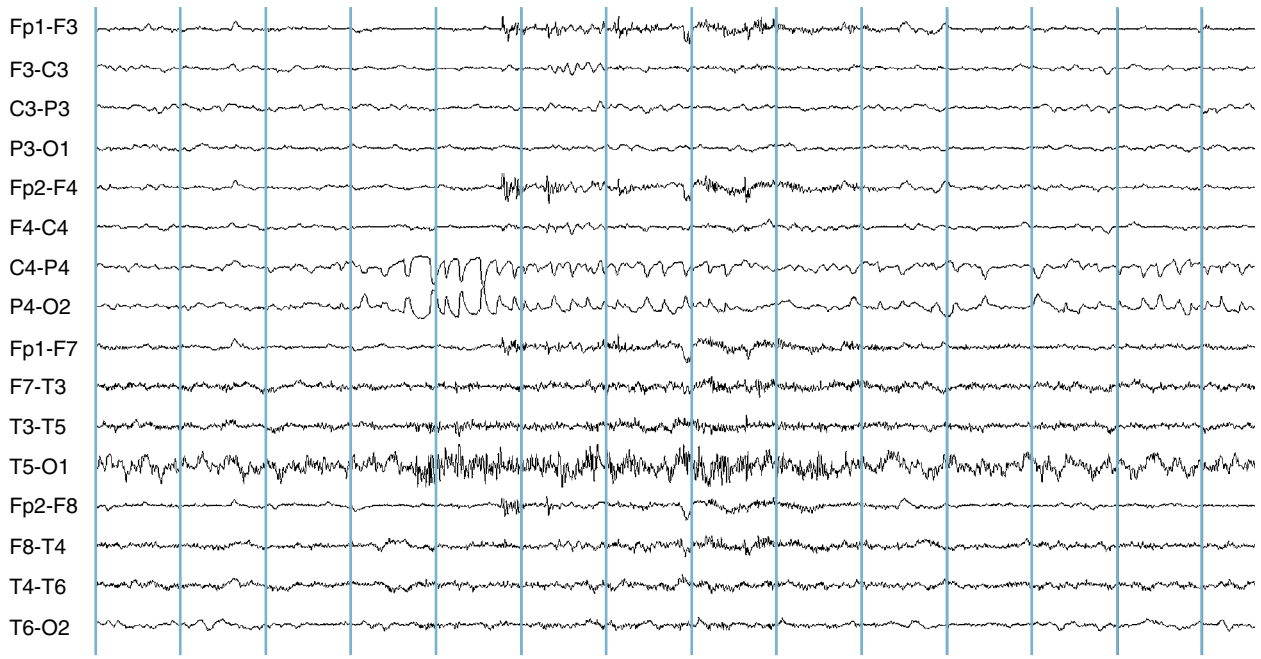


Figure 6-16 In this figure, there is a repetitive "popping" in the P4 electrode. Note that there is no electric field surrounding this event, confirming that it is an artifact—the surrounding electrodes, T4, C4, and O2 are seen to be quiet (while the deflection is seen in the P4-O2 channel, the adjacent T6-O2 channel is completely quiet). Given the repetitive nature of this artifact, it would be possible to mistake it for a seizure discharge. The fact that this waveform is restricted to a single electrode is the clue to its noncerebral origin. The "mirror image" configuration of the C4-P4 and P4-O2 channels is also characteristic.

same effect is seen in Figure 6-17—a deflection that has some resemblance to a spike-wave discharge in T4 is seen to be an electrode pop on closer analysis. Figure 6-18 shows a high voltage, complex waveform confined to the Fp2 electrode. Again, because this waveform is of moderately high voltage but cannot be detected in the adjacent channels (Fz-Cz, F4-C4, and F8-T4), these deflections are correctly interpreted as an artifact in the Fp2 electrode.

As with most rules, exceptions exist. Is it possible for a true cerebral event to be detectable only in a single electrode? True cerebral events isolated to a single electrode may occur, but they are relatively rare. The phenomenon is most likely to occur with a low-voltage event. Turning up the amplifier gain aggressively can help identify small deflections in adjacent electrodes if they are present. Events localized to a single electrode are probably more common in newborns than in older children or adults. The higher the voltage of a true cerebral event, the less plausible that it is localized to a single electrode.

The Single "Bad Electrode"

A poor electrode contact can cause a distinctive, high-voltage pattern in which adjacent channels in a bipolar montage appear to mirror one another (see Figure 6-19). In this example, the high-voltage pattern seen in the F3-C3 channel is mirrored in the C3-P3 channel implying a poor electrode contact at C3. It is especially useful to recognize the patterns caused by a poor electrode contact when it only occurs intermittently in a tracing. Knowing that there is a poor contact in C3, the alert reader immediately becomes suspicious of any high-voltage deflection that may be seen at any other time in that electrode, realizing that it could represent electrode artifact from the poor contact.

OTHER TYPES OF ARTIFACT

Muscle Artifact

Muscle artifact is one of the most commonly encountered artifacts in the EEG and is especially common during wakefulness. Muscle artifact is recorded most often by electrodes that overlie the muscles of the scalp: the frontalis, temporalis, and occipitalis muscles. The frontopolar and midtemporal electrodes, Fp1, Fp2, T7, and T8 are most frequently affected (as was seen in Figure 6-7), but most others can be affected as well. Because there is almost no muscle over the vertex of the skull, muscle artifact is usually not seen in the midline (Cz, and Pz) electrodes.

Muscle artifact appears as a fast wave that appears to "turn the channel black." Occasionally muscle artifact can be difficult to distinguish from a train of rapid spikes, although certain characteristics help make the distinction. Muscle artifact usually fires at a fast, uncountable rate, and it can be difficult to appreciate a specific wave morphology from within this rapidly firing, heterogeneous mixture of waves. Instead, muscle artifact usually consists of a mixture of spike-like potentials with variable shape and height. An interesting exception to this rule is the repetitively firing isolated motor unit. This type of artifact appears as a repetitive, low-voltage monomorphic spike that has a stuttering firing frequency and may persist for varying lengths of time. Although the firing rate does vary, it does not show sustained trends toward

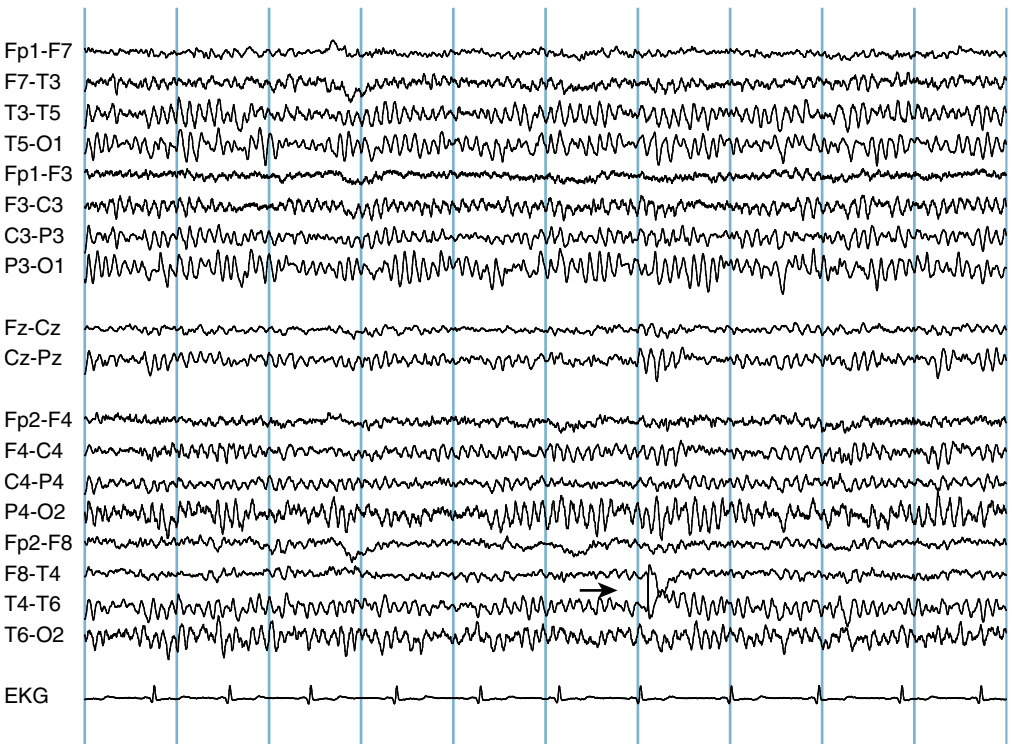


Figure 6-17 The discharge seen in the seventh second of this page in the T4 electrode has a morphology potentially suggestive of a spike-wave discharge (arrow). Note that even though the deflection is fairly large, there is no evidence of the discharge whatsoever in the adjacent electrodes (C4, T6, or F8). The presence of this relatively high-voltage event in a single electrode helps confirm that it is an electrode artifact rather than a spike-wave discharge.

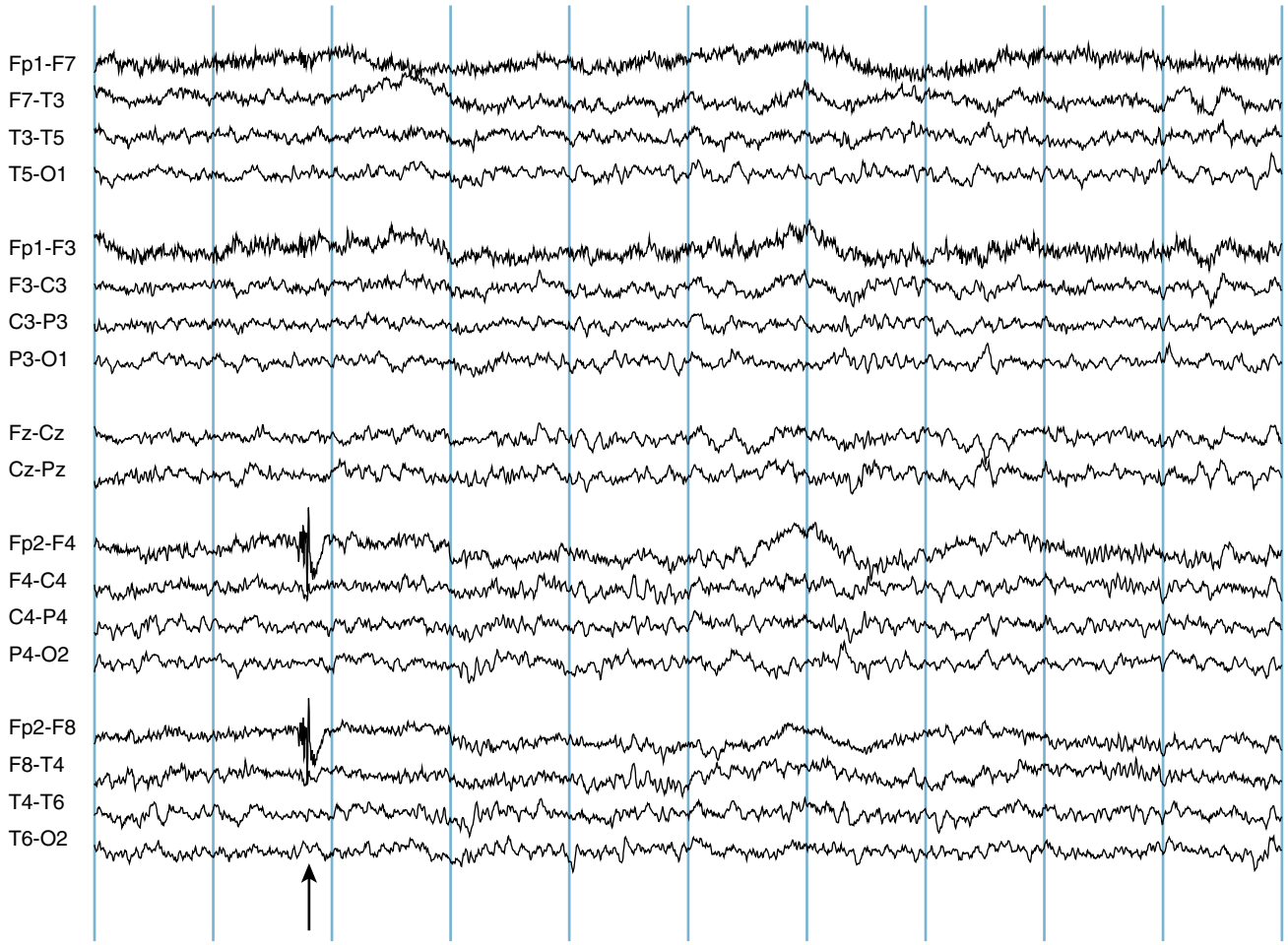


Figure 6-18 The fast, multiphasic waveform seen in the Fp2 electrode (arrow) is more complex than what has been seen in previous examples and clearly represents an example of electrode artifact. Once again, the fey feature is that the event is restricted to a single electrode. The cause of such artifacts is not always clear. Possible explanations include a poor electrode contact, the patient touching the electrode, or some other momentary electrical contamination of the electrode.

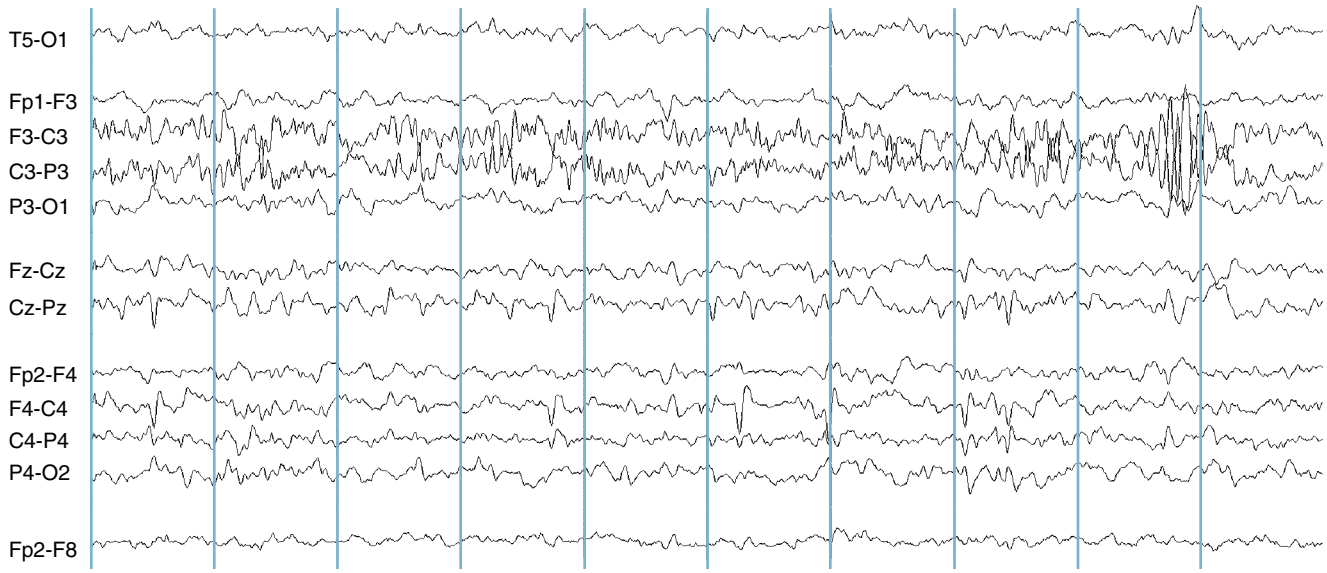


Figure 6-19 The waveforms of the F3-C3 and C3-P3 channels mirror each other and are of higher voltage than their neighbors. This characteristic pattern is caused by a faulty electrode contact at C3.

speeding up or slowing down as seizures tend to do (see Figures 6-20 and 6-21). Motor unit spikes usually do not have an electric field, that is, they are not seen in adjacent electrodes. When seen over the temporalis muscle, they may be terminated by asking the patient to relax the jaw, although the firing of an individual motor unit may not be easily under the patient's control.

Sweat Artifact

A patient who is sweating may manifest an artifact that consists of long duration, high-voltage potentials recognizable by their distinctive, prolonged shape (see Figure 6-22). This slow, swaying artifact is caused by electrolyte bridges that form between adjacent electrodes because of excess sweating. Sweat artifact can be eliminated by anything that stops the patient from sweating, including cooling the room where the EEG is being performed.

EKG Artifact

EKG artifact is usually easy to identify. Its most distinctive features are its shape and its rhythmicity. Most EEG tracings are recorded with an EKG channel. Suspicion that a spike is really an example of EKG artifact can be confirmed by matching the timing of the deflection in question with the QRS complex in the EKG channel. Also, the width of EKG artifact should be similar to the width of the QRS complex in the EKG channel (see Figures 6-23 and 6-24, *A* and *B*).

EKG artifact tends to occur in certain contexts. Because the heart is on the left side of the body in most patients and the left ventricle makes a bigger contribution to the QRS complex than the right ventricle, the left temporal electrodes are more likely to pick up this type of artifact. Patients with higher voltage QRS complexes from ventricular hypertrophy may have greater degrees of EKG contamination in the scalp; in those

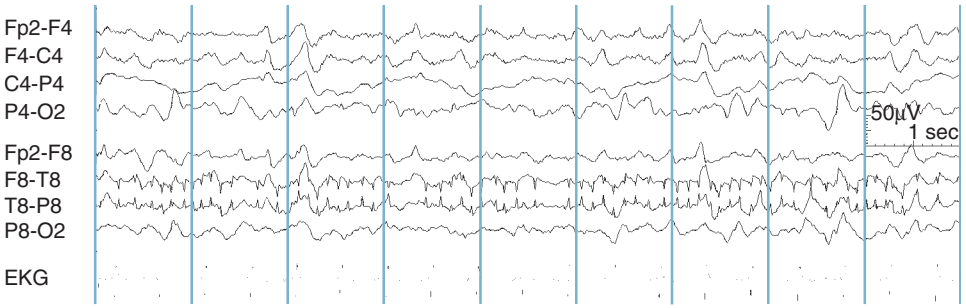


Figure 6-20 A spikelike discharge is seen in the T8 electrode and represents an artifact caused by a repetitively firing motor unit. The discharge does not maintain a trend toward speeding up or slowing down but has a sputtering characteristic. No electric field is detected by adjacent electrodes.

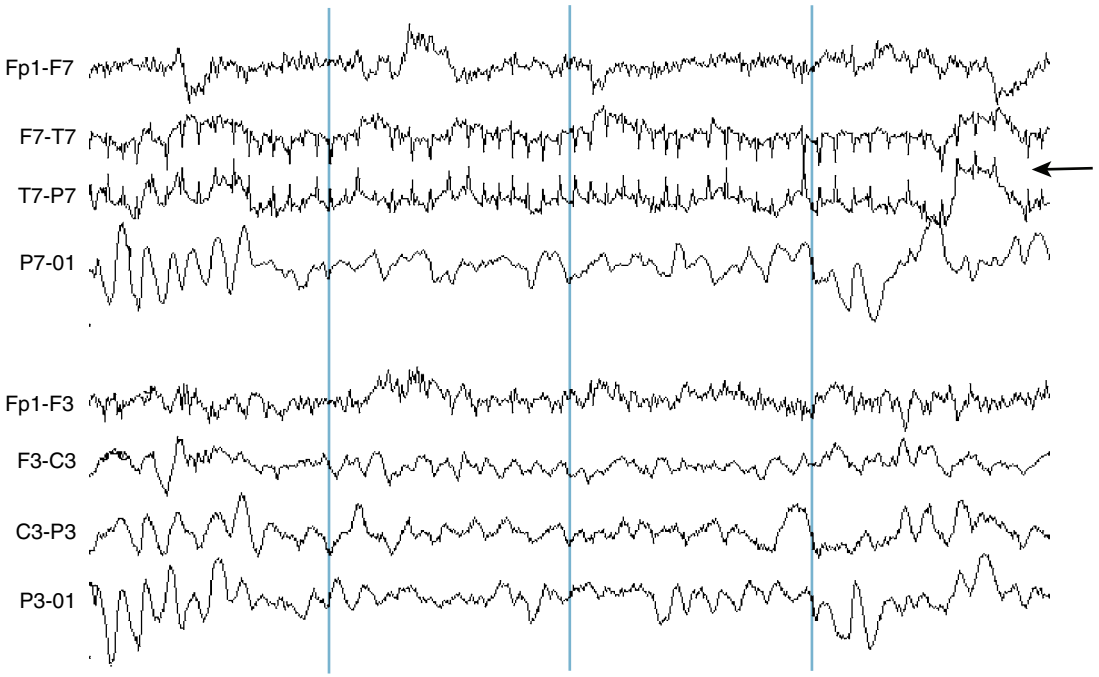


Figure 6-21 A continuously firing motor unit is seen in the channels shared by the T7 electrode (arrow). The unchanging morphology and the lack of a sustained tendency toward speeding up or slowing down help identify this as a motor unit artifact.

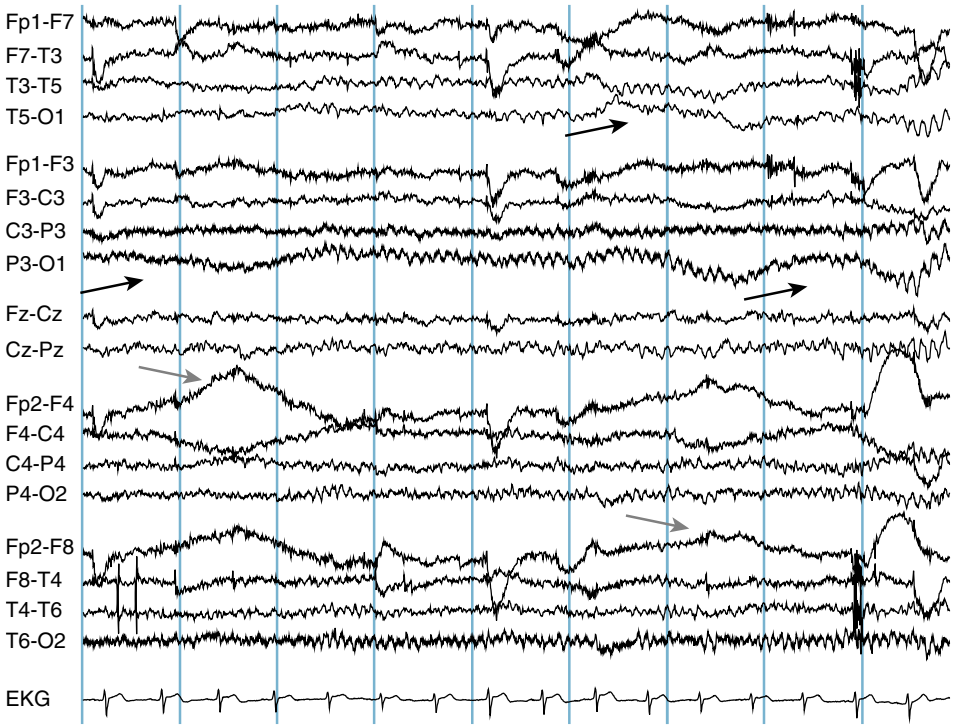


Figure 6-22 Sweat artifact usually appears as slow, wandering deviations of a channel's baseline. In this example, sweat artifact can be seen in the posterior channels on the left (black arrows). Anteriorly, the large deflections may be caused by a combination of some eye movement artifact and sweat artifact (gray arrows).

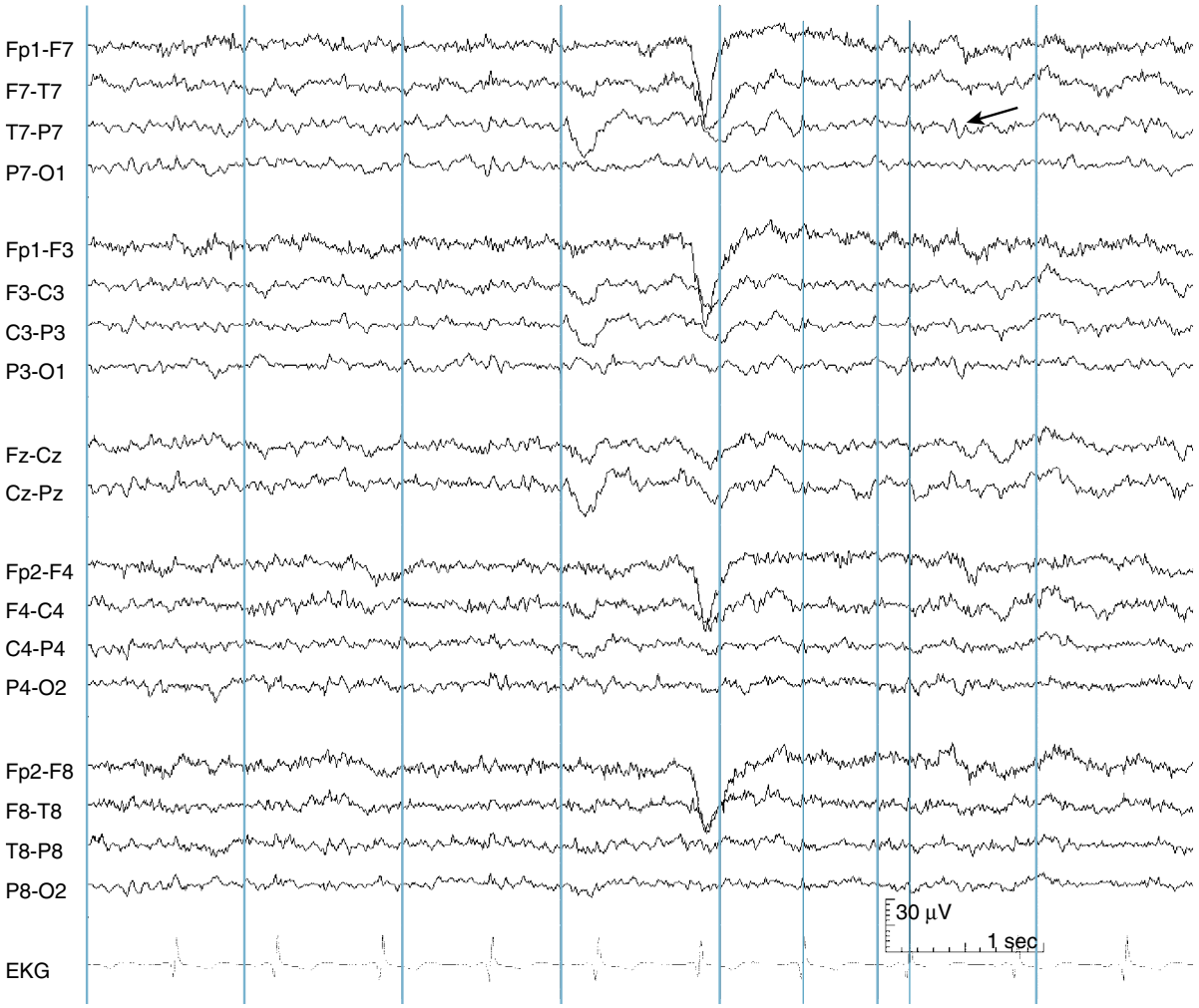


Figure 6-23 Electrocardiogram (EKG) artifact can be difficult to distinguish from low-voltage spikes. In this example, EKG artifact can be seen in several channels in synchrony with the QRS complexes seen in the EKG channel (see light blue vertical lines). The apparent low-voltage spike indicated by the arrow is not an example of EKG artifact because it is not aligned with an EKG complex; it likely represents a fragment of a mu rhythm.

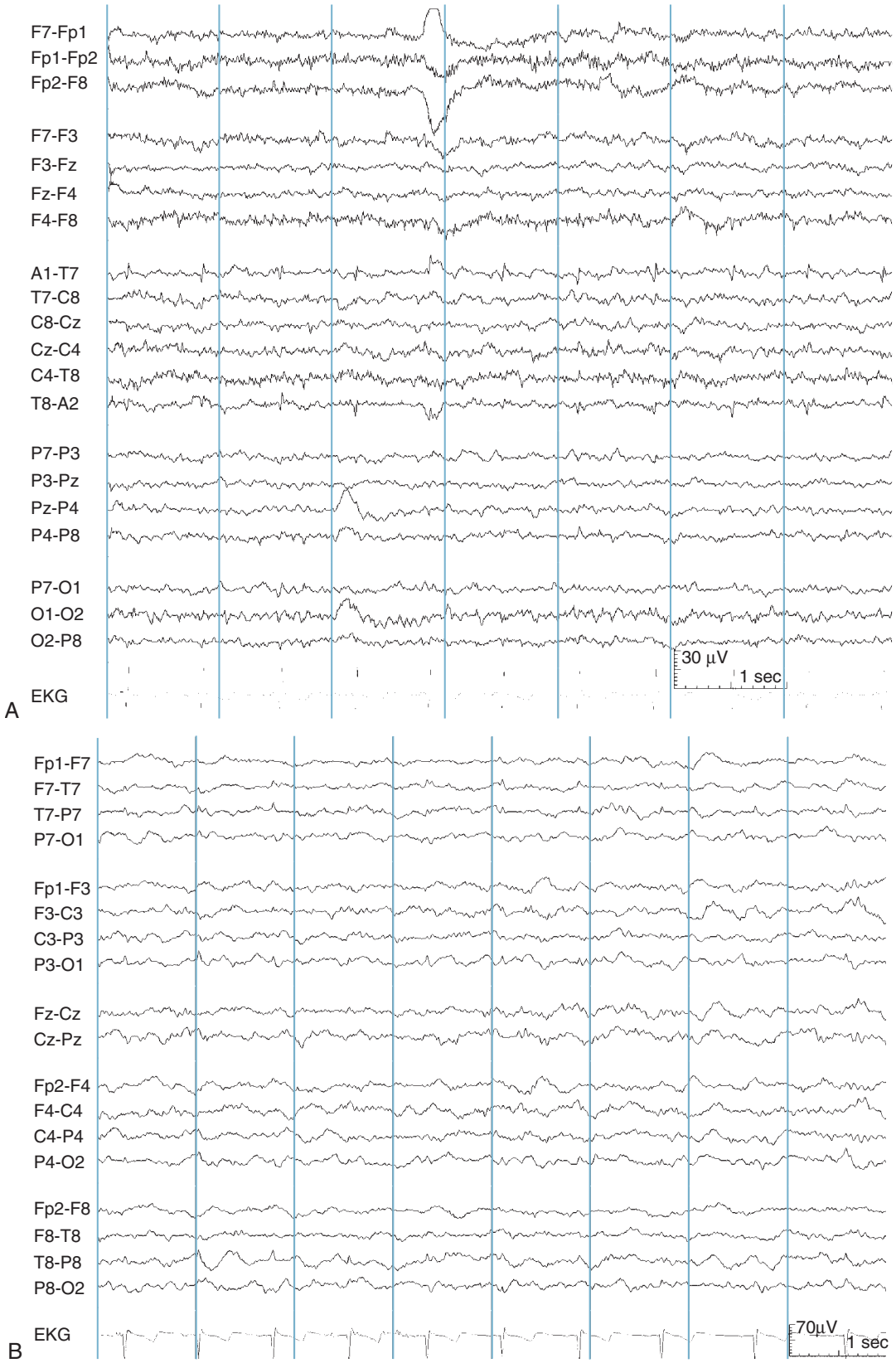


Figure 6-24 **(A)** Electrocardiogram (EKG) artifact is seen in the A1-T7 and T8-A2 channels. This spike-like artifact lines up with each QRS complex in the EKG channel. Because the amount of EKG signal differs between the earlobe and the midtemporal electrode, these two channels are a common location for EKG artifact. Unlike in this example, the artifact is often of higher amplitude on the left compared with the right. **(B)** Even without the aid of the EKG channel, the regularity of the low amplitude spike-like artifact seen in multiple channels makes it readily identifiable as EKG artifact.

with right ventricular hypertrophy, the EKG artifact may be more prominent on the right side of the head. Also, patients with wider necks tend to transmit EKG artifact more readily to the head. Finally, the height of the EKG complexes as measured on the scalp tend to vary with the respiratory cycle.

Given the fact that EKG signal is present everywhere on the scalp, it is worthwhile to consider why EKG artifact is not seen in every channel. Because almost exactly the same amount of EKG signal is present in adjacent electrodes, the EKG complex efficiently cancels itself out in bipolar comparisons of nearby electrodes. Even many reference electrodes have similar amounts of EKG signal compared with the scalp, yielding EKG-free referential tracings (although the choice of certain references may add an undesirable amount of EKG artifact to the tracing). The unexpected appearance of EKG artifact in a lone electrode can serve as a clue to a poor electrode contact. When two electrodes with good impedances are subtracted from one another, the EKG artifact cancels out efficiently. When there is a poor electrode contact, however, the poorly applied electrode picks up the EKG signal to a different extent than its properly applied neighbor. When the signal from the good electrode is subtracted from signal from the bad electrode, there is a net difference in the amount of EKG picked up by the two, and the net difference appears in the display as EKG artifact.

Apart from the situation of poorly applied electrodes, EKG artifact would be expected to appear when any two regions of the scalp are compared that carry different amounts of EKG signal for anatomical reasons. The most common example of this is seen in the transverse bipolar montage in the channel where the left earlobe is compared with the left midtemporal electrode (A1-T7). For anatomical reasons, the left earlobe carries less EKG signal than the left midtemporal area. Because of the left-sided orientation of the heart, the same effect is seen less dramatically on the opposite side, where T8 is compared with A2 (see Figure 6-24). Among all the electrode pairs in standard bipolar chains, these pairings are the most likely to manifest EKG artifact in normal patients with well-applied electrodes.

Pulse Artifact

Pulse artifact is seen when one electrode happens to be placed directly on an artery in the scalp (often the temporal artery), creating an artifactual waveform related to the pulsations. This type of artifact has also been called the hemoballistogram. In this case, a wide pulse artifact is seen in synchrony with the EKG. When an EKG channel is present, it is easy to link this artifact to the heart rate (note that the pulse artifact does not occur at the exact moment of the EKG but follows it by a set interval). Even when there is no EKG channel, this type of artifact is still usually easy to identify based on its heartbeat-like rhythm, the fact that it is much wider than the QRS complex of the EKG, and its presence in a single electrode (see Figure 6-25). More rarely, a related type of movement artifact may be seen that is synchronized to the patient's EKG in cases in which the patient's body makes a low-amplitude rocking movement with a hyperdynamic heartbeat.

60-Hz Artifact

The problem of 60-Hz artifact is discussed in more detail in Chapter 7, "Filters in the EEG," as a specific filter is available to help suppress this type of artifact. Small amounts of AC current from the power mains that surround the patient flow through the patient's body accounting for this type of artifact. (In countries that use line frequencies of 50 Hz, 50-Hz artifact is seen instead.) Because this small amount of 60-Hz current is present in similar amounts throughout the body, this signal is usually canceled out by the subtraction process used by the common mode rejection amplifiers in the same way that EKG artifact is canceled out as described earlier. Here again and similar to the case of EKG artifact, a poorly applied electrode is more likely to manifest 60-Hz artifact because of the difference between the amount of 60-Hz activity the poorly applied electrode picks up compared with its neighboring well-applied electrodes. As with the unexpected appearance of EKG artifact in an isolated electrode as described earlier, the presence of 60-Hz artifact only in channels that include a particular electrode suggests that that electrode may have a poor contact (see Figures 6-26 and 6-27).

Electrical Artifacts from External Equipment

Many machines used in medical settings give off a variety of electrical signals. Fortunately, it is the nature of most of these devices to give off signals in a regular fashion. Electronic intravenous fluid pumps are a well-known source of repetitive electrical artifact. Mechanical ventilators and electric hospital beds are other possible sources of interference. A wave form that occurs with precise regularity is almost never of biological origin. Even though the electrocardiogram is generally considered a regular wave, when carefully measured, small variations are always seen between beats. When electrical complexes are seen in the EEG with strict regularity (no variation in the interval between the complexes), these almost always represent signals from medical devices. Repetitive, highly stereotyped, complex waveforms are also suspicious for electrical artifact.

Special Types of Motion Artifact

Certain types of motion artifact are seen fairly frequently in the EEG. Respiratory artifact occurs when breathing motions cause a gentle rocking of the head with each breath. Usually, the electrodes in contact with the bed (often the occipital or temporal electrodes) are preferentially involved. Linking the occurrence of what is usually a broad artifactual wave to the respiratory cycle can establish the source of this type of artifact. This task is made considerably easier when a respiratory channel is in use. Ventilator artifact can be considered a subset of either respiratory artifact or motion artifact; wave artifacts are created by body movements induced by the ventilator. A special case of ventilator artifact is seen

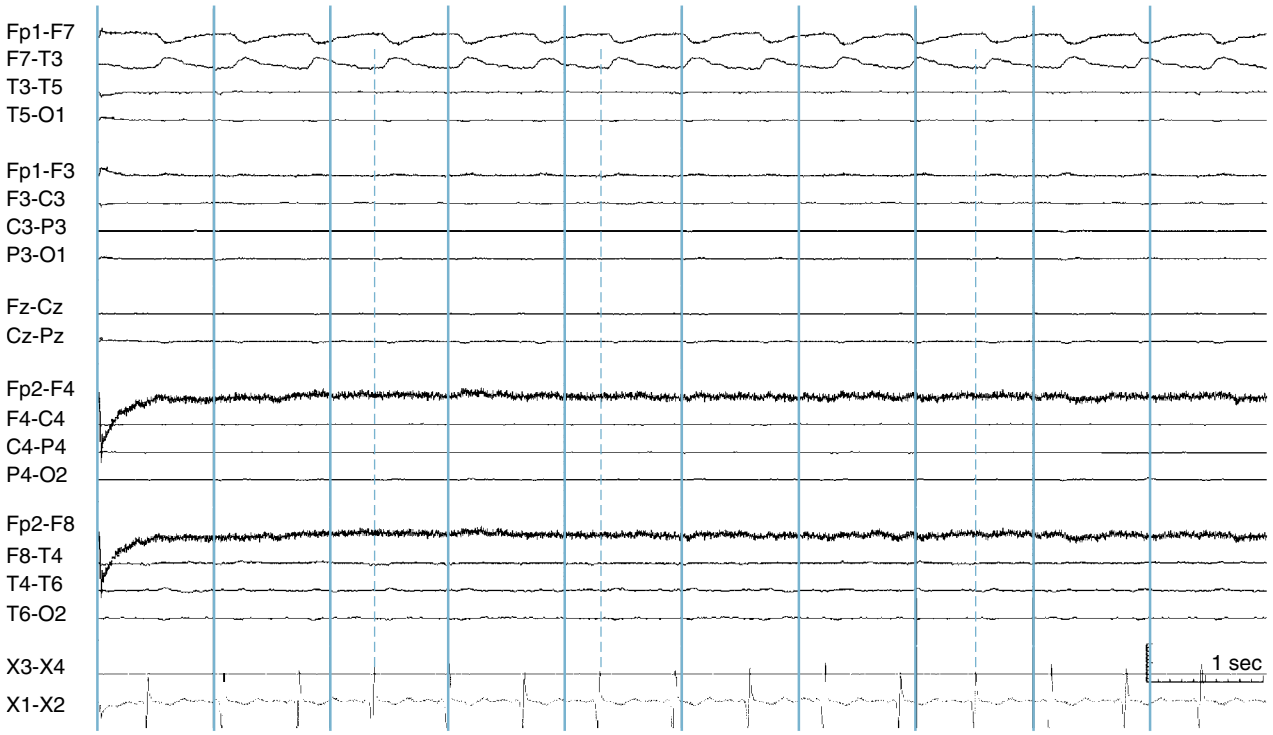


Figure 6-25 The regular deflections in the top two channels of this otherwise suppressed EEG represent pulse artifact in the F7 electrode, which likely lies on the temporal artery. Note that the EKG complexes consistently line up with the same phase of each wave (broken lines).

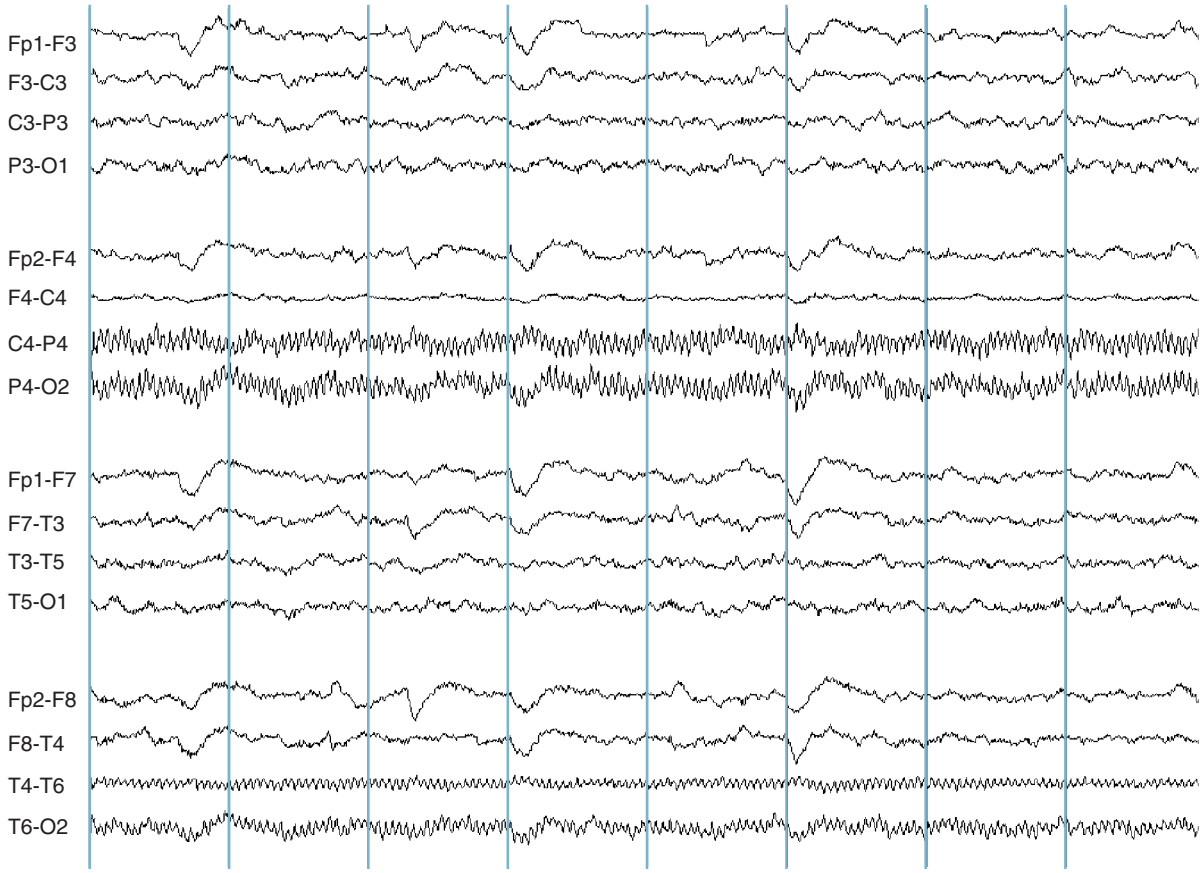


Figure 6-26 Prominent 60-Hz artifact is seen in the P4 and T6 electrodes. At first glance, 60-Hz artifact can be mistaken for muscle artifact, but the highly regular, sinusoidal pattern of the waves in these channels is not consistent with muscle activity. Sixty-hertz artifact may be seen at exactly 60 Hz, or as a subharmonic of 60 Hz. In this example the artifact has a frequency of exactly 20 Hz. Sixty-hertz artifact can often be eliminated after the fact with the use of a 60-Hz notch filter (see Chapter 7, "Filters in the EEG").

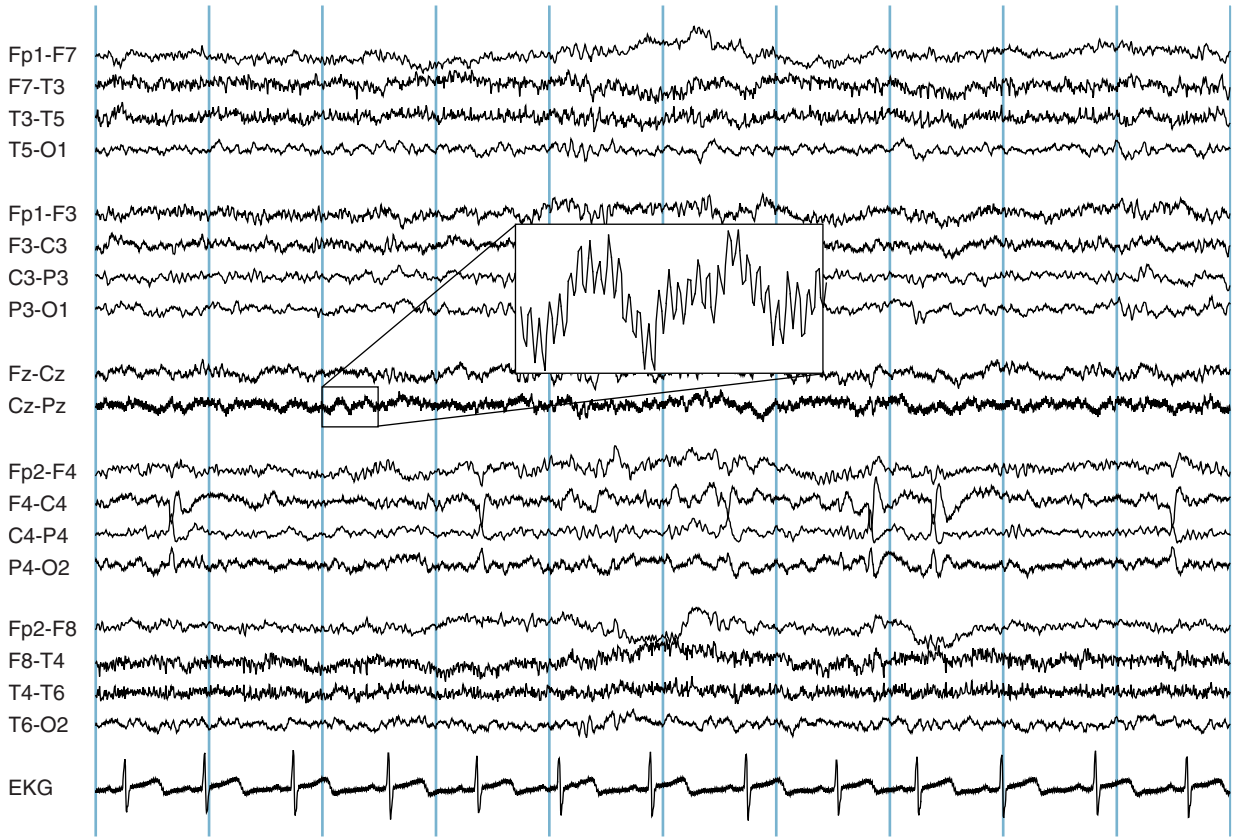


Figure 6-27 Sixty-hertz artifact in the Cz-Pz channel magnified to show the shape of the wave. Unmagnified, the channel simply looks "dark," but close inspection reveals a 60-Hz sinusoidal wave. The magnified area represents one half second. Because 30 waves are counted within the half-second area, the artifact's frequency is 60 Hz.

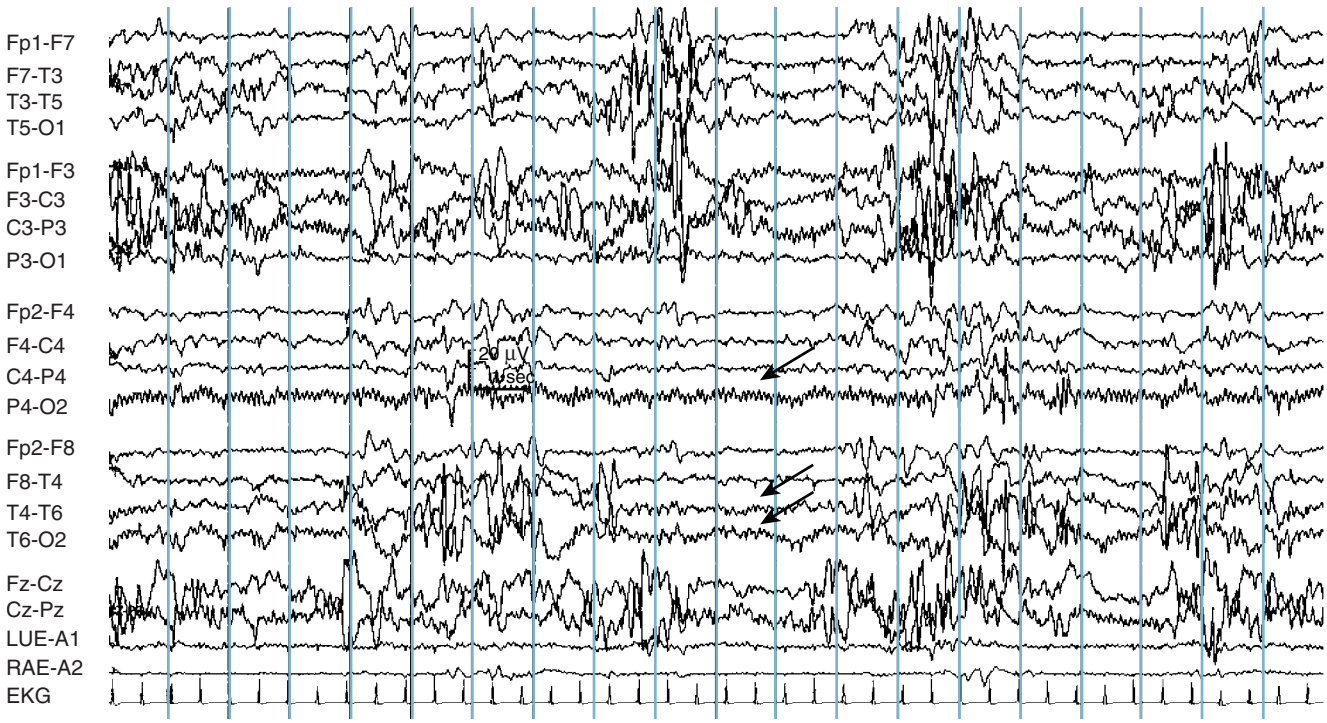


Figure 6-28 Motion artifact related to a high-frequency ("HiFi") ventilator is seen in three channels (arrows). The highly regular waveform is an indicator that this waveform is of mechanical rather than cerebral origin.

with high-frequency ventilators, which can cause high-frequency motion artifacts (see Figures 6-28 and 6-29).

Patting artifact occurs most frequently in children but is also seen in adults. When the patient's back is patted, a repetitive and regular motion artifact may appear in the EEG (see Figure 6-30). Chest physical therapy artifact may also cause a similar appearance. Usually, patting rhythms are maintained fairly regularly throughout their duration; it is uncommon for a caregiver's patting rate to increase or decrease substantially throughout its course, and abrupt pauses are more common (see Figure 6-31). This differs from seizure activity, which tends to gradually evolve in frequency throughout its course. The implied polarity of patting artifact waves usually is not "biologically plausible" for electrocerebral activity.

Hiccup Artifact

Hiccup artifact is a special type of motion artifact associated with hiccuping. Because each hiccup usually causes a similar movement in the patient, the EEG artifact caused by hiccuping is often stereotyped (see Figure 6-32). Occasionally hiccup artifact may resemble a polyspike discharge, but the field of the artifact is usually irregular enough that the distinction is not difficult.

Breach Rhythm

The breach rhythm is a distinctive rhythm that can be considered an artifact of a skull defect, usually from a previous neurosurgical procedure. Defects in the skull tend to allow preferential transmission of high-frequency activities. For this reason, higher voltage fast rhythms may be present over the area of a previous craniotomy site. Breach rhythms are discussed in more detail in Chapter 11, "Normal Variants in the EEG."

Chewing and Bruxism Artifact

Chewing artifact is usually easy to recognize, appearing as brief, repetitive muscle bursts, maximum in the temporal areas (see Figure 6-33). Chewing artifact can be seen while the patient is eating but sometimes occurs as a "nervous habit" even when the patient has nothing in his or her mouth. Chewing artifact may also be seen associated with chewing automatisms during complex partial seizures. Bruxism (tooth grinding) usually occurs during sleep, but may also be seen during wakefulness, especially in the mentally retarded population. Its appearance is similar to chewing artifact because both involve rhythmic tensing of the jaw muscles. Bruxism artifact is of longer duration and occurs at a slower pace than chewing artifact (see Figure 6-34).

Glossokinetic Artifact

Glossokinetic artifact is generated by movements of the tongue, which also has a net dipole (much like the globe of the eye), with the tip of the tongue being negative relative to the base. The appearance of this artifact in a tracing can be confirmed by having the patient reproduce it on the EEG by request. The patient is asked to move the tongue forward in the mouth repetitively by repeating syllables such as "la-la-la" or "ta-ta-ta." The field of glossokinetic artifact can extend over the whole scalp and can be recorded at highest voltage by electrodes placed on the face (see Figure 6-35).

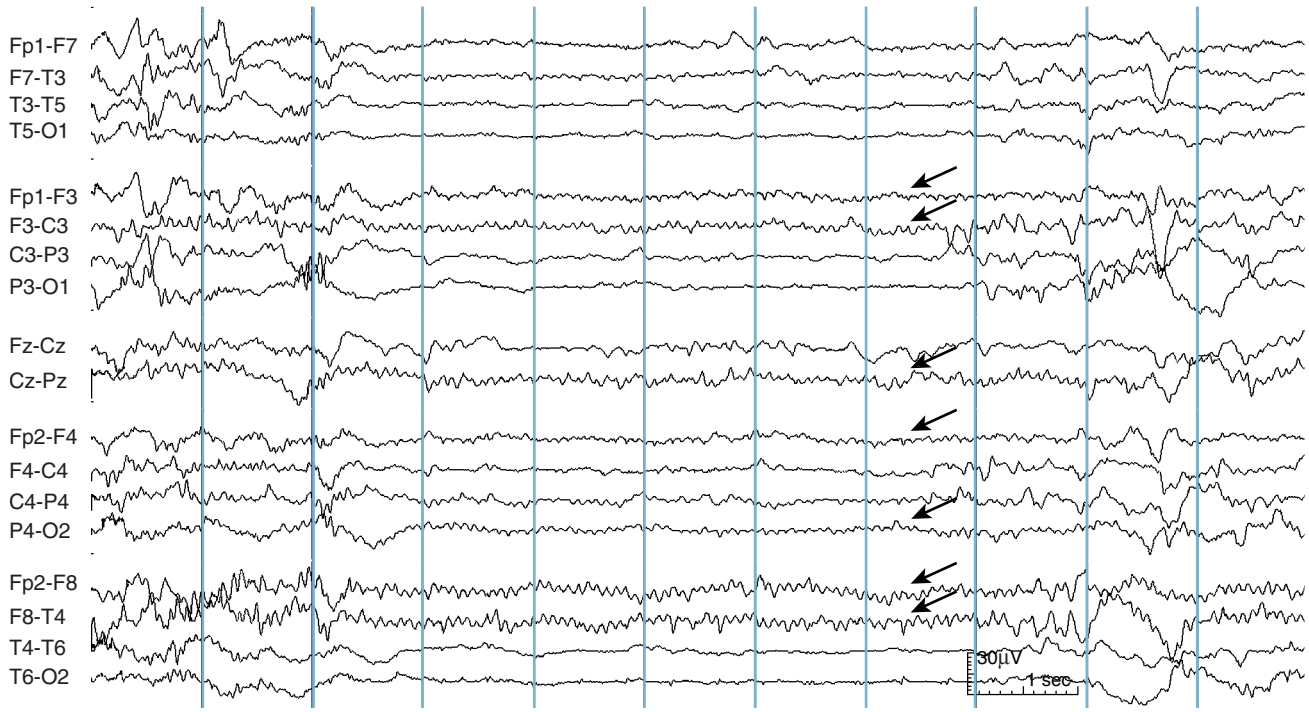


Figure 6-29 Highly regular, sinusoidal artifact caused by an oscillating ventilator can be seen in multiple channels (arrows). The long duration, "patchy" localization and lack of frequency evolution help to distinguish this waveform from a seizure discharge.

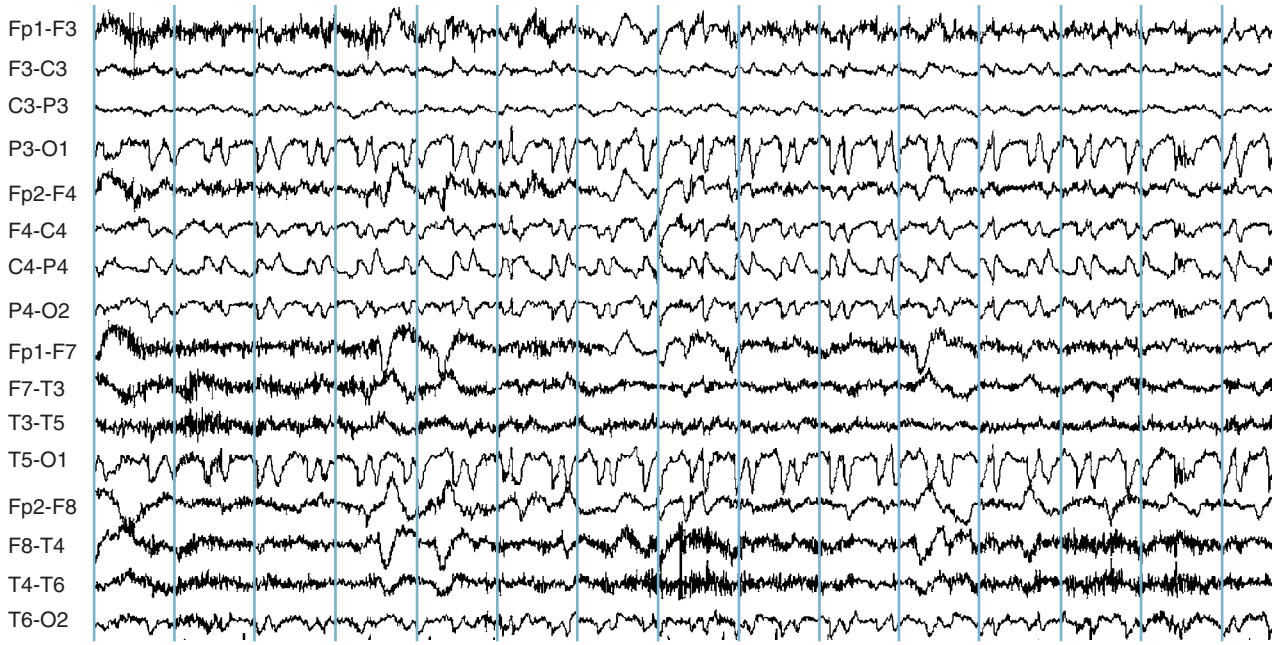


Figure 6-30 Rhythmic discharges are seen in several channels of this EEG, some of which resemble spike-wave complexes in morphology. Close analysis of the waveforms shows that the discharges do not make "topographic sense." Negativities are seen adjacent to positivities, and high-voltage deflections are seen in certain electrodes, whereas immediately adjacent electrodes are silent. These waves represent patting artifact rather than seizure activity.

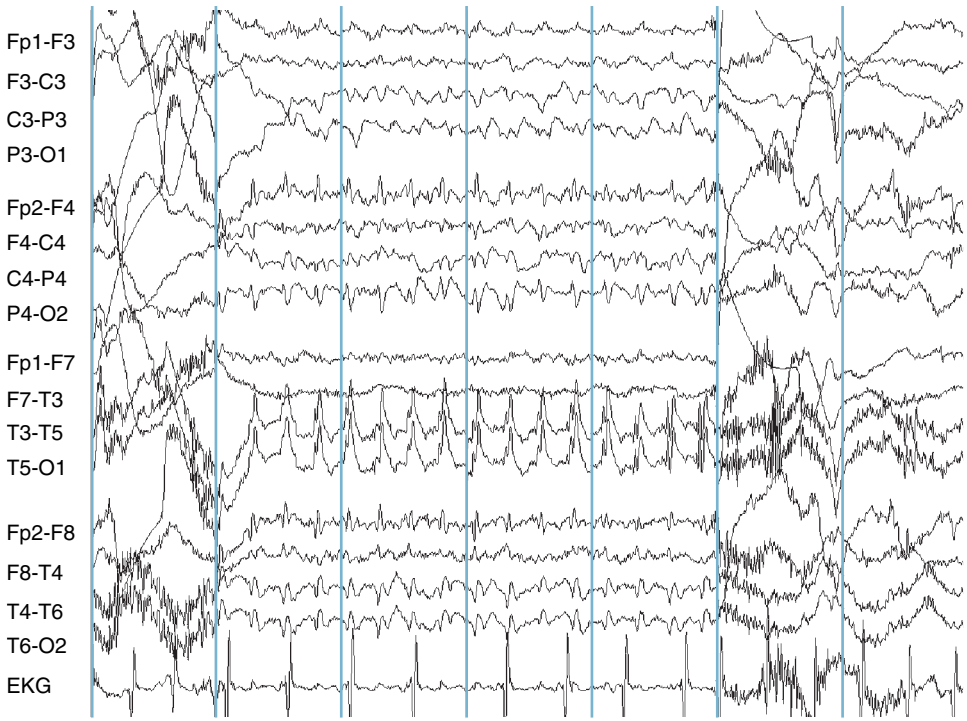


Figure 6-31 The large amount of motion artifact seen in the first second of this page is related to the patient crying. Rhythmic, high-voltage deflections that neither speed up nor slow down and do not have a plausible polarity represent patting artifact. After the patting stops, high voltage artifact in the last two seconds of the page indicate that the baby has resumed crying.

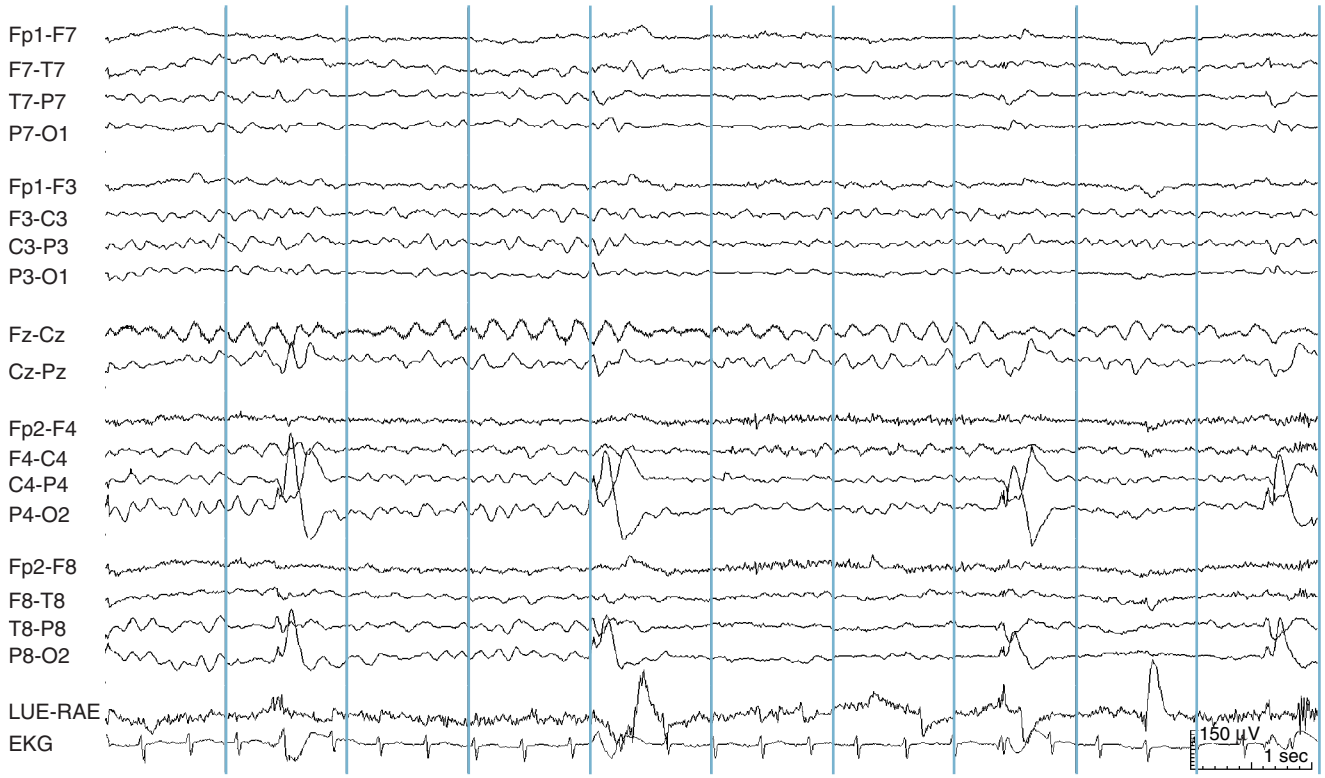


Figure 6-32 The four high-voltage deflections seen over the right posterior quadrant represent hiccup artifact. The patient is lying with her head turned to the right so that each hiccuping movement creates a relatively stereotyped pattern of motion artifact from the electrodes lying in contact with the bed.

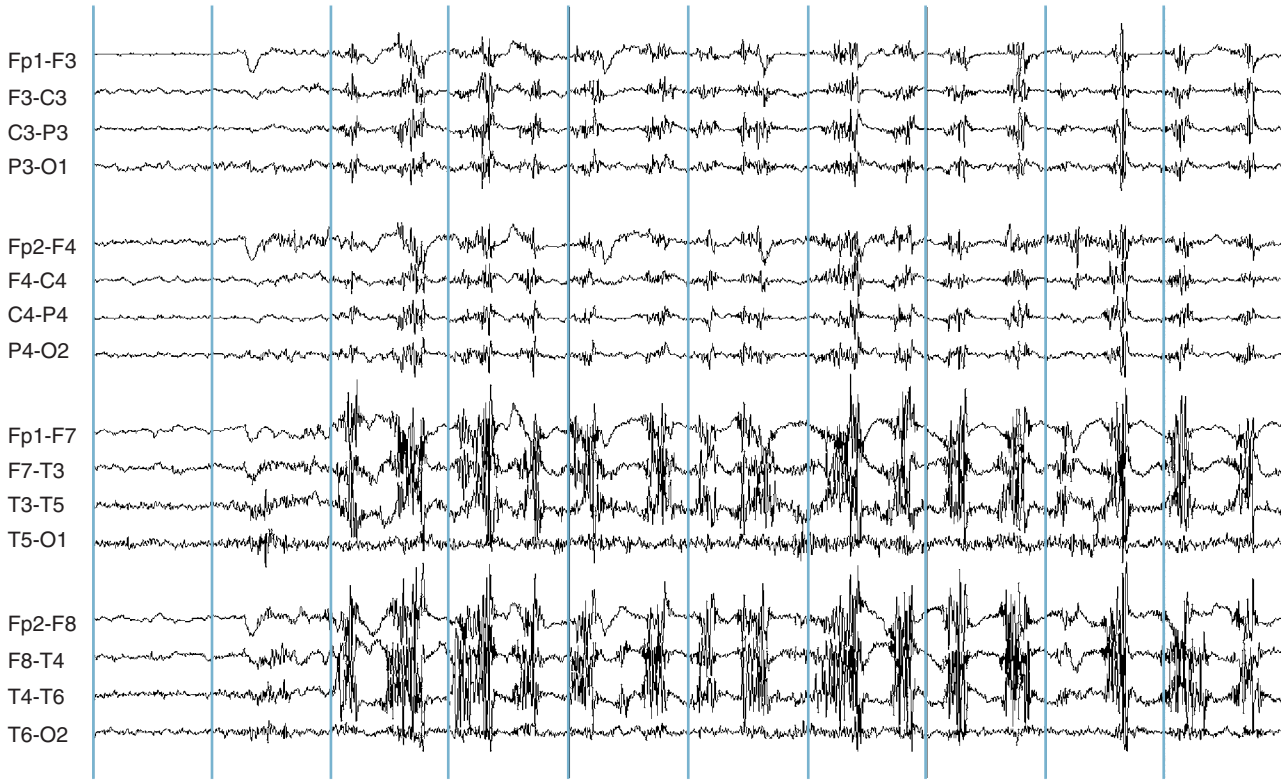


Figure 6-33 Chewing movements create rhythmic bursts of muscle artifact maximum over the temporalis muscle.

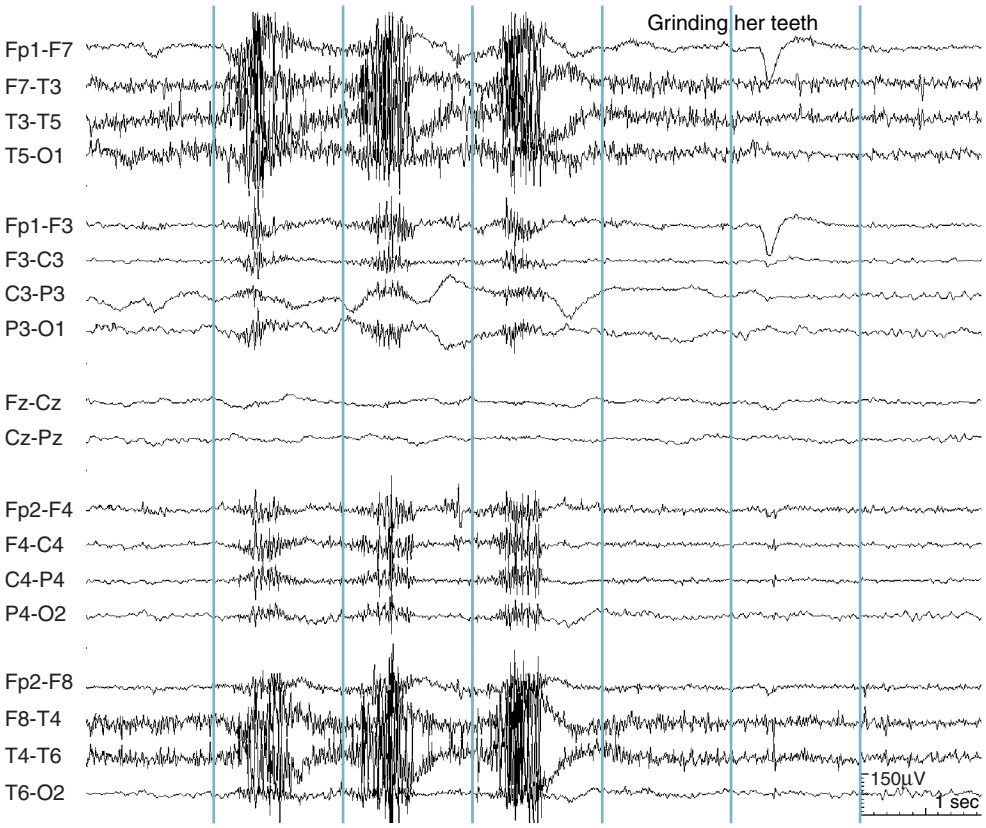


Figure 6-34 The bursts of temporal muscle artifact seen with bruxism are of slightly longer duration and have longer intervals than chewing artifact. Note that, despite the high-voltage muscle activity seen in the temporal areas, the midline electrodes (Fz, Cz, and Pz) are free of artifact because of the relative absence of scalp muscle in these areas.

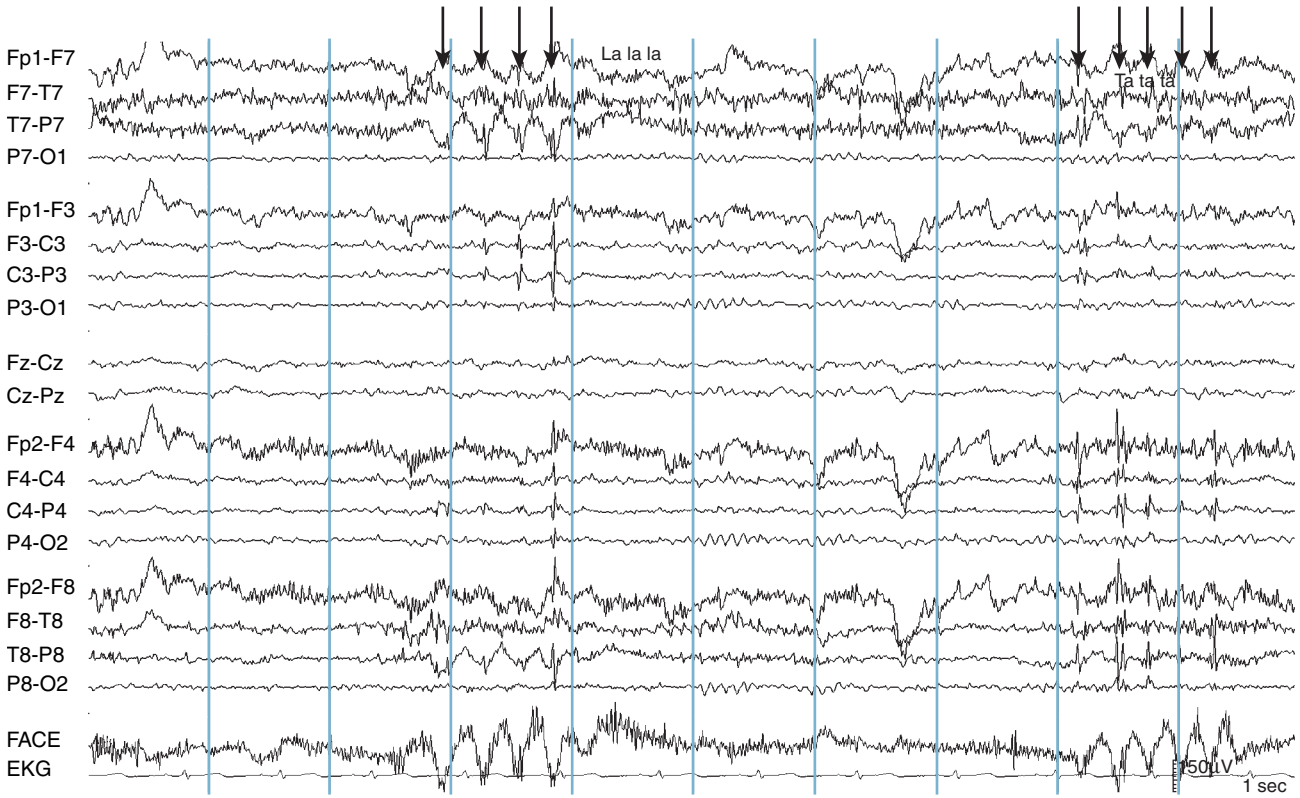


Figure 6-35 Glossokinetic artifact is caused by motion of the tongue, demonstrated in this case by asking the patient to speak repetitive syllables (arrows). The artifact may also appear spontaneously during tongue movements associated with speaking or swallowing. The glossokinetic artifact may have both slow wave and spiky components, as in this example. Characteristically, this type of artifact has a broad field across the scalp and especially over the face, as demonstrated in the second channel from the bottom labeled "FACE," derived from a special electrode pair placed on the face.

Chapter

7 Filters in the Electroencephalogram

The use of filters in recording and displaying EEG data is an indispensable tool in producing interpretable EEG tracings. Without filters, many segments of EEG would be essentially unreadable. As we shall see in this chapter, the use of filters can affect the EEG signal in ways that range from the subtle to the dramatic. The main benefit of filters is that they can appear to "clean up" the EEG tracing, making it easier to interpret and generally more pleasing to the eye. Certain filter settings can also be used to accentuate particular types of EEG activity. Filters can, however, be used improperly, and at times their use can lead to unintended consequences.

Some consider the study of how filters work an inherently dry topic. The purpose of this chapter is to provide a simple overview of how EEG filters work so that they can be used appropriately by the EEG technologist and reader. There also follows a brief discussion of simple circuit design for analog EEG filters, a topic that has traditionally been a part of electroencephalography training. The basis of some of the techniques used to filter digital EEG signals is also introduced. Although detailed knowledge of filter design is not necessary to interpret EEGs, understanding the circuitry or algorithms used to build these filters can enhance understanding of filter behavior and increase the level of sophistication of EEG reading.

Figures 7-1 and 7-2 illustrate the impact of filters on an EEG page. Figure 7-1 shows an EEG recorded during a moderate amount of patient movement, a "raw" EEG trace displayed *without* the explicit use of filters. Figure 7-2 shows the same page displayed with typical filter settings. Note that, despite the fact that muscle artifact still obliterates portions of the top four and bottom four lines of the EEG (the temporal areas), in the filtered example, the amplitude of that muscle artifact is reduced, making it easier to see adjacent channels. Indeed, in the filtered example, the presence of certain waveforms can be intermittently recognized within the areas of muscle artifact (this artifact is generated by contraction of the temporalis muscles) that otherwise would not have been detectable. Also note that the baseline of each channel is flatter, allowing for easier interpretation—each channel is more likely to stay within its own horizontal area after the filters are used.

There are also potential pitfalls in choosing filter settings. When using filters on a page of EEG that has a cluttered appearance, one might think that if a given filter setting works moderately well, then even more aggressive settings might work even better. With filters, however, the strategy of "more is better" often does not hold true because implicit to the act of filtering the EEG signal is the potential loss of information. Overzealous filter settings can overly "clean" the EEG, resulting in the filtering out and disappearance of waveforms that may be of interest to the reader. As we will see, some filter settings can change the shape of brain waves in a way that might suggest the presence of waveforms that are not really there.

THE BASIC STRATEGY BEHIND CHOOSING FILTER SETTINGS

The most ideal filter design would be one that removes all of the electrical noise or artifact from the EEG and only allows true cerebral activity to pass through. Unfortunately, no such "smart" filters exists; filters can only remove waves according to rigid mathematical rules. Luckily, there are good rationales for filtering out certain components of EEG signals using fairly simple mathematical assumptions. These assumptions are based on the idea that the brain only generates EEG waves within a certain range of frequencies and that any activity outside that range (unusually slow activity and unusually fast activity) is not likely to be of cerebral origin. Indeed, one of the general assumptions of EEG filter design is that activities well below 1 Hz and well above 35 Hz do not arise from the brain and likely represent electrical noise or artifact. Like many assumptions, this claim is mostly true but not completely true, as we shall see. On the basis of the concept that the frequency of almost all brain electrical activity of interest lies within a particular bandwidth, EEG filters are typically set up so that one filter rejects the majority of very high-frequency activity and another filter rejects the majority of very low-frequency activity. The range of frequencies between these unwanted high and low frequencies that is allowed to pass through the filter setup

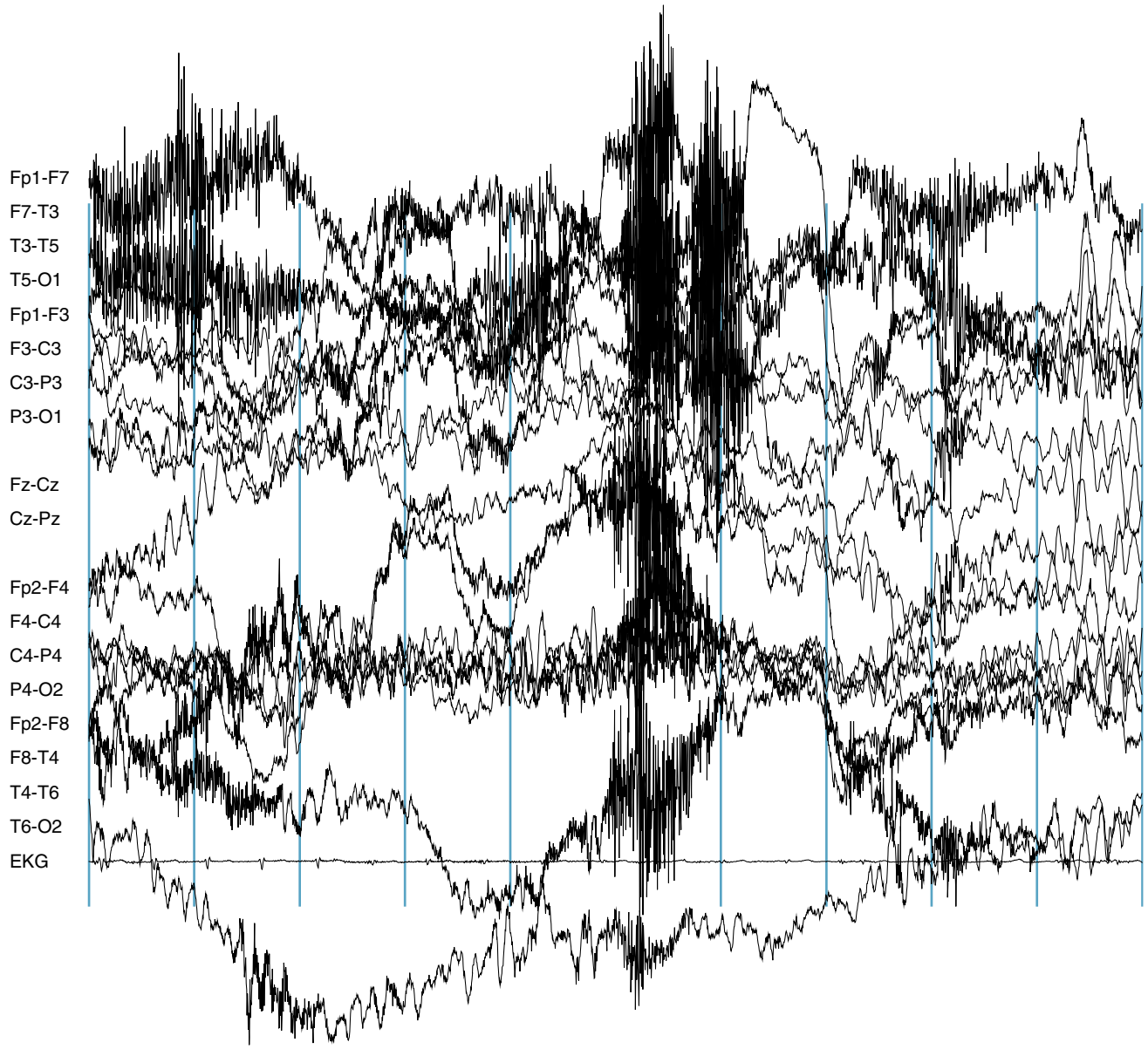


Figure 7-1 This EEG page was obtained without the explicit use of filters. Muscle artifact obliterates much of the temporal chains (the top four and bottom four EEG channels). The baselines of certain channels fluctuate so widely that they often obliterate other channels. Note that the bottom two channels even dip below the electrocardiogram channel. Compare to Figure 7-2.

is referred to as the *bandpass*. The way that different filter setups are associated with different bandpasses is illustrated here.

Rather than being used to reject spurious activity, occasionally filtering techniques can be used to bring out certain EEG activity that might otherwise have been hidden in other, higher voltage activity. In this application, the electroencephalographer may purposely attenuate the slow activity in a record (even though it represents true cerebral activity) to accentuate or "bring out" fast activity that would otherwise be lost in high-voltage slow waves. Examples of such special filtering techniques (which are not necessarily used during every EEG reading session) are illustrated in Figures 7-3 and 7-4. These figures show how aggressive use of the low-frequency filter can be used to bring out the presence of spike-wave discharges.

TYPES OF FILTERS

The three commonly used filter types in clinical EEG are low-frequency filters, high-frequency filters, and notch filters. The purpose of a low-frequency filter is to filter out low-frequency activity and to leave higher frequencies as they are. Because low-frequency filters attenuate low frequencies and allow high frequencies to "pass through," engineers often refer to low-frequency filters as *high-pass filters*. Likewise, high-frequency filters are designed to filter out high-frequency activity and

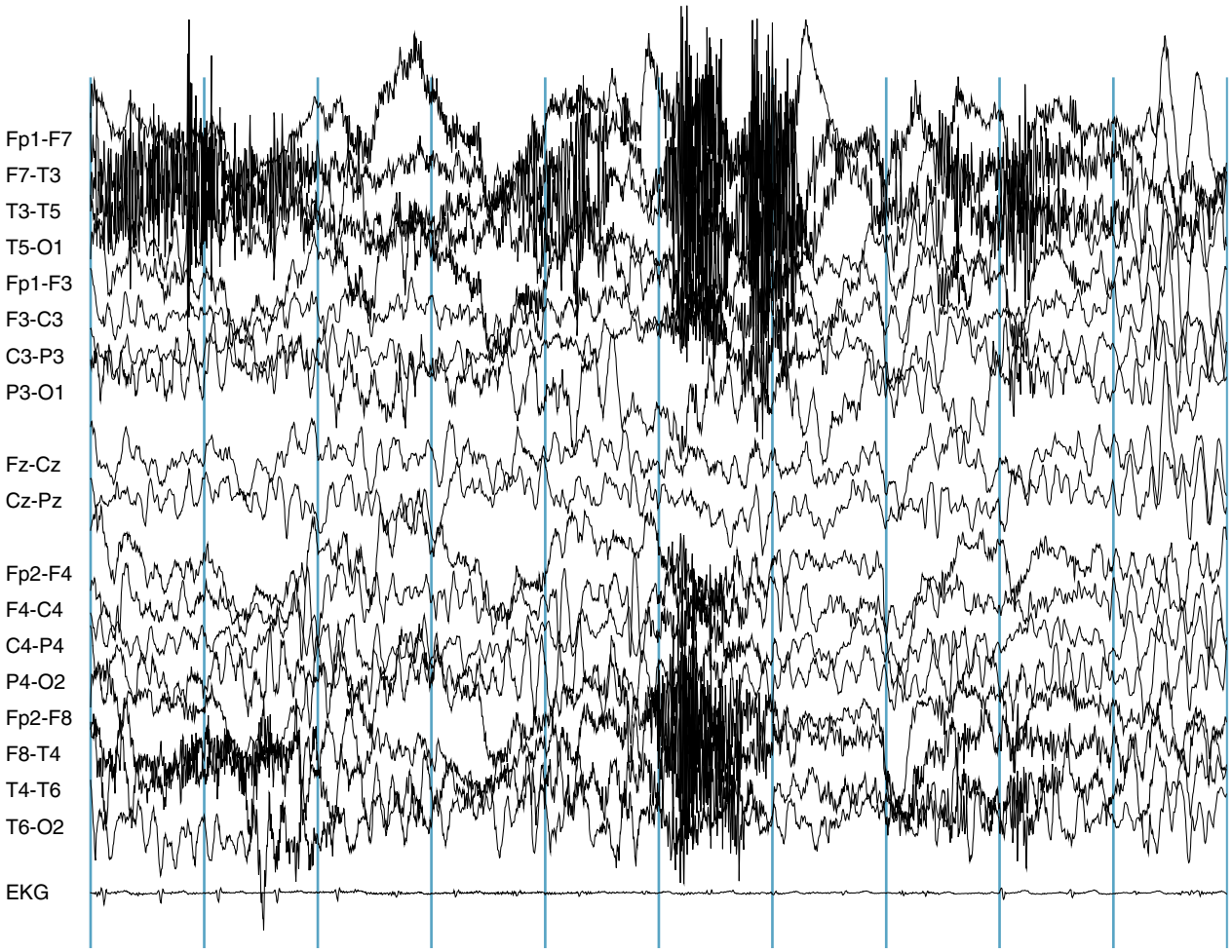


Figure 7-2 This is the same 10-second EEG page that was displayed in Figure 7-1 except that now the low-frequency filter is set at 1 Hz, and the high-frequency filter is set at 70 Hz. Note that the baselines of each channel are flatter. Also, the amplitude of the muscle artifact is significantly reduced throughout. Although there is still a large amount of artifact present on this page, the use of standard filter settings has rendered this page considerably more readable.

allow low-frequency activity to pass through and are sometimes referred to by engineers as *low-pass filters*. Although the use of the terms *high pass* and *low pass* to name filters is more common in the world of electrical engineering, these are not the preferred terms in clinical electroencephalography. In the world of clinical EEG, the alternate terms *high-frequency filter* (HFF—filters out the high frequencies) and *low-frequency filter* (LFF—filters out the low frequencies) are used, with the terms *high filter* (HF) and *low filter* (LF) sometimes used as shorthand abbreviations. Thus, HF is synonymous with HFF and LF is synonymous with LFF.

The *notch filter* is the third type of filter. Its purpose is to filter out activity at a specific frequency (rather than a frequency range). Because the alternating current in standard electric outlets in North America oscillates at 60 Hz, electric fields produced by the 60-Hz activity in the environment that surrounds us in our indoor environments frequently contaminates the EEG. Sixty-hertz notch filters (filters designed specifically to filter out 60-Hz activity) are used to attenuate or eliminate this unwanted signal. In countries where line frequencies are 50 Hz, 50-Hz notch filters are used for the same purpose.

FILTER NAMING CONVENTIONS

There are two different naming schemes for high- and low-frequency filters. A filter can be named after a frequency (e.g., a "5-Hz low filter") or after its time constant (e.g., a "low-filter with time constant of 0.1 seconds"). When a filter is named after a particular frequency, this is referred to as the *nominal frequency* or the *cutoff frequency* of the filter. Whether the filters on a particular EEG machine are named according to a cutoff frequency or a time constant is the decision of the manufacturer. Because referring to filters by their cutoff frequencies is becoming more common, and also because cutoff frequencies are easier to understand, we discuss the relationship between a filter's electrical characteristics and its cutoff frequency first.

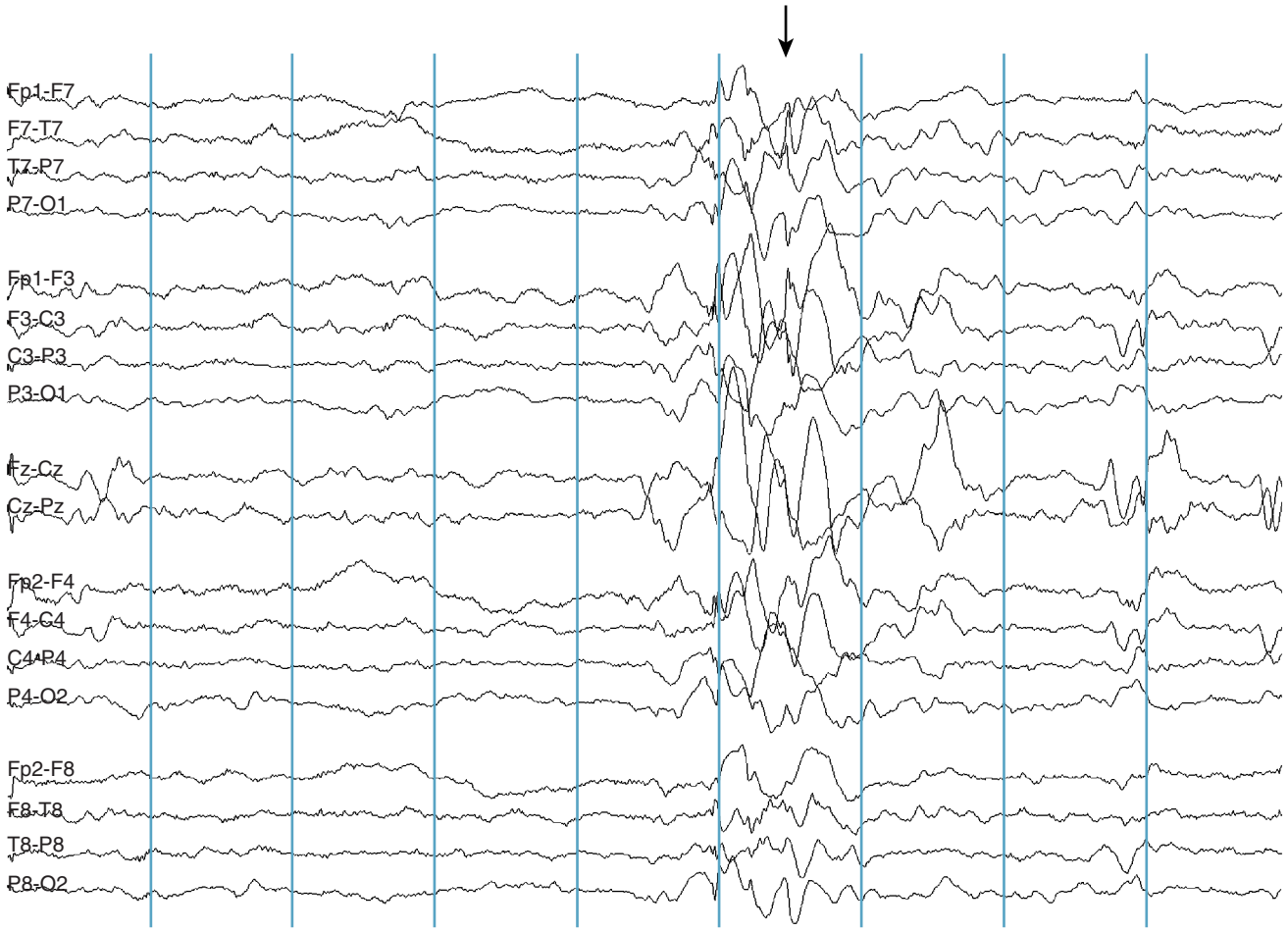


Figure 7-3 This recording is made during a transition from Stage I to Stage II sleep using the standard low filter setting of 1 Hz. A burst of high-voltage slowing is seen during the sixth second (arrow). Inspection of the burst suggests the possibility that some spike activity may be intermixed, but a definite determination as to whether spike activity is truly present is difficult. Compare to same EEG signals displayed with different filter settings in Figure 7-4.

Low-Frequency Filters

The term *cutoff frequency* conjures up the image of an all-or-nothing effect at the frequency named. For example, a class may have a particular cutoff grade for passing or failing—one point below the cutoff grade, and the student does not go on. An amusement park may have a particular height cutoff to go on certain rides—all individuals below that height are excluded from the ride. The behavior of low-frequency filters in terms of their cutoff frequencies is not at all so absolute as the behavior of classroom teachers or amusement park officials. In fact, it may be surprisins to learn how little a filter affects activity at its cutoff frequency. When a low-frequency filter encounters a sine wave that happens to be exactly at its cutoff frequency, it cuts down the amplitude of that wave by approximately 30%. Sine waves at frequencies somewhat below that frequency are reduced by somewhat more than 30%—the farther the wave's frequency is below the filter's nominal frequency, the more it is attenuated. Perhaps more surprising, sine waves at frequencies somewhat above the cutoff frequency are also reduced in size by the filter, although by somewhat less than 30%. Again, the more the sine wave's frequency exceeds the low-frequency filter's nominal frequency, the less it is affected by the filter.

Visual Effects of Low-Frequency Filters

When standard low frequency filter settings such as a cutoff frequency of 1 Hz or below are used for the low-frequency filter, the main effect is to help keep each EEG channel within its own horizontal area, eliminating large drifts upward or downward into the space of other channels. This is because this baseline drifting actually represents a very low frequency wave. More aggressive use of the low-frequency filter (higher cutoff frequencies such as 3 Hz or 5 Hz) initially begins to attenuate delta frequencies and, when even higher cutoff frequencies are used, may almost completely eliminate some slow activity, sometimes with the advantage of bringing out other features in the EEG (see Figures 7-5, 7-6, 7-7, 7-8, and 7-9). Examples of how different low-frequency filters might affect a simple trace of the posterior rhythm are shown in Figure 7-10. Note that when successively more restrictive settings are used for the low filter, the baselines of each channel become straighter, but faster activity is relatively preserved.

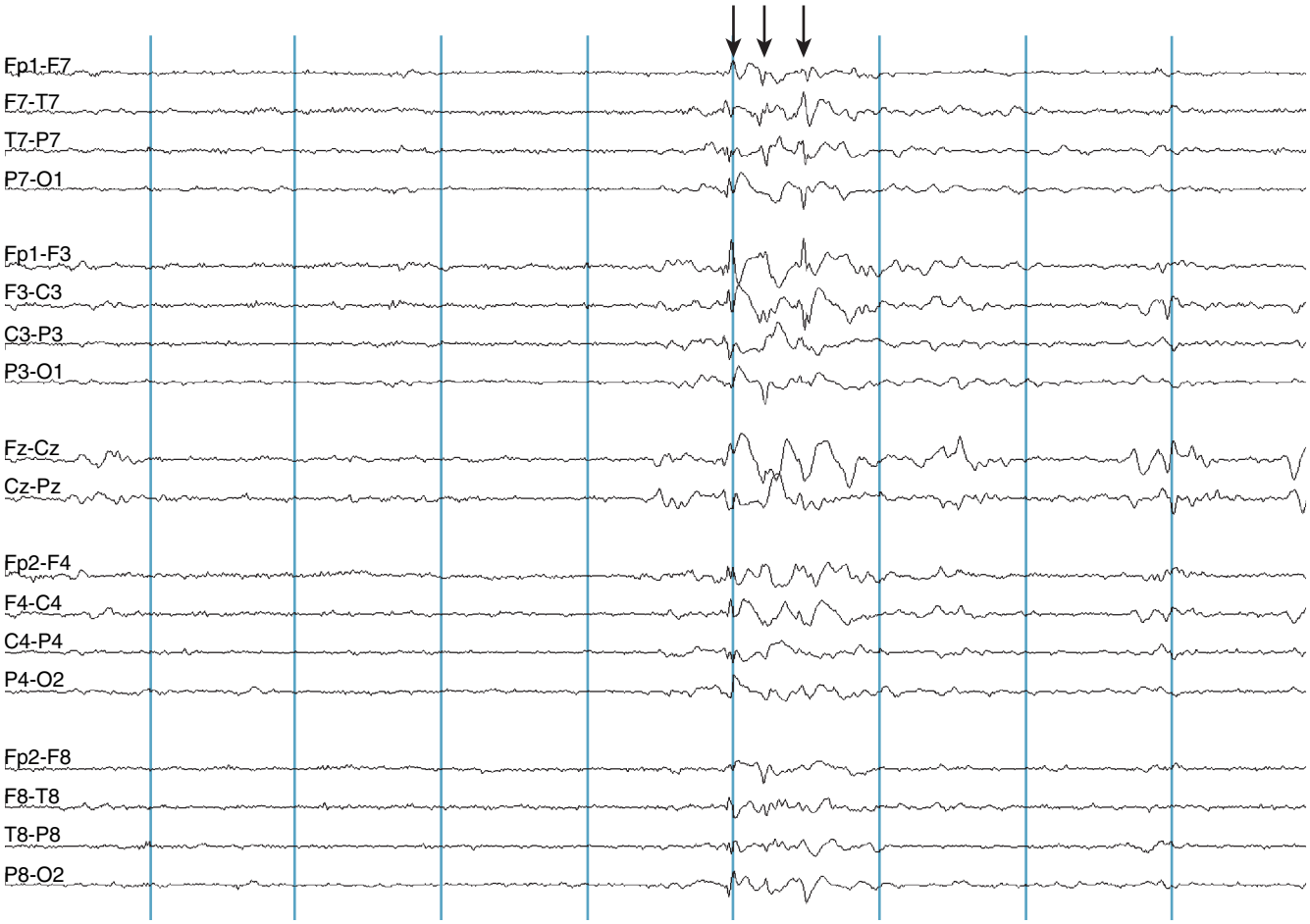


Figure 7-4 The same EEG page as shown in Figure 7-3, but this time with an aggressive low-filter setting of 10 Hz. Note that the bulk of the slow activity is suppressed, and because the slow activity is less prominent, faster activity—in particular, the spikes—is accentuated. With this filter setting, it is clear that there are three repetitive spikes with a broad field (below arrows) mixed into this slow-wave burst. If there was a question as to whether the initial slow-wave burst represented some type of motion artifact, the presence of these three embedded, rhythmically repetitive spikes with organized topography increases the likelihood that this represents an example of a diffuse, repetitive spike-wave discharge rather than motion artifact.

Roll-off Characteristics

The graph in Figure 7-11 illustrates how a 5-Hz LF would handle sine waves of varying frequencies. The curve describes what portion of a pure sine wave (y axis) at a given frequency (x axis) would be allowed to pass through the filter. Considering the example of this 5-Hz low-frequency filter in more detail, the curve shows that a 5-Hz sine wave presented to this filter will lose 30% of its amplitude after passing through the filter. (Why the amount of reduction at the cutoff frequency is specifically 30% is explained later.) If the original 5-Hz wave presented to the filter has an amplitude of 100 μV (the input wave), then the filter's output wave would only have an amplitude of 70 μV. What does the 5-Hz LF do with waves just above and just below 5 Hz? The roll-off curve for this filter shown in this figure indicates that a 4-Hz curve would be attenuated by 33%, but a 6-Hz sine wave would only be attenuated by 26%. The type of curve shown in Figure 7-11 that shows how a given filter processes pure sine waves of different frequencies is called the *roll-off characteristic* of the filter. Figure 7-12 shows how a 5-Hz LFF handles input waves of 10 Hz, 5 Hz, 2 Hz, and 0.5 Hz of the same amplitude, attenuating the lowest frequency waves dramatically but only causing a mild reduction in the amplitude of the 10-Hz wave. The exact amount of reduction at each frequency is given by the roll-off characteristic shown in the previous figure. Figure 7-13 shows the roll-off characteristics of 0.1-, 1-, 5-, and 10-Hz low filters.

Of course, when filters are applied to real EEG signals the waves presented to the filter consist of mixed frequencies. The filter attenuates each frequency component of the waves according to the rule of the roll-off characteristic even in wave mixtures. Low-frequency filters are especially useful for filtering out certain artifacts caused by patient motion that might shift a channel's baseline or other sources of low-frequency noise.

High-Frequency Filters

High-frequency filters also have a roll-off characteristic, but, logically, the curve rolls off in the direction opposite to the roll-off curves for LFFs, falling off toward the right (the direction of the higher frequencies). Just as

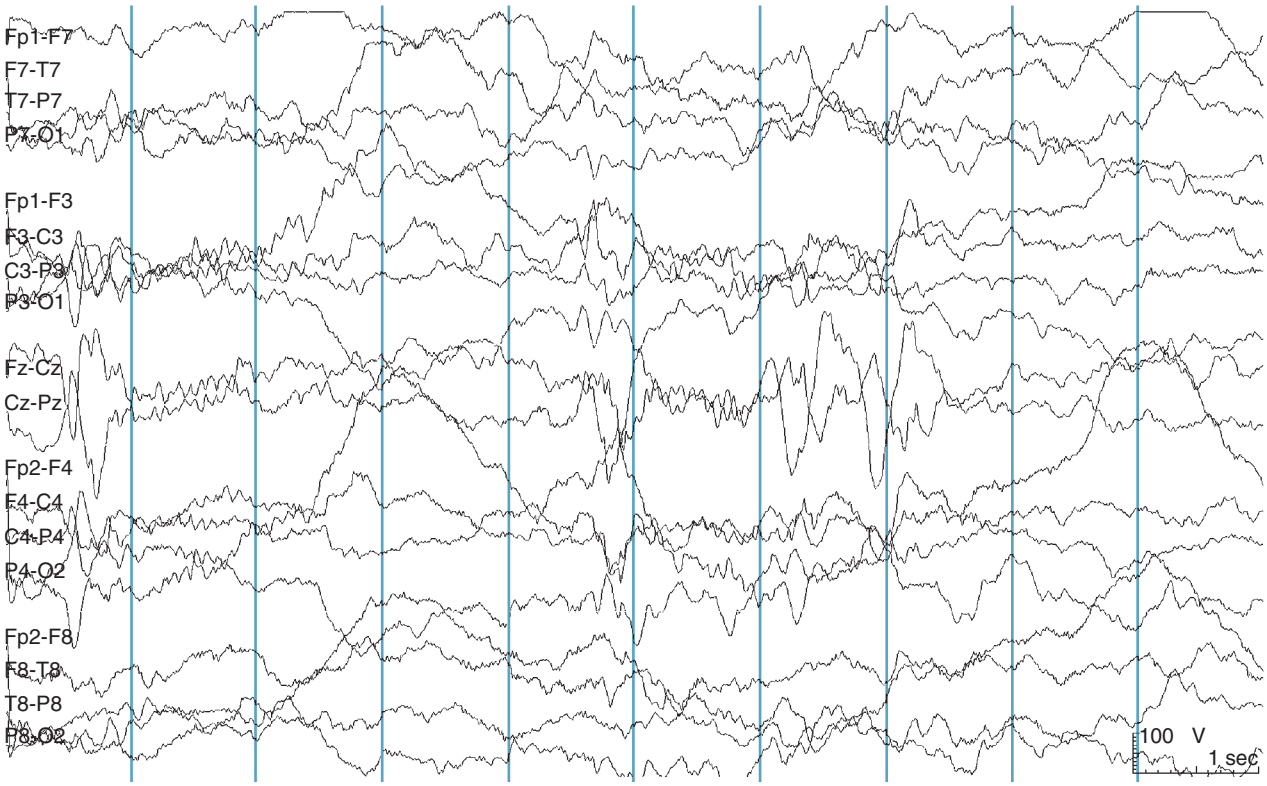


Figure 7-5 An example of Stage II sleep recorded with a conservative low-frequency filter setting of 0.1 Hz. Note that the baselines of several channels wander into the areas of adjacent channels making the tracing more difficult to read. Some of the largest and broadest slow-wave deflections may represent artifact rather than true electrocerebral activity.

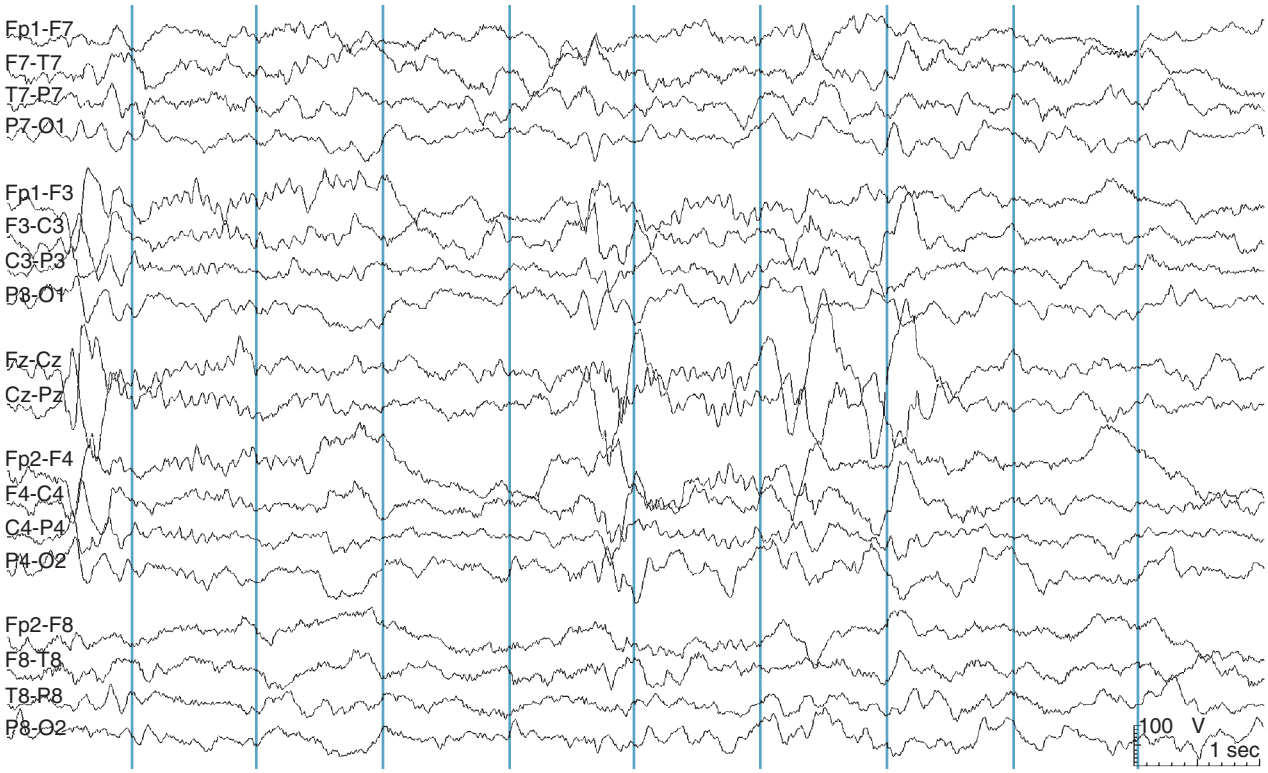


Figure 7-6 The same page of Stage II sleep from the previous example is shown, now displayed with the standard LFF setting of 1 Hz. The wide deflections in the baselines seen in Figure 7-5 of each channel are no longer present making the page more readable.

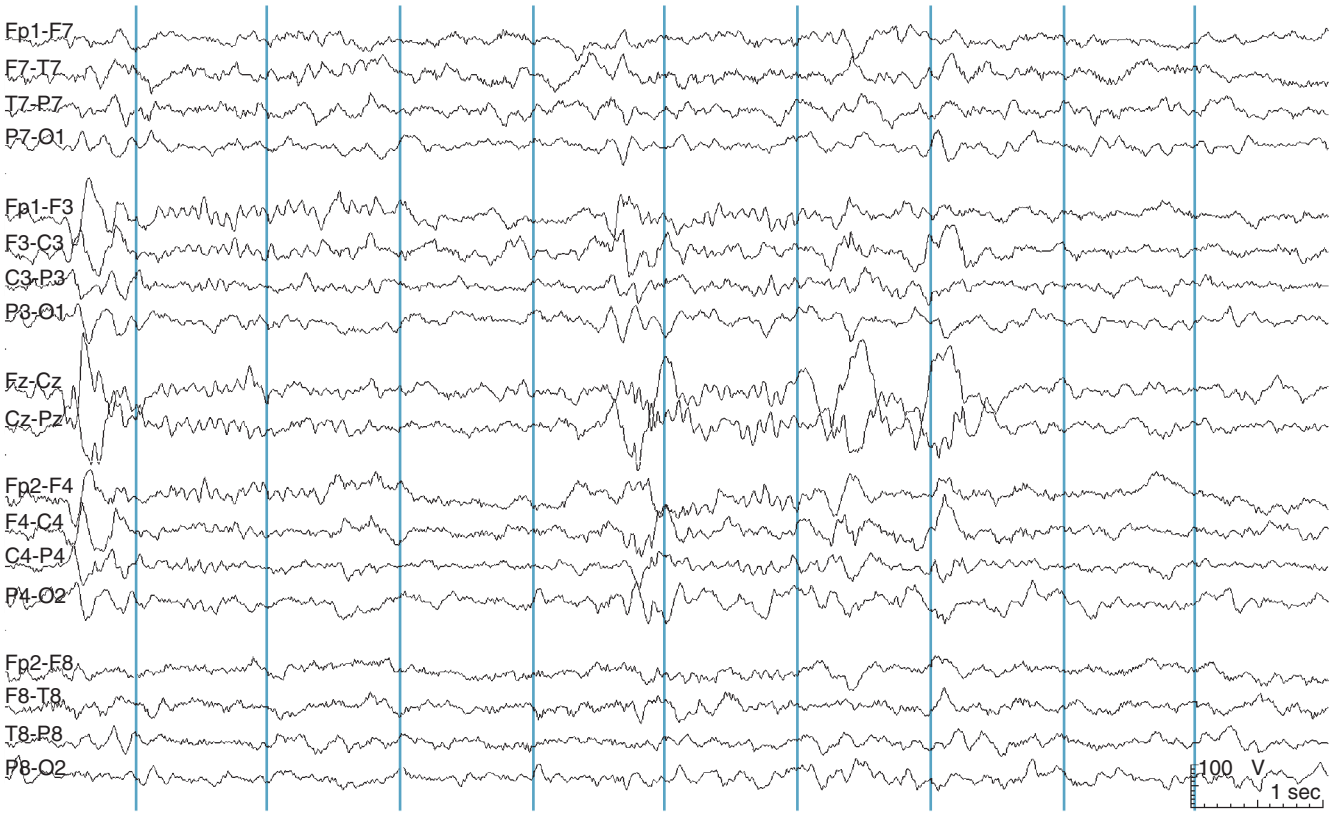


Figure 7-7 The same page of Stage II sleep from the previous examples is shown, now with a LFF setting of 3 Hz. The amount of slow activity detail is further decreased, but fast activity, such as the spindles and vertex waves, is still easily seen.

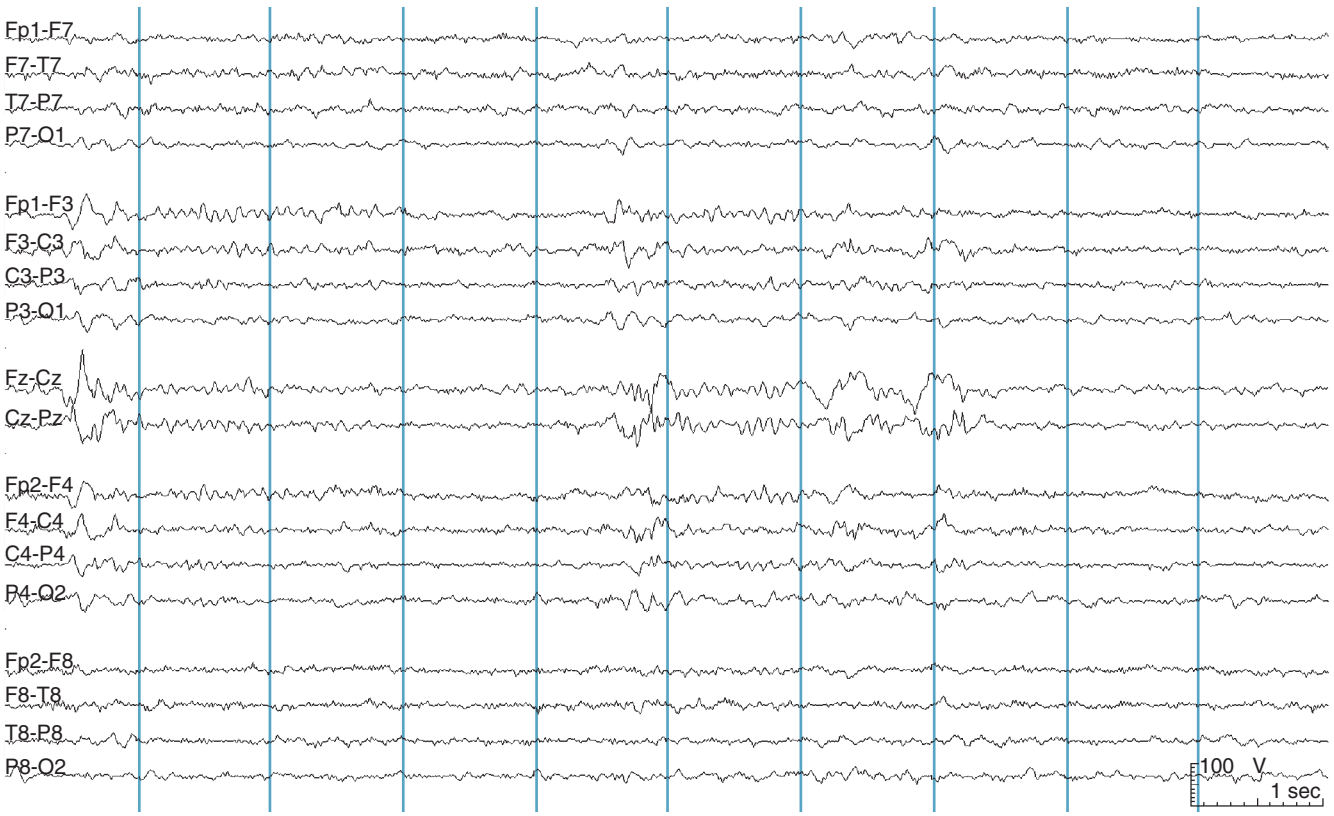


Figure 7-8 The aggressive LFF setting of 10 Hz used to display this same page of Stage II sleep is only occasionally used in routine EEG interpretation. Note that, now, most of the slow-wave activity is no longer evident. Faster events such as the spindles and vertex waves can still be appreciated.

was the case for LFFs, an HFF attenuates a sine wave at its nominal frequency by 30%; higher frequency waves are attenuated by even more than 30%, and lower frequency waves are attenuated by less than 30%. Figure 7-14 shows examples of roll-off characteristics for theoretical 70-, 35-, and 15-Hz HFFs. High-frequency filters are especially useful for filtering out muscle artifact and other high-frequency noise. Overly aggressive use of HFFs, however, will not only attenuate the height of high-frequency waves but can also change wave shape giving misleading results, as discussed later in the discussion of time constants.

WAVE ATTENUATION AND DECIBELS

The reason that filters are named after the wave frequency at which they attenuate the amplitude by 30% deserves some explanation. The amount that a filter reduces a given wave can also be stated in decibels. A *bel* is defined as the common logarithm of the ratio of the *powers* of two waves and a decibel is defined as 10 times that number:

$$dB = 10 \cdot \log \left(\frac{p_1}{p_2} \right)$$

where p_1 and p_2 are the powers of the two waves being compared.

Note, therefore, that the decibel unit is not an absolute measurement, but rather a comparison—a ratio or percent change. (Even when the decibel unit is used to describe the intensity of sound, it always represents a comparison to a defined, standard sound level.) Because the decibel unit is used to describe the change in *power* rather than the change in *voltage* (amplitude), but voltage measurements rather than power measurements are routinely used in EEG, the relationship between power and wave amplitude must be considered. In fact, the power of a wave varies as the square of its amplitude. For that reason, if amplitudes are used in the previous formula, their values must be squared to preserve the relationship:

$$dB = 10 \cdot \log \left(\frac{a_1^2}{a_2^2} \right)$$

where a_1 and a_2 are the amplitudes of the two waves being compared.

This equation can be rewritten taking the squared terms out of the parentheses:

$$dB = 20 \cdot \log \left(\frac{a_1}{a_2} \right)$$

The nominal or cutoff frequency of a filter is defined by the frequency at which the *power* is attenuated by

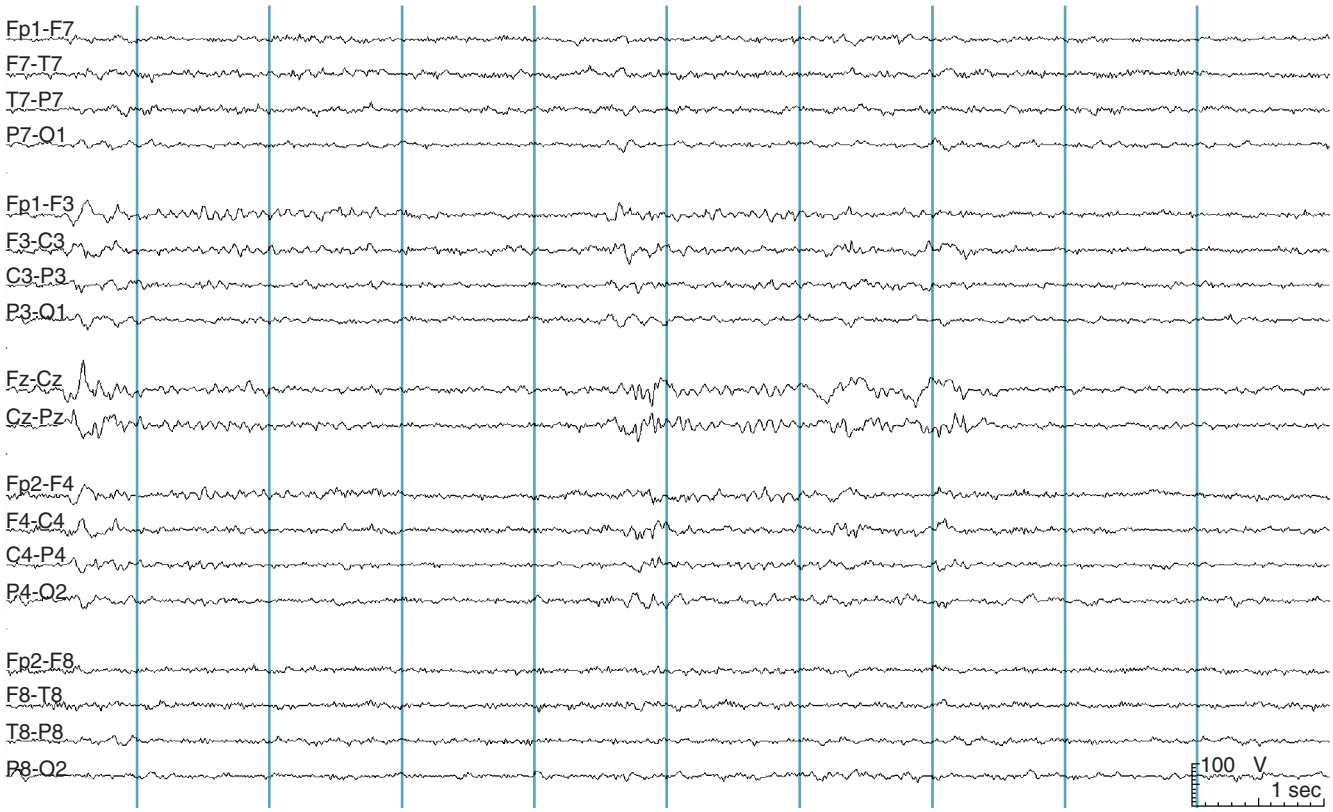


Figure 7-9 The LFF setting of 15 Hz used to display this same sample of Stage II sleep was chosen for illustrative purposes and is rarely used during routine EEG interpretation. This aggressive low-filter setting now begins to attenuate even the spindle activity. This setting eliminates a lot of important detail from the EEG tracing and is therefore seldom used.

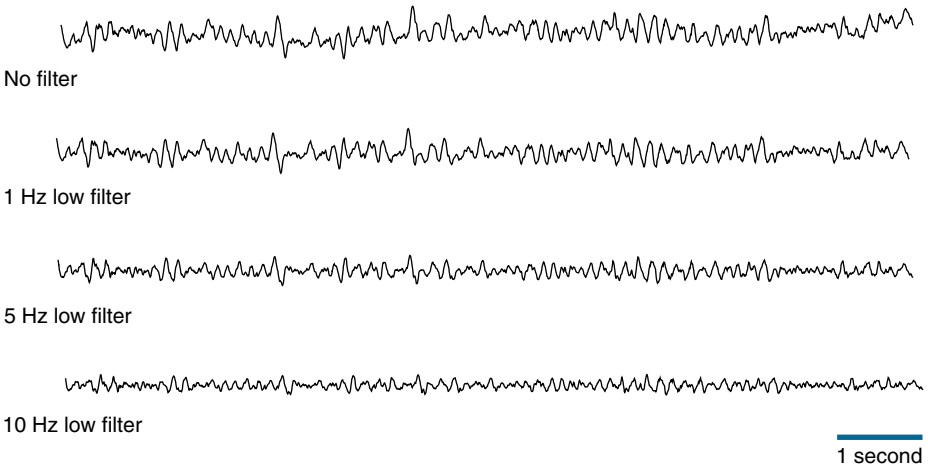


Figure 7-10 This figure illustrates the effects of the low filter on a sample of the posterior rhythm. The same segment of posterior rhythm is displayed with different low-frequency filter cutoff frequencies. Although an adult posterior rhythm generally does not represent an example of slow activity, some amount of slow activity is often intermixed with the posterior rhythm to a greater or lesser extent. Note that as the cutoff frequency of the low filter is steadily increased, the "waviness" of the baseline flattens, especially as the 5-Hz filter is used. After the cutoff frequency rises into the zone of the posterior rhythm itself, such as when the 10-Hz low filter is used, the filter begins to attenuate the waves of interest significantly (compare the amplitude of the posterior rhythm waves in the bottom line with those in the top line).

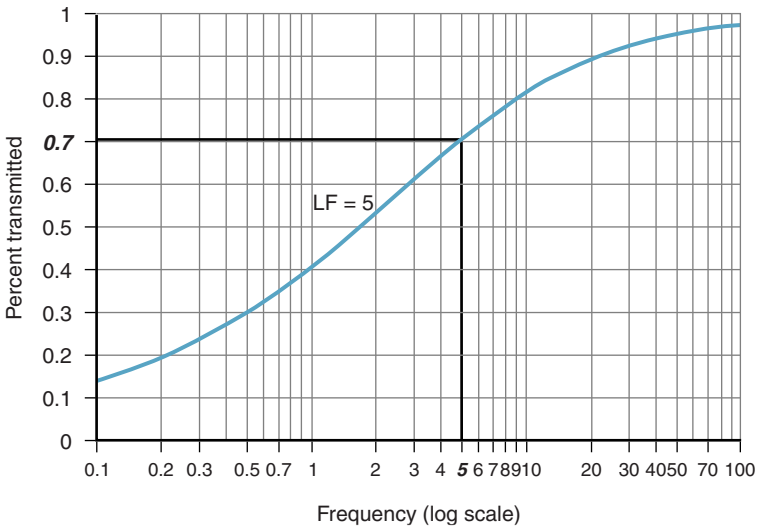


Figure 7-11 This curve describes the roll-off characteristic of a theoretical 5-Hz low-frequency filter. The graph shows the percentage of a pure sine wave that is allowed to pass through the filter (y axis) as a function of its frequency in hertz (x axis, plotted on a logarithmic scale). Note that waves at frequencies well below 5 Hz are attenuated substantially but waves at frequencies above 5 Hz are also attenuated to some extent, although the amount may be relatively minor. The curve shows that a sine wave of exactly 5 Hz is attenuated by approximately 30% (heavier bars). For an explanation of why wave amplitude is attenuated specifically by 30% at the filter's nominal frequency, see discussion in text.

50%. This frequency at which the power is reduced by 50% is also called the "3-dB point" of a filter because the formula tells us that a 50% reduction in power is equal to approximately 3 dB: when p_1 is twice p_2, 10 log (2) ≅ 3.01. What ratio of amplitudes corresponds to a 2:1 ration of powers? Because the amplitudes must be squared to get the power, the corresponding pair of amplitudes that would also manifest a 3-dB change would represent approximately a 70% reduction in wave height, such as from 10 to 7:

$$10 \log (10^2/7^2) = 10 \log (100/49) \cong 3 \text{ dB}$$

Because a reduction in amplitude of 30% corresponds to a 50% reduction in total power, filter cutoff frequencies are named after the point at which the filter decreases the power by 50% and, therefore, the amplitude by 30%.

The steepness of the roll-off characteristic of a filter is sometimes described in the units of *decibels per octave*. The term *octave* is best known in the music world, describing the difference between two notes such as "middle C" and "high C." In both the world of music and the world of electrical signals, an octave represents a doubling or a halving of a wave frequency. Therefore, 2 Hz is one octave below 4 Hz (and "middle C" on the piano represents a tone at 256 Hz, whereas the note one octave above, "high C," is the tone at 512 Hz). Simple low-filter frequency-response curves "roll off" at a maximum of 6 dB per octave, although this roll-off rate is not constant across the entire frequency band.

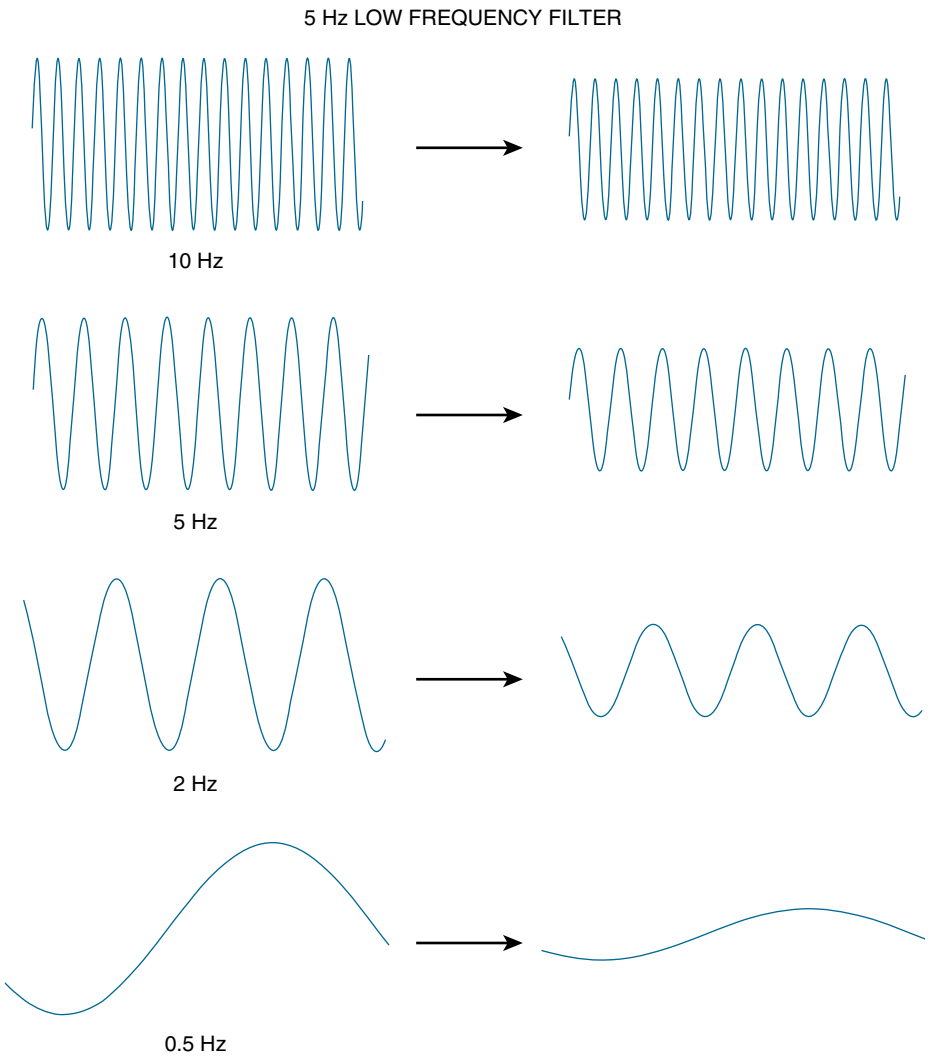


Figure 7-12 A 5-Hz low-frequency filter (LFF) attenuates waves of different frequencies to different degrees. Sine waves of the same amplitude but of different frequencies are shown on the left. The output wave after having been passed through a 5-Hz filter is shown on the right. Note that the 10-Hz sine wave shown at the top, even though above the filter's nominal frequency, is transmitted by the filter with a slightly decreased amplitude. The 5-Hz sine wave's amplitude is attenuated 30% by a 5-Hz LFF, as expected. Amplitudes of 2- and 0.5-Hz waves are reduced more substantially.

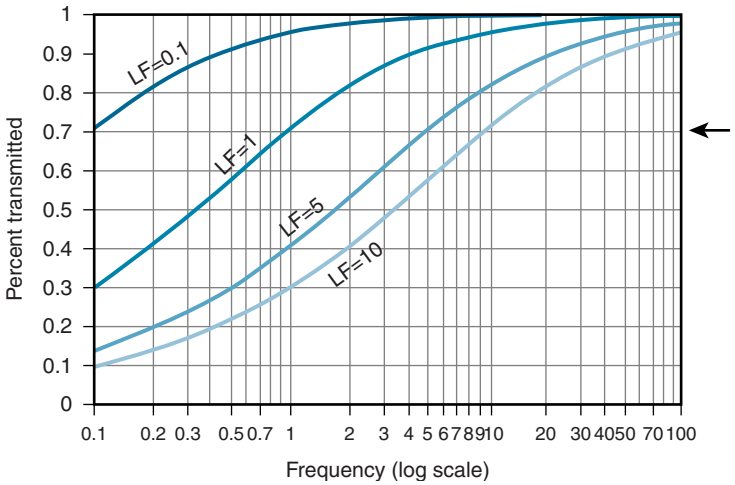


Figure 7-13 The roll-off characteristics of 0.1-, 1-, 5-, and 10-Hz low filters are plotted on a semilog plot. Note that a low filter setting of 0.1-Hz barely has an effect on the frequency range most commonly seen in conventional EEG recording—1 to 30 Hz. The 10-Hz low filter, however, has a major impact on frequencies in this range. Note also that the graph line representing 70% transmission passes through each roll-off curve at each filter's nominal frequency (arrow).

BANDPASS FILTERS

As mentioned earlier, an HFF and an LFF are typically combined to create a specific bandpass. Typical filter settings with which many routine EEGs are initially recorded include a pairing of an LFF set at 1 Hz and an HFF set at 70 Hz. Taking into account the fact that the roll-off characteristics for these filters are relatively gradual (rather than creating a "brick wall" where no activity below 1 Hz or above 70 Hz can pass), it is no surprise that a fair amount of EEG activity with frequencies above and below the nominal frequencies of 1 Hz and 70 Hz still may appear in the recording. Figure 7-15 shows two possible bandpass setups, one with LFF = 1 Hz and HFF = 70 Hz, and the other with LFF = 5 Hz and HFF = 70 Hz. Figure 7-16 illustrates the same bandpass setup, but plotting the curves with the frequency on a linear scale for comparison. Figure 7-17 illustrates the bandpass curve that uses a more aggressive HFF setting of 35 Hz. Note that, comparing the Figures 7-15 and 7-17, the shapes of the curves are similar, and the portions of the curves on the left (representing the low frequencies) are nearly identical. The portions of the curves on the right side (representing the higher frequencies) show more attenuation when the HFF is set to 35 Hz rather than 70 Hz.

50-HZ AND 60-HZ NOTCH FILTERS

Unlike HFFs and LFFs, which have a gradual roll-off curve, the purpose of a notch filter is to exclude a single frequency from the EEG signal. These notch filters are most useful when the field of AC current from the electrical wiring and outlets that surround the patient contaminates the record. The ideal notch filter's transmission curve shows a flat response for all frequencies, except for the nominal frequency of the filter, where there is a notch in the curve denoting near complete attenuation of any waves at that particular frequency (see Figure 7-18). Of course, in the world of working EEG, notch filters may not operate as perfectly as this description implies, but they typically do a good job of suppressing unwanted AC line voltage artifact. Figure 7-19 shows a sample of EEG with 60-Hz contamination in multiple channels. Figure 7-20 shows detail of

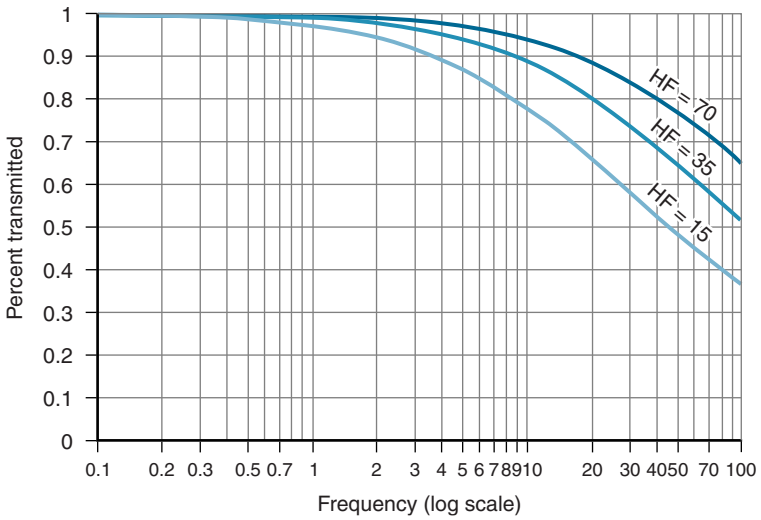


Figure 7-14 Roll-off characteristics for theoretical high-frequency filters (HFFs) with cutoff frequencies of 70 Hz, 35 Hz, and 15 Hz. Note that, as was seen with the low-frequency filters, each roll-off curve passes through the point of 70% transmission at its respective cutoff frequency. Higher frequencies are attenuated more dramatically, and frequencies lower than the cutoff frequency are attenuated by less than 30%. In this example, the 70-Hz HFF allows 90% or more of frequencies below 20 Hz to be passed through the filter.

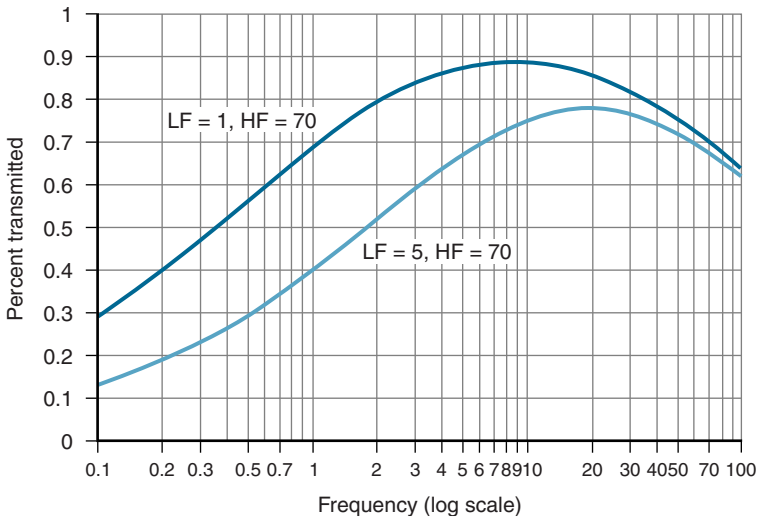


Figure 7-15 When a high filter and a low filter are combined, a particular middle range of frequencies is favored and higher and lower frequencies outside of that range are relatively excluded. This type of setup is called a bandpass. Two curves showing the bandpass characteristics generated by combining a 1-Hz low-frequency filter (LFF) and 70-Hz high-frequency filter (HFF; top curve), and a 5-Hz LFF and 70 Hz HFF (bottom curve) are shown. Because the difference between the two bandpasses illustrated is caused by a change in the LFF used, the left sides of the curves that describe the filters' effect on low frequencies differs, whereas the right side (high-frequency) portion of the curves is similar. Note that both bandpasses (especially the 1-Hz LFF/70-Hz HFF example) generally favor the frequency range of greatest interest in clinical EEG, the frequencies between 1 and 30 Hz.

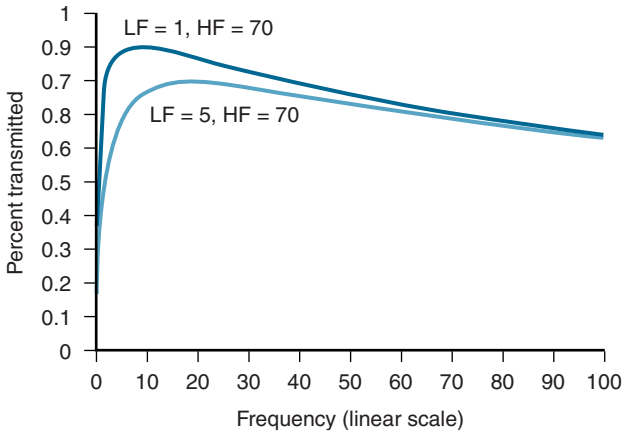


Figure 7-16 For the sake of comparison, the same two bandpass curves as are shown in Figure 7-14 are plotted on a linear frequency scale.

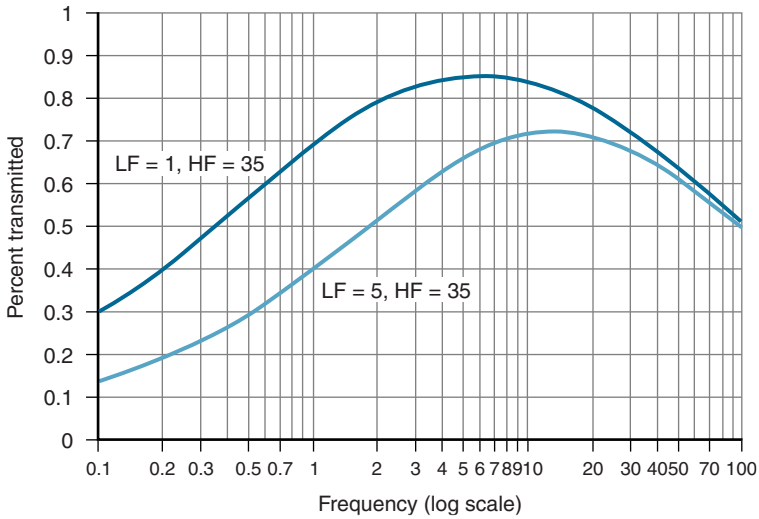


Figure 7-17 These curves illustrate the bandpasses resulting from pairing a 1-Hz LFF with a 35-Hz HFF and the pairing of a 5-Hz LFF with a 35-Hz HFF, respectively. Frequency is plotted on a logarithmic scale. Note that the right side of the curve falls off more steeply compared with the curves shown in Figure 7-14 because of the more aggressive (lower) HFF cutoff frequency used.

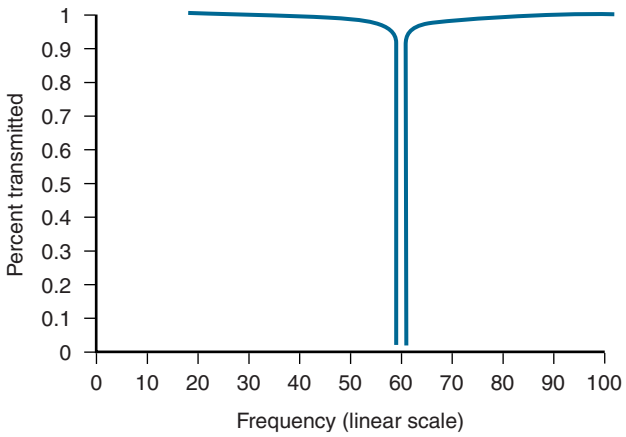


Figure 7-18 Illustration of an idealized roll-off characteristic of a 60-Hz notch filter. The goal of the notch filter is to allow all frequencies to pass except for activity exactly at the nominal frequency, in this case 60 Hz, which would be rejected completely.

the 60-Hz artifact. The reader should become adept at identifying the 60-Hz sine wave that comprises the fine structure of this type of artifact. Figure 7-21 shows the same page of EEG with the 60-Hz notch filter "in" (applied).

Some of the imperfections of notch filters can be overlooked, such as the fact that a 60-Hz notch filter will also attenuate some adjacent frequencies, such as 59 Hz and 61 Hz. The good news is that the electroencephalographer is rarely interested in these nearby frequencies because they are in a range much faster than the cerebral rhythms that are of interest during routine analysis; this type of shortcoming is rarely noticed. Also, a notch filter may have difficulty suppressing particularly large amounts of 60-Hz activity. The 60-Hz notch filter is useful in countries in North America and other locations where AC electricity is supplied at 60 Hz. The 50-Hz notch filter is used

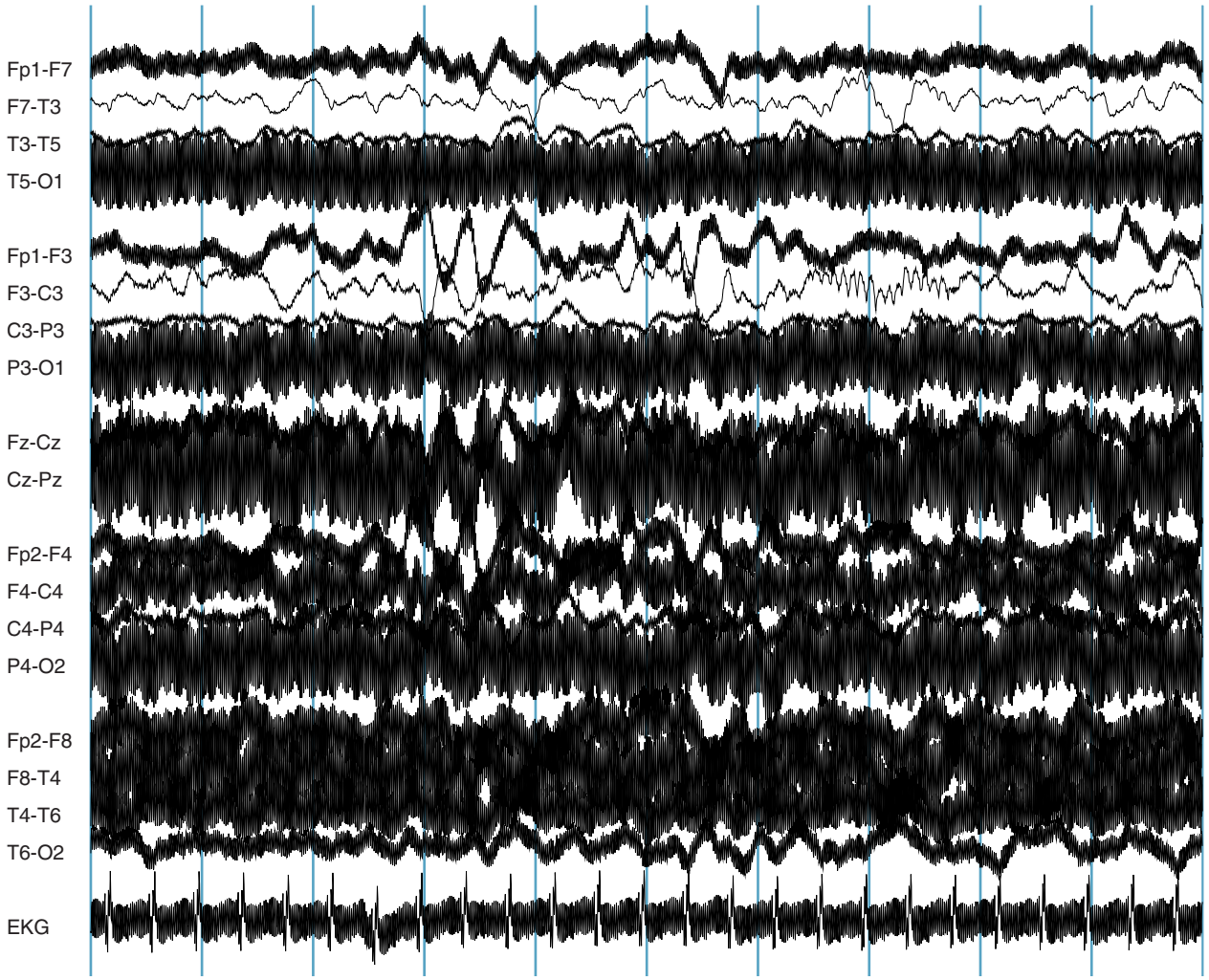


Figure 7-19 Multiple channels in this tracing are obscured by 60-Hz artifact. In general, 60-Hz artifact can be distinguished from muscle artifact by its highly regular, sinusoidal appearance. Close examination of the waves sometimes allows a 60-Hz wave to be discerned, which in this example has the appearance of vertical ribbing. Also, notice that the amplitude of the artifact tends to stay steady in each channel. Compare to Figure 7-20.

in the majority of countries where the line frequency is 50 Hz (in North America, the 50-Hz notch filter would serve no useful purpose).

ELECTRODE IMPEDANCE PROBLEMS

EEG technologists must perfect the art of applying EEG electrodes so that they can make an accurate recording of brain wave activity through the scalp. The procedure involves measuring the correct electrode location accurately followed by preparation of the skin, often by rubbing with an abrasive compound. Electrolyte solution or paste may be applied under the electrode to facilitate recording of electrical currents. After application but before the study begins, electrode impedance is measured for each electrode to assess how well the electrodes have been applied and to identify problem electrodes. Impedance is a measure of resistance that is partially dependent on the frequency of the wave being measured (discussed subsequently). Generally, electrode impedances should be under 5 kΩ (killiohms). It is good practice to recheck impedances both during and after the recording to document that electrodes have stayed well applied throughout.

Because it would never seem desirable to have 60-Hz artifact present in the EEG tracing, why not keep the 60-Hz notch filter in at all times? In fact, it is best technique to use the 60-Hz notch filter only when necessary (when 60-Hz artifact is seen to be contaminating the tracing). One reason for this is that the presence of 60-Hz artifact in one or more electrodes serves as a clue that there is an impedance problem in those electrodes. In reality, the dramatic appearance of the EEG in Figure 7-19 is also a red flag indicating that multiple electrodes have poor contacts. The presence of 60-Hz artifact in an electrode is an indicator of poor electrode contact for the following reason: even though 60-Hz activity may be present all over the head, we usually do not see it in the tracing because the activity cancels out during the subtraction of one electrode from another. If the two electrodes being compared

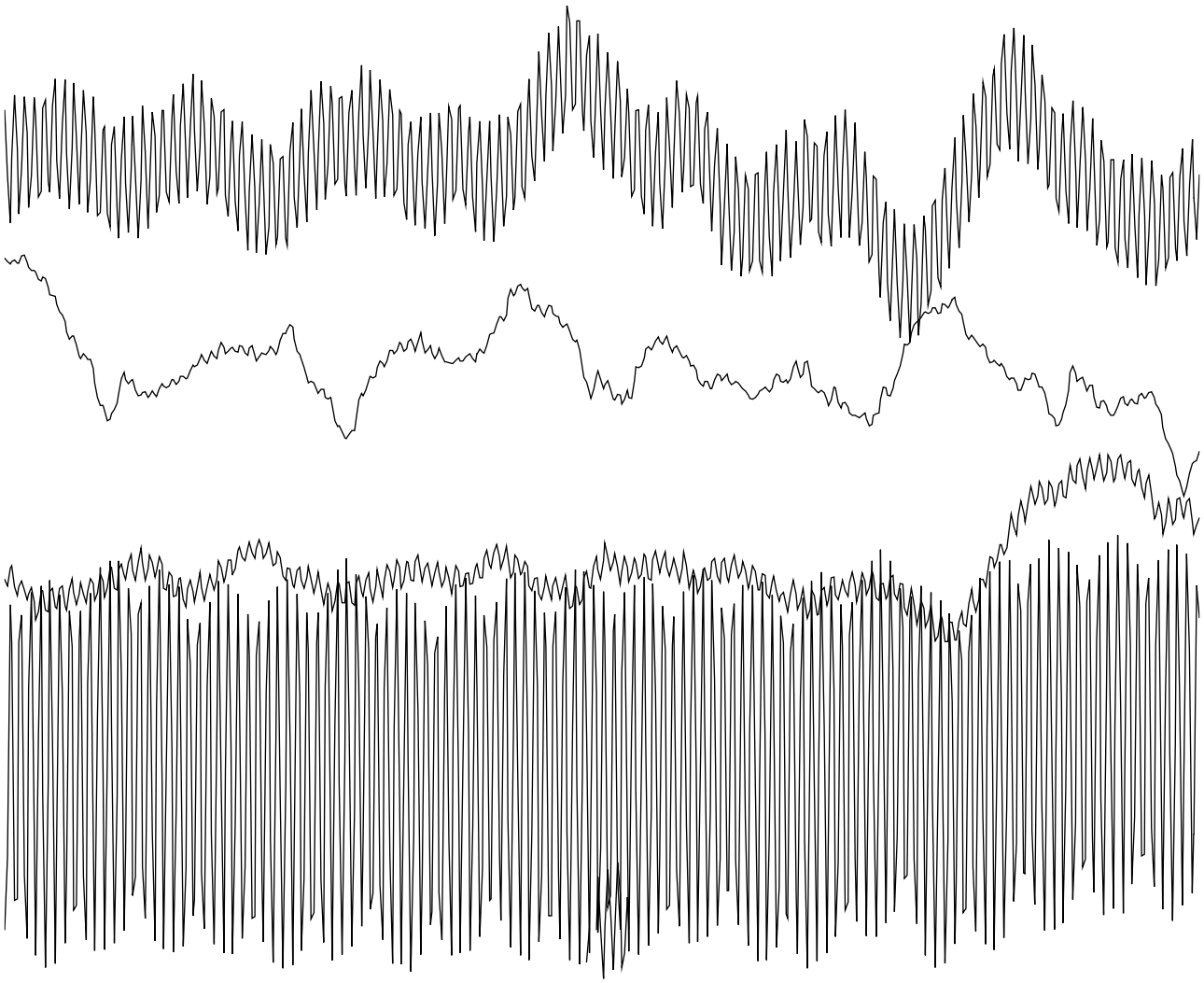


Figure 7-20 An enlargement of the top four channels from the previous figure is shown, revealing the detail of regular, well-formed 60-Hz sine waves. Each of the four channels shows different amounts of 60-Hz contamination. This artifact represents contamination of the patient's body with the alternating current sources that surround us in modern indoor environments.

have the same amount of 60-Hz artifact, then we will not see it in the channel that represents the subtraction. In contrast, if one of the electrodes in a subtraction is attached to the head in a different fashion than another (i.e., it has a different impedance than another), then the two electrodes will pick up the 60-Hz artifact to different degrees, and the artifact will be visible even after the subtraction. For this reason, the presence of 60-Hz artifact in an electrode is a clue to the possibility that that electrode may have a poor contact with the scalp. This is important to know because an improperly attached electrode may have a tendency to show voltages that are too high or low and may also be more prone to include other noise or artifact in the recording. Being alert to the fact that there is a poor electrode contact may prevent the reader from making the error of thinking that an abrupt electrode deflection represents true electrocerebral activity (such as a spike) rather than an electrode artifact due to a poor contact.

HIGH-FREQUENCY FILTERS, LOW-FREQUENCY FILTERS, AND RC CIRCUITS

High- and low-frequency filters are based on simple RC circuits, circuits that include both a resistor and a capacitor (hence the term "RC"). Although a detailed knowledge of the electronics behind circuits is not necessary to interpret EEGs, an acquaintance with the basic circuitry involved in the design of these filters can give insight into how the use of filters with different settings can affect EEG interpretation.

Following is a simple circuit with a single DC "battery" and a resistor (see Figure 7-22). Current flows through this circuit according to Ohm's law:

$$V = I \times R$$

Where V equals voltage, I equals current (measured in amperes) and R equals resistance (measured in ohms). Ohm's law is the major principle that helps us

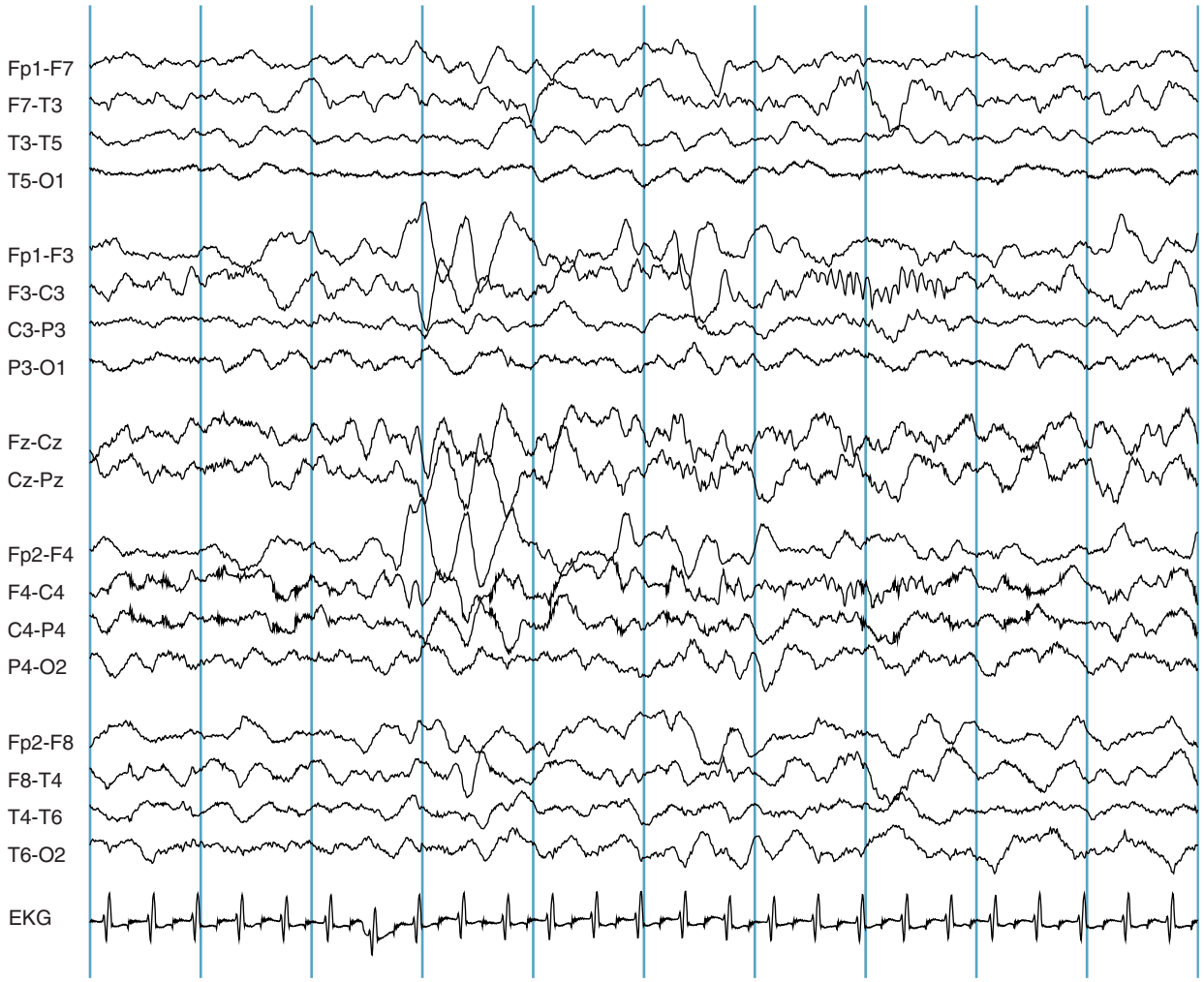


Figure 7-21 The same page of EEG as was shown in Figure 7-18, this time displayed with the use of a 60-Hz notch filter. The notch filter has dramatically "cleaned up" the EEG tracing, and the page now looks fairly unremarkable. Now that the page has been filtered, there is little to suggest to the reader that several of the electrode contacts probably have impedance problems.

understand the flow of current through a circuit at any given point. Choosing any two points in a circuit, Ohm's law should hold true, giving the relationships among the voltage difference, current flow, and resistance between those two points. The rearrangement of Ohm's law below makes intuitive sense:

$$I = \frac{V}{R} \qquad or \qquad current\,(amps) = \frac{voltage\,(volts)}{resistance\,(ohms)}$$

This way of writing Ohm's law highlights how a change in voltage or resistance, up or down, affects current flow, *I*. As the voltage difference between two points in a circuit increases, the current flow between those two points will increase. This makes sense because voltage is a synonym for the electromotive force (i.e., the electrical force that pushes electrons to move between one point and another). Likewise, when the resistance, *R*, increases at a particular location, this will slow down the current flow, *I*, as there is more resistance to the flow of electrons.

In addition to a resistor, a circuit may also include a capacitor (the 'C' in the term "RC circuit"). When the circuit is closed, the direct current (DC) battery in the circuit in Figure 7-23 will progressively charge the capacitor up to the maximum charge it can hold for that given voltage. After the capacitor reaches its fully charged state, the current flow will effectively stop. Particularly in the case of direct current, after the capacitor is fully charged, it essentially acts as a "brick wall" to further direct current flow.

An RC circuit with an alternating current (AC) source is a different story in terms of the capacitor's behavior. If the battery in our circuit is now replaced with an alternating current source, such as a standard AC current that resembles a sine wave (or an EEG signal which similarly oscillates), the effect of the capacitor on the circuit is quite different. This is because the alternating current will induce the capacitor plate to charge and discharge repeatedly, a behavior that the capacitor allows. The capacitor may never have the chance to act as a solid barrier to current flow as it did when the source was a DC battery because, before it gets to that point, the AC current has reversed and begins to discharge the capacitor again. One can imagine an RC circuit in which, if the current oscillates fast

enough, if the capacitor is big enough, and if the voltage oscillations are not too high, the presence of the capacitor may not change the behavior of an oscillating current appreciably. Thus, the extent to which a capacitor impedes the flow of current is significantly affected by the frequency of the oscillation.

One way to visualize this relationship is to consider a very, very slow alternating current. When the frequency of an alternating current is so low that it approaches zero, it begins to resemble a DC signal, which, we recall, will eventually be "blocked" by the capacitor after it becomes fully charged. However, an alternating current that oscillates very quickly is "blocked" much less by the capacitor—the capacitor simply goes through a repetitive charge-and-discharge cycle. Therefore, a capacitor provides varying resistance to current flow depending to a great extent on the frequency of the signal. The effective "resistance" that a capacitor provides to an alternating current presented to it is called the *impedance*, given by the formula:

$$Z = \frac{1}{2\pi fC}$$

where Z is the effective impedance in ohms, f is the frequency of the signal, and C is the capacitance of the capacitor given in farads. Note that the formula predicts, as expected, that as frequency rises, the impedance to current flow that the capacitor provides, Z, falls. At extremely high frequencies (f very high), the capacitor might provide little, if any, resistance to flow in the circuit. Likewise, as the capacitance of the circuit's capacitor rises (C very high), the impedance also falls. As we shall see later, basic filter design is based on this general principle that capacitors preferentially block flow to DC (or more slowly oscillating currents) and are more liberal in allowing higher frequency alternating currents to pass.

High-Frequency Filters

Figure 7-24 shows an example of a simple high-frequency filter. The input voltage is shown as an AC generator symbol because we will be considering the behavior of the filter with alternating currents (such as EEG waves) rather than just with direct currents. In this filter design setup, the output voltage comes from the terminals that arise from either side of the capacitor as shown. One way to understand this circuit is to imagine the behavior of the electrons (current) flowing around the circuit when they are oscillating at higher as opposed to lower frequencies. In the case of low-frequency activity, it is easiest to imagine a simple DC current as the most extreme example of low-frequency activity. In the case of a DC current, an electron would "prefer" to go down the output voltage pathway (after the capacitor is fully charged) because, as described earlier, the capacitor will eventually behave as a "brick wall" to the flow of DC current. However, if the electron is part of a rapidly oscillating current, it may be happy enough to be shunted in and out of the capacitor, which it can easily charge and discharge. In the case of such higher frequency signals, less

Figure 7-22 This simple circuit diagram shows a direct current source on the left and a resistor on the right.

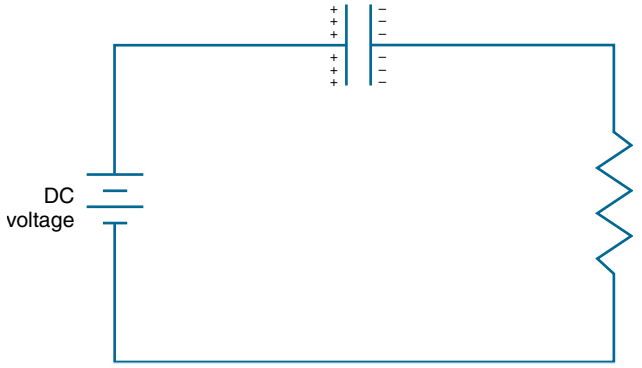


Figure 7-23 A capacitor has been added to the circuit from the previous example forming a simple RC circuit. When the circuit is closed as drawn, current flows until the capacitor fully charges, at which time current flow in the circuit drops toward zero.

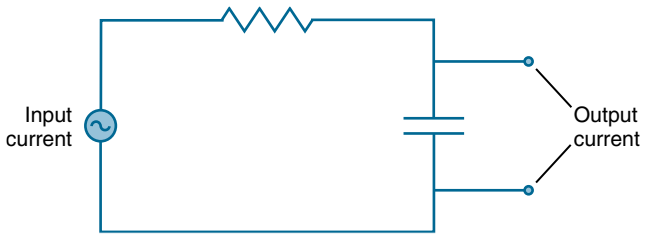


Figure 7-24 The diagram shows a schematic of a simple **high-frequency filter** (HFF). The direct current battery on the left side of the circuit has been replaced by an alternating current source which could also represent an EEG signal. A resistor and a capacitor are placed in series. The filter's output comes from the two poles placed on either side of the capacitor on the right, as shown. A voltmeter reading the potential difference across the two poles would represent the equivalent of the filter's output.

current would then flow down the output voltage terminals because it would prefer to flow in and out of the capacitor instead (see Figure 7-25). For this reason, this RC circuit setup filters out high-frequency signals but allows low-frequency signals to "pass" down the output terminals.

A more mathematical way of looking at this situation is that Ohm's law should hold across the two points defined by the output terminals. The output current of the filter is essentially the same as the voltage across the

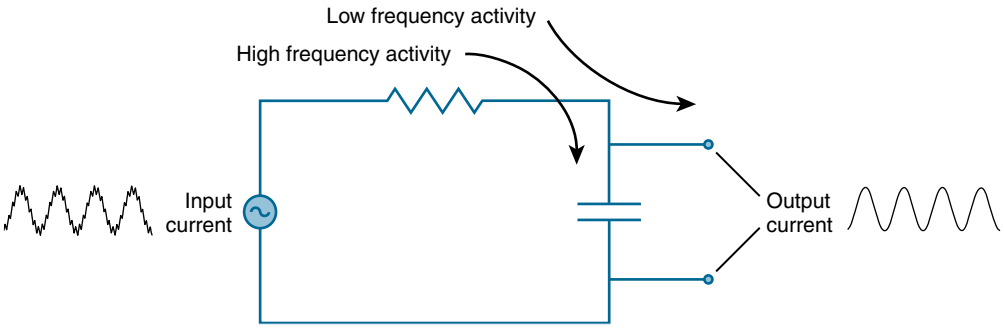


Figure 7-25 The input signal on the left consists of a mixture of a fast wave riding on a slow wave. Because it is easy for high-frequency activity to charge and uncharge the capacitor sequentially, a high-frequency signal "feels" relatively little resistance to flow across the capacitor. Because the capacitor presents little resistance (or impedance) to high-frequency activity, the voltage drop measured across the capacitor at the two output terminals is relatively low, and thus high-frequency activity in the output current is filtered out. In contrast, the low-frequency component of the activity sees the capacitor as a barrier (or higher resistance to flow) resulting in a higher voltage drop being measured across the output terminals and successful output of the low-frequency signal. Therefore, high-frequency components of the input current do not tend to appear at the output terminals, whereas low-frequency signals do.

capacitor (i.e., equivalent to the reading of a voltmeter attached to the two output current poles in the diagram). According to Ohm's law, $V = I \times R$, the voltage across any two points is a product of the current and the resistance between those two points. Because the effective resistance of the capacitor (impedance) increases in low-frequency situations, the voltage drop measured across the output terminals will increase when there is a low-frequency signal in the circuit. Because the resistance to current flow across the capacitor decreases markedly in the case of high-frequency currents, Ohm's law tells us that the voltage drop measured across the capacitor falls considerably with high-frequency signals. Because the voltage drop across the capacitor in this filter is essentially synonymous with the output voltage, a low-frequency input signal results in little change in the output voltage from this filter. High-frequency input signals result in more drastically attenuated output currents.

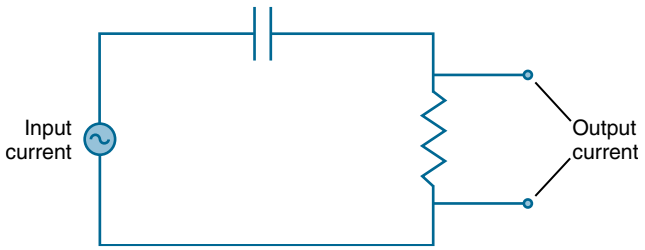


Figure 7-26 The design of the **low-frequency filter** (LFF) resembles that of the high-frequency filter (HFF), except that the positions of the capacitor and resistor are exchanged. Now the output current is measured by placing the equivalent of a voltmeter across the resistor in the circuit. Alternatively, the circuit diagram could have been drawn with the resistor and capacitor in the same positions as in the HFF diagram, but with the output terminals on either side of the resistor. The net effect would be the same.

Low-Frequency Filters

The low-frequency filter design shown in Figure 7-26, is similar to the design of the high-frequency filter except that the positions of the resistor and capacitor are exchanged. Another way of looking at this is that the output current terminals are being placed across the resistor in the circuit rather than across the capacitor (see Figure 7-27). Considering the behavior of the circuit shown in Figure 7-26 with different types of signals, we can imagine that the flow of a very low-frequency signal, one that resembles a DC current, will be significantly blocked by the capacitor, resulting in little current flow around the circuit in general (see Figure 7-28). With reduced current flow through the resistor, there will be little voltage change across the resistor (because $V = I \times R$) and thus little output voltage across the output terminals. In the case of a high-frequency signal, there will be much more current oscillation across the resistor and, therefore, a higher

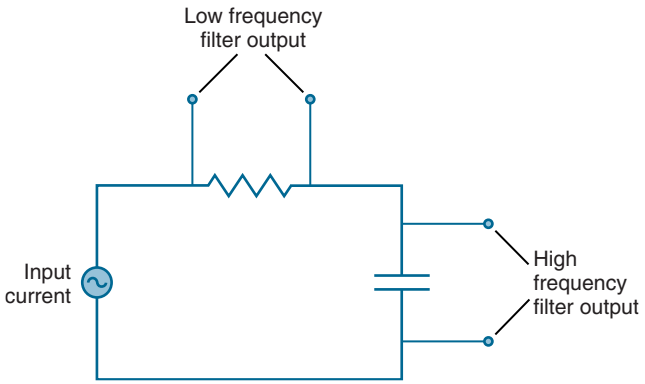


Figure 7-27 The difference between HFF circuit design and LFF circuit design is that the filter output represents the voltage measured across the capacitor or the resistor, respectively. Circuit diagrams can show this either by exchanging the position of the resistor and the capacitor as in the previous figures or by changing the points of voltage measurement as in this figure.

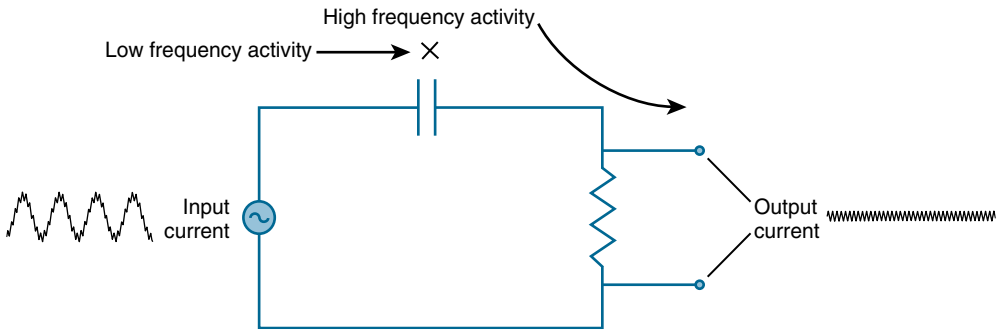


Figure 7-28 Again, the input signal on the left consists of a mixture of a fast wave riding on a slow wave. The output current of the low-frequency filter is equivalent to the measurement of the voltage across the resistor on the right side of the circuit. As discussed in the text, direct current or low-frequency activity is relatively blocked by the presence of the capacitor, resulting in lower current flow across the resistor and a lower voltage drop across the output current terminals. In contrast, the high-frequency component of the signal passes relatively without impediment through the capacitor, resulting in more high-frequency current oscillations through the circuit as a whole and, therefore, across the resistor, resulting in successful passage of high-frequency activity to the output terminals.

output signal will result across the resistor. In this way, this type of RC filter tends to filter out low-frequency signals and passes high-frequency signals. It is worth noting that, in the case of waves of mixed frequency, the RC circuits described earlier handle the high- and low-frequency components of mixed waves as if they were separate.

THE FAST TRANSIENT AND SQUARE WAVE RESPONSE OF FILTERS

In daily clinical use, the behavior of different filter setups is verified by the way they handle square wave inputs. The EEG instrument generates a square wave signal for the purpose of calibration in which the voltage is stepped up to a certain level, held at that level, then decreased to the original level, and so on, creating the square wave. The calibration signal allows EEG technologists and readers to verify that filters and amplifiers are functioning properly by examining how they affect the shape and size of square waves. High- and low-frequency filters affect the shape of a square wave input in different ways (see Figure 7-29). It is customary for the EEG technologist to document the proper behavior of the amplifiers and filters by running a square wave through the machine periodically and producing such calibration signals, which can be analyzed as described in the following sections.

High-Frequency Filter Handling of Transients

First, in the case of the high-frequency filter, what will the output wave be when the input current is a square wave? Remember that the output current of a high-frequency filter is essentially the measurement of the voltage across the capacitor in the RC circuit shown in Figure 7-24. At the initial moment of the upsweep of the square wave, the capacitor has just begun to charge and effectively provides no resistance to current flow because it is initially empty of charge. At that initial moment of no effective resistance, because $V = I \times R$, the voltage drop across the capacitor (which is the same as the output voltage of the filter) is zero. As the

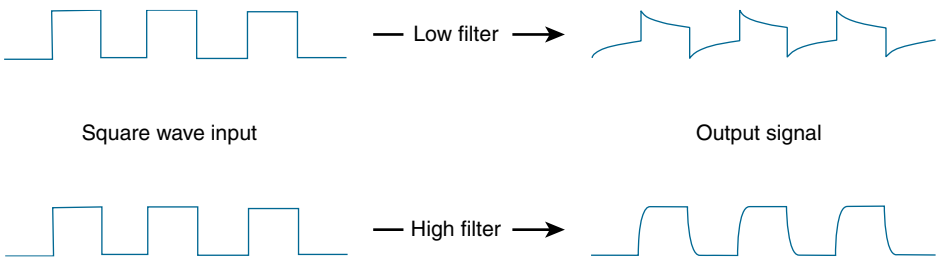


Figure 7-29 This figure depicts the separate effects of a high-frequency filter (HFF) and a low-frequency filter (LFF) on a square wave input. Note the transformation of the square wave brought about by the LFF in the upper output trace. The vertical portion of the square wave is unchanged but the horizontal portions of the wave tend to "sag" or "float" toward the baseline. LFFs tend to filter out any horizontal (DC) portion of a square wave that is away from the baseline by causing it to drift toward "zero" voltage. In contrast, the vertical component of the square wave can be thought of as a very fast wave and is not changed by an ideal LFF. The lower output trace shows the effect of an HFF on a square wave. Now note that the upswings and downswings of the square wave (the vertical components) are slurred and rounded off. The tendency of HFFs to curve or round off the fast or near-vertical components of a waveform can transform spikelike waves into waveforms that appear more sinusoidal. This effect is accentuated by more aggressive use (lower cutoff frequencies) of HFFs. As expected, the HFF does not affect the horizontal (DC) components of the square waves.

capacitor continues to charge it begins to provide resistance to current flow and an increasing voltage drop can be measured across it. After the capacitor becomes fully charged, the maximum voltage difference across its poles is attained and the output voltage curve seen in Figure 7-30 results. In fact, the quick step-up of the input voltage at the beginning of the square wave can be seen as a type of very high-frequency activity. In summary, besides decreasing the amplitude of high-frequency sine wave activity as discussed earlier in this chapter, high-frequency filters also tend to "round" the shape of the rapid upswings and downswings in waves (fast transient activity).

Low-Frequency Filter Handling of Transients

In the case of the low-frequency filter, a converse type of behavior is seen. Keep in mind that total voltage change across the circuit (which, in this example, is essentially the voltage of the square wave input at any given instant) must be the sum of the voltage change across the capacitor and the voltage change across the resistor (see Figures 7-30 and 7-31). Given the LFF filter design, the output wave is synonymous with the voltage change across the resistor as shown in Figure 7-25. At the initial instant of the step up in voltage of the square wave, the capacitor is uncharged and thus provides no resistance to current flow. Consequently, according to Ohm's law, there is initially no voltage change across the capacitor (V_1 in Figure 7-28 B). Thus, at the moment of the square wave upswing, the voltage across the resistor (which is synonymous with the filter's output voltage) is equal to the voltage of the square wave input–the output wave nearly exactly parallels the upswing of the square wave. As the capacitor begins to charge, however, it begins to offer increasing resistance to flow, and, according to Ohm's law, the voltage drop measured across it increases. Because the sum of the voltage drops offered by the capacitor and the resistor must remain steady, ($V_1 + V_2$ always equals V_{total}), it is clear that, as the voltage increases across the capacitor as it is acquiring charge and providing more resistance to current flow, the voltage across the resistor must concomitantly fall. This accounts for the decaying curve seen in Figure 7-32. If the square wave is held at peak voltage long enough, the capacitor will eventually become completely charged, and current flow in the circuit will stop. If there is no current flow across the resistor, there will be no voltage change across the resistor (per Ohm's law, $V = I \times R$), and the output wave curve in Figure 7-32 eventually drops to zero, as shown.

Therefore, in addition to decreasing the amplitude of slow-wave activity as discussed earlier in this chapter, low-frequency filters also change the shape of low-frequency activity, creating a tendency for a wave that otherwise would have been "horizontal" in shape above or below the x axis to veer toward zero. A horizontal wave above the x axis will tend to "sag" downward toward zero and a horizontal wave below the x axis will tend to "float" upward toward zero. This effect is illustrated in Figure 7-32, which shows the square wave input shown in light blue remaining horizontal above the x axis, but the low-frequency filter output of the square wave sagging toward zero. One advantage of the tendency of LFFs to cause the output voltage to sag or float toward zero is that they help keep EEG channels from straying far outside their horizontal areas for prolonged periods of time. A disadvantage is that, should there actually be a prolonged DC current (voltage held above or below zero for a prolonged period of time) arising from the brain, the LFF would tend to mask it.

Confirming the effect of high and low filters on square wave inputs, also known as the "transient response," helps demonstrate that an electroencephalograph is operating properly and according to the stated filter settings. An additional consequence of the filters' impact

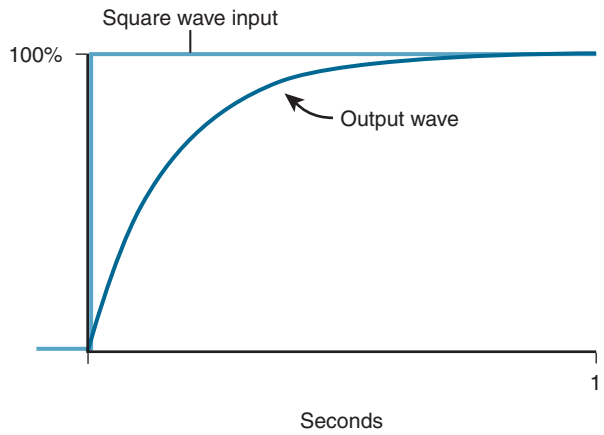


Figure 7-30 This figure shows the effect of a 1-Hz high-frequency filter (HFF) on a square wave input. The square wave input voltage is shown in light blue and can be considered an enlargement of a portion of the square wave shown in the previous figure. The output voltage is shown in dark blue. The exaggerated HFF setting of 1 Hz shown in this example is not used in clinical EEG but is used here for illustrative purposes. Imagining that the upswing of the blue square wave in this example represents a very fast spike in the EEG, the HFF would tend to round its shape considerably. Aggressive use of HFFs can misleadingly round out activity that would otherwise have a "spiky" shape.

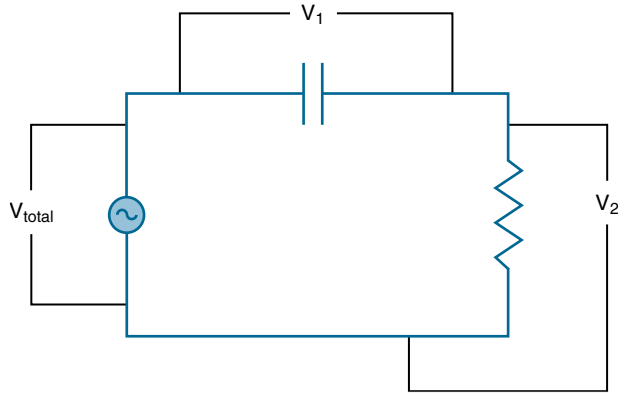


Figure 7-31 The sum of the voltage across the capacitor, V_1, and the voltage across the resistor, V_2, must always equal the total voltage across the whole circuit, V_{total}. In the case of a square wave for which voltage has stepped up to a certain value (the top of the square wave), V_{total} holds steady for that moment and V_1 and V_2 have shifting shares of the total voltage as the capacitor charges, as described by the curves in the previous figures.

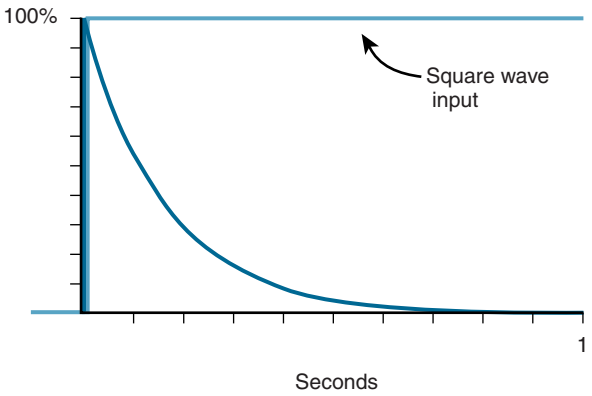


Figure 7-32 A 1-Hz low-frequency filter changes the shape of the same square wave input (shown in light blue) dramatically. The dark blue line shows how the fast initial upsweep portion of the square wave is not affected by the low-frequency filter, but the filter causes the horizontal portion of the square wave to "sag" toward zero. The filter handles the horizontal portion of the square wave, which is a step-up of voltage to a constant level held for a period of time, akin to very low-frequency activity. The horizontal part of the square wave can be thought of as a direct current with a frequency of zero.

on square wave shape is that filters can change not just the amplitude but also the shape of the input waves. The tendency of the high-frequency filter to round the shape of a fast transient (such as the upswing of the square wave) as illustrated in the idealized curves shown in Figures 7-30 and 7-31) can also be seen in changes in the shape of EEG waves recorded in practice. Figures 7-33 through 7-35 show the effect of increasingly aggressive HFF cutoff frequencies on a page of EEG. Note, in particular, the change of the appearance of the muscle artifact in the right temporal channels at the bottom of the page (F8-T8 and T8-P8). The use of aggressive HFF settings transforms what was obvious "spiky" muscle artifact to a wave that is sinusoidal in appearance and could easily be mistaken for brain wave activity.

An additional type of distortion produced by RC filters is phase shifting, which is a displacement of a wave to the left or to the right from its original position along the time axis. Although phase shifting is usually minor and does not present a major problem in clinical electroencephalography, the phase shifting effect can be more important in other applications, such as evoked potentials, in which absolute wave latencies are measured.

FILTER TIME CONSTANTS

The effect that high- and low-frequency filters have on square waves can be described in terms of a parameter called the filter's *time constant*. The time constant happens to be the product of the resistance and the capacitance of those two elements in the RC circuit and is represented by the Greek letter "tau": $\tau = R \times C$. As

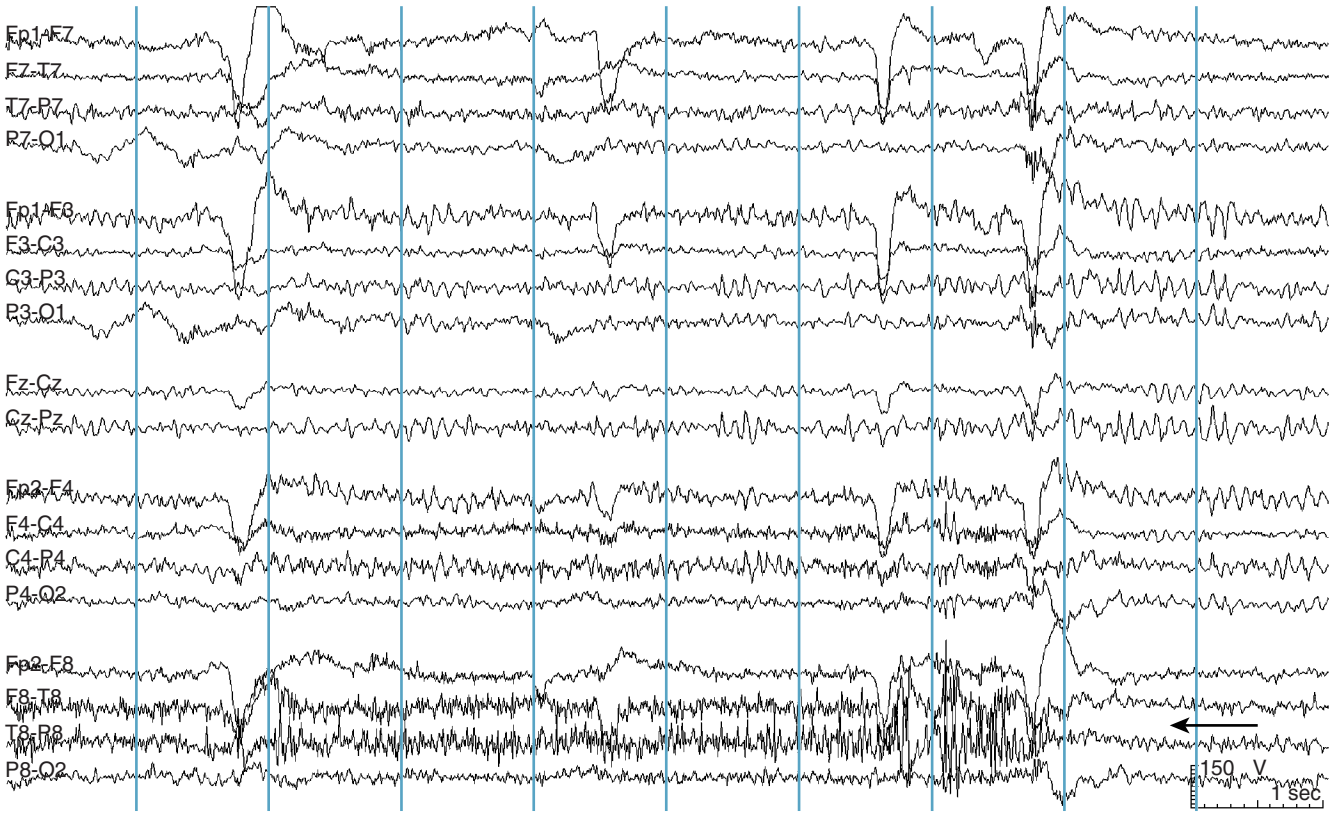


Figure 7-33 This page of EEG recorded with a standard high-frequency filter setting of 70 Hz shows an awake pattern with a posterior rhythm that is well seen and multiple eyeblink artifacts. The varying, spiky and sputtering nature of the fast activity seen in the F8-T8 and T8-P8 channels (arrow) are strong clues to its origin from muscle rather than from brain. The effect of lowering the HFF a toff frequency on these waves is examined in the following figures.

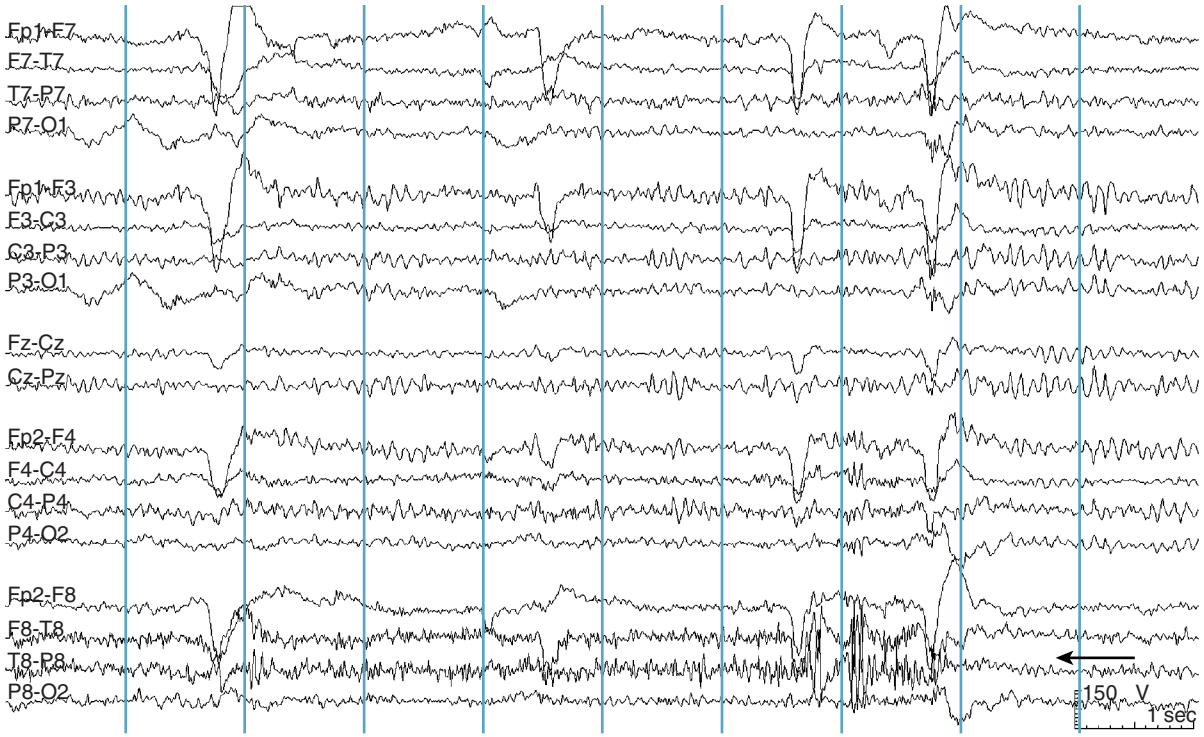


Figure 7-34 The same page of EEG shown in the previous figure is displayed with a more aggressive high-frequency filter setting of 35 Hz. Compare the appearance of the muscle artifact in the right temporal area to the previous figure. The ragged, spiky appearance of the right temporal muscle artifact is now less pronounced though, for the most part, the activity can still be identified as arising from muscle rather than from brain (arrow). Also note changes caused by the filter, sometimes subtle, elsewhere on the page.

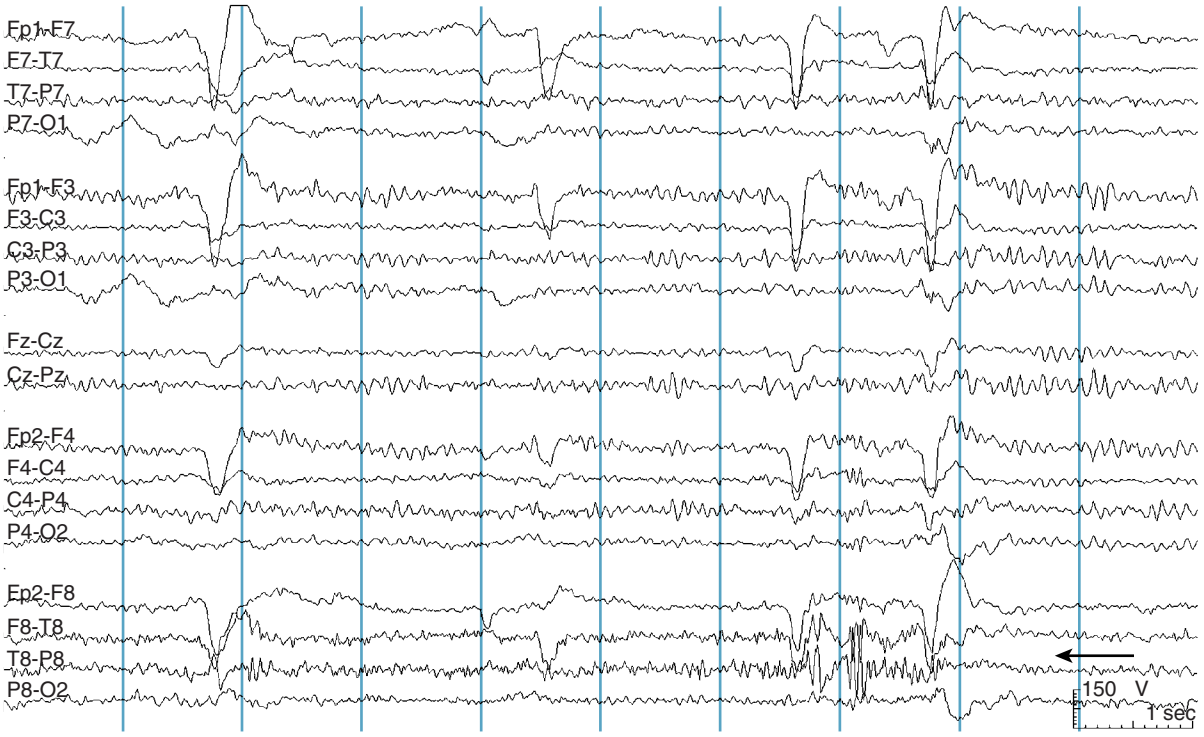


Figure 7-35 The same page of EEG as shown in the previous two figures is now displayed with a very aggressive high-frequency filter (HFF) setting of 15 Hz. This very low HFF cutoff frequency is rarely used in clinical practice because of its profound effect on wave shape but its effect is shown here for illustrative purposes. The HFF's tendency to round the shape of fast activity has converted the previously spiky muscle artifact into a sinusoidal waveform (arrow). It is no longer obvious that the source of the right temporal activity is muscle rather than brain activity. With these filter settings what we know to be muscle artifact could now conceivably be mistaken for right temporal spikes.

we shall see, τ gives the number of seconds it takes for a square wave to fall by 63% of its original value. In this discussion, we have been naming filters according to their cutoff frequencies, but some electroencephalographs (depending on the preference of the manufacturer) label high- and low-frequency filters according to their cutoff frequencies, and others label filters according to their time constants. Recently there has been a trend toward the use of cutoff frequency labels.

The equation below describes the output voltage for an HFF that is processing a square wave:

$$V_{output} = V_{input} \times \left(1 - e^{\frac{-t}{\tau}}\right)$$

where V_{output} is the output voltage of the filter, V_{input} is the input voltage of the filter, t is the number of seconds, $e \cong 2.718$ (the natural logarithm constant), and τ is the time constant of the filter.

Looking at this equation, it is clear that at time $(t) = 0$, the "$e^{(-t/\tau)}$" term $= e^0 = 1$, so the output voltage at $t = 0$ will equal zero. As t tends toward infinity, the "$e^{(-t/\tau)}$" term tends toward $e^{-\infty}$, or zero, and the output voltage approaches the input voltage. This is, indeed, what we saw in Figures 7-30 and 7-31.

Another key property of this equation is its behavior at the point where $(t) = \tau$ seconds. At that point, the output voltage will rise to $(1 - e^{-1}) = (1 - 0.36)$ or approximately 0.63 times its final value. This is the basis for the procedure used to check a calibration signal. The amount of time it takes for the upswing of the square wave signal to rise to 63% of its final value in the case of high filters or the amount of time it takes for the square wave signal to decay by 63% of its initial value in the case of low filters is measured. This measuring technique for checking low filters is shown in Figure 7-36. The time constant, τ of a filter can be easily derived from the filter's cutoff frequency and vice versa by means of the following simple relationship:

$$f_{cutoff} = \frac{1}{2\pi\tau}$$

or, exchanging the positions of the cutoff frequency and the time constant:

$$\tau = \frac{1}{2\pi f_{cutoff}} = (0.16)\frac{1}{f_{cutoff}}$$

The 0.16 term comes from taking $1/(2\pi)$ out of the fraction on the left side of the equation. For example, for a 5-Hz low-frequency filter, using the formula above, the corresponding time constant is $(0.16) \times (1 / 5 \text{ sec}^{-1}) = 0.032$ sec (see Figure 7-37). Likewise, for the example of the 1-Hz filter, the time constant should be 0.16 seconds. The equations

$$\tau = \frac{1}{2\pi f_{cutoff}} \qquad f_{cutoff} = \frac{1}{2\pi\tau}$$

can be used interchangeably to convert cutoff frequencies to time constants and vice versa. The effects of high- and low-frequency filters are summarized in Table 7-1.

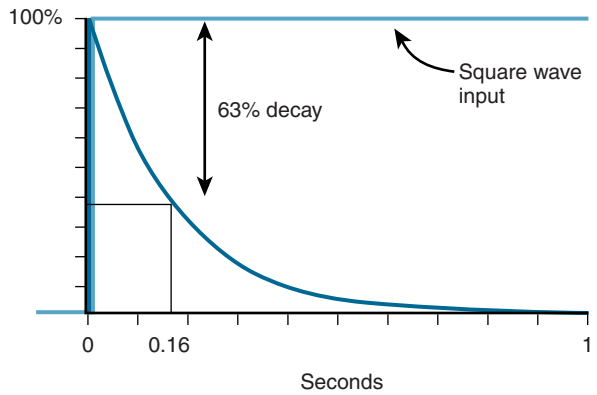


Figure 7-36 Proper behavior of the filter settings in use can be confirmed by measuring the changes in square wave calibration pulses. The time constant of a low-frequency filter is defined as the amount of time it takes for a square wave pulse passed through the filter to fall by 63% of its original value (to 37% of its original value). The square wave pulse generated by the instrument is shown in light blue. The reader measures the height of the calibration pulse and identifies the point at which it has fallen by 63%. The amount of time in seconds that it has taken for the wave to fall to this point is measured on the x axis, in this example, 0.16 seconds, and represents the filter's time constant. The value of the time constant, τ, can be plugged into the equation, which gives the relationship between the cutoff frequency and the time constant $f_{cutoff} = (1/2\pi\tau)$, which equals 1 per second (1 Hz) in this case. Therefore, the measurement of this calibration pulse implies that a low-frequency filter with a time constant of 0.16 seconds (i.e., a cutoff frequency of 1 Hz) is in use.

DIGITAL FILTERS

The complex science of digital filter design belongs to the field of digital signal processing and is beyond the scope of this text, but it is worthwhile to consider some basic techniques in digital filtering to have a general idea of the workings of these filters. The RC circuits described earlier are designed to work on real electrical currents and are therefore analog devices. The advent

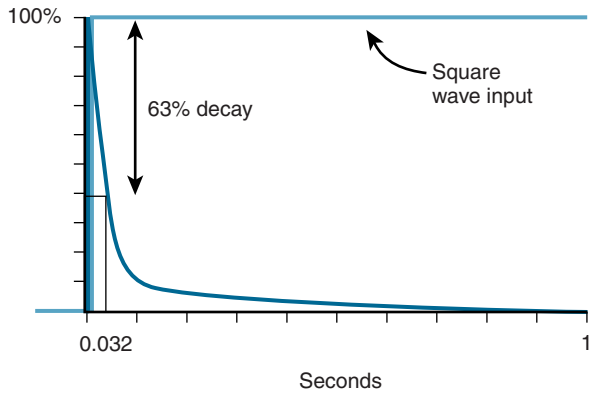


Figure 7-37 Here, the same square wave pulse (shown in light blue) is passed through the instrument. In this example, the pulse falls to 63% of its original value after only 0.032 seconds. When this time constant is plugged into the equation $f_{cutoff} = (1/2\pi\tau)$, $f_{cutoff} = 5$ per second, implying that a 5-Hz low-frequency filter is now in use.

Table 7-1 Effects of High- and Low-Frequency Filters

	Synonyms	Attenuation	Advantages	Effect on Wave Shape	Pitfalls of Use	Standard Settings
Low-frequency filter	Low-filter, high-pass filter	Attenuates a sine wave at cutoff frequency by 30%	Helps keep waves near baseline, reduces very low-frequency activity, which is often artifact	Causes waves to "sag" or "float" toward baseline	Reduces ability to appreciate slow waves and asymmetries	1 Hz
High-frequency filter	High-filter, low-pass filter	Attenuates a sine wave at cutoff frequency by 30%	Reduces very high-frequency activity, which is often electrical noise or artifact	Rounds off shape of fast transients (vertical elements)	Makes high-frequency noise (e.g., muscle artifact) look more like sine waves (brain waves)	70 Hz

of computerized digital EEG instruments has completely changed the methodologies used to design filters. It is important to remember that the waves displayed on digital EEG instruments really represent streams of numbers. Successive strings of numbers are clumped into "bins" and displayed essentially as the tops of histograms, as shown in Figure 7-38. The tops of the histogram are joined together to create a curve, the displayed EEG wave.

Consider the wave shown in Figure 7-39. A reader who is accustomed to analyzing EEG waves may see a combination of a slower wave and a faster wave superimposed wave in this pattern, but the computer "sees" something entirely different. Rather than the shape of the wave, the software that runs the digital EEG instrument has nothing more available to it than a stream of numbers. The stream of numbers that the wave in Figure 7-39 comprises is shown in Table 7-2.

Imagine how much more difficult it would be to recognize that the wave associated with this set of numbers consists of a mixture of a slow wave and a superimposed fast wave if only the list of numbers from Table 7-1 were available for your analysis. Likewise, the challenge of creating a filter that might filter out either the fast activity or the slow activity from the stream of digits that comprises this waveform represents a completely different type of problem from that of designing an RC circuit. Whereas analog electrical signals can be passed through RC circuits of the types depicted above, streams of numbers cannot. Instead, different types of mathematical manipulations are brought to bear on the type of digitized information shown in the table to filter out different types of activity.

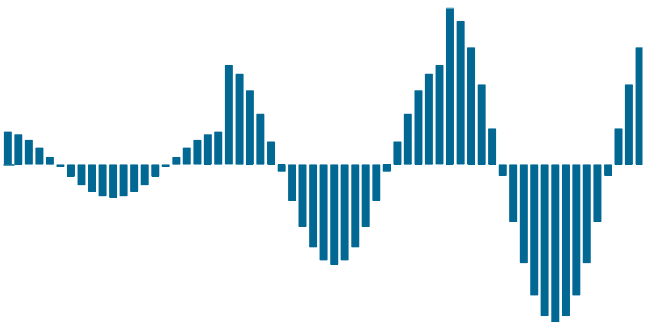


Figure 7-38 The waves that appear on the EEG display represent a graphical depiction of digital data. Typically, the EEG is sampled at a rate approximately between 200 and 500 times per second. Each sample or "bin" of data is then plotted graphically creating a curve. This figure shows how discrete digital data points can be resolved into a smooth-appearing curve.

Digital HFFs

First, we consider a simple algorithm to filter out fast activity from this digital waveform. The technique used here is similar to the technique referred to as "moving averages." (The technique of moving averages is also used by some financial analysts to track the historical movement of a stock's price over time.) With this technique, this digital wave is transformed by taking the average of a set number of the previous points or "bins" before it. In the example that follows, the wave shown in Figure 7-39 is transformed by replacing the 100th point by the average of the previous 99 points (Points 1 through 99). Likewise, the 101st point is replaced by the average of Points 2 through 100, and so on. Applying this technique to the stream of numbers shown in Table 7-2 that created the wave shape seen in Figure 7-39, the new wave seen in Figure 7-40 is generated. Note that the fast component of the wave is considerably attenuated, although it is still recognizable. The slow component of the wave is now its most prominent feature.

Digital LFFs

A simple digital algorithm for filtering out slow activity involves subtracting adjacent bins and replacing the original values with the differences. Using this technique, the second point would be replaced by the difference between the second point and the first point, the third point would be replaced by the difference between the third point and the second point, and so on. The wave depicted in Figure 7-41 shows the result of using this simple algorithm on our original digital wave from Table 7-2. Note that the slow activity is nearly completely filtered out, and the fast activity remains. The only remnant of the slow activity is a slight

Table 7-2 Digital Data for Waveform Shown in Figure 7-39

0.200	0.487	-1.607	-0.291	-0.777	-1.824	0.203	-0.770	-0.459	1.369	-0.091	1.067	1.661	-0.120
0.395	0.631	-1.468	-0.172	-0.780	-1.849	0.110	-0.947	-0.598	1.201	-0.233	1.030	1.636	-0.052
0.580	0.777	-1.315	-0.032	-0.755	-1.845	0.040	-1.109	-0.732	1.025	-0.391	0.968	1.583	-0.011
0.752	0.919	-1.151	0.126	-0.705	-1.811	-0.004	-1.250	-0.856	0.846	-0.562	0.884	1.501	0.004
0.906	1.053	-0.983	0.299	-0.631	-1.749	-0.022	-1.368	-0.966	0.669	-0.741	0.781	1.393	-0.009
1.038	1.175	-0.813	0.482	-0.537	-1.659	-0.012	-1.461	-1.058	0.499	-0.924	0.662	1.260	-0.049
1.146	1.280	-0.648	0.671	-0.425	-1.544	0.024	-1.526	-1.128	0.339	-1.104	0.532	1.107	-0.114
1.228	1.365	-0.492	0.861	-0.301	-1.406	0.088	-1.562	-1.174	0.195	-1.279	0.395	0.936	-0.203
1.281	1.427	-0.349	1.047	-0.168	-1.249	0.175	-1.570	-1.194	0.069	-1.443	0.256	0.751	-0.313
1.306	1.462	-0.223	1.225	-0.032	-1.076	0.286	-1.549	-1.185	-0.035	-1.591	0.120	0.558	-0.441
1.303	1.470	-0.118	1.389	0.104	-0.892	0.415	-1.500	-1.148	-0.114	-1.721	-0.008	0.361	-0.583
1.271	1.449	-0.036	1.536	0.234	-0.701	0.560	-1.427	-1.082	-0.166	-1.827	-0.125	0.164	-0.734
1.214	1.399	0.020	1.662	0.354	-0.508	0.717	-1.331	-0.989	-0.191	-1.909	-0.225	-0.028	-0.890
1.133	1.321	0.050	1.764	0.459	-0.318	0.880	-1.216	-0.870	-0.189	-1.963	-0.306	-0.209	-1.046
1.032	1.216	0.052	1.840	0.546	-0.136	1.046	-1.086	-0.728	-0.160	-1.988	-0.364	-0.376	-1.198
0.915	1.087	0.027	1.888	0.612	0.034	1.209	-0.946	-0.567	-0.105	-1.983	-0.397	-0.525	-1.340
0.784	0.936	-0.024	1.906	0.653	0.187	1.366	-0.799	-0.389	-0.026	-1.950	-0.404	-0.652	-1.469
0.646	0.766	-0.100	1.895	0.668	0.321	1.510	-0.650	-0.199	0.073	-1.888	-0.382	-0.754	-1.580
0.503	0.582	-0.198	1.855	0.655	0.432	1.638	-0.505	-0.002	0.189	-1.800	-0.333	-0.830	-1.669
0.363	0.388	-0.314	1.788	0.614	0.518	1.747	-0.368	0.199	0.318	-1.688	-0.257	-0.879	-1.734
0.228	0.189	-0.445	1.696	0.545	0.576	1.832	-0.243	0.397	0.457	-1.556	-0.155	-0.899	-1.773
0.104	-0.010	-0.587	1.583	0.450	0.607	1.892	-0.134	0.588	0.600	-1.407	-0.030	-0.891	-1.783
-0.006	-0.205	-0.736	1.450	0.330	0.610	1.924	-0.045	0.768	0.742	-1.245	0.115	-0.856	-1.764
-0.097	-0.392	-0.886	1.303	0.188	0.585	1.927	0.021	0.932	0.880	-1.075	0.277	-0.796	-1.716
-0.166	-0.564	-1.032	1.146	0.028	0.535	1.900	0.062	1.077	1.007	-0.902	0.452	-0.714	-1.640
-0.211	-0.720	-1.171	0.983	-0.148	0.462	1.845	0.076	1.199	1.121	-0.730	0.635	-0.612	-1.537
-0.230	-0.854	-1.298	0.819	-0.334	0.367	1.762	0.063	1.296	1.216	-0.564	0.822	-0.495	-1.409
-0.222	-0.964	-1.408	0.659	-0.527	0.256	1.653	0.022	1.365	1.290	-0.409	1.008	-0.367	-1.261
-0.186	-1.048	-1.498	0.508	-0.721	0.132	1.521	-0.045	1.406	1.340	-0.269	1.188	-0.232	-1.094
-0.122	-1.105	-1.565	0.370	-0.911	-0.001	1.369	-0.137	1.418	1.362	-0.147	1.358	-0.094	-0.913
-0.033	-1.133	-1.605	0.248	-1.093	-0.137	1.201	-0.251	1.401	1.357	-0.048	1.513	0.040	-0.723
0.080	-1.132	-1.618	0.147	-1.263	-0.273	1.022	-0.385	1.358	1.323	0.028	1.648	0.167	-0.529
0.214	-1.104	-1.602	0.070	-1.415	-0.404	0.835	-0.535	1.289	1.260	0.077	1.762	0.283	-0.335
0.365	-1.050	-1.557	0.017	-1.547	-0.524	0.647	-0.697	1.198	1.170	0.099	1.850	0.382	-0.145
0.531	-0.973	-1.483	-0.009	-1.656	-0.630	0.460	-0.865	1.088	1.054	0.094	1.911	0.462	0.035
0.706	-0.875	-1.383	-0.007	-1.738	-0.718	0.281	-1.037	0.963	0.914	0.062	1.944	0.519	0.200
0.886	-0.760	-1.257	0.022	-1.792	-0.784	0.114	-1.206	0.826	0.754	0.004	1.947	0.551	0.348
1.066	-0.633	-1.109	0.077	-1.817	-0.825	-0.037	-1.369	0.683	0.578	-0.078	1.921	0.556	0.475
1.242	-0.497	-0.943	0.156	-1.812	-0.840	-0.169	-1.520	0.539	0.389	-0.180	1.866	0.534	0.578
1.408	-0.358	-0.762	0.258	-1.779	-0.828	-0.278	-1.655	0.398	0.192	-0.300	1.785	0.483	0.654
1.560	-0.220	-0.570	0.378	-1.719	-0.787	-0.362	-1.770	0.264	-0.009	-0.433	1.681	0.405	0.703
1.694	-0.087	-0.373	0.514	-1.634	-0.719	-0.420	-1.863	0.142	-0.207	-0.575	1.555	0.302	0.724
1.807	0.035	-0.175	0.661	-1.527	-0.623	-0.449	-1.930	0.037	-0.400	-0.722	1.413	0.175	0.716
1.896	0.142	0.020	0.814	-1.401	-0.503	-0.451	-1.969	-0.048	-0.581	-0.868	1.257	0.027	0.683
1.957	0.231	0.205	0.969	-1.260	-0.360	-0.426	-1.979	-0.111	-0.747	-1.009	1.093	-0.138	0.624
1.990	0.298	0.378	1.121	-1.108	-0.198	-0.375	-1.960	-0.149	-0.895	-1.141	0.926	-0.316	0.542
1.994	0.341	0.534	1.265	-0.951	-0.020	-0.301	-1.911	-0.160	-1.019	-1.259	0.759	-0.503	0.441
1.969	0.357	0.669	1.397	-0.793	0.168	-0.206	-1.835	-0.143	-1.119	-1.359	0.599	-0.695	0.325
1.914	0.347	0.781	1.513	-0.638	0.363	-0.094	-1.733	-0.099	-1.192	-1.437	0.448	-0.885	0.197
1.833	0.308	0.866	1.608	-0.492	0.560	0.031	-1.607	-0.028	-1.236	-1.491	0.313	-1.070	0.063
1.727	0.242	0.924	1.681	-0.358	0.754	0.165	-1.462	0.067	-1.252	-1.518	0.195	-1.245	-0.074
1.600	0.150	0.954	1.727	-0.241	0.940	0.303	-1.300	0.186	-1.239	-1.517	0.099	-1.406	-0.208
1.454	0.034	0.956	1.745	-0.145	1.114	0.440	-1.126	0.324	-1.199	-1.488	0.028	-1.547	-0.335
1.294	-0.103	0.930	1.735	-0.071	1.271	0.572	-0.944	0.478	-1.135	-1.429	-0.018	-1.667	-0.450
1.125	-0.258	0.878	1.696	-0.023	1.409	0.693	-0.760	0.645	-1.047	-1.343	-0.036	-1.762	-0.549
0.950	-0.428	0.803	1.627	-0.001	1.522	0.801	-0.578	0.819	-0.941	-1.230	-0.027	-1.830	-0.629
0.775	-0.607	0.708	1.532	-0.006	1.610	0.890	-0.403	0.996	-0.819	-1.093	0.009	-1.869	-0.686
0.605	-0.792	0.595	1.411	-0.039	1.670	0.958	-0.240	1.171	-0.686	-0.936	0.070	-1.879	-0.718
0.444	-0.978	0.470	1.268	-0.098	1.701	1.001	-0.091	1.340	-0.547	-0.762	0.155	-1.860	-0.723
0.297	-1.159	0.336	1.105	-0.182	1.703	1.018	0.038	1.497	-0.406	-0.575	0.261	-1.813	-0.700
0.166	-1.331	0.199	0.928	-0.288	1.676	1.007	0.144	1.639	-0.268	-0.379	0.385	-1.739	-0.649
0.057	-1.489	0.062	0.740	-0.413	1.622	0.967	0.226	1.762	-0.137	-0.180	0.522	-1.641	-0.570
-0.029	-1.630	-0.068	0.545	-0.553	1.543	0.899	0.282	1.861	-0.019	0.019	0.668	-1.522	-0.466
-0.089	-1.750	-0.189	0.349	-0.704	1.442	0.805	0.309	1.935	0.084	0.211	0.819	-1.386	-0.337
-0.122	-1.845	-0.295	0.157	-0.863	1.322	0.685	0.309	1.982	0.167	0.393	0.970	-1.237	
-0.127	-1.914	-0.382	-0.028	-1.023	1.187	0.542	0.283	1.999	0.227	0.560	1.117	-1.079	
-0.106	-1.954	-0.448	-0.200	-1.180	1.041	0.379	0.230	1.988	0.262	0.709	1.253	-0.917	
-0.058	-1.966	-0.490	-0.355	-1.330	0.889	0.201	0.154	1.947	0.270	0.835	1.376	-0.756	
0.014	-1.947	-0.505	-0.490	-1.469	0.736	0.011	0.058	1.878	0.251	0.937	1.481	-0.601	
0.108	-1.901	-0.493	-0.602	-1.591	0.586	-0.185	-0.056	1.782	0.204	1.011	1.565	-0.455	
0.221	-1.827	-0.452	-0.688	-1.692	0.445	-0.384	-0.183	1.664	0.130	1.058	1.624	-0.324	
0.349	-1.728	-0.385	-0.746	-1.771	0.315	-0.581	-0.319	1.525	0.031	1.077	1.657	-0.211	

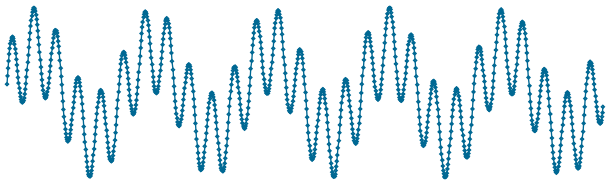


Figure 7-39 When the stream of data in Table 7-2 is displayed in graphical form the curve shown here is generated. Each diamond represents a data point from the table.

Figure 7-40 When the simple digital "moving average" technique is applied to the wave shown in the previous figure, this curve results. In this technique, each point is converted to the arithmetic average of the previous 99 data points from the original wave. This technique helps to smooth out the high-frequency oscillations and is a simple version of a digital high-frequency filter.

Figure 7-41 The wave shown is the result replacing each data point with the subtraction of adjacent data points of the wave shown in Figure 7-39. This simple technique removes most of the low-frequency oscillation and leaves most of the high-frequency component intact.

wavering of the baseline, which is still (but just barely) discernible.

Of course, the digital filtering algorithms used in a commercial EEG instrument are much more complex and sophisticated than the simple algorithms used in the previous examples, which show obvious defects. These examples are given to remind the reader that digital filtering involves complex mathematical manipulations on strings of digits as opposed to the behavior of analog circuits. Digital filters are designed to mimic the behavior of the RC circuits they have replaced. Digital filters have their own inherent imperfections depending on the specific filtering algorithms used.

Chapter

8 The Structure and Philosophy of the EEG Report

The essential purpose of recording and interpreting an EEG is to communicate information to the clinicians that will help guide the patient's care. When patients are referred for EEG testing, the referring physician often does not have the opportunity to review the EEG personally but usually relies completely on the report of the EEG to learn the findings and clinical implications of the test. Physical and time barriers and lack of EEG expertise may limit direct review of the record. Often, the EEG report becomes the *de facto* permanent record of the results of the study. For these reasons and others, considerable thought should be put into the content and wording of the EEG report, which is typically divided into a number of sections as described in this chapter.

IDENTIFYING INFORMATION

The EEG report generally starts with clinical identifiers, including the patient's name and date of birth, the name and location of the laboratory performing the study, the date of the study, and the name of the referring physician. Next, a brief clinical history is given that includes the general indications for which the study was ordered. This brief summary may reflect a combination of the clinical information that has been provided by the referring physician and additional history that has been obtained from the patient or family by the EEG technologist. The medications taken by the patient and the date of the most recent seizure may also be given, if applicable. This history is usually recounted in a concise fashion:

> This 60-year-old woman is referred because of episodes of confusion lasting 1 to 2 minutes that started approximately 1 month ago. There is a history of a left-sided stroke 3 years previously. The EEG is requested to rule out seizures.

This clinical description serves multiple purposes. First, it may alert the EEG technologist to the necessity of using specific recording techniques. For instance, absence seizures are suspected, the technologist may concentrate particularly on hyperventilation, perhaps even performing it twice. If temporal lobe epilepsy is suspected, the technologist may place extra electrodes over the temporal areas. Second, when the EEG report is completed, issues surrounding the clinical indications for the study are often addressed in the final "Clinical Correlation" paragraph at the end of the report. For example, if the referring physician suspected temporal lobe epilepsy, the EEG report may include additional pertinent negatives that directly address the clinical question posed, such as a comment that no epileptiform or slow-wave activity was noted in the temporal areas. Finally, the clinical history may also alert the technologist and the reader to special situations such as skull defects from previous surgeries, areas of the scalp that are inaccessible because of a bandage or other instrumentation on the head, or perhaps the fact that this is the fourth EEG in a sequence obtained on a patient in a coma.

TECHNICAL SUMMARY

Next, a technical description of the procedure used for the recording is provided. Because in any given laboratory most EEGs are recorded by a standard technique, this descriptive paragraph is usually standardized and only requires revisions when there are deviations from the laboratory's routine procedure. Because the technologist is responsible for the recording procedure, this paragraph is typically produced by the technologist. An example of a procedure description for a routine EEG is as follows:

> A 21-channel digital electroencephalogram was performed in the Clinical Neurophysiology Laboratory of *The Particular Hospital* at a sampling rate of 256 samples per second. The 10-20 international system of electrode placement was used and both bipolar, and referential electrode montages were monitored. Additional electrodes were placed at FT9 and FT10. The patient was sleep-deprived. No sedation was administered. The patient was recorded during the waking, drowsy, and sleep states. The total recording time was 41 minutes.

The next three sections represent the core of the EEG report and are produced by the interpreting electroencephalographer. These include a *description* of the

appearance and findings of the EEG, a summary of the findings or *interpretation* of the EEG (which may include an "abnormality list"), and a *clinical correlation* paragraph discussing the clinical implications of the findings. Each of these sections is now discussed in more detail.

DESCRIPTION

Here the electroencephalographer provides a concise description of the appearance of the EEG. Precise technical terms are used in this part of the report, including electrode names from the international 10-20 system and a variety of other EEG terminology. The goal of this portion of the report is to allow another electroencephalographer to visualize the appearance of the recording without actually having seen the original tracing. Provided with a well-written description, an experienced electroencephalographer should ideally be able to draw up the same abnormality list and clinical conclusions that would have been made had he or she personally reviewed the tracing. To provide this level of detail, the technical description paragraph may include EEG terminology that is not necessarily completely understandable by an internist or other general physician, or even in some cases by a neurologist who does not specialize in electroencephalography.

A good description allows a second electroencephalographer either to confirm the identifications of waveforms given in the interpretation paragraph or perhaps to disagree with them. For instance, if low-voltage sharps waves in sleep in the occipital areas with positive polarity were described as epileptiform activity, such a description may lead a second (more experienced) electroencephalographer to reject this interpretation and reidentify them as POSTS (positive occipital sharp transients of sleep), a normal variant (see Chapter 11, "Normal Variants in the EEG," for further discussion of POSTS). Formally, the description paragraph should consist of pure description of the visual appearance of the EEG; conclusions as to whether a described wave is normal or abnormal are not absolutely required in this paragraph and would usually appear in the interpretation section. In practice, for clarity's sake, some readers will flag findings as normal or abnormal in the description, especially if there are multiple findings, so that the message of the report is as clear as possible.

If appropriate to the EEG, the description is organized according to sleep state. Separate paragraph descriptions may be written for wakefulness, drowsiness, and sleep as needed. In the paragraph describing wakefulness, it is customary to quote the frequency and reactivity of the posterior rhythm, assuming it is identifiable. The amount of fast activity present during wakefulness is also commented on. A sleep paragraph would generally describe the presence of vertex waves and spindles if these are present. Any additional findings in each state would also be included in these sections. A typical description of normal wakefulness and sharp waves in sleep might include the following:

> AWAKE
> A moderate amount of 11- to 12-Hz medium-voltage rhythmic waves are seen posteriorly that suppress with eye opening. A small amount of symmetric 18–30 Hz low-voltage fast activity is seen anteriorly bilaterally.
>
> ASLEEP
> Stage II sleep is seen with vertex waves and a moderate amount of 14-Hz bicentral sleep spindles. Low- to medium-voltage sharp waves are seen occasionally in T8.

These two paragraphs describe the background activity during wakefulness and the presence of normal sleep elements. It is also clear that the right anterior temporal sharp waves were seen in sleep but not during wakefulness.

INTERPRETATION

This paragraph generally starts by clearly stating whether the EEG is considered normal or abnormal, assuming that this determination can be made. Terms such as "probably normal" or "probably abnormal" should be avoided whenever possible as they limit the usefulness of the report and are often found frustrating by the clinician who receives the report. Such noncommittal terms should only be used in the small minority of cases in which a determination of normality is not possible.

At this point in the report, it is also useful to give an abnormality list, an example of which follows:

> This EEG is abnormal due to the presence of
>
> 1. low- to medium-voltage spikes maximum in the right anterior temporal electrode during drowsiness and light sleep and
> 2. increased slow-wave activity over the right hemisphere during drowsiness, maximum in the right mid- and anterior temporal areas.
>
> No other asymmetries or epileptiform activity was noted.

Note that this portion of the report could have described "a discharge maximum in F8, with slow-wave activity maximum in F8 and T8." Because some future readers of the report may not be familiar with the official names of electrode positions and other technical EEG terminology, in this section of the report, it is preferable to use plain English terminology such as "right anterior temporal" rather than "F8." An abnormality list gives the report reader an opportunity to get a quick picture of the EEG findings by scanning the list. In reality, many report recipients may lack the time, patience, or expertise to go back and read the technical description

portion of the report. The goal is to write an interpretation paragraph that can stand alone and communicate the main findings of the EEG.

In both the description and interpretation paragraphs, the location and field of EEG events should be given with as much specificity as the tracing allows. For instance, if a spike has been found in the F8 electrode, it is insufficient to describe it simply as a "right temporal spike"; after all, there are *at least* three right temporal electrodes (anterior, mid-, and posterior temporal). In fact, a spike in the right anterior temporal area and a spike in the right midtemporal area may have significantly different clinical implications.

CLINICAL CORRELATION

The paragraph on clinical correlation is probably the most useful to the referring physician, yet it may be the most difficult to write, partly because many EEG findings are nonspecific (i.e., the patient's EEG findings usually do not establish a specific diagnosis). Therefore writing this paragraph is not as simple as stating "*Finding A* is present, which implies that the patient has *Diagnosis B*." The challenge of writing this paragraph lies in neither understating nor overstating the clinical implications of whatever findings are present. Rather, abnormal EEG findings may increase the chances that a diagnosis is present, yet they will rarely establish a particular diagnosis definitively. Thus, the clinical correlation paragraph must be worded with care. For example, it is well known that the presence of spikes in the EEG is not associated with a diagnosis of epilepsy 100% of the time. Many individuals go through life with spikes in the EEG, possibly never knowing they have them and never experiencing a single seizure. Nevertheless, the presence of spikes in the EEG makes it more likely that that person does have epilepsy compared with another person who does not have such spikes. The report should, therefore, communicate this concept of "increased risk" or "an association" rather than appear to be diagnosing epilepsy:

> Spikes in the right anterior temporal area suggest the possibility of a decreased seizure threshold in that area. Slowing over the right hemisphere suggests the possibility of an anatomical or functional (post-seizure) change in that region.

These sentences make it clear that the epileptiform abnormality seen in the EEG increases the chances that the patient has epilepsy, but does not establish the diagnosis. The slowing over the right hemisphere could have a number of possible causes, such as stroke, tumor, or the recent occurrence of a focal seizure from that hemisphere. The same type of thinking can be used when writing a clinical correlation for a normal EEG. Some electroencephalographers choose to signal to the referring clinician that a normal EEG tracing does not exclude the diagnosis of epilepsy.

The electroencephalographer must resist temptation to suggest further diagnostic studies in the report. For instance, in the earlier example, the combination of spikes in the right anterior temporal area associated with slowing over the hemisphere do suggest the possibility of a fixed right hemispheric lesion. The findings are potentially consistent with the patient having a right temporal lobe glioma (among other diagnoses). Why not suggest the patient undergo magnetic resonance imaging (MRI)? Because the electroencephalographer often does not know the patient firsthand, it may not be known that the patient had neuroimaging 4 months earlier. In such a case, a suggestion that an MRI be obtained may be confusing. Is the electroencephalographer saying that the MRI should be repeated? Decisions about obtaining neuroimaging are best made by the clinician who knows the entirety of the patient's story, including the EEG report.

Similarly, the EEG report should not suggest specific medications. The patient may have already been on a suggested medication in the past or could even be allergic to a suggested medication. Again, medication decisions should only be made by the treating physician who knows the whole story, not just the appearance of the EEG.

WHAT IS AN ABNORMAL EEG FINDING?

This question is not often asked directly, but when making the decision to label an EEG finding as normal or abnormal, the meaning of the word "abnormal" should be considered. As an initial thought, one might feel that a finding that is uncommon or rarely encountered should be considered abnormal. This is a poor definition for many reasons. For instance, in a given town, individuals who have red hair might be quite rare. Nevertheless, having red hair is not abnormal. The rarity of a given finding does necessarily indicate that it is abnormal. Rather, for the purposes of EEG interpretation, a finding should be considered abnormal if it is associated with *pathology* (disease or disability).

Consider the following hypothetical example: you have been reading EEGs for a year and have noticed a small group of patients with an atypical finding, for which you have invented the term "x-wave." Because x-waves look so unusual and so few patients have them, you are inclined to consider them abnormal. What threshold should be used to make a definite assertion of abnormality? You decide to conduct the following study: You collect 100 patients with x-waves and compare them to 100 patients of the same age who do not have x-waves. You then compare the two groups, looking to see whether one group has more epilepsy (or any other pathologic condition) than the other. You find that the rate of epilepsy is 1% in both groups. No other disorder is more common in one group than the other. Therefore, even though at first these x-waves did not "look right" to you, you finally conclude that x-waves represent a normal variant. Even though they are rarely seen, it is incorrect to call them abnormal because they are not associated with pathology or disease.

If, however, individuals with "x-waves" have more seizures, or mental retardation, or brain tumors than the 100 patients who lack them, then the fact of this association can be included in the reports of patients who have x-waves. In the real world, it is surprising how difficult it can be to perform this seemingly simple hypothetical study. Although it may not be particularly difficult to amass a large number of patients with one EEG finding or another in a busy EEG laboratory, the real challenge lies in assembling a "normal" comparison group. In reality, it is logistically difficult to obtain the recordings of 100 "normal" 50-year-olds or 100 "normal" schoolchildren. How does one choose 100 normal patients and then induce them to come to the EEG laboratory for a study? If 100 patients are invited and 50 refuse, are the remaining 50 still a "random" group, or may there be a difference between refusers and nonrefusers? How does one ethically handle the eventual occurrence of EEG abnormalities discovered in the so-called normal group?

The example of the hypothetical "x-wave" serves as a cautionary tale. It reminds us that even if an EEG tracing does not "look right" to us, we should not jump to the conclusion that it is abnormal. Properly, if abnormal, whatever does not "look right" should be categorizable as a known abnormality type. After it is categorized, the type of pathology that has been associated with that abnormality type can be cited in the Clinical Correlation section. If something does not "look right" in the EEG but it is not possible to categorize it as a known abnormality type, one must hesitate before calling it abnormal. The finding should still be described in the report, noting that it is of "uncertain clinical significance."

The histories of several of the known "normal variants" (see Chapter 11) have followed this course. Initially, because they "looked abnormal" and were recorded in individuals with epilepsy (because most EEGs are obtained in persons with epilepsy), they were presumed to be abnormal. Only later was it recognized that certain of these findings were present with significant frequency in the normal population, leading them to be reclassified as normal variants.

THE THRESHOLD FOR "ABNORMAL"

In the course of interpreting EEGs, from time to time the question arises as to whether a wave is sharp enough or an asymmetry asymmetrical enough to label abnormal. For instance, several waves are seen in the EEG, but it is not clear that they are sharp enough to be labeled as spikes. After a lot of thought, you estimate that the chances are fifty-fifty that the discharges represent an epileptiform abnormality. Is it best to err on the side of calling the discharge normal or abnormal? What is the potential impact of each type of judgment, which might represent a different type of error?

Imagine the following hypothetical story behind the patient described earlier with the "fifty-fifty spike." Although the EEG reader may not know it, the EEG was obtained because the patient had an episode of loss of consciousness. The referring physician is completely undecided as to whether the patient has had a syncopal event (a simple fainting spell) or an epileptic seizure. What would the consequences be of either type of reading error in this patient's EEG: calling a normal wave a spike or missing a true spike and calling the EEG normal?

First, consider the scenario in which the patient does not have epilepsy and the discharge is not really an epileptic spike, but you make the error of reporting that an epileptic spike is present. The referring physician reads your report and comes to the (erroneous) conclusion that the patient has epilepsy and considers starting an antiepileptic medication. The patient is labeled as having epilepsy and cannot drive for a period of time according to local law. In this case, the patient is now considered to have a potentially chronic disease where none exists and may be unnecessarily receiving antiepileptic medications. Perhaps the referring physician should realize that the finding of a spike in the EEG does not necessarily compel the diagnosis of epilepsy. Unfortunately, epileptiform findings in the EEG tend to carry more weight than they should among nonspecialists.

In the second scenario, the patient does have epilepsy but you make the "error" of calling the EEG normal. In this situation, however, the "opposite" sequence of the events does not necessarily occur. This is partly because it is known that individuals with epilepsy may still, from time to time (and sometimes repeatedly), have a normal EEG. The referring physician cannot exclude the diagnosis of seizure simply because of your report of a normal EEG. In reality, this patient does have a seizure disorder and your reading "error" may, at the very least, delay the diagnosis and incur any of the disadvantages to the patient consequent to that delay. If the patient does, in fact, have epilepsy, additional spells will probably occur, providing additional descriptions of the event that may be more clearly suggestive of seizure. There will also be additional opportunities to check the EEG. One may argue that this type of error could delay the ordering of neuroimaging that could reveal an important finding, such as a tumor, leaving the patient inappropriately untreated. In fact, if a tumor is present, additional symptoms or spells will probably occur, eventually prompting the ordering of neuroimaging studies.

Which type of error is worse, "overcalling" or "undercalling" the spike? In the scenario just described, the "overcalling reader" encountering 100 patients with a "50-50 spike" could be responsible for giving 50 patients an erroneous diagnosis of epilepsy. The "undercalling reader" will be responsible for delaying a true diagnosis of epilepsy in 50 people. Although neither is desirable, most would say that the first scenario in which fifty patients are given a diagnosis that they do not have is the less desirable outcome. Physicians are generally charged with alleviating disease, but in this scenario, the physician has effectively attributed to these patients a disease they do not have, possibly relegating them to unnecessary long-term therapy and other possible negative consequences. In the second scenario, the diagnosis of a seizure disorder, and its etiology may be missed or delayed.

Although this is not desirable, if disease is present, it will likely declare itself at some time in the future. If it does not, then no harm has been done. In summary, the type of error in which abnormalities are "undercalled" is usually the lesser of two evils: it is generally worse to *erroneously label* a patient with a disease he or she does not have than to *delay the diagnosis* of epilepsy in another patient, keeping in mind that a good physician knows that a normal EEG does not exclude the diagnosis of epilepsy. For this reason, it is recommended that readers lean to the conservative side in making judgments about findings that are not entirely clear. While giving an EEG tracing a normal interpretation, the electroencephalographer also has the option of including a comment in the EEG that reflects the difficulty of the decision:

Several waveforms seen in the O2 electrode attain sharp contours at times, but probably fall within the range of normal. Although these do not clearly represent an example of sharp waves, the question could be further investigated by obtaining a repeat study, if clinically indicated.

Although the reader should be able to categorize the large majority of EEG tracings as normal or abnormal, in the case of unclear findings the reader may wish to communicate to the referring physician that "a repeat study may provide additional useful information." This kind of wording and phrases such as "if clinically indicated" are chosen so as not to appear to compel the referring clinician to obtain a repeat study if one is not necessary when the patient's overall picture is considered.

THE CONCEPT OF INDEPENDENT OBSERVATIONS

Imagine that you are reading an EEG and you have a brief suspicion that there is slowing over the right hemisphere. By the time you get to the end of the EEG, however, you have convinced yourself that the asymmetry was not persistent enough and you have decided to make an overall interpretation of the EEG as normal. Before preparing the final report, however, by chance you come across the patient's MRI report, which describes a stroke in the right hemisphere. Should you reverse your decision and write your report so that it describes slowing in the right hemisphere? After all, the patient does have a stroke there! The answer is that you should not change your opinion of what the tracing shows based on the patient's clinical history. Often, clinicians refer patients for additional studies to confirm the importance of other findings and to see whether they "line up" with one another. In this case, it is possible that the referring physician has reviewed the MRI scan but doubts the presence of the stroke, thinking that the MRI was interpreted incorrectly. By ordering the EEG, the referring clinician was seeking an independent corroboration of the MRI finding. The purpose of the current EEG interpretation is not simply to echo the finding of what was found on a previous study. The ordering physician certainly has not referred the patient for EEG just to hear a repetition of the findings in the MRI report. Instead, to be useful, the results of your EEG interpretation should represent an independent observation.

To avoid the temptation of this type of error, many electroencephalographers do not read the patient's history before looking at the tracing. This is good practice. It also increases the sense of satisfaction the reader gets in picking up subtle right hemispheric slowing and learning that the patient has had a stroke on that side only after a conclusion about the EEG slowing has been reached. If the reader is already aware that the patient has a stroke on the right side before looking at the first page of the EEG, it may be psychologically difficult not to see slowing over the right hemisphere. The search for slowing over the hemisphere "where it's supposed to be" may absorb so much mental energy that other abnormalities may be missed because of the distraction. The best time to review the patient's history is after the EEG has been read, at least for the first time, and a preliminary conclusion has been reached. After the initial reading, it is not "dishonest" to review the record again in light of the patient's history to make sure that something has not been missed. If the interpretation is changed at this point in the process, the reader fully realizes that the finding was not evident to him or her without the additional clue of knowing the patient's history.

Chapter

9 The Abnormal EEG

An EEG is considered abnormal if it has findings known to be associated with a pathologic or disease state. As discussed in Chapter 8, "The Structure and Philosophy of the EEG Report," this distinction is designed to prevent the interpreter from calling an EEG abnormal simply because it includes a finding that "looks unusual" or is uncommon, because uncommon findings may not be abnormal.

When deciding which types of studies should be labeled abnormal, it is useful to consider the referring physician's purpose in ordering an EEG. Ideally, the clinician is confronted with a patient with a particular clinical picture and has formed a list of possible diagnoses, also called the "differential diagnosis," that might explain the patient's findings. If the result of an EEG could increase or decrease the chances that one or more entities from the differential diagnosis list is the correct diagnosis, then it may be reasonable to obtain the test. However, if the EEG is not likely to have an impact on the probability of any of the diagnoses on the list, then the test is probably not indicated. It is usually not a good idea to obtain an EEG just "to see what it looks like" without a specific question in mind that the EEG could potentially answer. A related concept is that an EEG is indicated if there is some likelihood that the result will affect the patient's treatment. Simply reporting that the EEG looks "odd" or "unusual" will not likely be helpful to the referring physician. Rather, an EEG should only be considered abnormal if it contains a finding that has some association with a disease or an abnormal state.

The majority of EEG abnormalities do not specifically lead to a single diagnosis. Only a small minority of EEG abnormalities are associated with a short enough list of disease entities that they can be considered "specific" for one diagnosis or another. The majority of EEG findings are diagnostically nonspecific and are associated with a list of disease states that is lengthy and diverse, so much so that specific diagnoses usually cannot be suggested based on the EEG result. An example of a dramatically abnormal but nonspecific EEG abnormality is generalized delta slowing, a finding that is associated with so many types of abnormal states (e.g., coma, post-seizure state, meningitis, anesthesia) that the clinical implications of the finding can only be stated in the broadest terms in the EEG report. The utility of the EEG comes in combining the EEG result with the patient's history and other findings to narrow the differential diagnosis.

The referring physician is usually best placed to put the EEG findings and the clinical story together to arrive at a clinical conclusion. Without the clinical history, the EEG is a considerably less powerful tool. Even when a clinical history has been submitted with the EEG request, the reader of the EEG should hesitate before suggesting specific clinical diagnoses. The submitted history may be incomplete and often lacks details such as the physical examination or certain laboratory or imaging findings. When reading the submitted history, the electroencephalographer usually cannot know whether that history is complete. For these reasons, the interpreting physician should resist the temptation to make specific diagnoses based on the EEG results. Rather, a list of diagnoses that have been associated with the observed abnormalities, a sort of "EEG differential diagnosis," should be given at the end of the report, if feasible. The discussion given in the final clinical interpretation section of the report should take into account the clinical history provided, but the EEG differential diagnosis offered should not be limited by that history.

EEG abnormalities can be categorized in a variety of ways. Abnormalities may fall into the categories of 1) abnormal expressions of normally occurring rhythms (e.g., asymmetries of normal rhythms), 2) inherently abnormal rhythms (e.g., "slow" delta and theta rhythms in an adult who is awake), 3) certain repetitive or *periodic patterns* (e.g., burst-suppression patterns), 4) epileptiform abnormalities (spikes, sharp waves, etc.), and 5) abnormal "super-architecture" (e.g., abnormal sleep state cycling). These various abnormality types are discussed in the following sections. The first two abnormality families, abnormalities of normal rhythms and abnormal rhythms, are discussed by frequency range, starting with slow activity. Basic epileptiform abnormalities are discussed in this chapter. Various epilepsy syndromes and the EEG findings associated with them are discussed in Chapter 10, "The EEG in Epilepsy."

ABNORMALITIES OF SLOW-WAVE (DELTA AND THETA) ACTIVITY

Descriptive Parameters for Slow Waves

Slow-wave abnormalities can be defined in multiple domains. The most obvious descriptor of a slow wave is its location. A slow wave may occur focally, such as in the left anterior temporal area or the right occipital area. Slowing may occur in broader regions, such as in the right posterior quadrant, in "anterior brain regions," or over a whole hemisphere (see Figures 9-1 and 9-2). Such regional slowing can be considered a subset of focal slowing. Finally, slowing may also be diffuse or generalized.

Slow waves may be rhythmic or irregular (nonrhythmic). When there is a tendency to rhythmicity but the waves cannot be considered truly rhythmic, the intermediate term "semirhythmic" may be used (see Figure 9-3). Slow waves can be of varying amplitude. If slowing is only observed in a certain sleep stage, such as drowsiness or slow-wave sleep, this fact should be described. Slow waves may occur intermittently (in brief runs or bursts) or continuously (in long, continuous runs with few pauses). These different parameters are worth remembering and should usually be included in the written description of slow-wave activity.

Parameter	Examples
Rhythmicity:	*Irregular* versus *semirhythmic* versus *rhythmic*
Amplitude:	*High voltage* versus *low voltage*
Sleep Stage Specificity:	Seen in *wakefulness* versus *drowsiness* versus *sleep*
Continuity:	*Intermittent* versus *continuous*
Localization:	*Focal* versus *regional* versus *generalized*

The parameters can be remembered using the mnemonic "RASCL." Certain combinations of these parameters can define specific, well-described slow-wave abnormalities. For instance, a slow wave that is frontal, occurs intermittently, is rhythmic, and is seen during wakefulness has been given the specific name FIRDA (frontal intermittent rhythmic delta activity), discussed further later in the chapter.

Focal Slowing

Focal slow waves are the classic sign of a lesion in the cerebral hemispheres. Before the era of modern neuroimaging, the electroencephalogram was an important tool for the localization of cerebral tumors. Today the

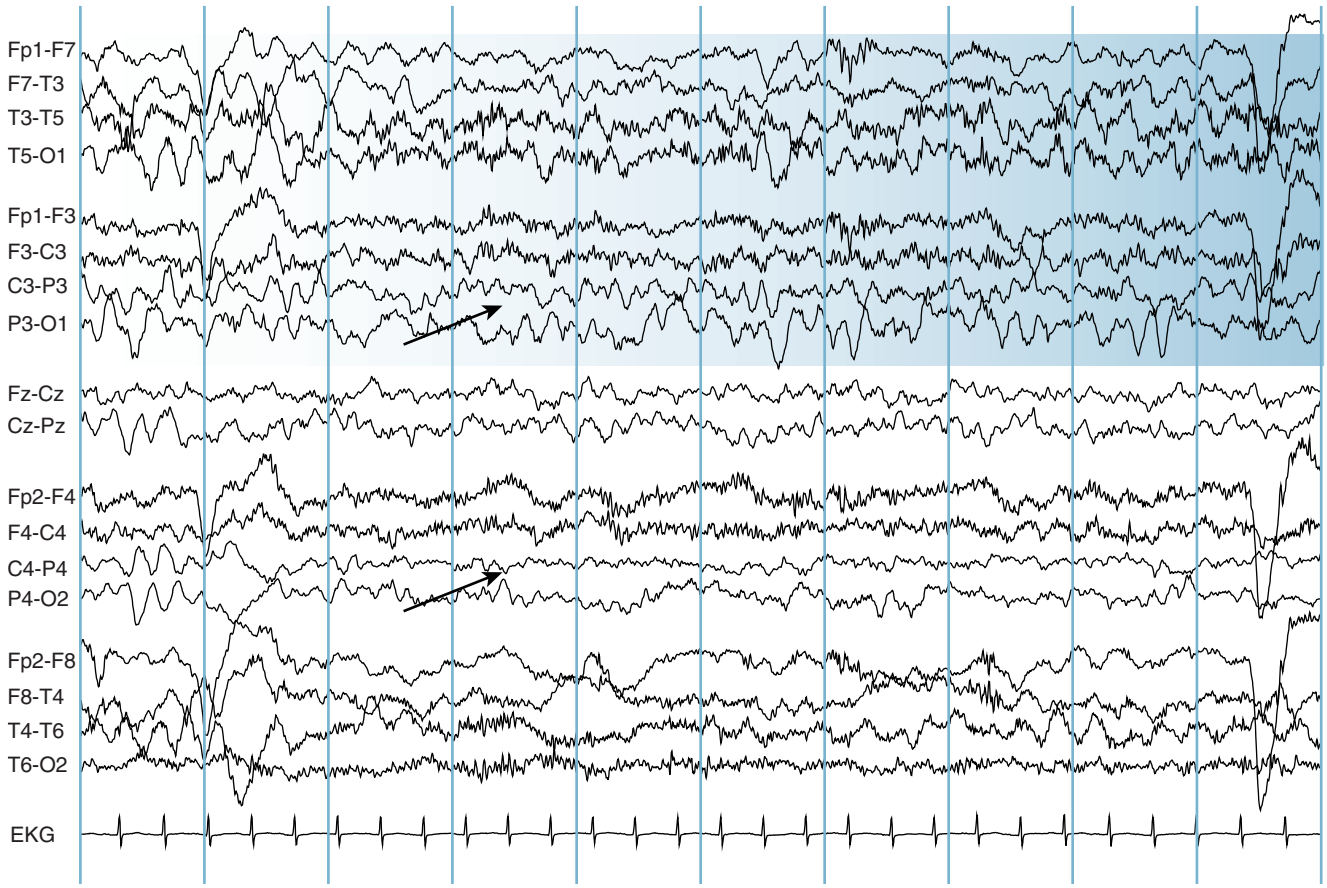


Figure 9-1 This page of waking EEG shows slowing in the delta range occurring predominantly over the left hemisphere. Compare the left hemisphere channels (shaded) to the right hemisphere (bottom eight channels, unshaded). Waves in both the theta and delta ranges are more plentiful and of higher voltage over the left. The difference is especially prominent comparing the left (top arrow) and right (bottom arrow) parasagittal areas.

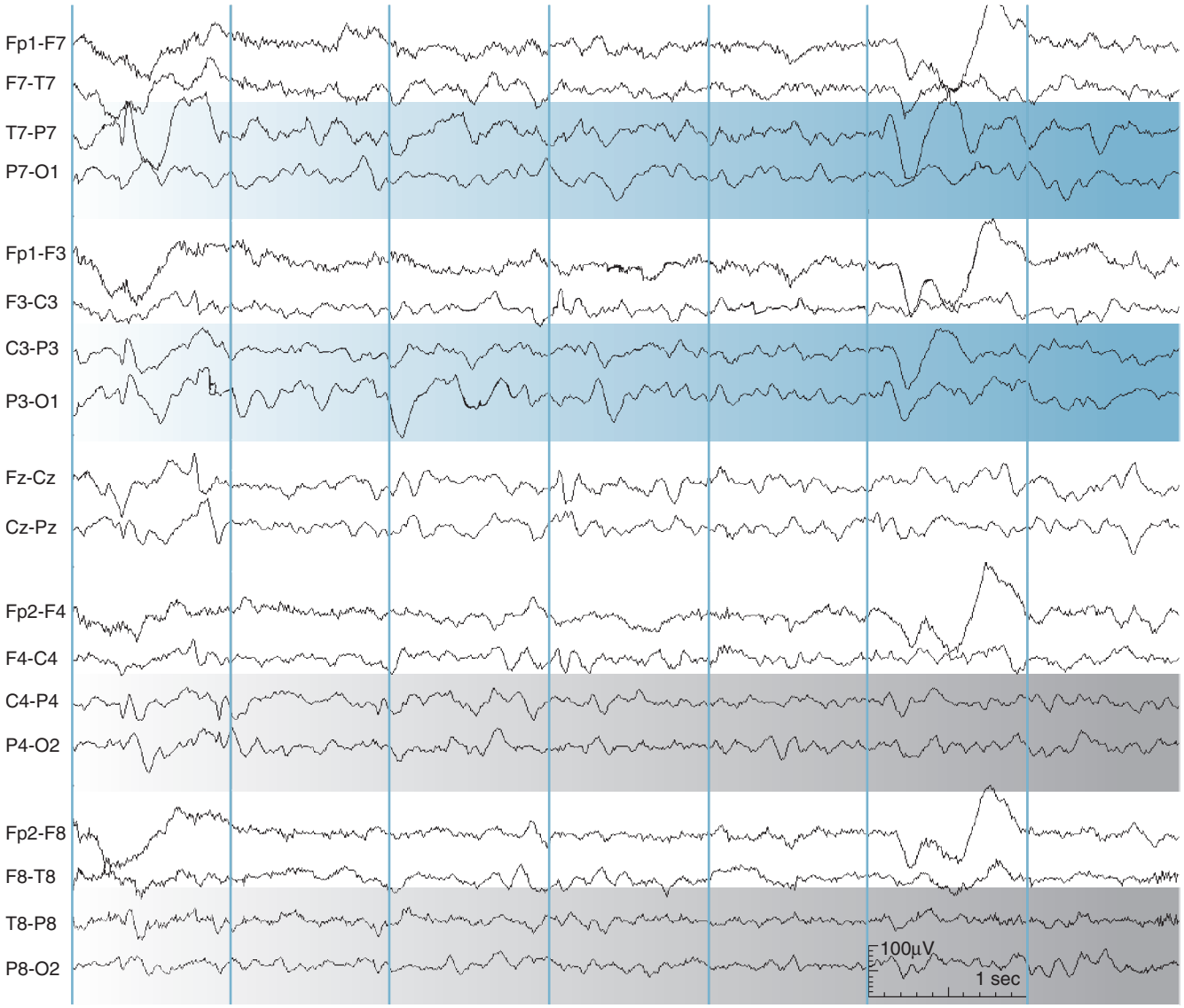


Figure 9-2 A mild but abnormal increase in slowing is seen in the left posterior quadrant (blue rectangles) compared with the right posterior quadrant (gray rectangles) during drowsiness.

magnetic resonance imaging (MRI) scanner has moved to the forefront in localizing tumors, but the EEG may still play an important role in identifying certain types of focal lesions that may or may not be evident on neuroimaging. The EEG has the advantage over imaging studies of identifying areas of electrical abnormality which may be "nonanatomical," that is, functional abnormalities of the brain that may not be visible on an MRI or computed tomography (CT) scan. Examples of "nonanatomical" slowing may include postictal slowing (see Figure 9-4), slowing from trauma that has not caused an MRI lesion (see Figure 9-5), or even migraine (discussed subsequently).

Focal slow waves may mark an area of previous, rather than acute, injury. Brain lesions that cause focal slowing in the absence of epileptiform activity may not necessarily be prone to seizures. Figures 9-6 and 9-7 show a left temporal slow wave abnormality; the perinatally acquired lesion causing the slow wave is shown in Figure 9-8. Figure 9-9 shows a subtle right occipital slow wave brought on by hyperventilation. If asymmetric slowing is only seen during hyperventilation, especially when the asymmetry is relatively mild as seen in this example, it is less likely to be associated with pathology than spontaneously occurring slow-wave asymmetries. Slow-wave asymmetries that alternate sides, whether spontaneous or elicited by hyperventilation, are much less likely to be clinically significant.

Focal slow-wave abnormalities have generally been associated with deeper lesions located at the level of the deep white matter (as opposed to more superficial gray matter lesions), although exceptions to this rule do occur. As discussed next, more superficial abnormalities of cerebral cortex are classically associated with decreases in beta activity.

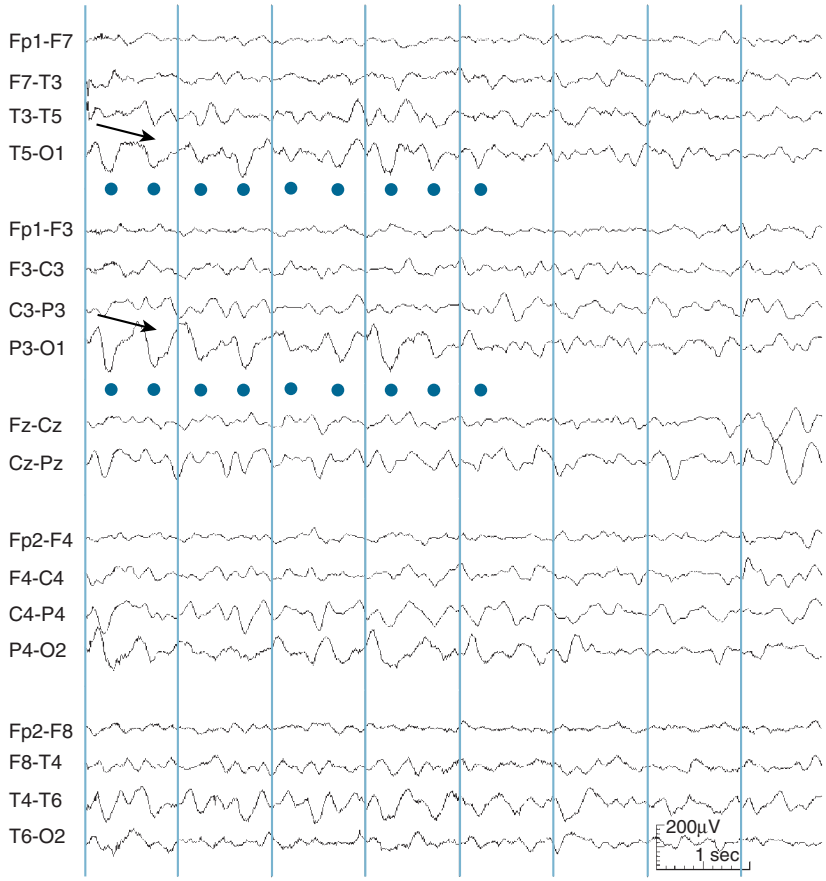


Figure 9-3 Although not perfectly rhythmic, the slow waves seen in the left occipital (O1) channels (arrows) are not completely irregular (dots). Some of the unpredictability of the appearance of these slow waves is due to intermixing with other rhythms. Waves of this intermediate degree of rhythmicity can be termed *semirhythmic*.

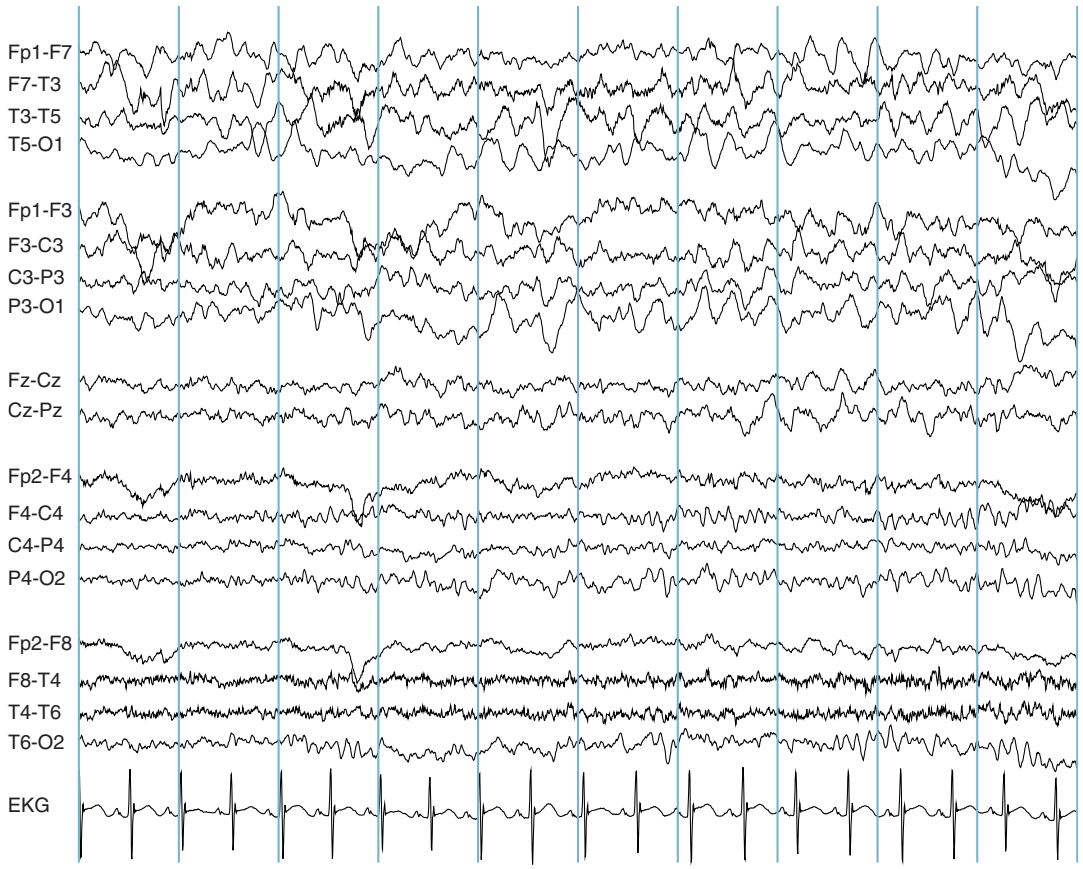


Figure 9-4 Significant slowing is evident in the top eight EEG channels (left hemisphere) compared with the bottom eight EEG channels (right hemisphere). The fact that this slowing represented a postictal change after a seizure was confirmed by demonstrating clearance of the slowing on a repeat EEG 2 months later. Depending on the nature of the patient and the intensity and duration of the seizure, postictal slowing may last from several seconds to as long as four weeks.

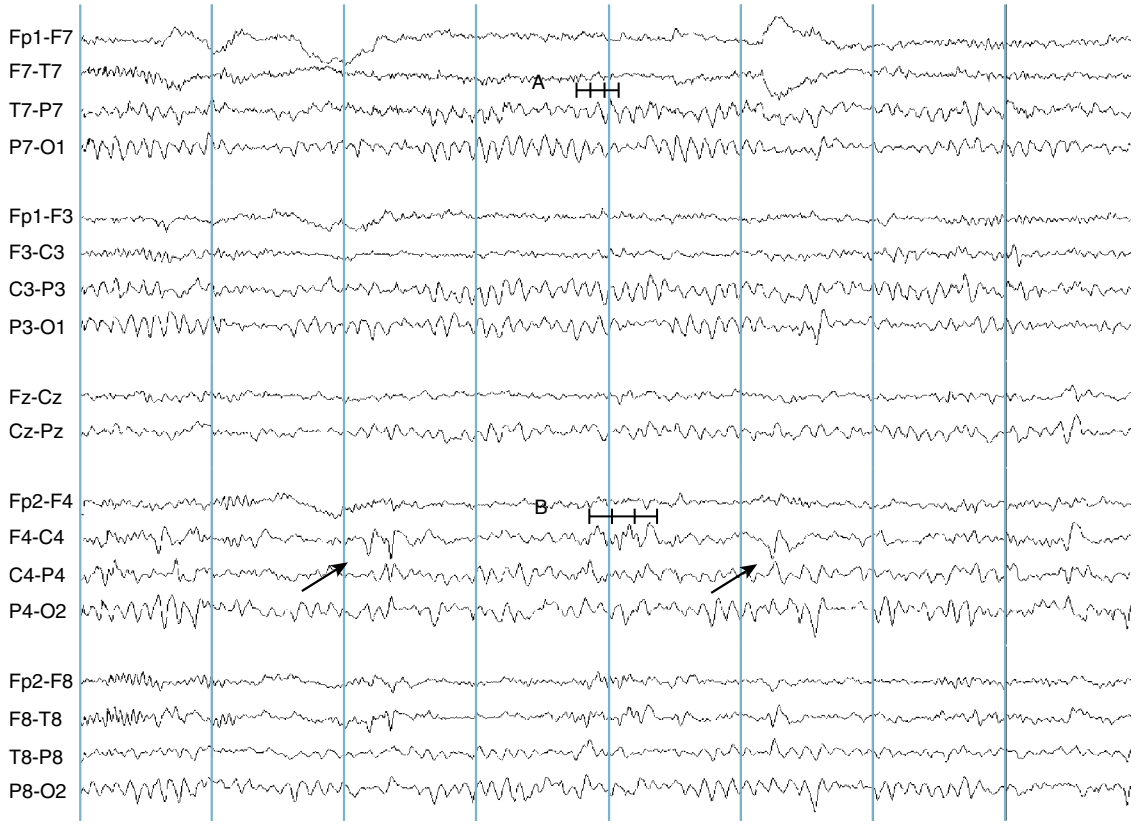


Figure 9-5 Slowing and sharp waves are seen over the right hemisphere in this patient following a brain contusion. Note the sharp waves (arrows) over the right hemisphere. Normal alpha range activity with short wavelengths (A scale) is seen on the left compared with slower, theta range activity with correspondingly longer wavelengths (B scale) on the right.

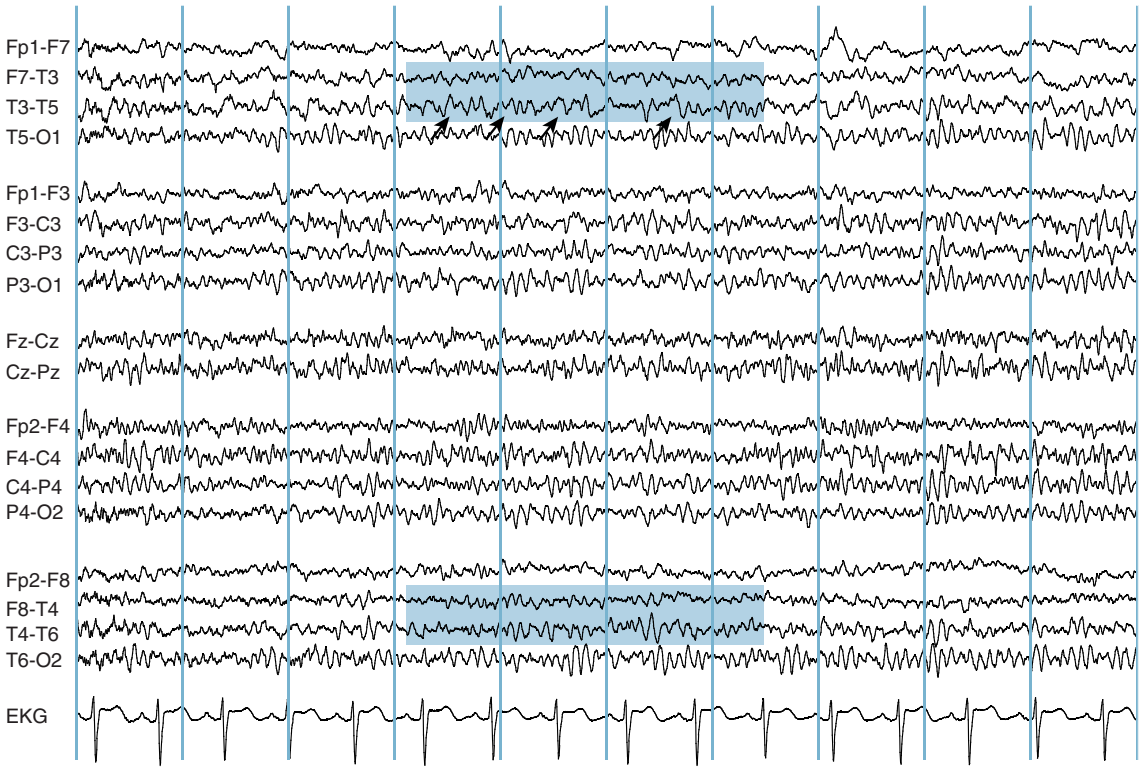


Figure 9-6 Close comparison of the temporal areas (shaded areas) shows an asymmetry of slow activity with increased theta waves on the left as a result of an old perinatal injury to the left temporal lobe tip in a 17-year-old girl. The arrows indicate individual theta waves in the left temporal area. The patient's scan is shown in Figure 9-8. The lesion resulted in complex partial seizures.

Intermittent Rhythmic Delta Activity

Frontal Intermittent Rhythmic Delta Activity and Occipital Intermittent Rhythmic Delta Activity

The term *intermittent rhythmic delta activity* (IRDA) refers to rhythmic delta activity occurring in brief bursts, usually lasting no longer than a few seconds, typically located either frontally or occipitally (see Figure 9-10). In some examples the bursts can be more generalized (see Figure 9-11), and occasionally they can be asymmetrical. IRDA is a pattern typically seen in wakefulness and is usually associated with processes of mild to moderate severity. Keeping in mind that the patient generally must attain some level of wakefulness to manifest frontal (FIRDA) or occipital (OIRDA) IRDA, these patterns would not be expected in patients whose recordings are restricted to more deeply sedated or comatose states.

One important feature of FIRDA and OIRDA is that these abnormal patterns do not suggest a specific localization. Surprisingly, the tendency for IRDA to occur either frontally or occipitally is not dictated by an anterior or posterior location of the patient's lesion but rather by the patient's age. Up to approximately 10 years of age, IRDA tends to occur in the occipital areas. By the early teenage years, IRDA tends to occur frontally. One reason that it is important to correctly identify an example of rhythmic slowing as IRDA is to avoid being trapped into inappropriately using IRDA as a "falsely localizing sign." As implied earlier, FIRDA is not particularly associated with frontal lesions, and OIRDA is not particularly associated with occipital lesions; either can be associated with anterior or posterior brain abnormalities. Indeed, the location of the IRDA tells us more about the age of the patient than the location of the lesion.

It is interesting to note that the predilection of the hyperventilation response for the occipital area in children and for the frontal area in adults parallels the age dependence of FIRDA and OIRDA. In older patients, the hyperventilation response may mimic the appearance of FIRDA, and in younger patients it mimics OIRDA, which is to say that younger children tend to manifest the intermittent rhythmic slow of hyperventilation in the occipital areas.

FIRDA and OIRDA are etiologically non-specific, and can be caused by a variety of toxic, metabolic, and other processes that affect the central nervous system. Their presence signals some type of cerebral disturbance, focal or diffuse.

Temporal Intermittent Rhythmic Delta Activity

Temporal IRDA (TIRDA) should be considered separately from FIRDA and OIRDA. The presence of intermittent, rhythmic trains of delta activity in either temporal lobe has been associated with temporal lobe epilepsy (see Figure 9-12). Furthermore, the side of the TIRDA, which is usually unilateral, indicates the side of the lesion if one is present. This localizing property of TIRDA distinguishes it from the major types of IRDA (FIRDA and OIRDA). Thus, unlike FIRDA and OIRDA, TIRDA is considered a potentially epileptogenic abnormality and has localizing value.

Occipital Intermittent Rhythmic Delta Activity in Childhood Absence Epilepsy

A specific type of occipital intermittent rhythmic delta activity is seen in children with childhood absence epilepsy. Intermittent rhythmic 3-Hz delta activity, similar to OIRDA, can be seen in what can be either brief or prolonged runs in the occipital area in children who have childhood absence epilepsy. This type of rhythmic delta activity distinguishes itself from typical examples of OIRDA in that the runs can be quite prolonged, lasting many seconds.

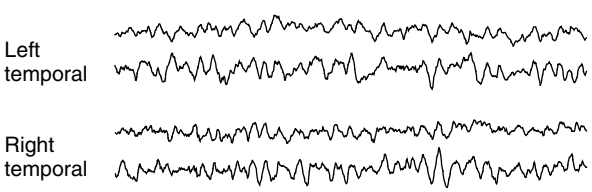


Figure 9-7 Close-up views of the left and right temporal channels taken from the shaded areas of the previous figure are shown. Note the mixture of slow waves with wider bases in the top channels compared with the faster alpha rhythms with narrower bases in the bottom channels.

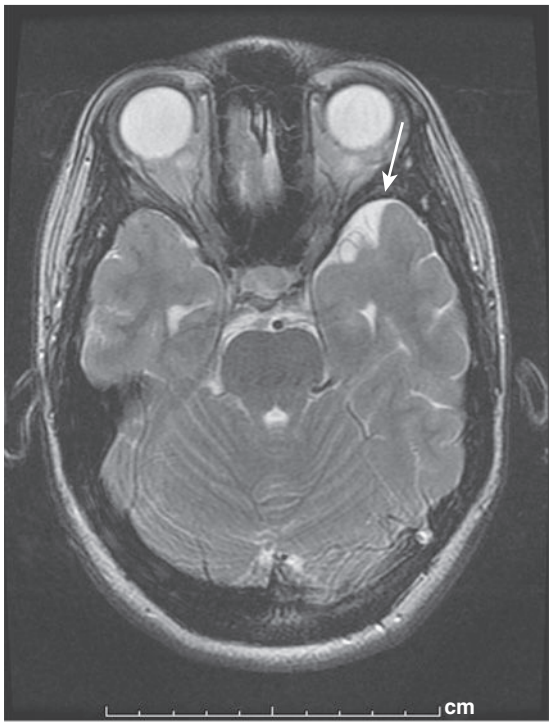


Figure 9-8 This is the T2-weighted magnetic resonance imaging (MRI) scan of the patient whose EEG is shown in the previous two figures. In this MRI sequence, the cerebrospinal fluid appears white. Note the loss of volume in the left temporal pole (arrow), which is responsible for the EEG asymmetry noted in the previous figures.

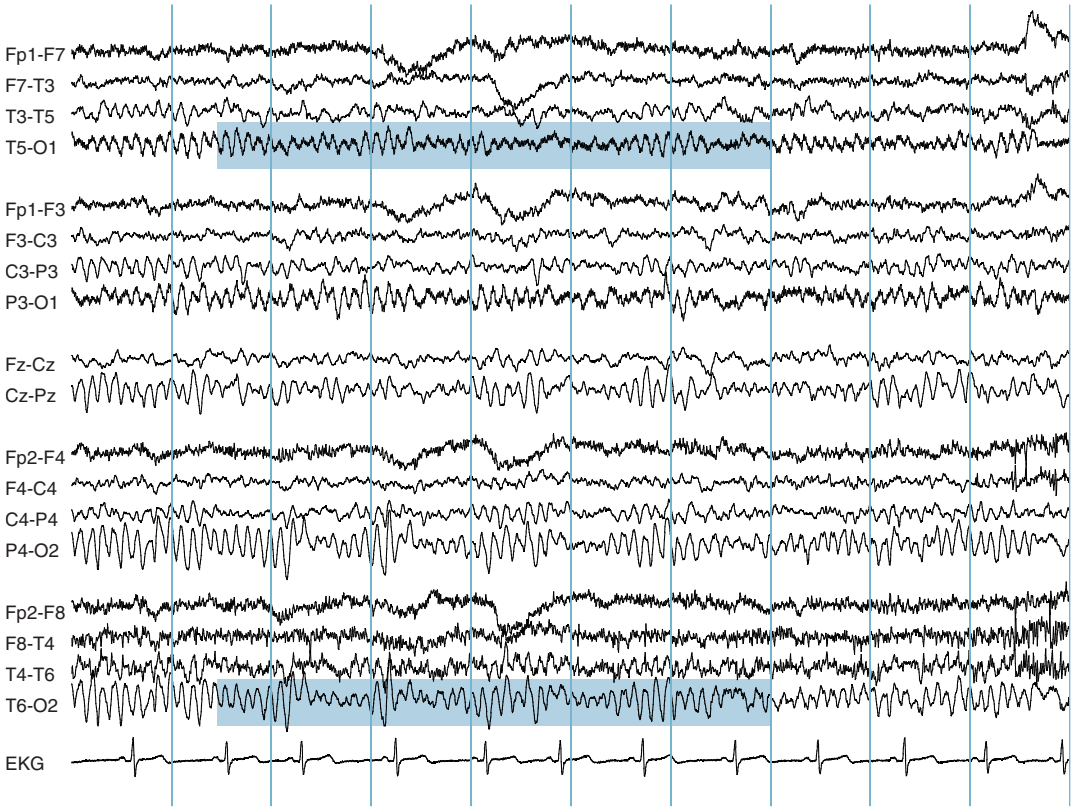


Figure 9-9 A subtle increase in slow-wave activity is seen in the right occipital area compared with the left (compare shaded areas). The posterior rhythm appears against a flat baseline in the T5-O1 channel (upper shaded area), but the posterior rhythm rides up and down on a low voltage wave in the T6-O2 channel (lower shaded area). The same comparison can be made between the P3-O1 and P4-O2 channels.

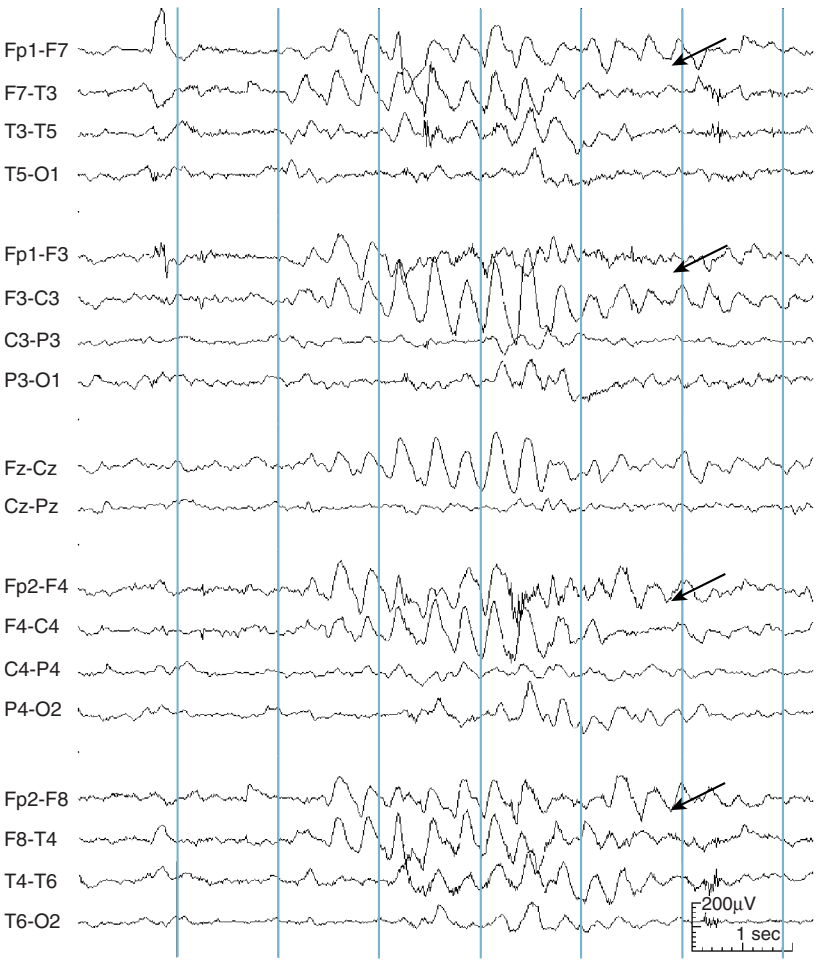


Figure 9-10 Frontal intermittent rhythmic delta activity (FIRDA) consists of rhythmic runs of delta activity in the frontal areas of varying duration (arrows). Although FIRDA appears frontally, the finding does not necessarily imply a frontal pathology (see text).

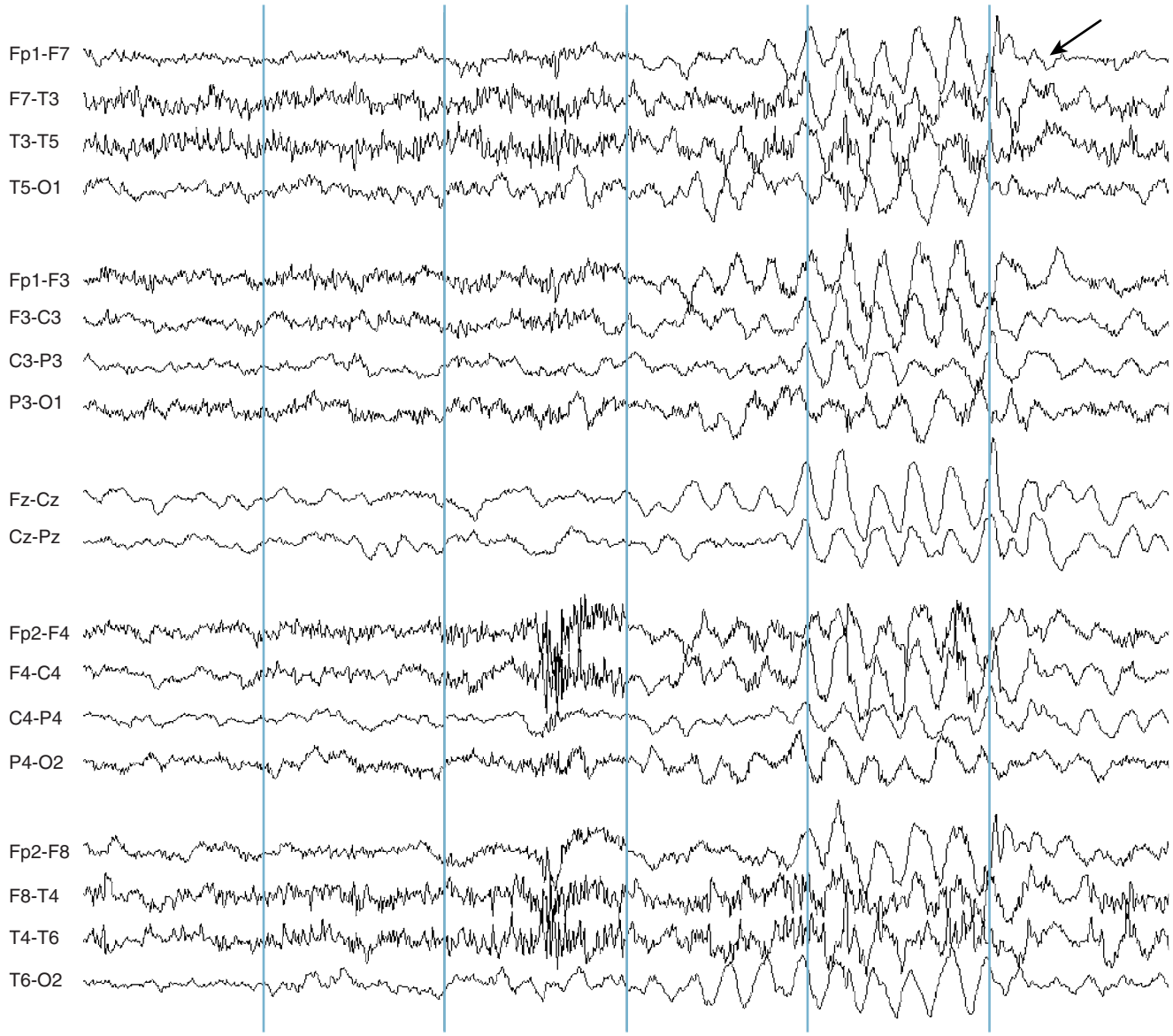


Figure 9-11 Compared with Figure 9-10, this burst of intermittent rhythmic slow (arrow) has a more diffuse (rather than frontal) distribution. It is seen equally well in the anterior and posterior channels.

Generalized or Diffuse Slowing

In general, theta and delta rhythms are not expected in the waking adult EEG. In younger patients; however, theta waves may be seen in the normal waking EEG. The range of normal posterior rhythms in childhood may serve as a useful reminder that theta frequencies are commonplace in the EEG of the awake child (see Table 2-2 in Chapter 2, "Visual Analysis of the EEG)." For instance, in individuals young enough that a posterior rhythm of 7 Hz is considered normal, other 7-Hz rhythms may also be seen elsewhere in the normal waking EEG.

The determination that an adult EEG is abnormal simply because theta waves are present is made more difficult by the fact that it is normal for theta waves to appear with drowsiness. Therefore, in one way or another, the electroencephalographer must establish that a patient is not simply drowsy at the time that theta waves are seen to label them abnormal. Interpreting an EEG as abnormal on the basis of the presence of theta waves that are, in reality, related to drowsiness would constitute a significant error in interpretation. Indications that a patient is probably awake would include the presence of a posterior rhythm, temporalis or frontalis muscle artifact, eyeblink artifact, or conversation. The technologist can document (or force) alertness by having the patient count or perform some other task to exclude drowsiness as an explanation for observed slow-wave activity.

The list of pathologic states that can cause diffuse slow-wave activity is long and includes almost any abnormal state that can cause a diffuse cerebral disturbance. The most common causes are postictal (postseizure) states, postanoxic states, diffuse traumatic injuries,

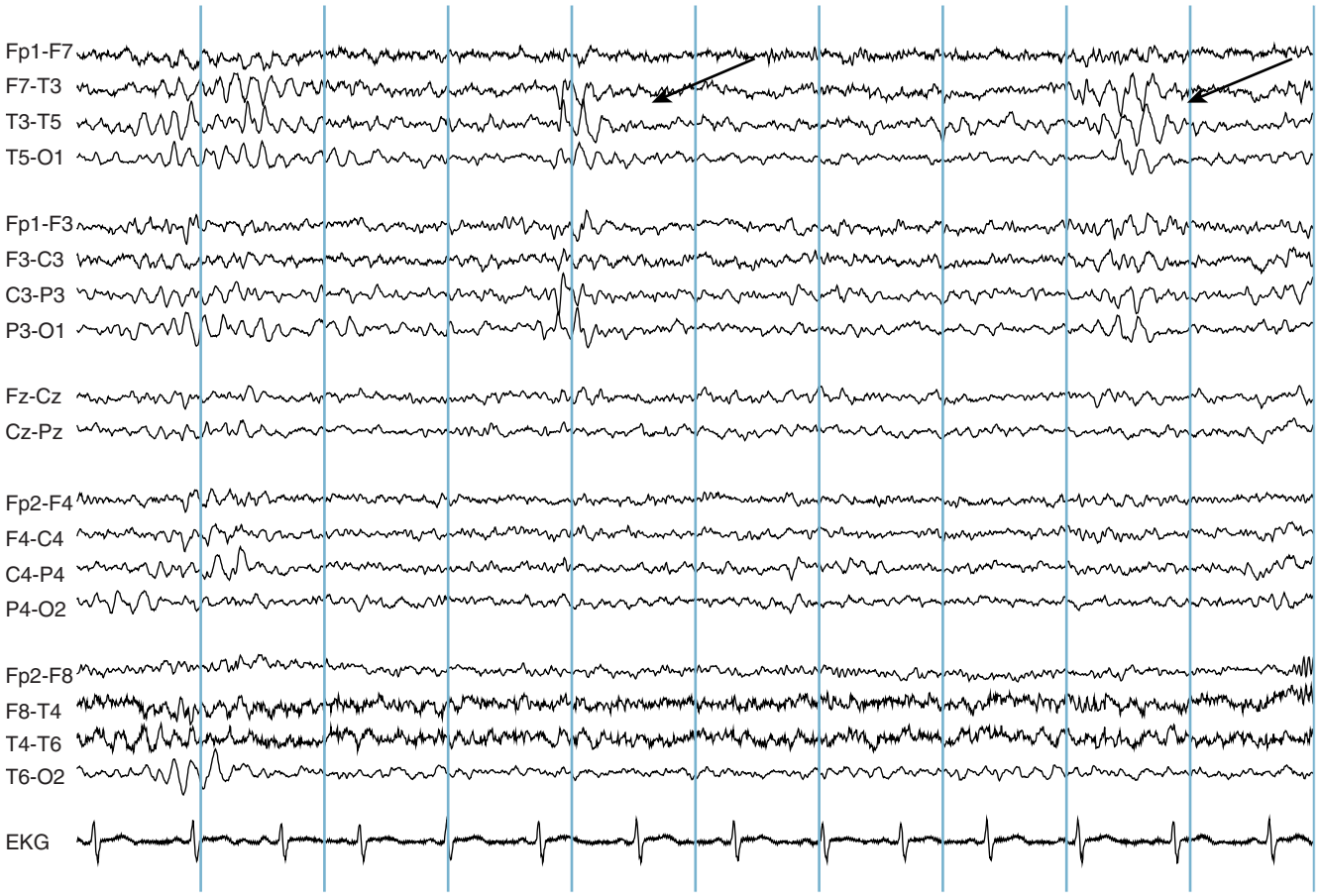


Figure 9-12 Temporal intermittent rhythmic delta activity (TIRDA) is seen in a patient with temporal lobe epilepsy. This rhythmic delta activity is seen best in the left temporal area (arrows), although the left parasagittal area is involved as well. In this example, some of the theta waves are sharply contoured, which is not a necessary feature of TIRDA.

infectious or inflammatory processes of the nervous system, toxic states related to drugs or other metabolic derangements (hepatic, renal, etc.), and a large number of other processes that have the potential to affect large portions of the cerebrum. Most of the many possible causes of coma are also on the list of possible causes of diffuse slowing in the EEG. For this reason, it is not feasible for the electroencephalographer to give a specific differential diagnosis for this pattern in the report. Often the interpretation will summarize the possible etiologies as a "diffuse cerebral disturbance" or a "diffuse encephalopathy." The interpretation may also specifically discuss any clinical entities that have been questioned in the clinical history. EEG patterns in coma, many of which consist of slow-wave patterns, are discussed in further detail in Chapter 12, "Electroencephalographic Patterns in Stupor and Coma."

Migraine

Because it is such a common disorder, the role of the EEG in migraine merits a separate discussion. A large number of abnormalities have been described in patients with common forms of migraine ("common" or "classic" migraine) *between* migraine attacks, so-called interictal EEG abnormalities in migraine. Many of these claims have been challenged because of the lack of well-controlled studies. The main problem is that the high frequency of migraine in the general population increases the chance that an individual who has any particular EEG abnormality may also incidentally have migraine. Some authors have claimed that focal slowing, and even epileptiform activity, are seen with increased frequency interictally in migraine patients. The "high-frequency photic response" (the ability to maintain a photic driving response at particularly high flash stimulation frequencies) has also been described in migraine patients. None of these phenomena, however, has been clearly proved to have an incidence in migraine patients above that seen in the general population. Because there are no EEG abnormalities that have proved to be useful in helping to diagnose uncomplicated migraine patients between attacks, EEG is not recommended as part of the routine evaluation of migraine patients. The EEG may be indicated, however, in the rare cases in which there is a question that a headache symptom may represent an epileptic seizure phenomenon.

Complicated Migraine

Focal slowing, either rhythmic or irregular, may be seen during the course of complicated migraine attacks. Figure 9-13 shows an example of high-voltage irregular slowing over the left hemisphere during an attack of hemiplegic migraine (a rare type of complicated migraine) in a 12-year-old girl. Confusional migraine, another rare type of complicated migraine that occurs predominantly in children, may cause bilateral slowing in the EEG. In some cases, migraine with visual aura can cause slowing during an attack, particularly in the posterior quadrants, although in many cases the EEG is not particularly remarkable during attacks.

In summary, EEG abnormalities, usually in the form of slowing, can be recorded in some patients during migraine attacks (particularly during complex migraine attacks). Whether certain abnormalities are characteristic of the EEG *between* attacks in migraine patients remains an unsettled question.

"Sharp Slow"

At times, a hybrid between sharp waves and delta or theta waves is seen consisting of sharply-contoured slow waves. An example of these slow waves with sharpened peaks is shown in Figure 9-14. The informal term for this type of wave is *sharp slow*. Whether such sharply contoured slow waves represent a true epileptiform abnormality or simply a variant of slow waves is unclear; some may be truly epileptiform and some may not. A variety of claims have been made regarding the significance of sharply contoured slow waves, including their association with vascular abnormalities such as small strokes, although none of these associations has been definitely proven.

Abnormalities of Alpha Activity

Alpha range frequencies make their most dramatic appearance in the EEG in the form of the posterior rhythm. Asymmetries of the posterior rhythm are common, and certain asymmetries are even expected: the posterior rhythm is usually of higher voltage over the right, nondominant hemisphere (see Figure 9-15). However, complete absence of the posterior rhythm may occur in a minority of otherwise normal individuals. It is also true that the posterior rhythm may be absent in individuals with brain injuries or other abnormalities, but in such cases the EEG usually shows other abnormal features. Therefore, an EEG whose only remarkable feature is absence of the posterior rhythm should be considered normal. Asymmetries of the posterior rhythm are discussed in more detail in Chapter 2, "Visual Analysis of the EEG."

In some patients, alpha rhythms take up much of the posterior quadrants of the EEG during wakefulness. This occurs either when the field of the posterior rhythm extends far forward or when the posterior rhythm blends with mu rhythms, a normal variant rhythm seen in the central areas that may also be in the alpha range. Mu rhythms are discussed in more detail in Chapter 11, "Normal Variants in the EEG."

ABNORMALITIES OF BETA ACTIVITY OR "FAST ACTIVITY"

Excess Fast Activity

Unusually high voltage or plentiful beta activity is the most frequently encountered abnormality of fast activity in the EEG. By far, the most common explanation

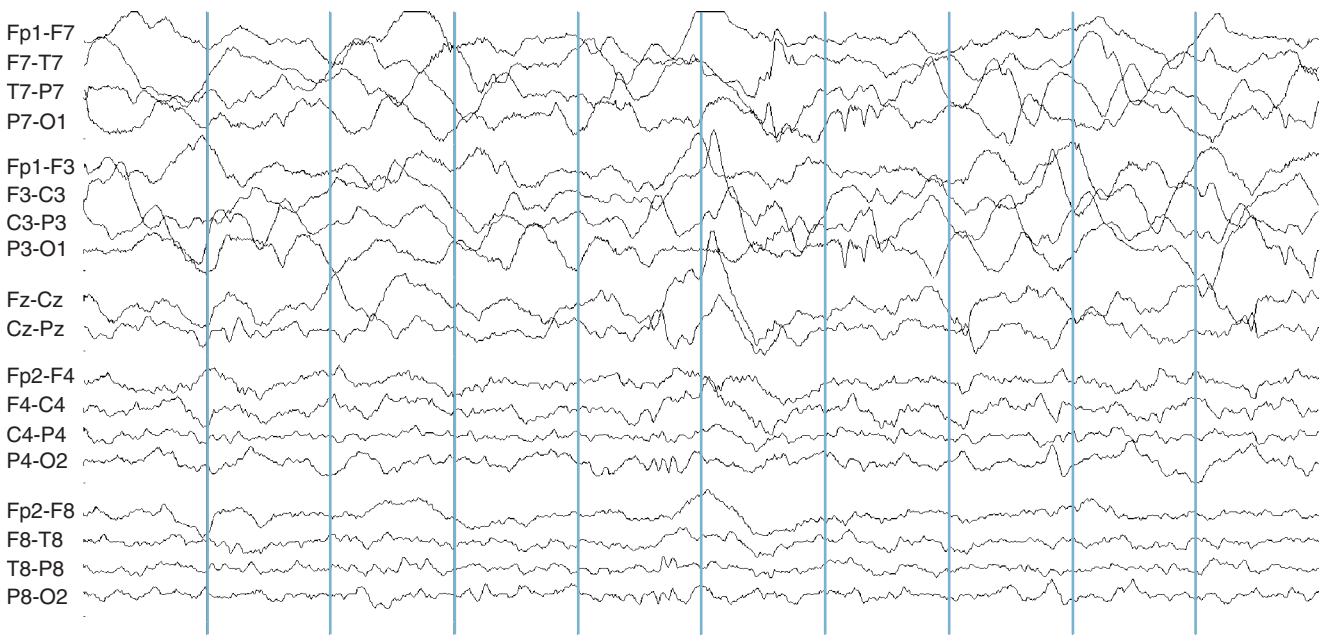


Figure 9-13 A 12-year-old girl recorded during an episode of hemiplegic migraine. Note the high voltage slowing over the left hemisphere (top eight channels) compared with the relatively more normal-appearing activity over the right hemisphere (bottom eight channels).

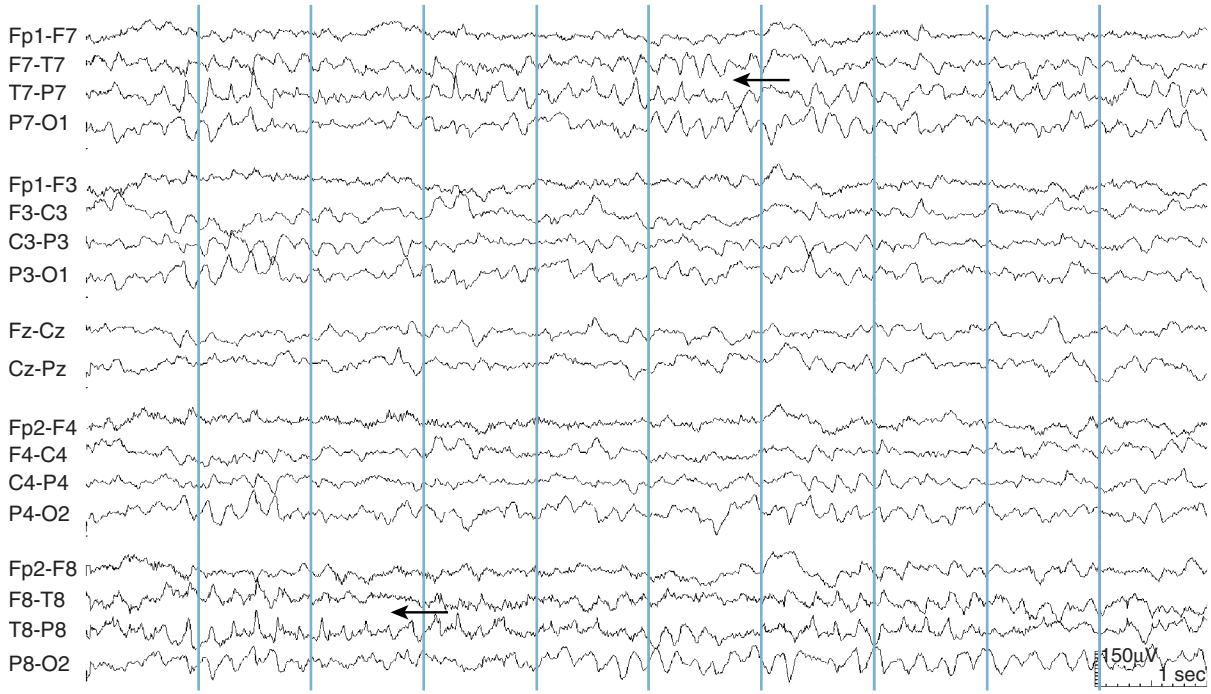


Figure 9-14 Sharp theta waves are seen in each temporal area (arrows). Note the varying degree of sharpness with some waves in the series appearing sharper and others appearing more rounded.

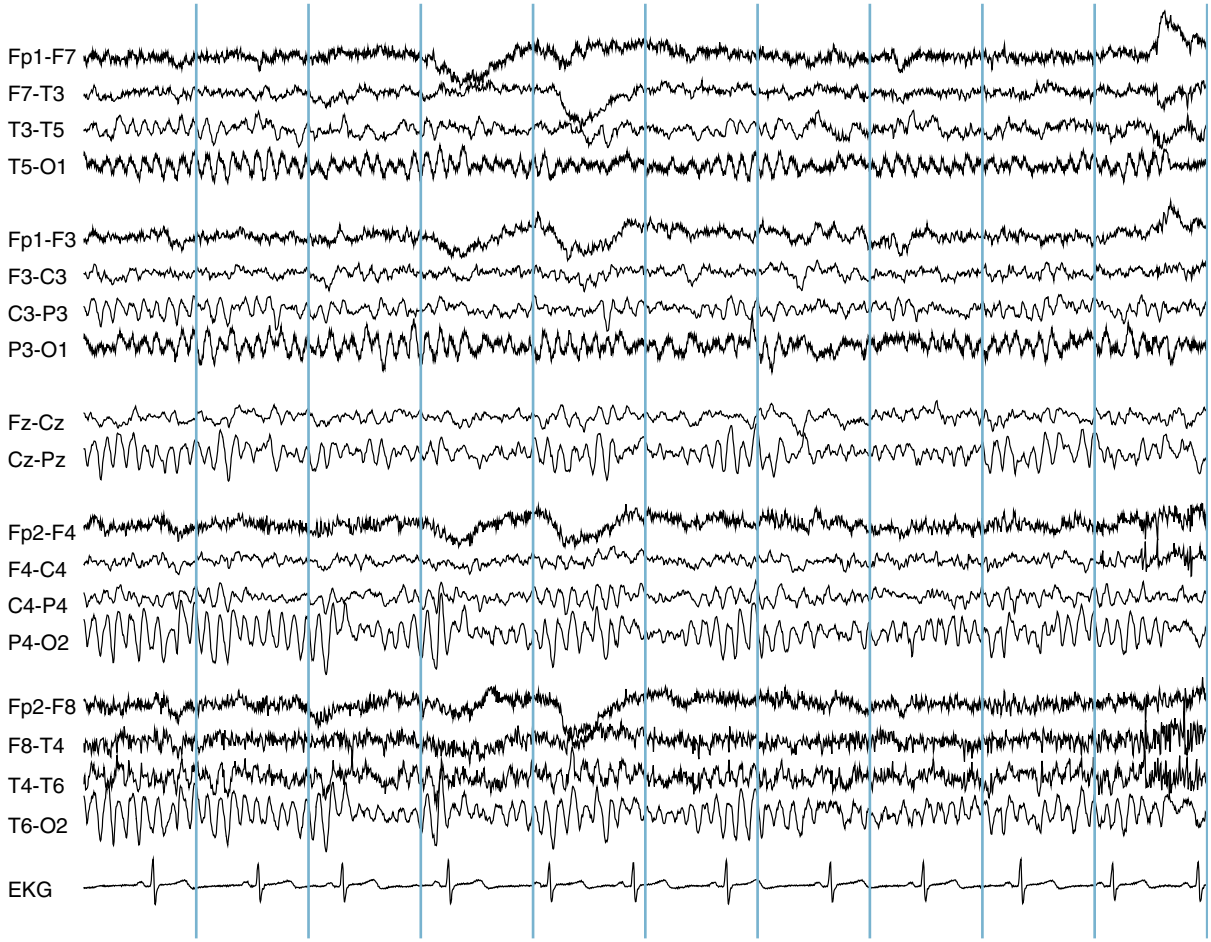


Figure 9-15 This EEG shows the typical asymmetry of the posterior rhythm. Higher voltages are seen in the right occipital area (P4-O2 and T6-O2 channels) compared with the left (T5-O1 and P3-O1 channels).

for excess fast activity in the waking EEG is pharmacologic effect. Benzodiazepines (such as diazepam, lorazepam, and clonazepam) and barbiturates (such as phenobarbital and pentobarbital) are the most common causes of increased fast activity in the EEG, although other categories of medication may cause this effect less frequently. Because these two classes of drugs are frequently used in both the acute and chronic management of seizures, it is common to see increased fast activity as a pharmacologic effect in the EEG. Drug-related increases in beta activity are usually diffuse but may also be frontally predominant (see Figures 9-16 and 9-17). The increased beta activity seen as a normal finding in many patients at onset of drowsiness usually subsides with deepening Stage II sleep.

Whether excess fast activity in the EEG as a drug effect should be considered an abnormal finding is, to some extent, a question of semantics. According to the definition used in this text—that a finding is abnormal only if it is associated with a pathologic or disease state—excess fast activity caused by drug administration would not represent a true abnormality; having received phenobarbital or diazepam should not be considered, in itself, a disease state. (A normal volunteer receiving these medications would manifest an increase in fast activity in the absence of a pathologic state.) Many readers note the presence of the fast activity in the interpretation without calling the tracing abnormal solely based on the finding of increased fast activity.

A relatively rare explanation for dramatically increased fast activity is developmental delay or mental retardation, especially when associated with a dysgenetic lesion of the brain (e.g., lissencephaly). Increased fast activity in the absence of other explanation should not prompt the reader to jump to the conclusion that a dysgenetic brain lesion is present. Often the medication list is incomplete, or, alternatively, barbiturates may have been administered previously and subsequently discontinued but may not yet have been cleared from the bloodstream. Fortunately, the increased beta activity that is seen with dysgenetic lesions usually occurs in the context of an otherwise significantly abnormal EEG and is easy to recognize. Also, in these cases the beta activity is often of a slower frequency than is seen in normal individuals.

"Slowed" or Absent Fast Activity

Although cortical fast activity recorded during wakefulness is usually within the range of 18 to 30 Hz, a reduction in the frequency of fast activity may also represent an abnormality. "Slow" fast activity is most commonly encountered in cases of diffuse cortical injury, as may be seen after an anoxic episode. In other cases, decreased frequency of fast activity may represent the pharmacologic effect of certain sedative or anesthetic medications, such as those used for pharmacologic induction of coma.

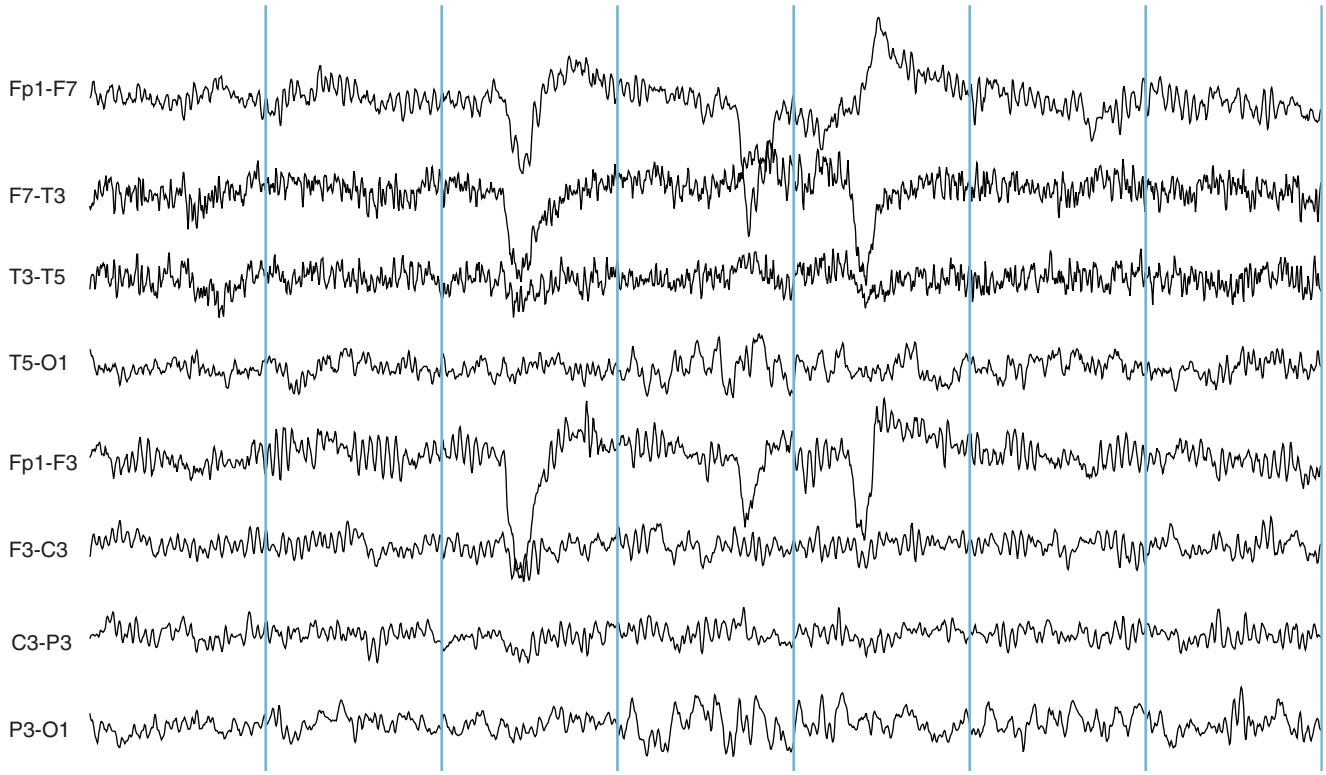


Figure 9-16 A large amount of beta activity is distributed across all channels. Scanning for low-voltage spike activity is made considerably more difficult by the large amount of fast activity.

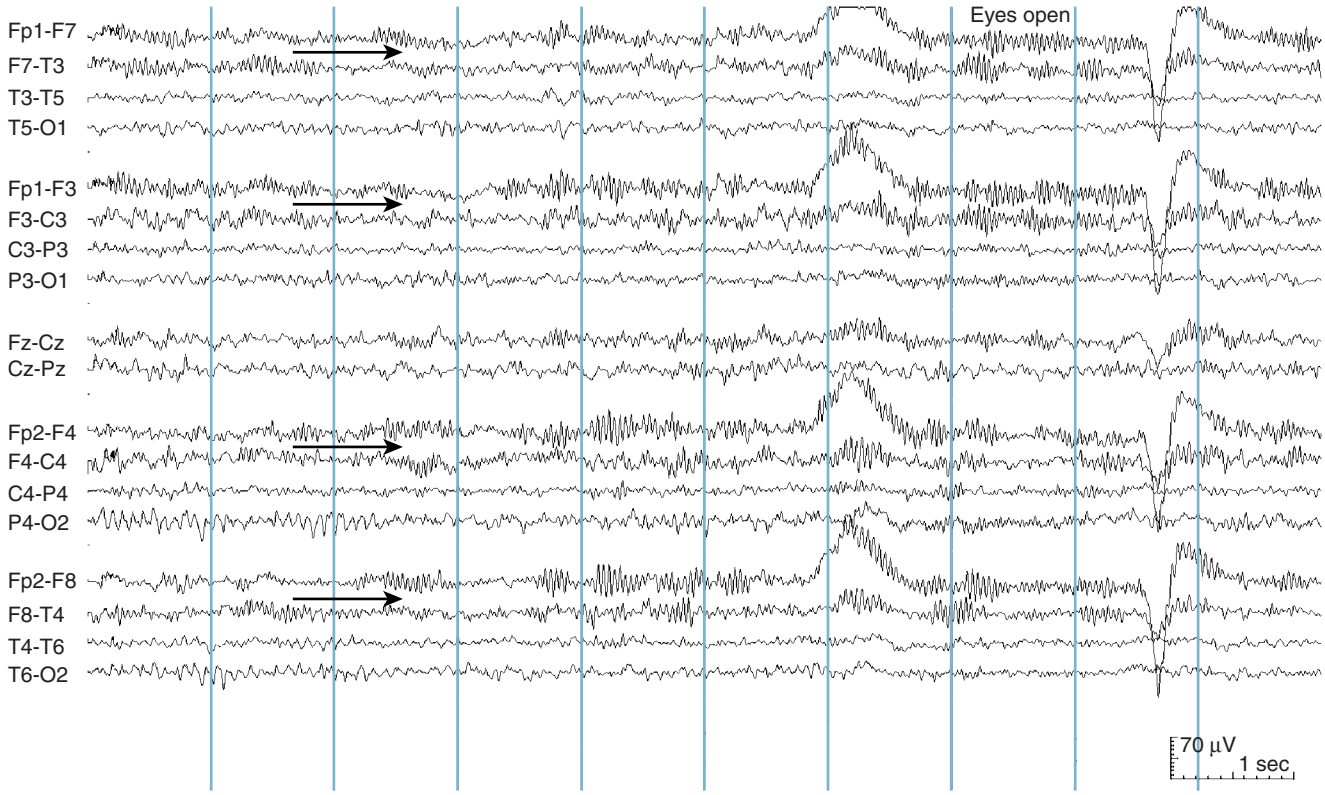


Figure 9-17 The amount of fast activity in this patient has been markedly increased by the administration of lorazepam. In this example, the fast activity is most prominent in the anterior quadrants (arrows).

A relative absence of fast activity in the EEG may also represent an abnormality. Absent fast activity may be seen in the setting of severe, diffuse cortical injuries (in which case, other abnormalities are typically present), although it must be appreciated that some patients have less fast activity in the EEG than others, and apparently decreased amounts of fast activity in the waking EEG may represent a normal variant in some individuals.

In assessing the amount of fast activity in an EEG, the reader should not be led astray by the situation of a tracing that is displayed with low amplifier gains. Tracings with large amounts of high-voltage slow activity are typically displayed with low amplifier gains so that adjacent channels will not collide and cross. At these lower gains, the lower voltage fast activity, although present in normal amounts, may not be visible in the display. This effect is discussed in more detail in Chapter 2, "Visual Analysis of the EEG," and illustrated in Figure 2-24.

Asymmetry of Beta Activity

A true asymmetry in fast activity between brain regions is an important abnormality. Fast waves are believed to be generated at the level of cerebral cortex, reflecting the activity of cortical circuits near the scalp surface (rather than activity arising from deeper levels, such as the deep white matter). Thus, asymmetry of fast activity can be an important marker of cortical damage (see Figure 9-18). Most often, the region with lower voltage fast activity marks the area of abnormality; in the region of a cortical stroke, it is expected that beta activity will be reduced. Much more rarely, cases are seen in which an area of abnormal cortex is associated with higher voltage fast activity.

ASYMMETRIC RHYTHMIC ACTIVITY

In cases in which not just a single frequency range but all frequencies of electrical activity are depressed in a certain area, the shorthand term "decreased rhythmic activity" can be used to refer to a decrease in activity of all frequencies (see Figure 9-19). There are many possible causes for decreased rhythmic activity in a particular region, and care should be taken not to suggest a list of possible causes that is too narrow because even noncerebral causes are on the list.

When rhythmic activity is decreased over a discrete area, an anatomical injury such as loss of brain parenchyma (e.g., a stroke or encephalomalacia) is an initial consideration. Abnormalities in the various anatomical spaces should also be considered, including the intraparenchymal space (the brain tissue), the extra-axial space (including the space between the brain and the skull), and the extracranial space (the scalp). Orderly consideration should be given to the possibility of fluid collections in the subdural, subarachnoid, or epidural spaces, which may consist of blood, pus, or cerebrospinal fluid. Individuals with large but asymmetrical subgaleal

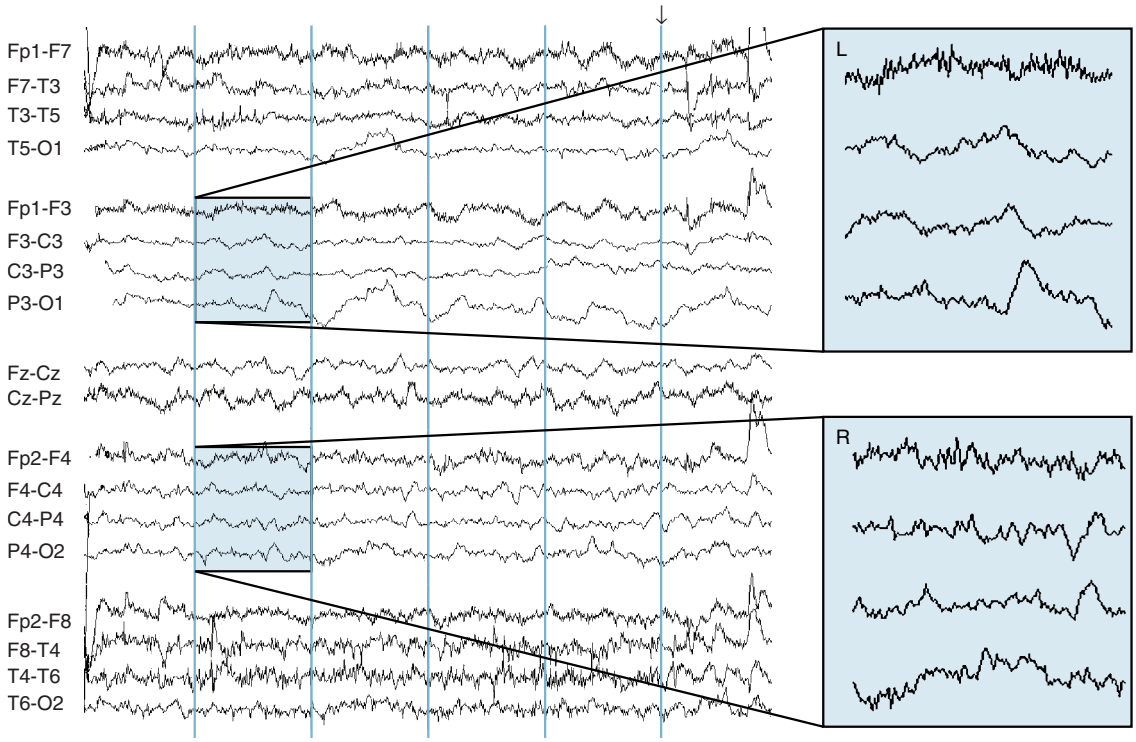


Figure 9-18 The waking EEG of this patient who has suffered a left middle cerebral artery infarct shows decreased fast activity over the area of the stroke. The close-ups emphasize the lower voltage of fast activity over the left parasagittal area compared with the right.

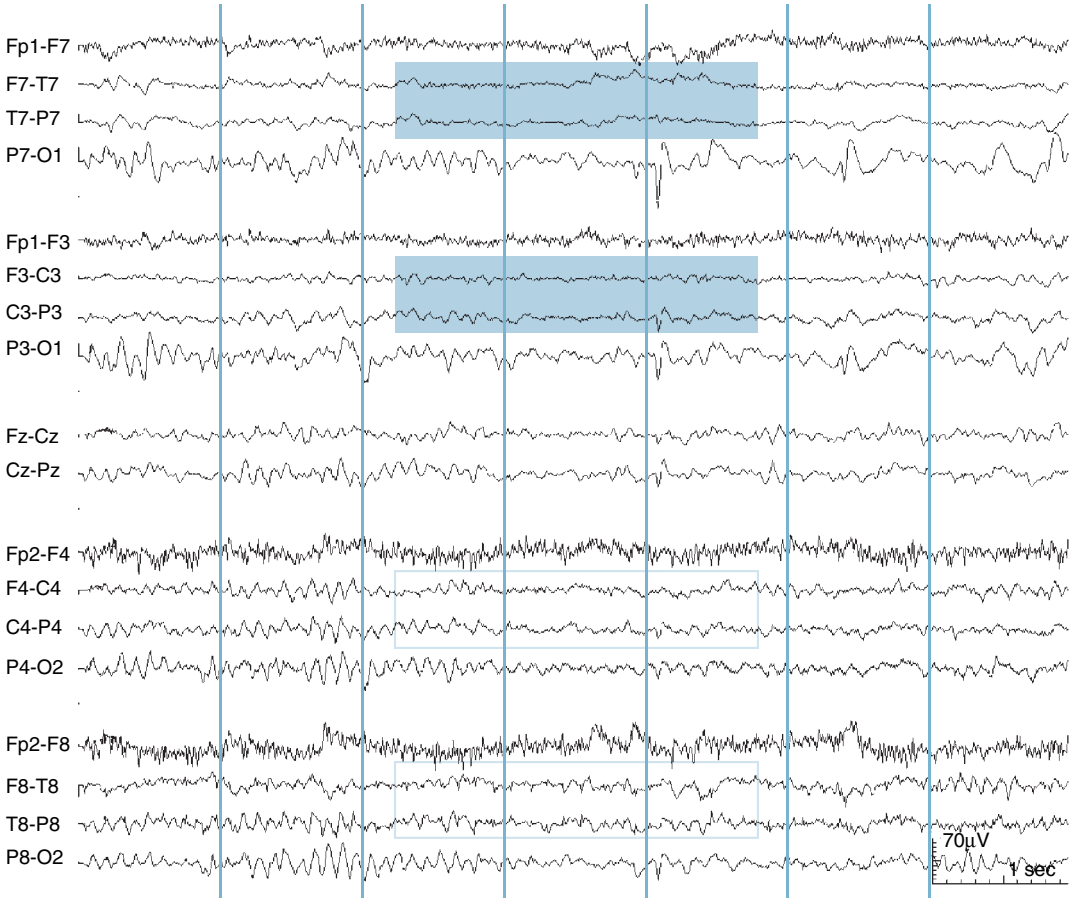


Figure 9-19 This patient, who has experienced a left hemispheric stroke, shows a more marked asymmetry of rhythmic activity (i.e., for waves in multiple frequency ranges) in the highlighted areas. Whereas in the previous figure the asymmetry was mostly restricted to fast frequencies, in this example all frequencies are relatively diminished over the left central and temporal areas (shaded rectangles), giving them a relatively flat appearance. Compare with the right central and temporal areas (unshaded rectangles). Incidental note is made of left occipital spike-wave discharges.

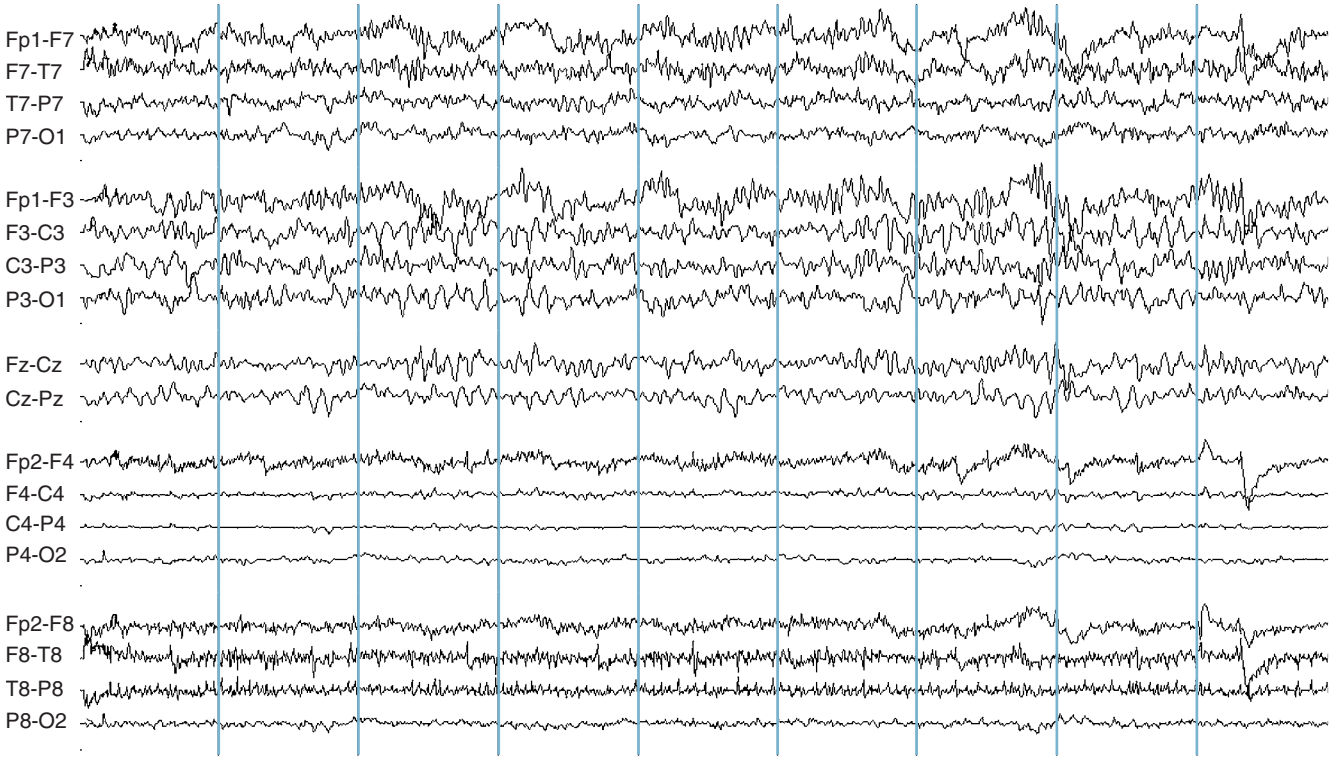


Figure 9-20 The most dramatic examples of voltage asymmetry can be seen in patients who have undergone surgical hemispherectomy. Note that all apparent rhythmic activity over the right hemisphere can either be ascribed to artifact or to projection of voltages from the left hemisphere (e.g., low-voltage alpha frequencies seen in F4-C4 and C4-P4). The patient's magnetic resonance imaging scan is shown in the next figure.

hemorrhages or other scalp swelling such as a caput succedaneum (a type of scalp swelling seen in the newborn soon after delivery) can cause dramatic voltage asymmetries even in the absence of cerebral abnormalities. It would be undesirable to make the error of interpreting the EEG of a patient who has been in a car accident as indicating a brain injury in the left hemisphere simply because of a large hemorrhage in the left scalp. Therefore, the whole gamut of intra-axial and extra-axial/extracranial abnormalities should be considered in the case of an EEG tracing with a voltage asymmetry that includes a wide band of frequencies. Figures 9-20 and 9-21 show a dramatic asymmetry of rhythmic activity as an aftereffect of surgical removal of the right hemisphere for refractory epilepsy.

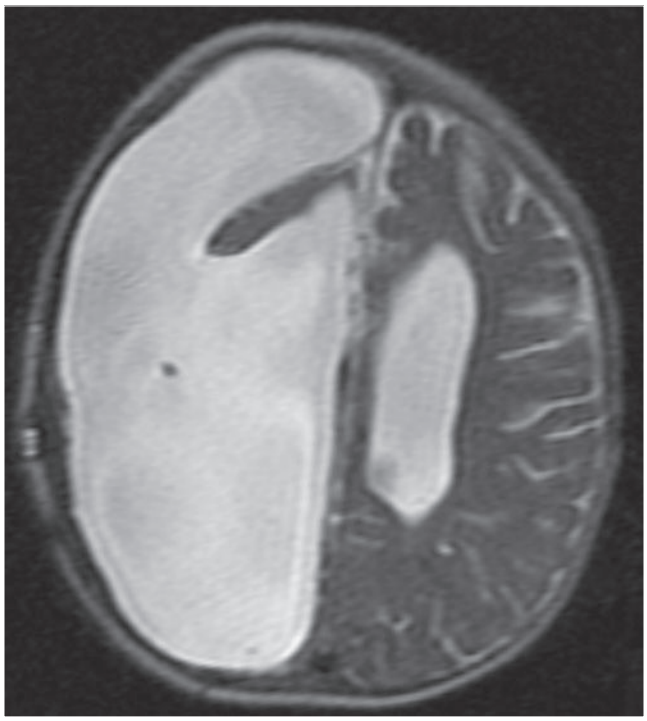


Figure 9-21 The magnetic resonance imaging scan of a patient who has undergone right hemispherectomy for intractable epilepsy is shown. A small tongue of disconnected frontal brain tissue remains. The patient's EEG is shown in the previous figure.

ASYMMETRIES OF SLEEP ELEMENTS

In the case of asymmetric sleep spindles or vertex waves, the magnitude and persistence of the asymmetry are important in deciding whether or not to label the asymmetry as abnormal (see Figure 9-22). Mild asymmetries in the amplitude of sleep spindles are occasionally seen and may be of little consequence. Spindle voltage asymmetries greater than one third to one half bring up the question of a true functional brain asymmetry (see Figure 9-23). In certain patients, occasional

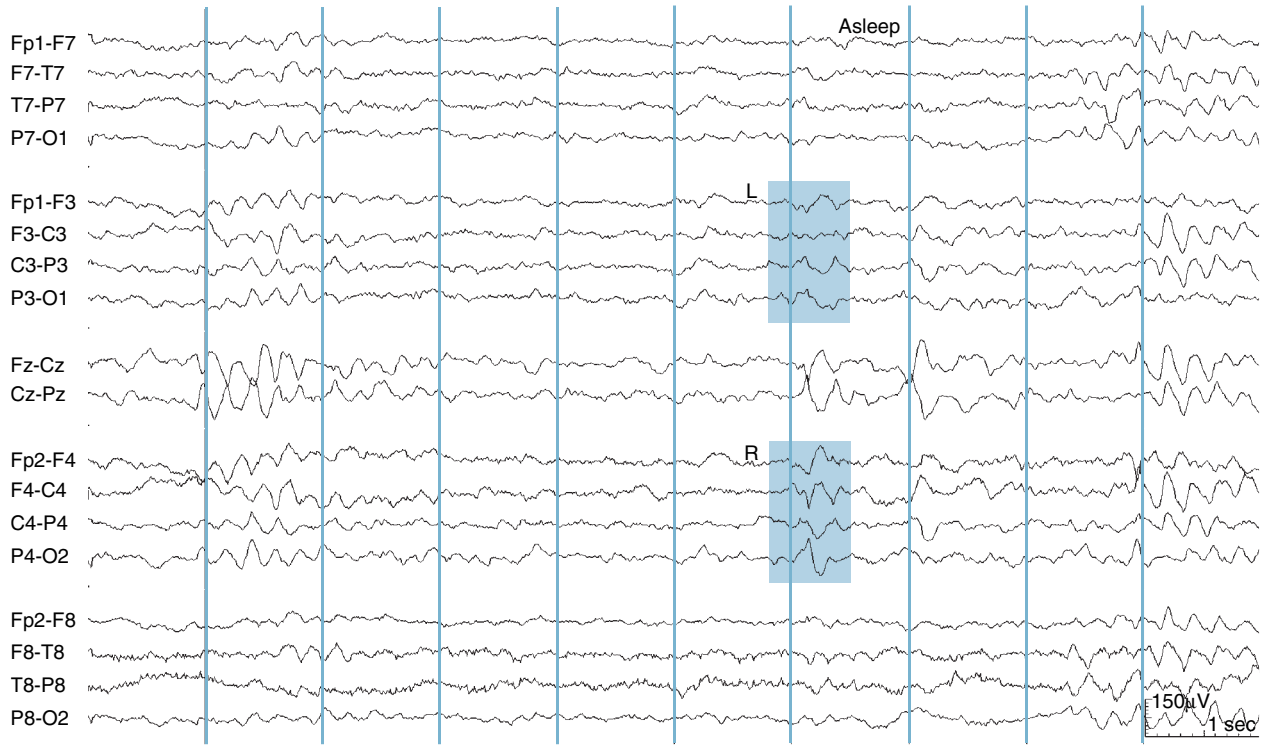


Figure 9-22 This EEG shows an asymmetry of vertex waves during Stage I sleep. Note that the vertex wave is well seen in the midline and in the right central area (shaded area marked "R") but not well seen in the left central area (shaded area marked "L"). Such asymmetries are only considered to be potentially clinically significant if they are persistent.

asymmetry of vertex waves is seen and may not be significant. Mild or nonpersisting shifts of vertex wave maxima to the left or right may not be clinically important. More persistent or dramatic asymmetries in the appearance of spindles or vertex waves may, however, reflect an asymmetry of the brain structures that generate these waves and therefore may be clinically significant. Asymmetry of sleep elements should be confirmed in a referential montage whenever possible.

Extreme Spindles

"Extreme spindles" is an exaggerated spindle pattern in sleep and represents a rare abnormality. These abnormally prolonged and persistent spindles have been described in mentally retarded children (see Figure 9-24). Rarely, this abnormal spindle pattern may even intrude into wakefulness.

EPILEPTIFORM ACTIVITY

In the 1974 glossary of EEG terms (Chatrian et al., 1974), epileptiform activity was defined as "distinctive waves or complexes, including spikes and sharp waves, that are distinct from background activity and resemble those recorded in a proportion of human subjects suffering from epileptic disorders." The word *epileptiform* itself suggests that these are waves that appear in the "form" of epilepsy but do not constitute an example of epilepsy (i.e., epileptic seizures) itself. By definition, epileptiform activity refers to abnormal discharges that occur between seizures—the formal definition specifically excludes epileptic *seizure* discharges themselves. Many epileptiform discharges (i.e., discharges that occur between seizures) do resemble the component discharges of actual epileptic seizures. The single interictal spike seen in a patient's EEG may resemble the component parts of the train of rapid spikes that constitutes that patient's epileptic seizure discharge, although this is not always the case.

Occasionally, it may be difficult to distinguish interictal activity from electrographic seizure activity (discussed in more detail in Chapter 10, "The EEG in Epilepsy"). The electroencephalographer must always be conscious of the distinction between discharges that take place during the seizure or "ictus," referred to as *ictal* activity, and activity that takes place between seizures, *interictal* activity. The definition of epileptiform activity has been extended by some to include certain paroxysmal bursts of slow-wave activity, reasoning that these bursts are the indicator of a decreased seizure threshold in certain patients (see the discussions of OIRDA associated with childhood absence and TIRDA earlier in the chapter). Keeping in mind that approximately 1% of asymptomatic adults and 3% to 4% of asymptomatic children will have epileptiform activity in the EEG, it is important to avoid the error of equating epileptiform activity in the EEG with the diagnosis of epilepsy.

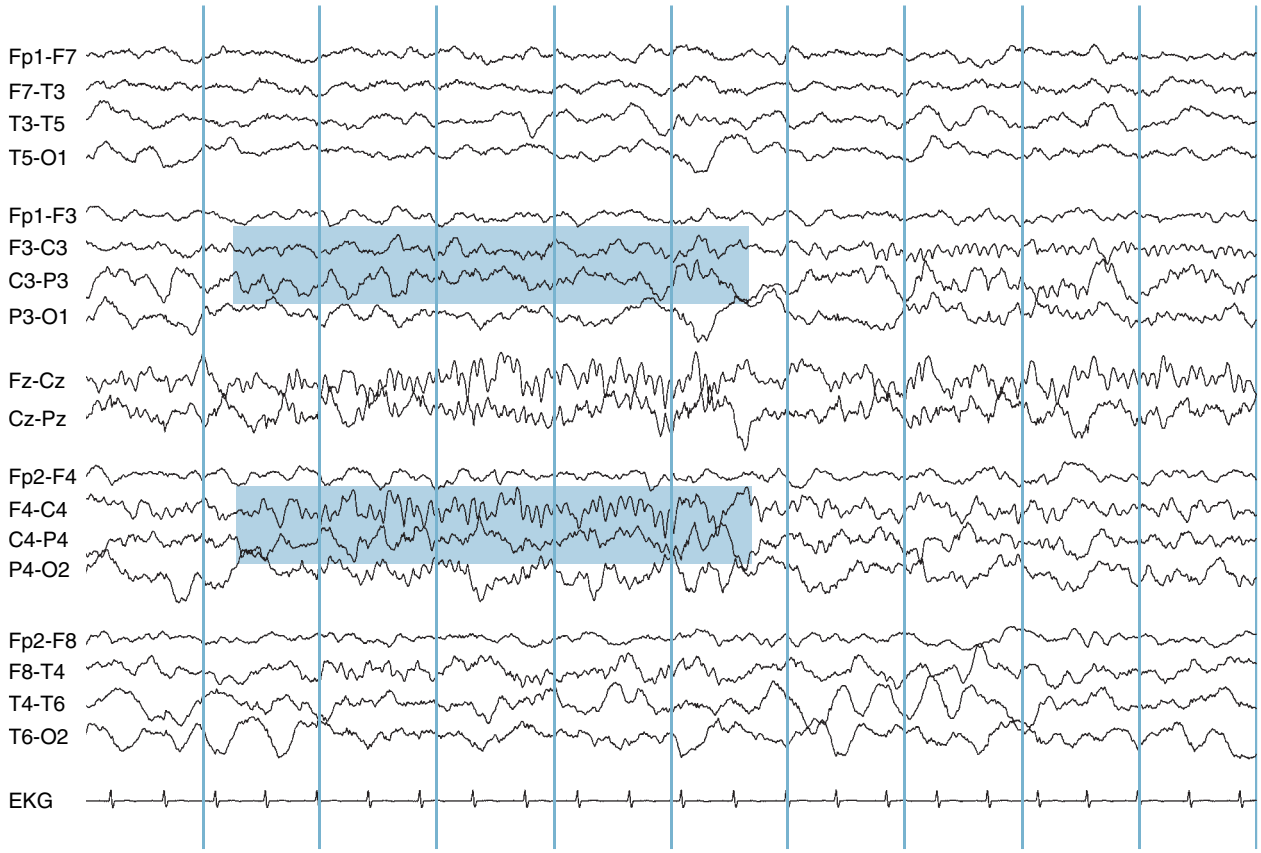


Figure 9-23 Persistent asymmetry of sleep spindles is considered abnormal. Note that the spindle visible in the right central region (lower shaded area) and the midline can barely be seen during this interval in the left central region (upper shaded area).

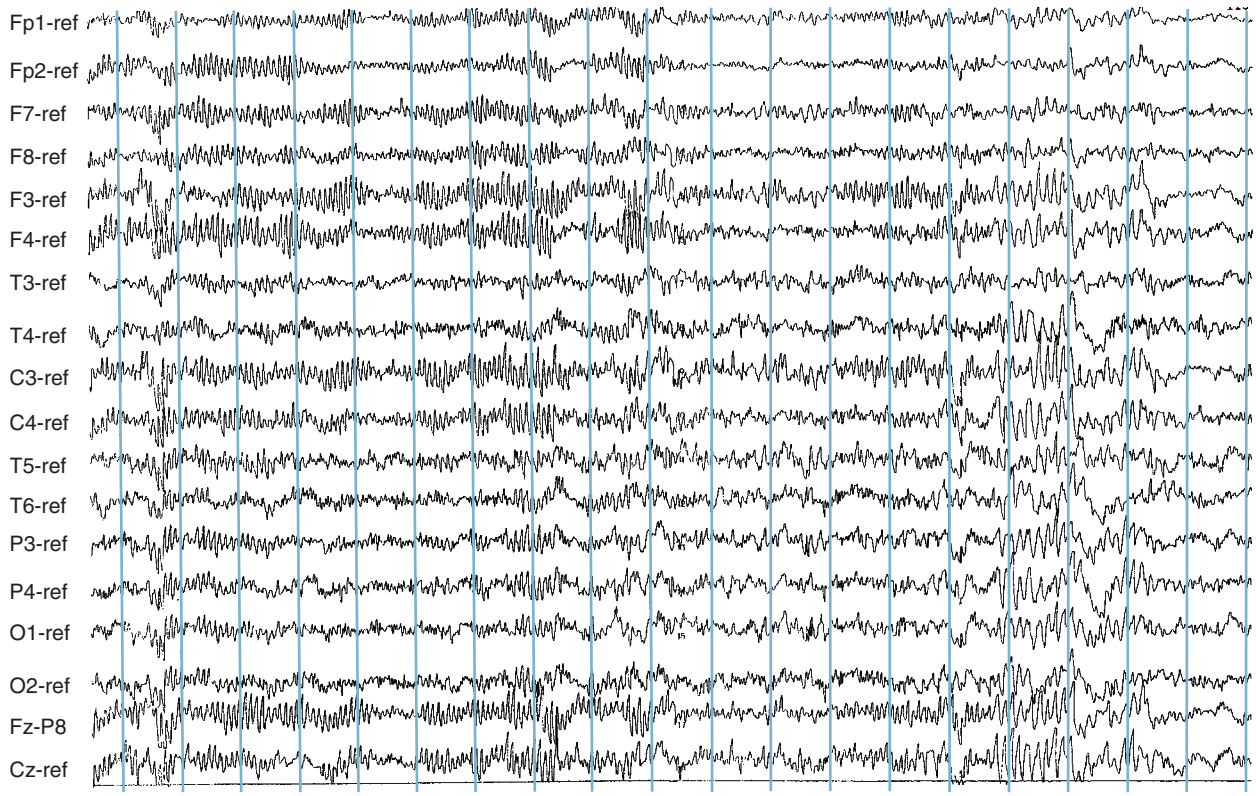


Figure 9-24 An example of extreme spindles is shown in a referential montage. Note that spindle voltages are highest in the C3 and C4, F3 and F4, and Fz and Cz electrodes, as expected.

Electrographic Versus Electroclinical Seizures

By definition, a seizure is an event during which a clinical change in the patient is accompanied by a characteristic abnormal change in brain waves. The clinical change may either occur outwardly and be observable by those surrounding the patient or the change may be experienced subjectively by the patient. The outward clinical change may be as obvious as generalized tonic-clonic seizure activity or more subtle, such as a mild slumping of the shoulders or slowed responsiveness. Internally experienced seizure manifestations, such as olfactory hallucinations or the psychic experience of fear, may be invisible to outside observers. Because most seizure events do not occur while a patient is being recorded on EEG, the brain wave portion of the definition implies a presumed change in the EEG at the time of the clinical event had the patient been recorded by an appropriate technique at the time.

The question may arise as to how to classify an EEG seizure discharge that is not accompanied by a known clinical change in the patient. EEG seizure discharges during which the patient's clinical status is unknown or apparently unchanged are termed *electrographic seizures*. To distinguish electrographic seizures from those seizures that meet the classic seizure definition of concurrent clinical seizure behavior and EEG seizure discharge, the term *electroclinical seizure* can be used. Imagine a patient who has 10 seizure discharges recorded in the EEG. Five are recorded during wakefulness and are associated with behavioral arrest, and the other five are electrically identical discharges recorded during sleep, a state during which behavioral arrest cannot be observed. The electroencephalographer may then report that five electroclinical seizures and five electrographic seizures were recorded during the study.

Whether a seizure is truly "electrographic" as opposed to "electroclinical" can sometimes approach a philosophical question. How does one deal with an electrographic seizure discharge during sleep as described in the preceding paragraph or in a child too young to describe experience? Similar questions arise when an individual recorded on video has an electrical seizure discharge but is not in the midst of an activity. In such a case, had the patient been speaking or carrying out an activity, perhaps a behavioral arrest or slowed response time would have been observed. Often such questions regarding individual events may not be answerable and the convention is to label any seizure discharge as an electrographic seizure if no behavioral or experiential change can be ascertained. Likewise, in some patients, it can be difficult or even impossible to determine whether a run of discharges represents an interictal discharge or an electrographic seizure discharge. This is a particular problem in patients with slow spike-wave discharges in which the ictal and interictal discharges can look quite similar.

Descriptions and definitions of epileptiform spikes, sharp waves, and spike-wave discharges are given in Chapter 3, "Introduction to Commonly Used Terms in Electroencephalography." Because the large majority of epileptic patients do not experience an actual epileptic seizure during routine EEG recording in the EEG laboratory, it is the interictal epileptiform discharges that are the main tool in further defining an individual's specific epilepsy diagnosis (see Figure 9-25).

Limitations to the Localization Value of Interictal Epileptiform Activity

Interictal discharges can be valuable in localizing the epileptogenic zone in epileptic patients, but there are instances when their localization value may be limited. Certain cerebral regions are particularly difficult to localize using scalp recordings. Some types of discharge may even be "falsely localizing," suggesting a localization that is different from their scalp source. This is especially true of discharges that arise from areas of cortex that are not adjacent to the scalp, such as the interhemispheric fissure, orbital surfaces, mesial temporal lobe, basal surfaces of the occipital lobes, or insular cortex.

A second potential problem is that the location of an epileptiform discharge, even if well ascertained, may not always indicate the seizure onset zone. Interictal discharges often arise from the seizure focus, but this is not true 100% of the time. Keep in mind that some individuals with interictal discharges may never have a seizure, which implies that at least some interictal discharges never give rise to seizure activity. Also, there are some patients with seizures who never have interictal discharges. Acknowledging these two situations, if it is possible for an area of the brain to generate interictal discharges but never originate a seizure and another area may generate seizures but be interictally quiet, it is easy to imagine an occassional circumstance in which an interictal discharge could occasionally "point to the wrong place" in some patients with seizures. Some individuals may have two separate foci of epileptiform activity, although seizures may only arise from one of the regions. In such a patient, the location of one of the discharges may suggest an incorrect localization. It is not hard to imagine how this could happen. Imagine a person who has experienced a patchy injury to the brain, perhaps from trauma or infection. Two areas are damaged, one in such a way that it generates abnormal epileptiform discharges but, by chance, not in a way that generates actual seizures. The second area is damaged in such a way that it generates both epileptiform activity *and* seizures. In such a patient, only one of the spike localizations is pertinent to understanding from where the seizures arise. Therefore, a spike localization often, but not always, corresponds to the area of seizure onset. Other factors such as the history, imaging results, and seizure semiology should be used to confirm localization.

A secondary use for localizing epileptiform activity, even in the absence of epileptic seizures, is to localize different types of lesions in the brain. In the past, electroencephalography was among the most useful tools for localizing brain tumors, but modern neuroimaging has supplanted electroencephalography

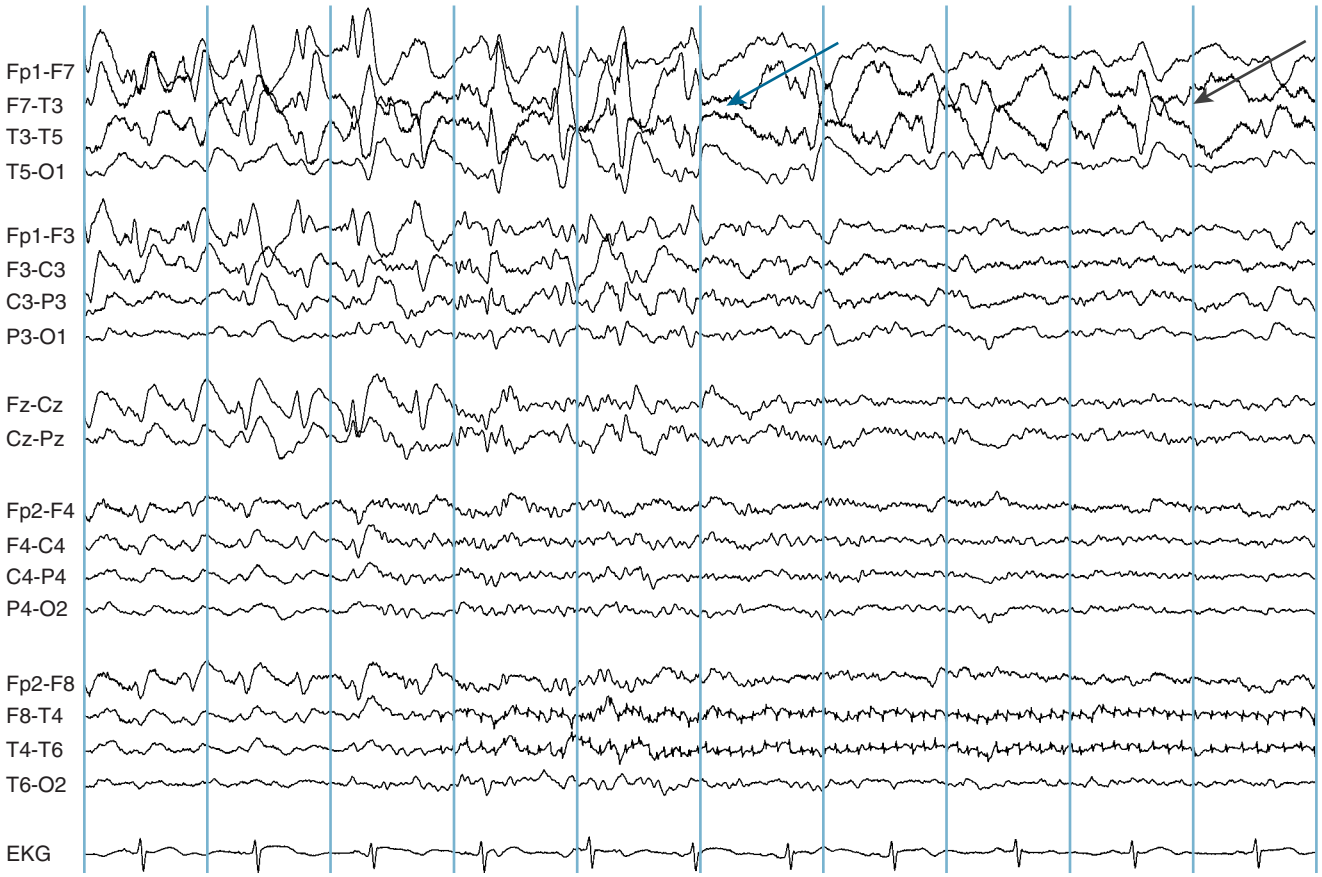


Figure 9-25 A run of spike-wave discharges is seen across the left temporal chain (black arrow) in a 15-year-old girl with complex partial seizures. The sharp slow waves seen at the end of the page (gray arrow) suggest a hybrid between slow waves and sharp waves, sharing the characteristics of both.

as a primary tumor localization tool. Still, despite the primacy of MRI in imaging tumors, epileptiform activity in the EEG may sometimes give the first clue to the presence of a tumor, as occurred in the patient discussed in the paragraph that follows on secondary bilateral synchrony.

Secondary Bilateral Synchrony

Secondary bilateral synchrony refers to the phenomenon of generalized discharges arising from a unilateral cortical focus. Typically we think of focal discharges in the EEG as associated with focal lesions and generalized discharges as associated with generalized processes or generalized epilepsies. Apparently generalized discharges in the EEG are not always associated with true generalized seizure onsets, however. Occasionally, a unilateral focal lesion can originate a discharge that spreads rapidly and synchronously across both hemispheres. Such discharges, although arising from a single focus, may spread throughout the brain so rapidly that they are virtually indistinguishable from a primary generalized discharge. Figure 9-26 shows an example of apparent generalized spike-wave discharges. Figures 9-27, 9-28, and 9-29 are taken from other pages of the same patient's EEG and give clues to the phenomenon of secondary bilateral synchrony. The patient's causative lesion is shown in Figure 9-30. Because of this phenomenon, it is good practice to consider the possibility that any generalized discharge might be an example of secondary bilateral synchrony and to look for clues to the latter. One such clue is voltage asymmetry of the discharge—the discharge may be of persistently higher voltage over one hemisphere as compared with the other. An even more compelling finding is a consistent "lead-in" of the discharge from one hemisphere, the hallmark finding of secondary bilateral synchrony. In these cases, the discharge is seen to originate from one hemisphere followed by rapid generalization.

Because close analysis of genuine generalized discharges may disclose an apparent onset that is several milliseconds ahead on one side compared with the other, the most compelling "lead-ins" are those of 2 seconds' or more duration that always occur on the same side. In some patients, it is not uncommon for genuine generalized discharges to alternate the

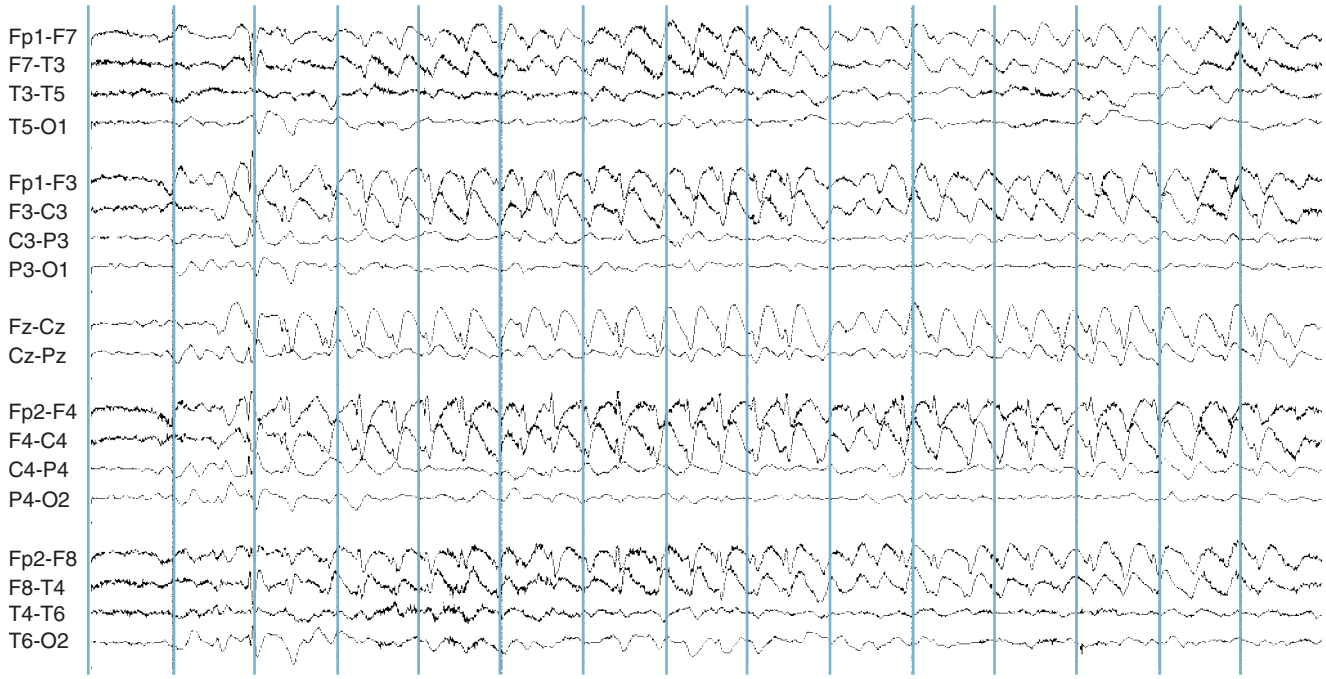


Figure 9-26 An apparent generalized, frontal-predominant spike-wave discharge is seen with no clear indicators to the contrary; no significant asymmetries or "lead-in" is seen. In fact, the discharge shown in this figure was triggered by a tumor in the right temporal lobe. The following figures show additional pages taken from the same EEG that do have clearer clues as to the discharge's focal onset.

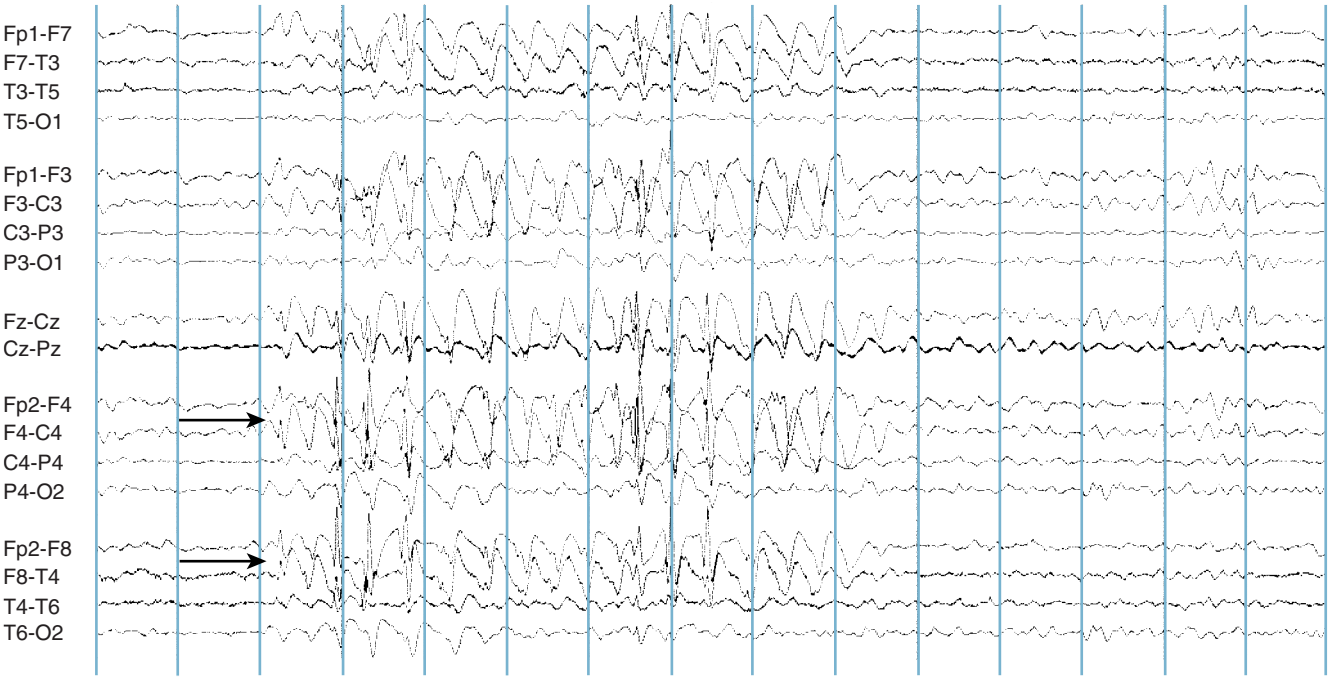


Figure 9-27 During this burst taken from the same EEG shown in the previous figure, higher spike-wave voltages are seen on the right side (arrows) compared with the left. There is also a question of a "lead-in" in the right frontal area. Although slight voltage asymmetries may occur in truly generalized discharges, the presence of a voltage asymmetry should bring up the question of secondary bilateral synchrony.

apparent side of onset with brief "lead-ins" seen in one hemisphere, then the other. When the side of the lead-in alternates, this decreases the chances that the generalized appearance of the discharge is due to secondary bilateral synchrony.

The criteria for a *definite diagnosis* of secondary bilateral synchrony are fairly strict and include a definite "lead-in" of the discharge from one side, a possible voltage asymmetry, or possible focal interictal epileptiform activity seen at other times in the record arising from

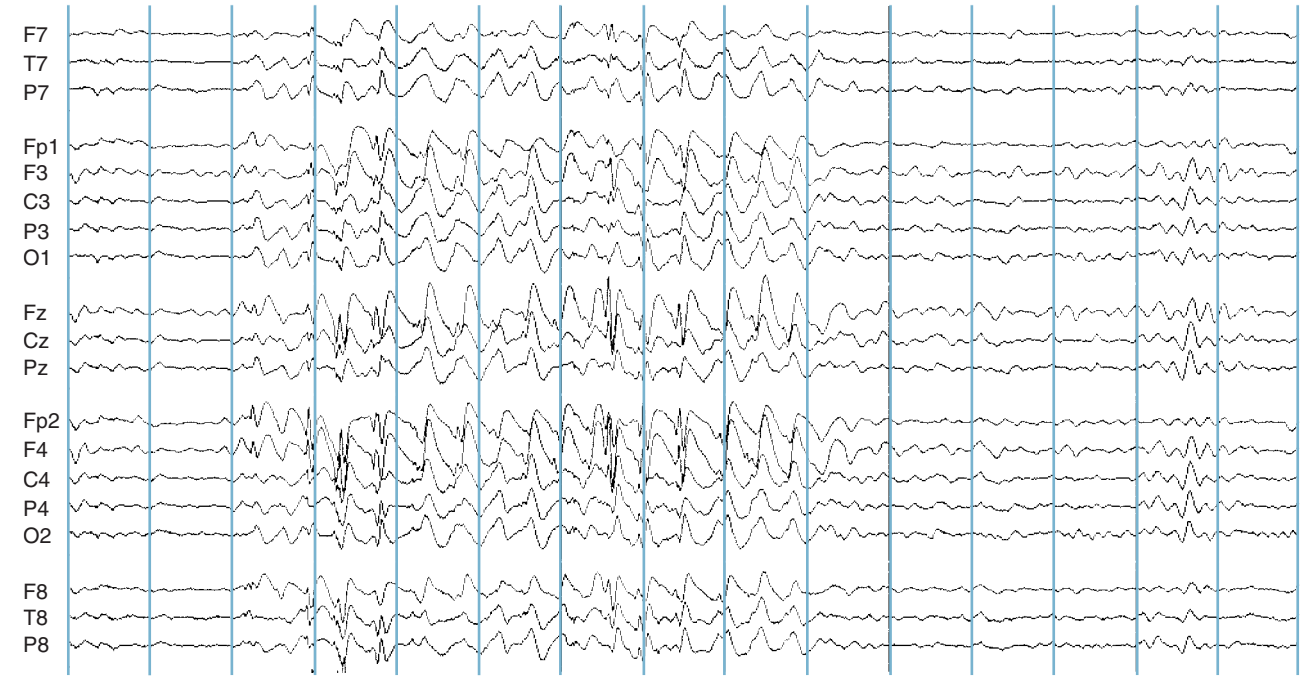


Figure 9-28 The same page of EEG as was shown in the previous figure is displayed in a referential montage, confirming the presence of a voltage asymmetry.

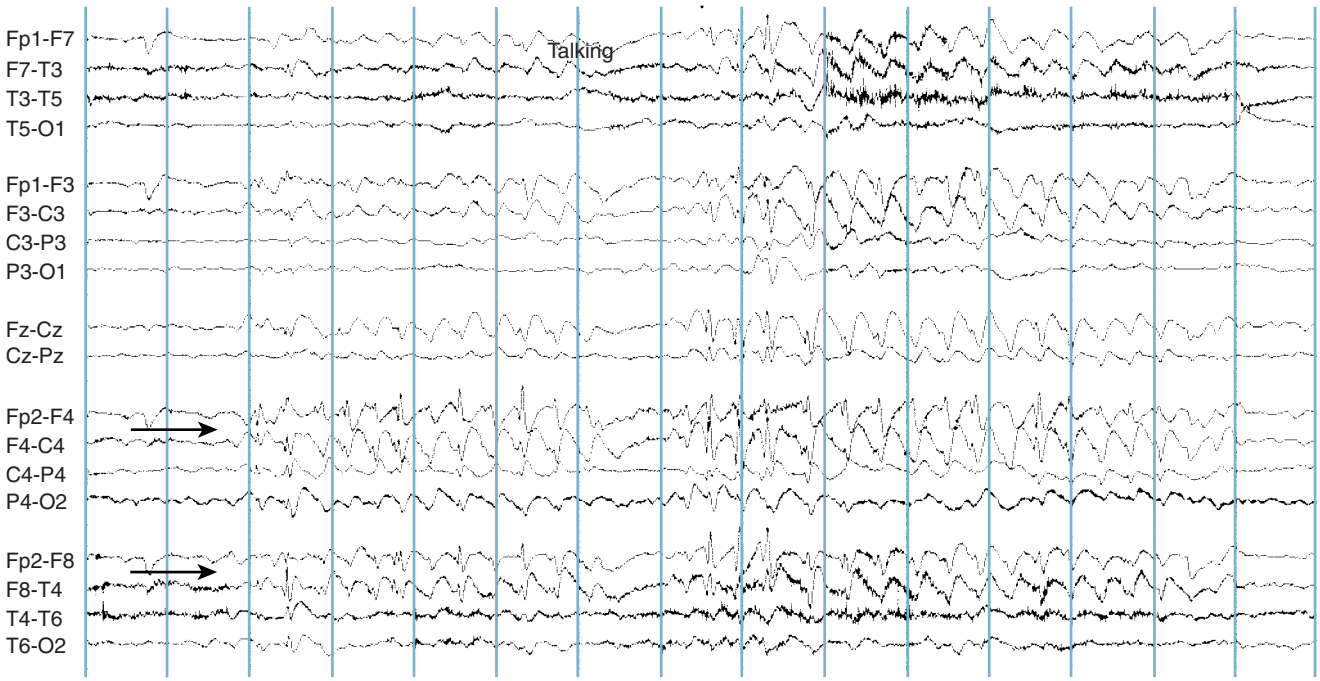


Figure 9-29 Another page of EEG is taken from the same tracing as shown in the previous three figures. This example of the patient's generalized spike-wave discharge now shows a clear and prolonged lead-in from the right hemisphere (arrows). An asymmetrical lead-in, especially if prolonged, is one of the most convincing clues to the presence of secondary bilateral synchrony.

the same side as the lead-in. The threshold for doubting that a generalized discharge is truly generalized (i.e., *suspecting* the diagnosis of secondary bilateral synchrony) is considerably lower. At times, focal lesions may cause discharges to generalize so rapidly that a lead-in is not identifiable. Therefore, every generalized discharge should be scrutinized for the possibility that it has a hidden focal onset, even if not meeting strict criteria for secondary bilateral synchrony. When other signs of a primary generalized epilepsy are not present

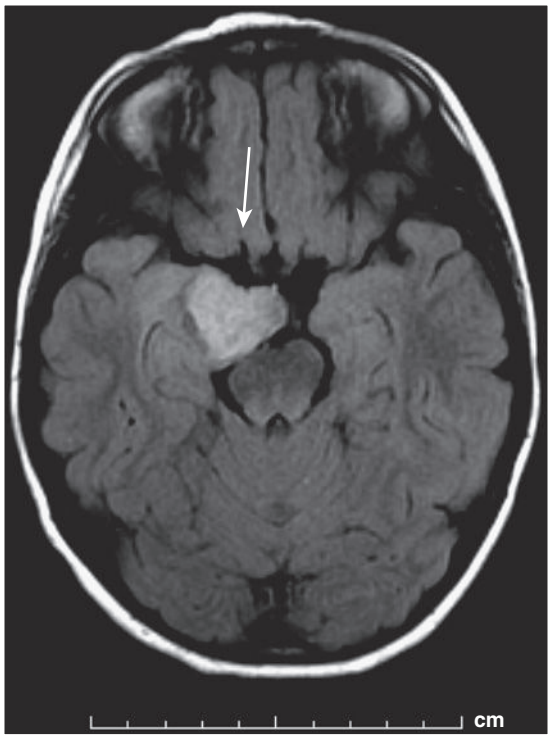


Figure 9-30 The magnetic resonance imaging scan of the child whose EEG is depicted in the previous four figures demonstrates the causative lesion. A glioma enlarging the tip of the right temporal pole is seen on this fluid-attenuated inversion recovery image (arrow). This unilateral lesion was responsible for generating a discharge which, at times, was indistinguishable from generalized spike wave.

in the EEG, the reader may choose to warn of the possibility of a focal onset, even when generalized spike-wave or other generalized discharges are seen. If appropriate, the clinical correlation paragraph can include wording to the effect that "the generalized discharges present in this EEG are potentially consistent with a primary generalized epilepsy, although the possibility of a focal onset with rapid secondary generalization cannot be completely excluded."

PERIODIC PATTERNS

The term *periodic pattern* refers to the presence of a periodic waveform in the EEG. The term *periodic* implies repetitive recurrence of the waveform with an *approximately* fixed interval between the waveforms. The term periodic may be compared with the term *rhythmic*, which implies a more fixed and regular interval. In fact, periodic patterns may sometimes be so regular as to appear rhythmic, but the term periodic implies the possibility of somewhat more variation in the intervals between waveforms.

A particularly useful classification system for periodic EEG patterns was described by Brenner and Schaul (1990). In this system, a periodic pattern is classified according to whether it occurs over a single hemisphere or both hemispheres (unilateral vs. bilateral), whether it occurs over both hemispheres in a synchronous or asynchronous (bilaterally independent) fashion, and whether the intervals between waves are less than or greater than 4 seconds (short interval vs. long interval). In practice, combinations of these three parameters result in four categories of patterns: 1) periodic lateralized epileptiform discharges (PLEDs), 2) bilateral periodic lateralized epileptiform discharges (BiPLEDs), 3) periodic short interval diffuse discharges (PSIDDs), and 4) periodic long interval diffuse discharges (PLIDDs). (See Table 9-1.)

PLEDs

Of the periodic patterns, the best known is PLEDs (see Figure 9-31). Of the four letters in this abbreviation, the "L" for lateralized is the most useful to keep in mind. The word *lateralized* is used in place of the word *focal*, reflecting the fact that, rather than being confined to a small area, the field of a PLED spreads across a whole hemisphere. If an apparent periodic discharge is focal rather than lateralized, the diagnosis of PLEDs should be questioned.

PLED waveforms, apart from being lateralized to a single hemisphere, may be simple, complex, or polymorphic. Typically, although not always, the PLED waveform contains sharp or spike-like features. PLEDs occur repetitively in the tracing approximately every 1 to 4 seconds. In general, when PLEDs are present, they are present for the entirety of the tracing; they do not tend to appear and disappear within a tracing. If a periodic waveform is seen only intermittently, the diagnosis of PLEDs should be questioned.

Etiologies associated with PLEDs include a list of acute and subacute processes with stroke, infection,

Table 9-1 Periodic Patterns

	Short Interval (0.5–4 sec)	Long Interval (4–30 sec)
Unilateral	Periodic lateralized epileptiform discharges (PLEDs)	—
Bilateral asynchronous	Bilateral periodic lateralized epileptiform discharges (BiPLEDs)	—
Bilateral synchronous	Periodic short interval diffuse discharges (PSIDDs)	Periodic long interval diffuse discharges (PLIDDs)

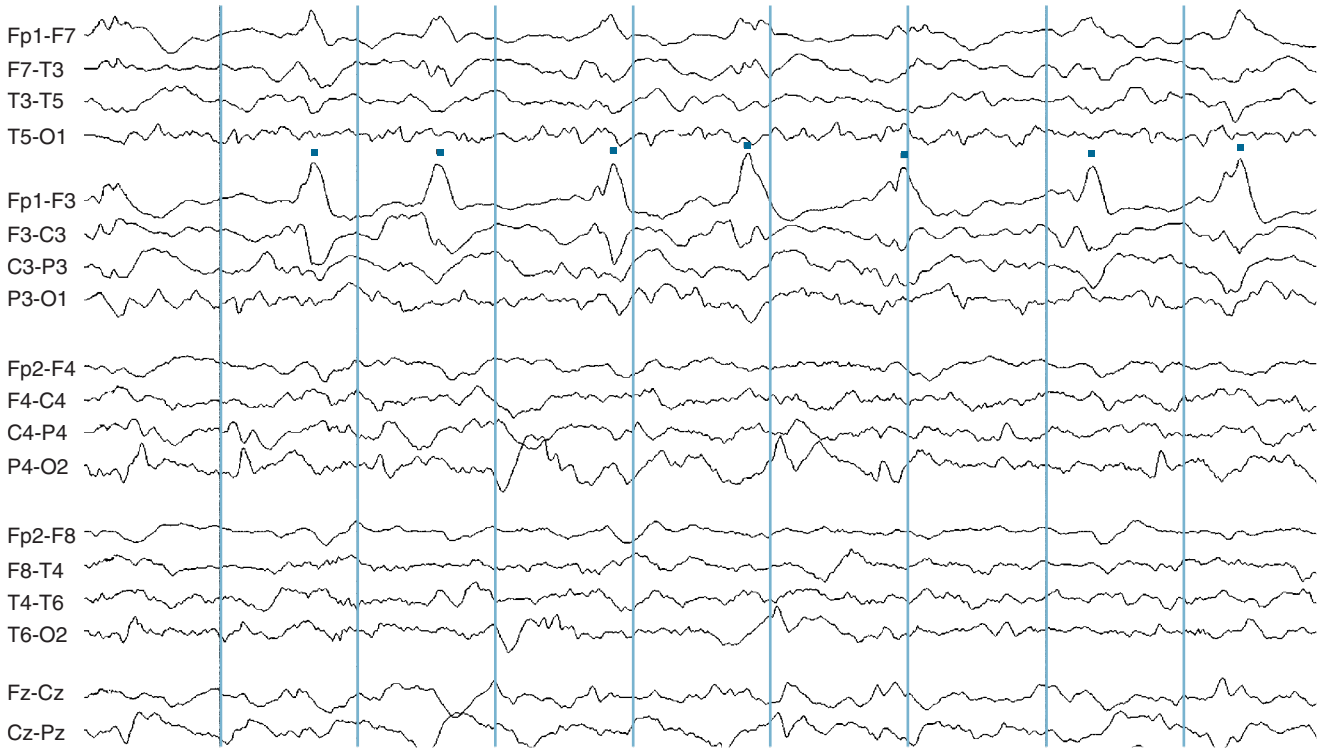


Figure 9-31 An example of periodic lateralized epileptiform discharges (PLEDs) is seen in the form of high-voltage, repetitive complexes over the left side (dots). The discharges have a fairly broad, hemispheric field. At times, the waveform seems sharp and at other times it appears more rounded. This duality is characteristic of PLEDs, which may or may not manifest obviously sharp features.

and tumor at the top of that list. Classically, the finding of PLEDs has a particularly well-known association with herpes simplex virus (HSV) encephalitis (although the finding is not pathognomonic). In HSV encephalitis, PLEDs may either be unilateral or bilateral but independent (BiPLEDs, discussed in the next subsection).

Occasionally, the distinction between PLEDs and seizure activity can be difficult. For instance, when PLEDs are associated with contralateral jerking movements of the body, the diagnosis of epilepsia partialis continua (EPC) is suggested. Unless there is such a contralateral motor response in association with the discharges, the electrical patterns associated with EPC can be indistinguishable from PLEDs. In general, however, PLEDs are not considered to represent an electrographic seizure discharge but rather an interictal pattern. This is borne out by the fact that PLEDs are usually resistant to treatment with antiseizure medications. Rare exceptions to this concept are seen in the form of confusional states associated with PLEDs that have cleared with the administration of antiseizure medications.

PLEDs may be seen less commonly after a prolonged seizure discharge, so-called postictal PLEDs. After the clinical seizure has ceased, periodic afterdischarges identical to PLEDs may be seen over a single hemisphere, eventually burning out with time. Except in unusual cases, PLEDs are a temporary phenomenon and do clear with the passage of time.

BiPLEDs

PLEDs that occur over both hemispheres simultaneously but in an asynchronous or independent fashion are termed *bilateral periodic lateralized epileptiform discharges* (BiPLEDs). The possible causes of BiPLEDs is similar to the causes of PLEDs but favor those processes that are more likely to be bilateral. Therefore, a tumor or a discrete, focal stroke, both of which tend to be unilateral processes, are not expected as causes of BiPLEDs, but anoxic encephalopathy (the equivalent of bilateral, diffuse strokes) is a more common cause. As mentioned earlier, BiPLEDs are particularly characteristic of HSV encephalitis which is usually a bilateral process.

PSIDDs

Periodic short interval diffuse discharges (PSIDDs) are periodically occurring waveforms that may consist of sharp waves, spikes, spike-wave discharges, or triphasic waves. As the name implies, the field is spread diffusely over the brain, and the interdischarge interval is 4 seconds or less (see Figure 9-32). The most common clinical association of PSIDDs is anoxic encephalopathy. Each discharge may or may not be associated with a body jerk (myoclonus). In general, the presence of PSIDDs in anoxic encephalopathy suggests a poor neurologic prognosis. PSIDDs may also occur as a seizure phenomenon (see the section on nonconvulsive status epilepticus later in the chapter).

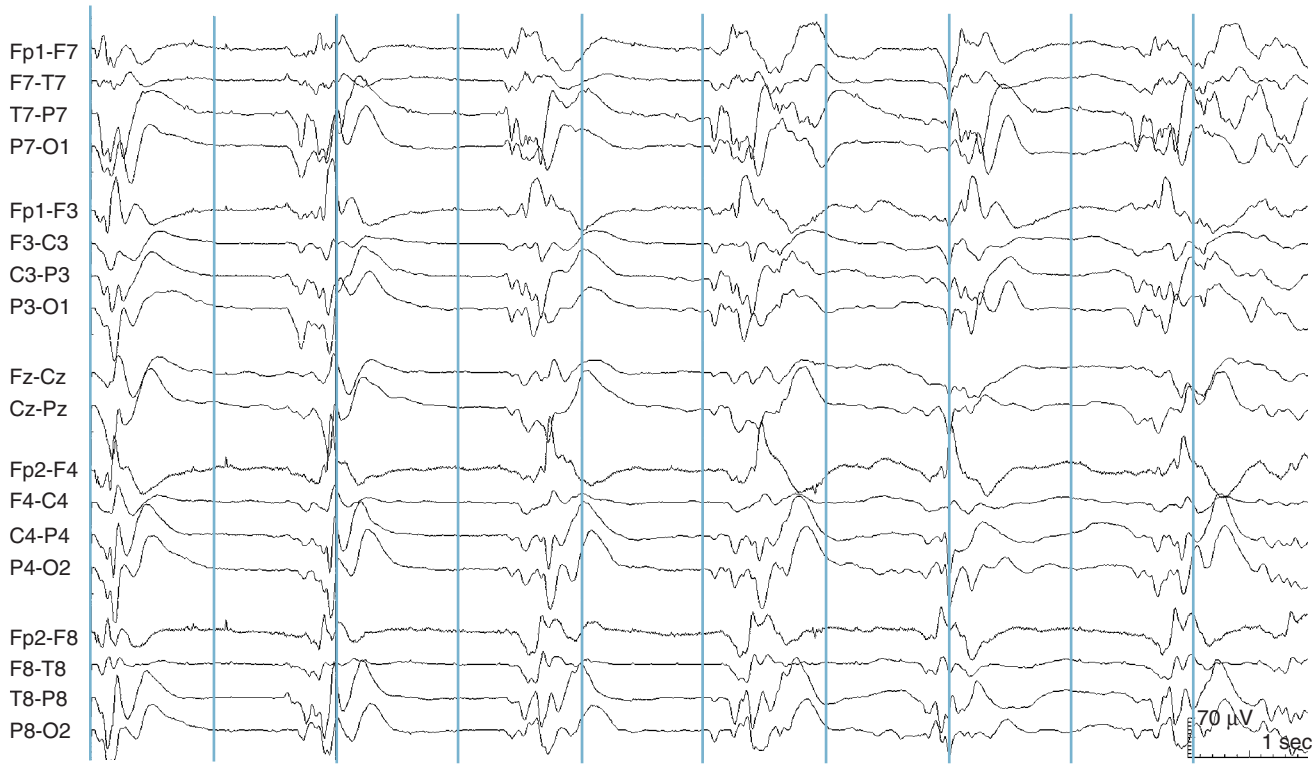


Figure 9-32 Periodic short-interval diffuse discharges (PSIDDs) are characterized by a repetitive discharge that occurs synchronously over both hemispheres with a relatively short interdischarge interval (approximately 1.5 seconds in this example). In this patient, the diffuse discharges have a relatively complex morphology.

Triphasic Waves

Triphasic waves may be considered a subset of PSIDDs. Triphasic waves are medium- to high-voltage complexes with a characteristic triphasic pattern, often with a sequence of negative–positive–negative deflections, repeating at rate of 1.5 to 2.5 Hz (see Figure 9-33). The first phase of the deflection has been said to resemble a blunted spike and the whole complex may resemble a blunted spike-wave discharge, although usually the initial deflection is too rounded for the complex to be considered a true spike-wave discharge. Triphasic waves usually occur diffusely and symmetrically, often with a frontal predominance. The most frequent clinical association is with hepatic encephalopathy or hepatic coma, but other metabolic derangements (such as renal failure) may also be associated with triphasic waves. Triphasic waves may also show the unusual manifestation of an anterior-to-posterior time lag in which the wave is seen frontally up to 200 ms before it is seen posteriorly.

Nonconvulsive Status Epilepticus

Generalized nonconvulsive status epilepticus (NCSE) may also technically fulfill the criteria for PSIDDs in that this pattern may consist of repetitive, diffuse sharp discharges (see Figure 9-34). Both the EEG appearance and the clinical context may aid in distinguishing between NCSE and other causes of PSIDDs, although at times the distinction is difficult. Because NCSE is an epileptic seizure pattern, its presence requires a different treatment approach aimed at terminating the seizure activity, usually with aggressive use of antiepileptic medications.

Creutzfeldt-Jakob Disease

Creutzfeldt-Jakob disease (CJD) is a rare disorder that consists of a progressive dementia characteristically associated with repetitive myoclonus. The disease may be transmitted by infectious particles called prions. CJD is often associated with the finding of PSIDDs in the form of sharp wave complexes, often with triphasic morphology (see Figure 9-35). The myoclonus typically associated with this disease may occur synchronously with the PSIDDs. The findings of PSIDDs and myoclonus alone cannot establish the diagnosis of CJD. Rather, the distinctive clinical picture of CJD should be considered supported by finding this characteristic pattern in the EEG.

PLIDDs

Periodic long-interval diffuse discharges, or PLIDDs, refer to repetitive waveforms that are separated by intervals of four to 20 seconds' duration. PLIDDs are the hallmark finding of subacute sclerosing panencephalitis (SSPE), a rare disorder related to a slow measles infection of the central nervous system. Measles

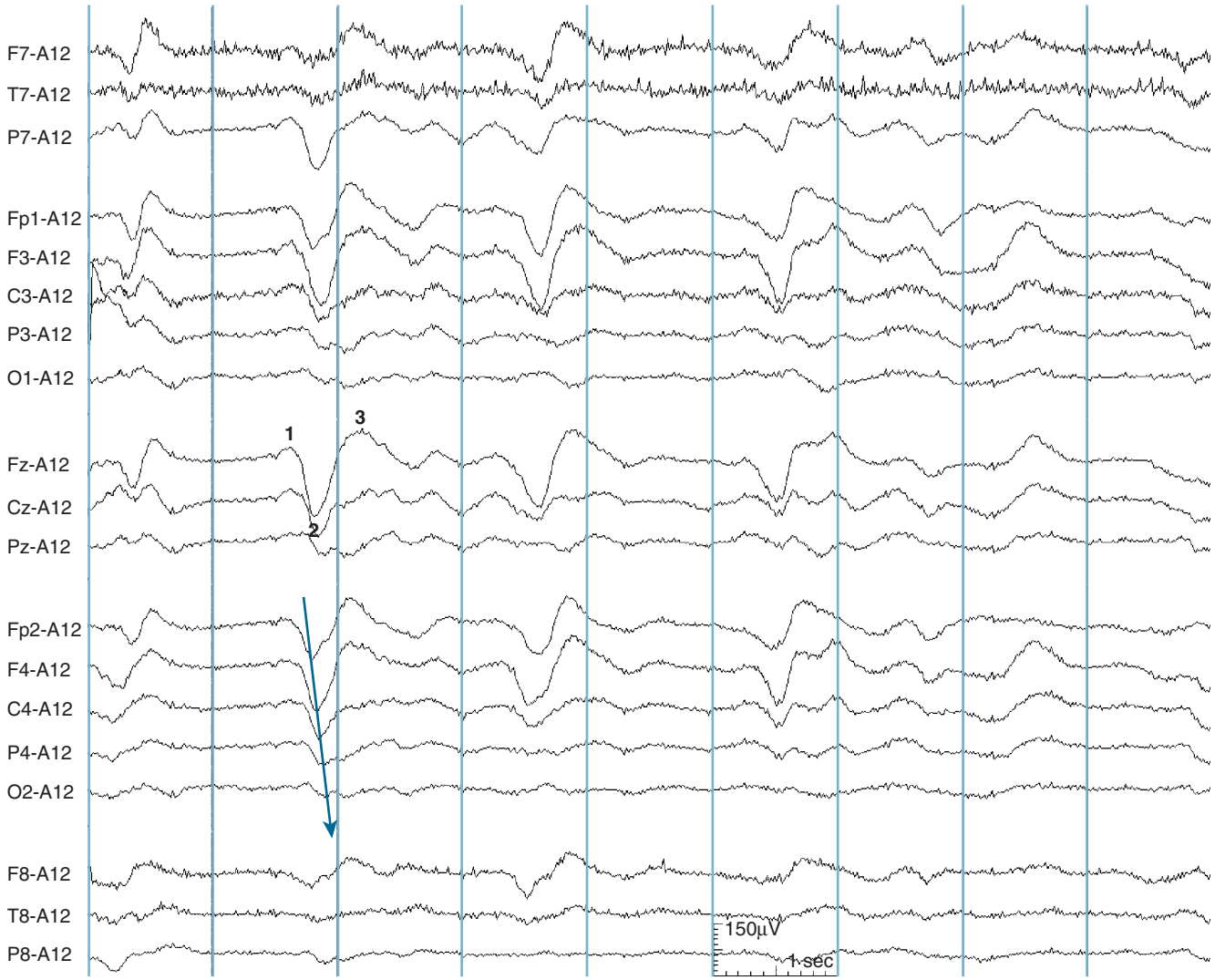


Figure 9-33 An example of triphasic waves is shown in a patient with hepatic failure. The waves are displayed in a referential montage using a reference that consists of the average of both earlobes, A1 and A2. The three separate phases of the triphasic waves ("up," "down," and "up") are labeled "1," "2," and "3," respectively. The characteristic time shift of triphasic waves is highlighted by the gray arrow. Note that the trough of the wave begins earlier in the frontal area and is delayed by several milliseconds in each successively more posterior channel. Although a classic feature of triphasic waves, this back-to-front delay is not seen in all cases of triphasic waves.

immunization has almost completely wiped out this disease in many parts of the world, although a low rate of SSPE persists even in the immunized population; a possible relationship between the slow virus infection and the vaccine is unclear. This fatal disorder begins with mental status changes and behavioral deterioration followed by dementia and myoclonic seizures that correlate with the periodic discharges. Over weeks or months, the disorder inexorably progresses to a vegetative state and death. The presence of such discharges in the appropriate clinical context is considered highly characteristic of SSPE.

Burst suppression patterns related to anoxia or drugs (including pharmacologic agents used in the intensive care unit) may fall into the category of PLIDDs (see Figure 9-36). Because the interval between bursts in burst suppression may be both less than or greater than 4 seconds in duration, burst-suppression patterns may fall into the category of either PLIDDs or PSIDDs. Burst suppression patterns are discussed in more detail in Chapter 12, "EEG Patterns in Stupor and Coma."

ABNORMAL SLEEP STRUCTURE

Abnormalities of sleep structure fall into a variety of categories, including sleep disorders that are beyond the scope of this text. Abnormal sleep structure, including the absence of normal sleep elements or the abnormal ordering of sleep staging, may be the result of an abnormal central nervous system, a disease process, or a pharmacologic effect.

Occasionally, patients are seen who lack the expected synchrony seen in normally occurring sleep elements. This type of abnormality has been associated with agenesis of the corpus callosum (see Figures 9-37, 9-38, and 9-39). Not all patients with agenesis of the corpus callosum manifest this finding, however.

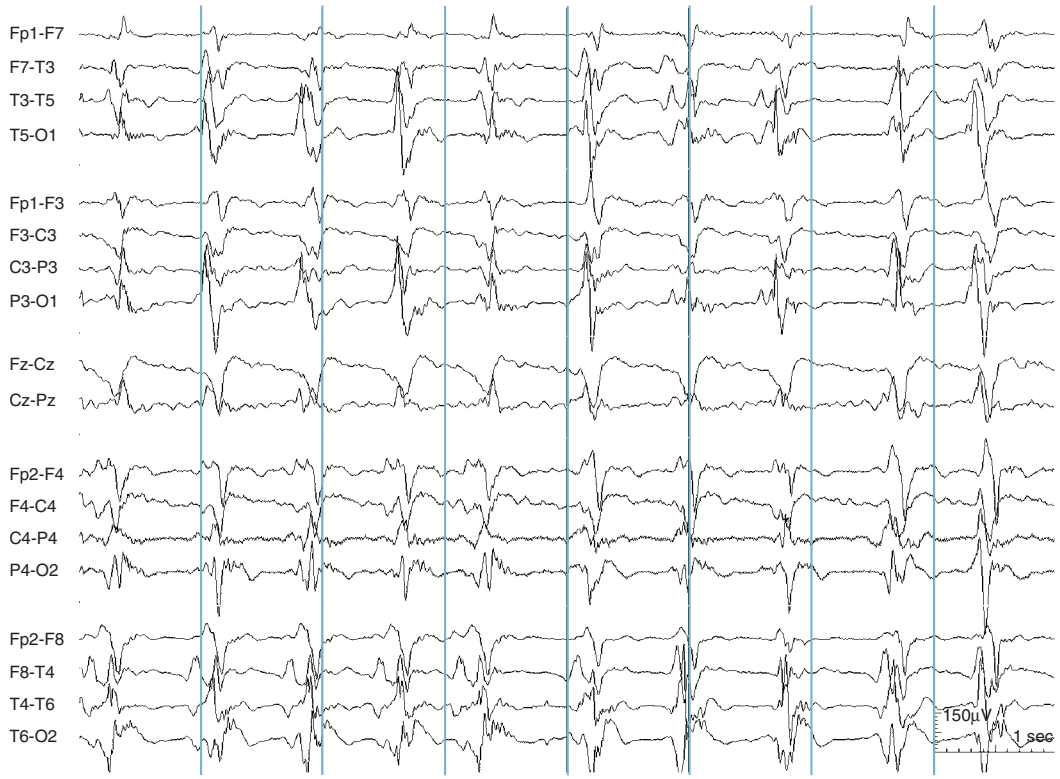


Figure 9-34 During the episode of nonconvulsive status epilepticus shown in this figure, this 19-month-old girl was motionless and unresponsive. This seizure pattern was eventually terminated with anticonvulsant medications.

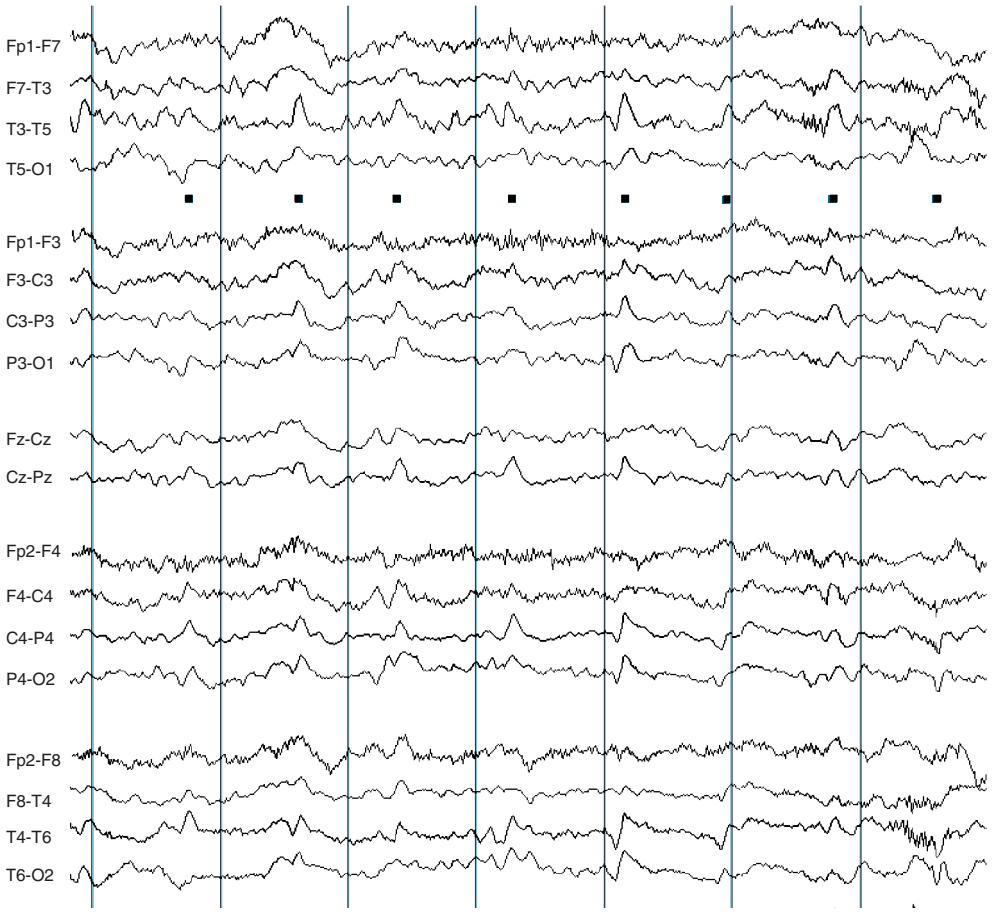


Figure 9-35 Periodic short interval diffuse discharges (PSIDDs) ranging from 30 to 50 µV are seen in a patient with Creutzfeld-Jakob disease (dots). Note that the field of the discharge is diffuse, although this patient's variant of the disorder is posteriorly predominant. (Image courtesy of Dr. Edward Bromfield and Dr. Barbara Dworetsky.)

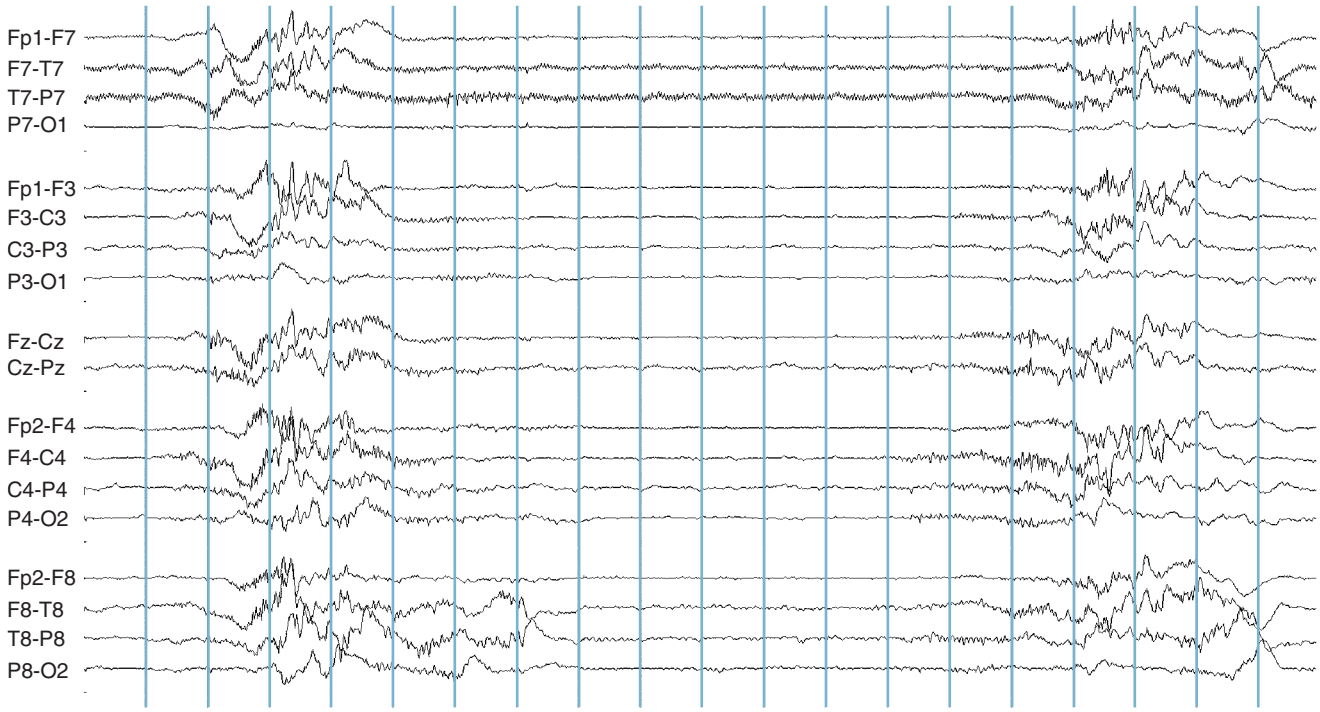


Figure 9-36 This burst-suppression pattern consists of 2- to 3-second bursts of mixed activity that includes sharp elements, separated by several seconds of diffuse suppression. Technically, this example falls into the category of periodic long interval diffuse discharges because of the long interburst interval, but the term *burst-suppression* is much more commonly used for this type of pattern.

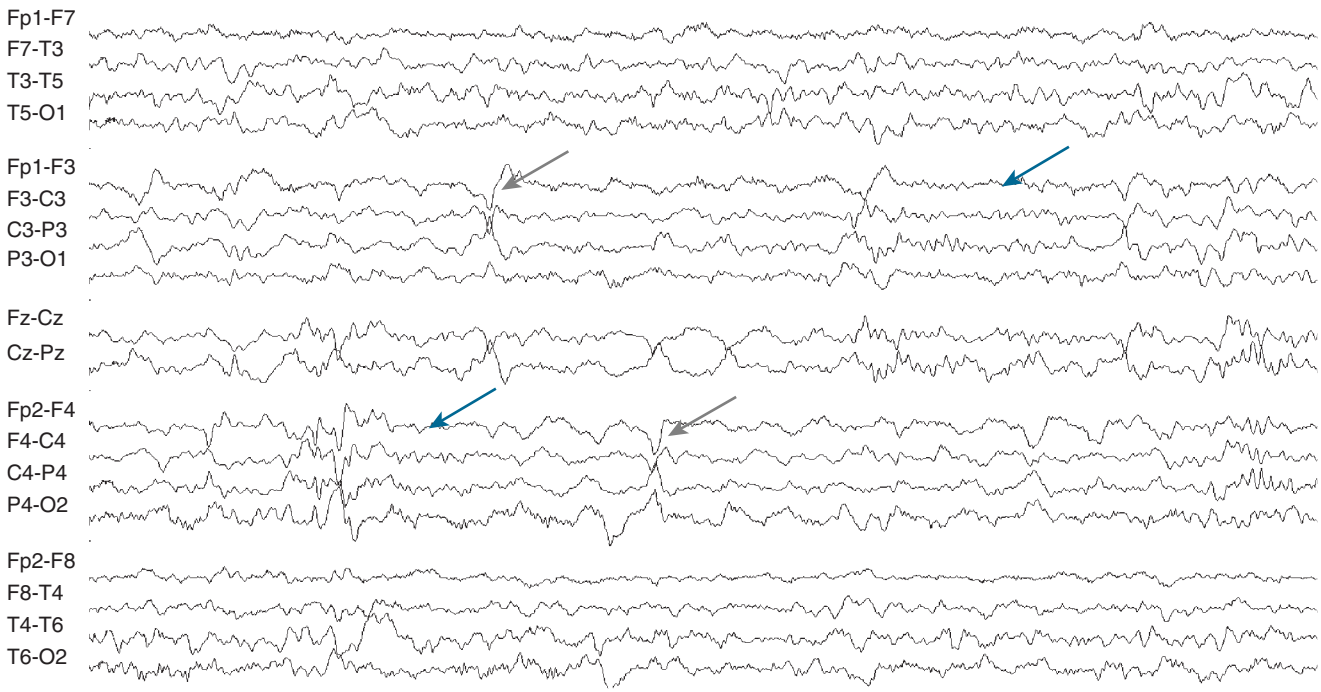


Figure 9-37 Both vertex waves (gray arrows) and spindles (blue arrows) are asynchronous in this 3-year-old boy with agenesis of the corpus callosum. Although it seems logical that vertex waves and spindles might not be synchronous in individuals who lack a corpus callosum, some patients with agenesis of the corpus callosum still do manifest vertex wave and spindle synchrony. Likewise, the presence of spindle and vertex wave asynchrony does not always indicate absence of the corpus callosum.

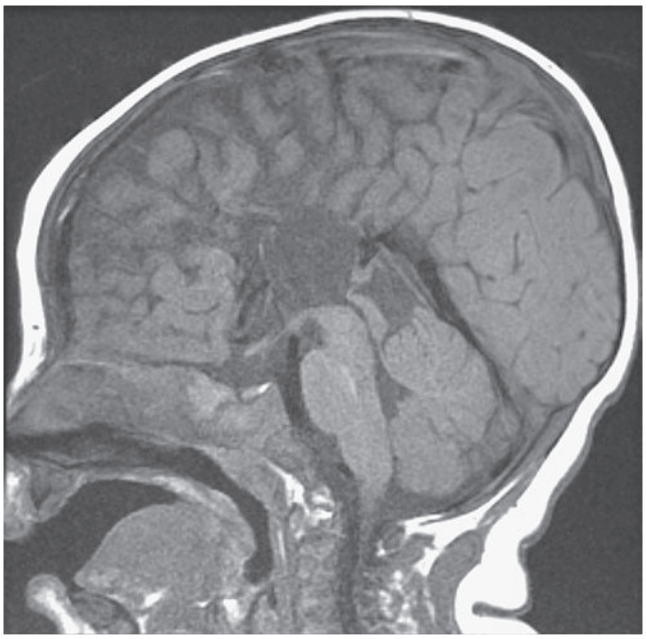


Figure 9-38 The magnetic resonance imaging (MRI) scan of the patient whose EEG was shown in the previous figure demonstrates absence of the corpus callosum. The next figure shows the MRI of an individual with an intact corpus callosum for comparison.

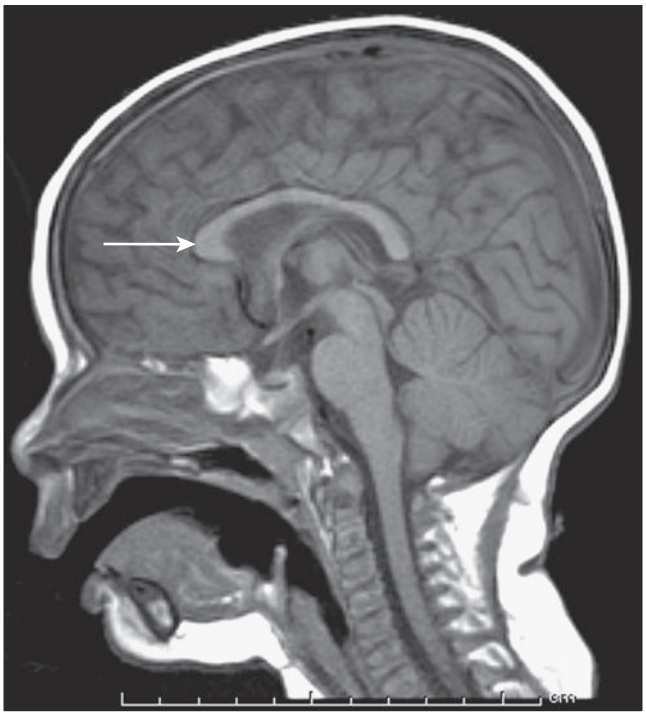


Figure 9-39 A magnetic resonance imaging scan of an individual with an intact corpus callosum is shown for comparison to the previous figure. The arrow indicates the lighter-colored horseshoe-shaped corpus callosum cut in cross-section. The corpus callosum is the large bundle of neurons that joins the two hemispheres. This structure is absent in the previous figure.

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Chapter

10 The EEG in Epilepsy

There are many indications for EEG testing, but the most common reason that an EEG is obtained is to assist in the diagnosis of seizures and epilepsy. Although the history is still the cornerstone of the diagnostic process, in some cases, the results of the EEG can make an equal or even greater contribution to the diagnosis of seizures, especially when some elements of the history are unclear. The EEG is a particularly powerful tool in helping to classify seizure types. Previous chapters in this book were written from the point of view of various EEG findings and discussed their potential clinical implications, including possible associations with epilepsy. This chapter provides a review of selected seizure types and seizure/epilepsy syndromes and discusses the EEG findings most commonly associated with each.

SEIZURE TYPES AND SEIZURE SYNDROME

Seizure Types

The distinction between *seizure types* and *seizure syndromes* is central to both the practices of clinical epileptology and clinical electroencephalography. It is worthwhile to consider the distinction between the two and how the diagnosis of each is made. The term *seizure type* refers to the classification of an individual seizure event. Ideally, a seizure type can be discerned from knowing three key features regarding the event: the patient's appearance during the seizure, the patient's subjective description (if any) of what the experience of the seizure was like, and the appearance of the EEG recording made at the time of the event. The details of all three of these features, of course, are not always available to the clinician, especially the simultaneous EEG recording.

The following hypothetical example illustrates how these three key features are used: a patient experiences an event that starts with a subjective report of a feeling of fear. Next, observers report that the patient begins to stare and is unresponsive, followed by rhythmic jerking of the left arm. Simultaneously, an EEG is recorded that shows a rhythmic discharge starting in the right temporal lobe and evolving to include much of the right hemisphere over a brief period of time (see Figure 10-1, *A, B,* and *C*). The combination of psychic aura reported by the patient, staring, the specific motor phenomena reported by observers, and observation of an EEG seizure discharge that starts in the right temporal lobe all establish the diagnosis of a specific seizure type: a complex partial seizure arising from the right temporal lobe. Note that the age and previous history of the patient are not of primary importance in diagnosing seizure type; rather, the behaviors observed during the episode and any recordings made at the time of the event are the most important elements in defining seizure type. We can assign the seizure type "complex partial seizure arising from the right temporal lobe" or "right temporal lobe seizure" without knowing whether the seizures are occurring in a broader context of possible posttraumatic epilepsy or cryptogenic temporal lobe epilepsy. This contrasts with the approach to diagnosing seizure syndromes, described next.

Seizure Syndromes

To discern a patient's *seizure syndrome*, it is useful to know the patient's age, history, neurodevelopmental or cognitive status, and the seizure types he or she has experienced. For instance, a 28-year-old man with normal intellect and neurologic examination and a history generalized convulsions and myoclonic jerks since the early teenage years likely has the diagnosis of the seizure syndrome known as juvenile myoclonic epilepsy. In this example, the patient's seizure syndrome includes two seizure types: myoclonic seizures and generalized tonic-clonic seizures. If necessary, the diagnosis of these seizure types could be confirmed by patient and observer descriptions and simultaneous video/EEG recording of the individual seizure events. The diagnosis of the seizure syndrome of juvenile myoclonic epilepsy, however, is best established by knowing the age at onset of the seizures, the seizure types the patient has, and additional pertinent history.

CLASSIFICATION OF SEIZURE TYPES

The most frequently used classification of seizure types was established by a committee of the International League Against Epilepsy (ILAE) in 1981. This classification divides seizures into partial and generalized

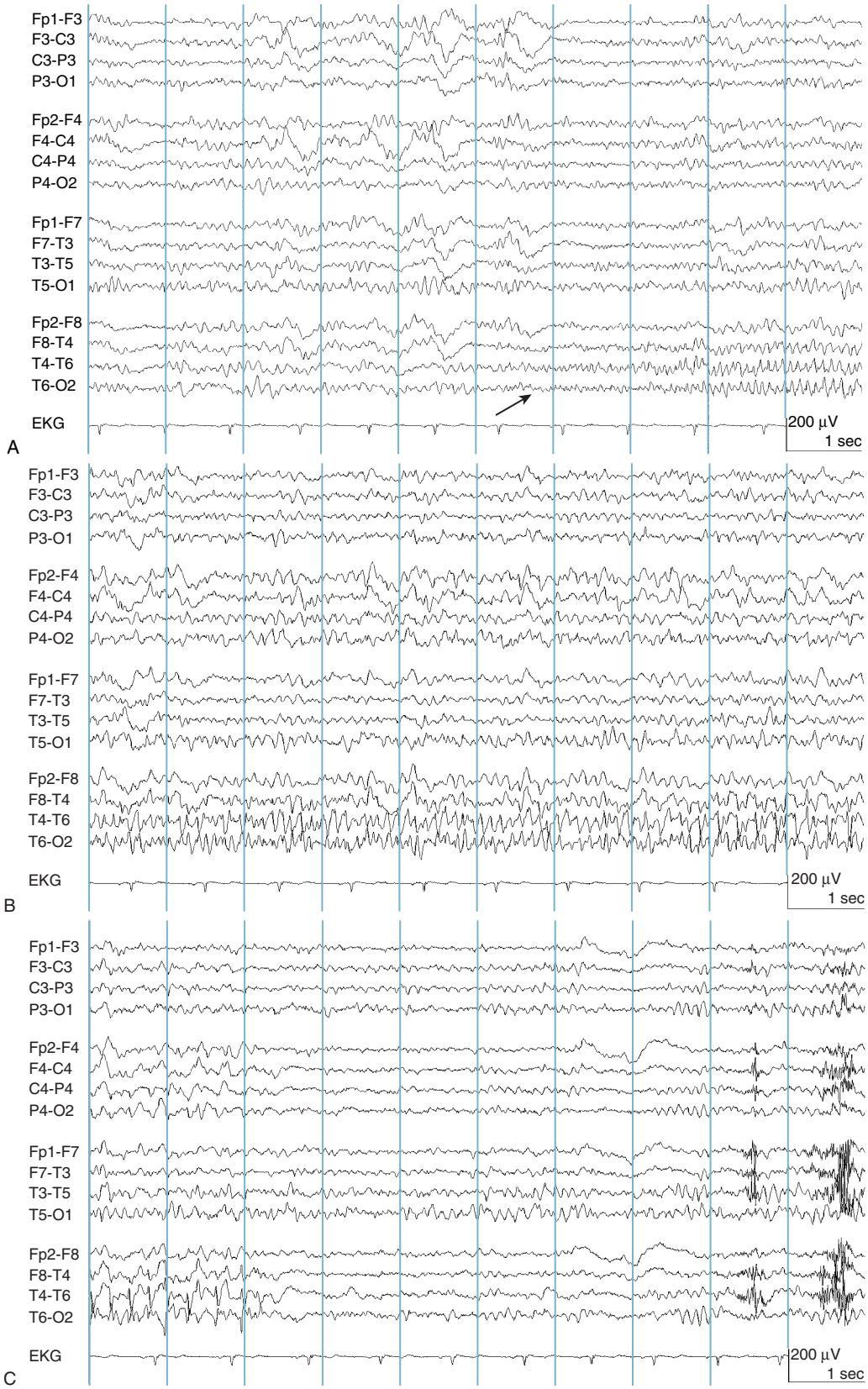

Figure 10-1 A focal seizure beginning in the right temporal lobe is shown (montage setup: left parasagittal chain over right parasagittal chain, left temporal chain over right temporal chain). The arrow indicates the location of seizure onset which consists of a low-voltage, fast rhythm initially confined to the bottom two channels (compare with homologous channels on the left: C3-P3 and P3-O1). This fast rhythm then spreads through the right temporal chain, subsequently involving the right parasagittal chain as well. The discharge slows in frequency and increases in voltage throughout its course before abruptly coming to an end.

categories and a smaller unclassified category. The process of splitting seizure types up into generalized or partial categories is the first step in seizure classification. Each category of seizure type is described briefly in this section, along with its characteristic clinical and EEG findings. The broad schema of the classification is shown in Figure 10-2.

Partial Seizures

Because a seizure can arise from nearly any location on the cortical surface, the range of potential seizure manifestations is quite diverse. Partial seizures may involve motor findings (such as jerking or stiffening of a limb), sensory findings (such as a sensation of tingling or pain over a region of the body, hearing a sound, smelling an odor, or seeing brightly colored shapes), psychic features (such as a sensation of fear or déjà vu), or autonomic findings (such as sweating or palpitations). It should also be kept in mind that a significant portion of the cortical surface is functionally relatively silent. When a seizure discharge starts in one of these "silent" cortical areas, it is possible for the discharge to occur on the cortical surface without clinical change as a clinically silent seizure rather than a clinical seizure. Frequently, a seizure discharge may begin in one of these "silent" areas and subsequently propagate to other areas such as the motor strip, at which time obvious clinical signs may appear such as clonic jerking of an extremity. Thus, when a patient manifests clonic jerking, we can infer that the seizure discharge has involved the motor strip, but this is not a guarantee that the seizure discharge *originated* in the motor strip—the seizure discharge may have started elsewhere and propagated to the motor strip. Figures 10-3 and 10-4, show examples of how two focal-onset seizures propagate.

Simple Partial Seizures Versus Complex Partial Seizures

Partial seizures are further subdivided into *simple* partial seizures and *complex* partial seizures. A complex partial seizure is a partial seizure associated with diminished consciousness or responsiveness. Some of the partial seizures may be associated with completely retained responsiveness, such as a simple partial seizure involving clonic spasms of the face or hand—the patient may remain completely alert and aware during such a seizure. The majority of partial seizures, however, are associated with diminished responsiveness and are properly classified as complex partial seizures. Indeed, it is only during a minority of partial seizures that complete responsiveness is maintained. During complex partial seizures, the patient may appear awake but is unresponsive (or only minimally responsive) to stimulation. Staring behaviors frequently indicate reduced responsiveness during complex partial seizures. Only rarely does a patient appear to lose consciousness completely during a complex partial seizure.

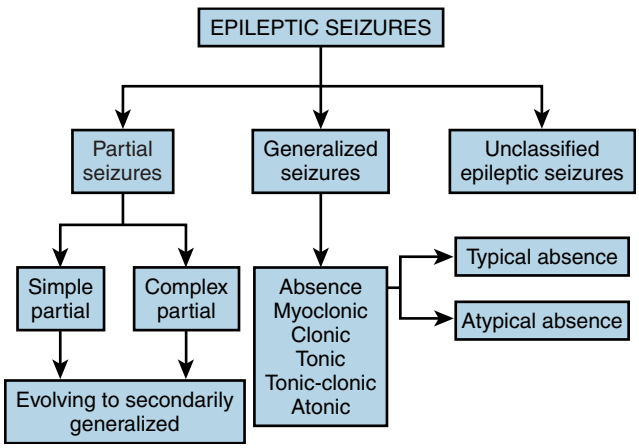

Figure 10-2 Seizure classification table.

Epileptic Auras

An epileptic aura is a subjective sensation that heralds the onset of a seizure. The sensations can be diverse, such as hearing a buzzing sound in the ears, experiencing déjà vu, or having a feeling of nausea immediately preceding a seizure. This initial, comparatively minor feature may then be followed by more dramatic seizure manifestations. Strictly speaking, an aura is not a preseizure warning or "prodrome." Rather, it represents the onset of the seizure itself. The onset of the aura is coincident with the onset of the seizure discharge.

During the aura phase of a seizure, the epileptic seizure discharge is usually confined to a small area, often in the temporal lobe. As it spreads out of its confined area, the clinical seizure manifestations may become more dramatic. Because the aura should properly be considered the onset of a clinical seizure, a true epileptic aura can be counted as a seizure. In some cases, the aura may not progress and represents the seizure in its entirety.

Recordable EEG Patterns Associated With Partial Seizures

Partial seizures are distinguished by EEG patterns that involve only subsets of the brain. In comparison, the EEG patterns of generalized seizures involve all brain areas at once. Although most epileptic seizures can be recorded using standard scalp electrodes, in some cases a seizure discharge may occur in a cortical area that is not readily accessible to recording with conventional electrodes. These include such areas as the mesial surfaces of the frontal, parietal, and occipital lobes, the orbitofrontal surface of the frontal lobe, the basal occipital and temporal lobes, and the mesial surface of the temporal lobe and insula, among others (see Figures 10-5 through 10-9). Therefore, most, but not all partial seizures are well recorded at the scalp. For these reasons, a negative EEG recording cannot, in and of itself, exclude the diagnosis of an epileptic seizure. In these occasional cases of definite epileptic seizure with negative scalp recordings, other factors must be taken into account to make the correct diagnosis, such as the specific features of the patient event and the history. In cases of partial epileptic seizures associated with

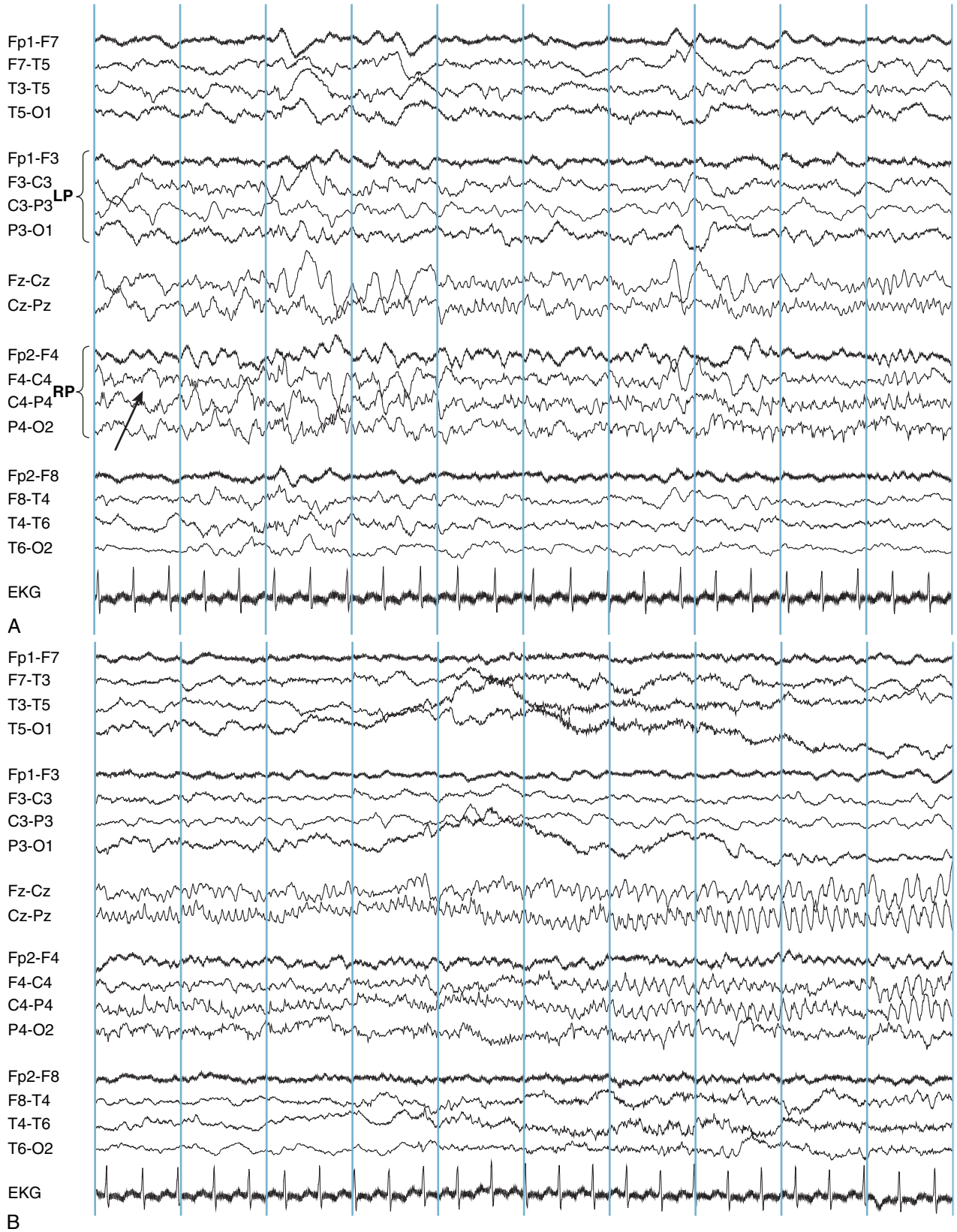

Figure 10-3 This seizure discharge begins in the right parasagittal (RP) area in the form of low-voltage, very fast activity (arrow) and spreads quickly to the midline electrodes (Fz-Cz and Cz-Pz). The seizure onset is detected by noting the first asymmetry between the right parasagittal and left parasagittal (LP) chains. The discharge then becomes most prominent and well formed in the midline channels (Fz-Cz and Cz-Pz) and involves the right parasagittal area, with the field spreading to the right temporal chain before termination.

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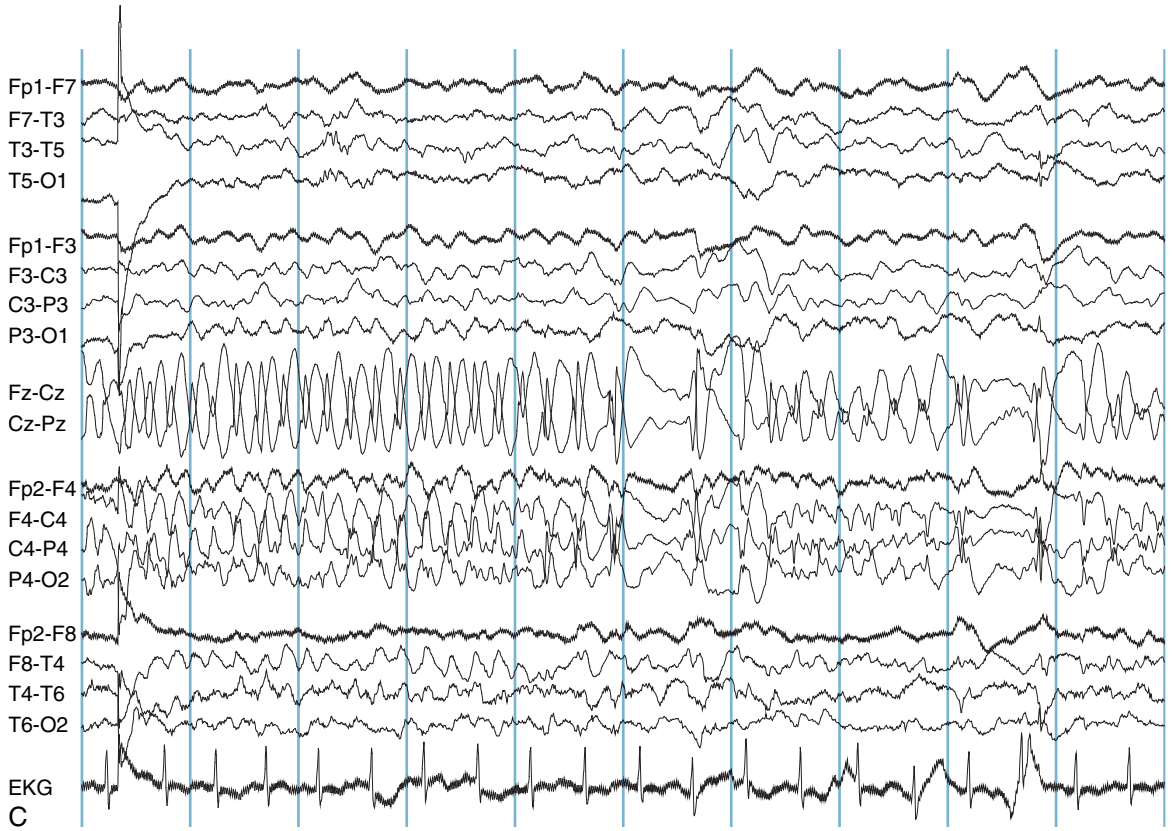


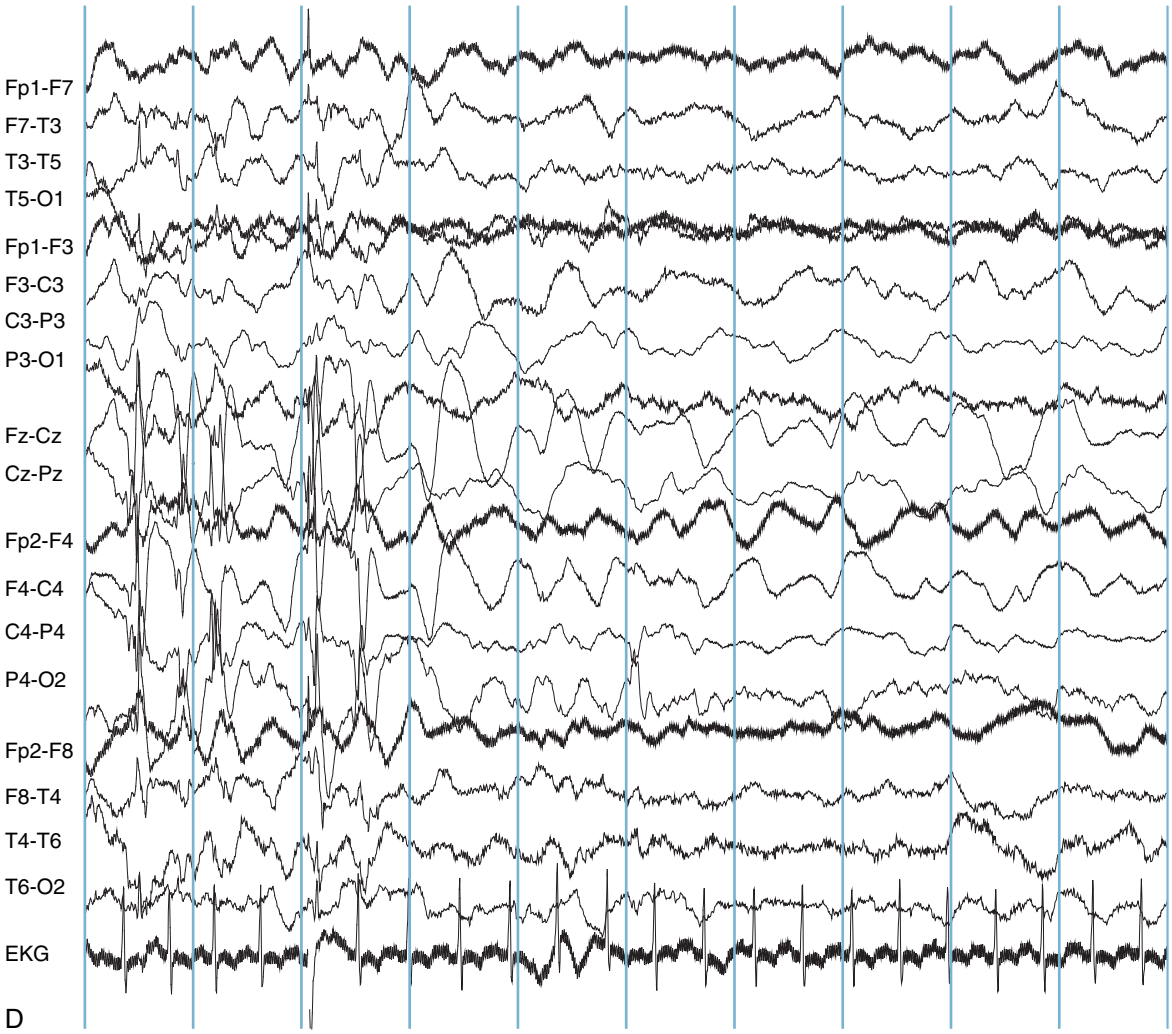


Figure 10-3, cont'd

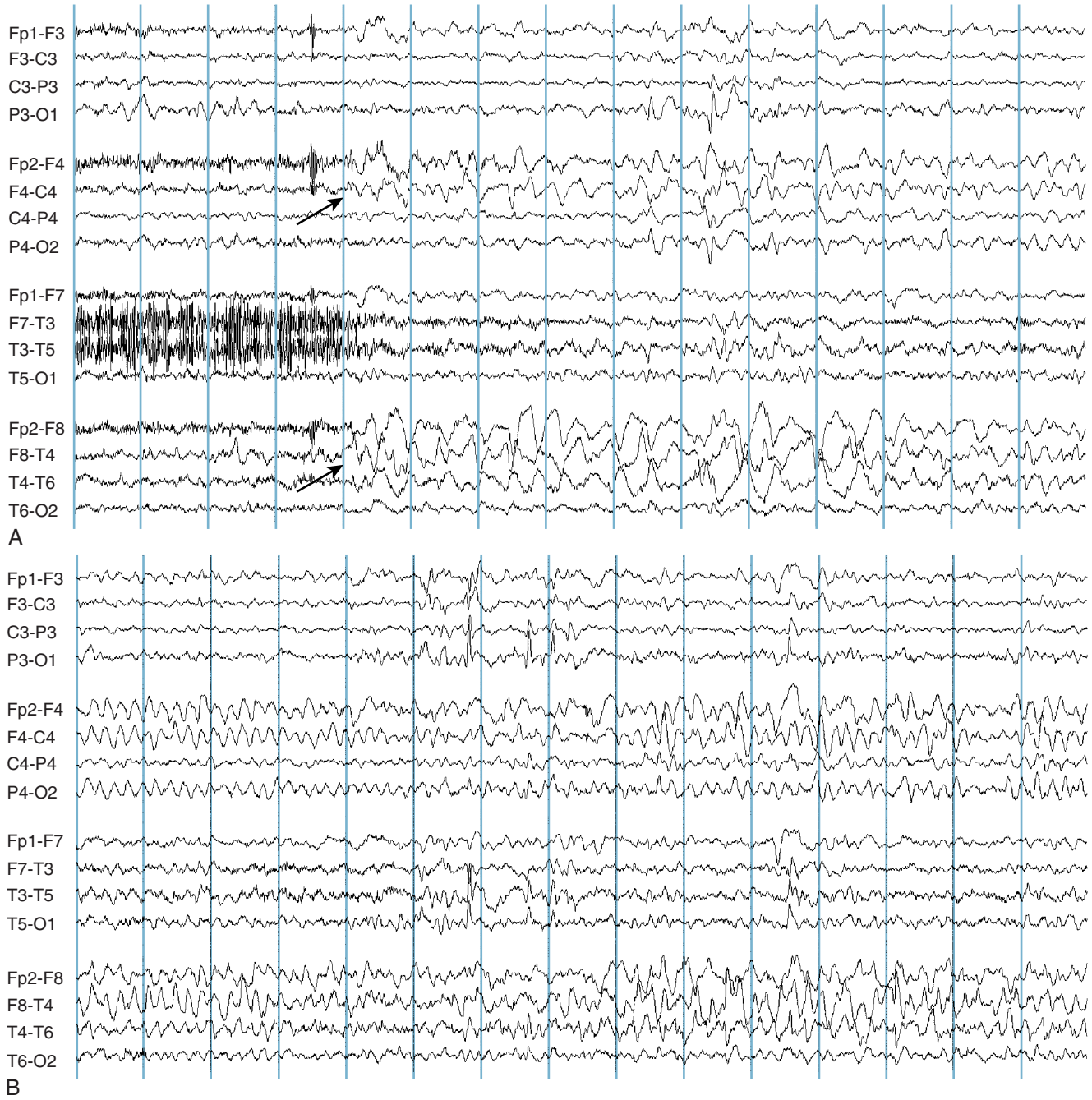


Figure 10-4 A seizure discharge (arrows) begins in the right temporal chain and right frontal area (montage setup: left parasagittal chain over right parasagittal chain, left temporal chain over right temporal chain). Initially, the seizure waveforms consist of higher voltage sharp forms, but as the seizure spreads to involve the whole of the right hemisphere, it attains a more rounded or sinusoidal morphology. This is an example of a discharge in which the frequency initially accelerates, then decelerates before termination (final termination not shown).

a negative EEG, the definition implies that there is some *theoretical* electrode placement that could record the seizure discharge, even if that placement location would have to be deep within the brain.

The EEG patterns associated with partial seizures usually consist of a rhythmic, sharp discharge over the affected area (e.g., spike or spike-wave discharges) as shown in the previous figures. However, partial seizure recordings do not only appear as a train of spikes. Especially when seizure sources are located deeper in the brain and at some distance from the recording electrode, the seizure may only appear as a rhythmic focal slow wave without obvious sharp features when recorded from the scalp. In the case of partial seizures, the epileptic discharge is usually unilateral. Although a unilateral partial seizure discharge may spread incrementally through the primary involved hemisphere, at such time as the discharge might cross to the opposite hemisphere, both hemispheres become simultaneously engulfed with seizure activity; there is no incremental

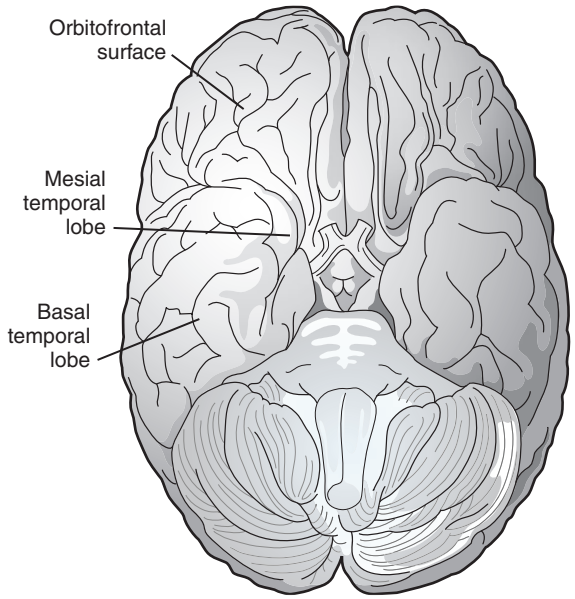


Figure 10-5 The basal surfaces of the brain are at some distance from the scalp and difficult to record well using routine techniques. The orbitofrontal surface of the brain lies on the floor of the anterior cranial fossa, above the eyes. The basal temporal lobe lies on the bone of the middle cranial fossa and the mesial temporal lobe, including the amygdala and hippocampus, also lie at some distance from the scalp electrodes.

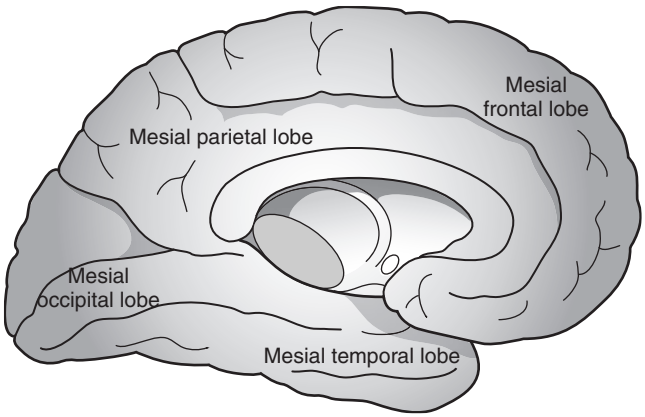


Figure 10-6 A section through the sagittal midline of the brain shows the mesial surfaces of the frontal, parietal, occipital, and temporal lobes which can be difficult to record.

spread through the opposite hemisphere. This process is referred to as secondary generalization.

An important exception to this rule is the example of temporal lobe seizures in which the seizure discharge may spread from one temporal lobe to the other without simultaneous involvement of the remainder of the hemispheres (see Figure 10-10). Therefore, in most cases, after the discharge becomes bilateral, the whole of both hemispheres is engulfed with the discharge and the discharge can be considered to have generalized. Complex partial seizures with bilateral temporal involvement represent the exception to this rule: the less typical example of a bilateral seizure discharge that is not truly generalized.

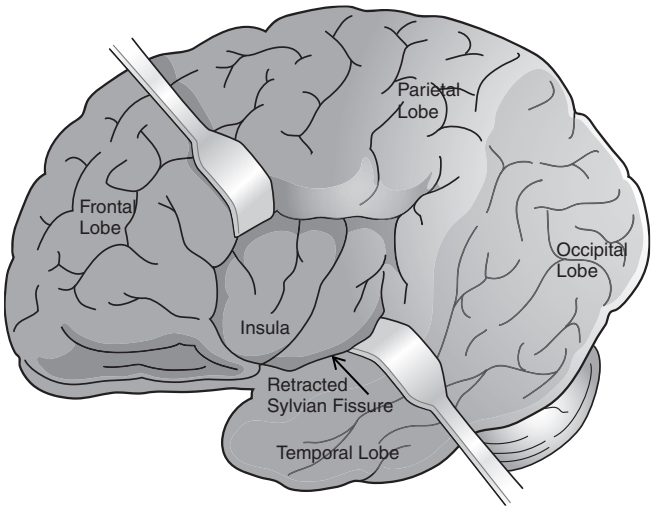


Figure 10-7 The insula (literally "island") represents an infolding of cerebral cortex covered by the lips (opercula) of the Sylvian fissure. The frontal and temporal opercula are retracted to reveal this hidden area of cortex.

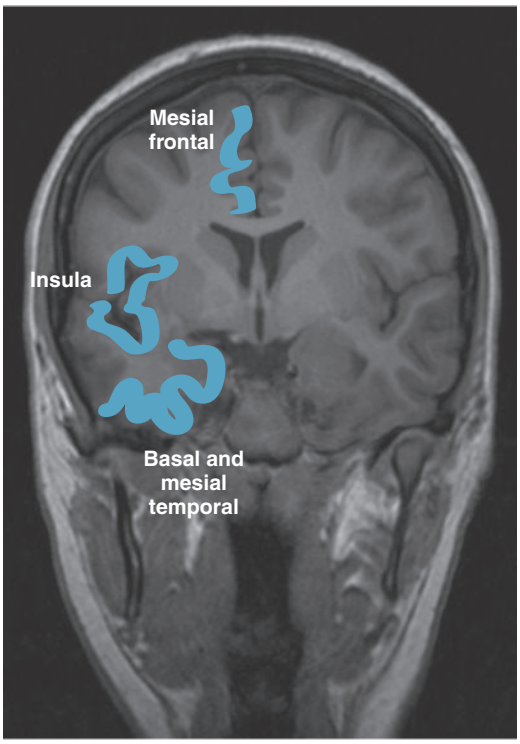


Figure 10-8 A coronal section of brain is seen on magnetic resonance imaging scan. The mesial frontal lobe, insula, and basal and mesial temporal lobes are highlighted.

Partial Seizures With Secondary Generalization

As described earlier, a seizure discharge may start focally and subsequently spread to involve all brain areas (generalize). This flow of the discharge from a subset of cerebral cortex to all of cerebral cortex is reflected both by the spread of the recorded discharge from a subset of EEG channels to all EEG channels and also by an evolution of the patient's seizure behavior

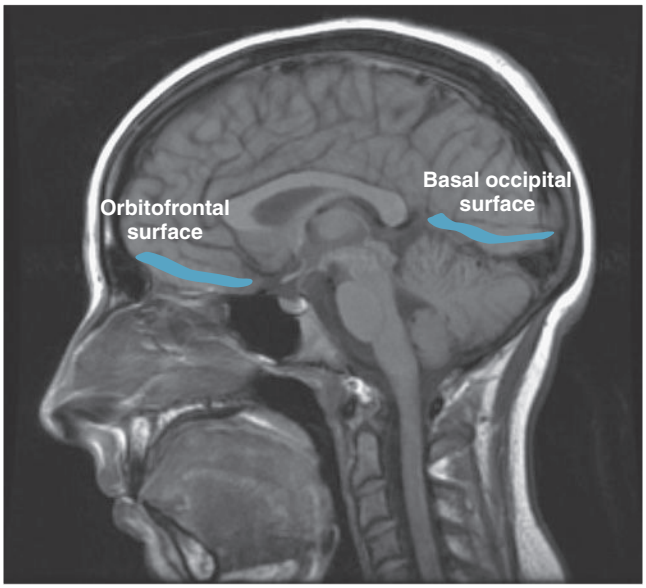


Figure 10-9 The basal frontal and occipital lobes are highlighted on a sagittal MRI scan. The gyri of the mesial surface of the cerebral hemisphere (frontal, parietal, and occipital) are also visible.

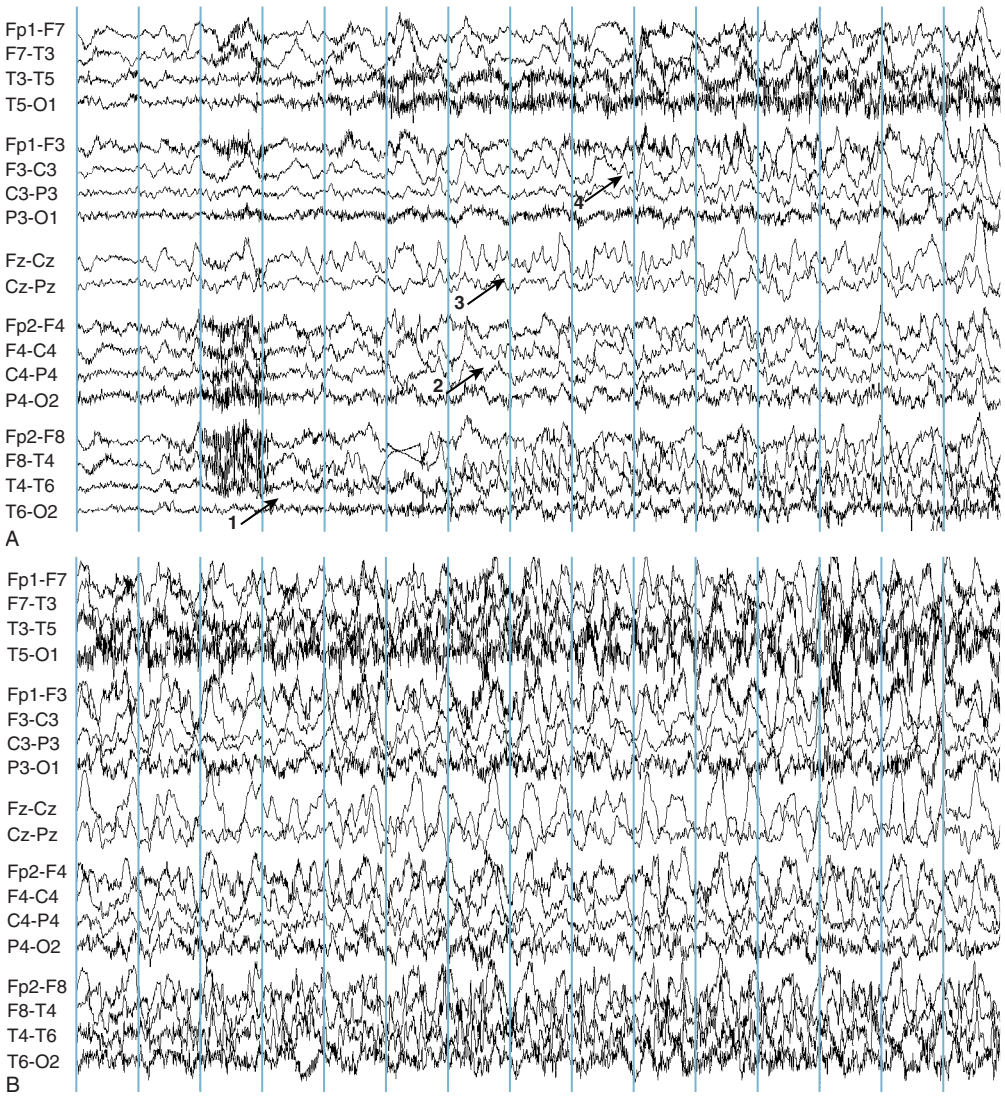
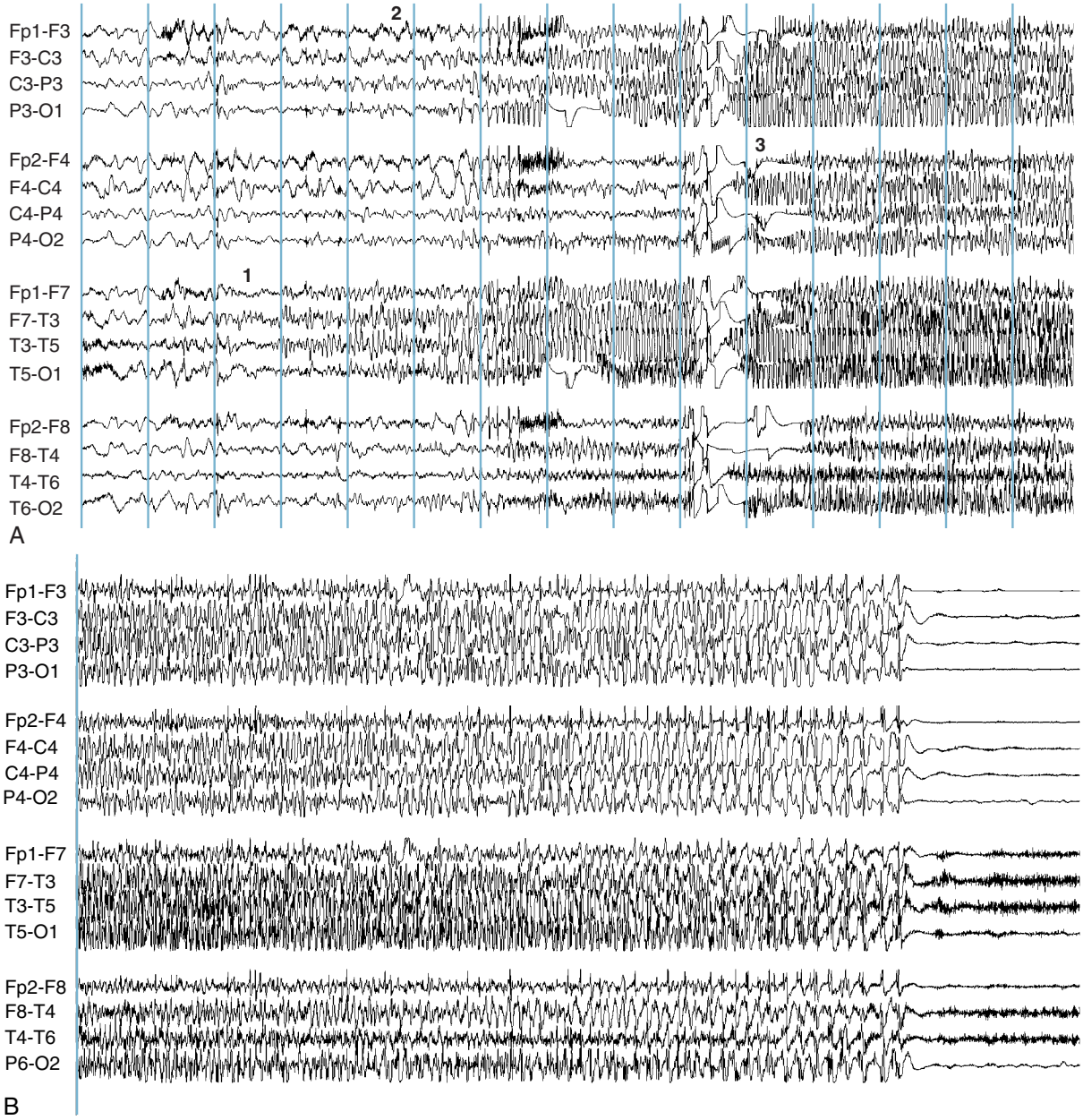


Figure 10-10 The beginning of the low-voltage sharp seizure discharge that is evident on the second half of the first page can be traced back to the right midtemporal area where the muscle artifact stops (1). After 3 seconds, the discharge spreads to the right parasagittal area (2) and then quickly to the midline (3). After 2 more seconds, the discharge has become bilateral and can be seen in the left parasagittal area (4). The sharp waves may be present simultaneously in the left temporal chain as well but would be difficult to discern because of the muscle and motion artifact in that area. As it nears its end, the seizure discharge increases in voltage and slows in frequency. The increased muscle and motion artifact is not unexpected as the clinical manifestation of seizures often includes muscle tensing and patient movement.

from a partial manifestation to involvement of the whole body. For instance, in the classic example of the type of seizure referred to as a "Jacksonian march," a patient's seizure may begin with clonic contractions in the right hand and arm and subsequently spread to the right face and leg. Thereafter, it may spread to the opposite side of the body so that bilaterally synchronous clonic activity of the whole body is seen. This clinical progression is mirrored by an electrographic evolution of the discharge from a small area in the left hemisphere, which includes the portion of the motor strip that is associated with the left hand, to the whole of the left hemisphere and then, finally, to involvement of both hemispheres (see Figure 10-11).

Partial seizures that secondarily generalize are classified among the partial seizures rather than the generalized seizures for diagnostic reasons. Partial seizures that do not generalize and partial seizures that do secondarily generalize have the same list of possible causes. In comparison, the list of causes of generalized seizures is distinctly different from the list of causes of partial-onset seizures. The tendency to a partial seizure to secondarily generalize usually has little to do with the etiology of the seizure.



Figures 10-11 A seizure in a child with severe myoclonic epilepsy of infancy (SMEI, or Dravet syndrome) is shown that begins in the left temporal chain (1) and quickly spreads to the left parasagittal chain (2). After a brief period of amplifier blocking (possibly related to patient movement), the discharge becomes bilateral (3). Although SMEI is related to the generalized epilepsies, this patient had similar focal seizure onsets arising from the opposite (right) side at other times.

Generalized Seizures

The classification of generalized seizures includes absence seizures, myoclonic seizures, clonic seizures, tonic seizures, tonic-clonic seizures, and atonic (astatic) seizures. Even though all of these seizure types fall into the category of generalized seizures, the manifestations of the different types of generalized seizures differ significantly.

Typical Absence Seizures

In its purest form, the sole clinical manifestation of the absence seizure is a pure stare. Absence seizures typically last some 3 to 15 seconds and are characteristically associated with complete unawareness of the environment. In practice, there is no real lower limit to the duration of an absence seizure apart from the ability of the observer or the patient to document unawareness or unresponsiveness for very brief periods of time; it may not be practical to document lack of awareness during discharges that last less than 1 second. Most absence seizures are brief, but they can be of any duration. To distinguish them from atypical absence seizures (described later), the term *typical absence seizure* may be used. When the "typical" or "atypical" modifier is absent, typical absence seizure is usually assumed. In the past, some have used the term "absence" to denote any seizure associated with staring. In modern usage, the term absence seizure refers to staring seizures associated with generalized spike-wave discharges as described subsequently—complex partial seizures associated with staring are excluded.

A simple typical absence seizure consists of staring alone. The most common modification of the pure stare of the simple typical absence seizure is the addition of rhythmic eye blinking occurring approximately three times per second (each blink occurring in synchrony with the generalized spike-wave discharge seen on EEG). In fact, this sometimes subtle, rhythmic clonic movements of the eyelids occurs with the majority of typical absence seizures; pure staring is relatively uncommon. Less frequent additions to the pure stare of typical absence are clonic or myoclonic movements of the upper body, which also occur in synchrony with the spike-wave discharges, or mild changes in tone during the absence. Although the patient is usually completely unaware of the environment during absence seizures, an occasional patient reports partial awareness and the ability to hear or see during the discharges. In the majority of cases, after an absence seizure, the patient is unaware that the episode has even occurred, the only potential clue being the subjective feeling that something has been missed in the observed sequence of events.

A fraction of patients may have an EEG pattern that is indistinguishable from the 3-Hz generalized spike-wave discharges that occur during clinical absence but may have no change in awareness at all during the discharges. For instance, these individuals may be able to continue a conversation without pause during the discharges, which then, by definition, do not represent clinical seizures (the definition of a clinical seizure requires that an objective or a subjective change occur in the patient at the time of the abnormal EEG discharge). Realizing that this phenomenon exists, the electroencephalographer must resist the impulse to assume that all 3-Hz generalized spike-wave discharges represent absence seizures. Even "classic" 3-Hz generalized spike-wave discharges may occur as an *interictal* abnormality.

Automatisms

Especially during lengthier absence seizures, individuals can manifest automatic behaviors during the seizure discharge. The expression of such automatisms (which may also be seen during complex partial seizures) varies widely. Examples include fumbling of the hands, running the fingers through the hair, or making humming sounds. Unlike other clinical features of the seizure, such automatisms are not driven directly by a seizure discharge in the way that clonic jerking would be driven by repetitive spikes in the EEG. Rather, automatisms represent the release of automatic, preprogrammed behaviors. When the cerebral cortex is functioning normally, these automatic programs are suppressed. When the cerebral cortex is involved with seizure activity, however, as during an absence seizure, such preprogrammed behaviors may be released.

EEG

The most frequent EEG correlate to typical absence seizures is the "classic" 3-Hz generalized spike-wave discharge (see Figure 10-12). When these discharges are analyzed closely, the maximum voltage of the spike component of the spike-wave complexes is most commonly seen in the superior frontal electrodes (F3 and F4). Less often, the spike maximum is seen in the occipital area, and even less frequently in other locations.

Although the term *3-Hz generalized spike wave* is well known and implies a consistent frequency, observed firing frequencies are not necessarily as consistent as the term implies. Often, the first few discharges fire at a frequency slightly faster than 3 Hz. After onset of the discharge, the firing frequency typically slows, often to 2.5 Hz and sometimes to 2 Hz before abruptly terminating (see Figure 10-13). One of the most characteristic attributes of the typical absence seizure is the abrupt onset and termination of the discharge. The classic 3-Hz generalized spike-wave discharge tends to occur against a normal background and has a clear time of onset and a fairly well demarcated termination. After termination, the EEG returns to the previous background after a few seconds or less.

Atypical Absence Seizures

As with typical absence seizures, the main clinical features of atypical absence seizures are staring and unresponsiveness. Atypical absence seizures differ, however, in that onset and termination of the episodes, both clinically and electrographically, are less clear, and firing rates are slower. Also, atypical absence seizures tend to occur in

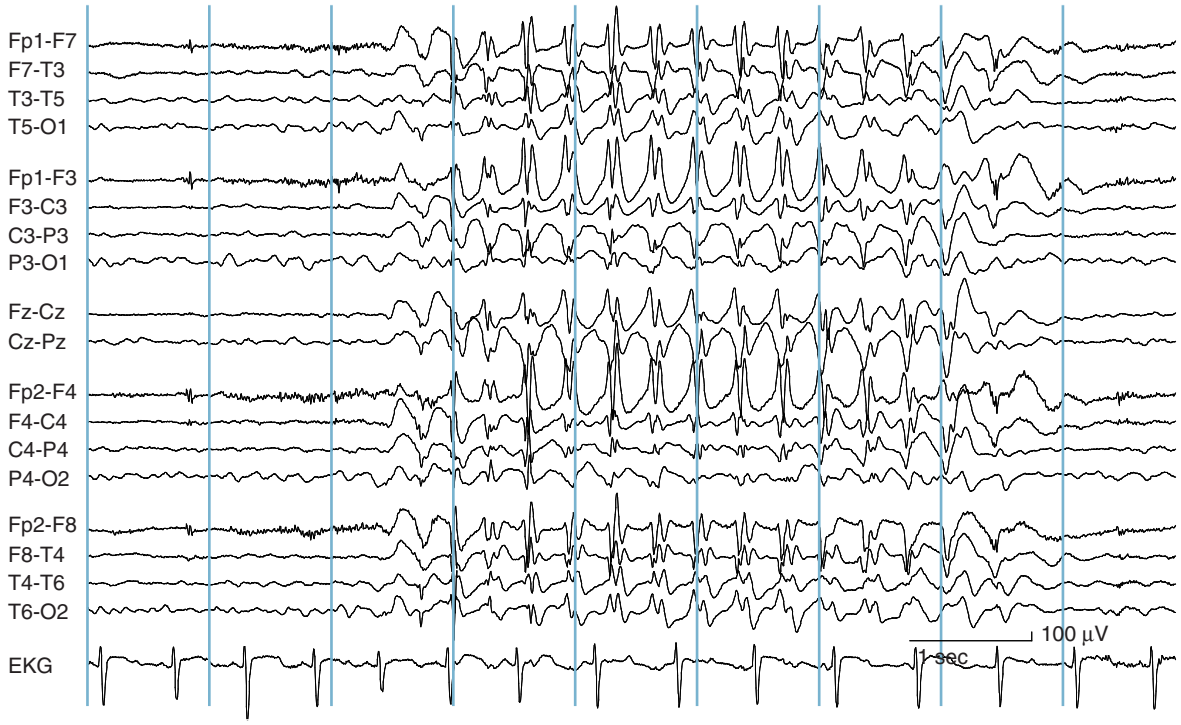


Figure 10-12 This 3-Hz generalized spike-wave discharge shows the abrupt onset and termination that is characteristic of absence seizures. Note the frontal maximum of the waveforms.

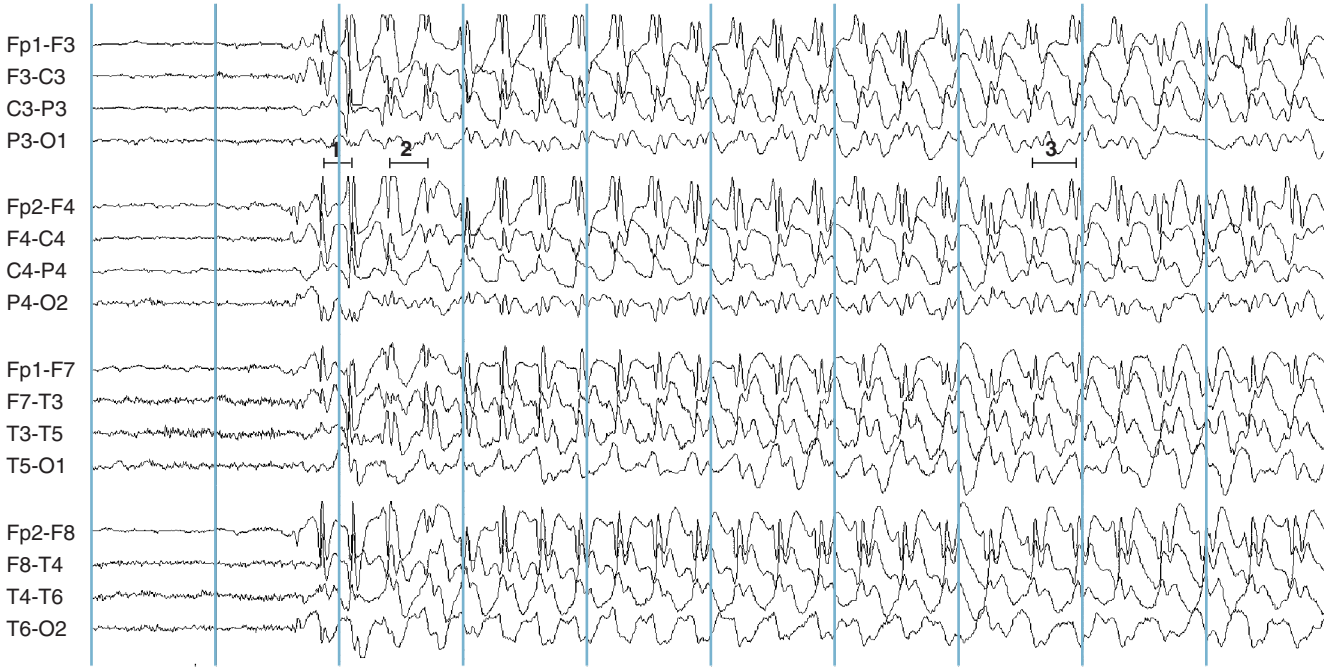


Figure 10-13 Although the discharge associated with this typical absence seizure is classified as 3-Hz generalized spike-wave, note that the discharge's firing frequency still evolves throughout its course. The first wavelength measured suggests a firing rate just above 4 Hz but (1), 1 second later at the time of the second measured wavelength, the firing rate has dropped to 3 Hz (2). Later in the discharge, the third measured wavelength implies a firing frequency of 2.5 Hz (3).

individuals with cognitive impairment or mental retardation, whereas typical absence seizures are more often seen in subjects who are cognitively normal. Changes in tone are more common during atypical absence seizures, with slumping of the head, shoulders, and sometimes the whole torso seen during some examples.

EEG

The EEG hallmark of atypical absence seizures is the slow spike-wave discharge. Slow spike-wave discharges differ from "classic" 3-Hz generalized spike-wave discharges in two important respects: slow spike-wave discharges, as their name implies, fire at a slower rate, usually 2.5 Hz or

less at onset; see Figure 10-14). Slow spike-wave discharges also lack the clear-cut onset and termination characteristic of typical absence seizure discharges. Finally, whereas slow spike-wave discharges are often generalized, asymmetries, both between the left and the right hemispheres and the anterior and posterior head regions, are more common. There is no single characteristic location for the discharge maximum for the slow spike-wave discharges associated with atypical absence seizures, and the location of the voltage maximum may differ even within the same patient at different times. Atypical absence seizures often occur against the backdrop of an otherwise *abnormal* EEG, which may include scattered epileptiform activity or a slowed background.

Slow-spike wave discharges are often associated with atypical absence seizures, but this is not always the case. Although slow spike-wave discharges are expected as the EEG correlate of atypical absence seizures, the converse is often not true: most slow spike-wave discharges are not associated with clinical atypical absence seizures. In practice, slow spike-wave discharges are often seen as *interictal* abnormalities in the EEG. The simple observation of slow spike-wave discharges in the EEG is no guarantee that the patient is actually experiencing an atypical absence seizure, although the finding does raise suspicion that the patient has this seizure type. When this pattern is seen, the concurrent observation of associated staring or some form of decreased responsiveness or change in tone is necessary to establish the diagnosis of an electroclinical seizure. This same phenomenon of electrical discharge without clinical change may also occur with "classic" 3-Hz generalized spike-wave discharges as described in the previous section, although much less often.

Myoclonic Seizures

Myoclonic seizures consist of a lightning-like or shock-like contraction of the muscles driven by an epileptic discharge. The appearance of a myoclonic seizure is similar to experiencing a brief electric shock. Myoclonus may consist of a single jerk or a quick series of jerks that occur in a burst, either rhythmic or non-rhythmic. Epileptic myoclonus may manifest as a muscle jerk in nearly any part of the body, although the most common location for epileptic myoclonus is the upper shoulder girdle. In such cases, the myoclonus usually consists of a quick series of abduction jerks at the shoulders. During the series of jerks, there may be a tendency for slight *net* abduction of the upper arms away from the body with each jerk. The most common EEG manifestation of epileptic myoclonus is a high-voltage polyspike-wave discharge, which may occur singly or in brief, repetitive bursts (see Figures 10-15 and 10-16).

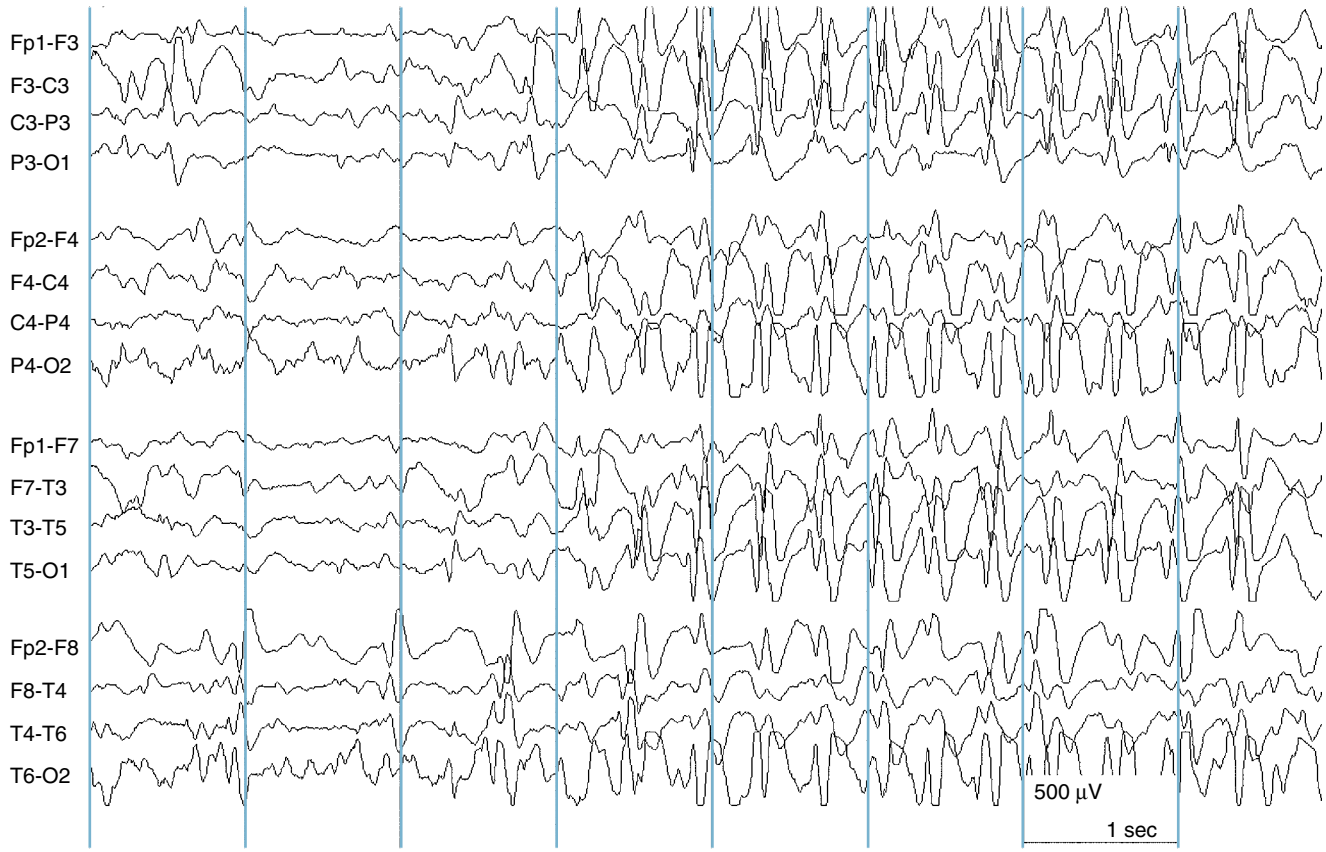


Figure 10-14 Slow spike-wave discharges are seen in a young man with mixed seizures. Note the slowed firing rate of the train of spike-wave discharges on the second half of the page and the scattered, single discharges in multiple locations seen on the first half of the page.

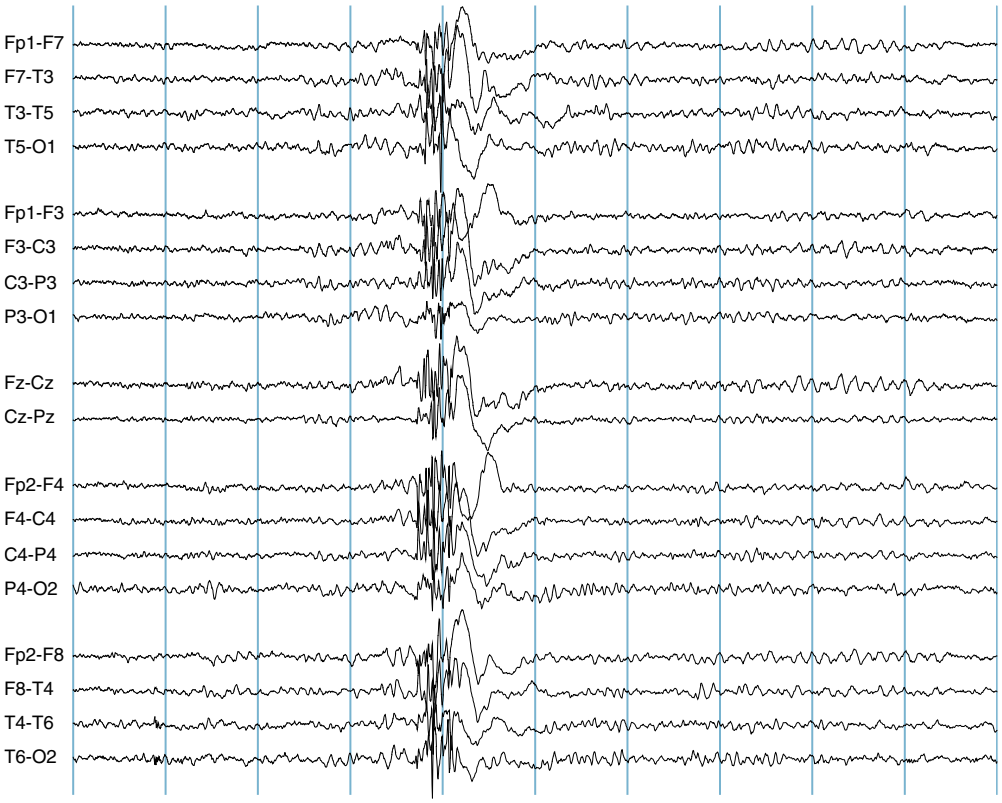


Figure 10-15 This 15-year-old girl was referred for tremor. The movements in question actually represented epileptic myoclonus driven by the high-voltage polyspike-wave discharge shown.

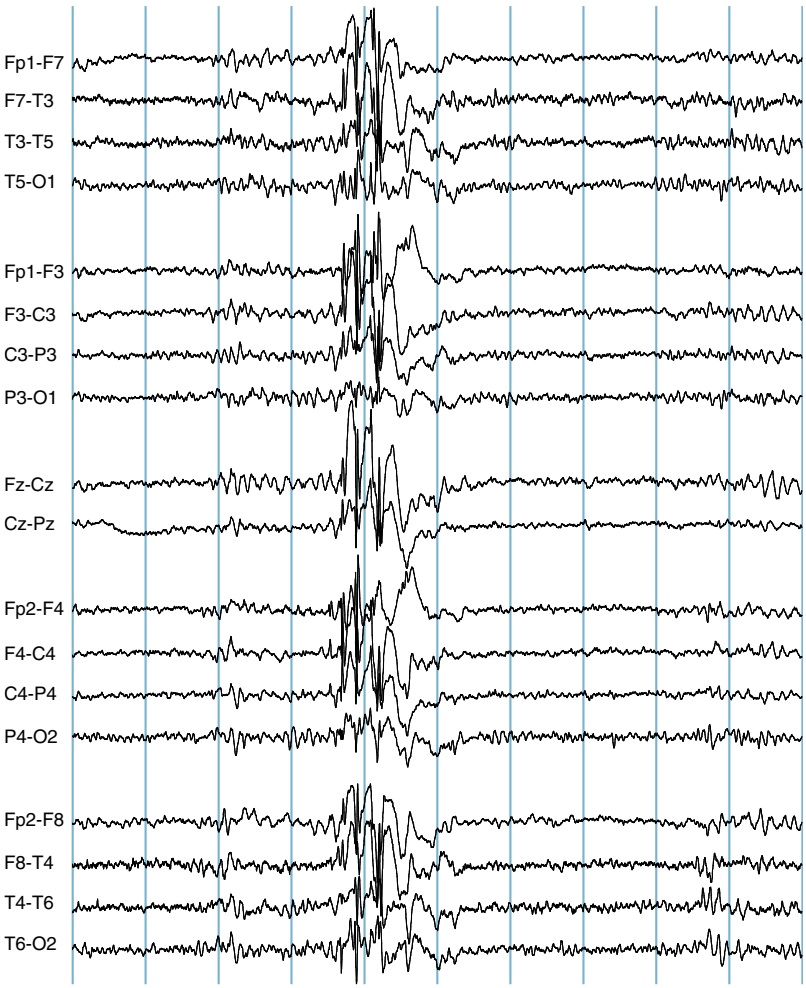


Figure 10-16 Myoclonic jerks often occur in quick succession. In this teenage patient, this quickly repetitive series of polyspike-wave discharges caused a series of quick abduction jerks at the shoulders.

It is important to keep in mind that not all myoclonus is epileptic. Nonepileptic myoclonus may originate in the central nervous system at levels below cerebral cortex, including the subcortical areas, brainstem, and even the spinal cord *(segmental myoclonus)*. The question of whether an instance of myoclonus is epileptic myoclonus is best confirmed by demonstrating the presence of a concomitant EEG discharge driving the movement. No EEG discharge would be expected to accompany nonepileptic myoclonus.

Clonic Seizures, Tonic Seizures, and Generalized Tonic-Clonic Seizures

Clonic Seizures

Clonic seizures are characterized by repetitive clonic jerks. These clonic jerks can occur in nearly any skeletal muscle group in the body depending on the cortical location of the discharge (usually including the motor strip). Clonic jerking from seizure activity tends to have a rhythmic quality. Because each clonic jerk is driven by an EEG discharge, a simultaneous spike or spike-wave discharge is expected with each clonic jerk. Therefore, the frequency of the EEG discharges typically matches the frequency of the jerks. Because clonic jerking is typically driven by discharges from the area of the motor strip, it is rare that scalp EEG electrodes will fail to record them, especially if they involve the face or hand.

Tonic Seizures

Tonic seizures cause tonic stiffening of a limb, several limbs, or the whole body. When the whole body is involved with a generalized tonic seizure, tonic stiffening in extension is most common; however, tonic stiffening with flexion of the hips, knees, or arms may also be seen. Especially with generalized tonic seizures, tonic contraction of the diaphragm may occur resulting in a forced inhalation or grunting sounds. Most often the EEG correlate of tonic seizures is a spray of rapid spikes in the affected area, often with an initial frequency of 10 to 25 Hz, which subsequently slows, similar to the onset of tonic-clonic seizures as described next. Other EEG correlates are not uncommon, including an abrupt desynchronization (flattening) of the EEG. When such flattening is seen, there is sometimes a suggestion of low voltage rapid spikes superimposed on the flattened pattern though these are not always identifiable (see Figure 10-17). Therefore, although most ictal patterns are dramatic and show high-voltage repetitive discharges, the electroencephalographer must also be alert to abrupt flattening of the EEG (an electrodecrement) as a seizure correlate. Other patterns are less common.

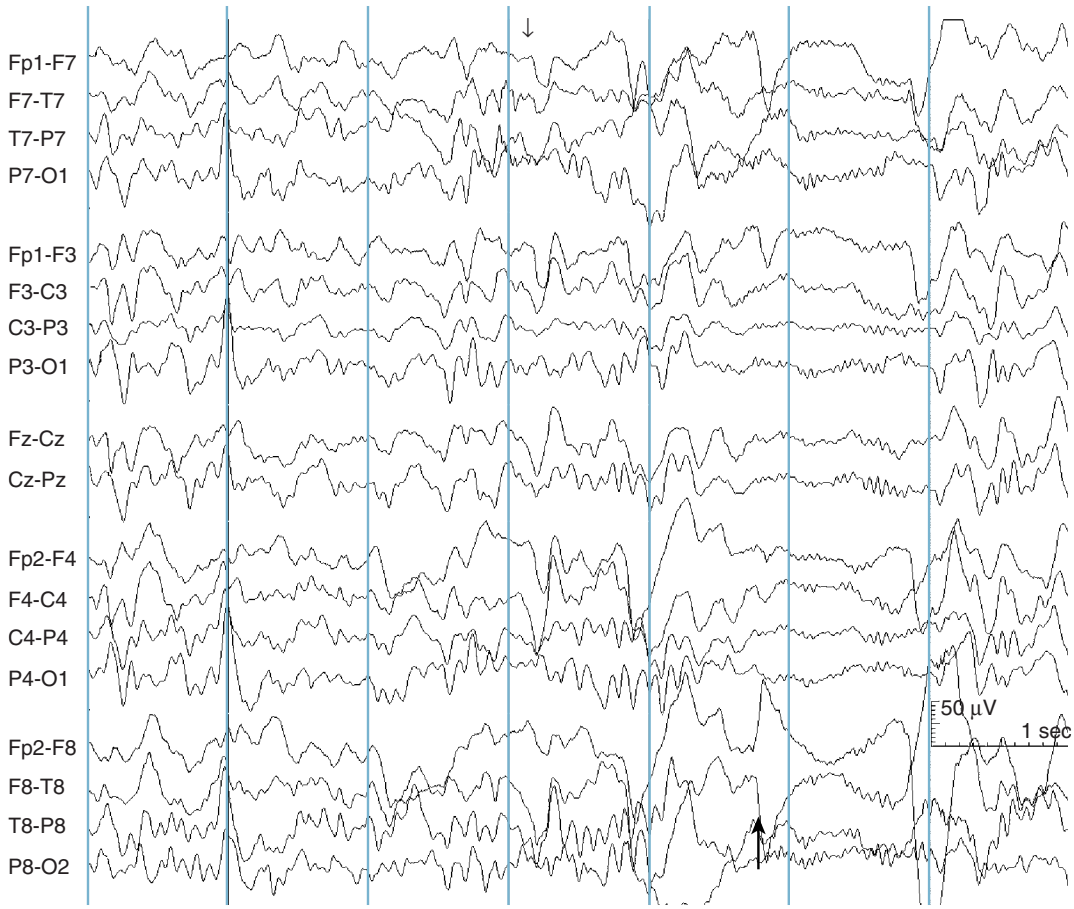


Figure 10-17 Electrodecremental seizure patterns consist of an abrupt flattening of the EEG, usually only lasting a few seconds. In some examples, low-voltage rapid spikes can be seen during the decrement (arrow), but in other cases spikes cannot be identified.

Generalized Tonic-Clonic Seizures

The generalized tonic-clonic seizure refers specifically to the sequence of whole-body tonic stiffening *followed by* clonic jerking. Unfortunately, this term is often used indiscriminately to refer to any generalized convulsion, a use which is technically incorrect; properly, the term *generalized tonic-clonic seizure* should be reserved exclusively for the sequence of whole-body stiffening followed by whole body clonic jerking. This distinction can be important as some clinical events which mimic seizures, such as convulsive syncope (nonepileptic stiffening and jerking body movements associated with brief episodes of significant hypotension, do not tend to manifest this specific sequence. The sequence of tonic body stiffening followed by clonic jerking, compared with other possible sequences of movements, is highly suggestive of epileptic seizure and should be duly noted.

The EEG correlate of the tonic-clonic seizure often begins with an abrupt onset of generalized rapid spikes which then slow in frequency over the course of the event. As the firing frequency of the spikes slows, the spikes may begin to manifest a clearer spike-wave morphology. From the clinical perspective, rapid spikes are often associated with tonic stiffening. At a certain point in the seizure, the firing rate of the spikes slows to a point that allows each spike or spike-wave discharge to generate a separate clonic jerk (see Figure 10-18). This is the reason that the progression from tonic stiffening to clonic jerking is so common. Over the course of a generalized tonic-clonic seizure the clonic jerking is seen to slow in frequency and blend into a slow-wave pattern: "postictal slowing." Less commonly, a clonic-tonic-clonic seizure may occur in which clonic jerking speeds up and melds into tonic stiffening, followed by the usual progression back to clonic jerking. As expected, the EEG correlate of this type of seizure often consists of spike-wave discharges that speed up to become rapid spikes, and then slow down again (see Figure 10-19).

Atonic Seizures

As the name implies, atonic seizures consist of a loss of tone, usually of the truncal muscles. In its mild form, an atonic seizure may simply consist of a subtle slumping of the shoulders. More frequently, an atonic seizure may manifest as a *head-drop* spell in which the patient's head slumps forward. The most dramatic version of an atonic seizure is the *drop attack* in which the patient collapses to the ground, possibly resulting in injury. An astatic seizure is a seizure resulting in a fall (discussed further in the section on seizure types in Lennox-Gastaut Syndrome). There are many possible EEG correlates to atonic seizures, including slow spike-wave discharges, EEG desynchronization (flattening), or polyspikes, sometimes followed by flattening.

Unclassified Seizure Types

There a few seizure types that do not fit into the foregoing classification. These include swimming or bicycling movements, apneas, and roving eye movements. In fact, it remains controversial as to whether many of these apparent seizure types represent true epileptic phenomena. It is possible that some of these behaviors represent automatic movements (automatisms) rather than seizure activity driven by an actual epileptic seizure discharge. The nature of such events therefore remains to be clarified. Infantile spasms (or epileptic spasms) represent a seizure type that is not easily classified as generalized or focal (see the later section on West syndrome).

Neonatal Seizures

Neonatal seizures have been given a classification scheme different from the general ILAE seizure classification described earlier. The most common neonatal seizure classification system in use today was described by Volpe in 1989. The seizure types described in this classification are actually quite similar to those of the ILAE classification, with the exception of an additional seizure category referred to as "subtle seizures," a subgroup that remains controversial as the epileptic nature of subtle seizure behaviors has not yet been firmly established. See Chapter 13, "The EEG of the Newborn" for further discussion.

CLASSIFICATION OF SEIZURE SYNDROMES

A patient's seizure syndrome is defined by the type or types of seizures experienced, age of onset, neurologic status (abnormal neurologic status before or after seizure onset), progression, family history, physical examination, and EEG patterns. Identification of a particular seizure syndrome will often suggest possible treatments and a specific prognosis. Select seizure syndromes are discussed below, generally in order of age of onset.

Epileptic Syndromes of Early Infancy Associated With a Burst-Suppression Pattern

Early myoclonic epilepsy (EME) and early infantile epileptic encephalopathy (EIEE) are the two major catastrophic epilepsies of early infancy. Although these two syndromes have much in common, they appear to represent two distinct entities.

Early Myoclonic Encephalopathy

Infants with EME present soon after birth with both fragmentary and massive myoclonic seizures. Other seizure types also occur. The magnetic resonance imaging (MRI) scan at birth is almost always normal, and a large fraction of these babies are eventually found to have a specific metabolic disorder, nonketotic hyperglycinemia (NKH). Those babies with EME who do not prove to have NKH may have other metabolic diagnoses, and it is felt that the remaining cases of EME may be caused by some metabolic entities

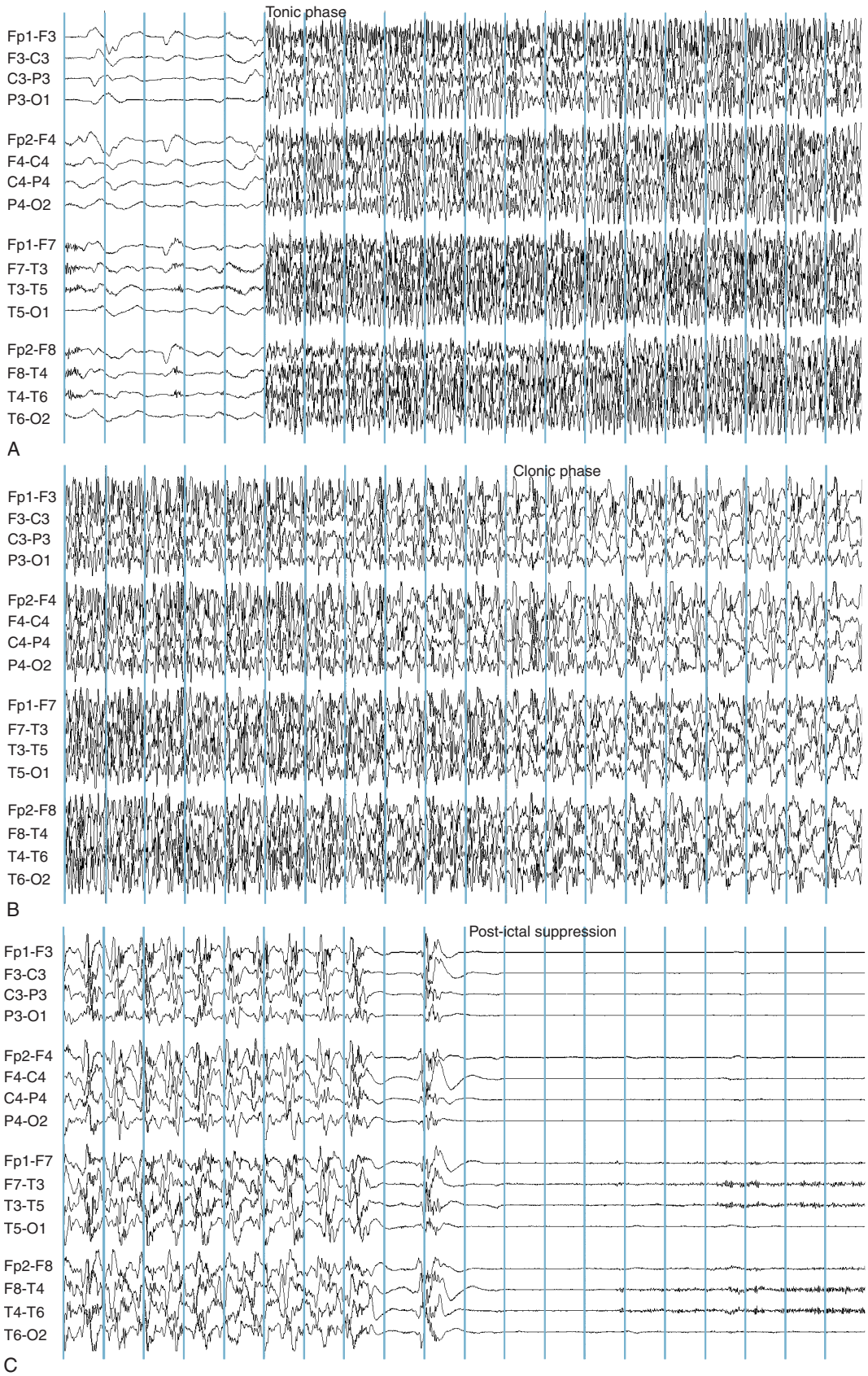


Figure 10-18 The classic evolution of a generalized tonic-clonic seizure is shown. There is abrupt onset of generalized rapid spikes at the start of the tonic phase. As the firing frequency of the spikes decreases the individual spikes become far enough apart from one another that each spike can generate a separate clonic jerk, representing the clonic phase of the seizure. In this case, there is a period of postictal suppression after cessation of the seizure discharge. After seconds or minutes, generalized slow-wave activity appears that may last from minutes to days depending on the duration of the seizure and the nature of the patient.

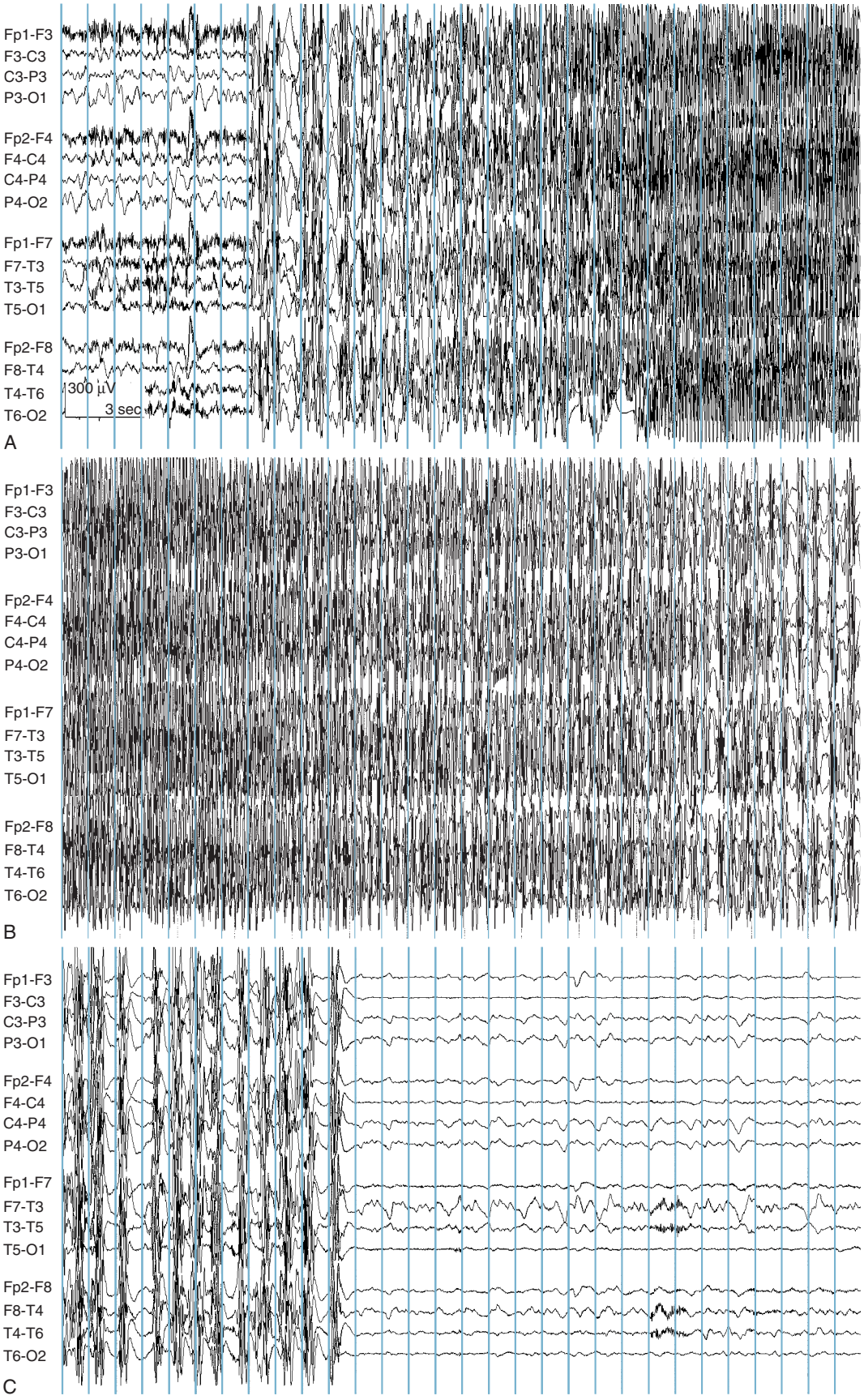


Figure 10-19 A clonic-tonic-clonic seizure discharge is shown. Note that, in comparison to the previous example, the initial rapid spikes appear in brief bursts (seconds 8–10 of panel A) each associated with a clonic jerk. After a few seconds, the rapid spikes consolidate and their firing frequency increases, corresponding to the tonic phase of the seizure. The seizure discharge then follows a pattern similar to that of the tonic-clonic seizure shown earlier, with slowing of the spike frequency associated with the clonic phase of the seizure. Postictal slowing is seen following this seizure discharge.

yet to be defined. This belief is based on the observations that EME babies have anatomically normal brains by MRI, and there is usually no history of a previous neurological injury. Babies with EME have complete developmental failure, and the seizures tend to be refractory to treatment. The EEG shows an unremitting burst-suppression pattern that may continue unabated through childhood (see Figure 10-20).

Early Infantile Epileptic Encephalopathy

EIEE, also known as Ohtahara syndrome, appears to be a "lesional" epilepsy syndrome and, in contrast to EME, is often associated with an abnormal MRI scan. Tonic seizures are more prominent in EIEE compared with EME. MRI abnormalities associated with this syndrome can include cerebral malformations, or cerebral injuries as may occur in babies with hypoxic-ischemic encephalopathy. Therefore, EIEE occurs as an epileptic syndrome that is believed to be symptomatic of a preexisting abnormality, be it a cerebral malformation or some type of brain injury. EIEE is more likely to evolve to West syndrome or the Lennox-Gastaut syndrome.

Like EME, EIEE is typically associated with a burst-suppression pattern on EEG. The EEG patterns of EIEE and EME are not easily distinguished without the benefit of the clinical history (see Figure 10-21). The burst-suppression pattern of EIEE is more likely to evolve into other EEG background patterns later in life, compared with the EME burst-suppression pattern, which may persist indefinitely.

Therefore, despite the many aspects they share in common (similar EEG pattern, intractable seizures, poor prognosis), EIEE distinguishes itself from EME in that EIEE is considered an acquired or lesional epileptic encephalopathy caused by a cerebral malformation or a cerebral injury. In contrast, children with EME are believed to have the disorder on a genetic or biochemical basis rather than from a postnatal event. In EME, MRI brain anatomy is typically normal.

The Concept of the Age-Dependent Epileptic Encephalopathies

EIEE is considered one of the *age-dependent epileptic encephalopathies*. This group of syndromes represents the result of different characteristic epileptic responses of the brain to injury seen at different ages. For instance, when the brain is injured in the neonatal period, the response to injury may present as the EIEE syndrome as described earlier. When the epileptic response to an injury or abnormality occurs later in infancy (typically after 3 months of age), the child may develop a pattern of infantile spasms or West syndrome (discussed later). The epileptic response to an injury that appears after 3 years of age may present as the Lennox-Gastaut syndrome (discussed later). Because different maturational states of the brain are only associated with certain syndromic patterns, West syndrome does not present in adults, and Lennox-Gastaut syndrome cannot present in early infancy; because of the way the human brain matures, it is

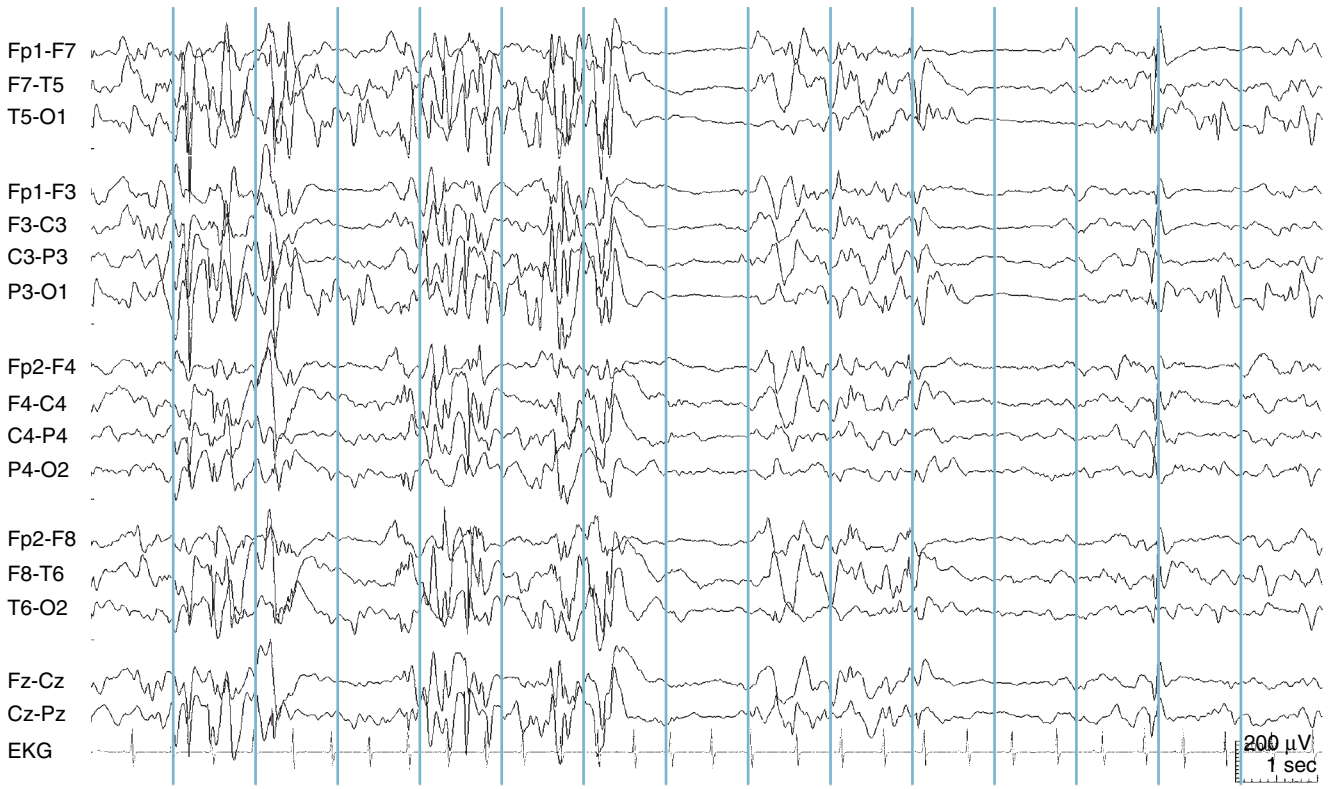


Figure 10-20 A dramatic burst-suppression pattern is seen in the EEG of this newborn with early myoclonic epilepsy (EME). No sleep cycling occurred in this recording, and every page of the record showed the same burst-suppression pattern. In patients with EME, this discontinuous pattern may persist for years.

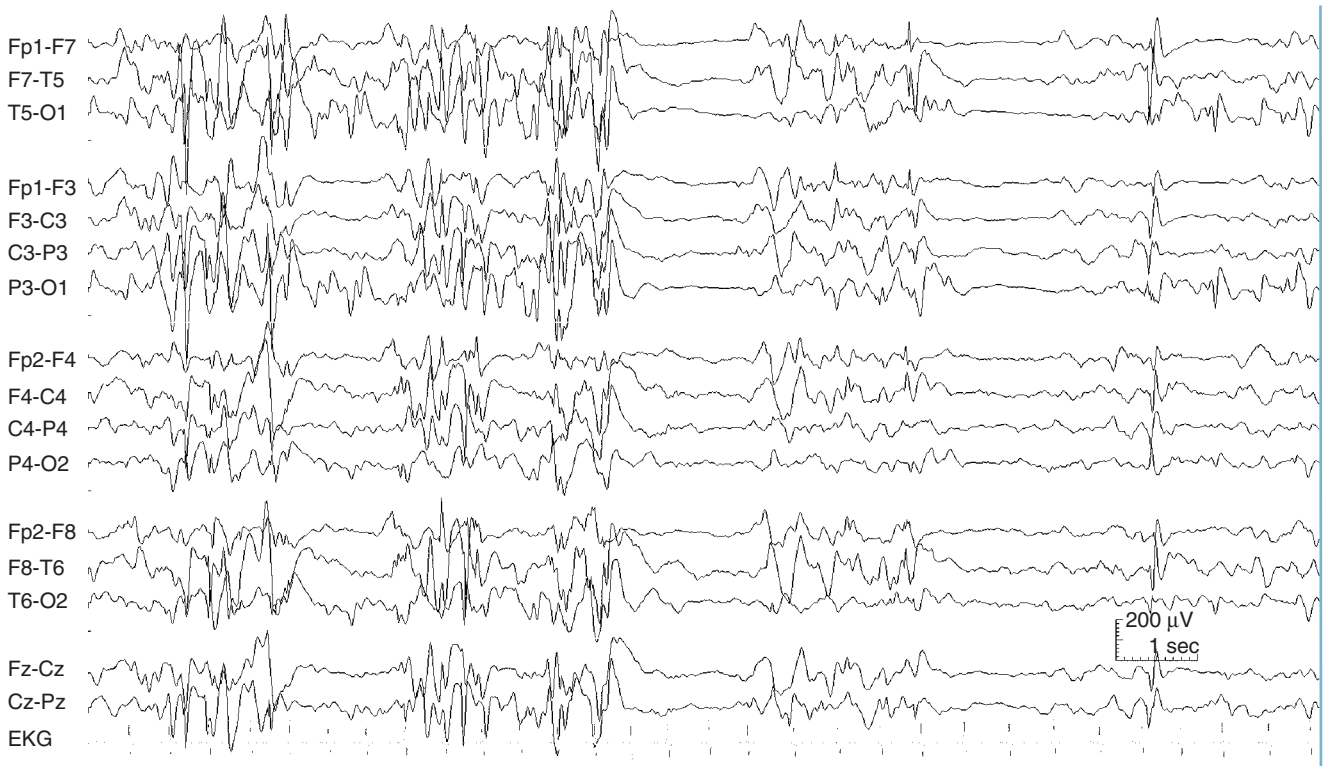


Figure 10-21 The burst-suppression pattern of a newborn patient with EIEE shown in this figure is essentially indistinguishable from the patterns seen in EME. In EIEE, eventual evolution to other patterns over months or years is not uncommon.

not able to mount those types of seizure patterns at those ages.

Pyridoxine-Dependent Seizures

The syndrome of pyridoxine-dependent seizures is extremely rare in its pure form. In the classic presentation of this disorder, a newborn is found to have refractory seizures associated with a persistent burst-suppression pattern on EEG. Administration of pyridoxine, sometimes in high doses, abruptly breaks the abnormal EEG pattern and promptly terminates the seizure activity. Without continued supplementation with pyridoxine, the seizures will relapse. Because this is such an easily treatable form of what would otherwise be a catastrophic epilepsy, pyridoxine infusion is routinely carried out on newborns with seizures who potentially match the phenotype of this disorder (in particular, those having a burst-suppression pattern on EEG without other explanation). Later onset and partial forms of this disorder exist and are even less commonly encountered.

Benign Familial Neonatal Convulsions and Benign Neonatal Convulsions

Benign familial neonatal convulsions (BFNC) and benign neonatal convulsions are the two important benign seizure syndromes of the newborn. The essence of both of these seizure syndromes is similar: seizures early in the newborn period in a previously well infant followed by a generally benign outcome. Although these two syndromes have significant similarities, they are discussed separately.

Benign Neonatal Convulsions

The syndrome of benign neonatal convulsions is also known to as "fifth day fits," a name that serves as a useful reminder that the fifth day of life is the most common age of onset for this syndrome. To some extent, the diagnosis of benign neonatal convulsions must be made in retrospect because a benign long-term course is a key part of the syndrome.

The expected presentation of benign neonatal convulsions consists of a newborn who appears normal at birth and who may have already been uneventfully discharged home from the hospital. The seizures begin in the first week of life, with the most common age of incidence being the fifth day of life; 90% of babies present between the fourth and sixth days of life. The seizures generally subside by the second month followed by continued normal development.

The family history for seizures is negative. There is no antecedent history of a difficult delivery or birth injury, and neuroimaging is normal. Apart from possible mild hypotonia, the interictal examination is normal. A search for central nervous system infection is negative, and no electrolyte or other metabolic disturbances are found. The clinician is left with a story of an otherwise perfectly well newborn with unexplained onset of seizures in whom all testing is normal.

In babies with benign neonatal convulsions, the EEG background pattern tends to be normal. A characteristic EEG finding has been described in such babies, termed *théta pointu alternant*. This is a pattern of sharpened theta waves occurring in brief runs, typically in each central area, and alternating sides (see Figure 10-22). Although this pattern is said to occur in the majority of patients with this seizure syndrome, it may be difficult to identify, and its presence is not necessarily diagnostic of benign neonatal convulsions.

Whether benign neonatal convulsions represents a single, distinct syndrome has been questioned. From one point of view, it should not be a surprise that the subgroup of newborns with seizures who have a negative history and normal testing would have a more favorable outcome than those newborns with seizures who have abnormal histories or abnormalities in neuroimaging or other testing. Whether or not benign neonatal convulsions represents a unique syndrome remains an open question. There is also a question as to whether the incidence of later epilepsy in these infants is somewhat higher than that of the unaffected population.

Benign Familial Neonatal Convulsions

The syndrome of benign familial neonatal convulsions (BFNC) has many elements in common with the previously described syndrome of benign neonatal convulsions. As the name implies, however, in such babies there is a positive family history of seizures in the newborn period. In this syndrome, the seizures tend to begin slightly earlier, typically on the second or third day of life, usually resolving by the second month. Unlike benign neonatal convulsions, in BFNC a 10% to 15% incidence of later epilepsy has been described. Also, mild developmental problems may occur with a slightly increased frequency compared with the unaffected population. A large proportion of affected individuals have been found to have linkage to the 20q13.3 gene locus corresponding to a mutation in the potassium channel gene KCNQ2. A smaller number of affected individuals have been found to have linkage to the 8q24 locus and a mutation in the KCNQ3 gene (both potassium channel genes). Still other kindreds appear to have no abnormality at either of these loci, suggesting that additional genetic abnormalities that cause this syndrome have yet to be identified.

The typical seizure in BFNC is the clonic seizure, preceded by tonic stiffening and apnea in some (Hirsch et al., 1993). Most commonly the interictal EEG is normal, however, the *théta pointu alternant* pattern, as has been described in benign neonatal convulsions, has been reported in some babies with this syndrome.

Febrile Seizures

Febrile seizures are seizures that occur with fever during childhood. There are mild variations in the age range that various groups have used to define febrile seizures, with ranges such as 3 months to 5 years and 1 month to 6 years being used. These stated age ranges are somewhat misleading in that it is uncommon for febrile seizures to *start* at the end of these age ranges; usually when a febrile seizure occurs after the age of 4 years, the child has already had previous episodes. The large majority of affected children have had the first febrile seizure by 3 years of age. According to the definition, the seizure should not have an obvious cause such as central nervous system infection, and children with previous unprovoked seizures are excluded from the definition. The fever should exceed 38.4 °C (101 °F), but this cutoff is flexible. The diagnosis of febrile seizures is usually made in a normal-appearing child with a normal nervous system. Nevertheless, there is no reason that children with preexisting neurologic abnormalities should be any less prone to febrile seizures than their normal counterparts leaving the underlying cause of the seizures more difficult to sort out in this group.

Although one may speak of a "febrile seizure syndrome," febrile seizures are generally not considered an example of an *epilepsy* syndrome. Epilepsy is defined as a tendency to recurrent, *unprovoked* seizures, and because the presence of fever is considered a provocative factor, febrile seizures are not considered examples of epileptic (unprovoked) seizures.

Febrile seizures are common. They occur in 3% to 5% of all children and, in the large majority, disappear during childhood. Only 2% to 4% of children with febrile seizures are destined to have future seizures without fever (epilepsy). Fever is known to be a common seizure-triggering factor for persons who do have epilepsy, and the question may arise as to whether an apparent febrile seizure episode really represents an epileptic seizure triggered by fever in a child who is destined to have epilepsy. It is known that certain features of a febrile seizure episode increase the odds that a child will later develop epilepsy. A febrile seizure is termed a *complex febrile seizure* when one or more "complex" features are present. These complex features have been defined by epidemiologic studies that have found these factors to be associated with an increased risk of developing later epilepsy (afebrile seizures). They include seizure duration longer than 10 or 15 minutes, focal (as opposed to generalized) febrile seizures, or two or more seizure episodes within a 24-hour period. The more of these "complex" features a child has, the higher the risk of developing later epilepsy. A preexisting abnormal neurological status at the time of seizure onset and a positive family history of epilepsy also independently increase the chance of later epilepsy in a child with febrile seizures.

A relationship between febrile seizures and temporal lobe epilepsy has long been suspected. Case-control studies of individuals with temporal lobe epilepsy appear to show an increased incidence of a history of febrile seizures, particularly *prolonged* febrile seizures, in temporal lobe epilepsy patients compared with control subjects. These findings suggest the possibility that prolonged febrile seizures may cause hippocampal damage (hippocampal sclerosis) and predispose to later temporal lobe epilepsy. An alternative interpretation is that those children destined to have temporal lobe epilepsy later in life

may automatically be more prone to prolonged febrile seizures in childhood and that the early febrile seizures may not be a causative factor in the later epilepsy.

The Role of EEG in the Evaluation of Febrile Seizures

Despite the fact that EEG abnormalities may be seen in children with febrile seizures, the EEG has not proved to be a particularly useful tool in predicting later epilepsy in these children. The most common epileptiform abnormality found in children with febrile seizures is generalized spike-wave discharges. This pattern is, of course, nonspecific in that it is also seen in the generalized epilepsies. Its presence in the EEG of a child with febrile seizures has not been clearly shown to increase the risk of later epilepsy. Increased slowing, especially in the posterior quadrants, may be expected within a few days of a febrile seizure. In general, because the EEG has not been found to be useful in predicting later epilepsy in children with febrile seizures, EEG testing is not routinely indicated in the evaluation of uncomplicated febrile seizures. Also, because febrile seizures are rarely treated with daily antiepileptic medications, it is highly unlikely that an EEG result, whether or not it is abnormal, would prompt the use of daily seizure medication.

Infantile Spasms and West Syndrome

Although the terms *infantile spasms* and *West syndrome* are sometimes used interchangeably, infantile spasms specifically refers to a seizure type (most often, but not exclusively seen as a part of West syndrome) and West syndrome to an epilepsy syndrome. The term West syndrome denotes a syndrome consisting of the triad of infantile spasms, an EEG pattern of hypsarrhythmia, and neurodevelopmental abnormality in childhood. The original clinical description of this syndrome appears in a letter to *Lancet* by Dr. W. J. West in 1841, who described the seizures in his son.

The hallmark seizures of West syndrome are infantile spasms. Infantile spasms may occur as flexor spasms, extensor spasms, or mixed (asymmetrical) spasms. Flexor spasms are the most common form, consisting of a brief tonic contraction in flexion of the body on the hips, flexion of the head on the neck, and tensing of the shoulders, sometimes in abduction. Because of the flexed position attained during the seizures, these episodes have also been referred to as "jackknife seizures" or "Salaam seizures." The position is typically held for approximately 1 second, followed by relaxation. The episodes tend to occur in clusters; a series of repeat spasms may last several minutes. Less commonly, spasms can result in extensor rather than flexor posturing. The presence of asymmetrical spasms should always prompt a search for an underlying focal lesion. Other, milder variants of spasms can be seen, including relatively subtle bobbing of the head with upward eye deviation and mild shoulder movement. Because epileptic spasms are seen in both apparently generalized and focal forms, this seizure type defies easy categorization into one group or the other.

Infantile spasms are occasionally erroneously classified as myoclonic seizures, but the episodes are not consistent with the lightning-like jolt that is the definition of myoclonus. Rather, the clinical spasm usually lasts approximately 1 second, too long to be classified as myoclonus. Although the large majority of spasms occur during infancy, occasionally spasms persist past infancy. In older children or adults, the term *infantile spasms* becomes awkward, and the term *epileptic spasms* is preferred.

The infantile spasms of West syndrome can be surprisingly difficult to diagnose. Because West syndrome is a rare disorder, many pediatricians have not encountered this seizure type in clinical practice. The tensing up seen during infantile spasms can mimic gastrointestinal discomfort or episodes of colic. Often, the primary care physician does not have the opportunity to witness the seizures personally but must rely on a verbal description of flexing up of the knees followed by crying, a history that may not initially suggest seizures. Features of the history that increase the suspicion of infantile spasms include the short duration of the individual tensing movements, the tendency for the episodes to cluster, and the tendency for the clusters to occur in the period after awakening from sleep.

The infantile spasms of West Syndrome usually begin between the ages of 3 and 18 months. Only rarely is onset of this seizure type seen outside of childhood. In many, the seizures resolve spontaneously but are often later replaced by other seizure types, such as focal seizures or the mixed seizures of the Lennox-Gastaut syndrome. Only a relatively small minority of patients are intellectually normal after developing infantile spasms and hypsarrhythmia.

EEG in the Diagnosis of Infantile Spasms and West Syndrome

Because West syndrome has distinctive ictal and interictal EEG signatures, electroencephalography is a central tool in the diagnosis of infantile spasms and West syndrome. EEG helps establish the diagnosis of infantile spasms, either by identifying the characteristic interictal pattern of West syndrome (i.e., hypsarrhythmia) or by recording the spasms and demonstrating an ictal discharge during the events. EEG may also be used to monitor the success of treatment.

The EEG term hypsarrhythmia is derived from the Greek meaning "high" or "lofty" rhythm. In fact, some of the highest voltages measured in electroencephalography are seen in babies with hypsarrhythmia. Compared with adult EEGs in which voltages typically do not exceed 200 μV, hypsarrhythmia EEGs may exceed 1 mV (1000 μV). The essential features of the hypsarrhythmic pattern are *high voltage* and *chaos*. In this context, chaos refers to the opposite of synchrony. A completely chaotic EEG pattern is a pattern in which different electrical events and rhythms are occurring in different brain regions at different times in an unsynchronized and seemingly unrelated fashion. In contrast, the generalized spike-wave discharge, although abnormal, represents a pattern with a high degree of synchrony

with all cortical areas acting in unison. In the chaotic hypsarrhythmia pattern, rapid spikes, high-voltage s low waves, and other abnormalities may occur in scattered locations at different times and in a seemingly random fashion (see Figure 10-23). Intermediate states between synchrony and complete chaos are also seen (see Figure 10-24).

Activation by sleep is an important feature of the hypsarrhythmia pattern. In fact, in some individuals, the waking tracing can be relatively normal with the EEG, only blossoming into a highly abnormal hypsarrhythmia pattern after the patient falls asleep (see Figure 10-25).

It can be challenging to describe the intensity of a hypsarrhythmia pattern in words. The hypsarrhythmia scoring system designed by Jeavons and Bower (1961; see Table 10-1) is a useful reminder of the various parameters of chaos and high voltage that are scrutinized during analysis of the hypsarrhythmic EEG. Note that the main factors are degree of chaos, magnitude of voltage, amount of bursting, number of locations of focal spiking, and presence of asynchronous delta activity.

EEG Recording of Infantile Spasm Events

Recording a hypsarrhythmia pattern is usually adequate to confirm the diagnosis of West syndrome in an infant with a history suggestive of infantile spasms. Because the seizures occur so frequently, often with multiple clusters per day that are linked to the sleep-wake cycle, it is not uncommon to record the seizures themselves. The ictal pattern associated with infantile spasms can vary, but most often it is seen as a "complex" slow wave of moderate voltage and approximately 1 second's

Figure 10-22 *Théta pointu alternant*, or alternating sharp theta, may be seen in each central area in both the syndromes of benign neonatal convulsions and benign familial neonatal convulsions. *(Modified from Dehan M, Quillerou D, Navelet Y, et al. Convulsions in the fifth day of life: a new syndrome? Arch Fr Pediatr 1977;34:730 742).*

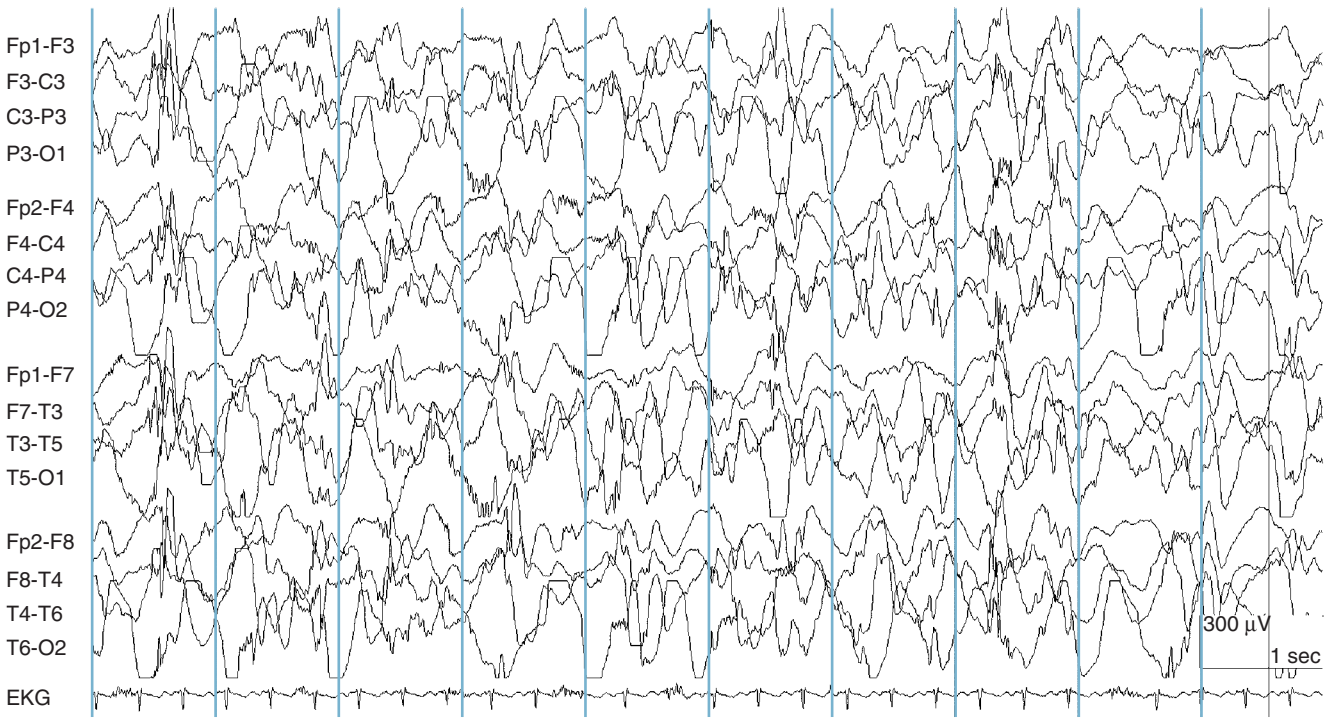


Figure 10-23 The high-voltage, chaotic pattern of hypsarrhythmia is shown. Note the "random delta" activity and the scattered spikes, occurring both singly and in brief runs in a chaotic fashion.

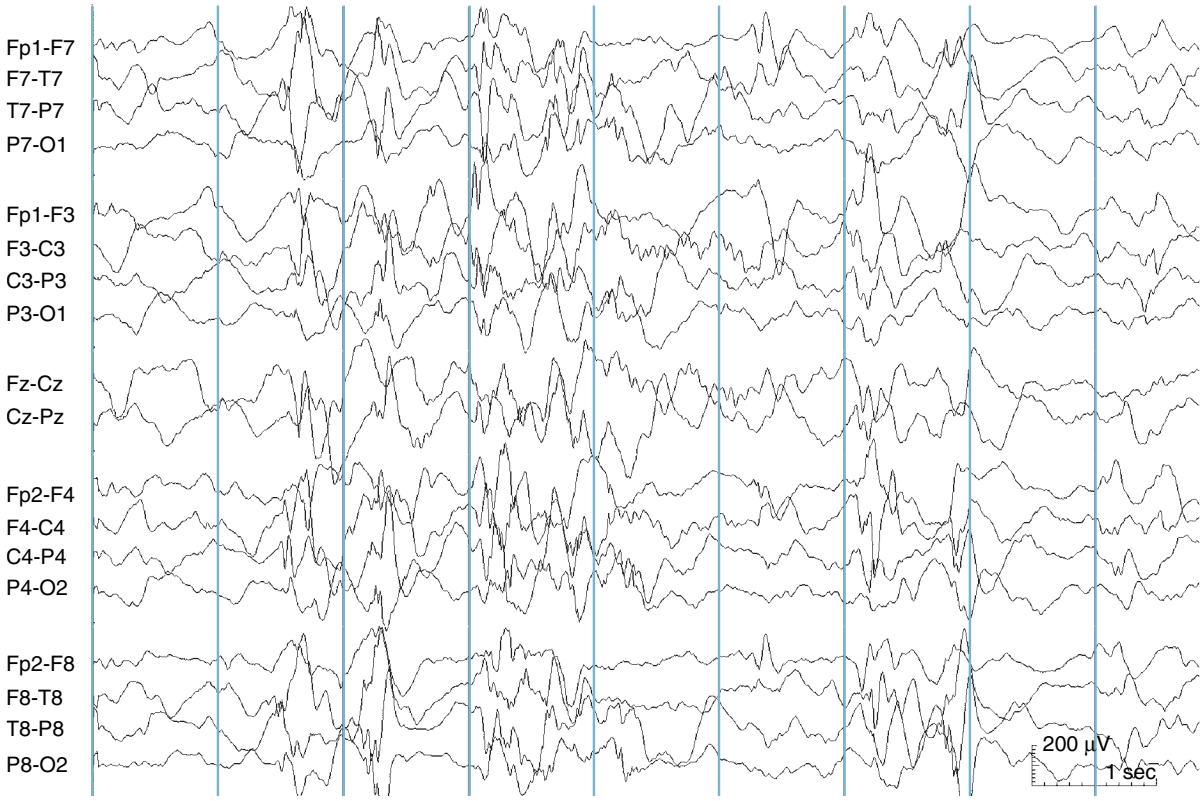


Figure 10-24 This hypsarrhythmia tracing also shows very high-voltage bursts and intermixed spikes. The pattern is somewhat less chaotic than the previous figure as the bursts show more bilateral synchrony. In the fifth second of the tracing, sleep spindles can be identified.

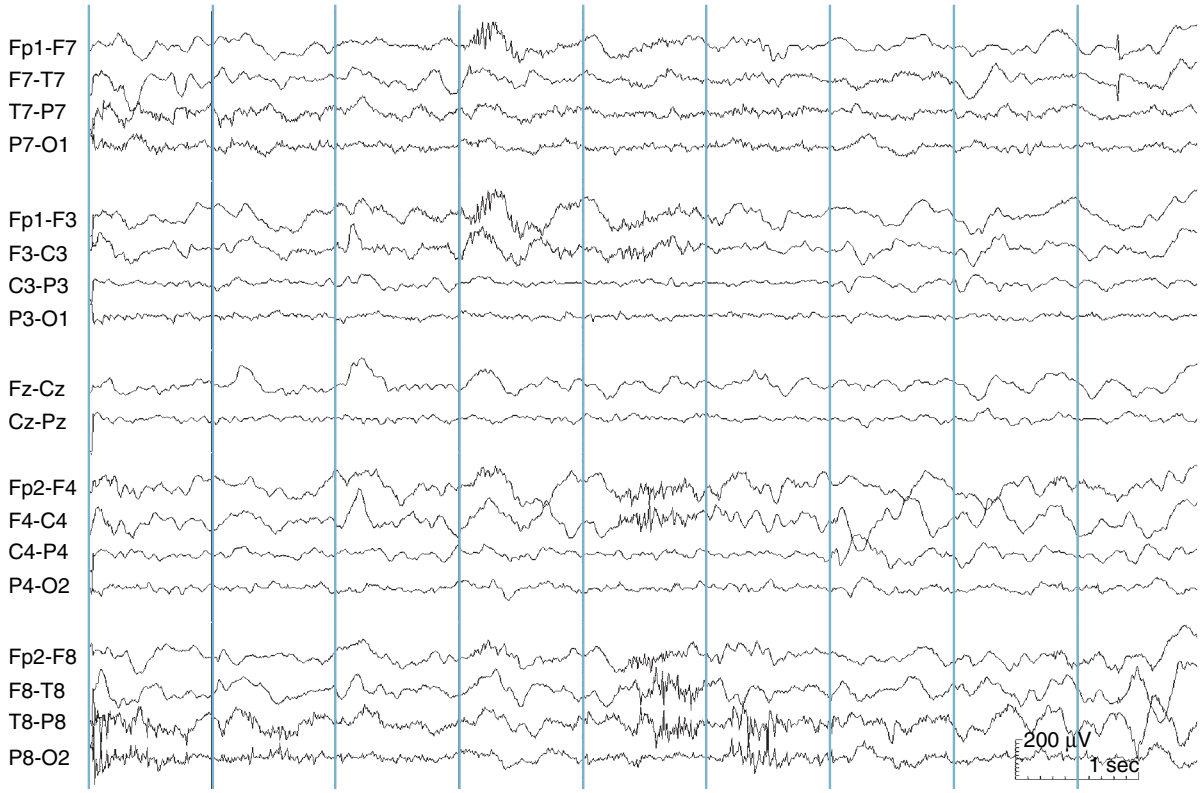


Figure 10-25 The EEG of the same patient whose hypsarrhythmia pattern was shown in Figure 10-23 is seen during wakefulness. While awake, almost all evidence of the hypsarrhythmia has cleared. The EEG diagnosis of hypsarrhythmia would almost certainly have been missed if this infant had not been recorded during sleep.

Table 10-1 Hypsarrhythmia Scoring System (Jeavons and Bower)

Grade	
Grade 1: 12 points	Complete chaos with total asynchrony, no organized discharges, and no normal background activity.
Grade 2: 8 points	Chaos, with some discernible synchronous bursts. The bursts are of chaotic makeup, and the episodic nature can only be seen by reducing the gain.
Grade 3: 6 points	Mainly chaotic, but with more bilaterally synchronous activity than in Grade 2.
Grade 4: 4 points	Bursts of chaotic makeup, some bilaterally synchronous discharges and a little normal background activity.
Grade 5: 3 points	Discharges mainly bilaterally synchronous, but some showing a chaotic makeup. Some normal activity.
Grade 6: 1 point	Bilaterally synchronous epileptic discharges (centrencephalic). No chaos. Normal background activity.
Grade 7: 1 point	Focal epileptic discharges. No chaos. Normal background activity.
Grade 8: 1 point	Nonspecific abnormality.
Grade 9: 0 points	Normal.
Voltage: 0–4 points	
100–200 μV	1 point
200–400 μV	2 points
>500 μV	4 points
Bursts of Very Chaotic Makeup containing delta waves from 0.75 to 3 Hz, with spikes of varying site and amplitude:	2 points
Bursts of Slow or Rapid Spikes Appearing simultaneously in all regions:	2 points
Focal Spikes	
0–4 points	
1 point each for:	
Left	1 point
Right	1 point
Anterior	1 point
Posterior	1 point
Completely Random Spikes	4 points
Completely Random Delta	2 points
Normal Background Activity	−1 point
Hypsarrhythmia	13–30 points
Modified hypsarrhythmia	9–12 points
Epileptic (centrencephalic)	2–8 points
Normal or nonspecific	0 or 1 point

Adapted from Jeavons P, Bower B: The natural history of infantile spasms. *Arch Dis Childhood* 36:17–22, 1961.

duration followed by very low-voltage rapid spikes. The slow-wave portion of the discharge is termed *complex* because the phase of the wave is often flipped up and down in different channels in a way that defies a single, accurate localization. Because the child is tensing up during the seizure and the rapid spikes, if present, are of low voltage, muscle artifact may obliterate any low-voltage rapid spiking activity. At times the ictal correlate may only consist of a slow wave followed by a brief, relative flattening of the background pattern. In other examples, the slow wave is not evident and only a simple flattening is seen (see Figures 10-26 and 10-27).

Because the hypsarrhythmia pattern is an interictal pattern seen between seizures, the electrographic seizure pattern of infantile spasms may actually interrupt the high-voltage, chaotic hypsarrhythmic pattern resulting in a relative flattening in the EEG at the time of the seizures (see Figure 10-28). This type of transition from high-voltage background to low-voltage seizure pattern contrasts with the more common occurrence of a seizure discharge attaining a higher voltage than the background from which it arises.

Modified Hypsarrhythmia

The term *modified hypsarrhythmia* has been used for EEG patterns that share many of the characteristics of full-blown hypsarrhythmia but do not meet the chaos or voltage criteria that would qualify as a fully hypsarrhythmic pattern. Some have attempted to create a classification of modified hypsarrhythmias; however, such classifications are cumbersome as there are so many ways that an EEG pattern can be similar to (but not quite an example of) frank hypsarrhythmia. Rather than simply using the nonspecific term "modified hypsarrhythmia" as if it referred to a specific entity, it is more useful to describe exactly what variations are seen from the classic pattern.

One common variation of hypsarrhythmia is presence of the pattern only during sleep. In these patients, the waking EEG pattern is not particularly remarkable but may show a few epileptiform discharges (see Figure 10-25). As the child falls asleep, the pattern blossoms into the typical high-voltage, chaotic pattern. The hypsarrhythmia pattern may also be suppressed during rapid eye movement (REM) sleep. For these reasons, it is mandatory that a portion of the recording be obtained in slow-wave sleep before excluding the presence of hypsarrhythmia. Another, less common but noteworthy variant of hypsarrhythmia is *hemi-hypsarrhythmia*. In hemi-hypsarrhythmia, the hypsarrhythmia pattern is essentially confined to a single hemisphere. When present, this pattern should prompt a search for a focal lesion.

West Syndrome as an Age-Dependent Epileptic Encephalopathy

Because onset of the combination of hypsarrhythmia and spasms in West syndrome is only seen during a specific age range, West syndrome is considered one of the age-dependent epileptic encephalopathies. The idea

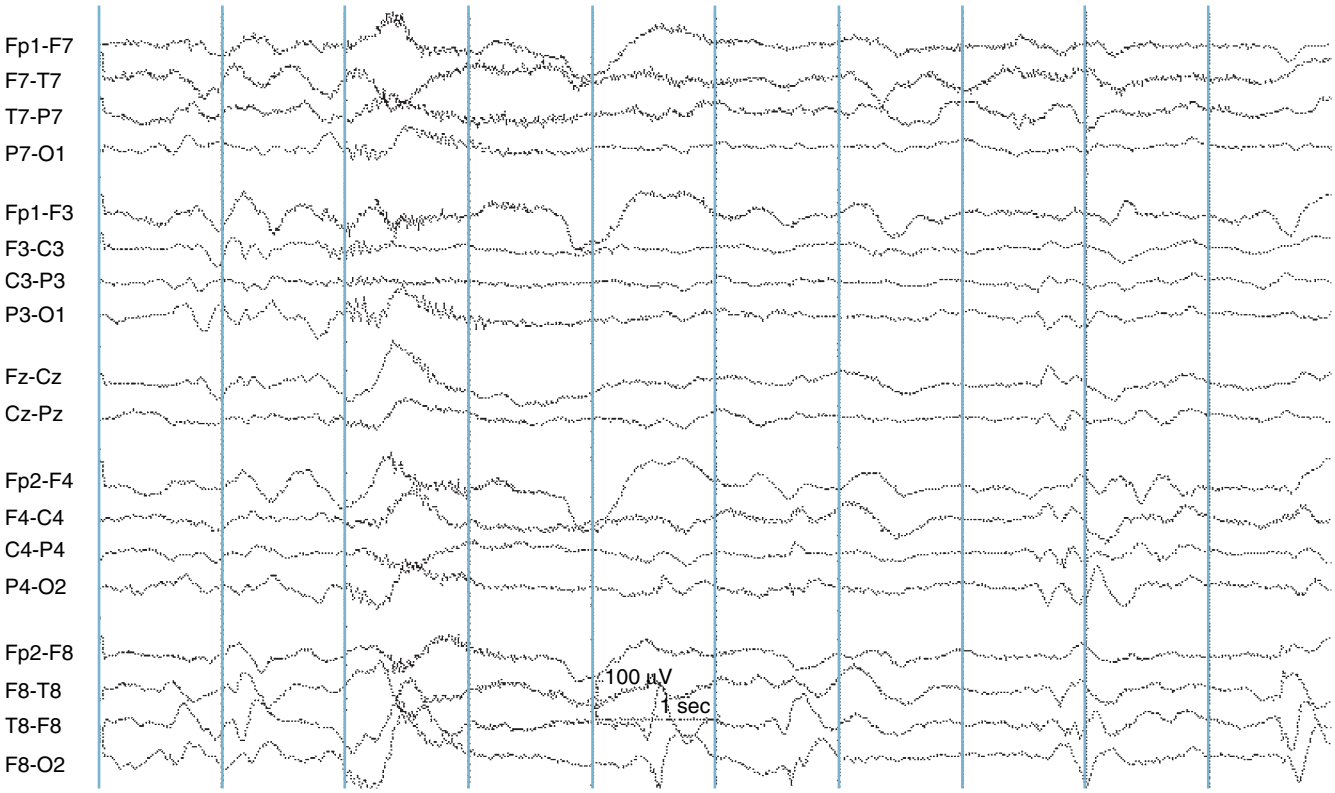


Figure 10-26 The rapid spikes seen in the third second of this tracing are associated with an infantile spasm. In some examples, the large slow wave at the onset of the seizure is more prominent.

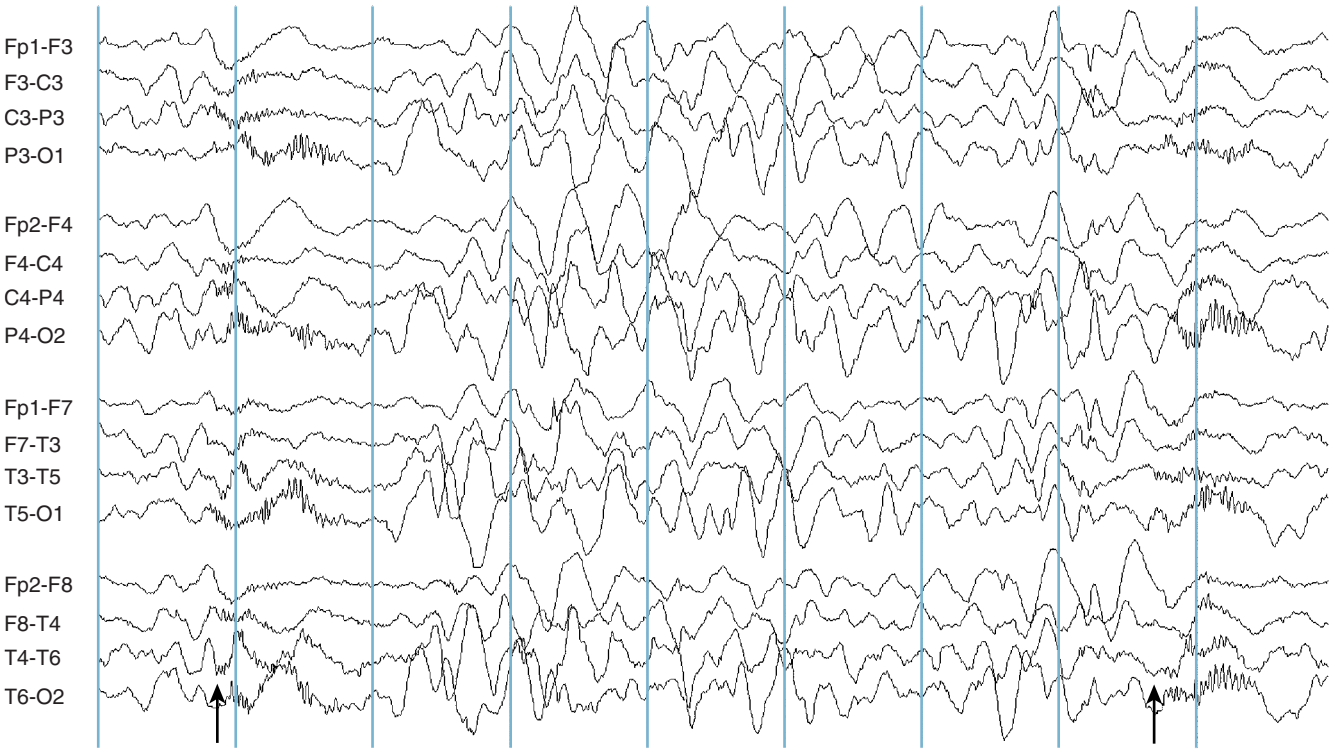


Figure 10-27 Two separate runs of rapid spikes associated with infantile spasms are seen (arrows) against the backdrop of a hypsarrhythmic EEG.

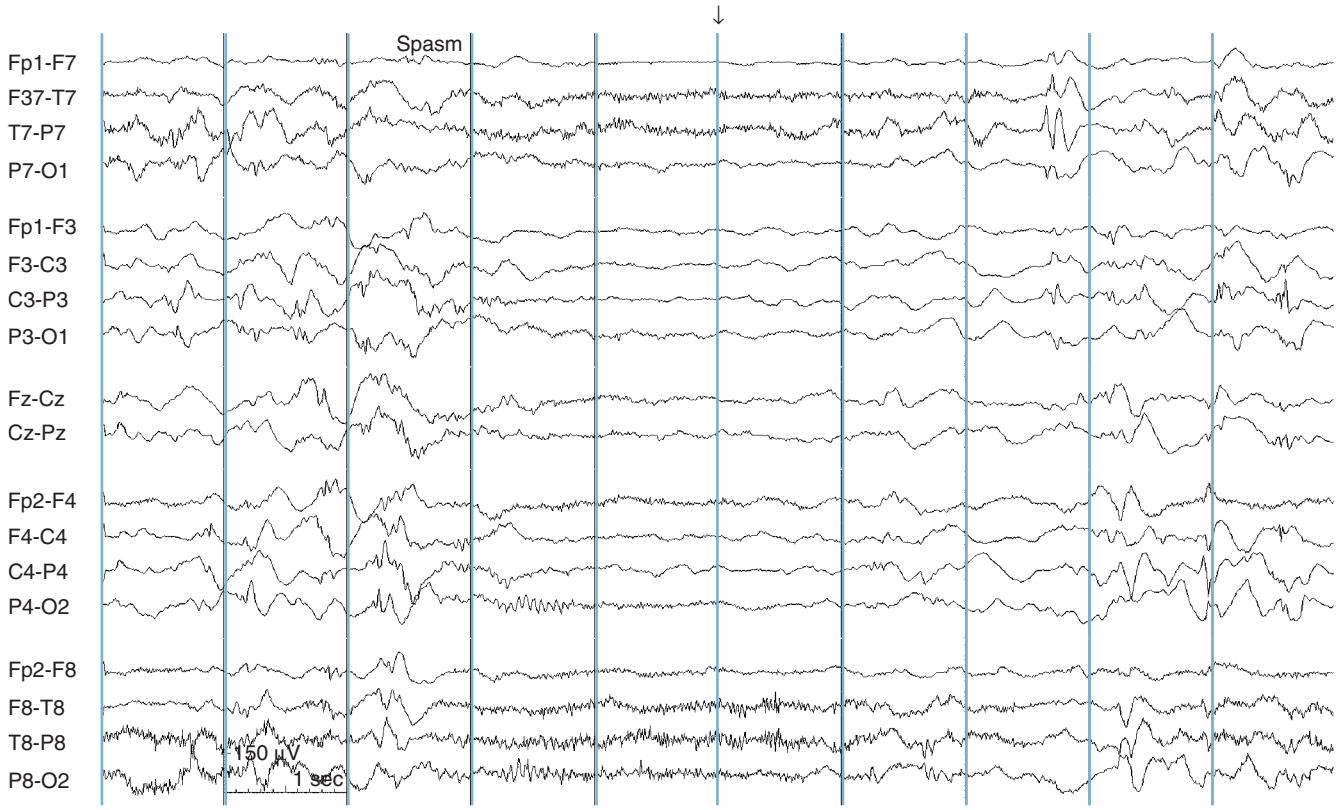


Figure 10-28 The initial clue to the occurrence of this infantile spasm is the abrupt flattening of the previous hypsarrhythmic pattern. Closer analysis shows two seconds of diffuse, rapid spikes. Although the rapid spikes are helpful in ascertaining the seizure pattern of infantile spasms, they are not always visible.

behind the concept of the age-dependent encephalopathies is that the brain mounts a characteristic response to the abnormal or injured state based on the age of the individual. This age-specificity is presumably based on differences in the physical and biochemical anatomy of the brain at different ages: seizure patterns that can be generated in early infancy may be different from those that are generated in later infancy, and again from those that are generated postinfancy. The brain of the child or adult does not respond to injury by generating the seizure patterns seen in West syndrome. As described earlier, EME and EIEE are age-dependent patterns seen in very early infancy, usually the neonatal period, at ages before West syndrome typically presents. Lennox-Gastaut syndrome, described later, is an age-dependent encephalopathy seen in later childhood and adulthood and may appear after the resolution of West syndrome in some cases.

Benign Partial Epilepsies of Childhood

The benign partial epilepsies of childhood are a group of seizure disorders associated with normal neurodevelopmental status, normal neuroimaging, a favorable long-term outcome, and a specific age range of occurrence. This is a group of disorders that are believed to be genetically transmitted and are often associated with distinctive EEG patterns.

In comparison to the majority of nonsyndromic focal epilepsies that are believed to be associated with one or more distinct cortical lesions such as an injury or a dysplasia, the benign partial epilepsies of childhood are considered nonlesional; no abnormalities are seen either on imaging or on pathologic examination. In the benign partial epilepsy syndromes, bilateral independent seizure onsets and bilateral independent EEG abnormalities are commonplace. This type of symmetrically bilateral presentation would not be expected in a lesional epilepsy because such a pattern would imply the presence of symmetrical cortical lesions in both hemispheres, an unlikely occurrence. For this reason, when bilateral focal discharges are seen during childhood in the right clinical setting, particularly in the case of a child with a normal neurodevelopmental status, the diagnosis of a benign partial epilepsy of childhood should be considered.

The two most common forms of benign partial epilepsy of childhood are benign childhood epilepsy with centrotemporal spikes (BCECTS; also known as benign rolandic epilepsy) and benign childhood epilepsy with occipital paroxysms. These two benign partial epilepsy syndromes are now discussed in more detail.

Benign Childhood Epilepsy With Centrotemporal Spikes or Benign Rolandic Epilepsy

BCECTS or benign rolandic epilepsy is one of the commonest forms of childhood epilepsy. The term *rolandic epilepsy* is based on the most frequent localization of

the EEG spikes, the rolandic sulcus (see Figure 10-29). Because the initial description of the syndrome was made by a Bavarian physician, Martinus Rulandus, some favor the term Rulandic seizures, which, by coincidence, is quite similar to the more commonly used term, rolandic seizures. Several other synonyms for this seizure type exist, including *sylvian seizures,* based on the localization of the discharges to the area around the sylvian fissure. The EEG spikes of benign rolandic epilepsy are usually of highest voltage in the central (C3 and C4) or midtemporal (T7 and T8) electrodes, hence the term *centrotemporal* spikes.

The Two Seizure Types of BCECTS

Two specific seizure types may occur in this seizure syndrome. The first is a partial seizure primarily involving the face, either with hemifacial clonic spasms or a pulling of the face to one side. The larynx, the cortical representation of which lies near the face area at the most inferior portion of the central sulcus, is also typically involved resulting in speech arrest. Children who are old enough to do so often describe a sensory change inside the mouth or on the inside of the cheek that heralds the seizure, presumably caused by the seizure discharge's involvement of the adjacent postcentral gyrus (face sensory area). This type of partial seizure tends to occur in the early morning. The classic description is of a child who walks into his parents' room in the early morning with hemifacial clonic contractions, drooling and unable to speak. Some children may experience these partial seizure episodes during sleep, often within the first few hours of falling asleep. Less often, the partial seizures occur later in the day, but this is the exception rather than the rule. When these spells occur during wakefulness, the child is often fully aware of the seizure. Complex partial versions of these seizures, associated with diminished awareness, occur more rarely.

The second type of seizure associated with benign rolandic epilepsy is a grand mal convulsion, which almost always occurs out of sleep, either during the night or less often during daytime naps. If a child has a grand mal convulsion that begins out of wakefulness, the diagnosis of rolandic epilepsy is unlikely.

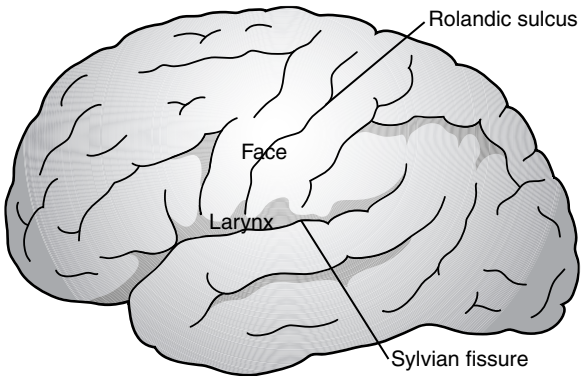


Figure 10-29 A lateral view of the left cerebral hemisphere shows the location of the rolandic (central) sulcus and the Sylvian fissure. The cortical areas for the larynx and face, which are near the Sylvian fissure, are shown.

Age Range in BCECTS

Most rolandic seizures occur between the ages of 6 and 13 years. It is rare to see the seizures persist past 13 years of age. Of all seizure types and seizure syndromes, the seizures of benign rolandic epilepsy are the type that most uniformly disappears with age and the only type for which the physician can more or less guarantee the patients and families that the seizures will "go away." Loiseau et al. (1988) were able to locate 168 of the 267 patients who were over age 20 years who they had earlier treated for rolandic epilepsy. Of these, 165 of the 168 were seizure-free; of the three with recurrences, two had only had a single additional seizure. The three cases of 168 who had experienced later seizures represents a prevalence rate that is not dramatically different from the rate of epilepsy seen in the general population.

EEG Findings in BCECTS

The EEG pattern seen in BCECTS is distinctive and this seizure syndrome is one of the few named after its EEG finding. Typically, high-voltage spike-wave discharges are seen in the centrotemporal areas (C3/T7 and C4/T8). In a given patient, the discharge maximum may be seen in the central electrode, the midtemporal electrode, or it may be shared between the two (see Figure 10-30). During wakefulness, the discharges may occur independently on each side; in sleep, the discharges often become bilaterally synchronous. It is particularly reassuring to find centrotemporal discharges over both sides of the brain when making the diagnosis of rolandic epilepsy, but in some patients the discharges are only recorded over a single hemisphere, a pattern that is still consistent with the diagnosis of BCECTS. In fact, such patients with unilateral discharges on the EEG may have discharges seen over the opposite hemisphere on the next recording. This ability of the discharges to switch sides or appear on both sides confirms the idea that benign rolandic epilepsy is not a lesional epilepsy but rather represents a genetic disorder; to say that a symmetrical, bilateral independent pattern is caused by a lesional epilepsy would imply that the patient has two separate lesions in the same brain location but on opposite sides, an unlikely occurrence.

Although, by far, centrotemporal spikes are the most common finding in benign rolandic epilepsy, other spike localizations may be seen less frequently. The most common alternate spike locations are the centroparietal areas (C3/P3 and C4/P4), the parietal areas (P3 and P4), and the central and parietal midline (Cz and Pz electrodes; see Figure 10-31). Occipital discharges are known to occur with increased frequency along with the centrotemporal spikes of rolandic epilepsy. Certain spike localizations, such as the anterior temporal areas (F7 and F8), are not expected in BCECTS. Interestingly but somewhat unexpectedly, generalized spike-wave discharges are also seen to coexist with centrotemporal spikes in the EEGs of patients with BCECTS much more often than would be expected based on chance (see Figure 10-32).

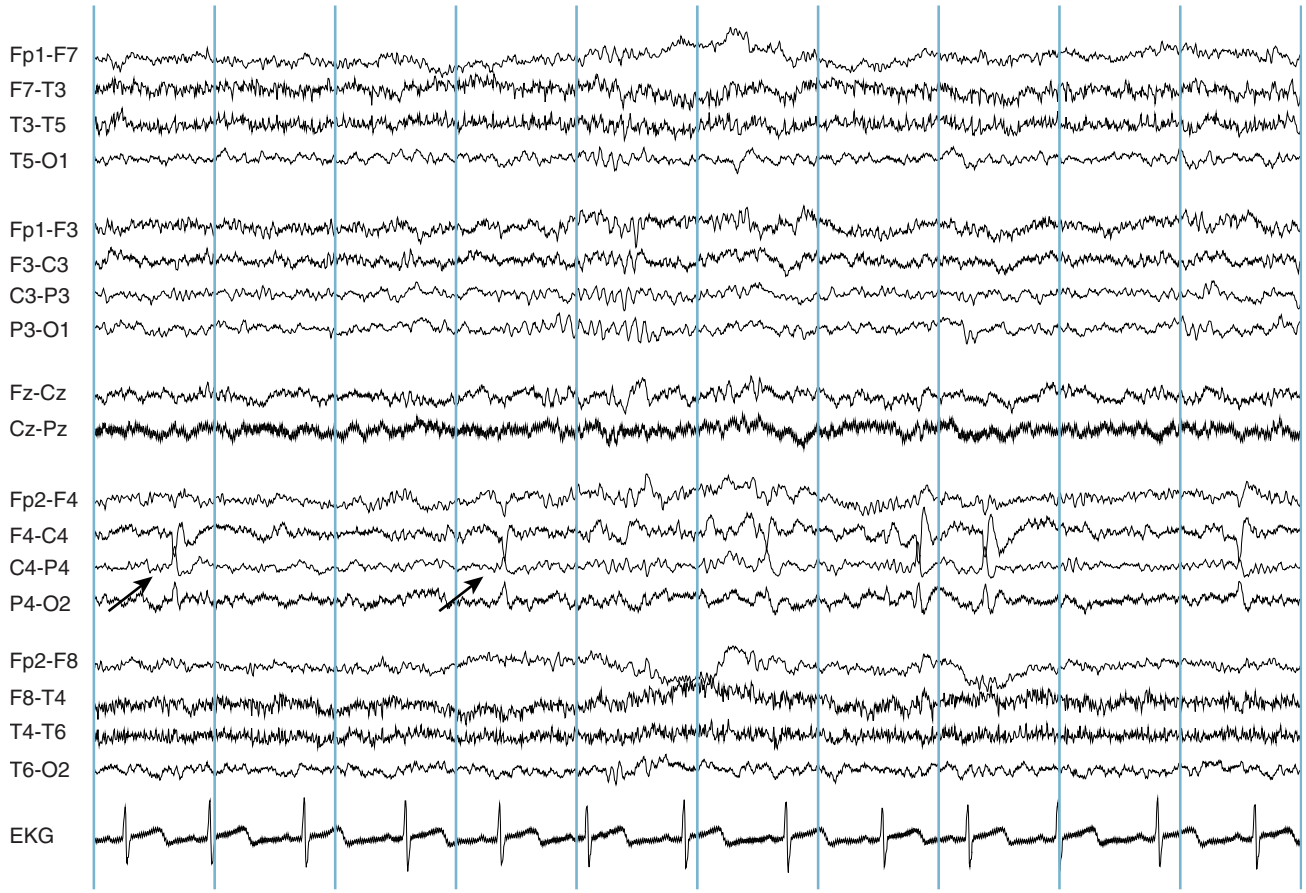


Figure 10-30 The typical appearance of right central rolandic spike-wave discharges are shown; two are indicated by arrows. These examples are highly localized to the C4 electrode, but in other cases the discharge may be maximum in the midtemporal electrode or the maximum may be shared between the two positions.

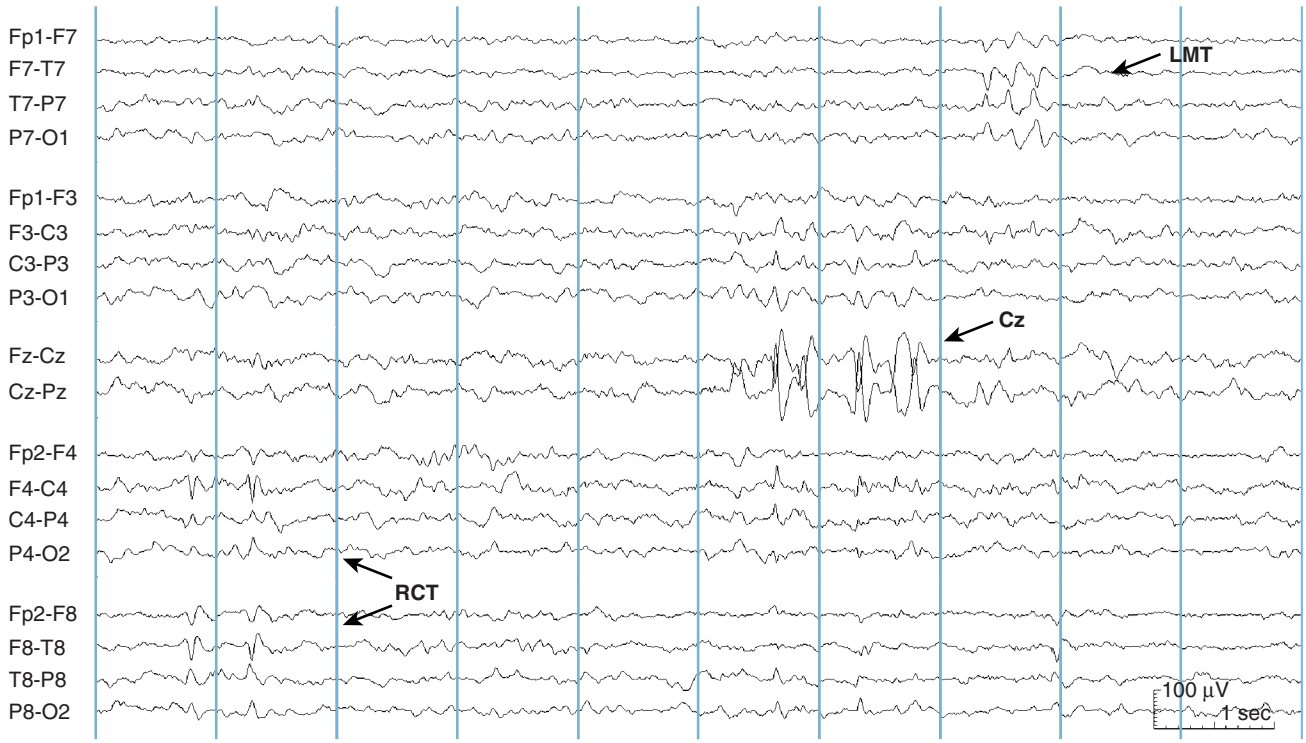


Figure 10-31 In addition to the more "standard" spike localizations in the right centrotemporal (RCT) and left midtemporal (LMT) areas, this 12-year-old boy also has spike-wave discharges in the central midline (Cz), a less common spike location for this syndrome.

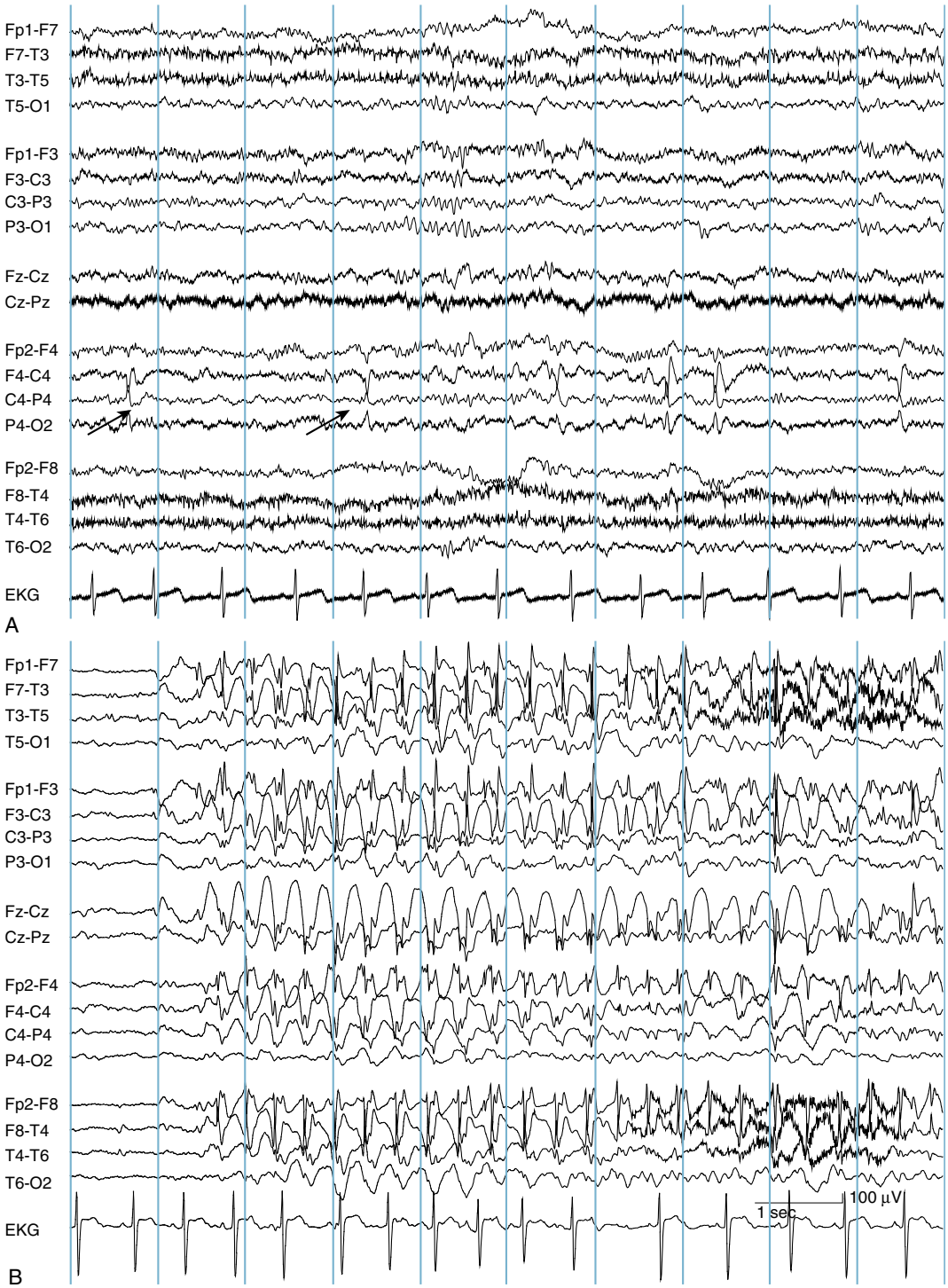


Figure 10-32 The first page **(A)** shows right central spike-wave complexes (arrows) in a patient with rolandic epilepsy. The second page **(B)** is recorded later in the same patient at sleep onset showing a run of generalized spike-wave discharges. Despite the apparent focal nature of rolandic epilepsy, many benign rolandic epilepsy (BRE) patients manifest generalized spike-wave in the EEG at some point in their course. Despite these generalized discharges, rolandic epilepsy patients rarely manifest frank absence seizures.

The centrotemporal spikes of rolandic epilepsy have a distinctive topography, referred to interchangeably as a "tangentially oriented" or "horizontal" dipole. The finding of a horizontal dipole further increases the certainty that a centrotemporal spike found in the EEG is associated with the benign rolandic epilepsy syndrome.

Spike Dipoles and the Horizontal Dipole of Rolandic Epilepsy

Considering the orientation of the dipole of any spike, it is clear from the laws of physics that any time a net negative charge is measured, there must be a counterbalancing net positive charge in some other

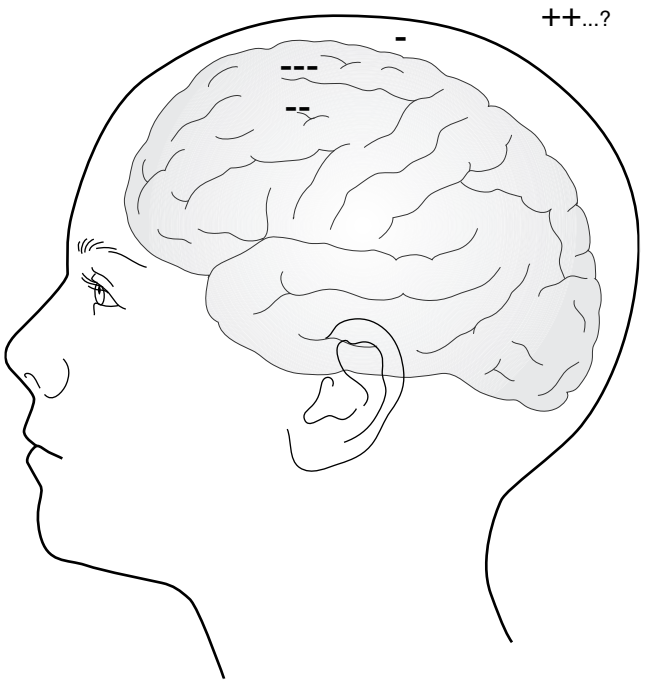


Figure 10-33 When a spike recorded from the scalp surface consists of pure negativities, the question arises as to the location of the unrecorded counterbalancing positivity.

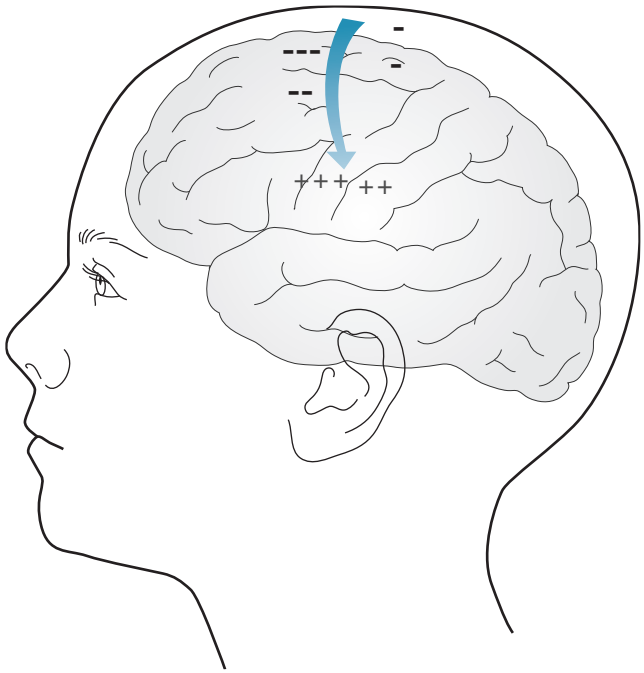


Figure 10-34 When pure negativities are recorded from the scalp surface, the "missing" positivities are presumed to lie at a location deeper in the brain. This implies the presence of a dipole that is oriented more or less parallel to one of the axes emanating from the brain's center. The "hidden" positivities are shown in blue, indicating that they cannot be recorded from the scalp surface. The large majority of scalp-recorded epileptic spikes are oriented as radial dipoles.

location (see Figure 10-33). In the practice of conventional EEG recording, the large majority of epileptic spikes manifest a negativity measured on the scalp, but no concurrent scalp positivity is measured. In such cases, it is inferred that there must be an unmeasured positivity somewhere deep to the scalp. The simultaneous coexistence of a superficial negativity and a deep positivity implies a radially oriented dipole, by far the most common orientation of epileptic spikes (see Figure 10-34).

In contrast, the dipole of the centrotemporal spikes of BCECTS often is not oriented in the direction of the more common radial dipoles described earlier, but rather manifests a *horizontal dipole*. At the same time as a negativity is measured in the centrotemporal electrodes, a simultaneous positivity is measured in the anterior electrodes (see Figure 10-35). The simultaneous recording of a negativity in the centrotemporal area and a positivity in the frontal area implies an electrical dipole oriented tangent to the scalp, pointing from the centrotemporal area to the frontal areas (see Figures 10-36 and 10-37). Thus, with the more commonly occurring radial dipoles, a negativity is measured on the scalp, and a positivity is *inferred* deep to the scalp. Discharges manifesting the more rarely occurring horizontal dipole manifest a scalp-to-scalp dipole, with a negativity in one scalp area and a visibly measured positivity in another scalp area.

Note that in this discussion and in the EEG literature in general, the terms *horizontal dipole* and *tangentially oriented dipole* are used interchangeably. Although both terms are commonly accepted, the latter is technically superior because the dipole being described

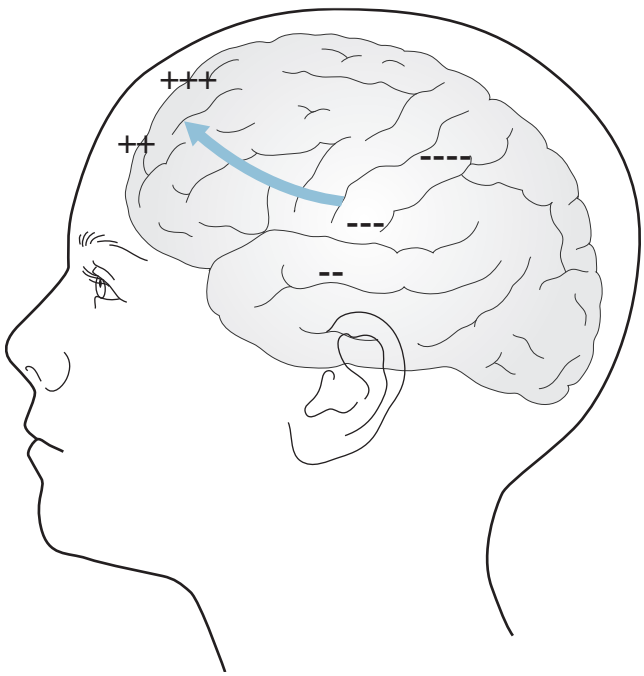


Figure 10-35 The classic discharge of benign rolandic epilepsy consists of a negativity recorded from the centrotemporal area(s) and concomitant positivities recorded from the anterior brain areas. This configuration of polarities suggests the presence of a dipole-oriented tangent to the scalp as shown.

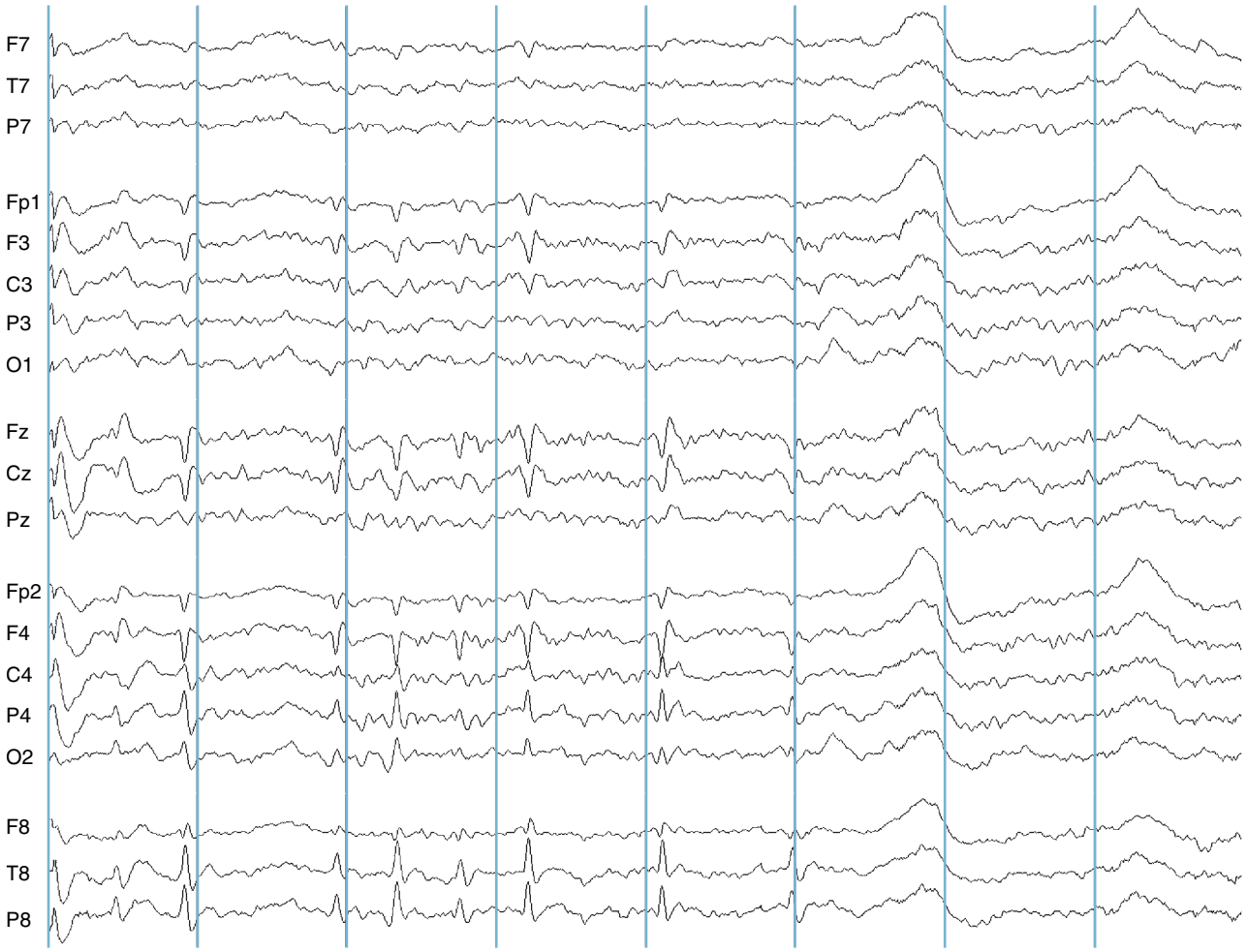


Figure 10-36 This EEG is recorded in a referential montage using the nose as the reference electrode. Negativities manifest as upgoing waves and the positivities as downgoing waves. The primary (highest voltage) negativity is seen in T8 with secondary negativities in P4 and P8. A smaller amount of negativity is picked up by the O2 electrode. What distinguishes this discharge from most scalp-recorded spikes are the positive deflections recorded by the anterior electrodes: Fp2, F4, Fz, Cz, and Fp1 and F3. The simultaneous recording of a negative charge in one scalp location and positive charge in another scalp location constitutes a tangential or "horizontal" dipole. The next figure shows a schematic representation of this dipole.

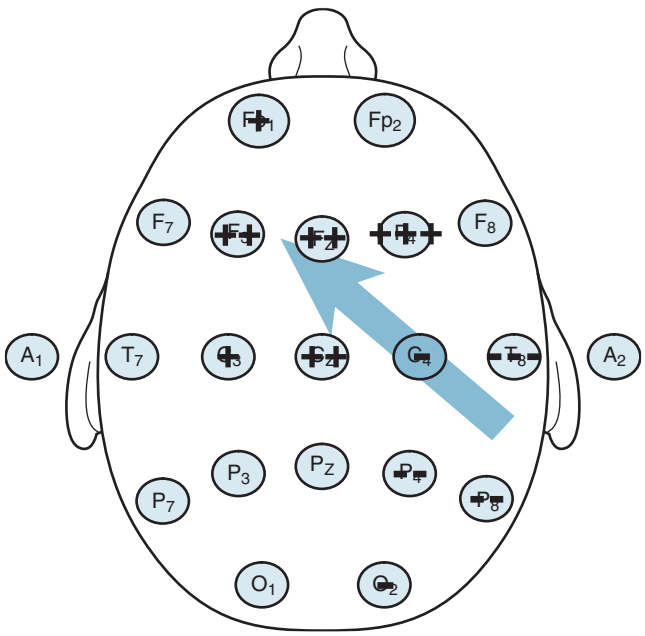


Figure 10-37 The net negativities and positivities recorded in the EEG trace from the previous figure are shown schematically. The presence of a peak negativity in the right midtemporal area and net positivities in the anterior electrodes suggests a dipole tangent to the scalp as depicted by the dipole arrow.

is always tangential to the scalp, no matter what the patient's head position. As patients move their heads, a tangential dipole will not always remain parallel to the horizon as the term horizontal implies. Nevertheless, the term horizontal dipole remains in common usage.

Pitfalls in the Identification of the Horizontal Dipole

The successful identification of a tangentially oriented dipole in the setting of rolandic epilepsy relies on the ability to detect the spike's frontal positivity. As it happens, this pattern is most easily demonstrated using referential montages. In referential montages with a well-chosen reference, the primary centrotemporal spike is seen as upgoing (negative) deflections in the centrotemporal area, and the anterior positivity is seen as simultaneous downgoing (positive) deflections in the frontal areas. As it happens, the pattern of the horizontal dipole (specifically, demonstration of the frontal positivity) is more difficult to appreciate in bipolar montages. In fact, even when a significant horizontal dipole is present, it usually cannot be appreciated in a bipolar montage (see Figure 10-38). In only a small minority of cases can a horizontal dipole be detected in a bipolar montage by way of an anterior *positive phase reversal* (see Figure 10-39). For that reason, a referential montage must be examined before the presence of a horizontal dipole can be excluded. Because a horizontal dipole provides important confirmatory information regarding a possible diagnosis of rolandic epilepsy, the presence or absence of such a dipole should always be sought when a discharge is seen in this location. The absence of a horizontal dipole does not, however, exclude the diagnosis of rolandic epilepsy because such a dipole is not demonstrable in approximately half of all cases.

As noted earlier, the horizontal dipole of the rolandic discharge is best demonstrated on referential montages. The choice of reference in this setting deserves special attention. The search for the positive component of the rolandic discharge entails identification of downgoing ("positive") deflections in the frontal channels. Consider,

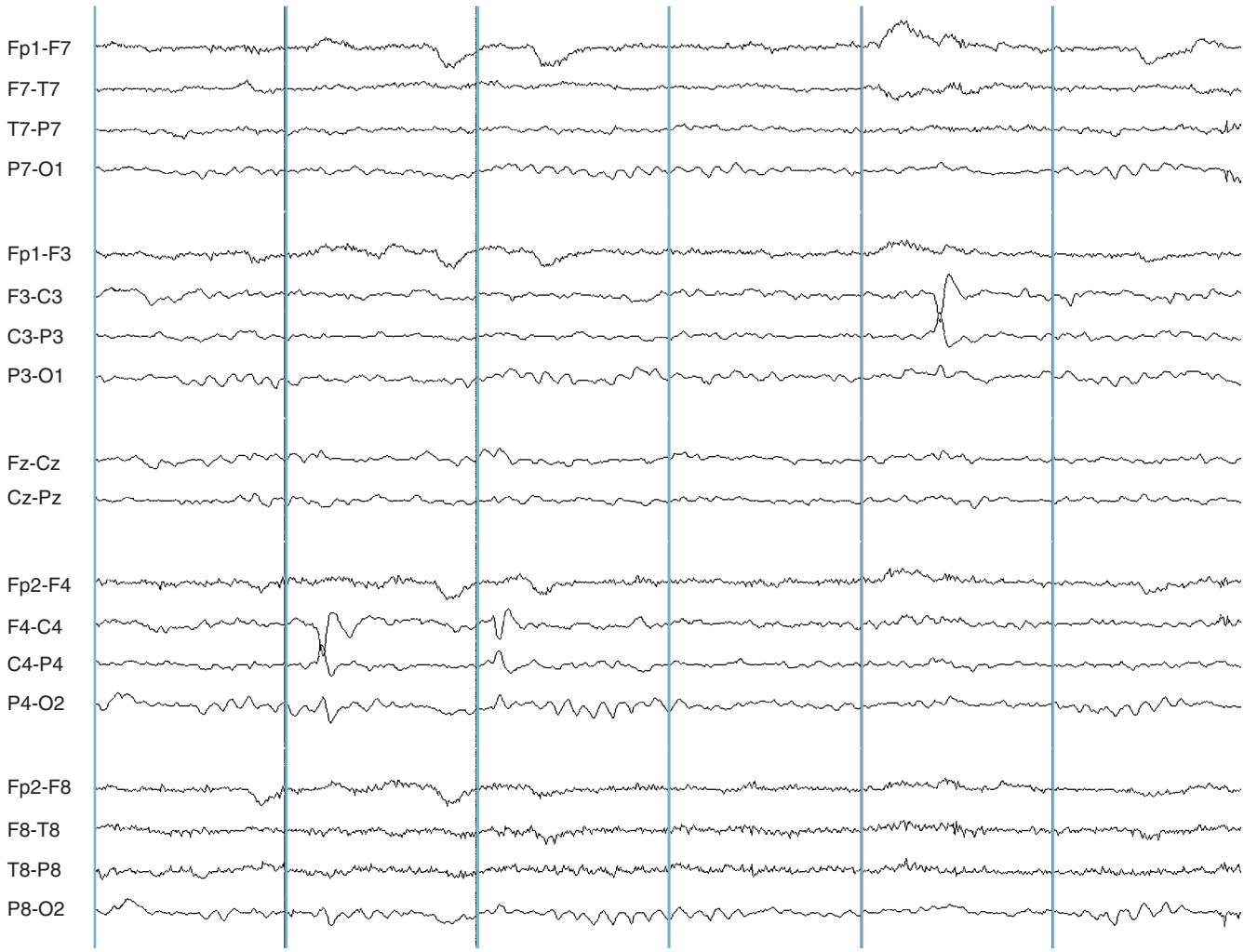


Figure 10-38 Even when centrotemporal discharges have a true horizontal dipole, the dipole orientation is not usually visible in bipolar montages. For instance, the horizontal dipole of these independent C3 and C4 discharges cannot be discerned from this tracing (compare to Figure 10-39).

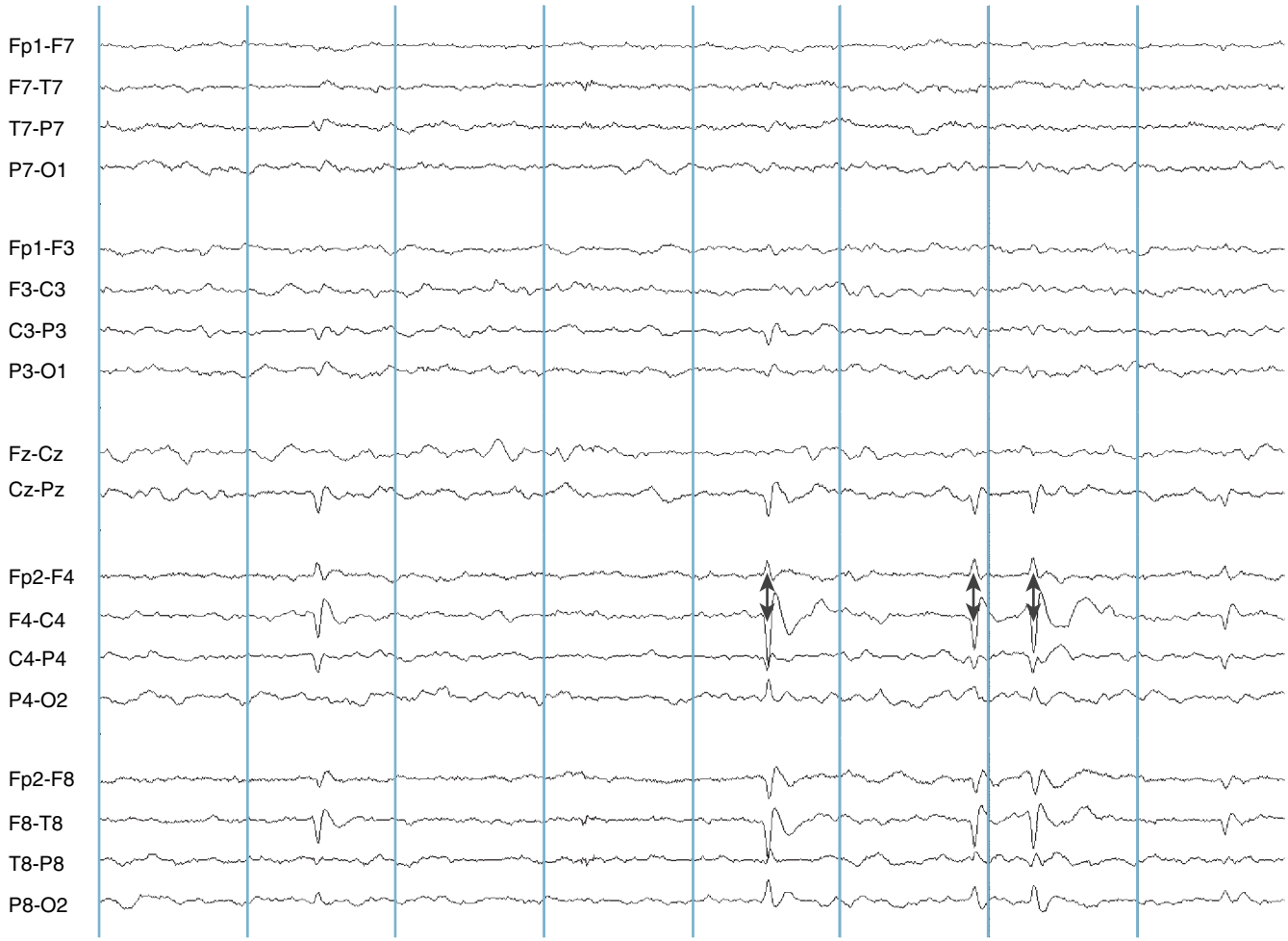


Figure 10-39 A right centrotemporal discharge is seen in a bipolar montage. The presence of an anteriorly oriented horizontal dipole is indicated by the positive phase reversals anterior to the peak negativities (arrows). In fact, this appearance represents the exception to the rule—even when present, horizontal dipoles usually cannot be appreciated in bipolar montages. The presence of a horizontal dipole can only be excluded with certainty by examining an appropriate referential montage.

however, the situation in which the reference chosen for a referential montage is contaminated with the active discharge. The example of a contaminated earlobe reference is easy to visualize. First, we examine the example of a nonrolandic discharge (that does not manifest a horizontal dipole) that is maximally negative in the left midtemporal electrode (T7). Recall that it is not uncommon for the adjacent earlobe electrode (A1) to pick up some portion of the electrical activity occurring in T7. If the A1 electrode is used as the reference electrode, A1 will partially record the negative event coming from the nearby centrotemporal area. As the voltage measured in A1 is subtracted from the frontal electrodes that, in this example, are truly neutral, the subtraction of the A1 reference will result in downgoing deflections in the channels corresponding to the neutral electrodes. The appearance of these downgoing waves is quite similar to the usual appearance of a true horizontal dipole, with negativities (upward deflections) in the centrotemporal area and positivities (downward deflections) in the anterior electrodes. In fact, in this type of case in which the reference is contaminated with the active discharge, downward deflections will also be present in the channels of the other neutral electrodes that use A1 as a reference (see Figures 10-40 through 10-42).

This type of error can be avoided first by choosing a reference that is distant from the active discharge and therefore inactive. When it occurs, this type of error can be detected by noting the distribution of downward deflections in *all* of the neutral electrodes' channels (rather than just the frontal channels) as is seen in Figure 10-42, thereby identifying those deflections as contamination of the reference rather than true positive events on the scalp. The same type of error can be made when an average reference electrode is used if the centrotemporal discharge is so large that it is visible in the average. The general problem of misinterpretation of discharge topographies based on contaminated reference electrodes is discussed in Chapter 4, "Electroencephalographic Localization."

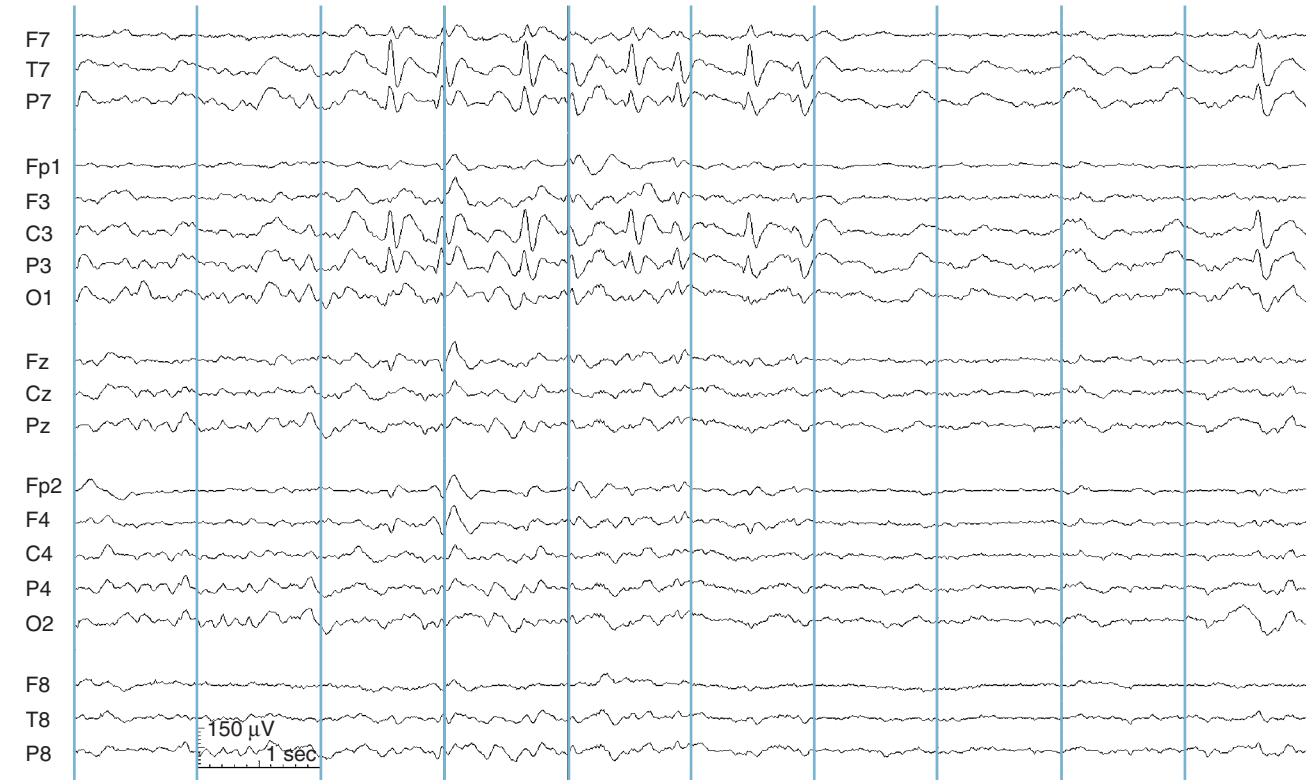


Figure 10-40 This left centrotemporal discharge, maximum in the T7 and C3 electrodes and recorded in a referential montage using the nose as the reference electrode, does not manifest a horizontal dipole. Note that all deflections are upgoing implying that only negative charges are present on the scalp (compare with Figure 10-36).

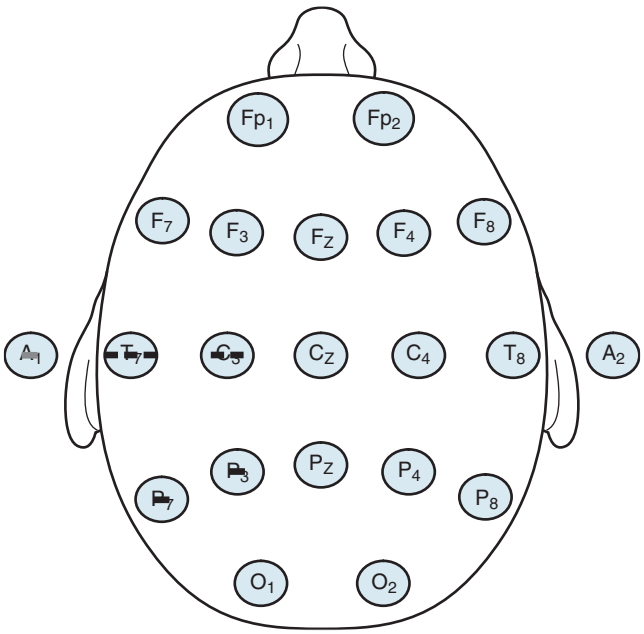


Figure 10-41 A schematic is shown of the purely negative discharge seen in the previous figure (Figure 10-40). Only negativities are recorded on the scalp implying that a positivity must be present deep to the scalp. Note the small amount of negativity overflows the left earlobe.

Rolandic EEG Trait

Almost all children with benign rolandic epilepsy manifest rolandic discharges during the course of their seizure disorder. Interestingly, it has long been known that the siblings of children with rolandic epilepsy may also manifest rolandic discharges in the EEG, even when those siblings do not have the seizure disorder. Therefore, the tendency to manifest rolandic discharges in the EEG appears to be a heritable trait, and only a subset of all children who have rolandic discharges in the EEG actually have rolandic epilepsy. The finding of rolandic discharges in the EEG in the absence of the seizure disorder is referred to as *rolandic EEG trait*. It may come as a surprise to learn that rolandic EEG trait is much more common than rolandic epilepsy itself. Some estimate that of all children in the general population with rolandic discharges in the EEG, as many as 90% only have rolandic EEG trait and do not actually have the epilepsy syndrome, that is, they do not have the seizures of rolandic epilepsy. Others estimate this percentage to be even higher. This fact has important ramifications in EEG interpretation. Rolandic EEG trait has such a high frequency in the population that the mere observation of rolandic discharges in the EEG should not automatically lead to the diagnosis of rolandic epilepsy or seizure disorder without careful consideration of the clinical history.

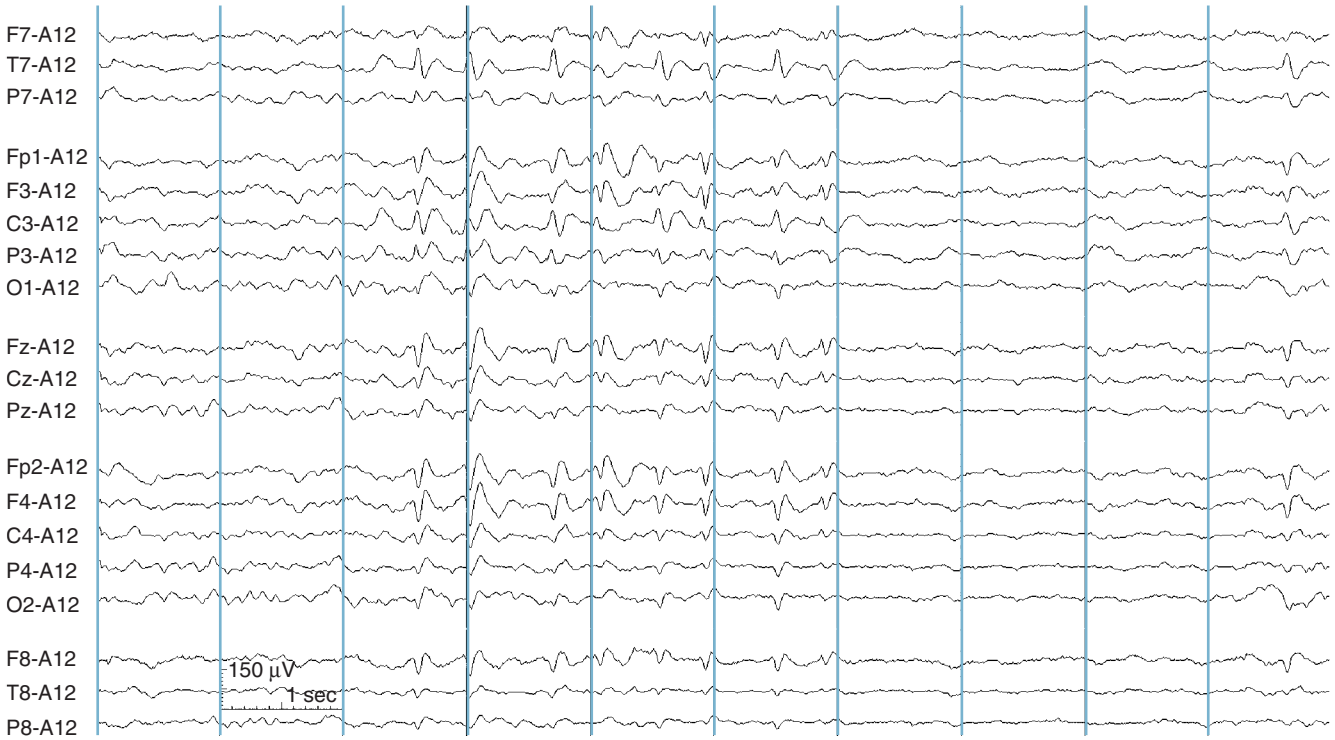


Figure 10-42 A left midtemporal (T7) rolandic discharge is recorded with suboptimal technique, giving the false impression of a horizontal dipole. The reference electrode used in this recording is an average of the earlobe electrodes, A1 and A2. Because A1 is adjacent to T7, it detects the negativity from the left midtemporal area. When this negativity is subtracted from neutral electrodes, an upgoing deflection results. Note that electrodes in areas that are presumably completely neutral (e.g., the right occipital area, O2) still manifest the upgoing deflection, which represents contamination of the reference rather than a true positivity. Compare this appearance to the true horizontal dipole shown in Figure 10-36 in which, because it was recorded with an appropriate reference electrode, distant electrodes are neutral.

Because of the relatively high frequency of rolandic EEG trait in the population, the presence of centrotemporal spikes in the EEG often represents an incidental finding. Consider the scenario of an 11-year-old girl with an episode of loss of consciousness in science class. An EEG is obtained that shows typical rolandic discharges with a horizontal dipole. In reality, the chances that this child's episode was an epileptic seizure are not increased by the abnormal finding in the EEG. As described earlier, the BCECTS syndrome consists either of partial seizures out of wakefulness or grand mal convulsions arising out of sleep. Because this child lost consciousness, this event is not likely to have represented a partial seizure, and, if it had represented an example of a grand mal convulsion, it does not fit with the rolandic epilepsy syndrome because it occurred out of wakefulness. Even though the EEG findings are considered abnormal, the finding of rolandic discharges does not match the clinical picture of the event. It is quite possible that this episode represented an example of syncope, despite the abnormal EEG finding. Because such a high proportion of individuals with rolandic discharges do not have epilepsy, the clinician should always confirm that the event history is consistent with the seizure types seen in the BCECTS syndrome before arriving at the diagnosis of epilepsy in this group of children.

Another way to look at this problem is to consider the difference in the rate of epilepsy between two groups of children who are found to have rolandic discharges: a group of children with rolandic discharges who have been referred to an EEG laboratory because of a possible seizure event and a group of children whose rolandic discharges have been identified during a hypothetical school-based study in which all children of a certain age group, regardless of their medical history, are studied by EEG. In the case of the school study, the large majority of children with rolandic discharges in the EEG will not have epilepsy. In the laboratory-referred group, however, the patient group is significantly enriched for epilepsy because the majority have been referred for seizure-like behaviors. In practice, some children are referred for EEG evaluation in whom suspicion for seizure is low, and the odds of having epilepsy, despite the presence of rolandic discharges, may be closer to the group from the school-based study example than the laboratory-referred group. In such cases, the mere presence of rolandic discharges should not compel a diagnosis of epilepsy. This is an excellent example of the maxim that "an abnormal EEG does not make the diagnosis of epilepsy." A properly written EEG report should reflect this fact by describing rolandic discharges as consistent with both rolandic epilepsy and rolandic EEG trait.

Childhood Epilepsy With Occipital Paroxysms

Two types of benign occipital epilepsy have been recognized, a more common, early-onset variety (also referred to as Panayiotopoulos type) and a late-onset variety (referred to as Gastaut type).

Early Onset Childhood Occipital Epilepsy

In the early-onset form of childhood occipital epilepsy, seizures begin between the ages of 3 and 6 years. The seizures of this syndrome are distinctive and include autonomic symptoms such as pallor, sweating, and irritability. The seizures may evolve to vomiting, eye deviation, and loss of consciousness, culminating in convulsions (either hemicolonic or generalized clonic). Even taking into account the fact that the ability to report visual manifestations may be limited in the children in this younger age group, visual phenomena are believed to be rare. Nocturnal seizures are somewhat more common than daytime seizures. Approximately two thirds of children manifest occipital spikes or occipital paroxysms (discussed later). In the early-onset version of this syndrome, seizures tend to be infrequent with some children experiencing only one or two seizure episodes throughout the syndrome's course. The seizures tend to remit within a few years of presentation. The small number of lifetime seizures and tendency toward natural remission reduce the necessity of treating with anticonvulsant medications.

Late-Onset Childhood Occipital Epilepsy

The late-onset version of childhood occipital epilepsy is felt to be considerably (fivefold) less common than the early-onset version. The mean age of onset in the late-onset form is 8 years, compared with 5 years for the early-onset form. In the late-onset form, seizures are much more frequent and tend to occur in the daytime. Unlike the early-onset form, visual phenomena are highly characteristic of the seizures in the late-onset form, consisting of elementary (or more rarely) complex visual hallucinations. Blindness may occur as either an ictal phenomenon or a postictal phenomenon. Headache and, more rarely, nausea and vomiting can be associated seizure symptoms.

EEG Findings in Childhood Occipital Epilepsy

Both the early and late forms of occipital epilepsy may show the classically described pattern of occipital spikes that suppress with eye opening, the so called "fixation-off" effect (so-called because the spikes "turn off" with visual fixation). In fact, any maneuver that interrupts visual fixation, such as shutting off room lights, can prompt the appearance of occipital spike activity. Although fixation-off occipital spiking is the classic EEG finding of the benign occipital epilepsies, it is not uniformly seen in these syndromes; it is estimated that occipital spikes are only seen in two thirds of children with benign childhood occipital epilepsy. In the remaining third, spikes may be seen in extraoccipital locations or the EEG may even be normal. Whereas some children may have the classic finding of paroxysmal occipital spikes, in others the spikes may occur singly. The spikes are often bilateral, but in some they may be unilateral (see Figure 10-43). The yield of finding EEG abnormalities is increased by obtaining sleep. Although it is the classic EEG finding of the childhood occipital epilepsy syndromes, fixation-off occipital spikes may also be seen in other types of occipital epilepsy outside of the benign group described here; the presence of fixation-off occipital spikes does not guarantee the presence of a benign occipital epilepsy syndrome.

Especially in the early-onset version of the syndrome, spikes may be seen in extraoccipital locations, including the centrotemporal areas. Like in BCECTS, generalized spike-wave discharges may also be seen in the childhood occipital epilepsy syndromes. The not infrequent co-occurrence of occipital spike-wave discharges and centrotemporal spike-wave discharges suggests the possibility of some commonality between benign childhood epilepsy with occipital paroxysms and benign rolandic epilepsy. The term *benign childhood seizure susceptibility syndrome* has been proposed to encompass the group of children who manifest a tendency toward the benign focal epilepsies, including children with benign rolandic epilepsy, rolandic EEG trait, childhood epilepsy with occipital paroxysms, and other more poorly defined benign focal epilepsies of childhood. Further delineation of the genetics of this group of disorders may help clarify whether the construct of a benign childhood seizure susceptibility syndrome is valid (Table 10-2).

Autosomal Dominant Nocturnal Frontal Lobe Epilepsy

Autosomal dominant nocturnal frontal lobe epilepsy (ADNFLE) is an unusual epilepsy syndrome that may have onset as early as infancy but as late as adulthood, although onset in the childhood years is typical. The seizures can be difficult to diagnose because they manifest as violent, uncoordinated thrashing occurring out of light sleep ("hypermotor seizures"). The spells are often mistaken for nightmares or other parasomnias, for a movement disorder, or sometimes for a psychiatric disturbance. Distinguishing between the seizures of ADNFLE and nocturnal movement disorders such as paroxysmal nocturnal dystonia can be difficult in some cases, and controversy still persists regarding the exact demarcation between these two syndromes. In a minority of cases, genetic abnormalities have been identified in the neuronal nicotinic acetyl-choline receptor.

EEG Findings in ADNFLE

The EEG findings in ADNFLE are variable; typically the interictal EEG is normal. When the interictal EEG is abnormal, anterior focal epileptiform discharges may be seen. Ictal EEG recordings can be difficult to interpret because they are often obscured by motion artifact

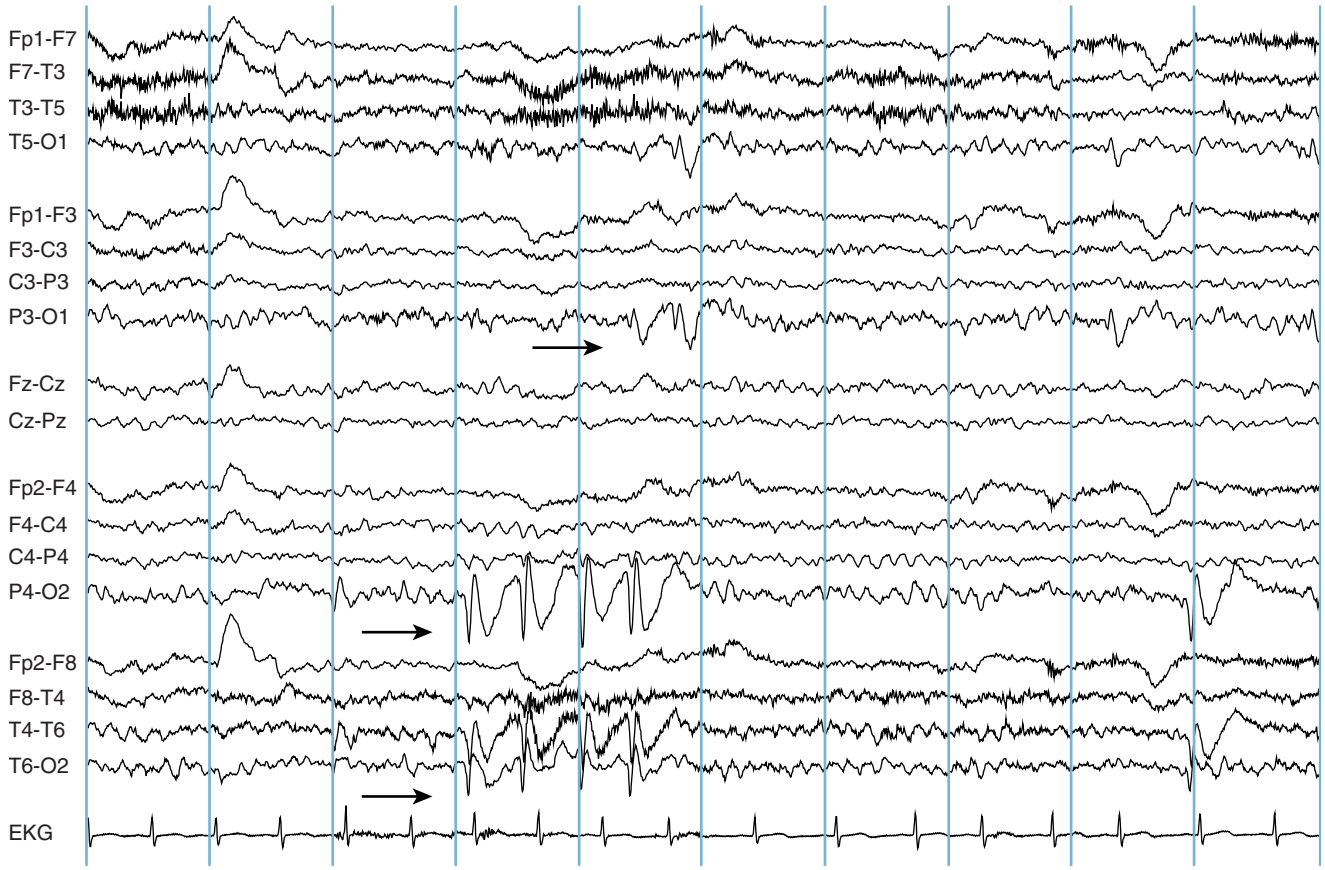


Figure 10-43 A paroxysm of occipital spikes is seen in the right occipital area (black arrows). Independent but lower voltage occipital spikes are seen in the left occipital area (lighter arrow). The paroxysmal bursting pattern seen here is consistent with that seen in the BCEOP (benign childhood epilepsy with occipital paroxysms) although the spikes are often bilaterally synchronous in those syndromes. Fixation-off spiking as may be seen in BCEOP is not demonstrated in this figure.

associated with the ictal hypermotor behaviors. A single intracranial electrode recording of an ADNFLE seizure has been made that demonstrated onset from the left operculoinsular cortex (Picard et al., 2000).

Primary Generalized Epilepsies

The primary generalized epilepsies are a group of genetic epilepsy syndromes characterized by generalized seizures and generalized spike-wave discharges in the EEG. The question of exactly how broad or restrictive the boundaries for this group of syndromes should be has still not been settled. The central entities in this syndrome group are those idiopathic generalized epilepsies that occur in individuals with otherwise normal neurologic and cognitive status: childhood absence epilepsy, juvenile absence epilepsy, juvenile myoclonic epilepsy, and epilepsy with grand mal seizures on awakening. Although some examples of these syndromes appear to conform to classic presentations and adhere to distinct boundaries, in other patients these syndromes may appear to blend into one another. For instance, individuals with juvenile myoclonic epilepsy may also have absence seizures, blurring the border

Table 10-2 Childhood Epilepsy With Occipital Paroxysms Subtypes

	Early Onset	Late Onset
Population Frequency	More common	Less common
Mean Age of Onset	5 years	8 years
Seizure Duration	Lengthy	Brief
Number of Lifetime Seizures	Few	Many
Timing of Seizures	Most at night	Most during day
Typical Seizure Semiology	Sweating, pallor, irritability, eye deviation, vomiting, culminating in convulsion (visual phenomena rare)	Pure visual phenomena without loss of consciousness
Long-Term Course	Early remission	Inconsistent remission during childhood, later course unclear

between juvenile myoclonic epilepsy and juvenile absence epilepsy, suggesting the possibility that some of these syndromes with different names may represent varying presentations of the same genotype.

The genetics and pathogenesis of the primary generalized epilepsies is just beginning to unfold. At least some of the generalized epilepsies have been demonstrated to be caused by sodium, chloride, or calcium-ion channel disorders ("channelopathies") and others by errors in synaptic receptors such as gamma-aminobutyric acid (GABA) receptors. As the genetics of these syndromes is further elucidated, many additional subtypes and biochemical errors will doubtlessly be identified.

A variety of terminology has been used to refer to the primary generalized epilepsies. The terms *primary corticoreticular epilepsy* and *centrencephalic epilepsy* are older terms that reflect the basic susceptibility in these patients to the occurrence of thalamocortical volleys that are the generalized spike-wave discharges characteristic of these syndromes. The same biochemical abnormality that allows for generalized spike-wave discharges to cause absence seizures is also presumed to be responsible for the more rapid discharges associated with generalized tonic-clonic seizures and the brief polyspike discharges that drive epileptic myoclonus in some patients, as described earlier.

A Note on the Terms Idiopathic, Primary, Cryptogenic, and Symptomatic

The term *idiopathic epilepsy* literally refers to an epilepsy "unto itself" (*idios*, Greek) meaning an epilepsy that has not occurred as a consequence of another condition or disorder (e.g., head trauma) but a circumstance in which epilepsy itself is the disorder. The primary generalized epilepsies fall into this group and in this usage, the term primary is essentially synonymous with idiopathic. The genetic focal epilepsy syndromes also belong to this category of idiopathic epilepsies—they are examples of epilepsy "unto itself," not a symptom of some other process. Note that the term idiopathic epilepsy does not imply an epilepsy with unknown cause, because the genetic underpinnings of the primary generalized (idiopathic) epilepsies are beginning to unfold, and genetic testing for many more of these epilepsies may be available in the near future.

Idiopathic epilepsy does not, however, simply apply to any epilepsy syndrome in which all tests are negative. In some instances, an underlying cause is suspected, although it cannot be specifically identified. Such cases are referred to as *cryptogenic epilepsy* (the cause is *hidden* but presumed to exist). For example, some individuals with temporal lobe epilepsy may have a normal MRI scan, but there is still strong suspicion of a microscopic abnormality in the temporal lobe. Because the abnormality is not detectable by currently available testing but is presumed to be present, this patient's epilepsy would be categorized as cryptogenic. Finally, the term *symptomatic epilepsy* is used for a seizure disorder that has a clear antecedent cause, for example, the seizures that follow a stroke.

Childhood Absence Epilepsy

Childhood absence epilepsy (CAE) usually has its onset between 4 and 8 years of age. The main seizure type of CAE is the typical absence seizure, described earlier, characteristically consisting of brief periods of staring, subtle rhythmic eye blinking, and unawareness of the environment. Children with untreated CAE have as many as 100 to 200 absence seizures per day. A large fraction of children grow out of the tendency to absence seizures during the teenage years, and in some 90% of CAE patients the absence seizures have cleared by the age of 20 years. Patients and families should be made aware, however, of the lifelong increased risk of generalized tonic-clonic seizures, even in CAE patients in whom the absence seizures abate during childhood.

Childhood absence epilepsy falls into the group of the primary generalized epilepsies. Such patients have a predilection for volleys of generalized spike-wave discharges, the EEG correlate of absence seizures. Typical absence seizures and generalized tonic-clonic seizures are strongly linked and likely have similar underlying mechanisms. Approximately 40% to 50% of children with CAE will also experience a generalized convulsion at some time during their lives. A minority of children may experience grand mal seizures either before or during the period of active staring spells.

EEG Findings in CAE

Confirmation of the diagnosis of absence seizures hinges on EEG demonstration of 3-Hz generalized spike-wave discharges with simultaneous staring/decreased responsiveness. Well-trained EEG technologists are adept at testing the child's responsiveness during 3-Hz generalized spike-wave volleys, usually by speaking a string of words during the discharge and asking the child to repeat the words back after the volley is over. In this way, awareness during the spell can be assessed. Although absence seizures are classicly associated with "3-Hz generalized spike-wave discharges", close examination of the discharges often reveals polyspike-wave morphology in many cases. The presence of spike-wave versus polyspike-wave discharges at the same frequency is not felt to have a diagnostic or prognostic impact in the setting of CAE (see Figure 10-44).

The diagnosis of absence seizures can be definitively established by recording the seizures in the EEG laboratory, something that is usually not difficult to do in the untreated child given how frequently the seizures occur. In addition to the 3-Hz spike-wave discharges that are associated with clinical seizures, patients usually have generalized spike-wave fragments that are not clearly associated with clinical change. In sleep, brief generalized polyspike-wave discharges may also appear. Most commonly, the voltage maximum of the generalized spike-wave discharges can be measured in a band across the frontal region, including the F3, F4, and Fz (superior and midline frontal) electrodes and the adjacent F7 and F8 (anterior temporal) electrodes.

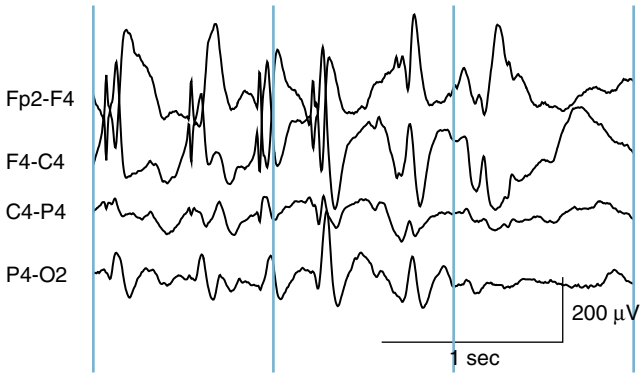


Figure 10-44 This close-up of a discharge associated with an absence seizure shows the multiphasic morphology of the spike component of the discharge. In fact, close analysis of many discharges casually referred to as "generalized spike wave" actually shows that these represent generalized polyspike-wave discharges.

Hyperventilation

Hyperventilation is an effective tool for demonstrating absence seizures. Hyperventilation is carried out during most conventional EEG recordings when it can be feasibly performed, depending on the patient's age, cognitive level, degree of cooperation, and absence of medical contraindications. The patient is asked to overbreathe for 3 to 4 minutes. It can be helpful to have younger children blow a pinwheel. In normal children, hyperventilation typically causes high-voltage hypersynchronous slowing, often dramatic, thought to be a consequence of the cerebral vasoconstriction associated with the lowered CO_2 levels caused by hyperventilation; a dramatic hyperventilation response is less common in adults (see Figure 10-45). In children with a tendency to generalized spike-wave discharges, the discharges are often provoked by the hyperventi lation technique. Among children with active absence seizures, hyperventilation will elicit an absence spell in approximately 80%. In some children, the absence seizures provoked by hyperventilation are more prolonged than those that occur spontaneously. At times, hyperventilation elicits a hypersynchronous response with some definite spike-wave discharges mixed in (see Figure 10-46), but no clinical seizure.

It is not uncommon for patients to become fatigued and to appear to lose alertness from the effort of hyperventilation and the malaise it may cause. It is important to be able to distinguish the "dreamy look" or lack of alertness brought on by the fatigue and discomfort of the hyperventilation exercise from actual clinical absence seizures. Certain clues can help distinguish between the two. First, during a true absence seizure, the patient almost always halts the hyperventilation effort with a noticeable change in the pace of respirations. Second, if the patient is asked to hold the arms outstretched during hyperventilation, a mild but distinct slumping of the arms is seen at the time of the seizures. This is easiest to observe when the patient hyperventilates in the seated position. Finally, and perhaps most important, the ability to repeat words spoken during an absence seizure is reduced or absent.

Generalized spike-wave discharges are often also seen in drowsiness as the patient passes into light sleep. Because responsiveness is impossible to evaluate in a patient who is falling asleep, whether these discharges represent electrographic seizures or electroclinical seizures remains an open question.

Patients and families who are aware that hyperventilation provokes absence seizures often ask whether exercise that causes heavy breathing should be avoided. Exercise is not expected to provoke absence seizure because of the physiologic differences between voluntary hyperventilation and hyperpnea during exercise. During exercise, CO_2 accumulates as a metabolic waste product, and blood CO_2 rises. The body responds to this increased CO_2 level by increasing minute ventilation in an effort to bring CO_2 levels back down to normal. During strenuous exercise with rapid breathing, the CO_2 level does not typically dip significantly below normal. In contrast, the patient who hyperventilates in the EEG laboratory presumably starts with a normal CO_2 level at baseline, and the hyperventilation effort brings the level significantly below normal, potentially provoking an absence seizure if the patient is prone to them.

Juvenile Absence Epilepsy

Although they share much in common, the syndrome of juvenile absence epilepsy (JAE) is distinct from CAE in several ways, beginning with a later age of onset of absence seizures in JAE. There is an important overlap between JAE and juvenile myoclonic epilepsy (described below) in that JAE patients may also experience generalized tonic-clonic seizures. In the JAE syndrome, the absence seizures tend to be less frequent, sometimes only occurring once or twice per week compared with the dozens of episodes per day seen in untreated CAE. The individual absence episodes tend to be of longer duration, with spells up to 20 seconds long not uncommon in the JAE group. The EEG findings in JAE are similar to those of juvenile myoclonic epilepsy, described later.

Epilepsy With Generalized Tonic-Clonic Seizures on Awakening

As the name implies, the syndrome of epilepsy with generalized tonic-clonic seizures on awakening (EGTCSA) consists of generalized tonic-clonic seizures in the period soon after awakening. EGTCSA is considered to be one of the primary generalized epilepsy syndromes. EGTCSA is similar to juvenile myoclonic epilepsy but, in its pure form, only includes generalized tonic-clonic seizures. There is a question as to whether this epilepsy syndrome simply represents the subset of JME patients who only experience generalized tonic-clonic seizures in the morning and who do not happen to have myoclonic seizures or absence seizures. Patients who have similar generalized tonic-clonic seizures that are not necessarily restricted to the period of awakening may resemble EGTCSA in every other aspect apart from the timing of their seizures. The EEG findings in this syndrome are similar to those found in the other primary generalized epilepsies.

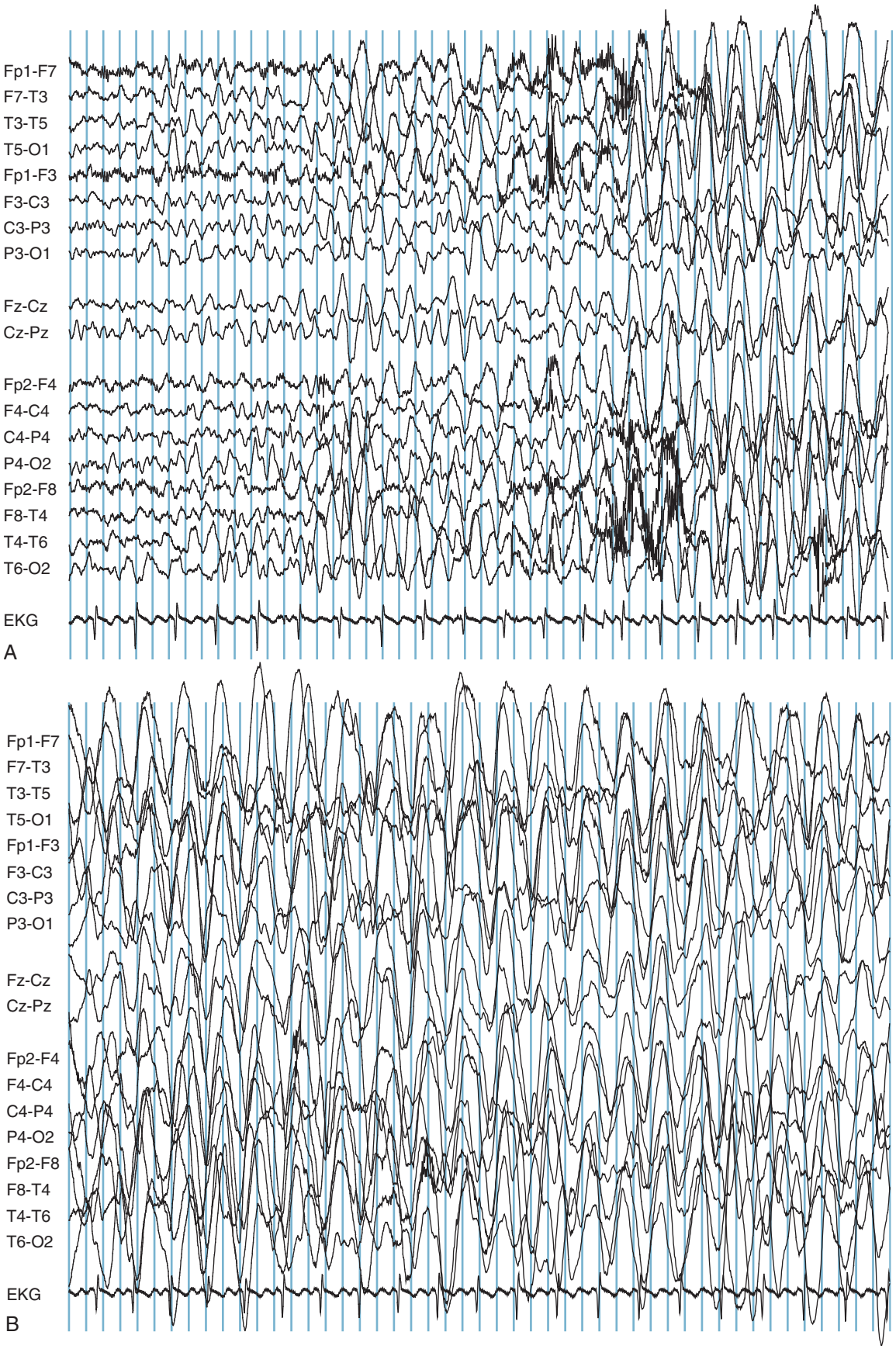


Figure 10-45 A-B The EEG response to hyperventilation can be dramatic. Factors associated with higher voltage hyperventilation responses include greater effort, younger patient age, and lower blood glucose levels. As long as no epileptiform (spike) activity is present, very high-voltage hyperventilation responses should not be considered abnormal.

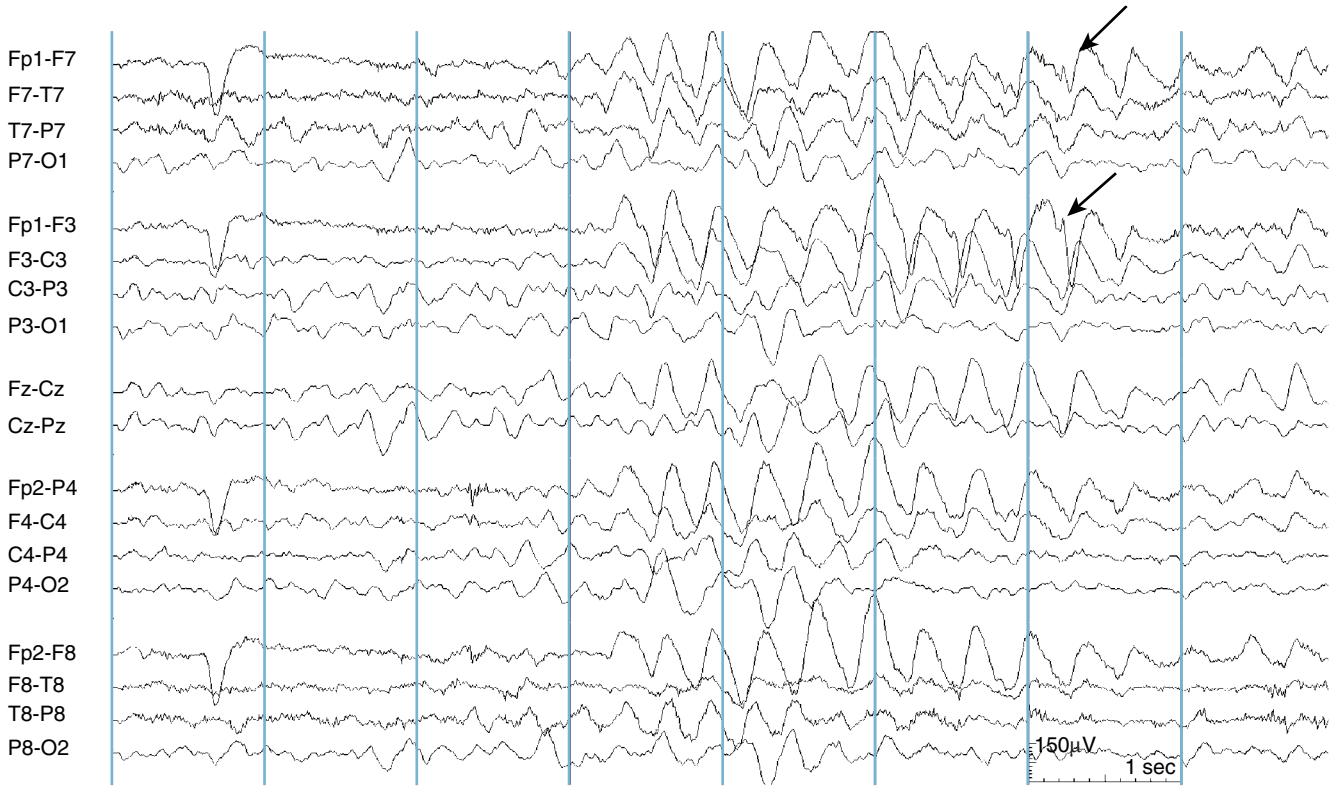


Figure 10-46 Definite spikes intermixed with a hyperventilation response are considered abnormal. A transient is most likely to represent a true epileptic spike when it is time-locked to the slow wave. Peaking or sharpening of the high voltage slow waves themselves during hyperventilation is less likely to be abnormal.

Juvenile Myoclonic Epilepsy

Juvenile myoclonic epilepsy (JME) is an epilepsy with onset in the second decade of life, most commonly during the early teenage years, and is a classic example of an idiopathic generalized epilepsy. The hallmark of this syndrome is the myoclonic seizure; generalized tonic-clonic seizures, and less commonly absence seizures, occur as well. Any subset of these three seizure types may be seen in an individual with JME though, by definition, myoclonic jerks should be present. On average, the myoclonic jerks begin at 12 years and the generalized tonic-clonic seizures begin at 13 years of age. Because the myoclonic jerks may start before the more dramatic generalized tonic-clonic seizures, in some patients these quick jerks may not initially be recognized as seizures and thus may not attract medical attention. Some patients only seek medical attention once a generalized tonic-clonic seizure has occured. For this reason, when younger patients present with generalized seizures, the clinician should specifically ask the patient about the presence of myoclonic jerks even if such a history has not been volunteered. In those patients in whom the diagnosis of JME is made based solely on the presence of myoclonic jerks before the occurrence of a generalized tonic-clonic seizure, effective antiepileptic therapy may prevent the patient from ever experiencing generalized tonic-clonic seizures.

The myoclonic seizures in JME characteristically consist of quick abduction jerks of the upper arms. The jerks have a predilection for the morning hours, and the patient may describe events such as involuntarily flinging a glass of orange juice into the wall because of the jerks. Consciousness appears to be preserved during these very brief episodes. Occasionally, a succession of myoclonic jerks can culminate in a generalized tonic-clonic seizure. This is one of the rare situations in which individuals appear to report observing the onset of a generalized tonic-clonic seizure in themselves. They may remember the initial series of myoclonic jerks but they should not be able to recall the ensuing generalized tonic-clonic seizure; an observer may have a difficult time determining when the myoclonic jerks stopped and the grand mal seizure began. The clearance of myoclonic seizures can be used as a barometer of the effectiveness of drug therapy in JME patients. Approximately one quarter of patients with JME will also manifest absence seizures, which tend to be less frequent but of longer duration than the absence episodes that occur in the context of CAE (similar to those described earlier in JAE).

EEG Findings in JME

The classic EEG findings in JME are *fast spike-wave discharges* and high-voltage polyspikes in sleep. The polyspikes may be either brief or more lengthy, well organized or poorly organized (see Figures 10-47 and 10-48). *Fast spike wave* refers to 4 to 5 Hz generalized spike-wave discharges that resemble classic 3-Hz generalized spike-wave discharges except for their faster firing rate (see Figure 10-49). A minority of patients may also

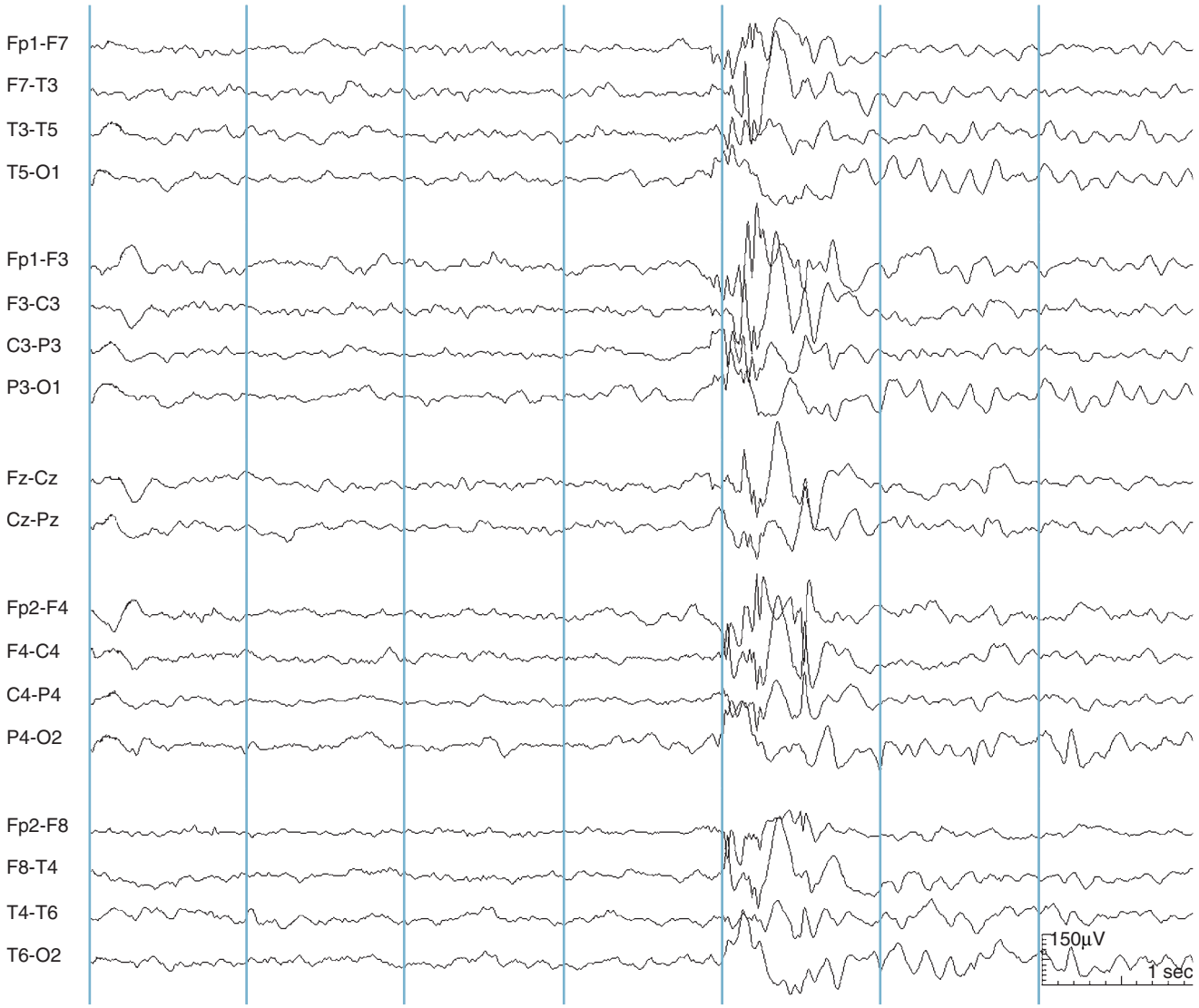


Figure 10-47 A relatively brief polyspike-wave discharge is seen during Stage I sleep in a patient with juvenile myoclonic epilepsy.

exhibit a photoparoxysmal response: spike-wave discharges that are triggered by strobe light stimulation (discussed later). The epileptiform findings of JME are activated both by hyperventilation and by slow-wave sleep; during REM sleep, the discharges are relatively suppressed. A normal background is expected. Unless caused by a recent seizure, the presence of abnormal slowing calls the diagnosis of an idiopathic generalized epilepsy into question. Some patients with JME may have a normal EEG study, but abnormalities are usually discovered with repeated studies. The diagnostic yield of the EEG in JME can be increased by the use of hyperventilation and strobe activation techniques. Recording sleep, which may be facilitated by sleep-depriving the patient before the study, also increases diagnostic yield.

At times, patients with JME may appear to manifest focal discharges. This occurs when asymmetric or fragmentary versions of the generalized discharges are noted (see Figures 10-50 and 10-51). When spike asymmetries are seen in a presumed primary generalized epilepsy patient, the apparent focal discharges should not occur consistently on one side but should appear to be somewhat evenly distributed on both sides. Focal spike-wave discharges that represent fragments of generalized spike-wave discharges should occur at the same locations as the patient's generalized spike-wave discharge maxima, most commonly the superior frontal electrodes, F3 and F4. Therefore, apparently focal discharges that are present at the same maximum points as the generalized spike-wave discharges are still potentially consistent with an idiopathic generalized epilepsy, especially when those apparent focal discharges appear to alternate sides—they should not be reported as separate focal abnormalities.

Strobe Stimulation and the Photoparoxysmal Response

Strobe stimulation, or intermittent photic stimulation, is a routine activation procedure carried out during most EEGs performed in the EEG laboratory. During

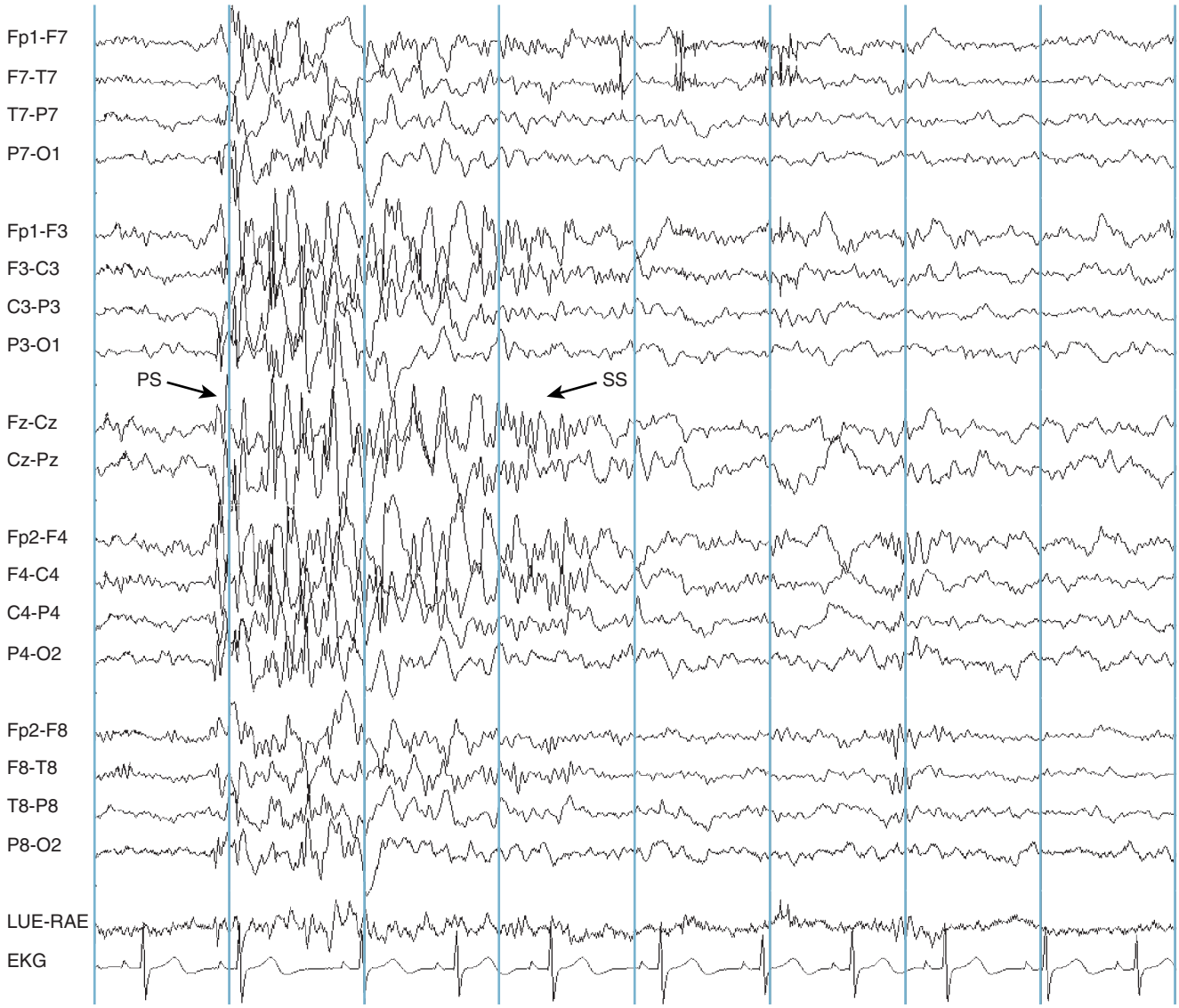


Figure 10-48 More prolonged polyspikes may be seen in patients with primary generalized epilepsy. This polyspike discharge (PS) merges with a sleep spindle (SS). The polyspike portion of the discharge can be distinguished from the spindle on the basis of the respective wave frequencies (14 Hz in the case of the sleep spindle).

the photic stimulation procedure, a strobe light is flashed in front of the patient at varying frequencies, usually between 1 and 35 Hz, for 5 to 10 seconds at each frequency, separated by pauses of similar length. The EEG is then analyzed for a photic driving response (a normal finding) and a photoparoxysmal response (an abnormal finding).

Visual Evoked Potentials and the Photic Driving Response

A visual evoked potential is a positive-polarity wave seen in the occipital area approximately 100 msec after a visual stimulus such as a flash. The photic driving response consists of a *train* of these occipital waves driven by repetitive strobe flashes. Each individual wave of the driving response can be considered a type of visual evoked potential. The photic driving response differs from visual evoked potentials in that it measures the ability of the occipital lobe to "follow" strobe flashes at varying frequencies with repetitive responses (see Figure 10-52). The strobe may be able to entrain occipital waves at low flash frequencies, but the driving response may "drop out" at higher flash frequencies. Occasionally, the driving response is seen at a subharmonic of the strobe frequency, usually half the strobe frequency (see Figure 10-53). The ability to produce driving at very high strobe frequencies, the *high frequency photic response* or *H response* has been reported to be associated with migraine and certain other clinical states, although the usefulness of this sign has been questioned.

A photic driving response can usually be observed in a calm subject, but absence of an identifiable driving response is not considered abnormal. Amplitude asymmetries in the driving response may be seen. Because

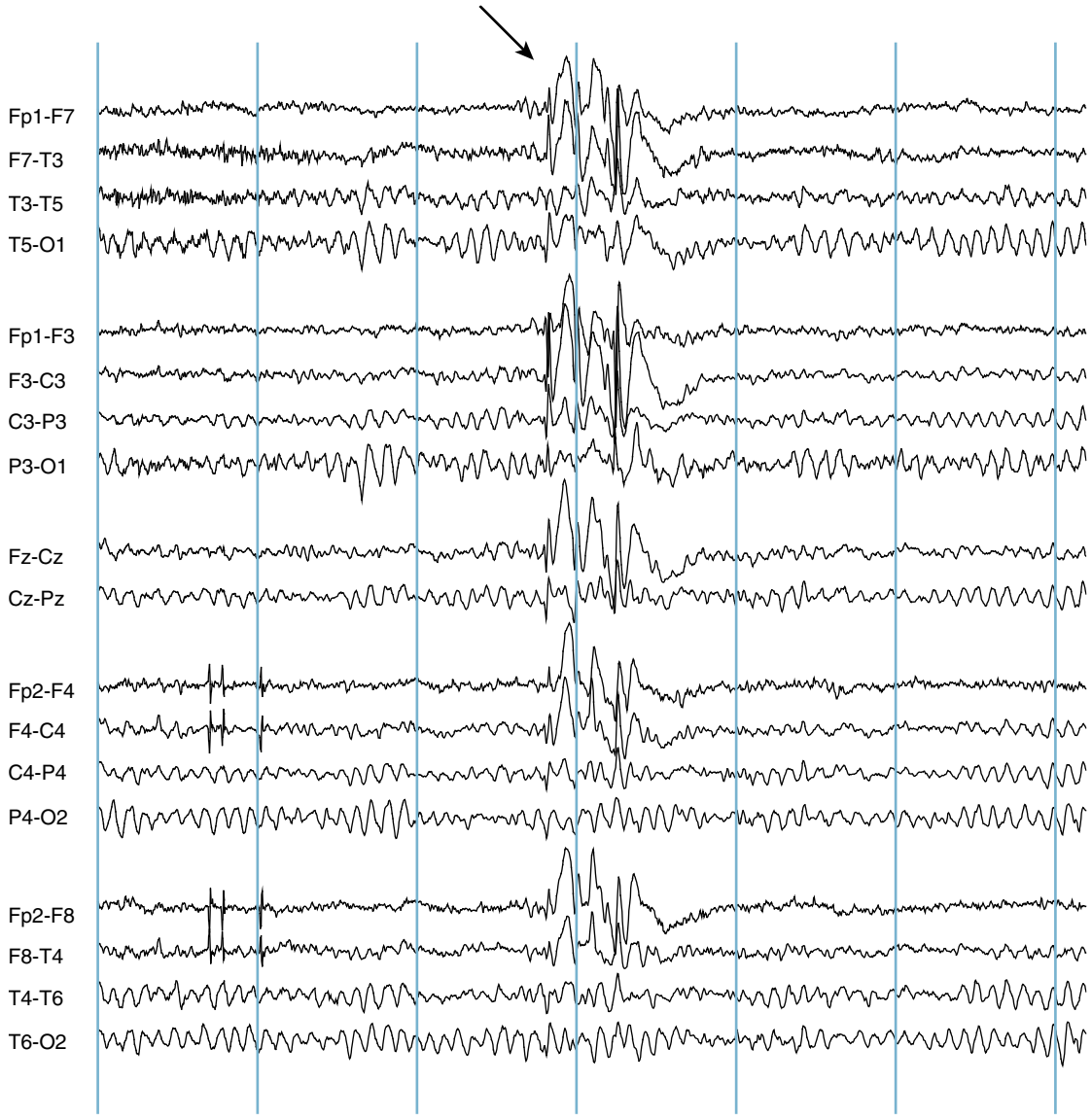


Figure 10-49 A brief, generalized fast spike-wave discharge is seen firing at approximately 4 Hz. Fast spike wave is particularly characteristic of juvenile myoclonic epilepsy and juvenile absence epilepsy syndromes. The spike-like discharges seen in the first second of the tracing in the Fp2-F4, F4-C4, Fp2-F8, and F8-T4 channels represent artifact.

each occipital lobe responds to photic stimulation from the opposite visual hemifield, some observed asymmetries may be related to asymmetric retinal stimulation from off-center strobe placement or eye or head turning by the patient. Although it is true that structural asymmetries of brain may cause an asymmetry of the photic driving response, many neurologically normal individuals may have an inherently asymmetrical driving response. Therefore, isolated asymmetries of the photic driving against the backdrop of an otherwise normal EEG are not considered abnormal.

High-Amplitude Photic Response

Certain disorders have been associated with a particularly high-amplitude photic driving response, particularly at low strobe frequencies. Such high-voltage driving responses are probably the equivalent of abnormally high-voltage visual evoked potentials. The high-amplitude photic response has been described in the early stages of the late infantile form of neuronal ceroid lipofuscinosis (Jansky-Bielschowsky disease), a neurodegenerative disease of infancy. Later in the course of this disease, the photic response is completely lost because of ongoing retinal degeneration and visual loss. A high-amplitude photic response may also occasionally be present in other neurodegenerative conditions.

The Photoparoxysmal Response

The phenomenon of intermittent photic stimulation eliciting generalized spike-wave discharges is referred to as a photoparoxysmal response (PPR; see Figure 10-52). Strobe stimulation can provoke a range of epileptiform activity in the EEG. Different features of the response

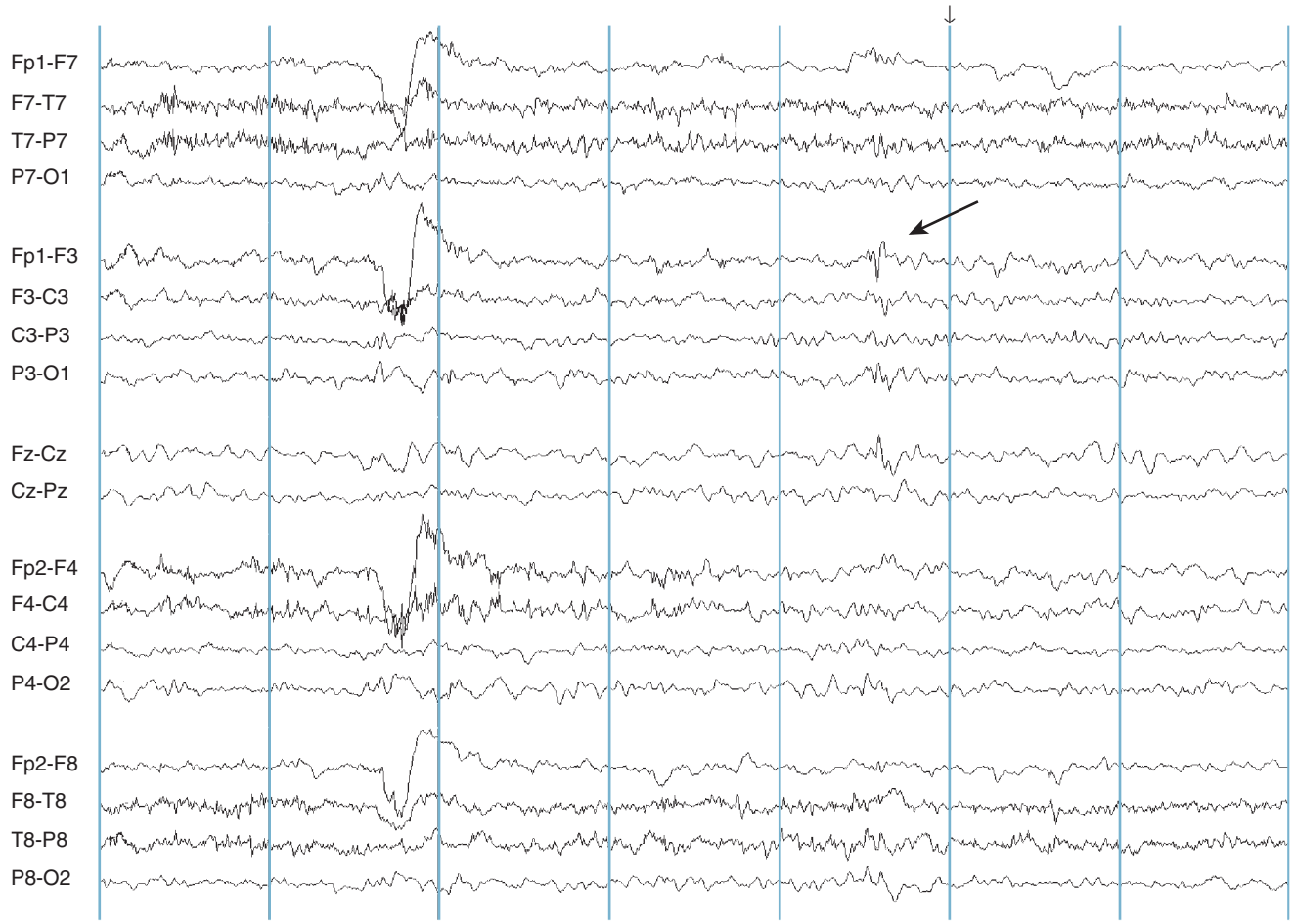


Figure 10-50 A single apparent focal spike-wave discharge is seen (arrow), maximum in the left superior frontal area. When seen as an isolated event, this spike-wave complex suggests a focal discharge. When seen in the context of the generalized spike-wave discharges seen elsewhere in the tracing of this patient with absence epilepsy, it is recognized as a fragment of a generalized discharge without focal significance (see Figure 10-49).

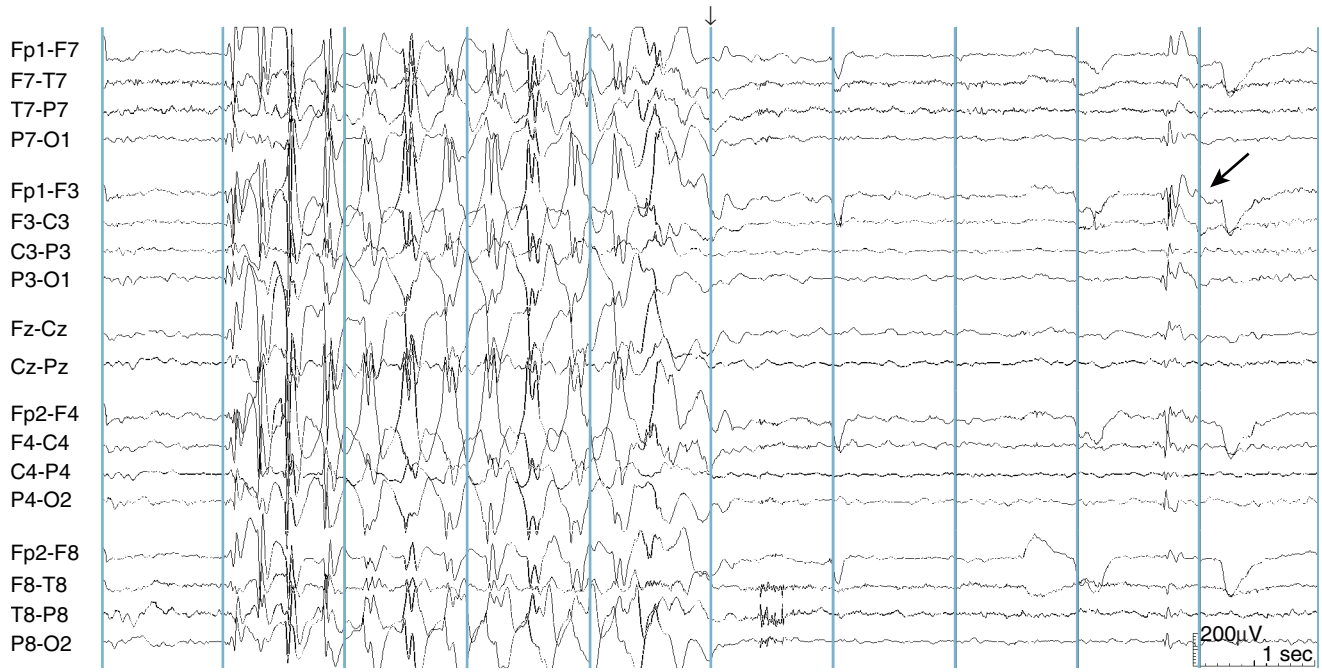


Figure 10-51 A high-voltage generalized spike-wave discharge is seen on a different page of the same EEG that was shown in the previous figure. Of note, later in this page, a spike-wave discharge is seen (arrow), again representing a fragment of the generalized discharge. When several such fragmentary discharges are seen, approximately the same number is expected on the left as on the right.

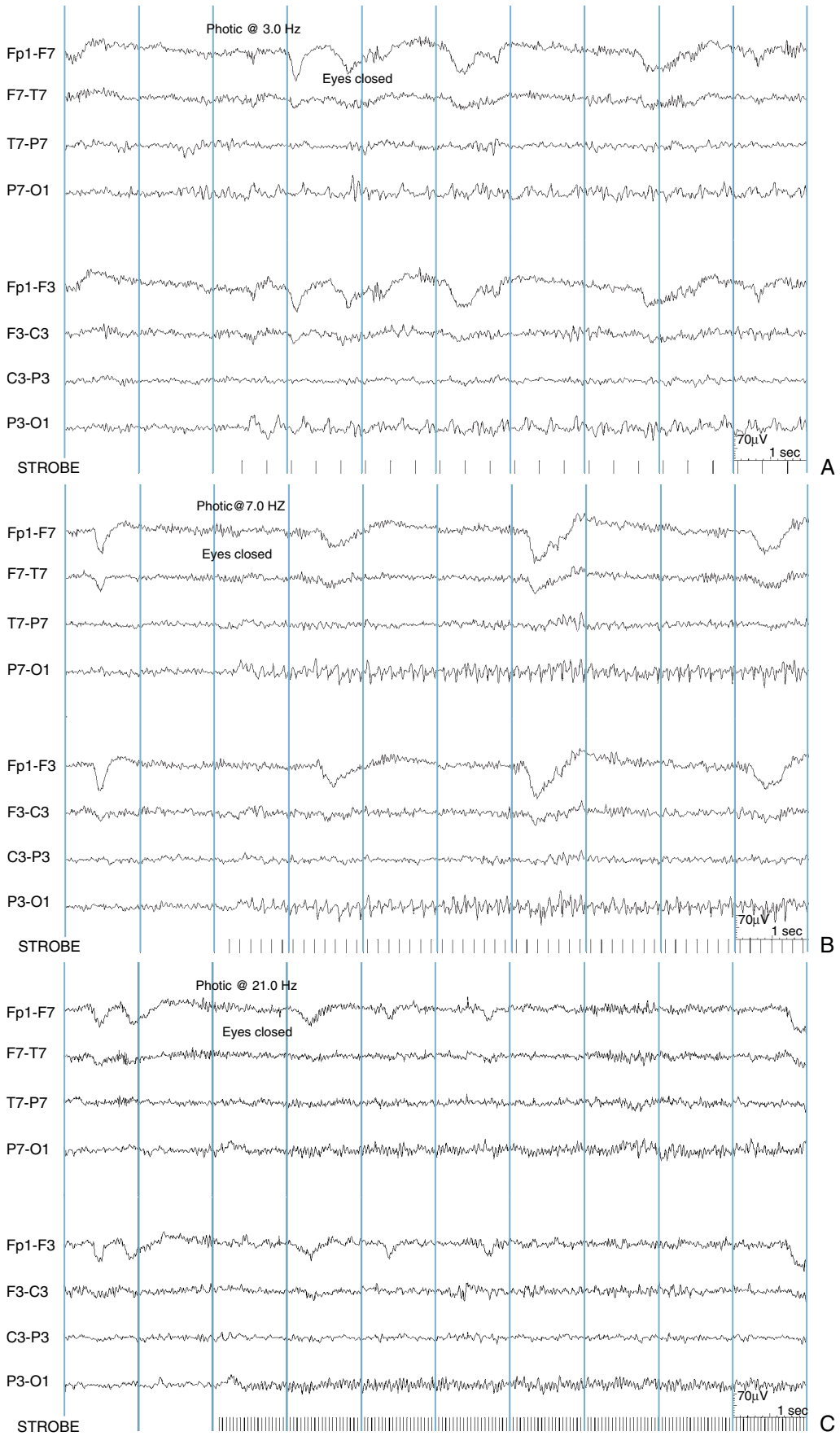


Figure 10-52 The strobe flashes of photic stimulation are denoted by each vertical line in the bottom strobe channel. A photic driving response can be seen in the occipital channels (P7-O1 and P3-O1) in the form of low-voltage, repetitive rhythmic waves that closely follow the strobe flashes. A similar response was also seen in the right occipital area but is not shown.

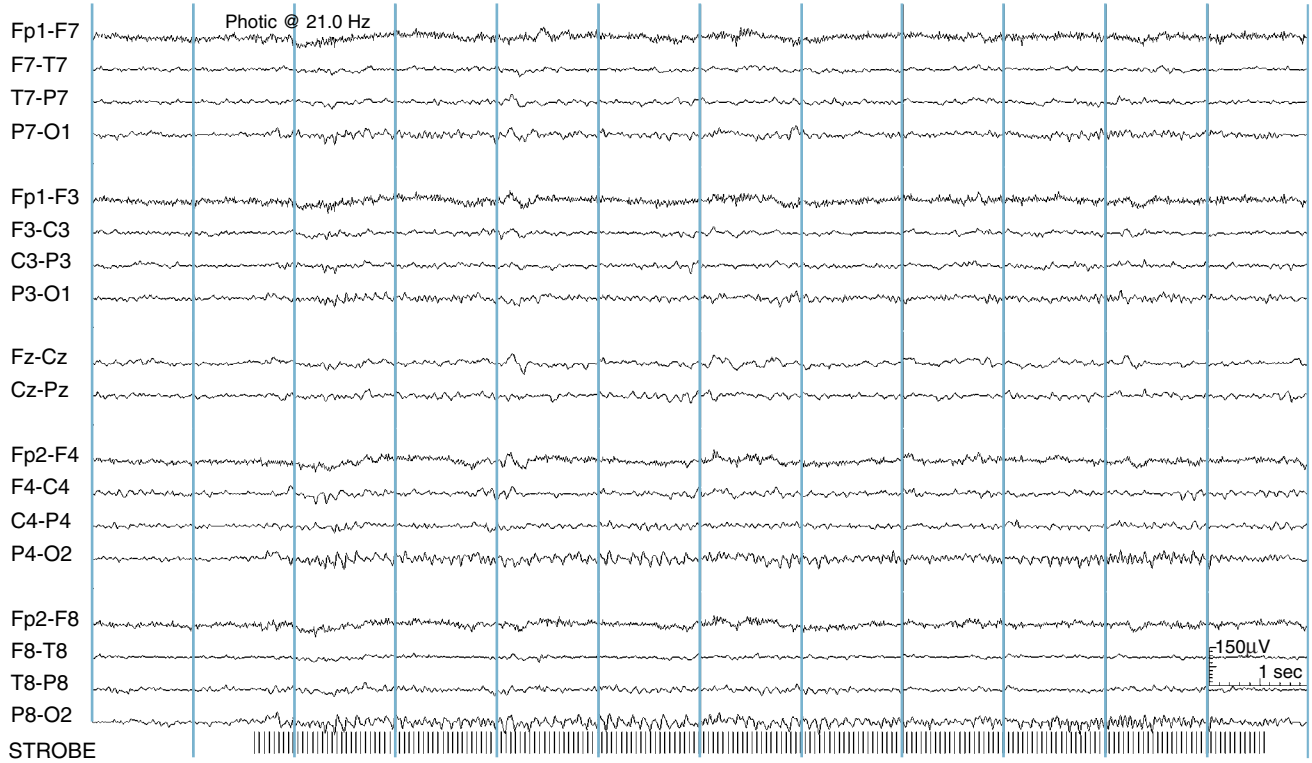


Figure 10-53 The photic driving response occasionally follows the strobe at a subharmonic (half) frequency. In this example, strobe flashes at 21 Hz generate a 10.5-Hz driving response.

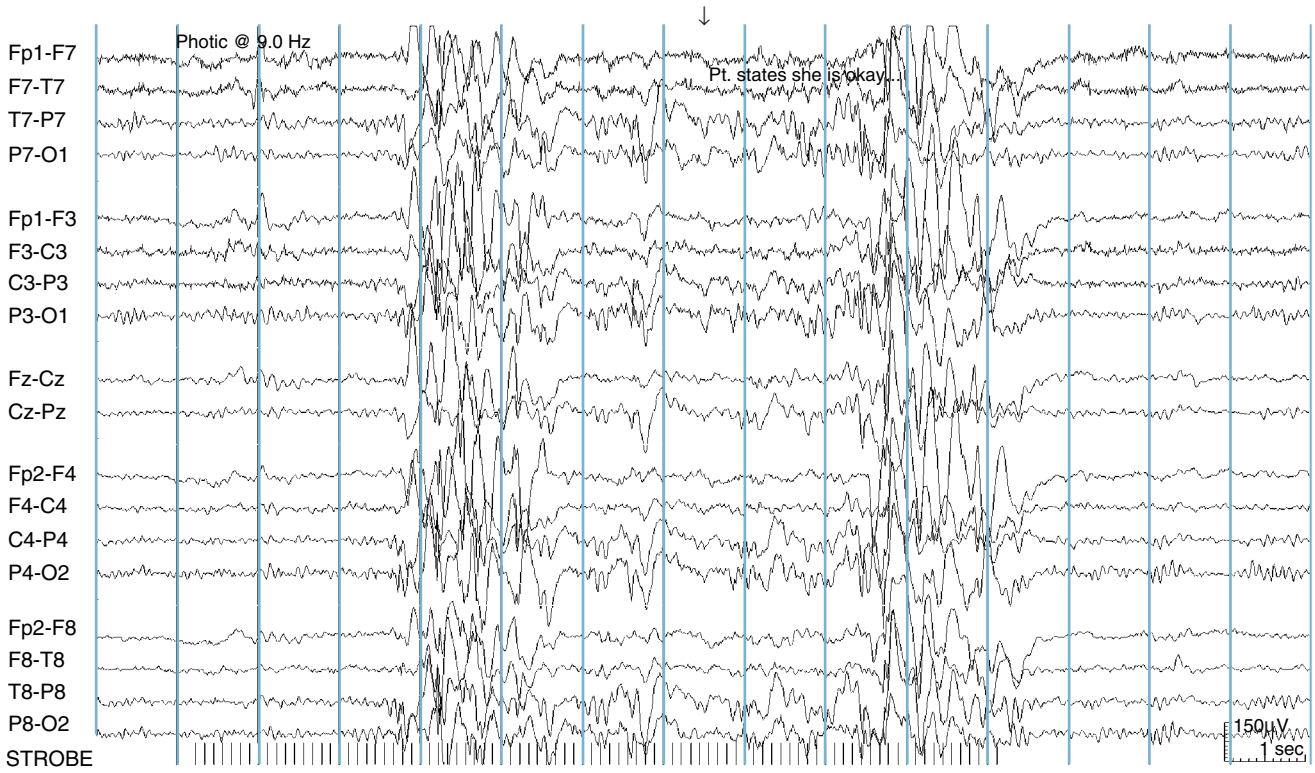


Figure 10-54 Two generalized spike-wave bursts are elicited by strobe stimulation. The generalized (rather than primarily posterior) distribution of the discharge and its high-voltage increase the chance that this photoparoxysmal response is associated with epilepsy.

are felt to increase or decrease the chances that the elicited activity is abnormal (i.e., associated with seizures). Generalized or frontal-predominant spike-wave discharges are felt to be more abnormal than posterior spike-wave discharges. Higher voltage discharges are felt to be more abnormal than lower voltage discharges. Finally, spike-wave discharges that outlast the strobe stimulus are felt by some to be more significant than those that end with the stimulus. Some of these, parameters, especially the distinction between posterior responses and responses in other locations, are designed to distinguish atypically exuberant (but non-epileptic) driving responses from true epileptiform bursts.

In patients with seizures, the PPR is most often seen in the primary generalized epilepsies such as JME and JAE. When present, the PPR is most commonly seen during the adolescent and teenage years and tends to become less prominent with age. Presence of a PPR is not synonymous with photosensitive epilepsy. Photosensitive epilepsy refers to actual seizures (as opposed to spike-wave discharges) elicited by flashing lights (see Figure 10-55). Examples of light stimuli encountered during daily life include flashing strobe lights, driving down a sun-dappled street, or flashing lights from video screens. Patients with photosensitive epilepsy often manifest a PPR on the EEG. However, of all patients with a PPR on the EEG (such as JME patients), most do not have a history of photic-induced seizures. The PPR may also be seen as an EEG trait in the siblings of patients with primary generalized epilepsy. A PPR is occasionally encountered in patients with focal epilepsies.

Generalized Epilepsy With Febrile Seizures "PLUS": GEFS+

The GEFS+ syndrome is a familial genetic syndrome in which different family members may have widely varying clinical presentations. Perhaps the most remarkable aspect of the GEFS+ syndrome is that the same single gene mutation can cause such a wide array of seizure types and seizure syndromes in different members of the same kindred. Individuals with the same GEFS+ mutation may manifest several combinations of (usually) generalized seizure types including febrile seizures, absence seizures, myoclonic seizures, generalized tonic-clonic seizures, and, in the most severe phenotype, seizures indistinguishable from those that occur in myoclonic-astatic epilepsy (Doose syndrome, discussed later). In the general population, febrile seizures usually subside by the age of 6 years. Individuals with the GEFS+ syndrome may have febrile seizures that continue past this age, hence the term *febrile seizures "plus."* At the time of this writing, more than five genetic mutations associated with sodium channel or GABA receptor errors have been identified that can cause the various phenotypes of the GEFS+ syndrome in different families. In a single family, multiple carriers of a given mutation can have strikingly different phenotypes.

It is surprising to see that a single gene can cause seizure syndromes as apparently disparate as a febrile seizure syndrome and the syndrome of myoclonic-static seizures (discussed later). Indeed, some obligate carriers of the GEFS+ gene are asymptomatic. It is hypothesized that the dramatic variation seen in the clinical expression in carriers of the same mutation is related to other modifying genes in affected individuals. Patients with this syndrome may manifest seizure disorders that are indistinguishable from JME, CAE, or pure generalized tonic-clonic seizures. Surprisingly, occasional individuals with this genotype may also manifest focal seizures. EEG findings in individuals with GEFS+ generally resemble the EEG patterns expected in the particular seizure syndrome the patient manifests as described earlier; there is no GEFS+-specific EEG pattern.

OTHER MYOCLONIC EPILEPSY SYNDROMES

Lennox-Gastaut Syndrome

Lennox-Gastaut syndrome (LGS) consists of the triad of mixed seizures, a slow spike-wave pattern on EEG, and cognitive dysfunction beginning in early childhood. Seizures seen in LGS include atypical absence seizures, atonic seizures, tonic seizures (occurring particularly during sleep), and myoclonic seizures. Some authors classify LGS as a myoclonic epilepsy, although myoclonic seizures are not an obligatory part of the syndrome, and others classify it as a symptomatic generalized epilepsy. Although LGS includes many seizure patterns that appear to be generalized, in many patients the EEG also shows significant focal or multifocal abnormalities. Despite its possible classification as a symptomatic generalized epilepsy, seizures of clearly focal onset may occur among the mixed seizures of LGS. Assignment of LGS to the group of generalized epilepsies may underemphasize the importance of focal or multifocal etiologies found in many patients.

The seizures of LGS are often refractory to treatment. In most, cognitive problems are already evident at the time of onset of the LGS syndrome, although a minority of children have an apparent normal neurodevelopmental status at the time the seizures start and subsequently lose ground as the seizures continue. In some patients, the LGS begins against the backdrop of a previously recognized encephalopathy, such as a pre-existing history of stroke, meningitis, cerebral dysgenesis, cerebral palsy, or West syndrome. In others with LGS, the syndrome appears to occur de novo.

Seizure Types and EEG Findings in Lennox-Gastaut Syndrome

Patients with LGS have a mixture of seizure types. The atypical absence seizures of LGS are associated with a simultaneous slow spike-wave pattern on the EEG that usually has a diffuse distribution. With atypical absence, neither the outwardly apparent staring spell nor the slow spike-wave pattern seen on EEG necessarily shows a sharply demarcated onset or termination. Making the

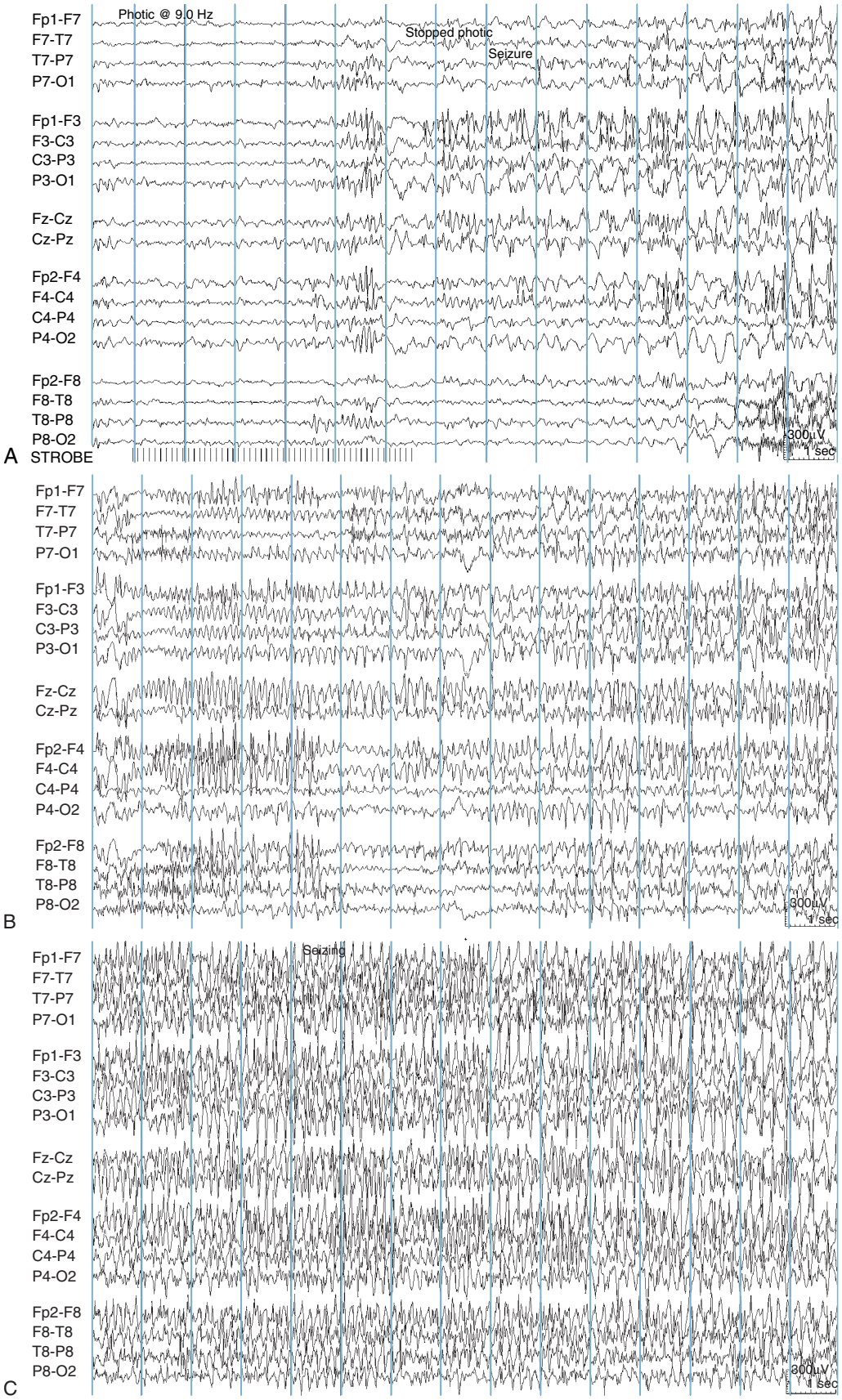


Figure 10-55 A generalized seizure is elicited by strobe stimulation. The phenomenon of an actual clinical seizure triggered by strobe flashes represents a true photosensitive epilepsy.

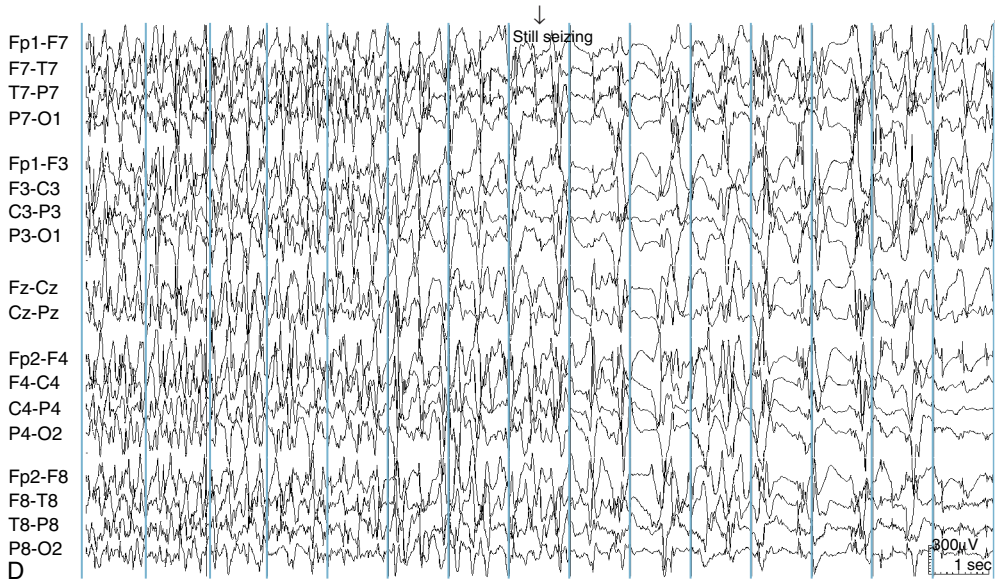


Figure 10-55, cont'd In contrast, many patients with a photoparoxysmal response as seen in Figure 10-54 may never actually have a clinical seizure triggered by flashing lights.

situation yet more complicated, slow spike-wave discharges are also frequently seen as interictal abnormalities in LGS. Therefore, when a run of slow spike-wave discharges is seen on the EEG, it is generally not possible to say whether the discharges represent an electroclinical seizure or an interictal abnormality without concurrent evidence of loss of awareness or responsiveness in the patient.

The atonic seizures in LGS may occur in their mildest form as a brief bobbing of the head or slumping of the shoulders. More severe versions may occur, such as dramatic "head drops" in which the face may fall forward and hit a table in front of the patient. The most severe version is the drop attack in which the patient falls to the ground. Patients with "drop seizures" may need to wear a helmet for head protection. Multiple EEG correlates are seen to drop seizures or atonic seizures (discussed earlier). Tonic seizures may occur at any time of the day, but the tonic seizures of LGS occur most characteristically during sleep and are typically associated with brief runs of generalized rapid spikes (see Figure 10-56).

Not all seizures that cause a head drop or a fall necessarily belong to the category of atonic seizures as seizures with tonic or myoclonic components may also culminate in a fall. Careful studies with the use of EMG electrodes have documented that many such "drop seizures," instead of being caused by a loss of tone, are actually associated with an initial tonic muscle contraction. Some prefer the more general term *astatic seizure* for such episodes (literally meaning the loss of the ability to stand—*astasia*), which can properly be used for any seizure type in which loss of posture is the primary feature.

The interictal EEG in LGS typically consists of generalized and/or focal slow spike-wave or polyspike-wave discharges (at 1–2.5 Hz) that are sometimes activated by sleep, a slow background, and brief bursts of rapid spikes in sleep. Many patients also manifest multifocal spikes or sharp waves.

Epilepsy With Myoclonic-Astatic Seizures or Doose Syndrome

In 1970, Doose et al. described an epilepsy syndrome with myoclonic seizures and falling spells that appeared to be distinct from LGS. Whereas children with LGS usually appeared to have a symptomatic epilepsy, those with this new syndrome had no clear etiology for their seizures but did have a strong family history of epilepsy. Therefore, a genetic etiology was suspected.

The most characteristic seizure type seen in myoclonic-astatic epilepsy is the myoclonic-astatic seizure which consists of one or more brief myoclonic jerks followed by a loss of posture, often culminating in a fall. There is brief loss of consciousness during the attacks followed by quick recovery. Seizure onset is usually between 1½ and 5 years of age. A large variety of other generalized seizure types also occur including generalized tonic-clonic seizures, absence seizures, and tonic seizures. Response to treatment and prognosis vary widely in this group, with some patients fairly responsive to treatment but others refractory to therapy and with a downhill developmental course.

EEG Findings in Myoclonic-Astatic Epilepsy

The EEG may be normal at onset. Often, the background shows slowing in the 4- to 7-Hz range. In sleep, slow spike-wave discharges can be seen, although these are not usually as prominent as those seen in LGS. Both the myoclonic and myoclonic-astatic seizures are typically accompanied by a brief burst of 2- to 4-Hz generalized spike-wave or polyspike-wave discharges, often one to three discharges in quick succession.

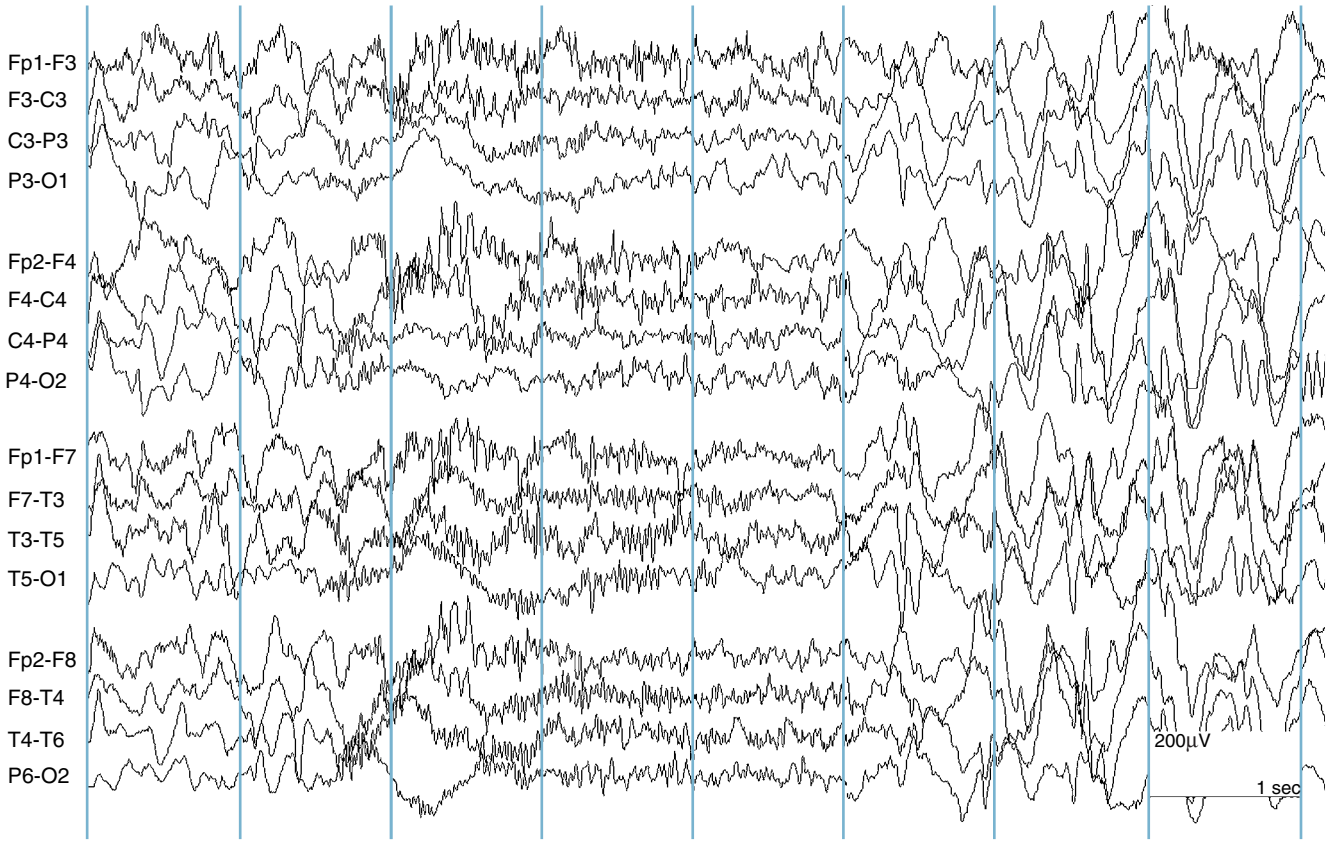


Figure 10-56 A tonic seizure occurs in sleep in a patient with the Lennox-Gastaut syndrome. The background pattern of slow spike-wave discharges is interrupted by a brief train of generalized rapid spikes associated with whole body tonic stiffening.

Benign Myoclonic Epilepsy of Infancy

Benign myoclonic epilepsy of infancy (BMEI) is a syndrome consisting mainly of myoclonic seizures typically presenting between the ages of 1 and 5 years in an otherwise normal child. The ictal EEG shows a brief series of generalized polyspikes driving rapidly repetitive myoclonic jerks which are usually relatively mild and occur only a few times a day. The syndrome usually remits spontaneously, but learning and developmental difficulties may be seen more often in affected individuals compared with the general population. Strobe stimulation may elicit the jerks in a subset of infants. Because of the occasional occurrence of generalized tonic-clonic seizures in children with this syndrome, benign myoclonic epilepsy of infancy may exist on a continuum with other primary generalized epilepsy syndromes.

Reflex-Induced Myoclonic Epilepsy

Reflex-induced myoclonic epilepsy may represent a subset of benign myoclonic epilepsy of infancy in which the myoclonic jerks are elicited by sound or touch. In certain infants, auditory or tactile stimulation such as a loud sound, brushing the cheek, or patting the back may elicit a train of spike-wave discharges associated with a brief run of rapid myoclonic jerks (see Figure 10-57). Reflex-induced myoclonic epilepsy, too, is generally expected to remit during childhood.

Severe Myoclonic Epilepsy of Infancy (Dravet Syndrome)

Severe myoclonic epilepsy of infancy (SMEI) is a childhood-onset epilepsy that is refractory to treatment and associated with an ongoing neurological deterioration that may be due to the occurrence of seizures themselves (epileptic encephalopathy). The typical presentation begins with repeated, prolonged febrile seizures during the first year of life. Despite the original name of the syndrome, the presence of myoclonic seizures is not invariable. For that reason, the eponym *Dravet syndrome* is increasingly used. Despite its relationship to the generalized epilepsies, focal seizures that can arise from either hemisphere are frequent, often with secondary generalization as was seen in Figure 10-11. Tonic seizures are uncommon.

Many cases of SMEI are caused by mutations in the SCN1A gene, the same gene that has been implicated in some GEFS+ kindreds. Borderline versions of SMEI are seen and the syndrome may lie on the severe end of the spectrum of primary generalized epilepsies.

Early in the course, the EEG may be normal. Later, high-voltage spike-wave and polyspike-wave complexes appear, sometimes generalized but in some examples surprisingly focal and concentrated over one hemisphere (see Figure 10-58). The discharges are activated by sleep and may manifest photosensitivity. The EEG pattern in SMEI is variable and not highly characteristic.

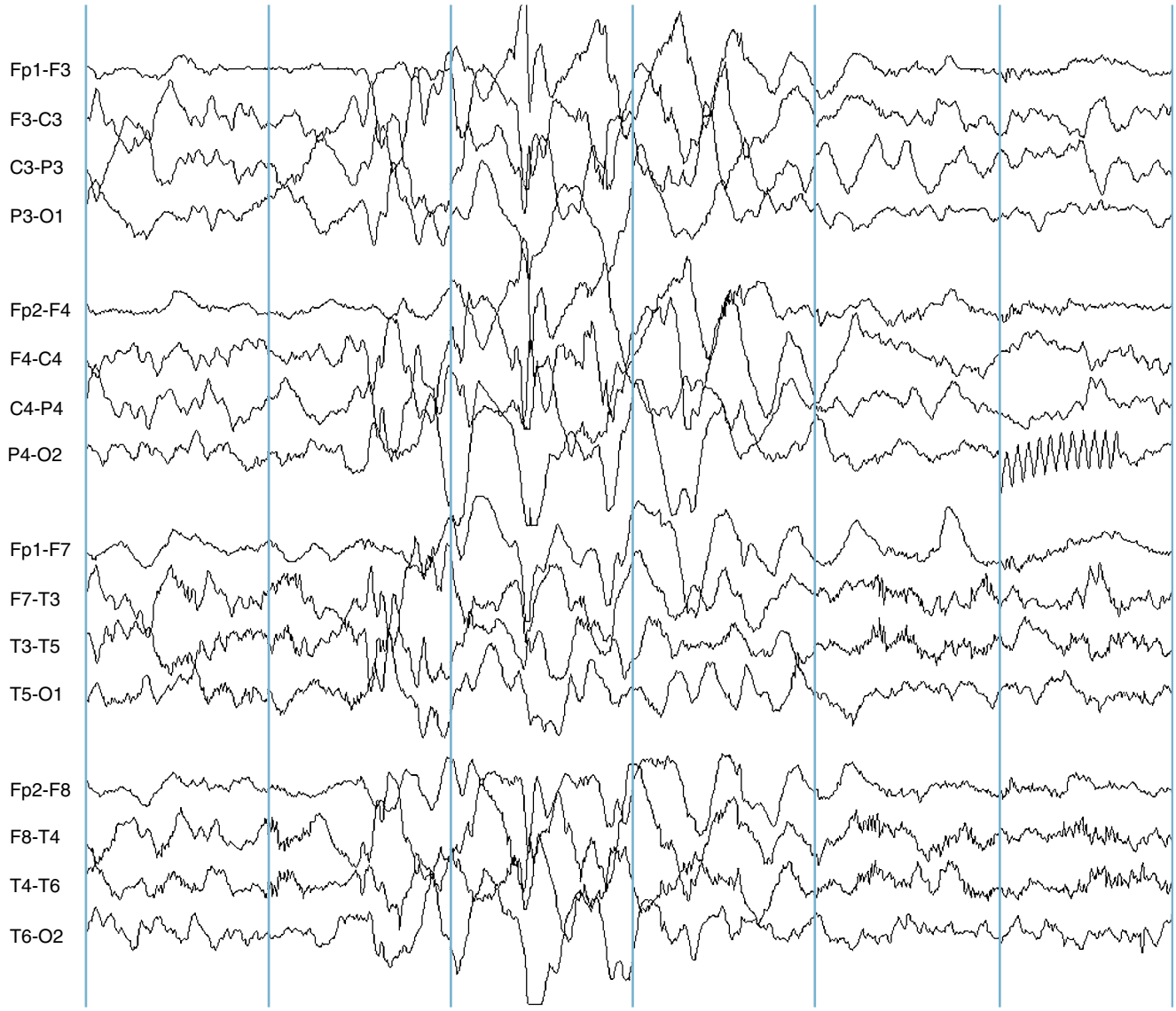


Figure 10-57 A brief, generalized spike-wave discharge is elicited by patting this toddler with reflex-induced myoclonic epilepsy on the back. The artifact in the P4-O2 channel on the last second of the page is an event marker made by the child's caretakers at the time of a clinical event.

The relationship of the seizures to fever, especially early on, is a key diagnostic feature.

Epilepsy With Continuous Spike Wave of Slow Sleep and the Landau-Kleffner Syndrome

Epilepsy With Continuous Spike Wave of Slow Sleep

Continuous spike wave of slow sleep (CSWS) is a relatively rare epileptic encephalopathy associated with neuropsychological impairment and a variety of possible seizure types, both generalized and focal. It shares many clinical features with Landau-Kleffner syndrome, described next. CSWS is associated with a distinctive EEG pattern during slow-wave sleep termed *electrical status epilepticus of sleep* (ESES). The term CSWS is used to refer to the clinical syndrome, whereas ESES refers specifically to the EEG pattern. The ESES pattern consists of continuous, rhythmic 1.5- to 2.5-Hz spike-wave discharges, often with a diffuse field but occasionally unilateral, that appear in non-REM sleep. The essence of this sleep-activated EEG pattern is the appearance of spike-wave discharges so rhythmic that they could potentially be mistaken for an electrographic seizure (see Figure 10-59). Despite the name of the syndrome, which seems to imply that the pattern should only be present during the sleep stages with slow waves, Stages III and IV, the ESES pattern appears with onset of Stage I or early in Stage II sleep. Therefore, ESES is essentially seen in the non-REM sleep stages. It probably is not necessary to insist on recording Stage III and IV sleep to exclude the presence of the ESES pattern—indications of the ESES pattern should be present by onset of Stage II sleep. In REM sleep, the continuity of the discharges is significantly disrupted. Scattered, focal

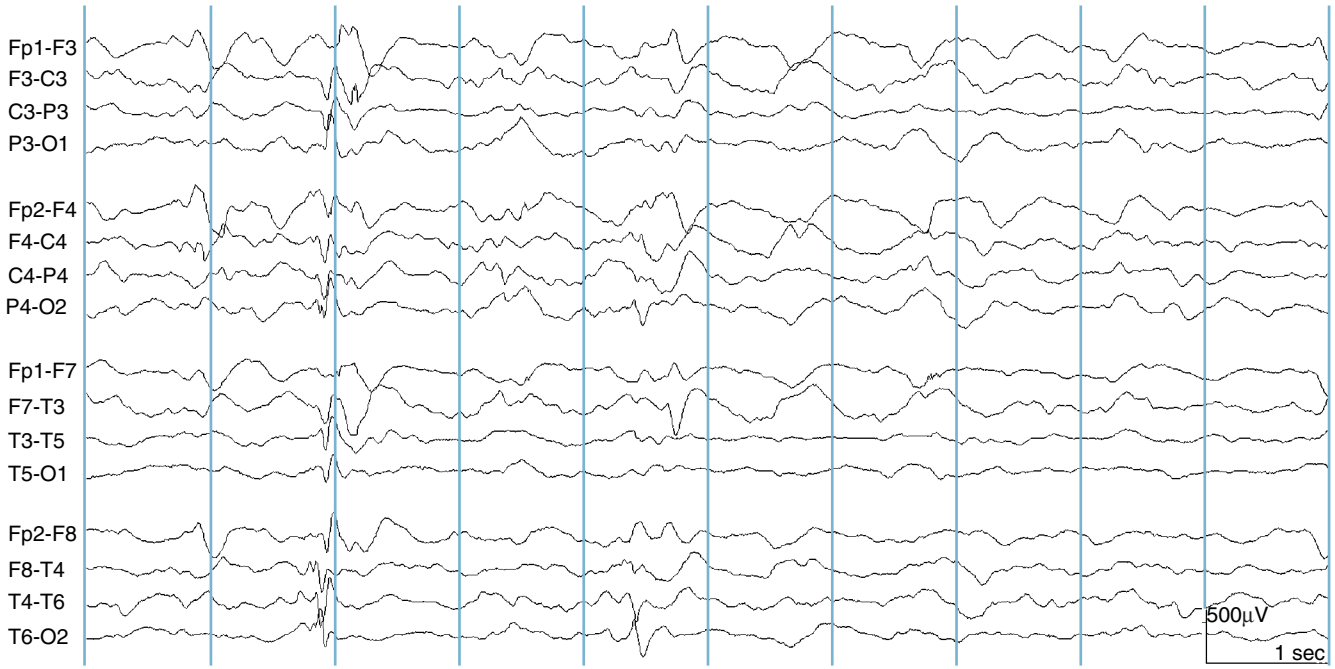


Figure 10-58 This child with severe myoclonic epilepsy of infancy (SMEI) has both generalized and focal discharges. EEG findings vary widely among patients with SMEI.

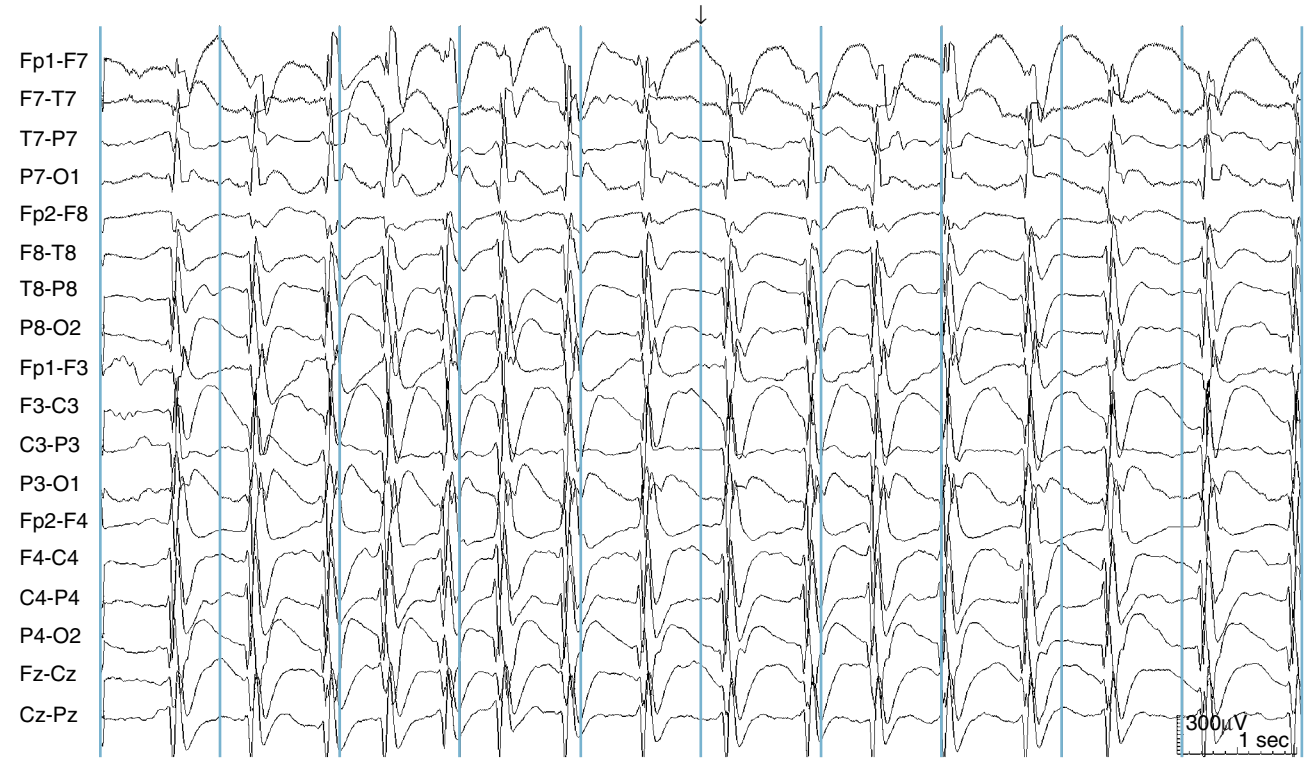


Figure 10-59 This highly rhythmic generalized discharge mimics a seizure discharge; however, it is present exclusively during non–rapid eye movement (REM) sleep and dissipates during REM sleep and on awakening. The ESES pattern consists not only of spikes activated by sleep but of a pattern so rhythmic and continuous that it could be mistaken for a seizure discharge.

discharges may be seen in REM sleep, similar in density to what is seen during wakefulness.

The patterns of clinical seizures seen in this syndrome range from rare or no seizures to seizures that occur several times per week. A wide variety of seizure types is seen, including nocturnal seizures, either hemiclonic or apparent generalized tonic-clonic, typical absence seizures or, more rarely, atypical absences that may also be associated with falls. Tonic seizures as are seen in LGS generally do not occur. Although epilepsy with CSWS is generally considered an idiopathic syndrome with no causative lesion expected on the MRI, in some children, a clinical picture otherwise similar to idiopathic CSWS may be seen as a symptomatic syndrome related to preexisting lesional abnormalities. The question of whether the classification should separate such symptomatic cases from the main CSWS group has yet to be resolved.

By what may seem to be an arbitrary definition, the ESES pattern should be visible during 85% or more of non-REM sleep. The term *electrical status epilepticus* reminds us that the pattern should have sufficient rhythmicity as to mimic a seizure discharge. The term ESES does not apply to patterns in which there may be a very high density of interictal activity during sleep but without the rhythmicity seen in seizure discharges. When making the diagnosis of ESES, the reader should have the impression that the discharge is so rhythmic that it could actually represent electrical seizure activity but that its duration, lack of evolution, and disappearance in REM sleep make this unlikely. Whether EEG patterns with a spike-wave density significantly below 85% or that manifest less rhythmicity and continuity should be included with ESES is controversial in some circles. In straightforward cases of ESES, it is visually obvious that the spike-wave pattern is present for more than 85% of the non-REM tracing, and it is usually unnecessary to make a formal count of the percentage of involved seconds.

Epilepsy with CSWS is associated with a global cognitive impairment which may include the behavioral, language, and motor realms. A variety of patterns of cognitive deficits may be seen. The presence of the ESES pattern appears to be age-dependent, even in those who have organic lesions on imaging. The continuous spike-wave pattern tends to "burn out" by the early teenage years independent of treatment. In some individuals, the EEG may even normalize. Long-term outcome may depend on a variety of factors, particularly the underlying etiology. Many children improve significantly after CSWS has run its course, although some amount of residual disability is the rule. The ESES pattern may be resistant to treatment; however, high-dose diazepam therapy can result in periods of remission and clinical improvement of varying duration in some (De Negri et al., 1995).

Acquired Epileptic Aphasia or the Landau-Kleffner Syndrome

The Landau-Kleffner syndrome (LKS) or acquired epileptic aphasia is a childhood epilepsy syndrome, which probably exists in the middle of a spectrum that includes benign rolandic epilepsy on one end and epilepsy with CSWS on the other. In children with LKS, the language disturbance predominates. In contrast to the majority of language disturbances observed in childhood in which expressive language lags behind the ability to comprehend, in LKS, difficulties with language comprehension are more prominent than expressive difficulties.

The most characteristic pattern is of a child who develops language normally and then acquires an aphasia that is most pronounced in the receptive realm. Concurrently or soon after, an EEG abnormality is noted over one or both temporal lobes, strongly activated by sleep. Behavior and attention may also be adversely affected. Children with LKS may also have seizures, but these are usually sporadic and few in number. In rare instances, LKS may begin before significant language has developed, and the "acquired" nature of the syndrome may not be evident. Such children may appear to present as having pure language delay. How often LKS plays a role in the language and behavioral regression seen in children with autism has been a matter of some debate. It does not appear that LKS explains more than a very small percentage of the larger population of children with autistic disorders.

The interictal EEG in LKS may show focal discharges, often in the centrotemporal areas or the posterior quadrants and may also occasionally show generalized spike-wave discharges (see Figure 10-60). In a minority, the discharges may have a frontal localization. In some, the waking EEG is normal. At some point in the disorder, not always at onset, bilateral generalized spike-wave activity is seen in a pattern consistent with ESES with more than 85% of the slow-wave sleep tracing involved with continuous spike-wave activity. This ESES pattern, when present, is essentially indistinguishable from the patterns that can be seen in epilepsy with CSWS, suggesting the possibility that LKS actually represents a subset of CSWS. In LKS, more unilateral or focal patterns of persistent spike and wave are more common compared with the CSWS syndrome, in which discharges tend to be more bilateral and diffuse. Clinically, deficits in LKS are more concentrated in the language sphere whereas the deficits seen in CSWS tend to be more global.

Because the discharges seen in LKS often show voltage maxima in the centrotemporal areas, a relationship between this syndrome and benign rolandic epilepsy has been proposed, although not yet proved. In fact, some children with benign rolandic epilepsy show so much sleep activation on the EEG that they may strongly resemble, at least from the EEG point of view, the EEG patterns seen in LKS. In children with LKS, initial EEGs are often interpreted as being consistent with benign rolandic epilepsy because of the presence of centrotemporal spikes. The fact that some children with benign rolandic epilepsy may manifest a developmental language abnormality also supports the possibility that the syndromes are related. Although occasional cases of an EEG pattern consistent with LKS have been reported after acquired brain lesions, in general, those with LKS have a normal MRI and, apart from the EEG, other diagnostic testing is normal.

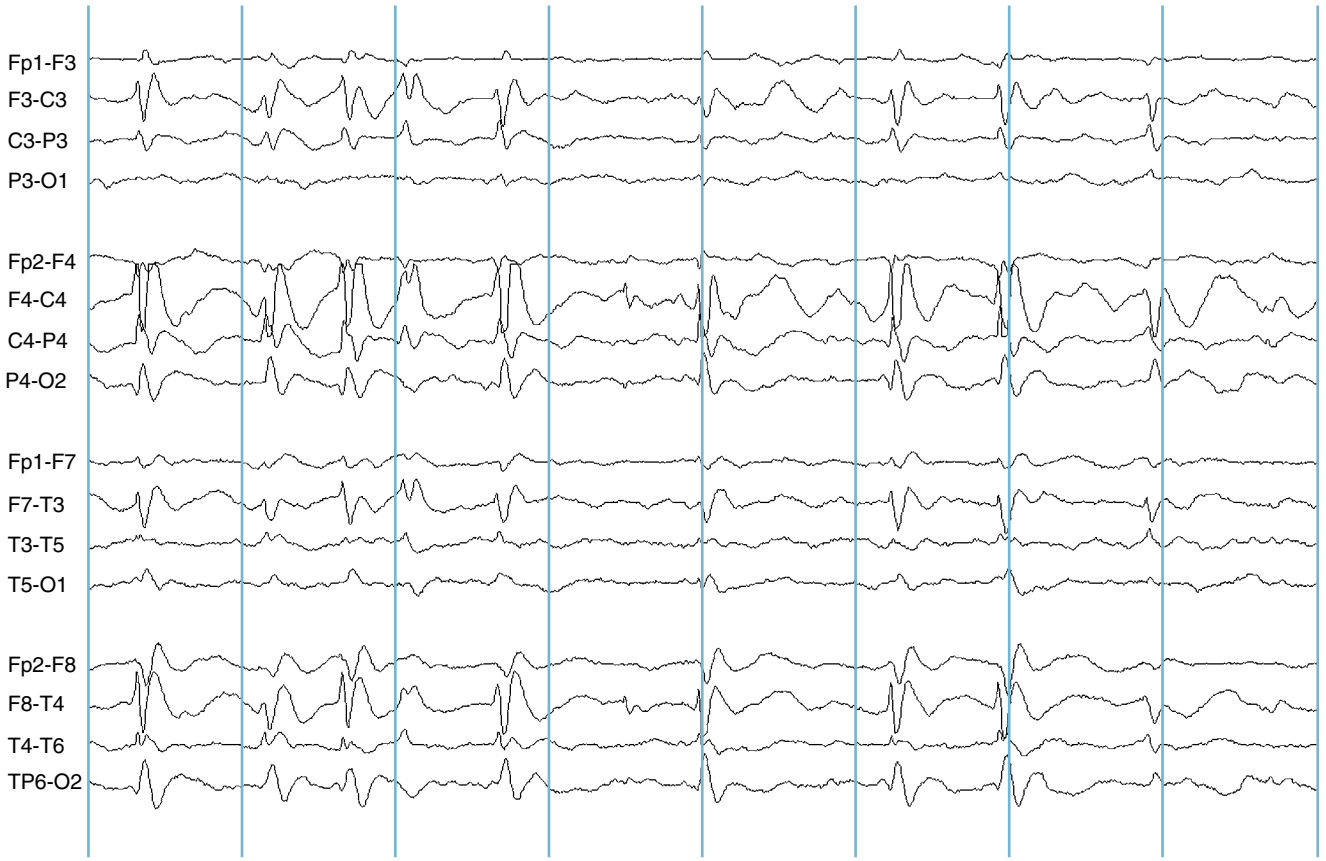


Figure 10-60 The EEG findings of Landau-Kleffner syndrome (LKS) are more widely variable than true ESES patterns. The component discharges seen in LKS may resemble rolandic discharges and are typically activated by sleep. They may be seen over one or both hemispheres. Although the discharges shown here are semirhythmic, they would not be mistaken for continuous electrographic seizure activity.

Localization-Related Epilepsies

Temporal Lobe Epilepsy

Temporal lobe epilepsy is the most common localization-related (focal) epilepsy. The seizure type most often associated with temporal lobe epilepsy is the complex partial seizure. When a cause for temporal lobe epilepsy can be identified, such as a glioma, hamartoma, or a dysgenetic lesion in the temporal lobe, temporal lobe epilepsy can be considered a symptomatic epilepsy. In cases in which testing is normal, temporal lobe epilepsy may be classified as a cryptogenic epilepsy. In cases in which mesial temporal sclerosis is present, this categorization is less clear because it is not always known whether the sclerosis is the cause or the result of the temporal lobe seizures.

The term *complex partial seizure* should not be equated with temporal lobe epilepsy. Most, but not all, seizures associated with the temporal lobe can be classified as complex partial seizures. Complex partial seizures can, however, arise from any lobe of the brain—frontal, parietal, and occipital foci can all result in complex partial seizures.

The classic temporal lobe seizure may begin with staring, possibly associated with automatisms such as fumbling movements of a hand, chewing, or lip-smacking automatic behaviors. If there is a seizure prodrome, it may include psychic symptoms such as a feeling of fear or déjà vu, or hallucinations involving the senses of taste or smell. Here, the term *prodrome* is misleading because such psychic symptoms actually represent the beginning of the ictal discharge rather than a warning that one is to occur. Dystonic posturing of a hand, when it occurs, is typically seen contralateral to the seizure focus. Conversely, automatic behaviors in a hand such as fumbling or picking movements usually occur ipsilateral to the side of the seizure focus. These automatic movements are not actually driven directly by the seizure discharge but rather occur as a release phenomenon. Temporal lobe seizure activity may spread to the ipsilateral motor cortex and include clonic or tonic movements of the contralateral side of the body, or the seizure may secondarily generalize and evolve to a generalized convulsion.

EEG Findings in Temporal Lobe Epilepsy

Because most temporal lobe epilepsy originates in the anterior temporal lobe, the classic EEG finding of temporal lobe epilepsy is the anterior temporal spike or sharp wave. An isolated spike in either the F7

or F8 or adjacent electrodes can aid significantly in localizing a seizure focus to the temporal lobe. Focal temporal discharges may be activated by sleep. An associated slow-wave abnormality may also seen in the temporal lobe, either in the form of intermittent slow (temporal intermittent rhythmic delta activity, or TIRDA) or continuous slowing. The use of additional electrodes over the anterior temporal lobes, such as the T1 and T2 positions (or the similar FT9 and FT10 positions) can enhance the recording of discharges from the temporal lobes. Because the yield of invasive electrodes such as sphenoidal and nasopharyngeal electrodes is not significantly better than these additional surface temporal electrodes, the use of these invasive has become much less common.

Temporal lobe seizures can be associated with highly focal discharges as described earlier, with spikes or sharp waves that manifest a broader field across the temporal lobe, or with discharges that are triggered by irritability in the temporal lobe but that express themselves across the whole of the involved hemisphere. In some cases, a focal temporal lobe abnormality can set off a generalized discharge (secondary bilateral synchrony) resulting in generalized spike-wave discharges with varying amounts of interhemispheric symmetry (see Chapter 9, "The Abnormal EEG" for a more detailed discussion of secondary bilateral synchrony).

Other Lesional Epilepsies

Although the temporal lobe is the most common location for lesional epilepsies, lesions that cause focal-onset seizures can be located in any area of cerebral cortex and in deeper areas as well. Lesions that cause localization-related epilepsies may be either congenital or acquired. Congenital lesions include cortical dysplasias and other malformations, including the hamartomas associated with tuberous sclerosis or pre- of perinatally acquired cortical scars. In childhood and adulthood, seizure foci may be created by a variety of processes including traumatic injury, vascular injury (including stroke and complications of vascular malformations), tumors, inflammation including infectious and autoimmune phenomena, and neurodegenerative processes.

The EEG signs of lesional epilepsy in nontemporal locations are similar to those of temporal lobe epilepsy as described earlier. Focal spikes or sharp waves arising from the epileptogenic area are the most characteristic signs of a focal epilepsy. Associated slow-wave abnormalities may also be present.

Occasionally, generalized spike-wave discharges are seen in which the spike focus does not have a clear localization (see Figure 10-61). Although this type of spike-wave discharge may appear to be generalized,

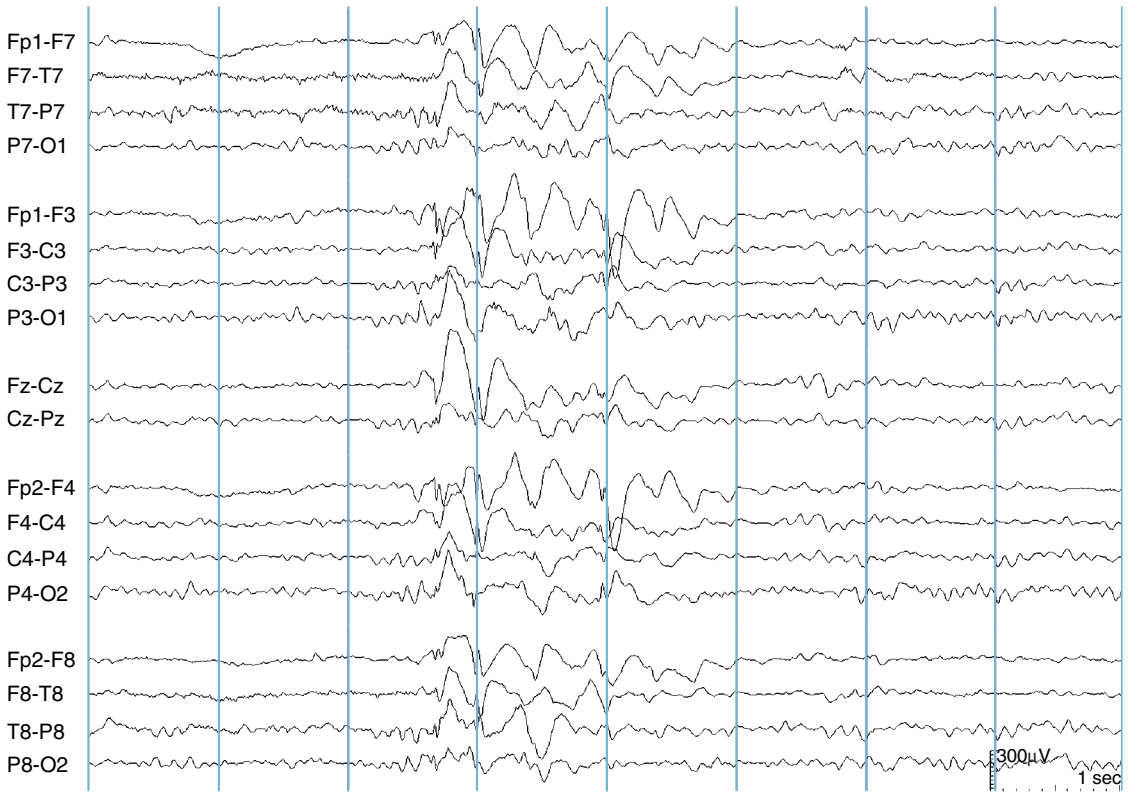


Figure 10-61 The burst seen in the middle of this page could be classified either as generalized spike wave with unclear spike localization or a burst of rhythmic slowing with intermixed spikes. These poorly organized spike-wave bursts may have either a generalized or a focal mechanism of onset.

the mechanism of onset is potentially either focal or generalized.

Figures 10-62, 10-63, 10-64, and 10-65 show examples of focal discharges associated with lesional epilepsies. Depending on the location of the seizure focus, the semiology of focal seizures varies widely. The subjective and observed manifestations of seizures provide important localization information in addition to EEG data.

Multifocal Spikes

EEGs with multifocal spikes are usually found in patients with some degree of developmental delay or mental retardation. Spikes or sharps are usually considered multifocal when three or more independent localizations are seen, usually with at least one location in each hemisphere (see Figure 10-66). This finding is generally felt to be indicative of an *epileptic encephalopathy*, a multifocal or diffuse epileptic process associated with a decreased seizure threshold and usually with a history of seizures. One important apparent exception to this rule is the patient with a benign developmental epilepsy, such as BCECTS. In addition to bilateral independent centrotemporal discharges, these patients may also manifest occipital discharges and occasionally discharges in other locations. Technically, these children have multifocal spikes, but they usually do not manifest clinical signs of a true epileptic encephalopathy and should be separated from the general group of patients with multifocal spikes.

Identification of Seizure Discharges

The large majority of seizure discharges are rhythmic discharges. The converse is not true: by far, most rhythmic discharges are not seizure discharges. How, then, to distinguish which rhythmic discharges are seizure discharges? The key features that mark a seizure discharge are a sudden change from the preceding background and gradual variations of the discharge in frequency and amplitude. Of the latter two parameters, the variation in frequency is most useful. Typically, a seizure discharge will show an increase or a decrease in its firing rate throughout its course. Sometimes the change in rate is subtle enough that the spiking frequency must be formally counted (see Figure 10-67). Occasionally a seizure discharge maintains an unvarying firing rate (see Figure 10-68), although a tailing-off of frequency may still be observable at the end of the discharge.

The most typical evolution of a seizure discharge is from lower voltage, faster activity to higher voltage, slower activity. This pattern may be seen in both gener-

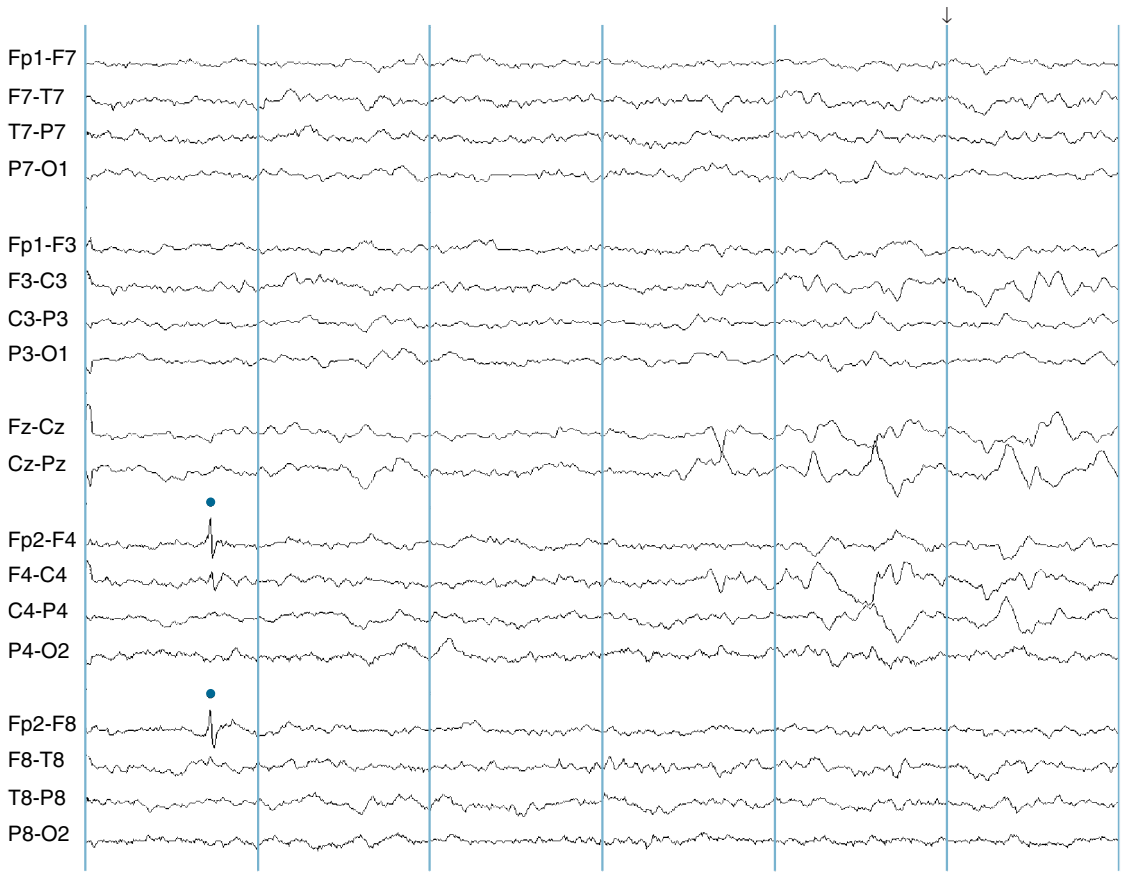


Figure 10-62 A right frontopolar spike-wave discharge seen in Stage Ib sleep (dots) is related to a focal lesion in the anterior right frontal lobe.

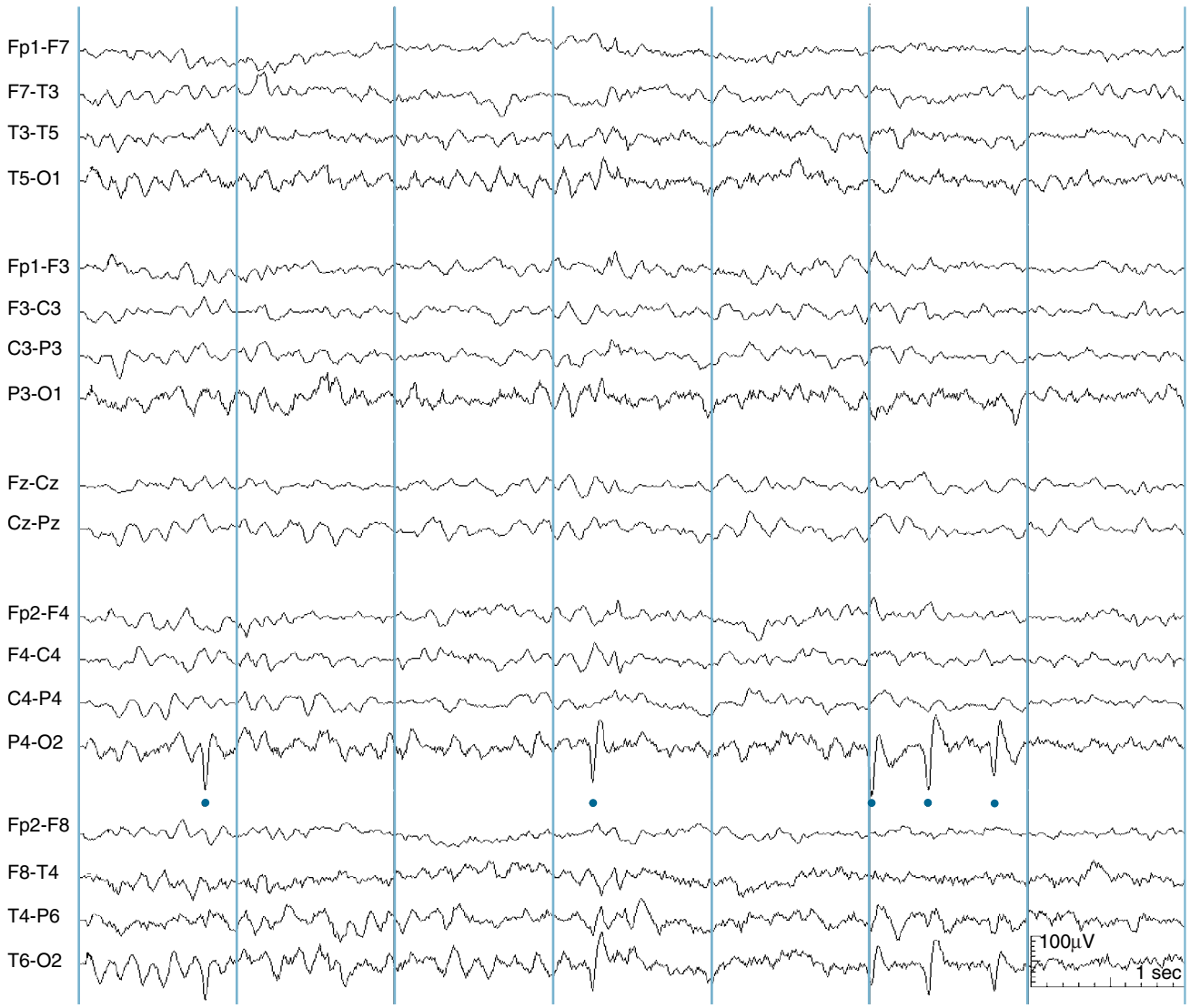


Figure 10-63 Focal right occipital spike-wave discharges (dots) are seen at the transition from wakefulness to drowsiness.

alized discharges, as was seen in Figure 10-18, and in focal discharges (see Figure 10-69). Other patterns of frequency change are possible and are more likely to occur with lengthier discharges, including both accelerations and decelerations of the firing frequency. Occasional seizure discharges do not manifest this characteristic evolution in frequency and must be recognized by their sudden onset and their clear contrast from preceding background rhythms. When suspicion arises that a brief, nonevolving discharge may represent a seizure discharge, it is important to search for similar rhythms elsewhere in the tracing. If a run of suspicious 20-Hz beta activity is seen, but similar 20-Hz rhythms are identified elsewhere in the tracing's background, it is much less likely that the rhythm represents a seizure. It is important not to mistake the waxing and waning of an underlying background rhythm for an EEG seizure.

Wave amplitude typically shows some variation throughout a seizure discharge's evolution, but this sign alone is not reliable in distinguishing seizure from nonseizure activity. Rhythmic waves that vary only in amplitude and not in frequency often do not represent seizure activity. Arousal rhythms may sometimes resemble seizure discharges, partly because they can appear as an abrupt change from the previous sleep pattern. Abrupt change from the background is characteristic of seizure discharges, but is also characteristic of the process of awakening. A suddenly appearing high-voltage arousal rhythm usually maintains a steady frequency that helps to distinguish it from a seizure discharge. The high-voltage hypersynchronous responses to hyperventilation and arousal patterns are both good examples of this phenomenon. These dramatic but benign wave patterns often show waxing and waning amplitudes but steady frequencies, helping to distinguish them from seizure discharges (see Figure 10-70).

The component waves of a seizure discharge often, but not always, show epileptiform morphology. Commonly,

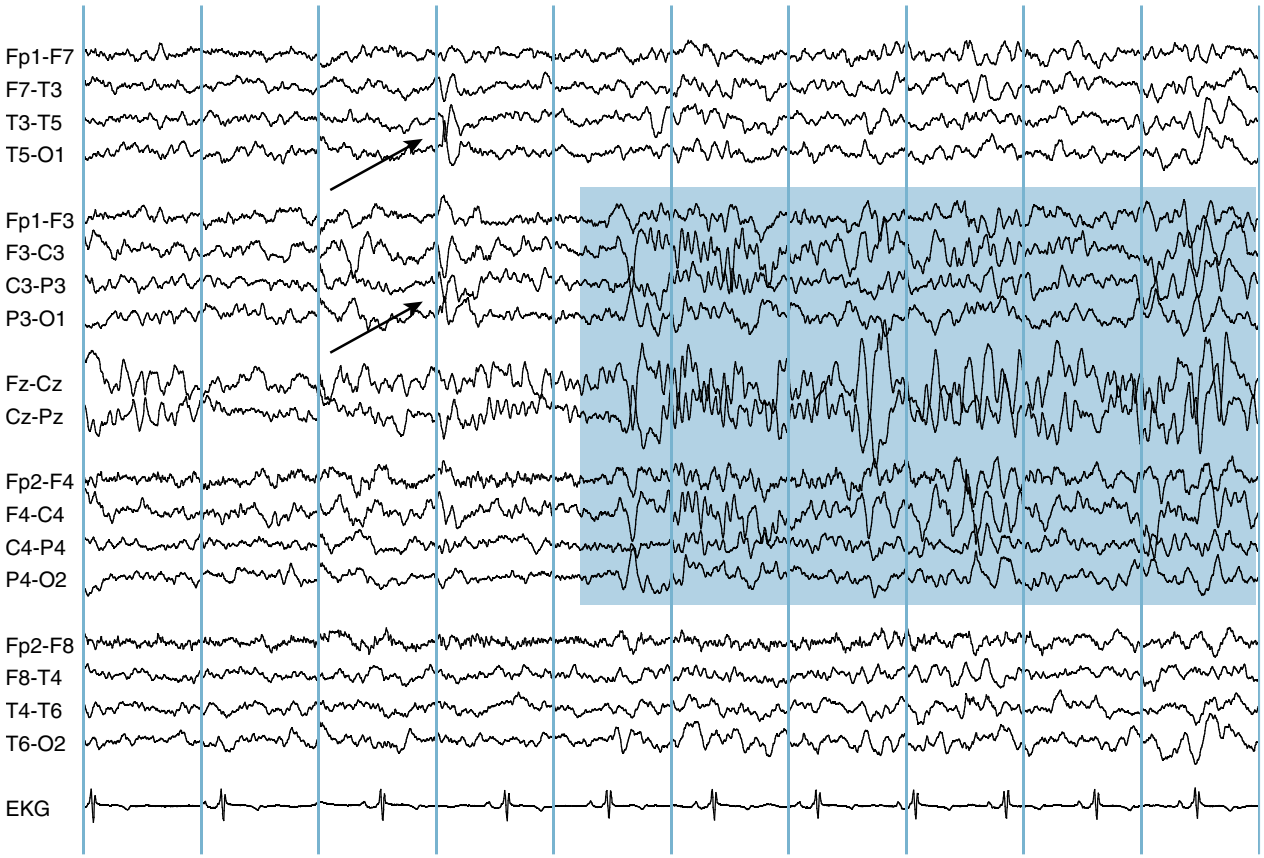


Figure 10-64 A focal spike-wave discharge is seen with a maximum in the left posterior temporal and parietal electrodes during Stage II sleep (arrows). Vertex waves and sleep spindles (blue rectangle) are distinct from the temporoparietal discharge.

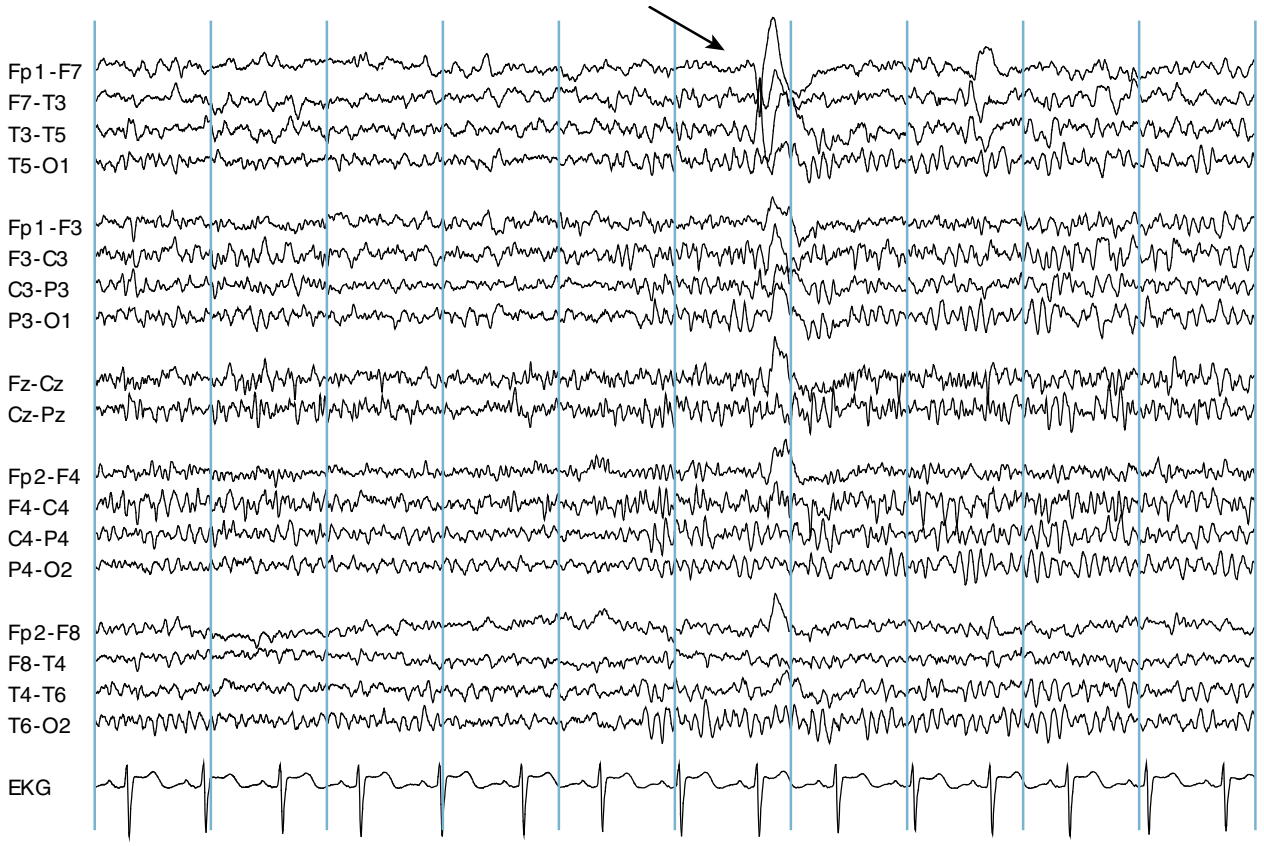


Figure 10-65 A left anterior temporal spike-wave discharge is seen in a 22-year-old woman with temporal lobe epilepsy (arrow).

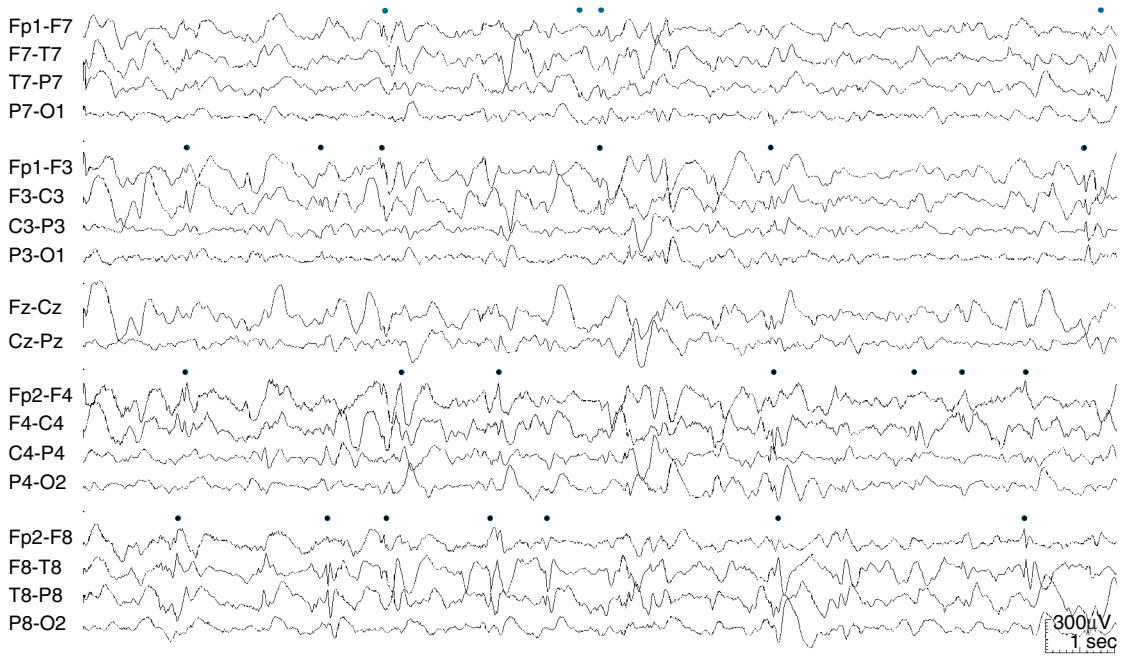


Figure 10-66 A pattern of multifocal spikes is seen (dots) during light sleep. Unless the different spike foci can be shown to belong to a single benign developmental epilepsy syndrome (on the basis of the patient's history and the location of the spikes), the multiplicity of spike locations seen here suggests the presence of a diffuse epileptic encephalopathy, usually associated with some degree of cognitive abnormality.

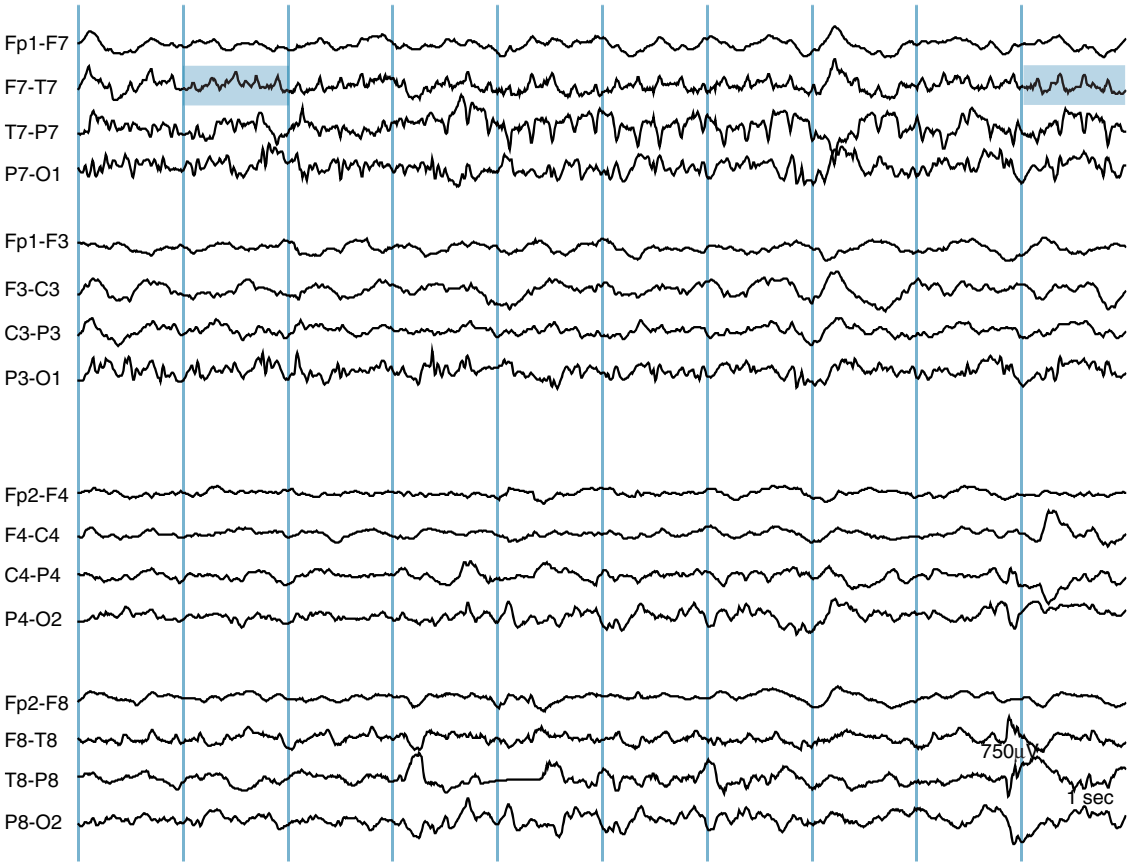


Figure 10-67 The seizure discharge seen in the left temporal chain seems, at first glance, to have a fairly constant firing frequency. When the spike frequency is formally measured at the beginning of the page and at the end of the page (see shaded rectangles), it can be seen that the discharge has slowed from 5 Hz to 4 Hz over these 8 seconds.

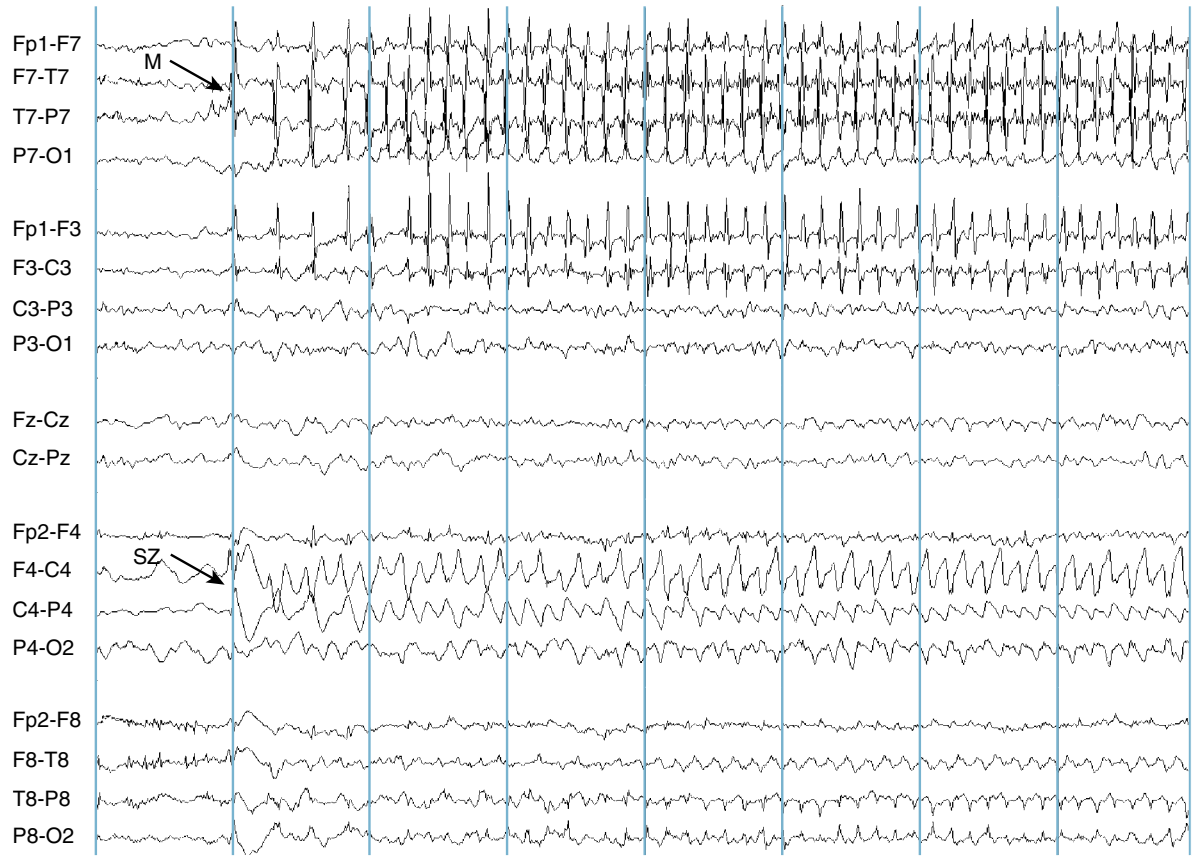


Figure 10-68 A seizure discharge (SZ) is seen beginning in the right central area in a teenage boy with Rasmussen encephalitis, a degenerative disorder that usually involves a single hemisphere. Because this discharge happens to involve the face area of the motor strip, the rhythmic left facial twitching caused by the seizure can be seen as repetitive muscle artifact related to the twitching (M) over the left frontotemporal area.

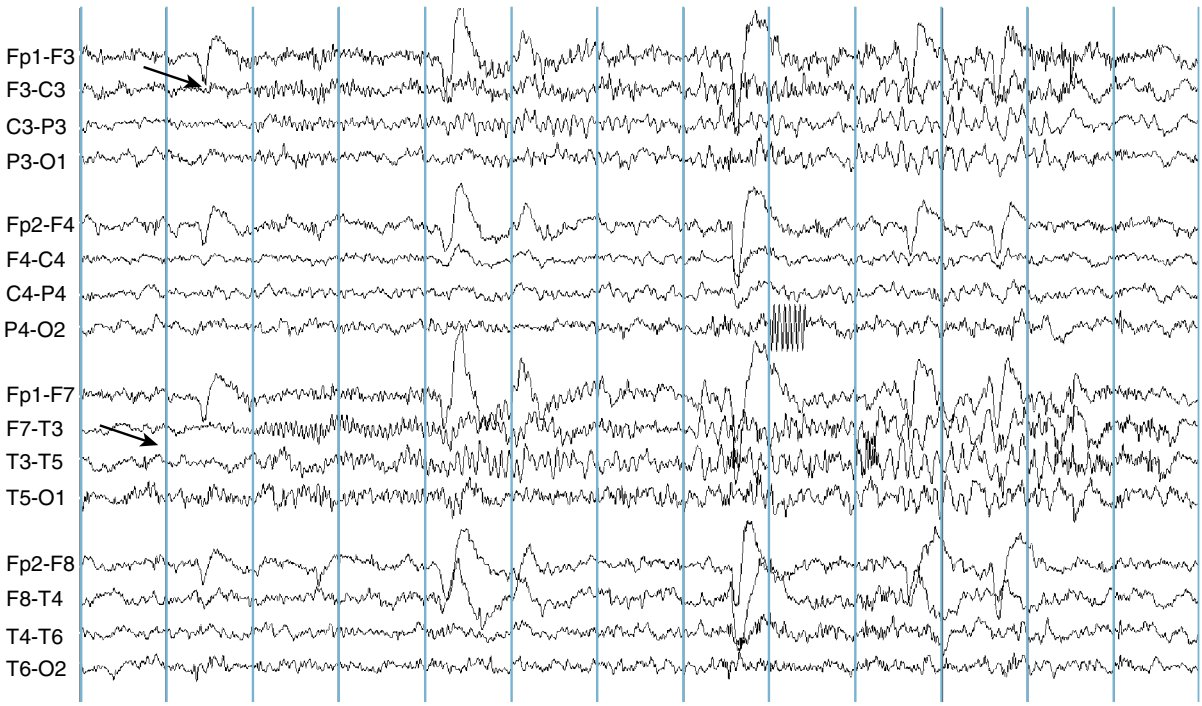


Figure 10-69 A fast seizure discharge is seen to begin in the left temporal area quickly spreading to the left central area (arrows; montage setup: left parasagittal chain over right parasagittal chain, left temporal chain over right temporal chain). Throughout the course of the discharge, the firing frequency slows and the discharge terminates by the last second of the page.

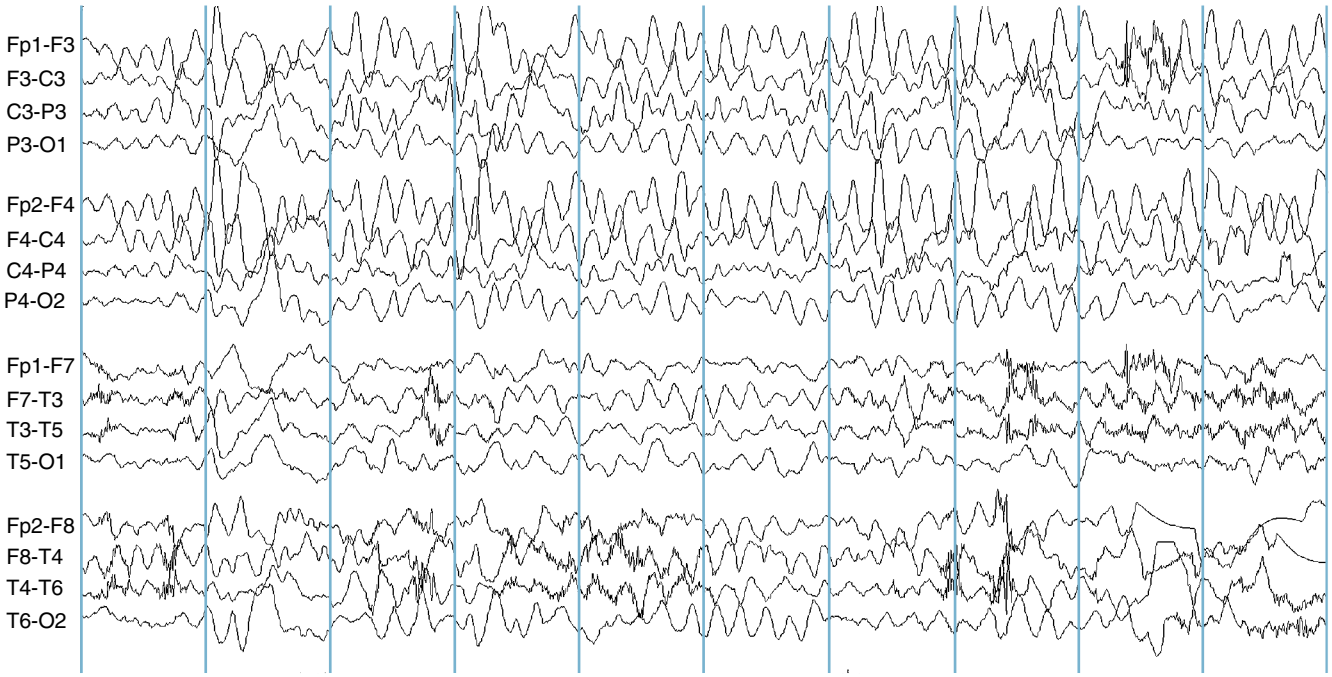


Figure 10-70 These high-voltage, hypersynchronous waves represent an arousal hypersynchrony rather than a seizure discharge. The fact that the waves retain a steady frequency throughout helps distinguish between the two possibilities.

seizures manifest as a spray of rapid spikes or spike-wave discharges. Nevertheless, some seizure discharges may have no sharp features, appearing as rounded, sinusoidal waves. In general, the closer the seizure is to the recording electrode, the more likely it is to manifest sharp features. Seizure discharges that show more rounded (as opposed to sharp) features are assumed to arise at a greater distance from the recording electrode. During a focal seizure discharge, slow waves may appear in the opposite hemisphere.

The foregoing discussion presupposes that the rhythmic waveform being analyzed in the EEG is true cerebral activity and not some type of artifact. As with the assessment of epileptiform activity, the first step in assessing a rhythmic waveform is to exclude artifact. Many types of rhythmic movements may occur in patients creating an artifact that may mimic a seizure discharge. Repetitive movements from patting or chest physical therapy, toothbrushing, head scratching, and head rocking may all resemble seizure discharges (see Figure 10-71). As with evaluation of epileptiform activity, a seizure discharge should have a plausible electric field. The process of distinguishing artifact from cerebral activity is discussed further in Chapter 4, "Electroencephalographic Localization," and Chapter 6, "Electroencephalographic Artifacts."

A minority of seizure discharges do not match the patterns of lengthy, evolving seizure discharges described earlier. As discussed, myoclonic seizures are very quick seizures often associated with bursts of polyspikes that may be too short to show evolution or rhythmicity in the typical sense. The characteristic, brief electrographic pattern of infantile spasms was described earlier and occasionally consists of a single, diffuse slow wave with complex topography. As already shown, some seizures may be associated with an electrodecremental pattern in the EEG.

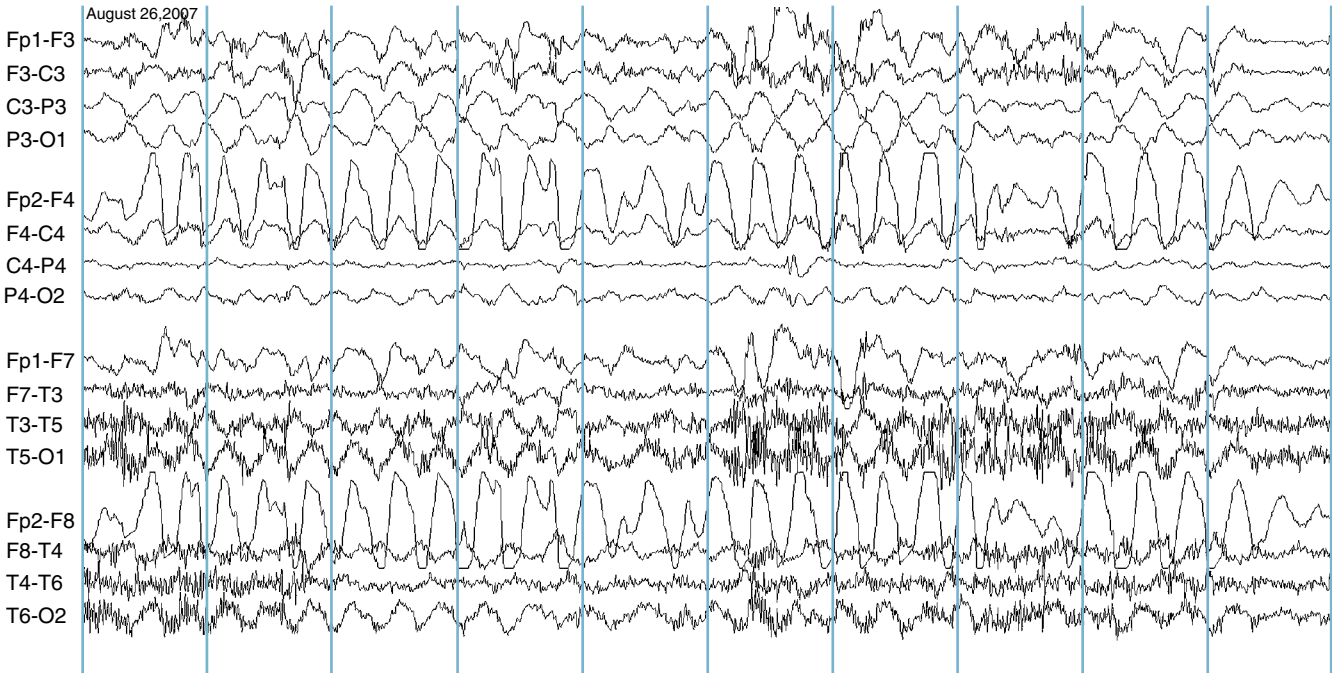


Figure 10-71 The repetitive and rhythmic waves seen on this page are caused by chest physical therapy artifact rather than a seizure discharge. Because the head movement caused by the rhythmic chest percussion jostles each electrode in an unpredictable way, high-voltage deflections are seen from some electrodes that are adjacent to electrodes that manifest little or no deflection, indicating that the apparent discharge has no plausible electric field and is the result of motion artifact.

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Chapter

11 Normal Variants in the Electroencephalogram

This section deals with a group of waveforms that may mimic abnormal waves but have now been recognized as normal variants. By definition, a normal variant is not associated with disease, be it epilepsy or another abnormal state. A few of the waveforms discussed in this chapter are of uncertain clinical significance: they are known to occur frequently in normal individuals but may be seen more often in people with epilepsy.

Many of the normal variant patterns described here bear some resemblance to epileptiform activity. The importance of developing proficiency in recognizing these patterns is to avoid mistaking them for epileptiform abnormalities. The basic features of these normal variants should be committed to memory so as to avoid the pitfall of describing one of these variants as an epileptiform abnormality (Table 11-1).

NORMAL VARIANTS THAT MIMIC SINGLE EPILEPTIFORM WAVES

Posterior Occipital Sharp Transients of Sleep

Posterior occipital sharp transients of sleep (POSTS) are one of the most common normal variants seen in the EEG and can be considered one of the normal elements of sleep. The acronym POSTS tells the story of these distinctive waveforms: POSTS are of Positive polarity, they are seen in the Occipital areas; they have a Sharp Transient waveform, and they occur in Sleep. POSTS are "triangular" or V-shaped wave that are particularly prominent in light sleep (see Figures 11-1 and 11-2). If not recognized as POSTS, these low-voltage discharges could potentially be mistaken for occipital sharp waves. Because POSTS are so common, the polarity of any low- to medium-voltage occipital sharp wave seen in sleep should be assessed before deciding that it is abnormal. Displaying POSTS in an appropriate referential montage should confirm their positive polarity and correctly identify them as POSTS rather than epileptiform discharges. POSTS usually appear in a bilaterally synchronous fashion, although normal POSTS may manifest asymmetrical amplitudes. POSTS may occur in brief, semirhythmic runs. Although POSTS usually consist of low-voltage, V-shaped waves, they may occasionally assume a more spiky appearance (see Figure 11-3).

Lambda Waves

Lambda waves are discussed with POSTS because their morphology and location are similar. The two are easily distinguished because lambda waves occur exclusively

Table 11-1 Summary Table of Selected Normal Variants

Posterior Occipital Sharp Transients of Sleep	Positive-polarity, low-voltage occipital sharps occurring in sleep, a normal sleep element
Lambda Waves	Low-voltage occipital sharps during wakefulness associated with searching eye movements
Small Sharp Spikes/Benign Epileptiform Transients of Sleep	Low-voltage temporal spikes, synchronous or independent, unilateral or bilateral, with broad field seen in adults during drowsiness and light sleep
Mu Rhythms	Arch-shaped rhythm in central areas during wakefulness, suppresses with contralateral hand movement
14- and 6-Hz Positive Bursts	Medium- to high-voltage, positive-polarity, arch-shaped bursts during drowsiness and light sleep in posterior temporal and occipital areas, mostly in children
Wicket Spikes/Wicket Rhythms	Arch-shaped rhythm of temporal areas during drowsiness and light sleep, mostly in adults
Breach Rhythms	Rhythm of increased fast activity, often with spiky appearance, seen over craniotomy sites
Rhythmic Temporal Theta Bursts of Drowsiness/Psychomotor Variant	Sharpened theta rhythm in temporal areas during drowsiness and light sleep
6-Hz Spike and Wave/Phantom Spike and Wave	Brief 6-Hz rhythms with inconsistent spike component seen in wakefulness and drowsiness, mostly in adults, following WHAM and FOLD patterns
Posterior Slow Waves of Youth	Theta and delta waves intermixed with posterior rhythm until mid-teenage years

FOLD, Female whose pattern has an Occipital emphasis, Low in amplitude, and seen in the Drowsy record; WHAM, Waking record, High in amplitude, Anterior in location and especially in Males.

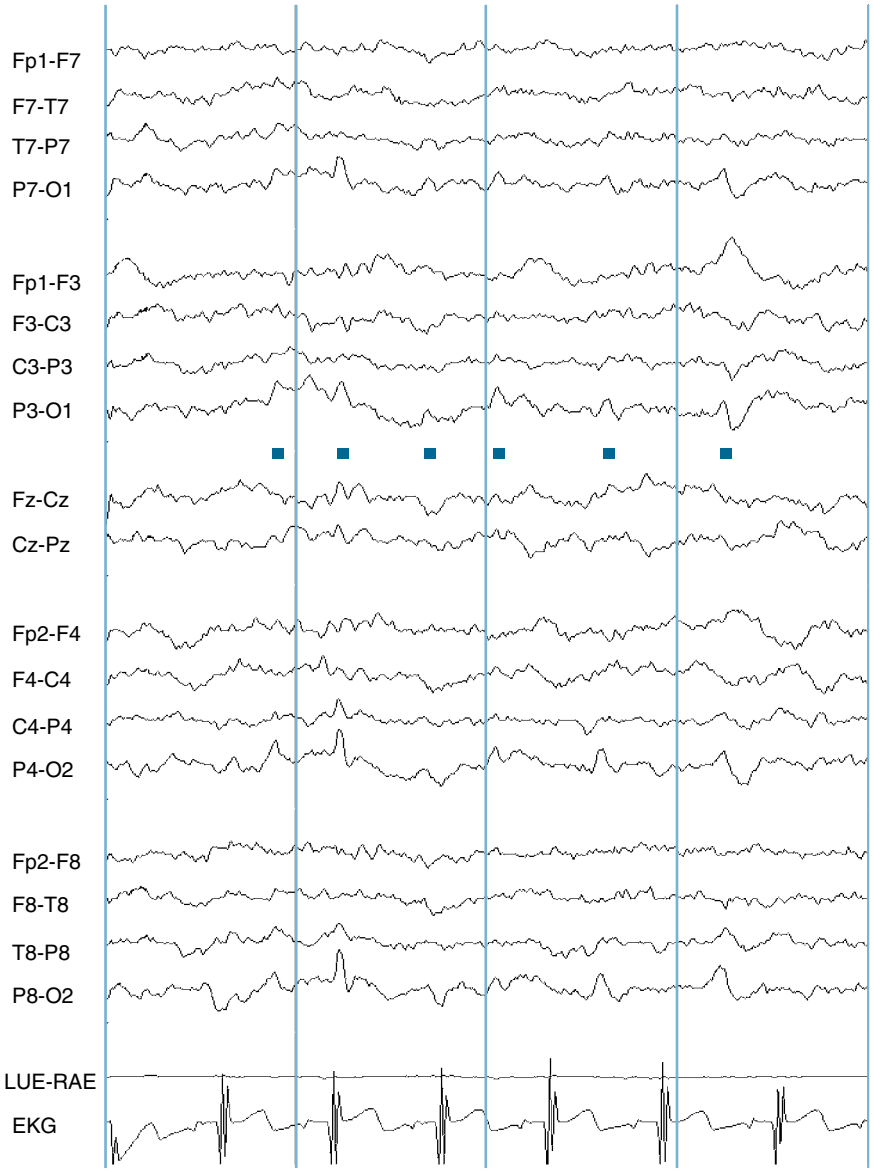

Figure 11-1 The distinctive triangular waves seen in the four occipital channels (P7-O1, P3-O1, P4-O2, and P8-O2) are examples of posterior occipital sharp transients of sleep (POSTS; indicated by dots). The upgoing deflection of these waves in the occipital channels of this bipolar montage suggests two possible polarity/localization combinations. The first is that the upgoing wave implies that the parietal electrodes, P3 and P4, are "more negative" than O1 and O2 and that these waves could be caused by a negativity in the parietal areas. No clear phase reversal is seen anterior to the upgoing waves, however, raising the suspicion that a parietal negativity does not explain the waveform. The second possibility is the correct explanation: O1 and O2 should be considered "more positive" than P3 and P4, and a positivity in the occipital area is causing the deflection. The absolute polarity of the event—in this case, an occipital positivity—is most easily confirmed by displaying it in a referential montage (see Figure 11-2).

during wakefulness and POSTS are seen during sleep. Lambda waves also appear as low-voltage triangular waves in the occipital areas, reminiscent of the Greek letter λ, but they are distinctive in that they occur at the time of lateral searching eye movements. Confirmation that a low-voltage occipital sharp transient wave is a lambda wave is made easier by finding evidence of concurrent lateral eye movement artifact. Lambda waves may be either surface positive or surface negative in the occipital area (see Figure 11-4). They are not as common as POSTS and are seen more frequently in children than in adults. Because they are related to searching eye movements, lambda waves are generally seen when the patient's eyes are open and the posterior rhythm is suppressed. Voltage asymmetry of lambda waves is not necessarily considered abnormal.

Small Sharp Spikes and Benign Epileptiform Transients of Sleep

The terms small sharp spikes (SSS) and benign epileptiform transients of sleep (BETS) are synonymous. These quick, low-voltage spikes are usually seen in the temporal areas with a broad gradient across the temporal chain. The upward and downward phases of the transients are usually of similar amplitude. They occur

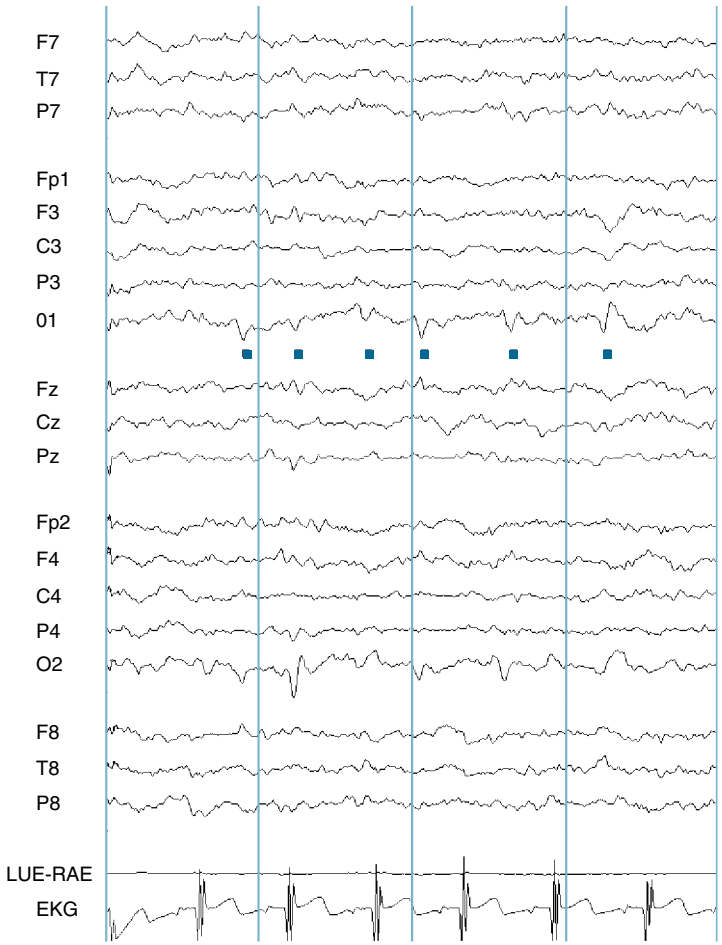

Figure 11-2 The same page of EEG shown in the previous figure is displayed in a referential montage. The clear downgoing deflections in the O1 and O2 channels (dots) clarify the positive polarity of these occipital discharges and confirm that they are an example of posterior occipital sharp transients of sleep.

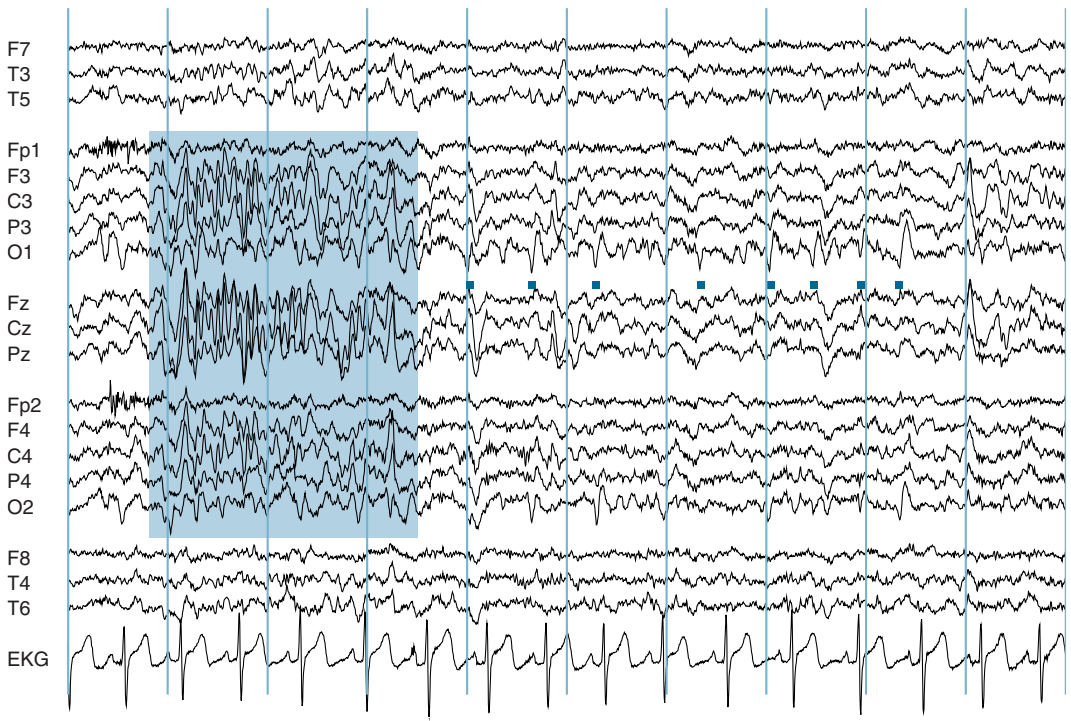

Figure 11-3 An example of Stage II sleep is shown in a referential montage (note the sleep spindles in the shaded area). Posterior occipital sharp transients of sleep (POSTS) are seen in the O1 and O2 channels (dots). These POSTS have a more spike-like morphology than those seen in the previous example; the initial downgoing deflections indicate their positive polarity. These occipital waves are not synchronous with the electrocardiogram (EKG) complexes and therefore do not represent EKG artifact.

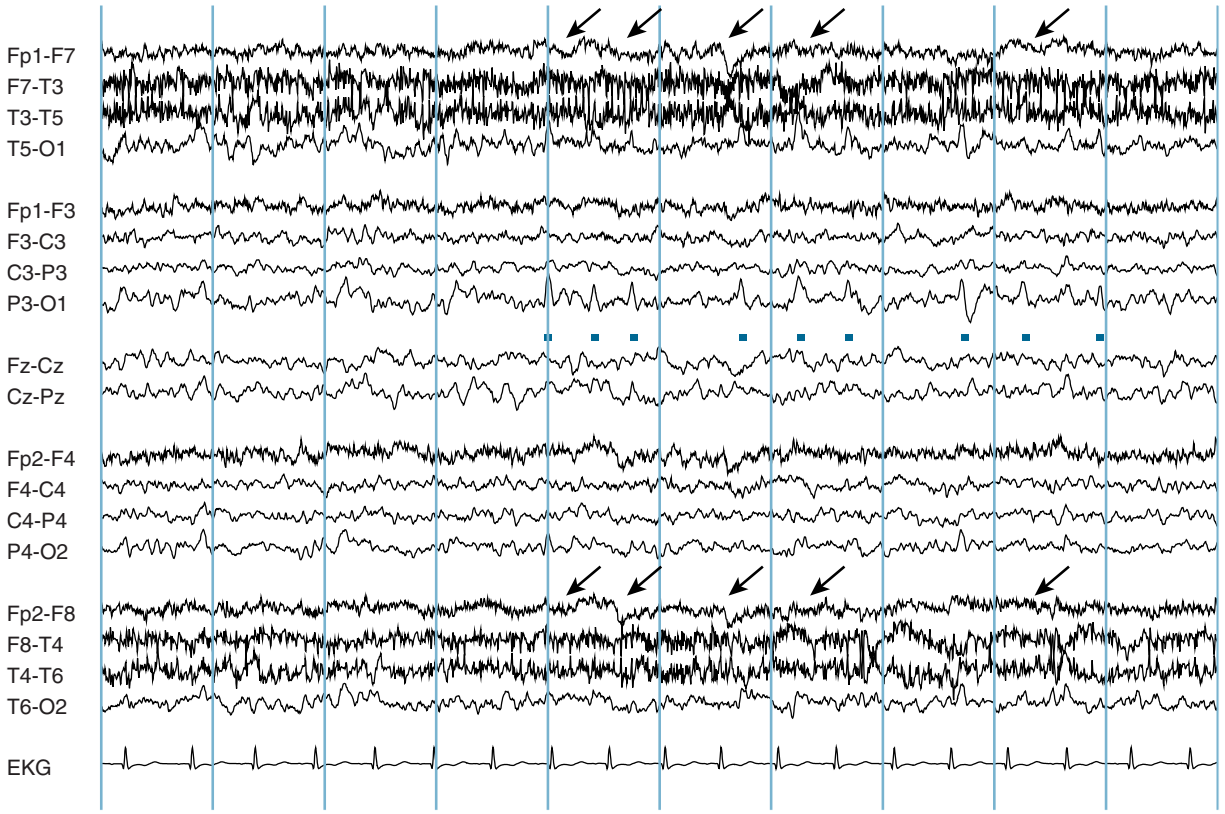

Figure 11-4 The triangular-shaped waves seen in the occipital channels are examples of lambda waves (dots). Lambda waves are associated with horizontal searching eye movements. Subtle lateral eye movement artifact (arrows) is seen in the frontal/anterior temporal channels with opposite polarity on each side (see Chapter 6, "Artifacts") for further description of eye movement artifact).

either unilaterally or bilaterally and, when bilateral, they may occur either synchronously or independently (see Figure 11-5). SSS are seen in drowsiness and light sleep and tend to disappear with deepening sleep. SSS are considered by many to represent a normal variant but some authors still contend that the finding suggests an increased degree of epileptogenicity.

NORMAL VARIANTS THAT MIMIC REPETITIVE EPILEPTIFORM WAVES

Mu Rhythms

Mu rhythms are commonly encountered rhythms seen in the central areas during wakefulness, best recorded by the C3 and C4 electrodes. They are most often seen from later childhood into the adult years, although they are occasionally seen in very young subjects. The mu rhythm has a distinctive arciform (arch-like) or "comb-like" morphology (see Figure 11-6). Because the mu rhythm is suppressed by voluntary motor activity in the opposite hand, the technologist can establish that a sharp central rhythm is a mu rhythm by requesting that the patient move the contralateral hand and demonstrating that the rhythm disappears. Although classically suppressed by moving the contralateral hand, movement of the ipsilateral hand or planning to move the hand may also suppress the mu rhythm in some subjects.

Because this arciform rhythm is sharpened on one side and rounded on the other, there is some potential to mistake it for epileptiform activity. When mu rhythms occur in trains, it is not difficult to identify them correctly on the basis of their location, morphology, and suppression with movement, if necessary. Occasionally, fragments of a mu rhythm may resemble low-voltage spike activity (see Figure 11-7). Apparent low-voltage central spikes can be confirmed to be a mu phenomenon by showing that the morphology of the spike fragment is similar to the mu waves when they occur in trains.

Mu rhythms may be seen either unilaterally or bilaterally. They may suppress independently. Asymmetrical expression of mu rhythms is not considered abnormal. The mu rhythm tends to occur at a frequency similar to that of the patient's posterior rhythm and therefore, varies with age. In some patients, the posterior rhythm's field blends into the field of the mu rhythm creating large zones of alpha activity in the posterior quadrants. Because of the similar frequencies and amplitudes of the two rhythms, in such cases, it is not always clear where the posterior rhythm ends and the mu rhythm begins.

The mu rhythm and the posterior rhythm are the two main idling rhythms of the EEG: the mu rhythm is only seen during contralateral motor inactivity and suppresses with movement. Similarly, the posterior rhythm is only present during visual inactivity and suppresses with eye opening or visual attention. A mu-shaped rhythm that does not necessarily suppress

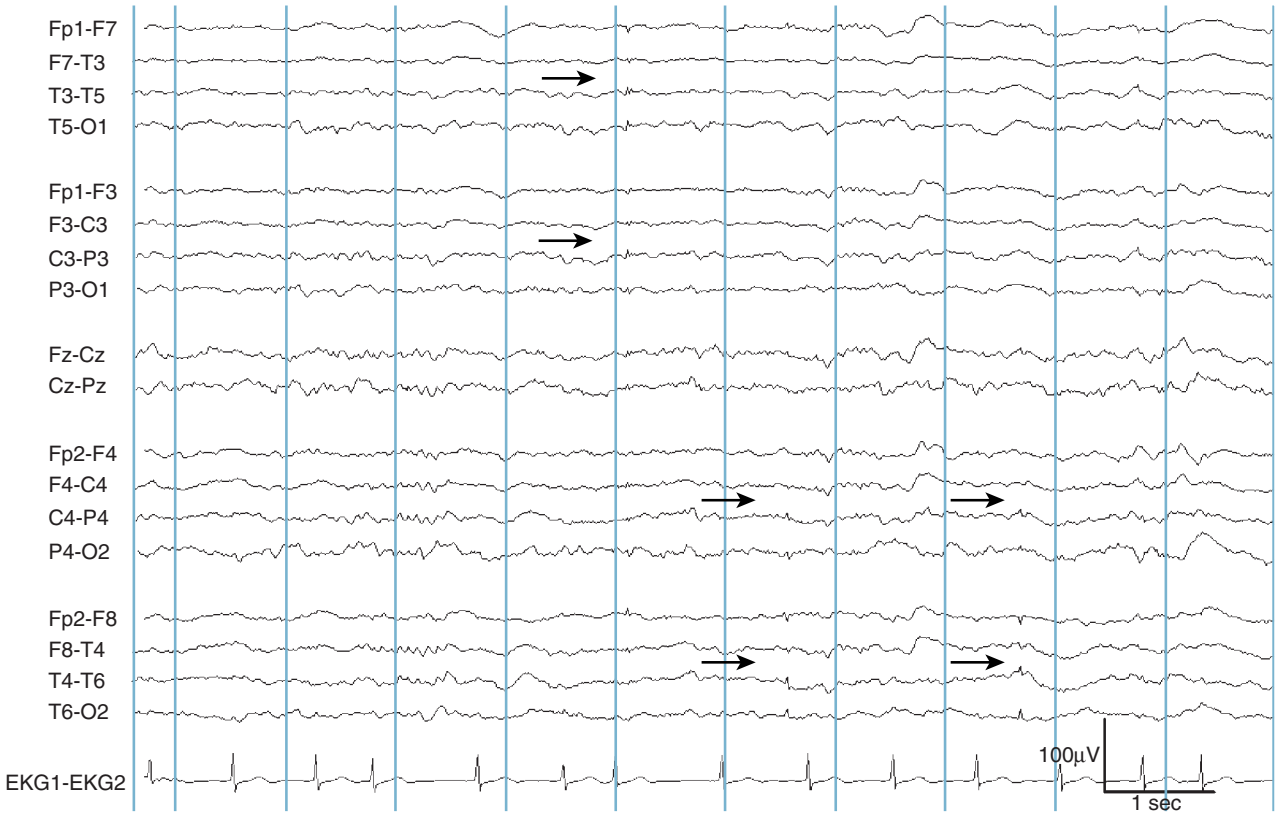

Figure 11-5 Small sharp spikes are seen in sleep, consisting of low-voltage spikes with a broad gradient across the temporal chains (arrows). In this typical example, the spikes are seen in both temporal areas independently.

with movement is occasionally seen in the central midline (Cz) and is referred to as a *midline theta rhythm.*

Wicket Spikes and Wicket Rhythms

Because their morphology is quite similar to that of mu rhythms, wicket rhythms are discussed with mu rhythms. Wicket rhythms differ from mu rhythms in that they are seen in drowsiness and light sleep rather than wakefulness and have a predilection for the temporal rather than the central areas (see Figure 11-8). Their arciform morphology is similar. Wicket rhythms range from 6 to 11 Hz with a voltage range of 60 to 200 μV (Reiher and Lebel, 1977). Similar to the situation seen with mu rhythms, it is possible to mistake a fragment of a wicket rhythm for epileptiform activity rather than a normal variant. Such fragments are called *wicket spikes.* Wicket spikes are distinct from temporal spike-wave discharges in that there is no aftercoming slow wave and they do not disrupt the underlying rhythm. The confirmation that a temporal spike is a wicket spike is best made by noting that the waveform is similar to that of the spikes when they occur in a train (as a continuous wicket rhythm) found elsewhere in the same tracing.

14- and 6-Hz Positive Bursts

Often referred to simply as "14 and 6," 14- and 6-Hz positive bursts are seen most frequently in adolescence. The term *ctenoids* (a word that means shaped like the teeth of a comb or like overlapping fish scales) has also been used for this phenomenon in the past but is no longer the preferred term. As the name implies, two versions of this variant are seen, one firing at a rate of 14 Hz and the other at 6 Hz. The 14-Hz form is more common (see Figures 11-9 through 11-11). The discharges are most prominent in the posterior temporal and occipital areas. The bursts consist of fast, arciform, or comb-shaped rhythmic discharges of low, medium, or high voltage in which the sharp phase has positive polarity and the rounded phase has negative polarity. It was initially asserted that 14 and 6 was associated with a variety of pathologic states, including epilepsy, but these bursts are now classified by most as a normal variant. The 6-Hz version of 14 and 6 is less commonly seen but felt to have the same significance; some patients manifest both forms in the same tracing.

Although 14 and 6 positive bursts may occur bilaterally, they usually do not fire synchronously. Asymmetrical occurrence of 14 and 6 positive bursts is not considered abnormal. Some authors believe that the 6-Hz component of 14 and 6 actually fires at 7 Hz and represents a subharmonic of the fundamental 14-Hz frequency, although true 6-Hz examples are seen. The bursts usually last 1 second or less, and there is no evolution in firing frequency during the burst. The distinctive wave morphology, frequency, and positive polarity help to confirm examples of 14- and 6-Hz positive bursts.

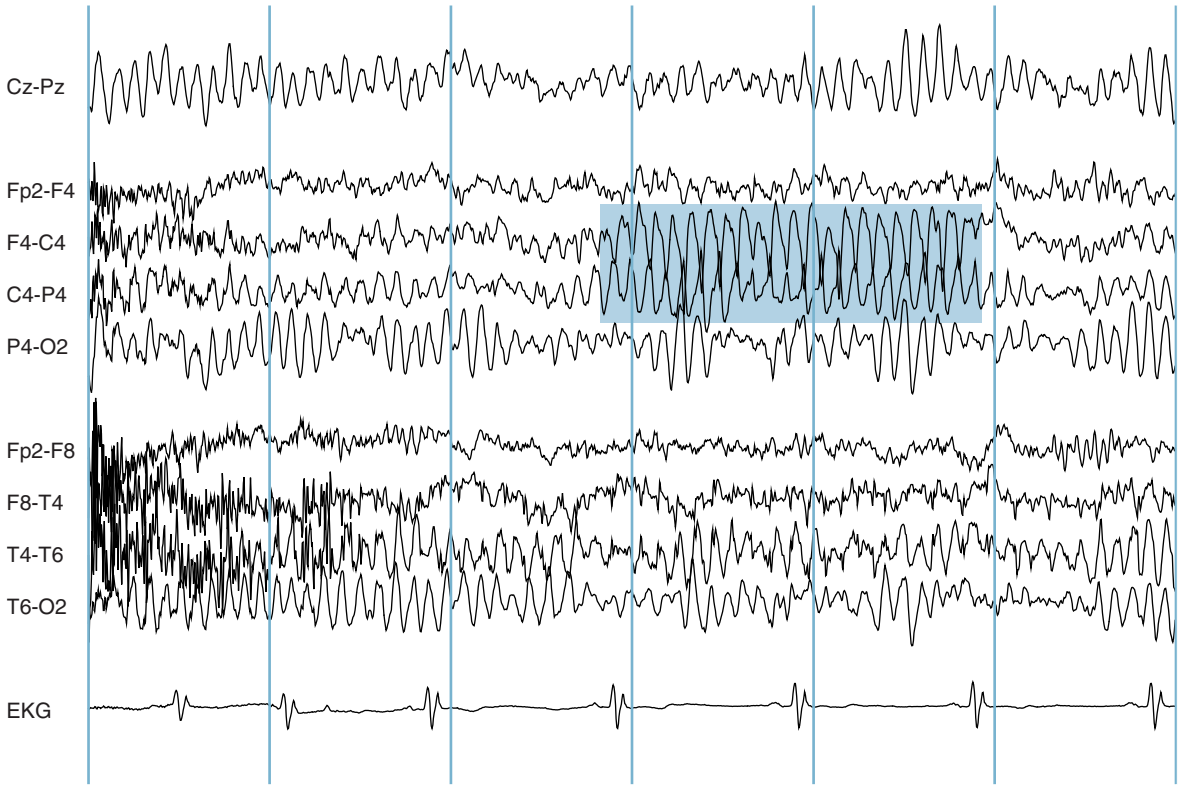

Figure 11-6 A mu rhythm is seen with maximum frequency in the right central (shaded) area, maximum in the C4 electrode. Note the typical morphology of the mu waveform, an arciform or comblike rhythm, rounded on one side and sharpened on the other.

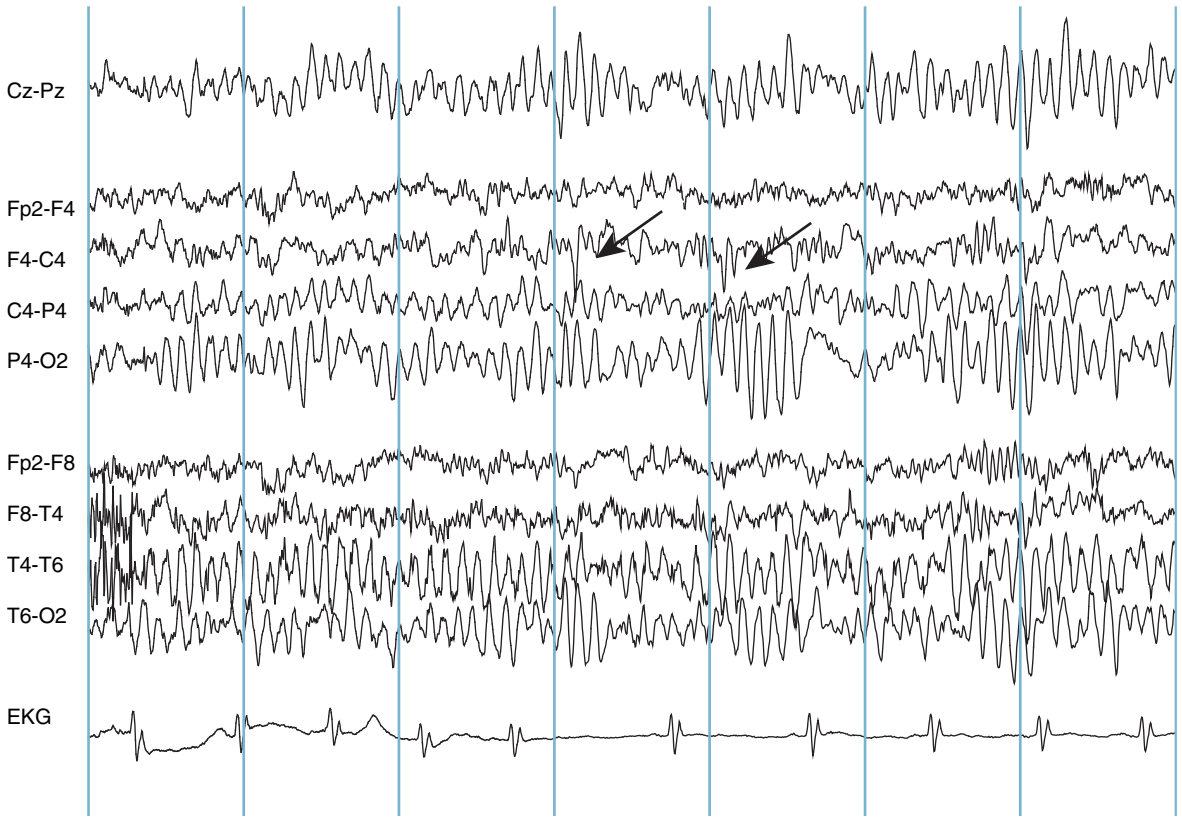

Figure 11-7 The two low-voltage transients (arrows) taken from the same tracing shown in the previous figure could be mistaken for low-voltage spikes. Comparing these transients to the mu rhythm shown in the previous figure, it becomes evident that these waveforms represent fragments of the patient's mu rhythm.

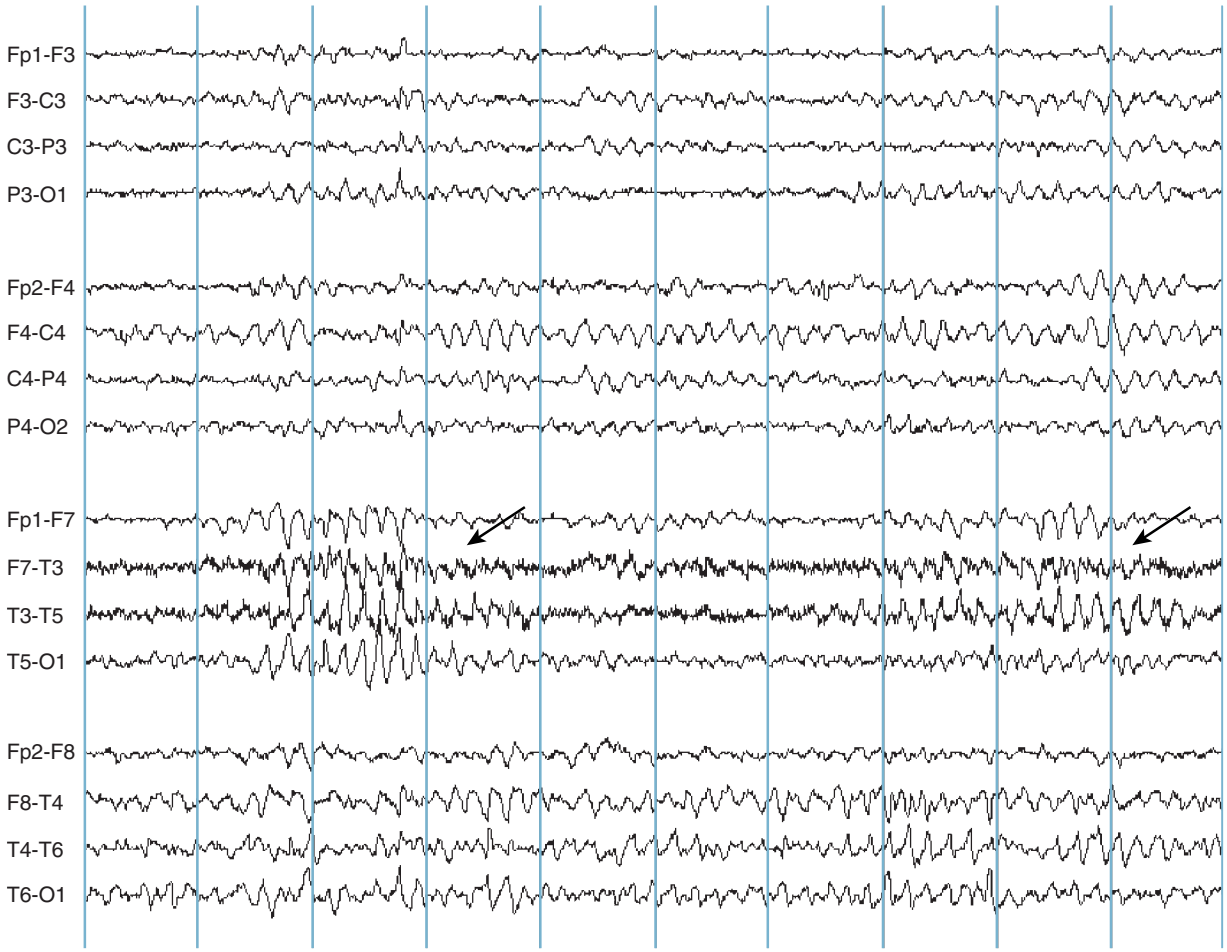

Figure 11-8 Brief trains of wicket spikes are seen in the left temporal area (arrows). Note the arciform morphology. A lower voltage wicket rhythm is present on the right (bottom four channels).

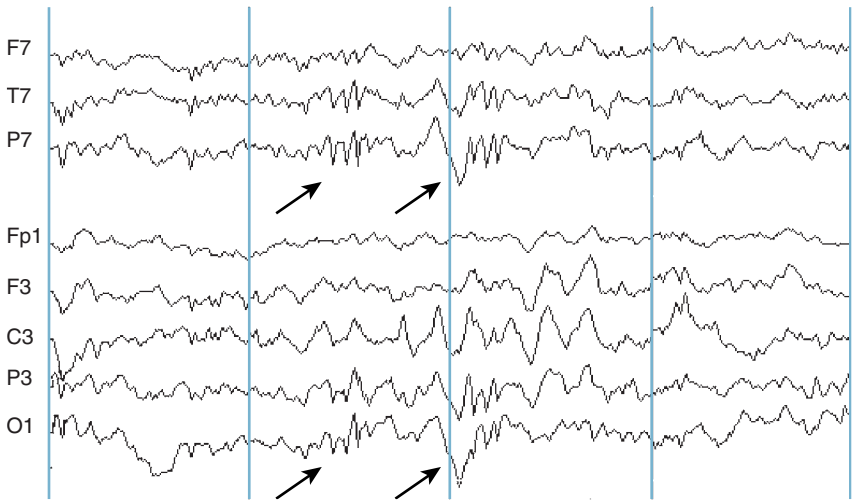

Figure 11-9 A close-up of 14 and 6 positive bursts is shown in a referential montage (arrows). Maximum positivity (indicated by downgoing deflections in a referential montage) of the 14-Hz bursts is seen in the left posterior quadrant (P7, T7, O1, and P3 electrodes).

Breach Rhythms

A breach rhythm results from a change in the transmission of EEG waves through the area of a skull defect, usually a postsurgical craniotomy site (see Figures 11-12 through 11-14). For reasons that are not fully understood, faster activity is transmitted preferentially through the region of skull defects, causing breach rhythms to have a sharpened appearance. Because the voltage asymmetries caused by breach rhythms could be hard

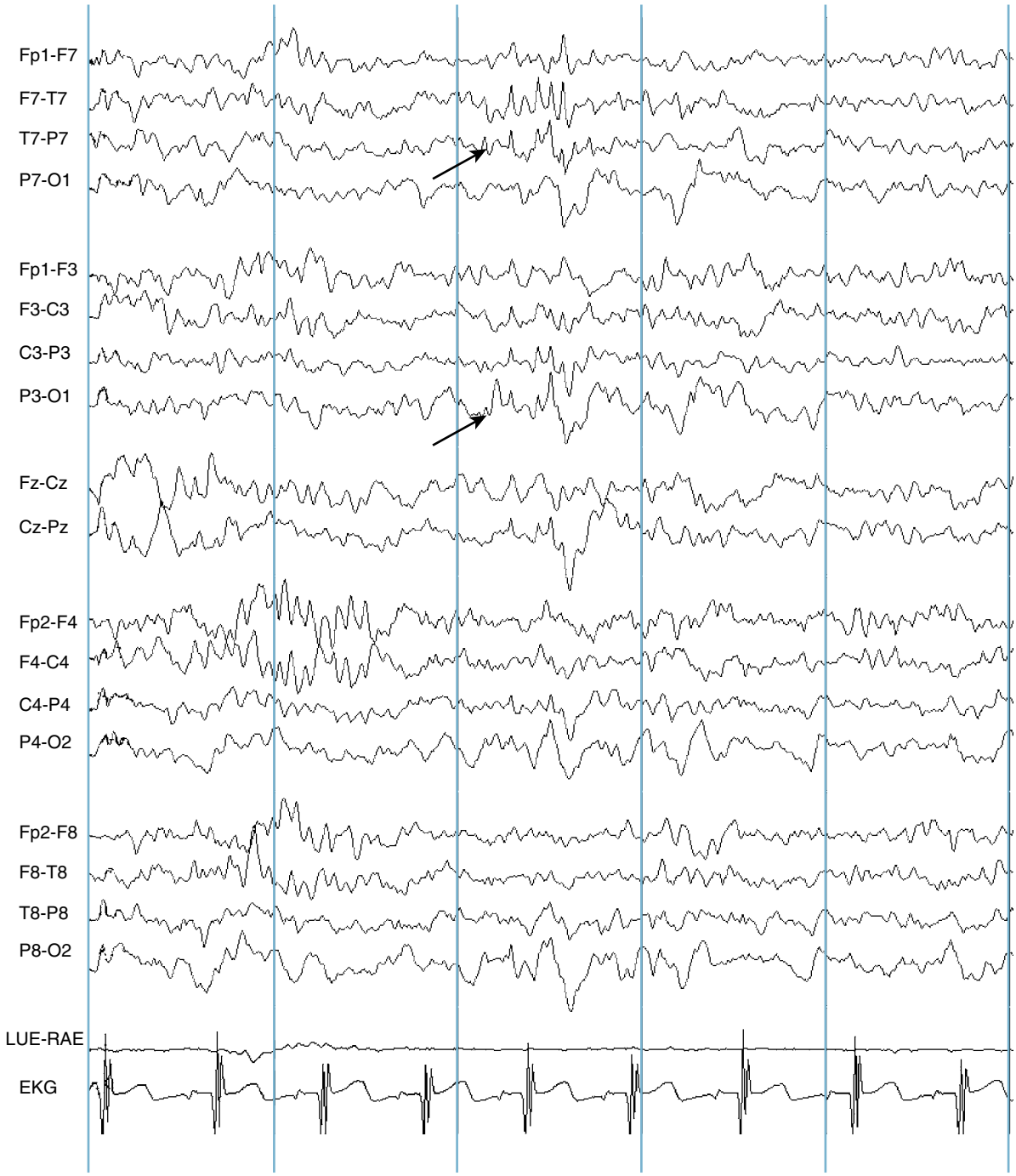

Figure 11-10 An example of 14- and 6-Hz positive bursts is shown in a bipolar montage (arrows). Note that the polarity and localization of the burst in this montage is somewhat ambiguous, appearing to be more anterior in the temporal chain than is actually the case. This appearance is due to the positive polarity of the bursts, which is more easily understood when they are displayed in a referential montage (see Figure 11-11).

to interpret without knowledge of the patient's skull defect, EEG technologists are asked to include information about craniotomy scars along with the clinical history. There is still some question as to whether breach rhythms are simply caused by a reduction in the skull's insulation effect at sites where it has been surgically disrupted or are instead caused by some change in the underlying cortex caused by the previous surgical procedure. Breach rhythms sometimes bear a resemblance to mu rhythms or wicket rhythms, and some feel that they represent overexpression of these natural rhythms through the skull defect. It is important to identify breach rhythms so as not to misinterpret any observed voltage asymmetry and to avoid mistaking fragments of the breach rhythm for spikes.

Rhythmic Temporal Theta Bursts of Drowsiness or Psychomotor Variant

Originally also referred to as rhythmic midtemporal-discharges (RMTD), rhythmic temporal theta bursts of drowsiness are also called psychomotor variant because

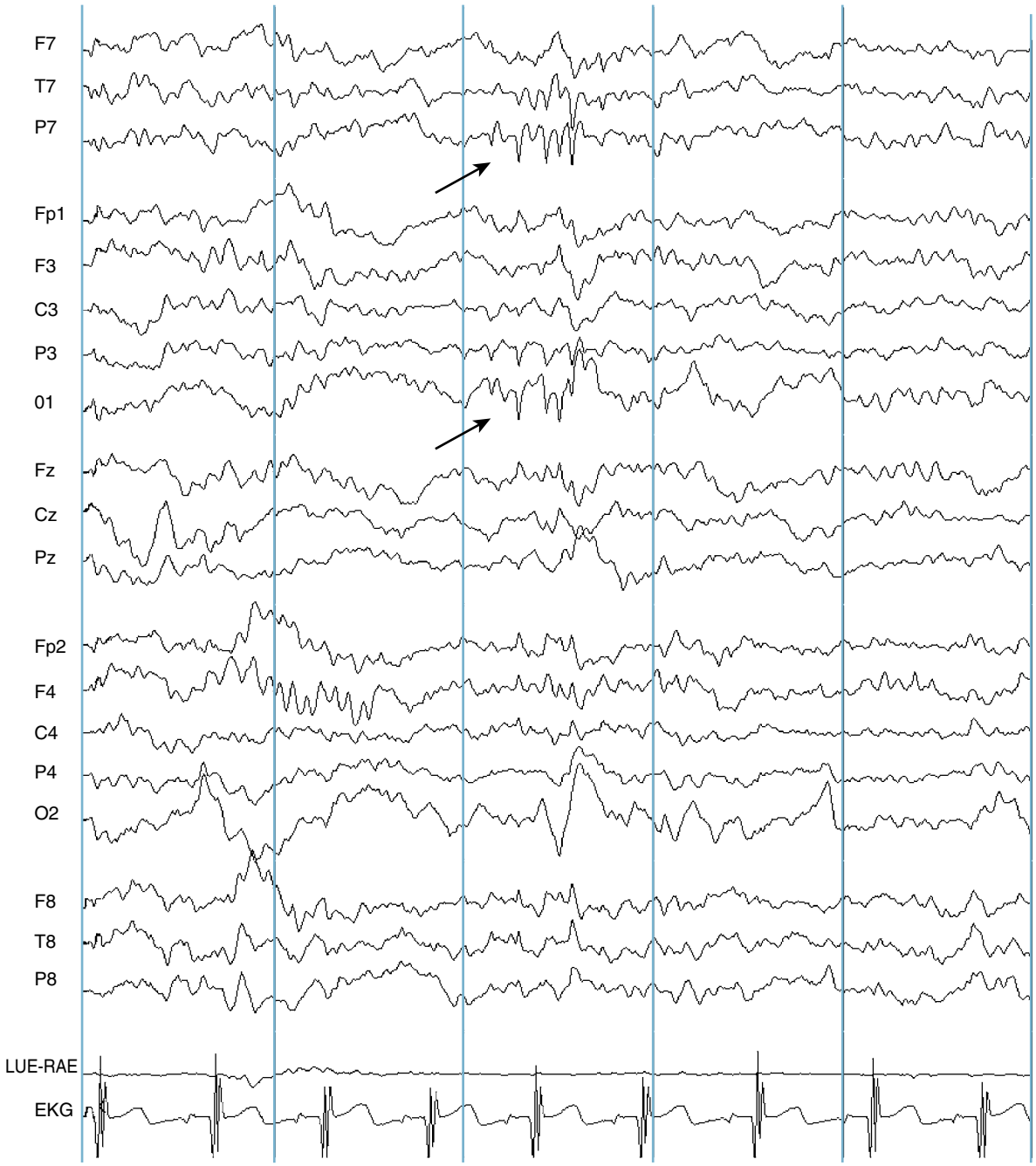

Figure 11-11 The same example of 14- and 6-Hz positive bursts from the previous figure is now displayed in a referential montage (arrows). The downgoing spikes in the posterior channels clarify the positive polarity of the bursts and the localization of their field to the left posterior quadrant.

of the discharges' resemblance to a temporal lobe seizure discharge. The use of the original term RMTD may persist because of the ease of using the abbreviation. Rhythmic temporal theta bursts can usually be easily distinguished from seizure discharges in that the frequency, amplitude, and morphology of the waveform do not vary throughout its course (see Figure 11-15). These theta bursts are seen in both children and adults and may be seen in either or both hemispheres. The discharges have a distinctive morphology with trains of waves with rounded tops and sharpened bottoms as seen in the figure. This variant is seen in drowsiness and light sleep and is not felt to be associated with epilepsy.

Six per Second Spike-Wave Complexes or Phantom Spike and Wave

Six per second spike-wave complexes are also referred to as "phantom spike wave" because of their short duration, usually 2 seconds or less. Phantom spike and wave is seen in wakefulness and mild drowsiness. The fact that it disappears with deeper sleep helps to distinguish it from epileptiform activity. These discharges

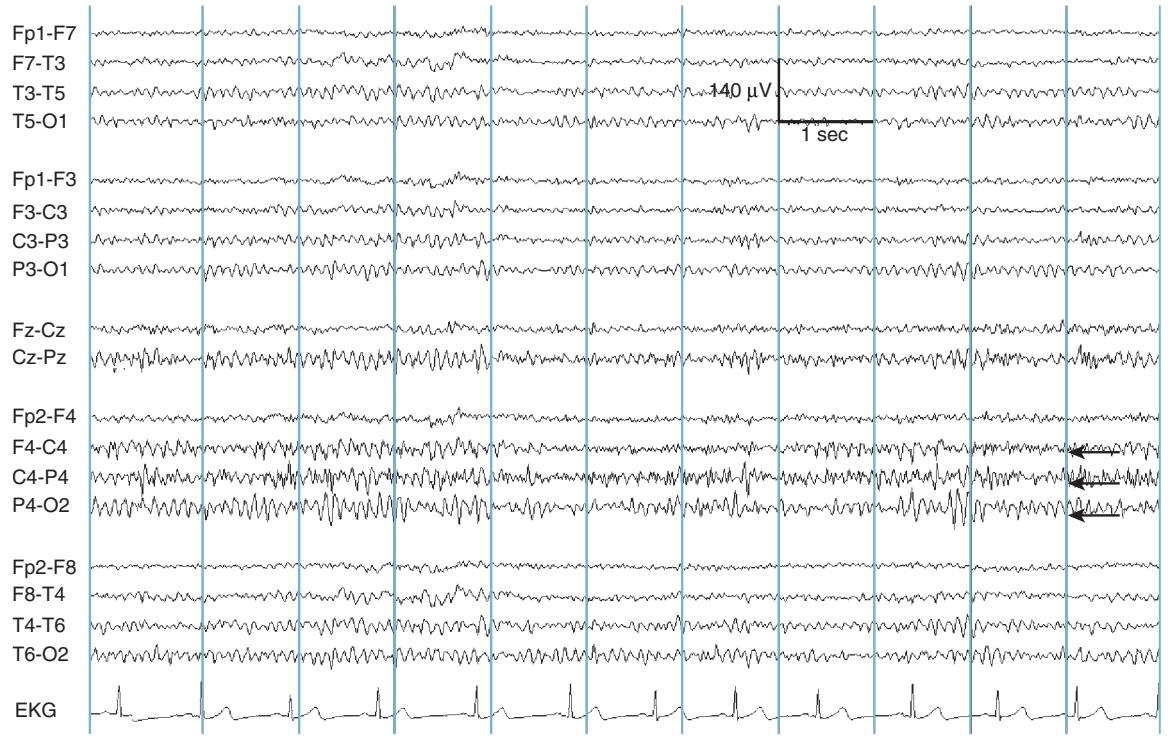

Figure 11-12 A breach rhythm is seen in the right parasagittal area (arrows) after a right-sided craniotomy. Note the higher voltage, sharpened rhythm in the F4-C4, C4-P4, and P4-O2 channels. *(Image courtesy of Dr. Edward Bromfield and Dr. Barbara Dwortesky.)*

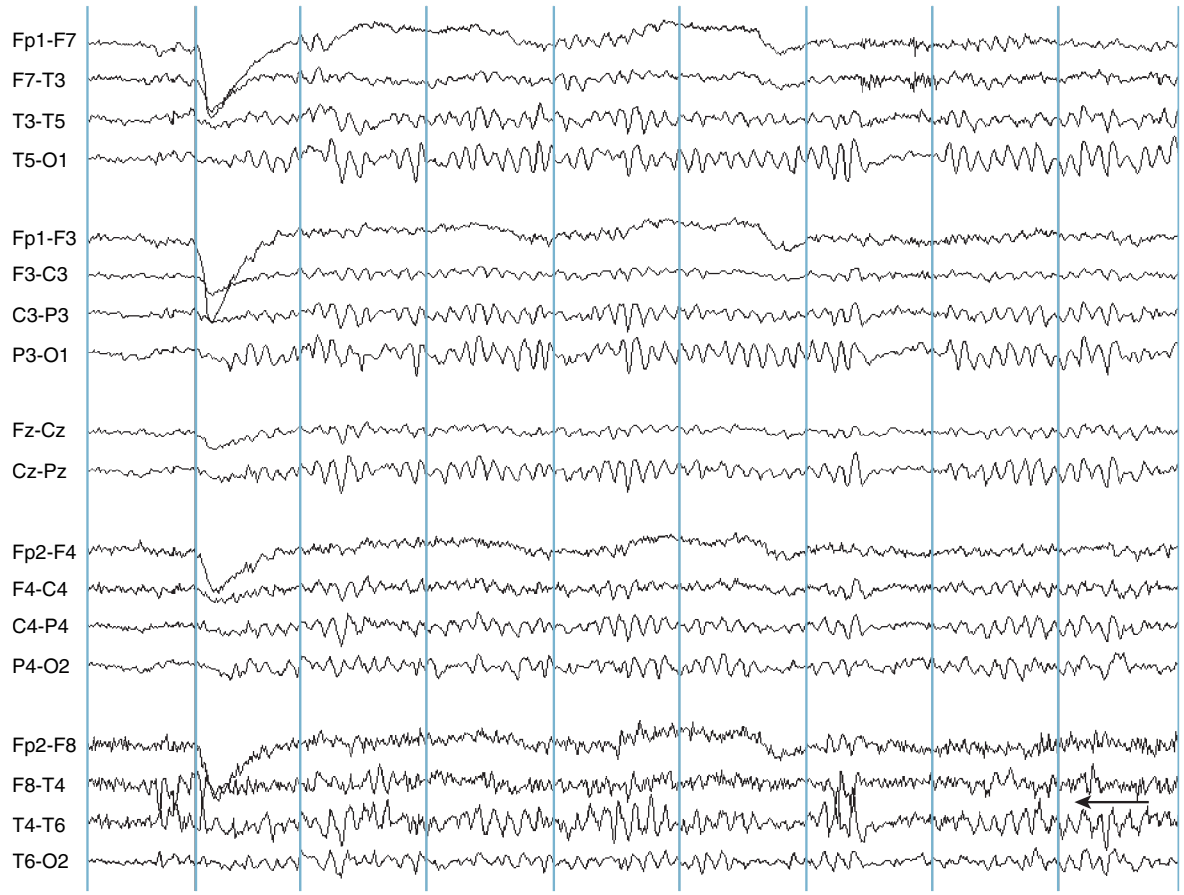

Figure 11-13 A breach rhythm is seen in the right midtemporal area (arrow) after a right temporal craniotomy. Note the arciform nature of the rhythm and compare with the homologous left-sided (T3) electrode. *(Image courtesy of Dr. Jong Woo Lee.)*

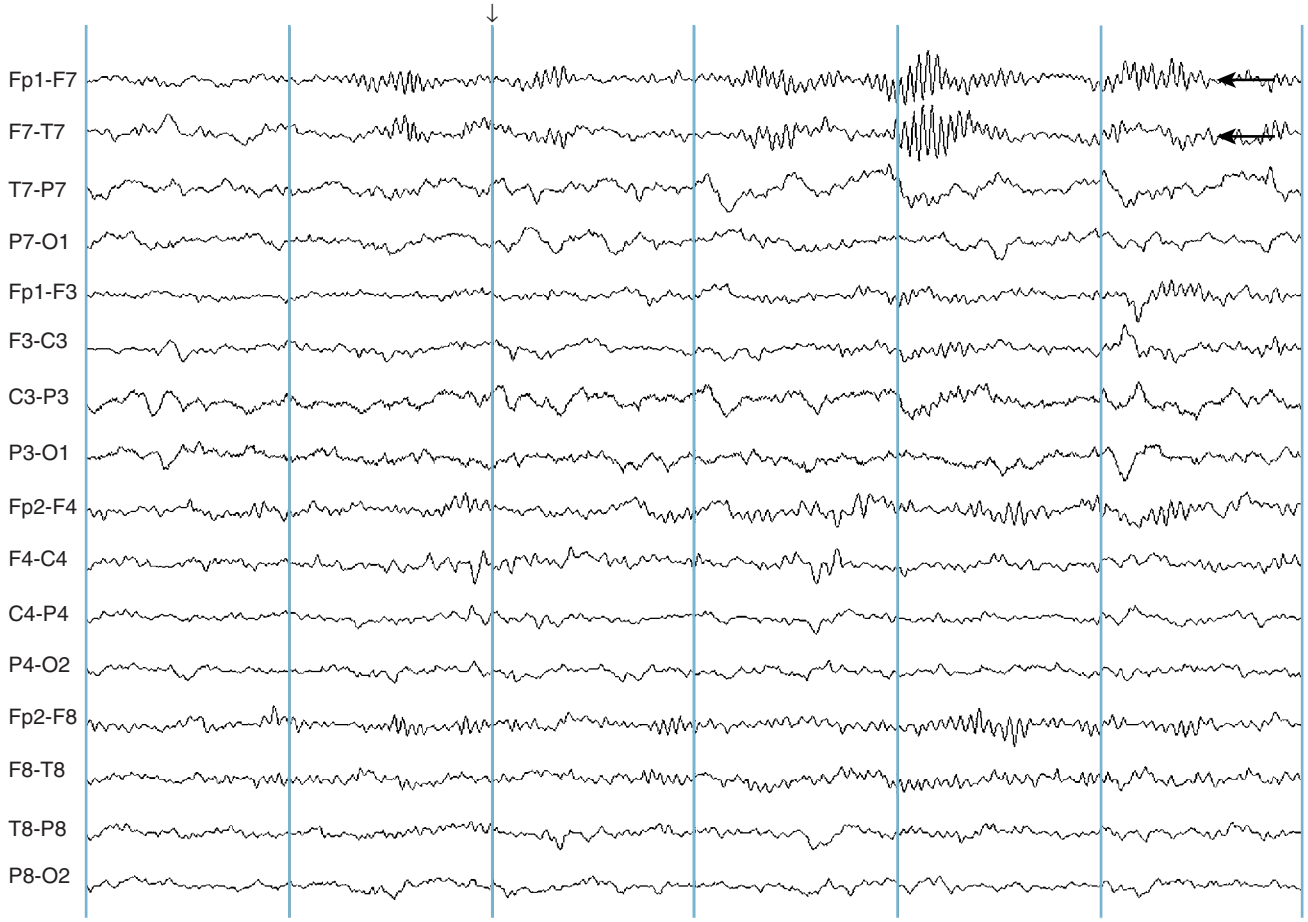

Figure 11-14 A breach rhythm is seen in the left anterior temporal area after a temporal lobectomy in a teenage boy (arrows). Compared with the previous examples, this rhythm has a more sinusoidal appearance.

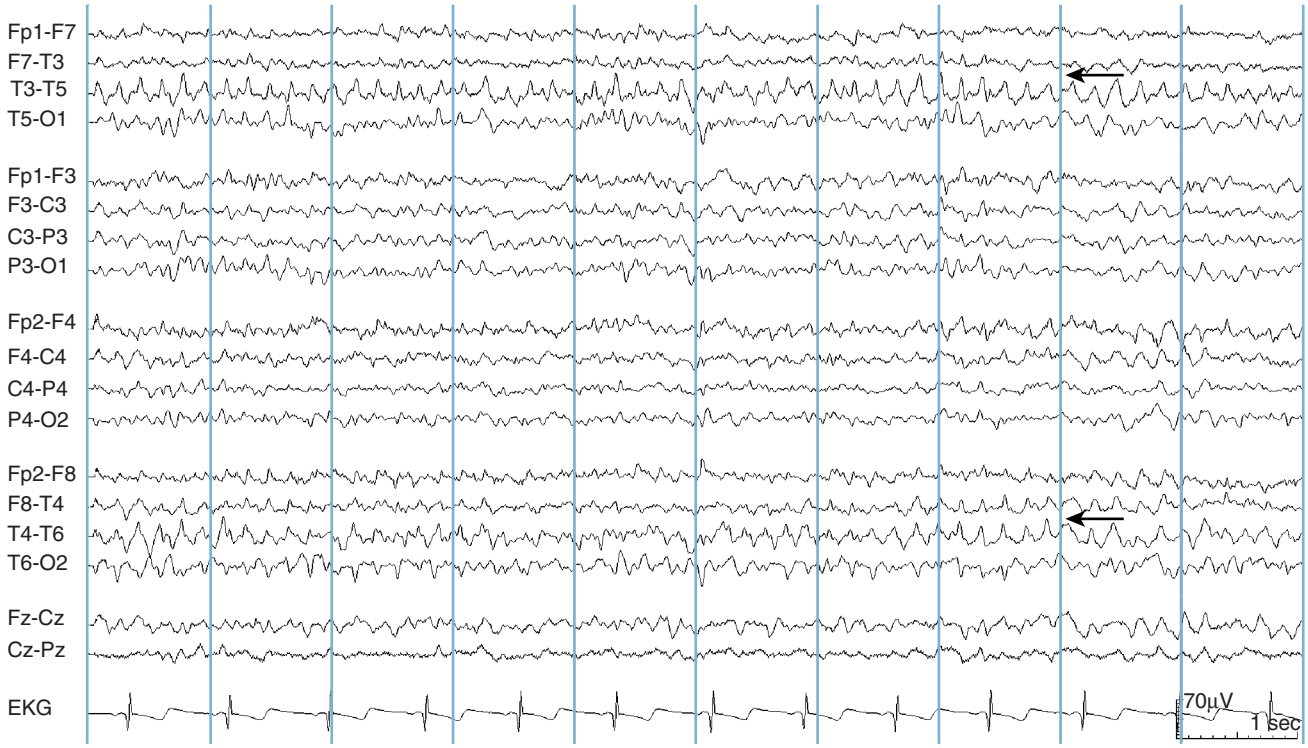

Figure 11-15 Rhythmic temporal theta bursts of drowsiness, also known as psychomotor variant or RMTD, is seen in each temporal area (arrows). The waves are sharp on one side and rounded or flat-topped on the other. The unchanging morphology and the constant frequency help to distinguish this from a seizure discharge—the "firing rate" is the same during the first second and the last second on the page.

are quick to appear and disappear, and the spike component of the spike and wave may only be intermittently evident.

In 1980, Hughes studied a large group of individuals with six per second spike-wave complexes. In this group, patients with certain characteristics, in particular, those with high amplitude, frontal-maximum discharges, were more likely to have epilepsy. Individuals with lower voltage, posterior discharges were more likely to have been referred for "neurovegetative symptoms" such as headache, dizziness, and vertigo or other psychological complaints as opposed to seizures. He distinguished the two groups using the mnemonics WHAM (Waking record, High in amplitude, Anterior in location and especially in Males) and FOLD (Female whose pattern has an Occipital emphasis, Low in amplitude, and seen in the Drowsy record; see Figure 11-16).

GENERAL INDICATORS OF NORMAL VARIANTS

Especially for the beginning EEG reader, memorizing the various characteristics of the normal variants just described may seem a daunting task. Even if all of the normal variant patterns have not been committed to memory, certain features of apparent epileptiform discharges should prompt the reader to question the possibility of a normal variant.

High Frequencies

Many of the normal variants have fast firing rates. Whereas "classic" generalized spike wave discharges have a firing rate of 3 Hz and even "fast" spike-wave discharges have a firing rate of 4 to 5 Hz, many of the normal variants have firing rates of 6 Hz or above. Mu rhythms, wicket rhythms,

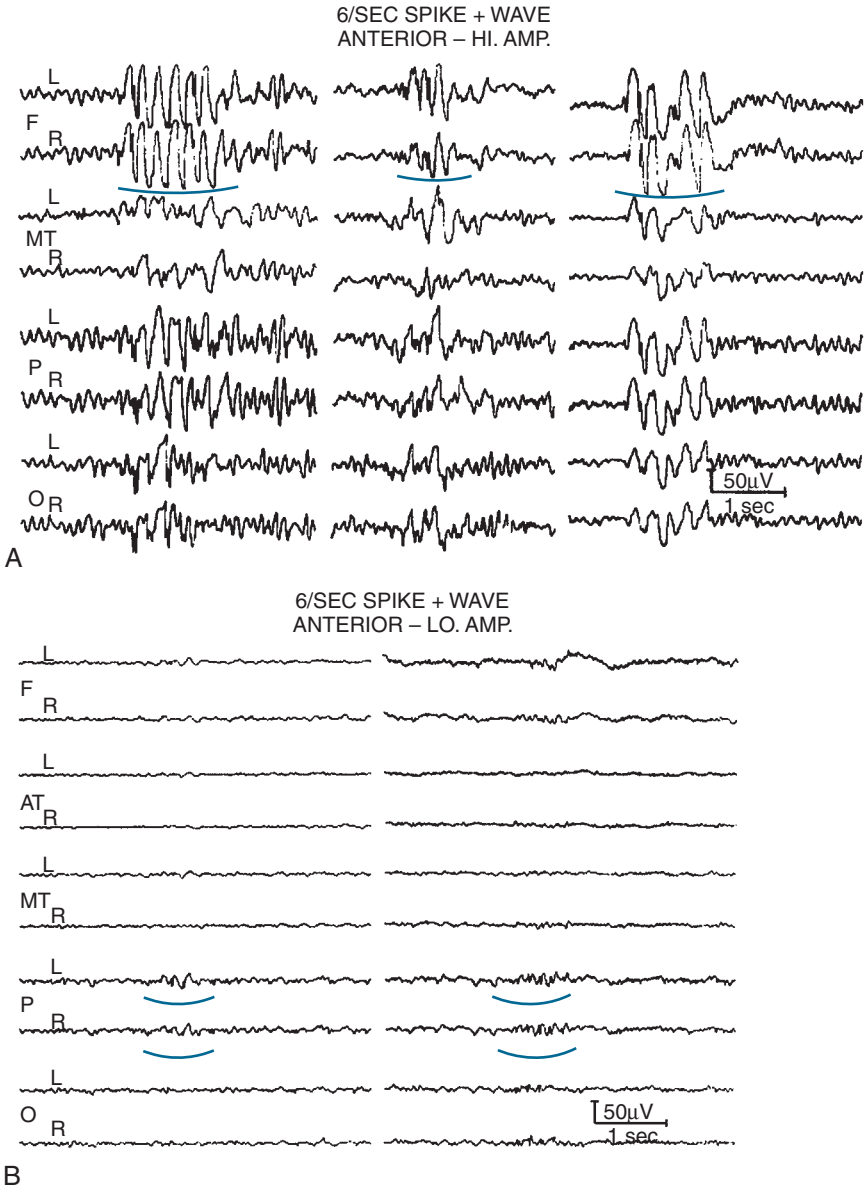

Figure 11-16 **(A)** Examples of frontal-maximum six per second spike and wave recorded from three separate patients (WHAM version) with higher amplitudes and anterior localization. **(B)** Examples of the occipital-maximum, lower voltage six per second spike and wave (FOLD version). *(Adapted from Hughes JR: Two forms of the 6/sec spike and wave complex.* Electroencephalogr Clin Neurophysiol *48:535–550, 1980.)*

14 and 6 positive bursts, psychomotor variant, and phantom spike and wave all have firing rates of 6 Hz or higher. Therefore, high-frequency spiking should cause the reader to consider whether the discharge may fit into the group of normal variants. True epileptiform spike-wave discharges occasionally have fast firing rates, but more moderate firing frequencies are more common.

Monomorphic Rhythms

The normal variant discharges tend to consist of repetitive waves of similar shape and wavelength (breadth). Because wavelength is directly proportional to frequency, truly monomorphic waves do not vary in frequency. This feature of normal variants helps to distinguish them from seizure discharges and sometimes even from epileptiform activity. A run of the psychomotor variant pattern can be distinguished from seizure activity because it does not vary in frequency even during lengthy trains. In contrast, a hallmark feature of a seizure discharge is a speeding up or slowing down in firing frequency throughout its course. Even interictal polyspike discharges tend to slow down slightly in frequency over the course of a burst, whereas 14- and 6-Hz positive bursts do not. The reader can confirm this by showing that the width (wavelength) of the first wave of a 14-Hz positive burst is the same as the last.

Disappearance in Deeper Sleep

The normal variants described earlier appear in wakefulness or drowsiness and light sleep and tend to disappear with deepening sleep. In contrast, epileptiform abnormalities often increase with deeper sleep. Disappearance of a possible epileptiform discharge with deepening sleep should cause the reader to consider whether the discharge in question matches any of the normal variant patterns.

OTHER NORMAL VARIANTS

Posterior Slow Waves of Youth

Posterior slow waves of youth are theta and delta waves that intermix with the posterior rhythm in younger subjects (see Figures 11-17 and 11-18). They are confined to the occipital areas and suppress with

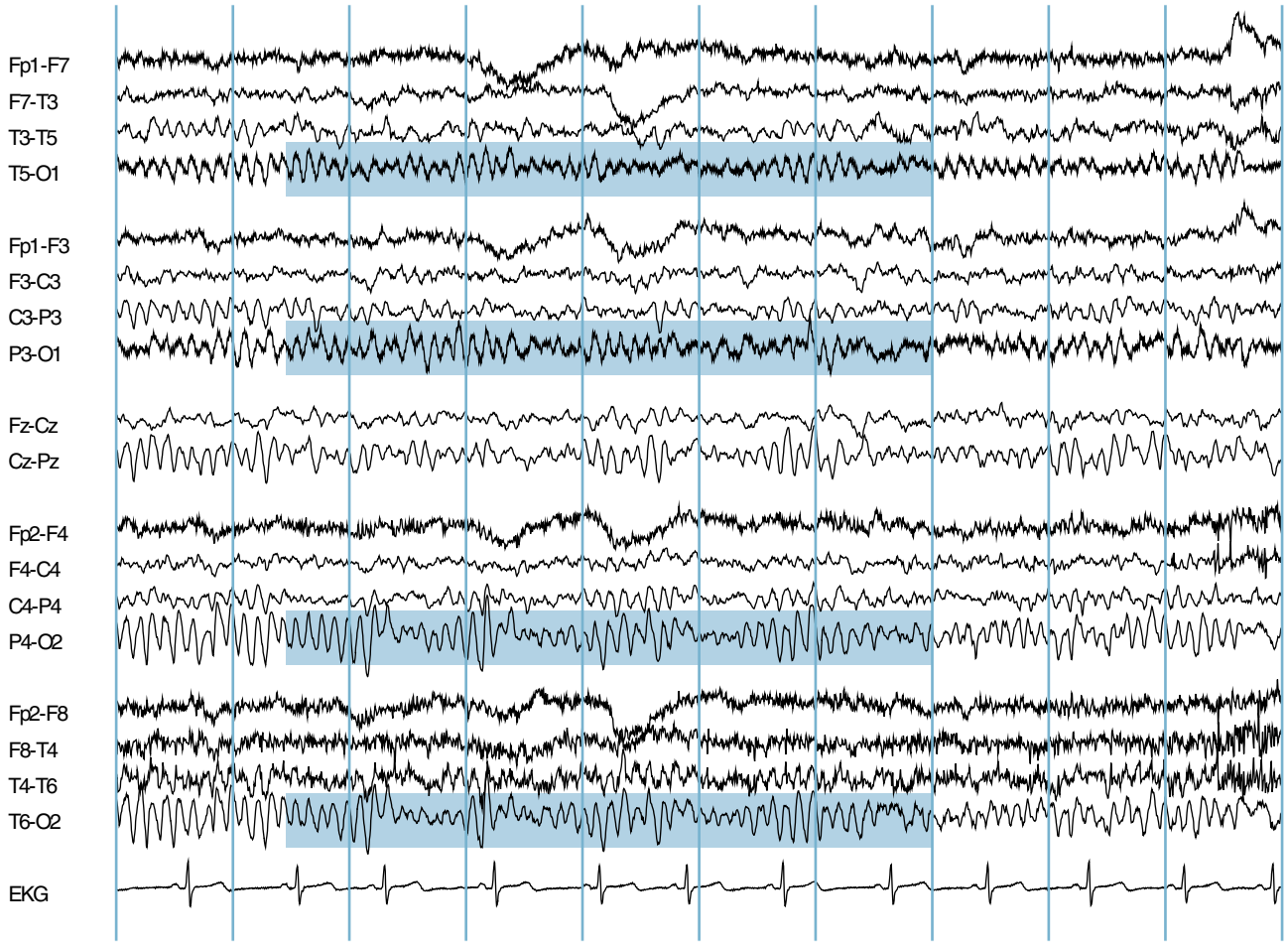

Figure 11-17 Posterior slow waves of youth are seen in the occipital channels (shaded areas). Note that the posterior rhythm does not have a flat baseline but instead rides up and down on low-voltage delta and theta waves. This degree of posterior slowing during wakefulness is commonplace from adolescence into the teenage years.

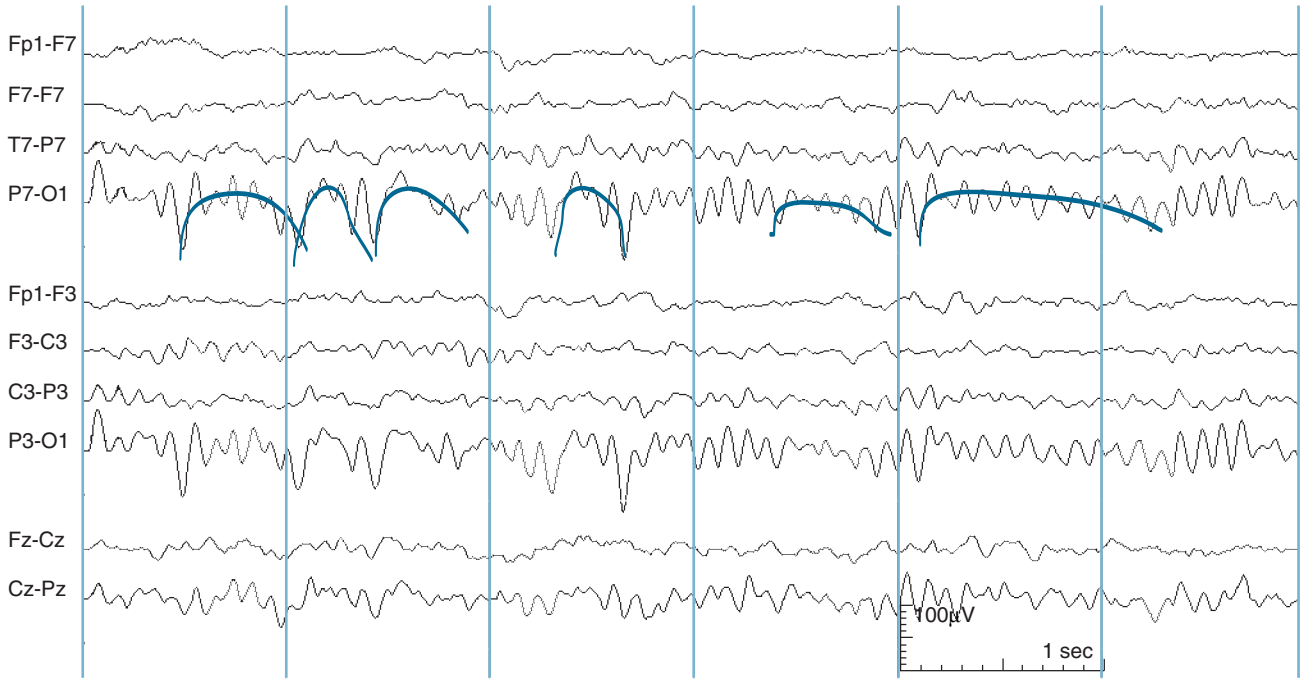

Figure 11-18 Posterior slow waves of youth are seen prominently in the P7-O1 and P3-O1 channels. Some of the intermixed delta and theta waves are marked with pencil in the P7-O1 channel to highlight the shifting baseline of the posterior rhythm caused by the posterior slow waves.

eye opening along with the posterior rhythm. Typically, these posterior slow waves briefly interrupt sustained runs of the posterior rhythm, sometimes making it difficult to count. Posterior slow waves of youth appear after age 7 years and are commonly seen up to the middle of the second decade. They become distinctly less common during the late teenage years and are only rarely seen after age 20, by which time posterior slow waves may be abnormal. Posterior slow waves of youth usually occur singly and do not exceed the amplitude of the posterior rhythm by more than 50%. Amplitude asymmetries are not uncommon with these waves and are not necessarily abnormal.

K-complexes and Related "Evoked Responses"

The k-complex is a normal element of sleep. K-complexes may occur either spontaneously or in response to an outside stimulus such as a noise in the environment. Occasionally, the bursting characteristic of a k-complex could be mistaken for a generalized spike-wave discharge because of embedded sharpened features. Occasional intermittent increases in voltage during drowsiness or sleep caused by an environmental stimulus may represent a type of evoked response; these may, indeed, represent brief arousal rhythms. Figure 11-19 shows the EEG response of a 33-year-old woman to a noise in the environment during drowsiness. The EEG technologist should note the relationship of such bursts to environmental stimuli, as appropriate, to aid in interpretation.

Exaggerated Hyperventilation Response

Hyperventilation responses are typically more dramatic in children compared with adults. The hyperventilation response is also easier to elicit when blood sugar is lower. In the absence of dramatic asymmetrical findings or clear epileptiform discharges, there is no defined upper limit of voltage for a "normal" hyperventilation response (see Figure 11-20) and hyperventilation hypersynchronies should not be considered abnormal based solely on voltage criteria at any age.

Hypnogogic and Hypnopompic Hypersynchronies

Hypnogogic (on falling asleep) and hypnopompic (on arousal) hypersynchronies are highly rhythmic, medium- or high-voltage waves seen diffusely across the EEG either on transition into sleep or on arousal. At times, these hypersynchronies can be dramatic and may potentially be mistaken for epileptiform activity or seizure activity (see Figure 11-21). Such hypersynchronies are usually easily distinguished from seizure activity on the basis of their occurrence at the time of sleep transitions and their monorhythmic nature: the frequency of these rhythmic waves holds steady throughout their course, which helps distinguish them from seizure activity.

Photomyoclonic Response

The photomyoclonic response is the result of a reactive twitching of the facial muscles to strobe stimulation (see Figure 11-22). This results in bursts of muscle

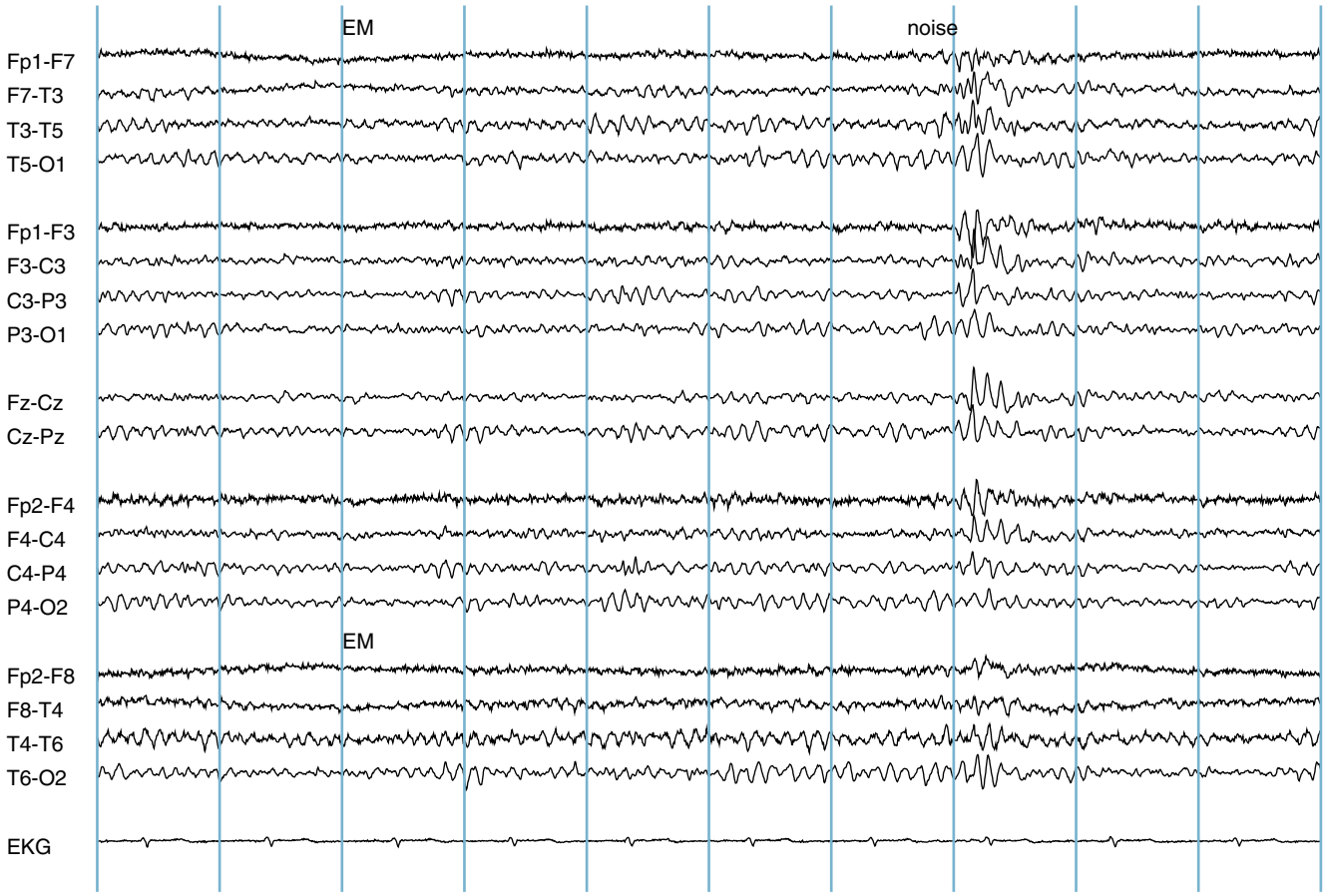

Figure 11-19 A brief increase in EEG voltage occurs in a 33-year-old woman in response to a noise in the EEG laboratory. Lateral eye movements (EM), indicated by a slight approximation of the Fp1-F7 and F7-T3 channels and a slight bowing apart of the Fp2-F8 and F8-T4 channels, and intermittent dropout of the posterior rhythm signal light drowsiness. Such brief increases in voltage from an outside stimulus may represent a brief arousal rhythm.

artifact occurring in tandem with the strobe flash. In some instances, the muscle potential bursts could be mistaken for spike-wave discharges. When the muscle spikes and eyeblink artifacts are visually removed, no other abnormalities are seen. The photomyoclonic response is not associated with seizures and is therefore considered a normal variant.

Spiky Alpha

The posterior rhythm is usually a sinusoidal rhythm, but a variant morphology, "spiky alpha," may be seen in a minority of patients. Spiky alpha waves are rounded on one side and spiky on the other (see Figure 11-23), similar to the arciform pattern seen with mu and similar rhythms. Spiky alpha variant is usually easy to recognize as such; the danger comes in finding fragments of spiky alpha and mistaking them for occipital spikes.

Slow Alpha Variant and Fast Alpha Variant

The slow alpha variant is a variant of the posterior rhythm in which a subharmonic frequency of the posterior rhythm (half the frequency) is either superimposed on or replaces the posterior rhythm itself. Figure 11-24 shows a patient with a 9-Hz posterior rhythm. Later in the page, a prominent 4.5-Hz rhythm is seen. Replacement of the posterior rhythm with a subharmonic frequency is considered a normal variant and should not be considered a slow-wave abnormality. More rarely, the posterior rhythm may be replaced with a higher harmonic frequency, typically a doubling of the fundamental posterior rhythm. This phenomenon is referred to as *fast alpha variant* (see Figure 11-25).

Cascading Vertex Waves

Vertex waves during Stage II sleep typically appear in a periodically repetitive fashion, often in conjunction with a spindle. In some patients, highly repetitive or "cascading" vertex waves may be seen for periods of time without pauses (see Figure 11-26). Although their appearance may be dramatic, this pattern can be considered a normal variant of vertex wave expression.

Spindle Fragments and Posterior Rhythm Fragments

Sleep spindle rhythms typically appear in runs lasting from one to several seconds. Occasionally, a fragment of this normal waveform can appear, giving a misleading impression. Figure 11-27 shows two apparent sharp

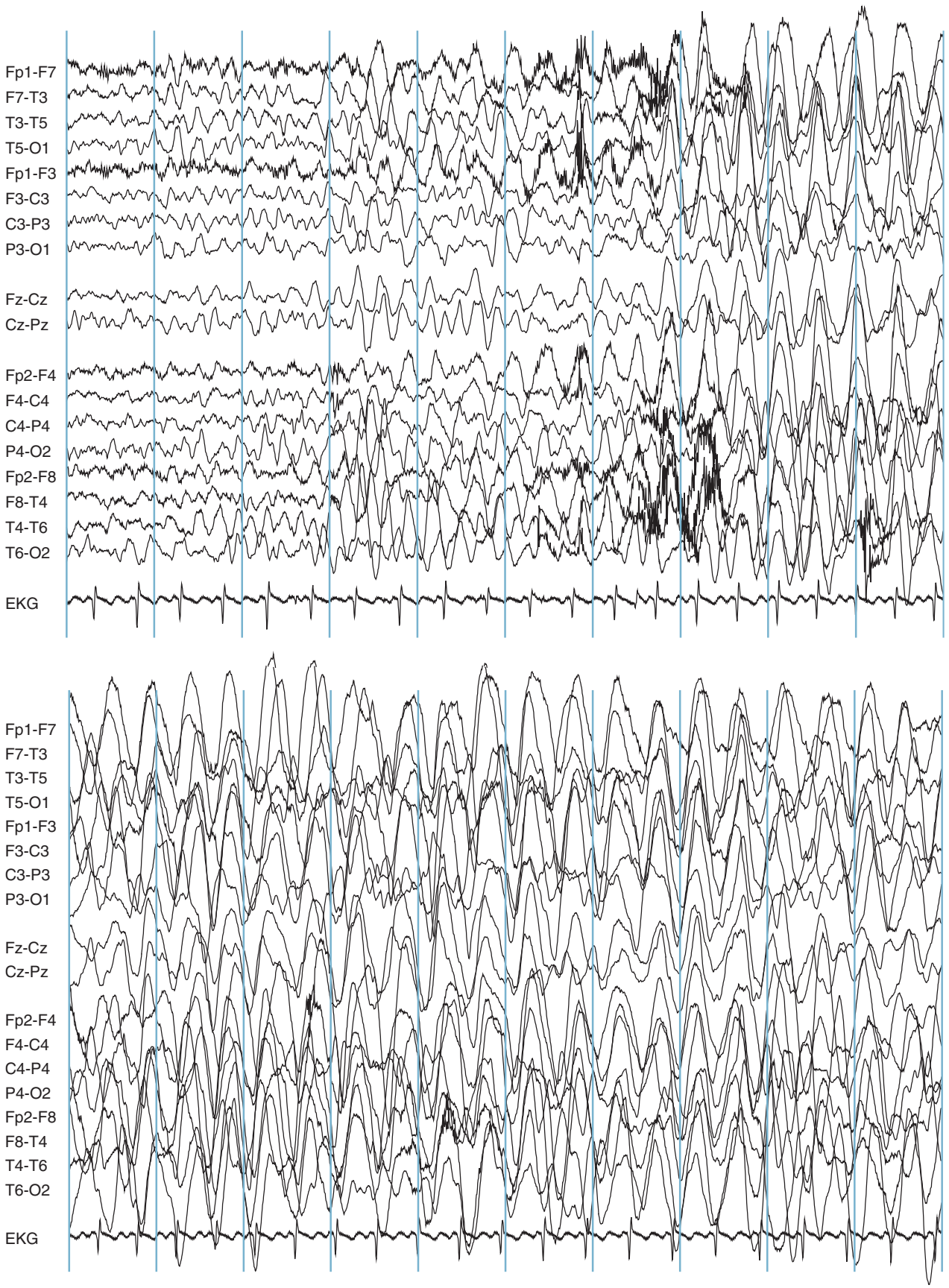

Figure 11-20 Even in patients who do not have epilepsy, the hyperventilation response can be dramatic, as it is in this young patient. In the absence of definite epileptiform features, very high-voltage hyperventilation responses should not be considered abnormal.

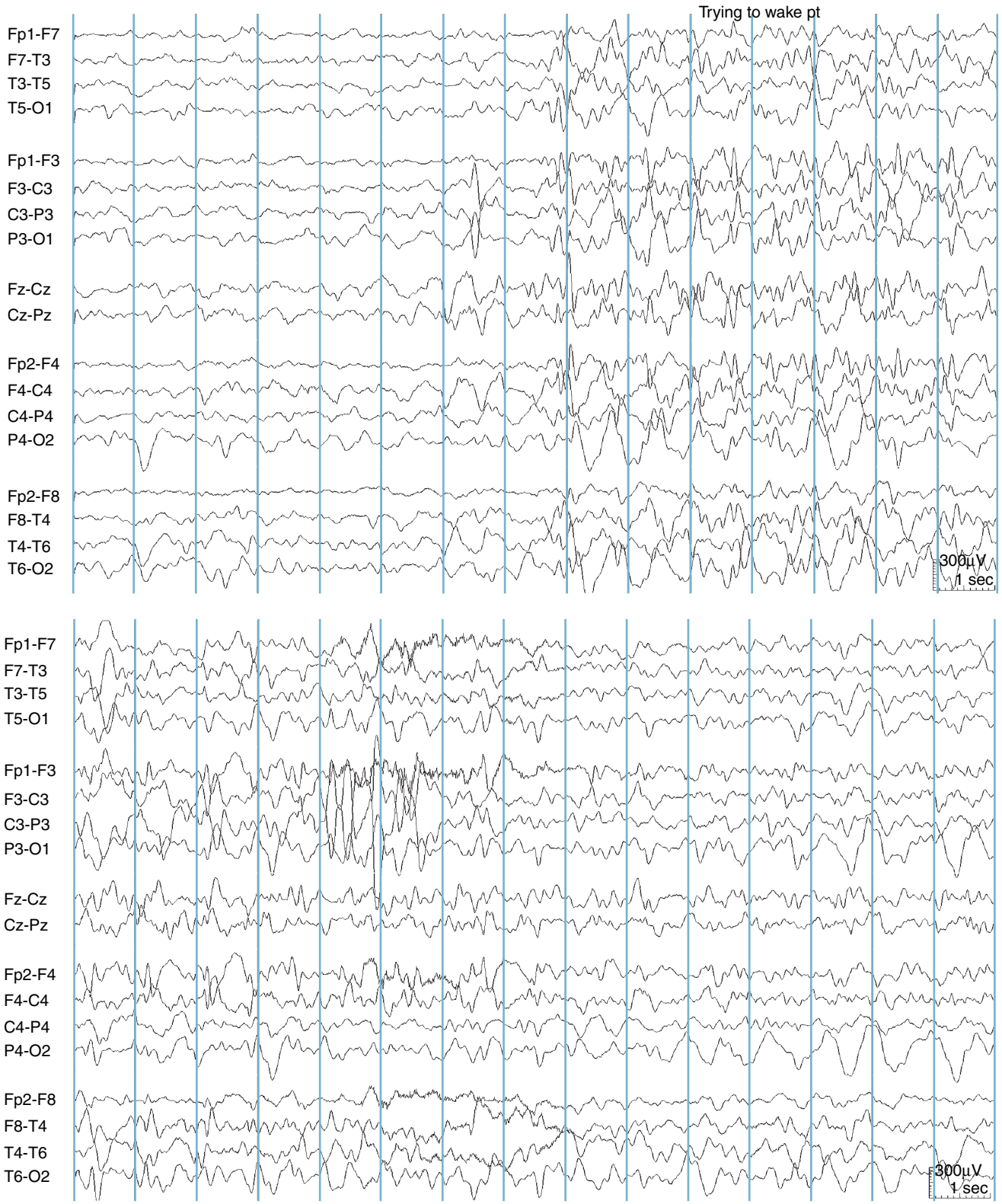

Figure 11-21 This example of a hypnopompic hypersynchrony (hypersynchrony occurring on arousal) occurs out of Stage II sleep when the technologist awakens the patient. This particular pattern consists of a mixture of rhythmic, high-voltage 1-Hz delta with a lower voltage 6-Hz theta rhythm. The fact that these frequencies do not evolve helps to exclude a seizure discharge. Some of the high-voltage deflections seen in the C3 electrode are probably due to a poor electrode contact.

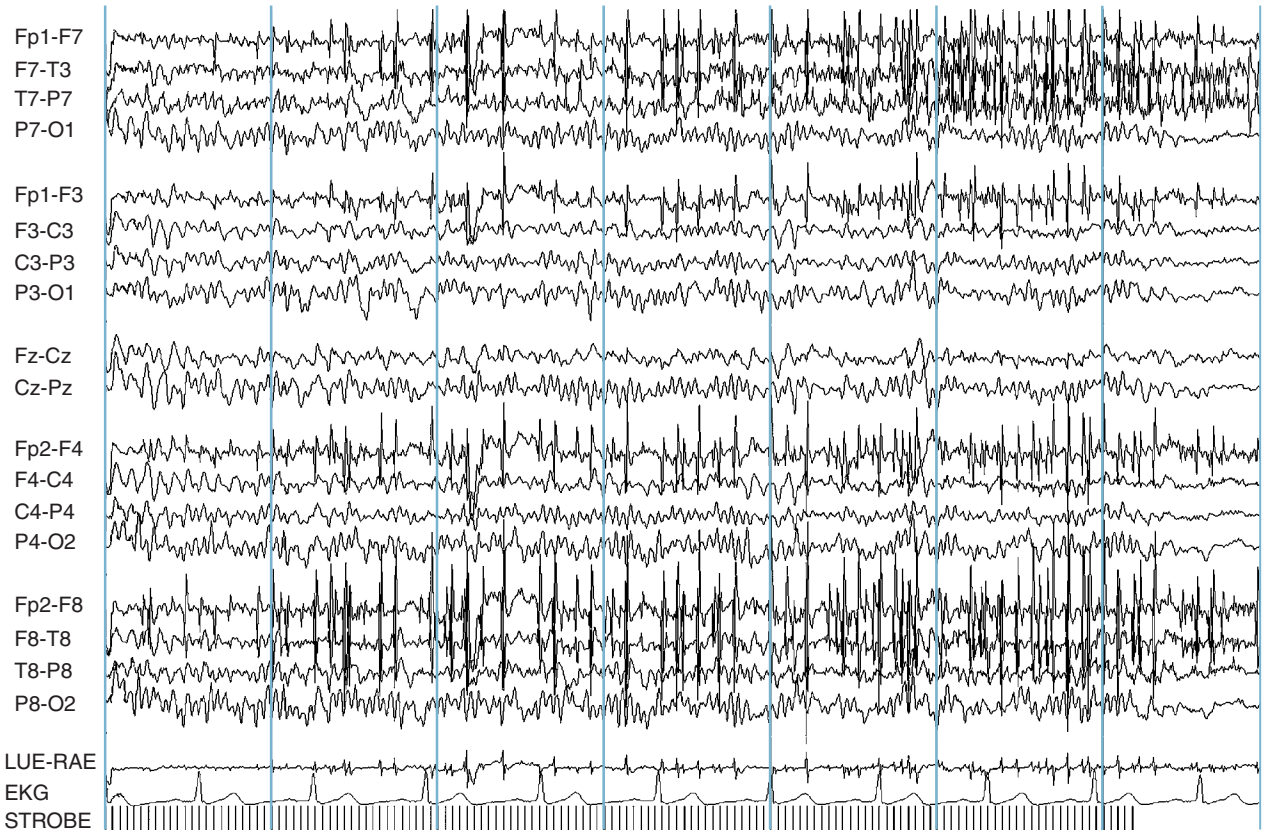

Figure 11-22 Repetitive muscle spikes are seen in the frontal areas during strobe stimulation. These artifactual spikes should not be mistaken for spike-wave discharges. The photomyoclonic response is seen more commonly in subjects with increased tension or anxiety. Visual observation of the patient shows that it is caused by rhythmic eye-twitching/blinking in time with the strobe. The timing of the strobe flashes is shown by the vertical lines in the bottom channel. A normal photic driving response is well seen in the channels that include the O1 and O2 electrodes.

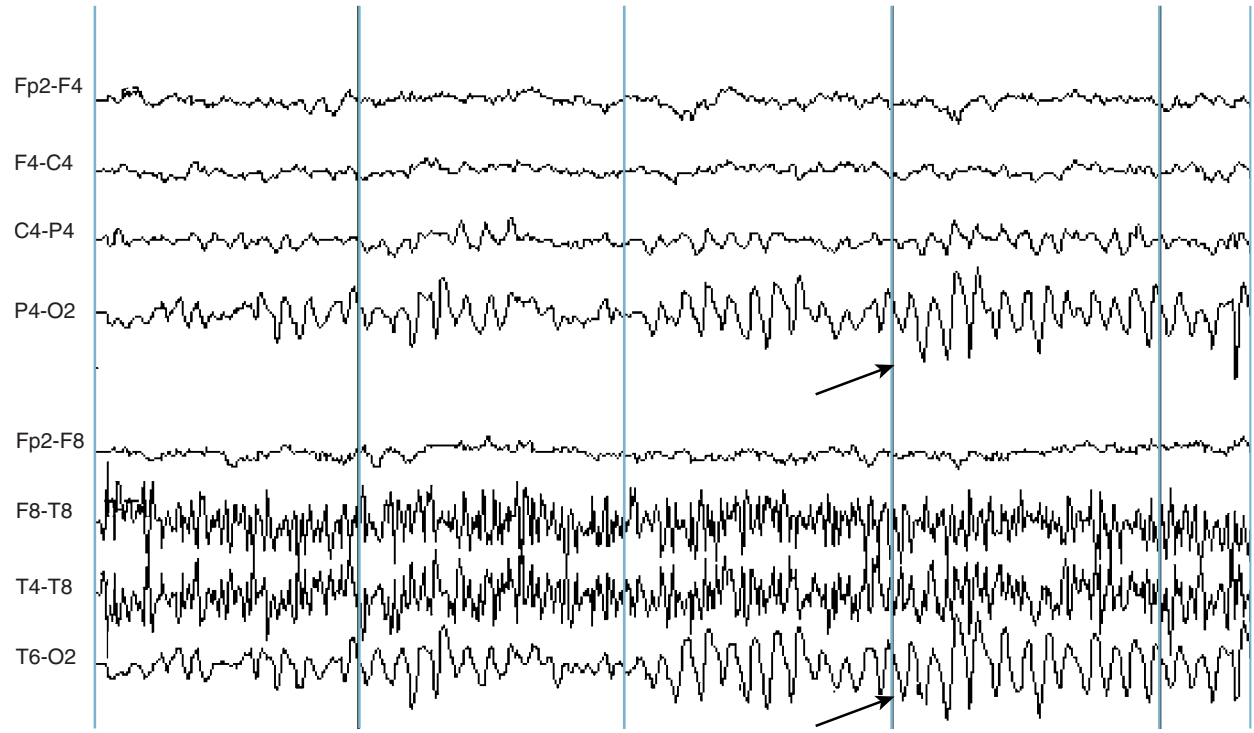

Figure 11-23 The posterior rhythm may become sharpened on one or both sides in some patients (arrows). This normal variant of the posterior rhythm is called *spiky alpha variant*.

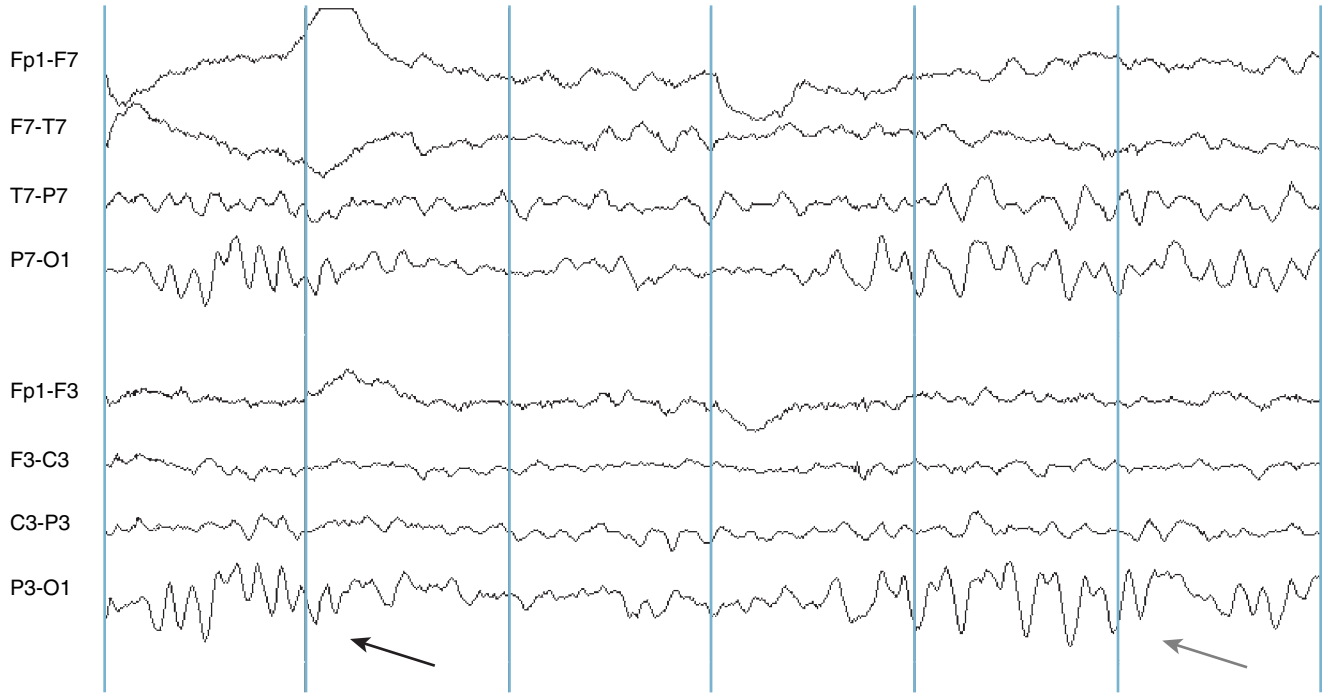

Figure 11-24 This patient has a fundamental posterior rhythm of 10 Hz (black arrow). At times a prominent and higher voltage 5-Hz rhythm is seen in the same region (gray arrow), representing a subharmonic of the fundamental 10-Hz rhythm. In this example, both the 5-Hz and the 10-Hz rhythm (in the form of notching of the 5-Hz rhythm) can be seen at the same time. In other patients, a pure subharmonic of the fundamental rhythm can be seen without notching (e.g., a pure 5-Hz rhythm taking the place of the posterior rhythm). In such cases, the presence of slow alpha variant can be confirmed by finding the fundamental posterior rhythm frequency elsewhere in the record, which should be a higher multiple of the slower frequency.

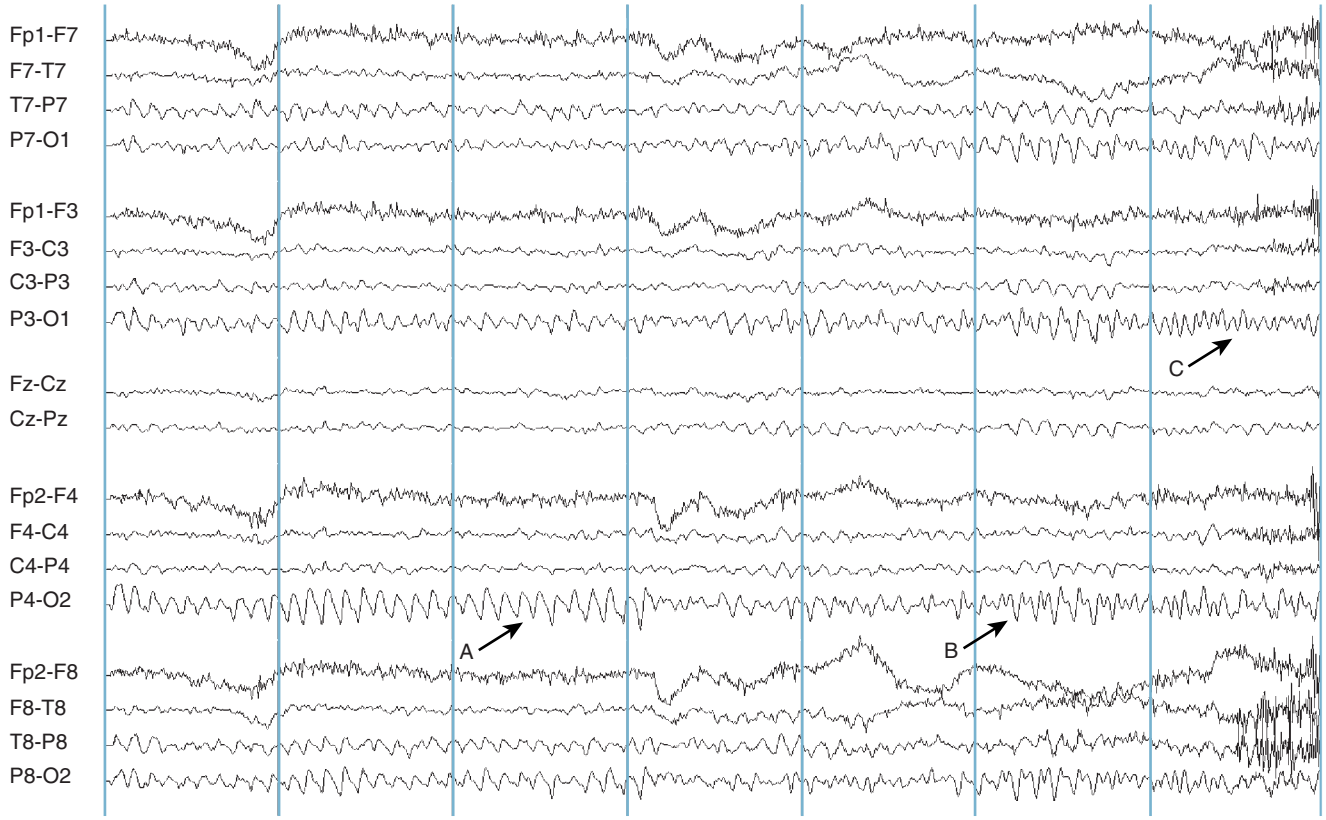

Figure 11-25 **(A)** This patient's fundamental posterior rhythm frequency is well seen. **(B)** A combination of the fundamental posterior rhythm and it first harmonic frequency (double the fundamental frequency) is seen in the form of a "notched" version of the posterior rhythm. **(C)** A pure version of the harmonic frequency, the "fast alpha variant."

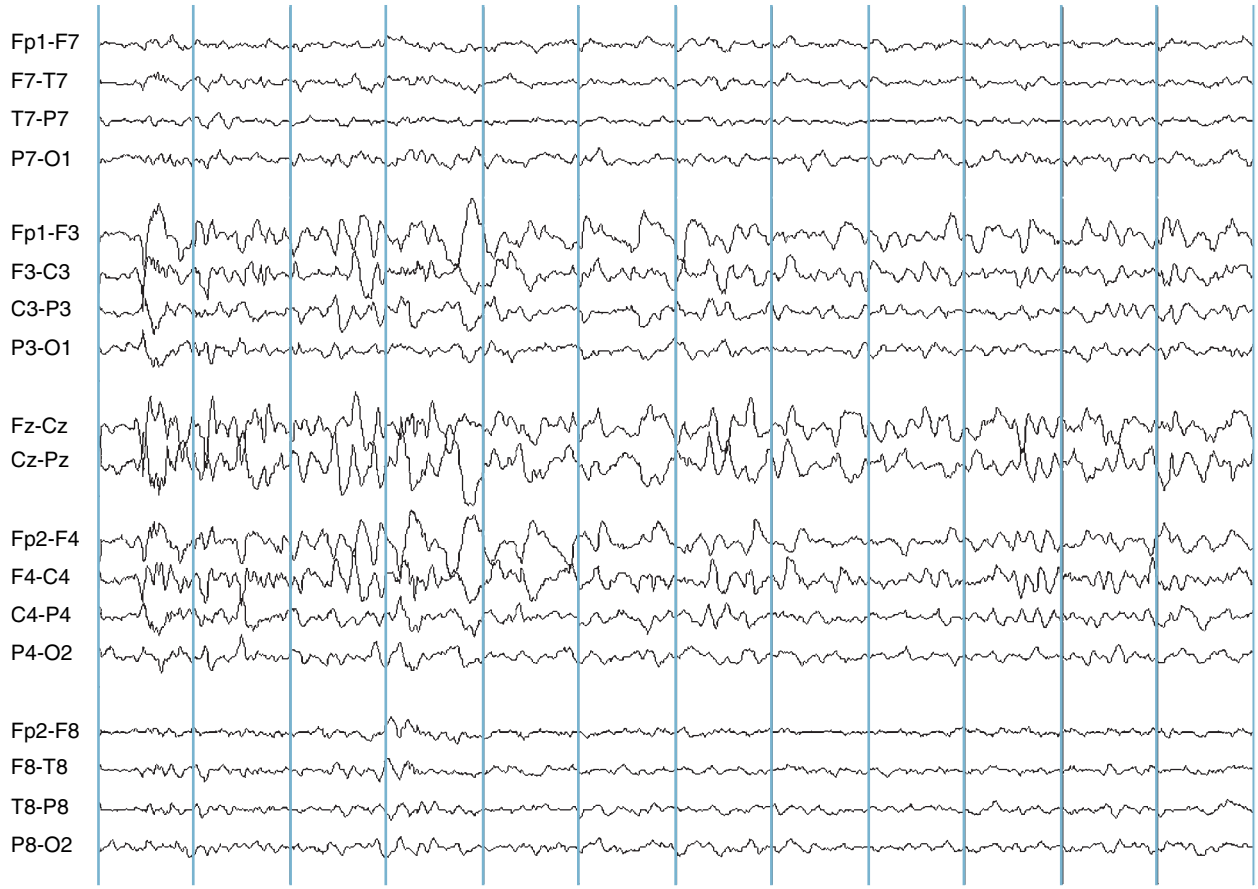

Figure 11-26 Although vertex waves usually occur in a repetitive, on-and-off pattern, less commonly they may be seen as relatively continuously appearing discharges. The maxima of these waves at Cz, C3, and C4 and the relative exclusion of the waves' field from the temporal areas help to identify them as vertex waves of sleep, despite their repetitive nature.

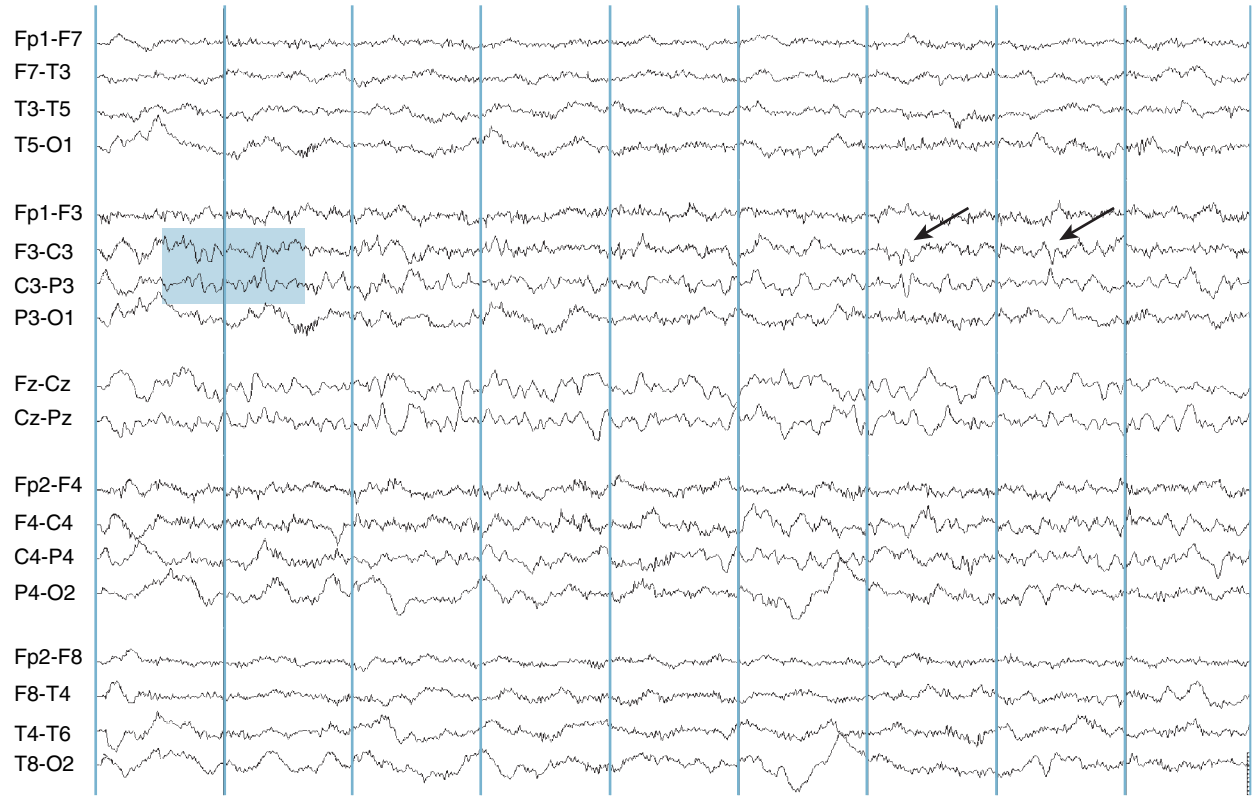

Figure 11-27 Two possible low-voltage spikes are noted in the left central region (arrows). The possibility that these represent low-voltage spikes can be excluded after it is recognized that they have the exact same morphology as the spindle wave seen at the beginning of the page (shaded area) and are examples of spindle fragments.

waves in the left central area. In fact, these are fragments of a sleep spindle, seen in a more typical form at the beginning of the page. Occasionally, fragments of the posterior rhythm may also appear singly, suggesting a single sharp wave (see Figure 11-28). When the morphology of the suspected wave fragment can be seen to match perfectly with previous, easily recognizable examples of the posterior rhythm, the benign nature of the wave can be confirmed. Similar phenomena involving wave fragments of mu rhythms and wicket rhythms are discussed earlier in the chapter.

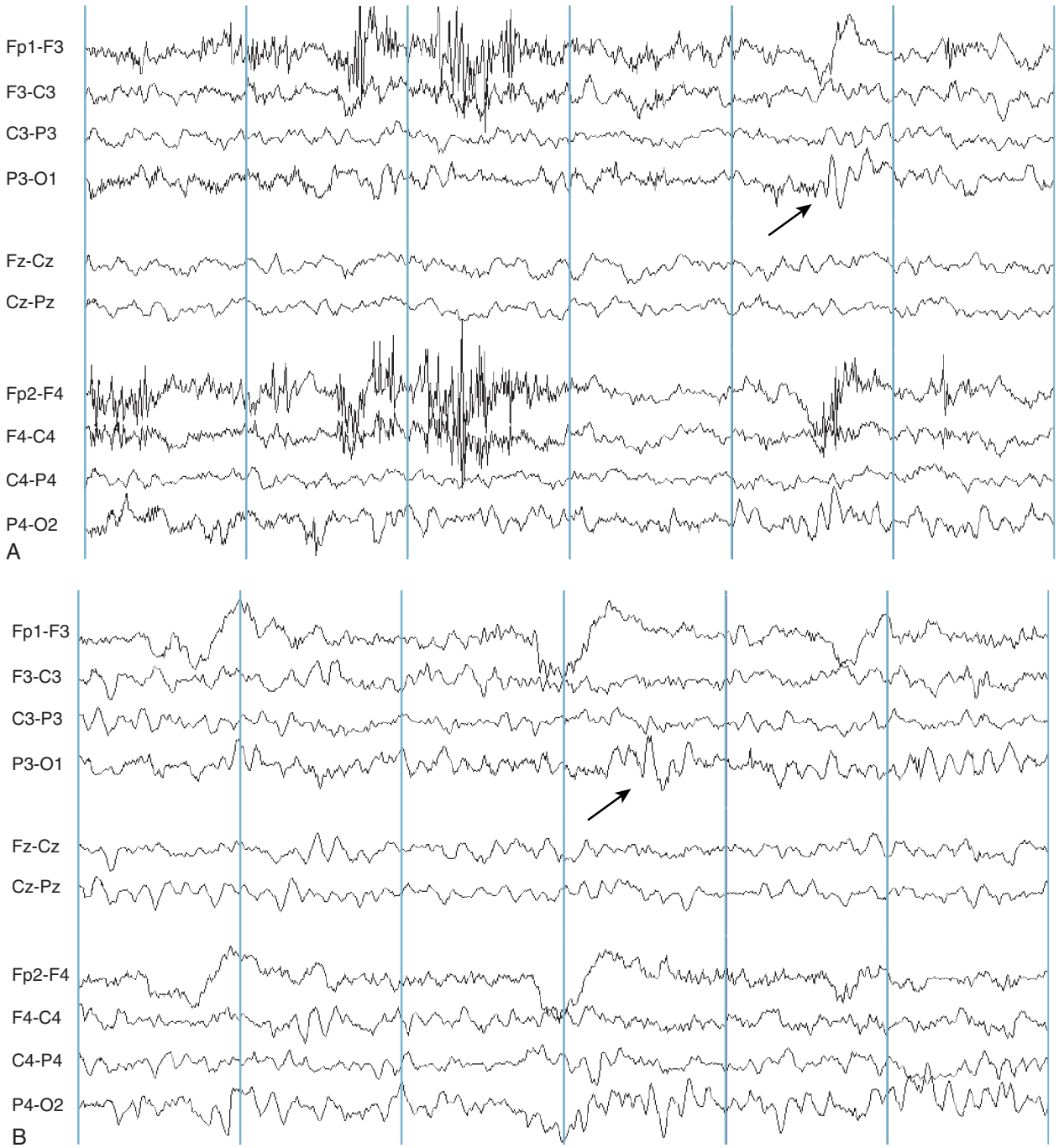

Figure 11-28 **(A)** An apparent sharp wave is seen in the left occipital area (arrow). **(B)** The same wave is now recognized in the context of the posterior rhythm (arrow), confirming that it is a posterior rhythm fragment rather than a sharp wave. The posterior rhythm fragment seen in Panel A was likely caused by rapid eye closure (note the eyeblink artifact in the frontal channels occurring at the same time as the wave fragment).

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Chapter

12 EEG Patterns in Stupor and Coma

The term *coma* refers to a state in which a person is unaware of self and surroundings, even if stimulated from the outside. Between consciousness and deep coma, there is a continuum of possible levels of responsiveness and awareness. *Encephalopathy* is a broad term that may be used to indicate a decrease in awareness; a patient who develops confusion and decreased awareness can be said to be "encephalopathic." Because many of the nuances of the neurologic examination are lost in the comatose patient, the EEG plays a special role in ascertaining the depth of coma. In patients who have been pharmacologically paralyzed, a common practice in intensive care units (ICUs), the neurologic examination yields limited information. In such patients the EEG may be the principle source of information regarding the patient's neurologic state.

Broadly speaking, the EEG may contribute information in the setting of coma in three ways. First, the pattern seen on a single EEG "snapshot" may suggest the depth and severity of the coma. Second, trends seen in repeat or serial EEGs can be a useful indicator of improvement or deterioration in a patient's status. The specific EEG parameters used to follow such trends and their implications are discussed in this chapter. Third, in a minority of cases the EEG pattern seen in coma can suggest its specific cause, such as the association of triphasic waves with hepatic and other metabolic encephalopathies or the unexpected discovery of continuous subclinical seizure activity.

INDIVIDUAL PARAMETERS OF THE EEG IN COMA: Voltage, Frequency, Reactivity, and the Presence of Normal Sleep Elements

There is a general correspondence between EEG coma patterns and the depth and severity of the coma. A variety of EEG attributes can be followed on serial testing to track a patient's progress in the comatose state. In patients who have a deteriorating neurological status, a parallel deterioration in the EEG is expected. Likewise, in patients with progressive neurologic improvement, a concomitant improvement in the EEG is expected. Thus the EEG can serve as a useful adjunct to the clinical examination.

Slow-Wave Voltage

Low-voltage slow waves intermixed with the patient's baseline background activity may be the first EEG sign of encephalopathic change (see Figure 12-1). An increase in the amount or amplitude of slow-wave activity suggests an increase in the severity of the encephalopathy. With deepening coma, slow-wave amplitude may continue to increase, and very high-voltage slow-wave patterns may be seen. Rather than intermixing with the background activity, the high-voltage slow-wave activity becomes the background. As cerebral function is increasingly affected, however, slow-wave amplitude can only increase to a certain point. With yet more severe cortical dysfunction, cortical rhythms begin to decrease in amplitude. With the most severe neurological processes cortical function becomes depressed and the brain becomes less able to maintain slow-wave voltages, resulting in diminished background activity and voltage. Thus, very low-voltage patterns in coma *(voltage depression)* are considered more ominous than high-voltage slow-wave patterns. The EEG patterns associated with the most severe degrees of cortical dysfunction show marked suppression of voltages or even electrocerebral inactivity.

Given this described sequence of initially increasing, then decreasing slow-wave amplitude with increasingly severe encephalopathy, a linear relationship between slow-wave amplitude and severity of encephalopathy cannot be assumed. When amplitudes are seen to decrease, this could represent either a trend toward normalization or signal a trend toward voltage depression and increasing dysfunction. In such cases, other EEG features (discussed later) such as frequency and reactivity of the background may help clarify the meaning of the change (see Figures 12-2 and 12-3).

The evolution of slow-wave activity during the improvement phase of a neurologic process may be less tightly linked to the patient's neurologic status. The clearing of slow-wave activity often lags behind the patient's clinical improvement. In a patient who is recovering from a dramatic encephalopathy, EEG slow-wave activity may still be present even as the patient wakes up, sits up, and begins talking. The persistence of slow-wave activity in the face of an improving neurologic picture is not necessarily a poor neurologic sign as long as there is a trend toward EEG improvement. Likewise, the slow-wave activity that follows a seizure

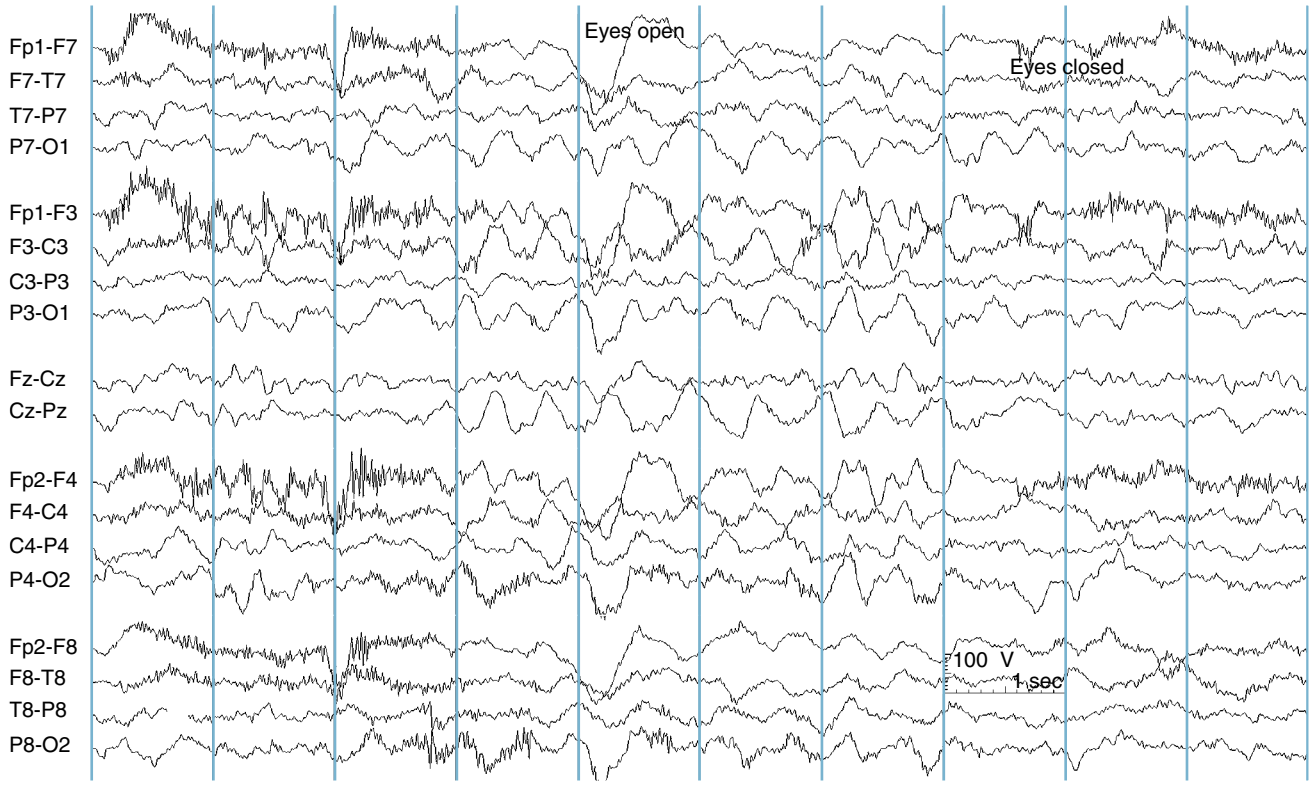

Figure 12-1 Low- to medium-voltage delta activity is seen superimposed on an otherwise unremarkable background in a stuporous 12-year-old boy with meningitis.

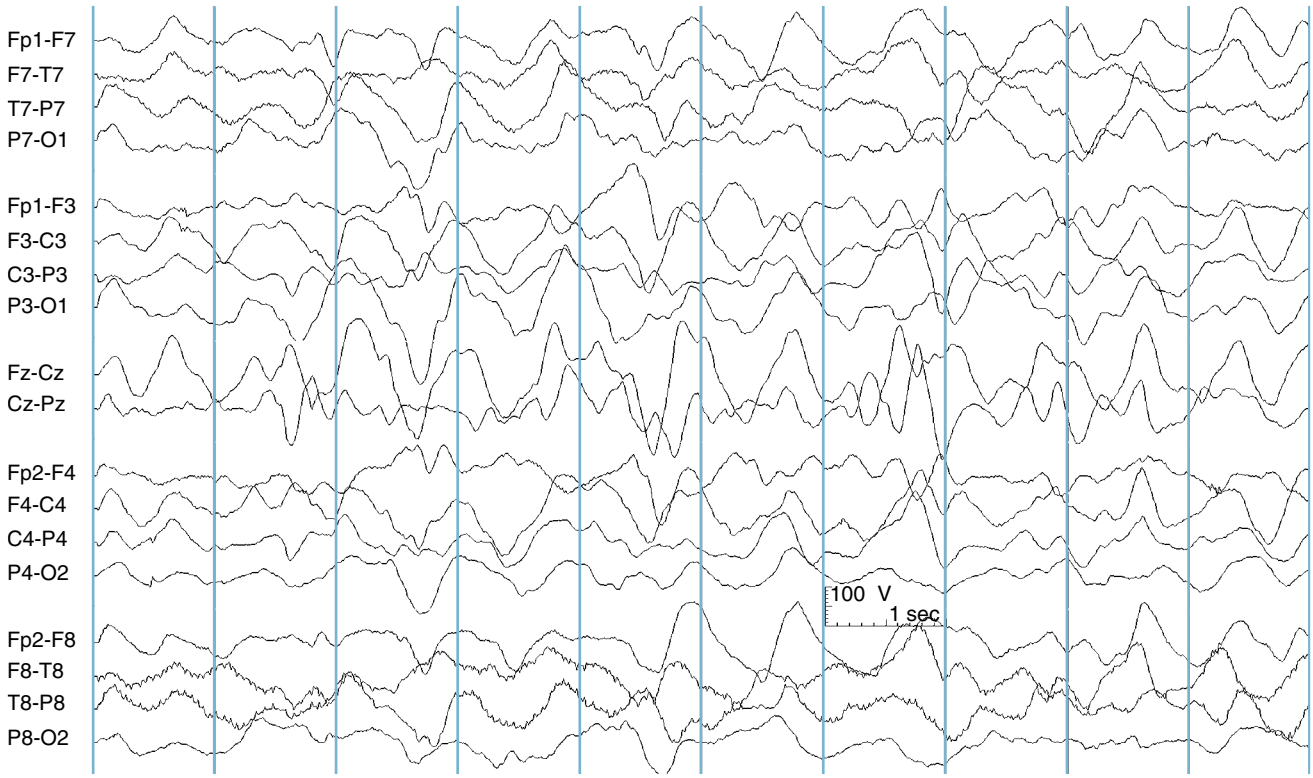

Figure 12-2 A typical slow-wave pattern in coma is shown with high-voltage semirhythmic delta waves. A small amount of intermixed theta activity is also seen, particularly near the vertex and in the occipital areas. Compare to Figure 12-3.

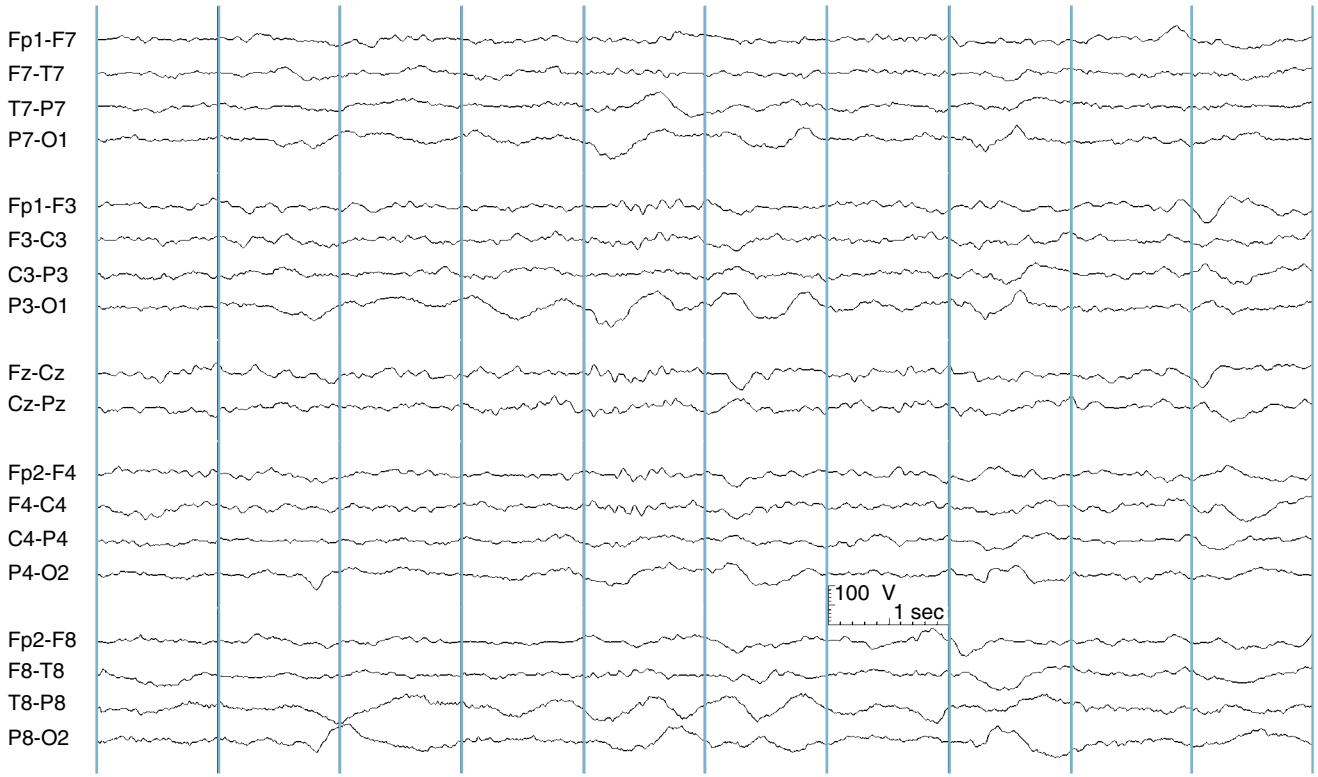

Figure 12-3 The EEG of the same patient seen in Figure 12-2 recorded 48 hours later. As discussed in the text, a decrease in slow-wave voltage in coma can signal either an improvement or a deterioration in the patient's state. In this tracing, the appearance of faster rhythms accompanies the decrease in amplitudes, clarifying that the drop in voltages represents a trend toward improvement.

(postictal slowing) may persist well past the point that patients report feeling back to their preseizure baseline. Slow-wave activity may persist after a seizure for hours, commonly a few days, but occasionally for as long as 3 to 4 weeks depending on the type of seizure, the duration of the seizure, and the general neurologic health of the individual.

Slow-Wave Frequency

The relationship of slow-wave frequency to coma severity is more straightforward than it is for slow-wave amplitude. In general, decreasing slow-wave frequencies suggest increasing severity of encephalopathy. A decrease in slow-wave *amplitude* can be associated with either improvement or deterioration in neurologic status as described earlier. Counting wave frequency is a useful tool for distinguishing between the two possibilities. If background frequency is increasing, this is a good sign; slower slow waves suggest deterioration. A similar approach is taken when comparing two hemispheres with slow-wave activity—one with higher voltages than the other. Higher voltage slowing may mark the more affected hemisphere, but it may be that the opposite hemisphere manifests lower voltages because it is the more abnormal side. In such cases, comparing the frequencies generated by each side may clarify which is the relatively "healthier" hemisphere, identified by its higher frequency.

Reactivity

EEG reactivity is an additional useful feature in assessing the depth coma. The EEG is monitored for change when the patient is stimulated. The stimulus may be as simple as calling the patient's name or could include purposeful noxious tactile stimulation. Intensive care unit procedures such as endotracheal tube suctioning or venipunctures also provide an opportunity to observe EEG reactivity. An unreactive EEG is one that shows no change in response to stimulation. Reactive EEGs show a change with stimulation, such as an increase in amplitude and rhythmicity in low-voltage tracings or a relative flattening of the background in higher voltage tracings.

Presence of Normal Sleep Elements

The presence of identifiable sleep elements in the EEG in coma is felt to be associated with a relatively better neurologic prognosis. The presence of sleep features implies that there is enough cerebral structure intact to generate these elements. Sleep spindles are the most commonly identified sleep feature in this setting. In rare cases, the higher centers that generate sleep elements are intact, but there has been a severe injury at lower levels of the central nervous system, resulting in a poor outcome despite the persistence of sleep elements.

SPECIFIC EEG PATTERNS IN COMA AND NEUROLOGIC PROGNOSIS

The prognostic impact of the EEG patterns discussed here must always be interpreted in the context of the coma's underlying etiology. Although various coma patterns have different reputations in terms of the severity of the encephalopathic state that they imply, even the most severe patterns can have a good final outcome if the etiology of the coma is inherently reversible. A good example of a reversible process is drug overdose. Patients with drug overdose may show, at least for a period of time, otherwise ominous EEG patterns such as burst suppression, voltage depression, or even "flat" EEG patterns. After the drug effect has cleared, assuming no permanent brain injury, the patient (and the EEG) may recover completely. This stands in contrast to the patient who shows a burst-suppression pattern or voltage depression after a prolonged cardiac arrest, a type of injury that is less likely to be reversible. In this group of patients, these EEG patterns have a more ominous significance.

Some of the most useful studies that have examined the prognostic impact of different EEG patterns in coma have limited the study group to patients with anoxic insults, such as those caused by cardiac arrest. This approach has the advantage of excluding the important variable of coma etiology from long-term outcome; however, the conclusions of these studies should only be extrapolated outside this etiologic group studied with caution. It is no surprise that two patients with the same EEG pattern in coma, such as a drug overdose patient and a patient with a malignant brain tumor, may have very different neurologic outcomes but similar EEG findings. Because EEG patterns are dictated more by the function of the cerebrum than the brainstem, the minority of patients with devastating brainstem injuries but relative sparing of the cerebrum may have misleadingly benign EEG findings. The order that specific coma patterns are listed in the following subsections should not imply a strict ranking, although they are generally described in order of increasing severity.

Intermittent Rhythmic Delta Activity

Among EEG findings in encephalopathy, intermittent rhythmic delta activity (IRDA) is considered to lie at the milder end of the spectrum of encephalopathic EEG patterns. IRDA may appear in patients who are awake or who are mildly lethargic or stuporous; IRDA patterns are not associated with deeply comatose states. IRDA tends to occur in the frontal regions in adults (frontal intermittent rhythmic delta activity, or FIRDA) and in the occipital regions in younger children (occipital intermittent rhythmic delta activity, or OIRDA; see Figure 12-4). When encephalopathic states become more severe, IRDA patterns may be replaced by continuous slow-wave patterns. Various types of IRDA are discussed in more detail in Chapter 9, "The Abnormal EEG."

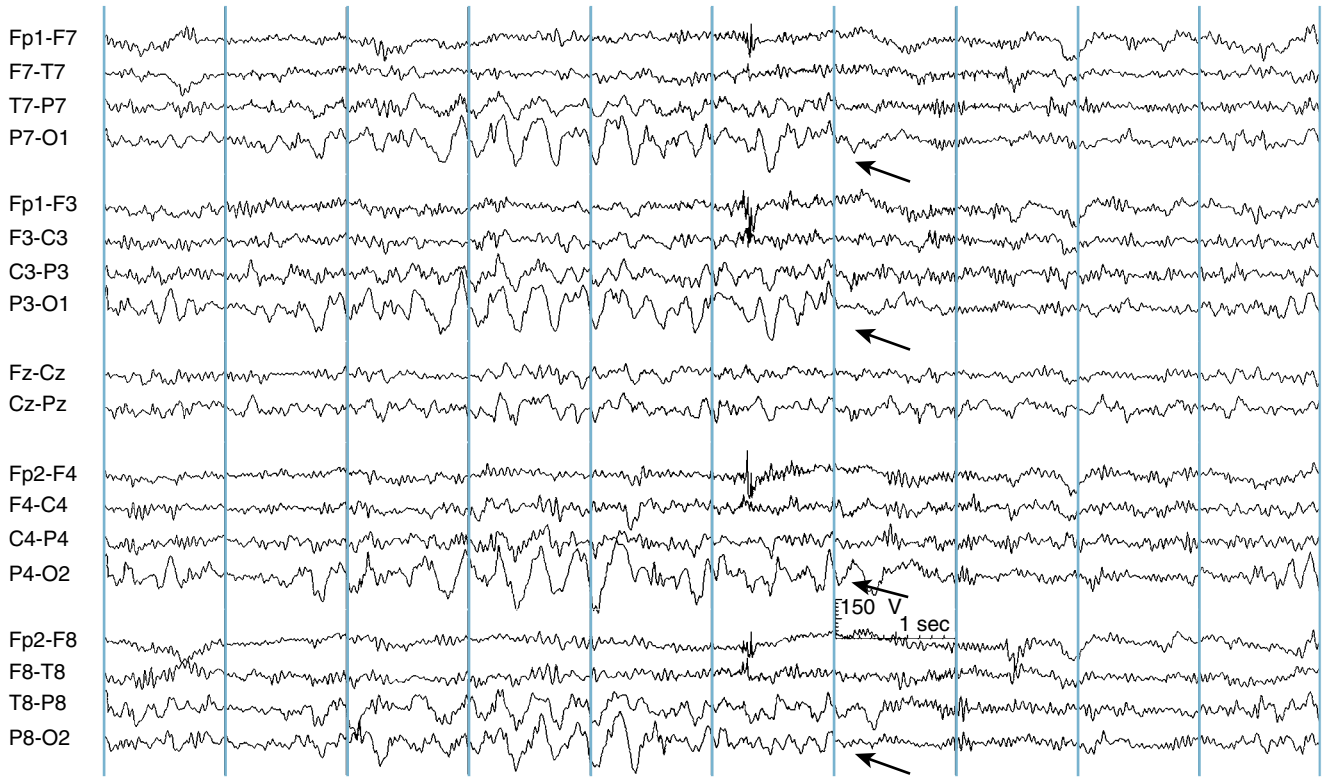

Figure 12-4 Occipital intermittent rhythmic delta activity (OIRDA) and excess fast activity are seen in a 9-year-old intensive care unit patient with postictal confusion (arrows). The increased fast activity is the result of treatment with lorazepam. Intermittent rhythmic delta activity is usually associated with mild encephalopathies.

Spindle Coma

Spindle coma may be indistinguishable from normal Stage II or III sleep (see Figure 12-5). The term may be applied to generalized slow-wave patterns obtained in comatose patients in which sleep spindles can be identified, although in most examples of spindle coma, the amount of spindle activity is exaggerated. Coma patterns that include normal sleep elements usually fall into better prognostic groups. This makes intuitive sense because the ability to generate normal sleep elements implies that the centers responsible for generating spindles, located in the diencephalon and above, are functionally intact. Some cases of spindle coma with poor outcome may be explained by patterns of damage that involve brainstem structures but have left higher cerebral structures relatively unaffected. Spindle coma can be distinguished from alpha coma (discussed later) in that, in spindle coma each spindle has a discrete duration and spindles should be maximally expressed in the frontocentral regions; alpha patterns in alpha coma (discussed later) are more diffuse and continuous.

Continuous Slow-Wave Patterns

Diffuse slow-wave patterns are among the most frequently encountered EEG patterns in coma. Just as there is a continuum among alert, stuporous, and comatose states, so is there a continuum between the normal EEG and EEG patterns with various degrees of diffuse slowing. Diffuse slow-wave patterns are usually comprised of delta frequencies, but theta frequencies may be seen as well. Slow-wave patterns in coma are usually nonrhythmic. Rhythmic slow-wave patterns are more often seen in the setting of metabolic encephalopathies.

As described in the previous section on slow-wave voltage in coma, higher slow-wave voltages are generally considered "more healthy" than lower voltages, but the relationship between voltage and depth of coma is complex, because declines in slow-wave voltage may potentially be associated with either clinical deterioration or improvement. EEGs that show reactivity to noxious stimulation are generally associated with a better neurologic prognosis than those that are unreactive, and those with higher frequencies are generally prognostically better than those with lower frequencies.

As with other coma patterns, the underlying cause of the coma is probably the strongest predictive factor in outcome, often more important than the specific EEG pattern itself. Outcome after an anoxic event associated with delta slowing may be quite different from the outcome seen after a generalized seizure that is followed by similar delta slowing in the postictal period; there is the expectation that the latter pattern may be completely reversible.

Asymmetric voltages in slow-wave patterns suggest that an asymmetric process is at work. Diffuse processes, such as metabolic derangements, are usually associated with symmetrical patterns. The exception to this rule is the case of a symmetrical process acting on an asymmetrical brain. For instance, although most patients with hyperosmolar coma would be expected to show a symmetrical slow-wave pattern, a patient with hyperosmolar coma who has suffered a previous stroke may

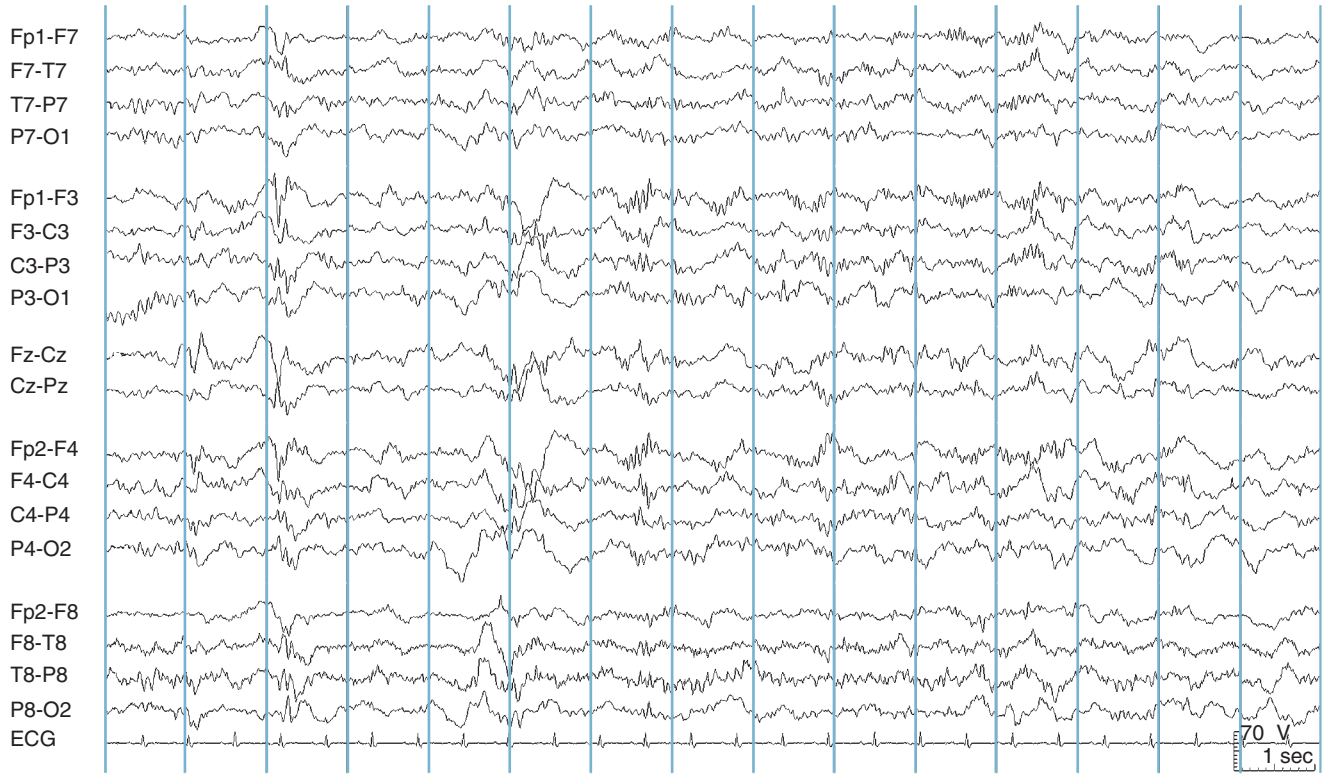

Figure 12-5 Exaggerated spindles are seen along with vertex waves in this comatose patient, representing an example of spindle coma.

show an asymmetric pattern in reaction to the metabolic derangement—healthy brain regions may react differently to the metabolic abnormality compared with previously injured regions. As discussed earlier, it may not always be clear which side is more severely affected when slow-wave patterns are asymmetrical (see Figure 12-6). Other findings superimposed on a slow-wave background such as periodic lateralized epileptiform discharges (see Chapter 9) or epileptiform activity may suggest additional diagnoses.

Alpha Coma

The alpha coma pattern consists of diffuse alpha activity in the range of 8 to 13 Hz (see Figure 12-7). Most reports have associated the alpha coma pattern after anoxic insult with a relatively pessimistic outcome, although with some exceptions. A similar pattern of diffuse alpha activity may be seen in toxic and metabolic encephalopathies, especially drug intoxications. In contrast to the postanoxic state, the alpha coma pattern after drug intoxication is felt to have a relatively more favorable prognosis.

Various theories have been put forward to explain the genesis of alpha rhythms in alpha coma. Some have suggested that the pattern is related to the spindle generator. Others have suggested that this alpha activity represents a paradoxically retained alpha-range activity related to the posterior rhythm. An additional possibility is that, because of diffuse cortical injury, the alpha activity of alpha coma represents a slowed version of the beta activity that is usually generated by the normal cortex. The dependence of neurological outcome on the cause of coma after alpha coma is a reminder of the general importance of considering etiology in assessing prognosis in coma.

Triphasic Waves

The appearance and significance of triphasic waves is discussed in more detail in Chapter 9, "The Abnormal EEG" and shown in Figure 9-34. The presence of triphasic waves is almost always associated with a state of depressed consciousness. Triphasic waves are usually caused by a metabolic derangement such as hepatic coma or renal failure but are occasionally seen after an anoxic insult. The prognosis of patients with triphasic waves depends on the course of the underlying process.

Nonconvulsive Status Epilepticus

Among EEG coma patterns, prompt diagnosis of nonconvulsive status epilepticus (NCSE) is important because it is a cause of coma that may be amenable to treatment

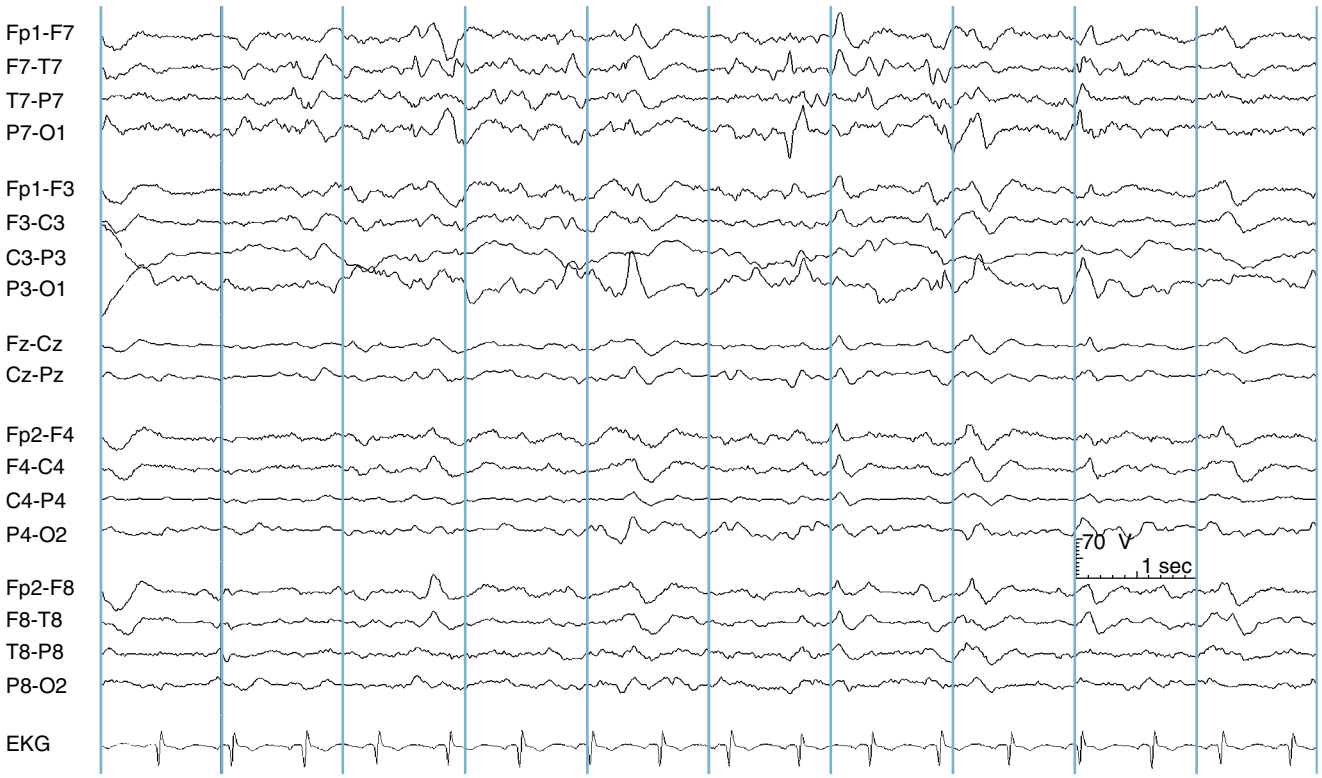

Figure 12-6 In this comatose patient, asymmetric slowing is seen with higher voltages over the left hemisphere compared with the right. Some sharp features are also evident over the left hemisphere. When slow-wave patterns are asymmetric, it is not always obvious which is the more severely affected side. In this example, fewer theta rhythms are seen over the lower voltage right hemisphere, suggesting that this side may be more severely affected.

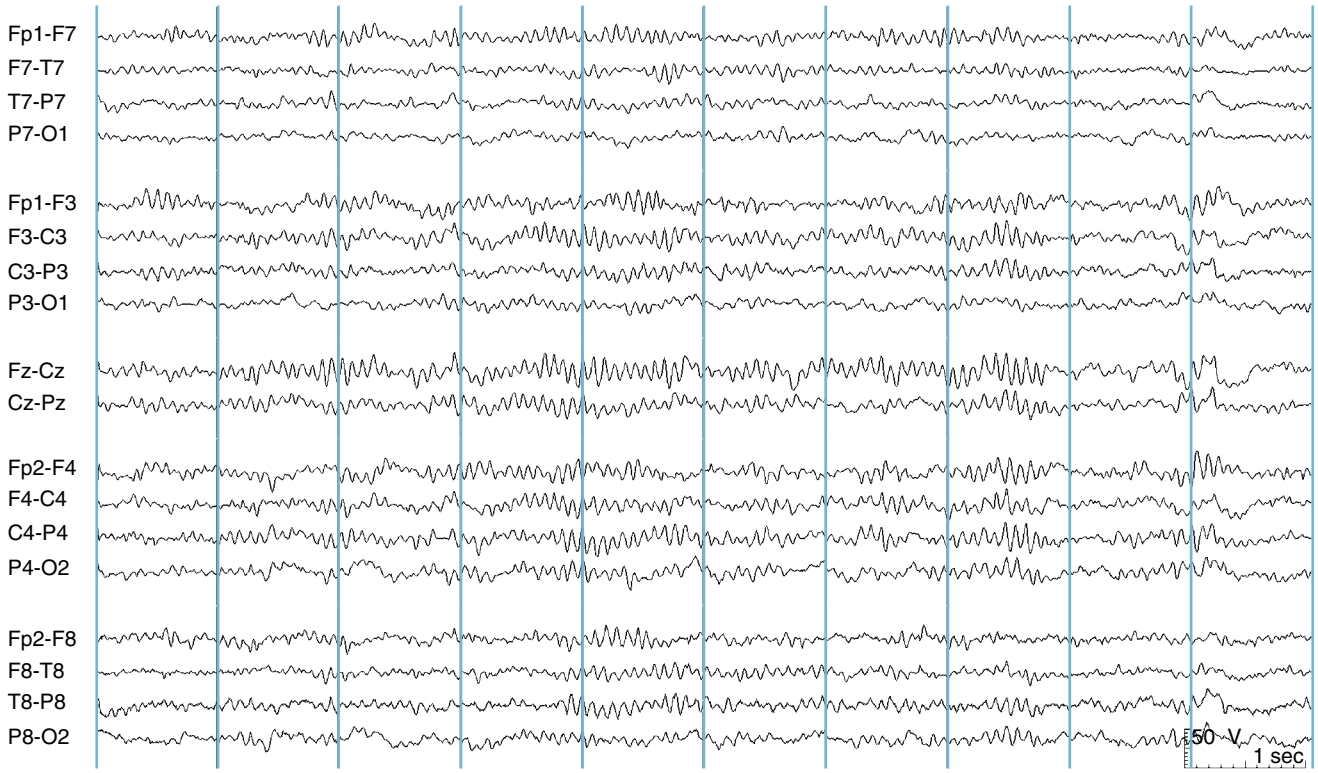

Figure 12-7 An alpha coma pattern is seen with diffuse alpha activity. Prognosis in alpha coma differs in cases caused by anoxia as opposed to those caused by drug effect.

with antiseizure medications. Both generalized and complex partial status epilepticus eventually evolve into states that resemble coma. Patients with continuous generalized seizures often begin by having repetitive, outwardly observable generalized convulsions. As the generalized seizure discharges repeat, however, the convulsive movements become less prominent, eventually culminating in a state in which the patient lies unresponsive and motionless even as the electrographic seizure discharges continue. Patients may transition to this nonconvulsive state within 30 minutes or less of seizure onset. Similarly, prolonged complex partial seizure activity (complex partial status epilepticus) will evolve to a stuporous or comatose state, sometimes with cyclical fluctuations in responsiveness (see Figures 12-8 and 12-9). Careful observation of patients with either generalized or complex partial status epilepticus may reveal intermittent, subtle rhythmic movements in the face or limbs, but absence of this finding cannot be relied on to exclude the diagnosis. Absence status epilepticus can present as a confusional state, although many patients retain the ability to walk and converse, albeit in a confused fashion, despite the presence of continuous discharges. Therefore, the different types of NCSE are important elements in the differential diagnosis of coma.

NCSE may have a variety of causes ranging from the patient with idiopathic epilepsy who experiences a seizure that fails to terminate to the patient in whom NCSE occurs as a terminal event following severe anoxic, metabolic, or neoplastic processes. When NCSE is caused by a severe, irreversible injury, the electrographic seizure pattern may evolve to a low-voltage pattern and eventually to a "flat" EEG.

Burst-Suppression Patterns

Burst-suppression patterns consist of periodic bursts of polymorphic activity, often containing sharp features, separated by periods of voltage suppression (see Figure 12-10). In a minority of patients, a myoclonic movement may accompany each burst. Burst-suppression patterns have been associated with anoxic injury and are generally associated with a poor prognosis for neurologic recovery. In infants and children, and especially in the minority of patients in whom the pattern improves promptly, outcome may be somewhat better. Burst-suppression patterns can also be caused by drugs, either by drug overdose or the purposeful use of drugs, such as barbiturates, to induce coma. The postanoxic and pharmacologic versions of burst suppression are usually easily distinguished by history and laboratory testing. Pharmacologic burst-suppression patterns have the potential for complete reversibility and are therefore in a separate prognostic group.

The purest form of burst-suppression is an unrelenting pattern that does not cycle to other patterns, is present on every page of the EEG, and is unaffected by outside stimuli. Lower voltage bursts and longer and flatter interburst intervals correlate with increasing severity. Transitional versions of burst suppression that

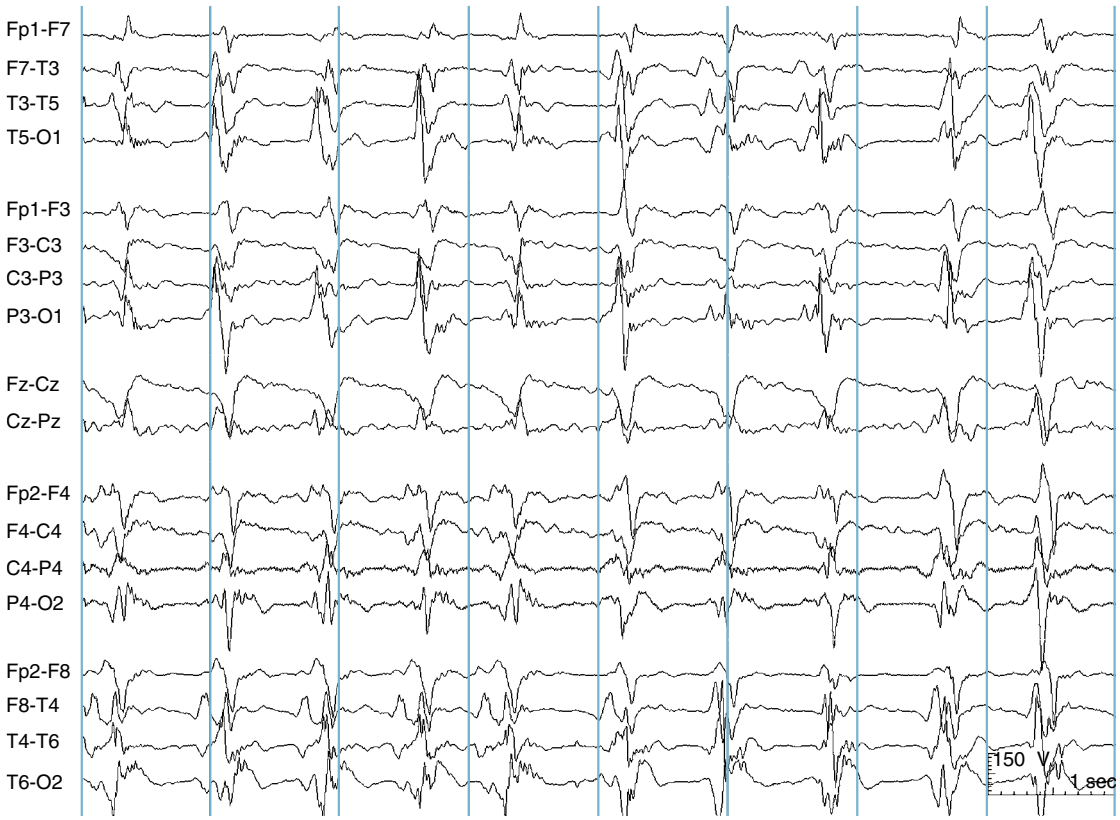

Figure 12-8 The EEG reveals that this patient's unresponsiveness is caused by continuing electrographic seizure activity or nonconvulsive status epilepticus.

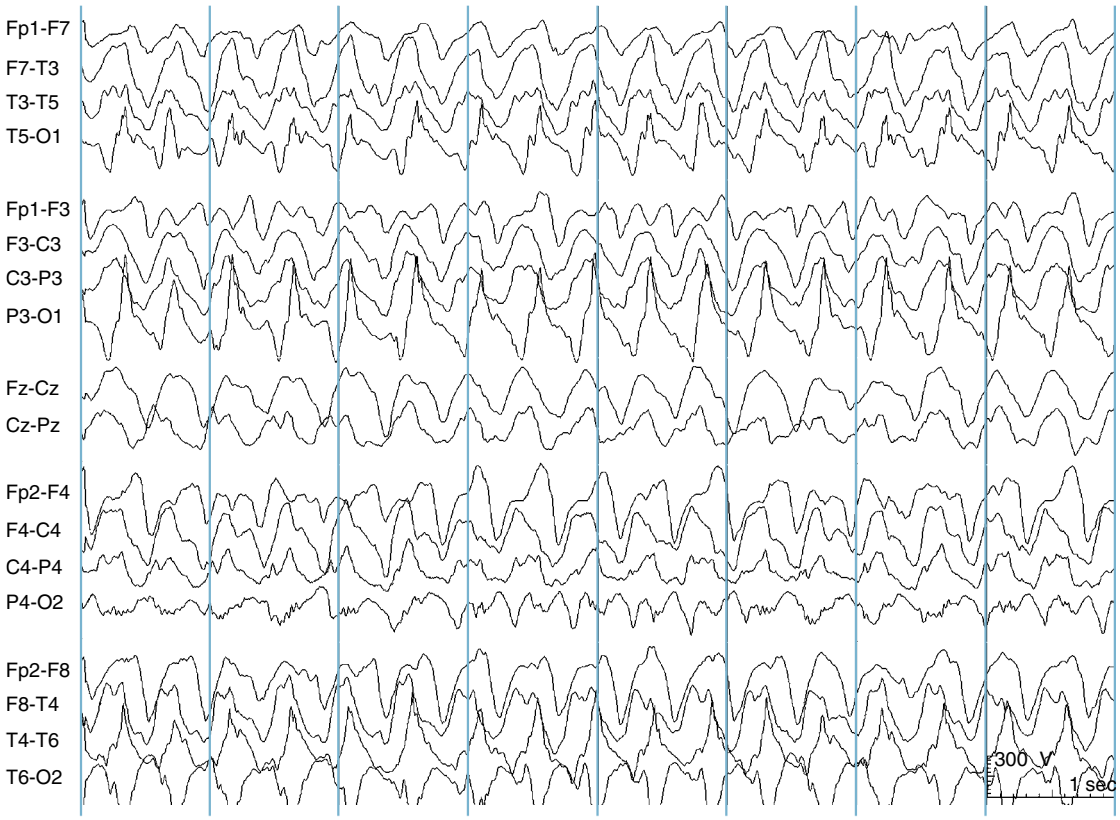

Figure 12-9 This highly rhythmic pattern with sharp features in this comatose patient is also consistent with nonconvulsive status epilepticus.

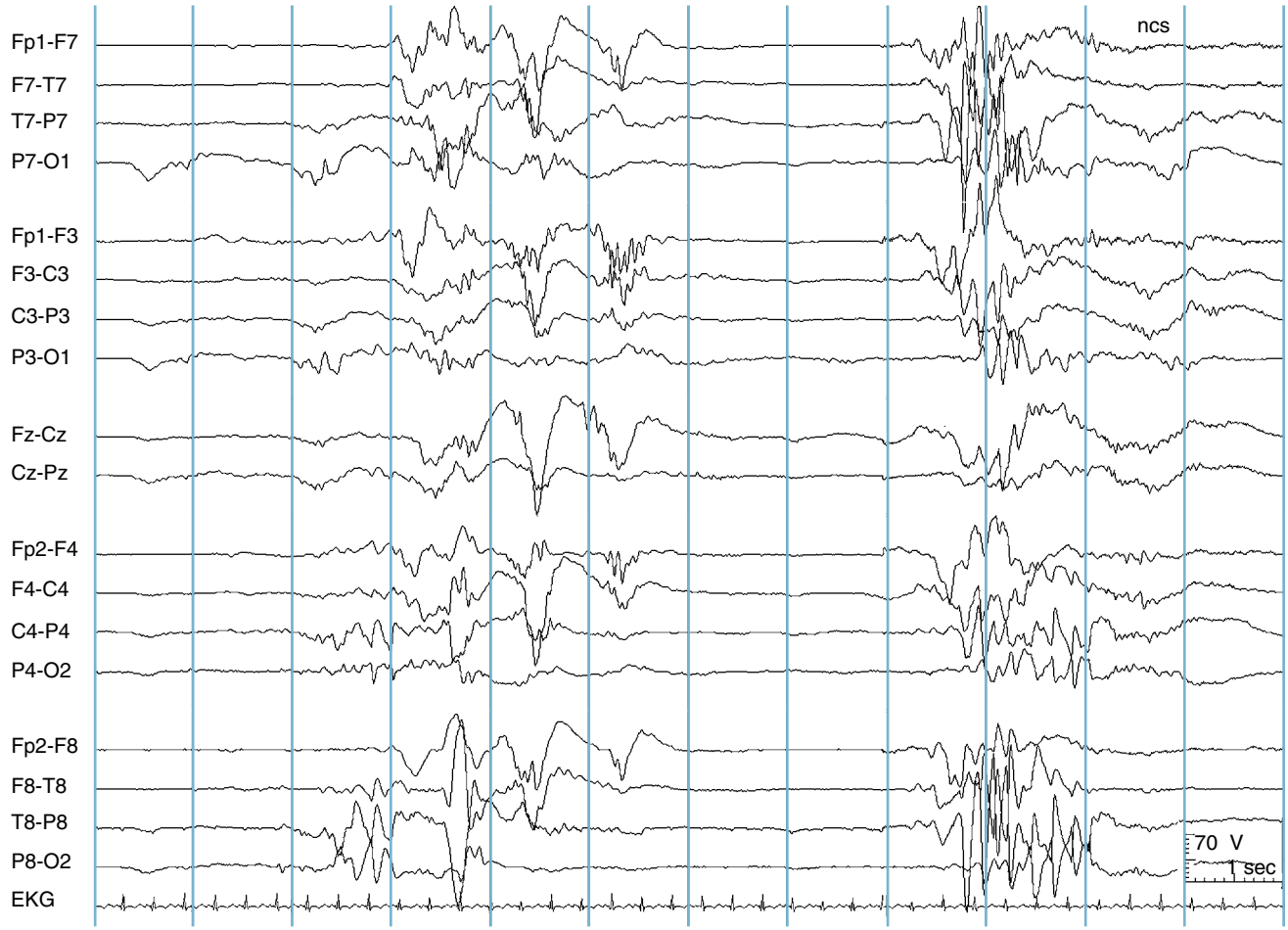

Figure 12-10 A burst-suppression pattern is seen with high-voltage polymorphic bursts separated by periods of suppression. NCS: no clinical signs.

change, or even pause, with outside stimuli (so-called reactive burst suppression) are probably associated with a better prognosis than the pure form of the pattern.

Voltage Depression

Low-voltage records or *voltage depression* in coma (tracings in which voltages persistently do not exceed 20 μV in any head region) are considered severely abnormal. The pattern suggests a degree of injury so severe that the cerebrum cannot generate significant voltages. Voltage depression may be seen after continued deterioration of a burst-suppression pattern in which the bursts have disappeared leaving only the periods of suppression. It may also be seen near the end of a sequence of continued voltage decline as described earlier.

Certain pitfalls in the diagnosis of voltage depression should be avoided (see Figures 12-11 and 12-12). The term implies a persisting pattern; some patients have a period of voltage depression after a seizure, but this period should be short-lived. Voltage depression also may occur transiently after anoxia. Therefore, very short tracings are inadequate to establish a diagnosis of voltage depression. Standard instrument settings may hide some features of a low-voltage EEG and may even suggest electrocerebral inactivity unless appropriate adjustments to amplifier gains are made. Finally, a small percentage of normal adults may have a low-voltage EEG pattern during wakefulness; the foregoing discussion of low-voltage patterns only applies to recordings obtained from patients in coma.

EEG RECORDING IN SUSPECTED CEREBRAL DEATH

The role of the EEG in the patient with suspected brain death is not straightforward. The original definition of death as the cessation of all vital signs became impractical with the advent of modern intensive care and mechanical ventilation. In response, an *ad hoc* committee was convened at Harvard Medical School in 1968 to establish a definition of irreversible coma or *brain death* that could replace the older definition when necessary in intensive care settings where mechanical ventilation may be in use. The guidelines attempted to identify individuals with "no discernible central nervous system activity" ("A definition of irreversible coma," 1968). The guidelines are designed to create a new legal definition of death such that when the criteria set forward for the diagnosis of brain death are met, the patient can be declared legally dead and removed from the ventilator.

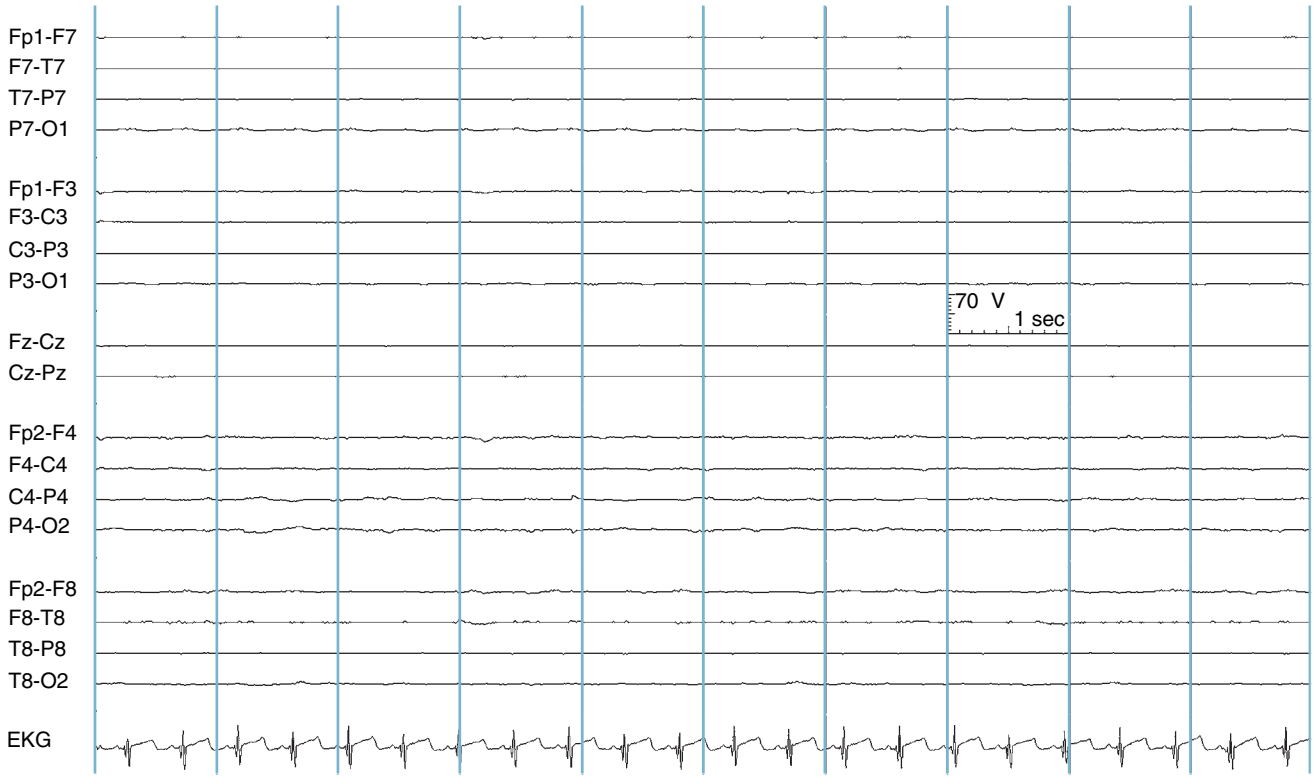

Figure 12-11 This EEG of a comatose patient is displayed at a sensitivity of 7 μV/mm and appears flat. Especially when electrocerebral inactivity is suspected, sensitivities of 2 μV/mm should be used (see Figure 12-12).

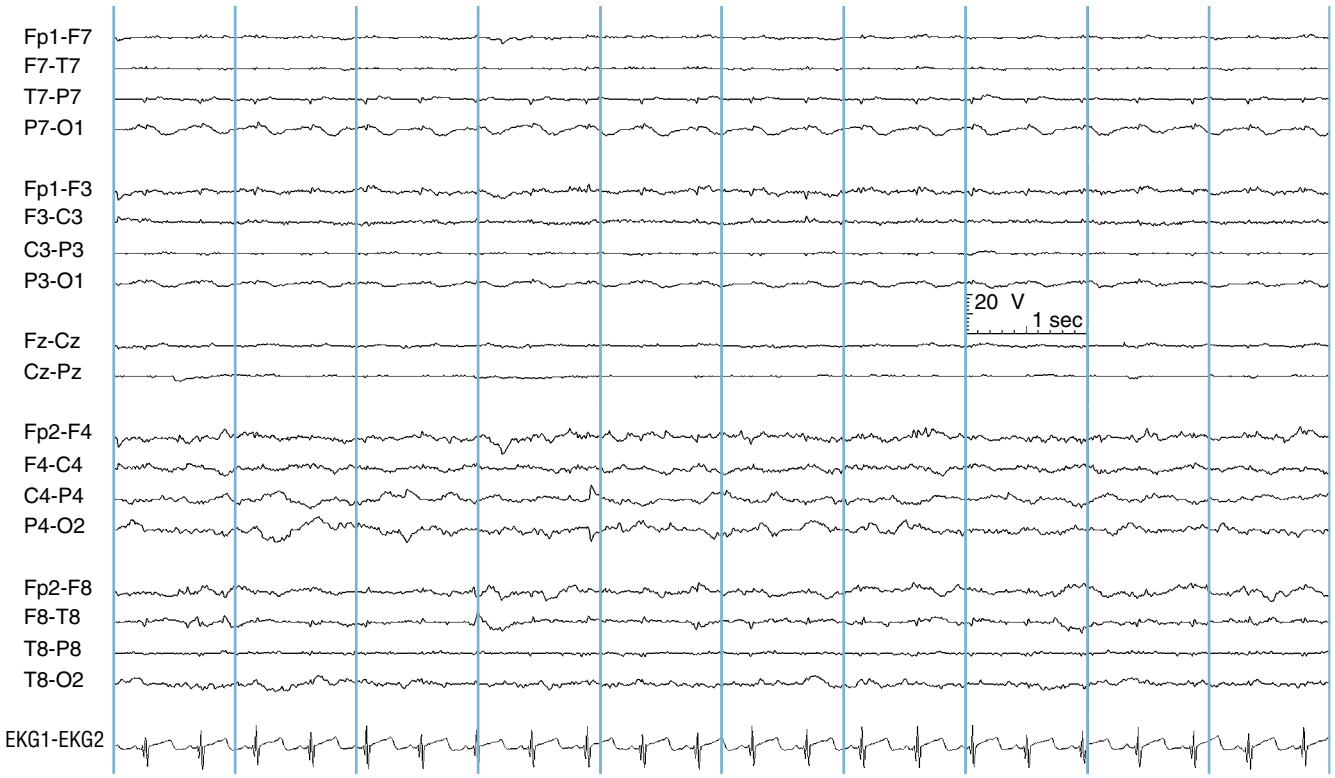

Figure 12-12 When the same page of EEG as was shown in Figure 12-11 is displayed at a sensitivity of 2 μV/mm, a small amount of definite electrocerebral activity is seen over the right hemisphere (bottom eight EEG channels). No electrocerebral activity is seen over the left hemisphere and midline, however. Pulsation artifact is seen in the channels that include O1.

The specific guidelines for determination of brain death vary among countries, jurisdictions, and even among individual hospitals. Almost all official criteria require a complete lack of responsiveness, lack of patient movement, lack of respiratory effort when the patient is taken off the ventilator during a trial period, and absent brainstem reflexes. The role of EEG in diagnosing brain death varies by location; an EEG showing *electrocerebral inactivity* (ECI) is usually not required to make the determination of brain death but can serve as an adjunct to the diagnosis. Patients with true ECI recordings, especially when two such recordings are obtained 24 hours or more apart, rarely experience neurologic recovery. An ECI recording considered alone, i.e., apart from the context of the patient's history and examination, should not be considered synonymous with brain death.

EEG recordings performed in the setting of suspected cerebral death are almost always carried out in ICUs. The large amount of electrical equipment in most ICUs makes this setting an electrically hostile environment and increases the challenge of obtaining clean EEG recordings at the high amplifier gains necessary for determination of ECI (see Figure 12-13). Nevertheless, with careful technique, satisfactory EEG tracings for this purpose are obtainable.

Because of the gravity of the question at hand, an EEG performed with the goal of establishing a complete lack of brain wave activity or ECI should meet certain minimal technical standards. The most recent guidelines from the American Clinical Neurophysiology Society were published in 2006 ("Guideline 3," 2006) and are summarized in the following list.

Electrocerebral inactivity is defined as a complete **lack of EEG activity over 2 μV** when the following appropriate recording techniques are used.

1. A **full set of electrodes** should be used, including the midline electrodes, Fz, Cz, and Pz, with the exception of areas that may be inaccessible because of recent surgery or trauma.
2. Interelectrode impedances should be **between 100 ohms and 10,000 ohms.**
3. The integrity of the recording system should be verified to confirm that the apparent low-voltage tracing was not caused by a disconnection in the recording apparatus. This is done by **tapping the individual electrodes** and confirming the presence of the tapping artifact on the recording.
4. A **double-distance montage** with some interelectrode distances greater than 10 cm should be used during at least some portion of the recording. Greater interelectrode distances increase the chance of detecting low-voltage activity. An extracephalic electrode placed on a limb (e.g., the right hand) can help identify artifacts. An electrocardiogram (EKG) channel should also be applied to help identify EKG artifact.

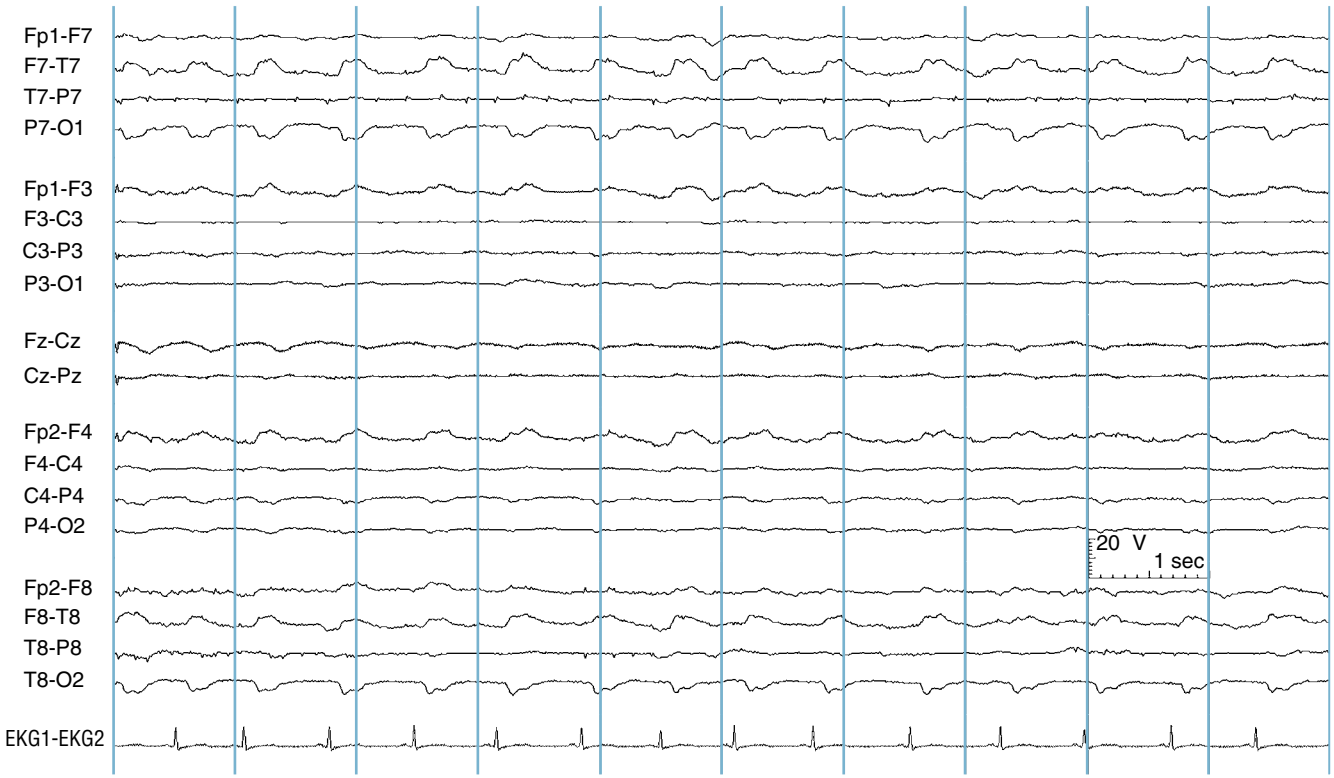

Figure 12-13 This page of EEG is displayed at 2 μV/mm and shows electrocerebral activity (ECI); none of the waves seen are of cerebral origin. Tracings displayed at the amplifier gains necessary for determination of ECI are prone to large amounts of artifact. On this page, several channels show pulsation EKG artifact. Beyond the use of high amplifier gains, several other procedural requirements must be met, as described in the text, before the EEG diagnosis of ECI can be made.

5. The record should be recorded at a sensitivity of **2 μV/mm for at least 30 minutes** to minimize the possibility of missing a 2 μV signal.
6. Appropriate filter settings should be used with a **bandpass of 1 to 30 Hz** or wider (low-frequency filter set at 1 Hz and high-frequency filter set at 30 Hz).
7. **Additional monitoring techniques** should be used to distinguish artifact from brain wave activity at the high amplifier gains used. This may include an EKG and a respiratory channel, if necessary, to monitor ventilator artifact.
8. There should be **no EEG reactivity** to intense tactile, auditory, or visual stimuli.
9. The recording should only be made by a **qualified EEG technologist.**
10. If the diagnosis of ECI is **uncertain, the recording should be repeated.**

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Chapter

13 The Electroencephalogram of the Newborn

Newborn EEG interpretation is considered a particularly challenging area. An understanding of the appearance of the normal newborn EEG was achieved considerably later than for EEGs of childhood and adulthood. In fact, before the 1960s, it was not generally accepted that there was scientific or clinical value to be found in the analysis of the EEGs of newborns.

The relatively slower progress in the field of neonatal electroencephalography has been related to several factors. In almost any laboratory, the number of newborn EEG studies performed is considerably smaller than the number of studies performed in older age groups. Thus any given reader likely has less clinical experience reading tracings from the neonatal age group compared with older children. Also, to establish the basic foundations of neonatal EEG interpretation one must know the appearance of the normal neonatal EEG, which, in turn, requires that we know which patients are neurologically normal. Neurologic normality is more difficult to ascertain in newborns because of the inherent limitations in our ability to assess newborns neurologically; the question of whether certain findings in the newborn EEG may be normal has remained controversial. In general, newborns are considered neurologically "normal" when the history, examination, and other neurological studies are normal. This definition is more difficult to apply in practice because most babies who have had an EEG have had it for some clinical indication, and the presence of an indication immediately brings up the possibility that something is amiss. Finally, there was an early bias toward believing that typical premature tracings were abnormal because their discontinuous appearance resembled patterns such as burst-suppression that are known to be abnormal in older individuals.

The Concept of Postconceptional Age

The EEG of newborns is strikingly different from that of older children and adults. In fact, the best known elements of the mature EEG (posterior rhythm, sleep spindles, vertex waves) do not make their first appearance until 6 to 8 weeks after term. In the context of electroencephalography, a newborn's degree of prematurity is stated in terms of postconceptional age (CA). The CA at birth is equivalent to the gestational age and is usually estimated using the date of the mother's last menstrual period, but other information such as early fetal ultrasounds and the baby's physical examination can be used to modify the estimate. By definition, a full-term newborn has a CA of 40 weeks and newborns delivered before 37 weeks are considered premature. Note that a 3-week-old newborn who was delivered at 38 weeks gestational age is considered to have a CA of 41 weeks for the purposes of EEG interpretation. The current CA is derived by adding the gestational age at birth to the current age in weeks (time since birth or "legal age"). One of the underlying assumptions of neonatal electroencephalography is that the expected appearance of a healthy newborn's EEG is based on its CA. Whether it was born prematurely or not, the EEG is generally assumed to evolve at the same rate whether the baby is inside or outside the womb. Certain pathological processes may, however, interrupt this orderly maturation. Therefore, a normal baby born at 41 weeks CA is generally expected to have an EEG structure similar to that of a normal 5-week-old baby who was at born 36 weeks CA.

From extreme prematurity to term to the postterm period, the appearance of the neonatal EEG evolves dramatically. In fact, on the basis of the various EEG features described here, an experienced neonatal electroencephalographer should be able to estimate the CA of a newborn to within approximately 2 weeks from the appearance of the EEG record. It has been claimed that when the CA estimate suggested by an otherwise normal neonatal EEG differs from the estimate based on the baby's physical examination, the EEG-based assessment is more likely to be correct. Figures 13-1 and 13-2 show the striking changes in the appearance of the cortical surface between 31 weeks CA and 40 weeks CA (term). It should come as no surprise that the appearance of the EEG evolves rapidly in premature babies.

Recording Technique

Opinion varies as to whether a full or reduced electrode set should be used for neonatal recordings. Some authors assert that the head is smaller, and therefore it is reasonable to apply fewer electrodes to the smaller

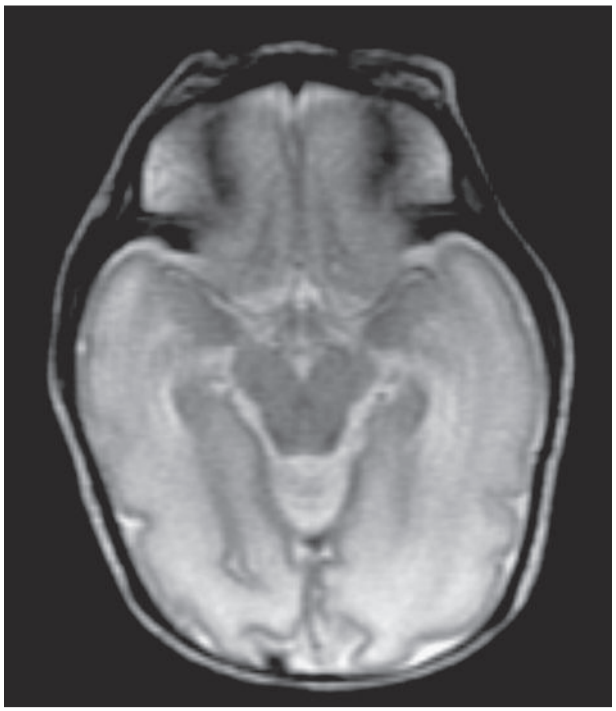

Figure 13-1 A T2-weighted magnetic resonance imaging scan of the brain of a normal baby at 31 weeks CA shows the relatively smooth appearance of the cortical surface and the rudimentary gyral pattern seen at this gestational age. The small amount of cerebrospinal fluid over the surface of the hemispheres appears white in this sequence. Prematures between 24 and 30 weeks CA have an even less developed cortical folding pattern.

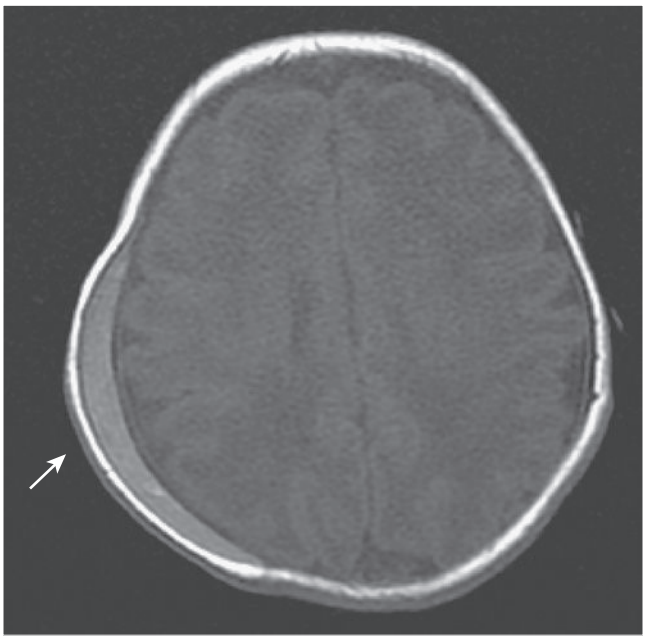

Figure 13-2 A T1-weighted magnetic resonance imaging scan of a term baby (40 weeks CA) shows a significant increase in the complexity of the gyral pattern compared with the 31 week CA brain shown in the previous figure. At term, the complexity of the cortical gyral pattern approaches that of the adult pattern. The cerebrospinal fluid over the brain surface appears dark in this scan sequence. This scan also shows a subgaleal hematoma over the right parietal area (arrow). The white signal encircling the head represents scalp fat rather than bone; this scalp hemorrhage lies outside the bony cranium. Such extra-axial collections may decrease the recorded voltages over affected areas.

head of the newborn. The opposing view holds that if the neonatal brain is conceptualized as a shrunken version of the adult brain, each lobe, gyrus, and cortical circuit is proportionally smaller, and the electric fields of discharges will be correspondingly smaller, requiring the usual (nonreduced) number of electrodes to achieve the same anatomic resolution of electric fields. Our laboratory uses a full complement of electrodes from the 10-20 system in newborns and even in most premature infants; reduced electrode sets are only used for premature infants with the smallest head sizes. Although reduced *(double-distance)* electrode applications have been shown to record the majority of normal and abnormal EEG activity and may also be better tolerated by the premature infant whose scalp skin is more sensitive, occasionally a highly focal seizure discharge or other highly focal finding may be missed. In addition, difficulties with artifact identification represent a hidden pitfall of the use of sparser electrode arrays. When a deflection is seen in a single channel, denser electrode arrays help determine whether an electric field surrounds the event, increasing or decreasing the chances that it is of cerebral origin as opposed to an electrical artifact.

Additional leads are applied to help assess sleep state; to some extent, a neonatal EEG recording resembles a polysomnogram. The added leads may include a nasal thermistor to measure respirations, ocular leads (one placed just above the outer canthus of one eye and the other just below the outer canthus of the other eye), and a submental electrode to monitor chin muscle (EMG) activity. Additional leads may include a strain gauge placed on the abdomen to record respiratory muscle effort and limb leads to document movements. Notations made by the recording technologist on the EEG record should also carefully document the appearance of the baby. Notations such as "appears asleep," "has hiccups," "feeding," "eyes closed," or "moving" help the reader assess sleep state and evaluate artifacts (see Figure 13-3).

Traditionally, newborn EEGs have been recorded at "half" paper speed (15 mm/sec). Although this practice may have originally been motivated in part by the urge to save paper on long recordings, the compression of the EEG resulting from slow paper speeds can make it easier to identify some discontinuous or bursting patterns, both normal and pathological. Certain delta patterns are easier to appreciate when displayed at slow paper speeds. For these reasons, slow paper speeds are still preferred by many readers for review of newborn EEG recordings. Ideally, a neonatal EEG record should include all stages of sleep—wakefulness, quiet sleep, and active sleep—which often requires recording times over 1 hour to allow assessment of sleep architecture.

A "QUICK TOUR" OF THE MAJOR NEONATAL EEG SLEEP STAGES

Similar to the "quick tour" of the adult EEG shown in Chapter 2, "Visual Analysis of the EEG," what follows is a brief overview or "tour" of the main sleep stages of the

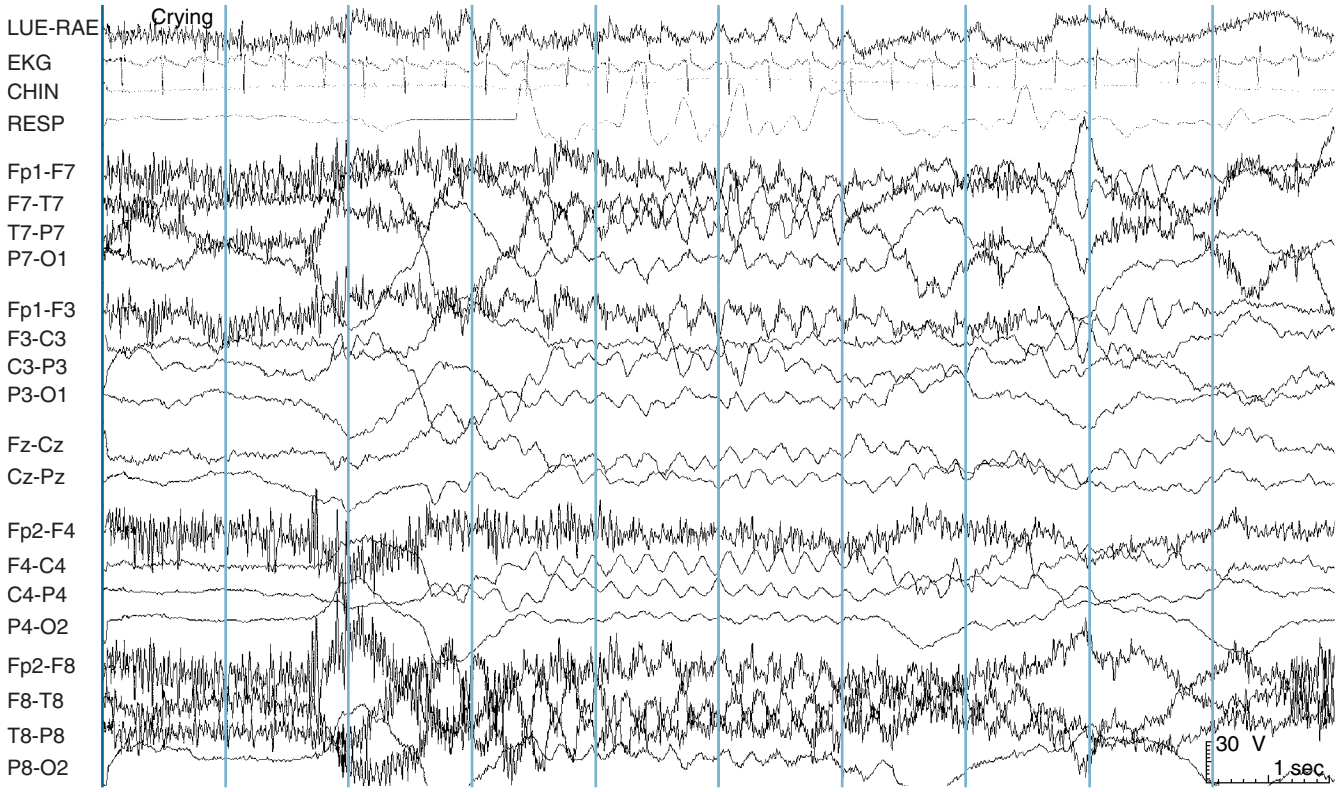

Figure 13-3 Rhythmic waves seen in the fourth to seventh seconds of this neonatal recording represent patting artifact. Because caretakers often attempt to soothe a crying baby by patting, patting artifact is a common finding in the newborn EEG. This type of rhythmic artifact may, in some cases, mimic an electrographic seizure.

newborn EEG and also how the technique of neonatal EEG recording differs in a few ways from that of older patients. Because the appearance of the newborn EEG evolves considerably through prematurity and approaching term, no single tracing can demonstrate all of the key findings.

In children and adults, the appearance of the EEG itself more or less defines sleep state. In newborns, however, one EEG background may be associated with several sleep states, and individual sleep states are associated with a variety of EEG backgrounds. Information from polysomnographic channels and behavioral observations are often necessary to define the current sleep state. We start by reviewing the five main background patterns of the newborn EEG, followed by the three main newborn sleep stages and how the described background patterns relate to the different sleep stages.

THE FIVE COMMON EEG BACKGROUND PATTERNS SEEN IN NEWBORNS

The features that we are most accustomed to seeing in the waking and sleep EEGs of older patients, such as the posterior rhythm, sleep spindles, and vertex waves, are not seen in newborns. Rather, specific types of EEG background patterns and elements are seen at different stages of maturity. These five principle EEG background patterns were originally described by the "French School" of neonatal electroencephalography. Although this system has not remained in common usage in all laboratories, it remains a useful construct for interpreting and describing neonatal EEGs. Inherent to the categorization of EEG backgrounds into these five groups is both the benefits and disadvantages of simplification, trading off ease of use with the problem of loss of nuance, in addition to the inevitability of encountering patterns that may not easily fit into one of the proscribed categories. Nevertheless, this system works surprisingly well, especially for normal or near-normal newborn EEGs near term. Additional characteristic waveforms that appear at specific CAs and are superimposed on these patterns, referred to as EEG graphoelements, are described later.

Normal neonatal EEG background patterns may be either continuous or discontinuous. The first step in classifying a background pattern is assessment of the degree of continuity. A discontinuous pattern is a pattern in which EEG activity seems to alternately "turn on" and "turn off" for varying amounts of time. In a continuous pattern, there are no recognizable

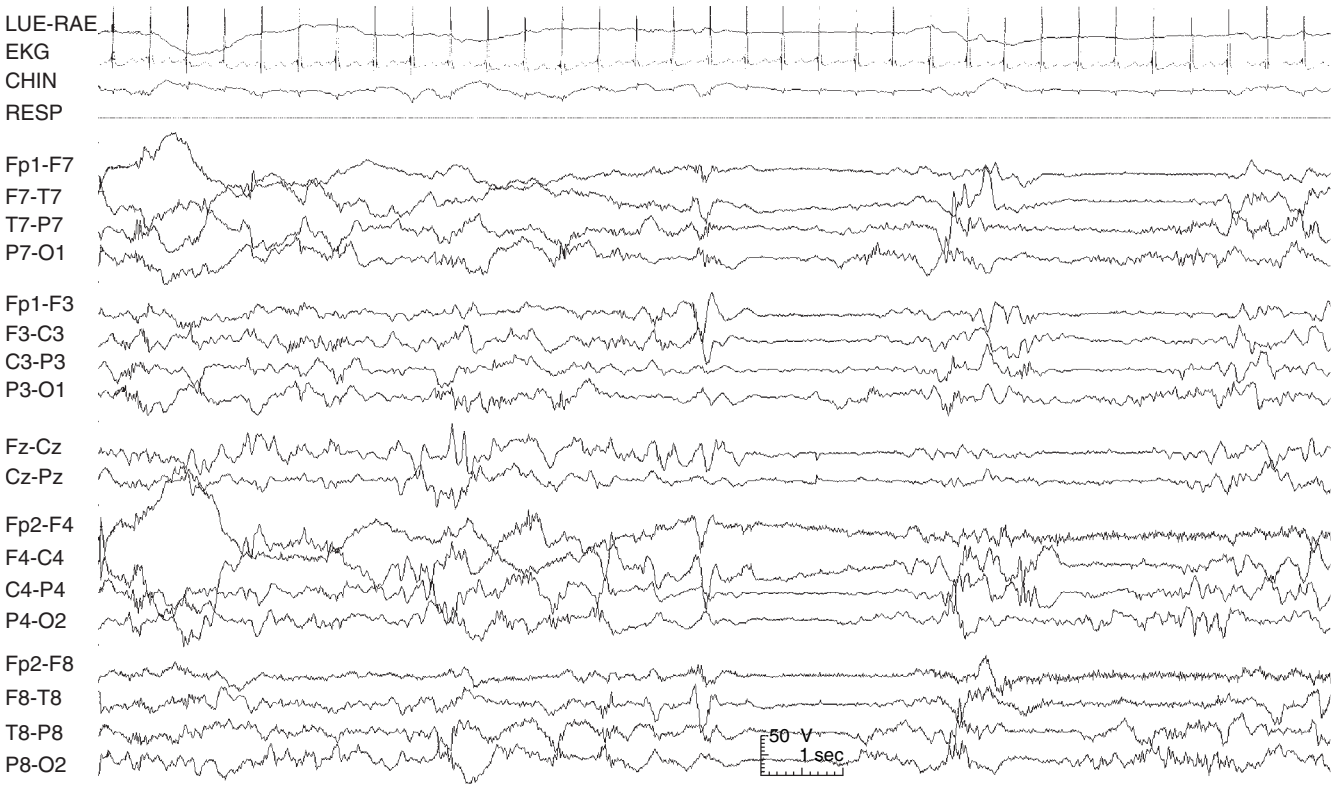

Figure 13-4 This page of EEG shows a transition from a continuous pattern, seen on the left half of the page, to a discontinuous pattern, seen on the right half of the page. The two periods of relative flattening (interburst intervals) seen on the right half of the page mark this as a discontinuous tracing. LUE, left under eye; RAE, right above eye.

regional pauses in activity (see Figure 13-4). The first three background patterns described here are continuous patterns, and the final two patterns are discontinuous patterns.

The Low-Voltage Irregular Pattern

As the name implies, this pattern consists of continuous low-voltage irregular (LVI), mixed frequencies, with delta and theta activity most prominent. Voltages generally range from 15 to 35 μV. An example is shown in Figure 13-5. As described later, the LVI pattern is seen during both wakefulness and active sleep. The LVI pattern is not expected to be seen during quiet sleep.

The Mixed (M) Pattern

The M pattern is similar to the LVI pattern, but with somewhat higher voltages and a more prominent contribution of slow activity. Continuous mixed frequencies are seen with a mixture of voltages (see Figure 13-6). The M pattern can be seen during any sleep stage. During active sleep, the LVI pattern is most characteristic, but the somewhat higher voltages of the M pattern may also be seen. Similarly, during wakefulness either the LVI or M pattern may be seen. In quiet sleep, the tracé alternant and high-voltage slow (HVS) patterns (described next) are most characteristic, but the M pattern may also be seen. Because it is possible to see the M pattern in any stage of wakefulness or sleep, polysomnographic findings and observed behaviors are key to correct determination of sleep stage.

The High-Voltage Slow (HVS) Pattern

The HVS pattern is characteristic of quiet sleep; it only rarely makes an appearance in other sleep stages. Like the LVI and M patterns, the HVS pattern consists of continuous, irregular mixed frequencies, but with higher voltages (50–150 μV). Delta frequencies are more prominent (see Figure 13-7). As described below, discontinuous patterns (tracé discontinu and tracé alternant) are the primary patterns of quiet sleep from the earliest post-conceptional stage to 38 weeks CA. As the baby gets closer to term, the tracé alternant pattern is replaced by the HVS pattern.

The LVI, M, and HVS patterns all consist of continuous irregular, mixed frequencies. The main distinguishing feature among these three continuous patterns is voltage.

The Tracé Discontinu Pattern

The tracé discontinu pattern (French for "discontinuous tracing") is a pattern of early prematurity, seen primarily at 30 weeks CA and before. As the name implies, tracé discontinu is a highly discontinuous pattern consisting of very high voltage polymorphic

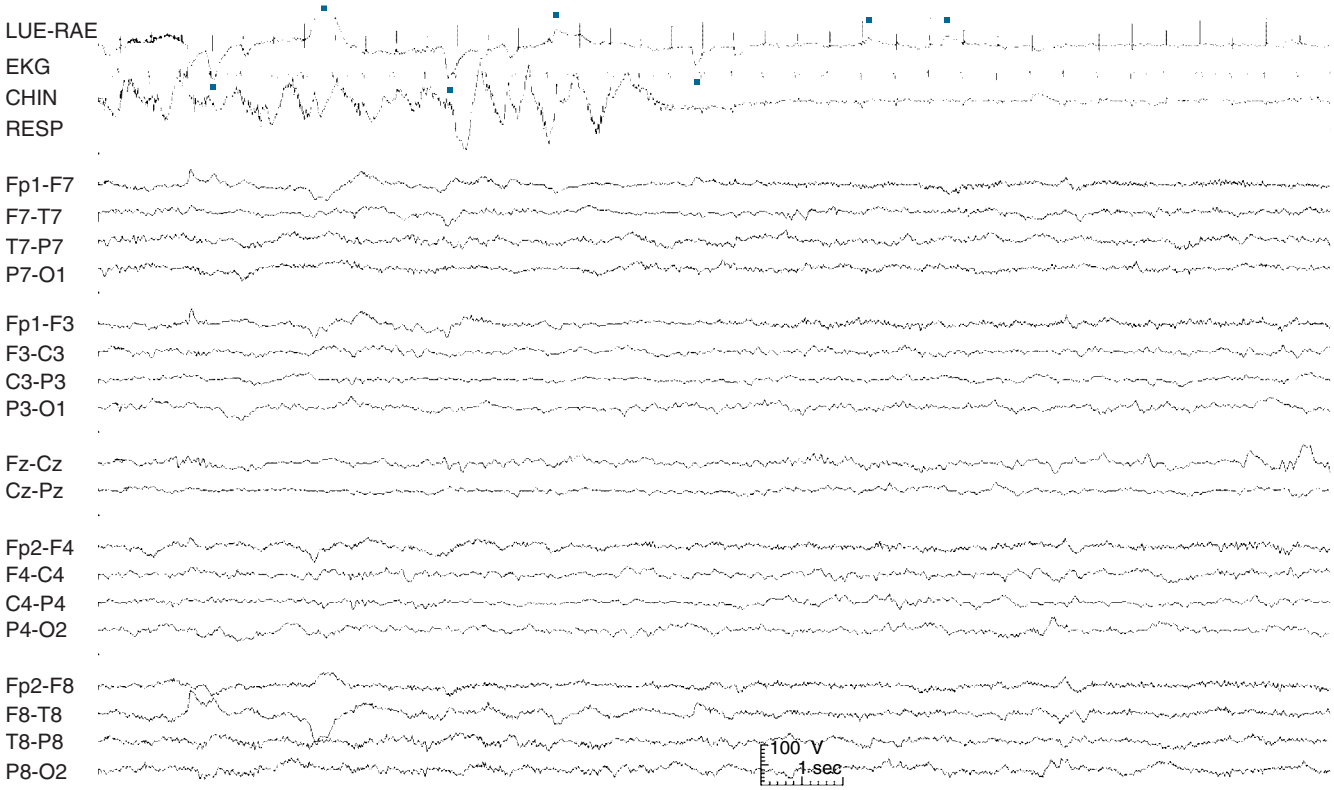

Figure 13-5 An example of a low-voltage intermixed (LVI) pattern is shown, with relatively nondescript mixed frequencies. The LVI EEG pattern is characteristic of both wakefulness and of active sleep. The triangular deflections seen in the top (ocular) channel represent rapid eye movement sleep (dots) indicating that this is an example of active sleep. LUE, left under eye; RAE, right above eye.

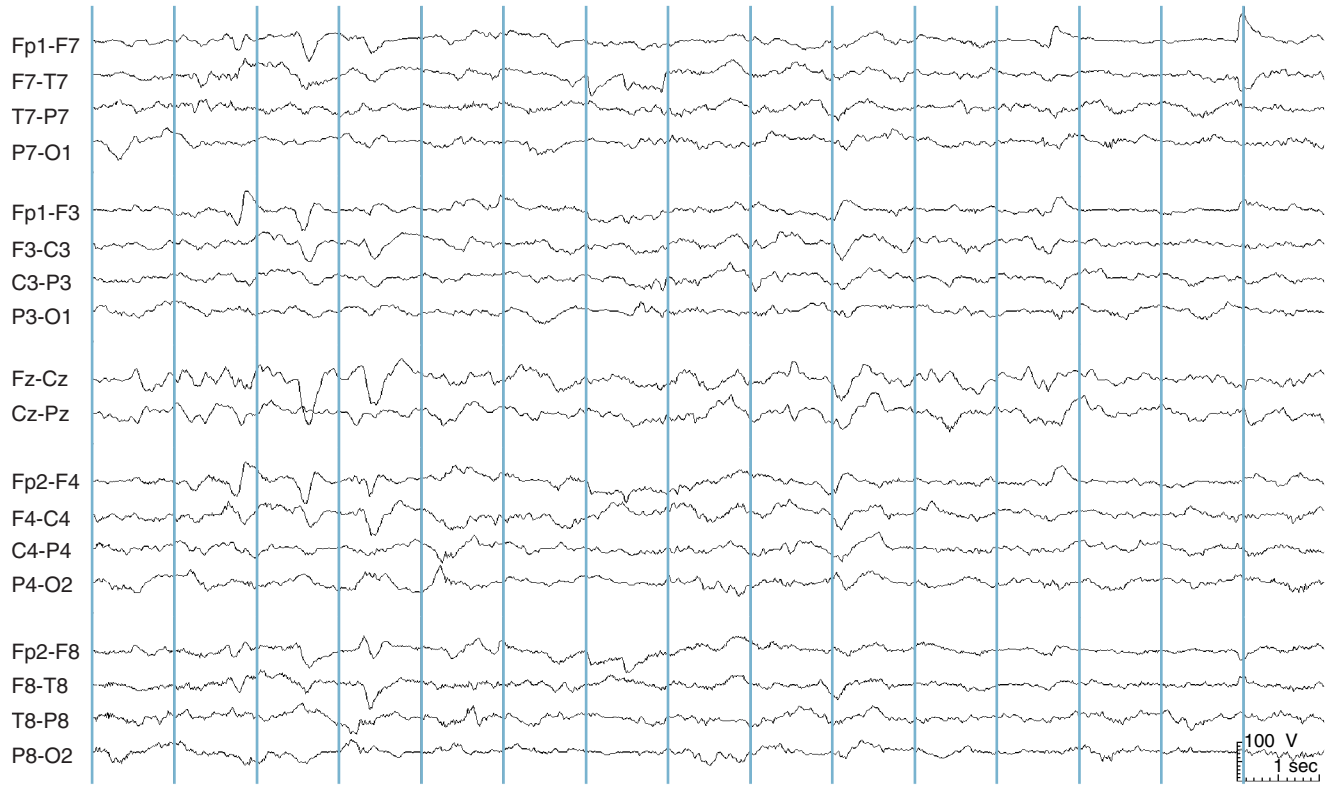

Figure 13-6 The mixed or "M pattern" is similar to the low-voltage intermixed (LVI) pattern, but with higher voltages. The M pattern may be seen in any of the sleep stages, including wakefulness, quiet sleep, and active sleep. Assignment of sleep state when the M pattern is present depends on other recording parameters such as technologist observations and information from the polysomnographic channels.

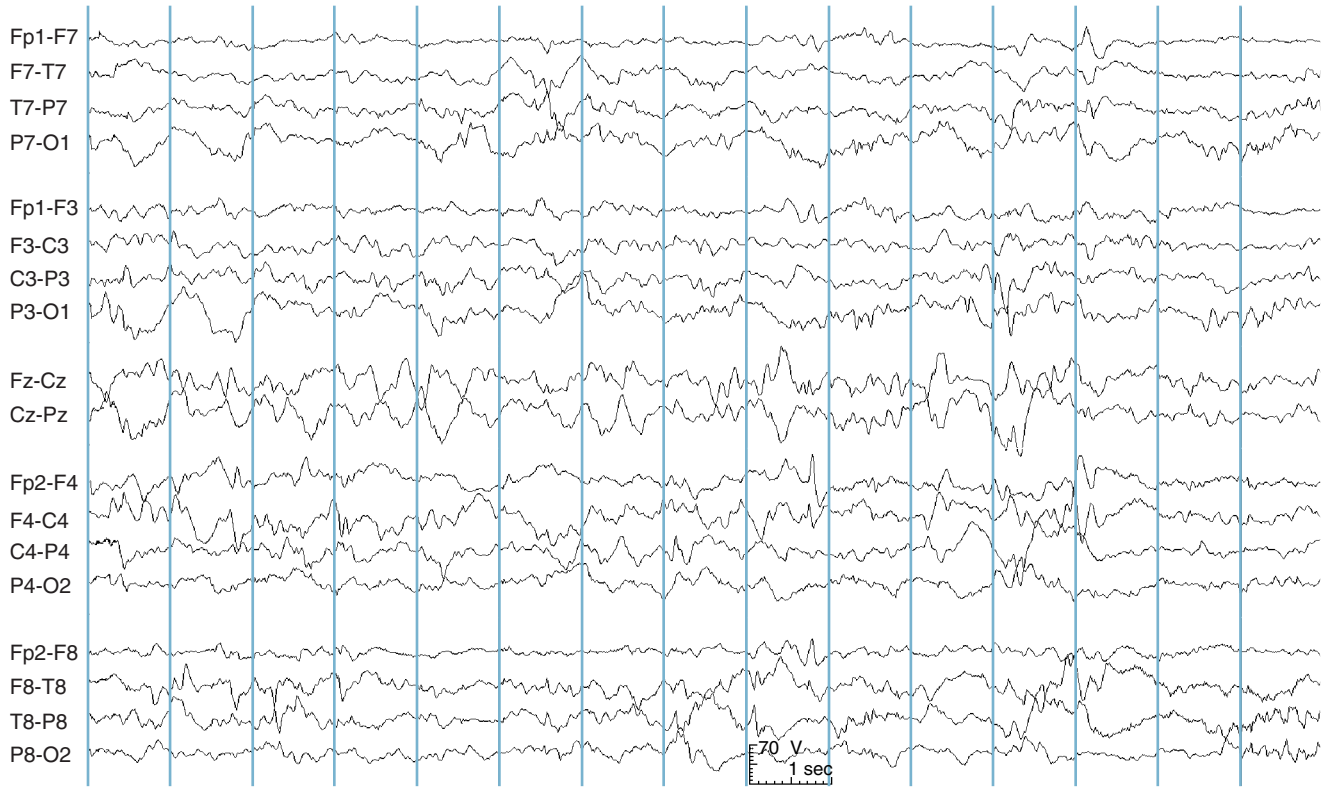

Figure 13-7 This segment of high-voltage slow (HVS) pattern was obtained during quiet sleep in a term newborn. Voltages are higher than were seen in the previous two patterns, but frequencies remain mixed and the waves are irregular. The HVS pattern is particularly associated with quiet sleep.

bursts, often containing large numbers of sharp features that may even resemble high voltage polyspikes (see Figure 13-8). The dramatic bursts of tracé discontinu are separated by equally dramatic flat periods that may exceed 10 to 20 seconds in length in the most premature babies (see Figure 13-9). Because of its resemblance to burst-suppression, a well-known pathologic pattern in adult EEG, it took some time for neonatal electroencephalographers to confirm that this was a normal pattern of early prematurity.

The Tracé Alternant Pattern

Tracé alternant (French for "alternating tracing") is the hallmark pattern of quiet sleep in newborns. Tracé alternant is a discontinuous pattern consisting of bursts of mixed activity lasting 2 to 8 seconds with interspersed flatter periods referred to as "interbursts" lasting 4 to 8 seconds (see Figure 13-10). Generally, the bursts and interbursts are of similar duration. The bursts normally contain a variety of activity, including sharp transient activity and also delta brush activity in more premature babies (described later).

When tracé alternant makes its first appearance after the 30 weeks CA, the quiet interburst periods are longer and flatter than at later CAs. Also, early on, the bursts show the least amount of synchrony between the two hemispheres. As the baby approaches term, the tracé alternant pattern evolves in three ways. First, the bursts are not as widely separated (the interburst intervals are shorter). Second, the periods between the bursts evolve from being relatively flat showing only small amounts of activity to showing increasing amounts of activity, so much so that as term approaches, it may become difficult to tell where a burst ends and a quiet period begins. Finally, the degree of interhemispheric synchrony of the tracé alternant bursts increases toward term, although it may never reach complete synchrony. The pattern shown in Figure 13-11 has, indeed, achieved complete synchrony, although this does not always occur. Even after term the degree of interhemispheric synchrony of tracé alternant is never required to exceed 75%, meaning that in normal babies, a small amount of asynchrony may always be seen.

The differences between tracé alternant and tracé discontinu are both qualitative and quantitative. Quantitative differences include longer interburst intervals, more sharp activity within bursts, and near complete synchrony in tracé discontinu compared with tracé alternant. Qualitatively, in tracé discontinu the interburst intervals are expected to be essentially flat, whereas varying amounts of continuous activity are expected during the interburst intervals of tracé alternant. Between 30 and 34 weeks CA, the evolution of tracé discontinu to tracé alternant during quiet sleep occurs on a continuum.

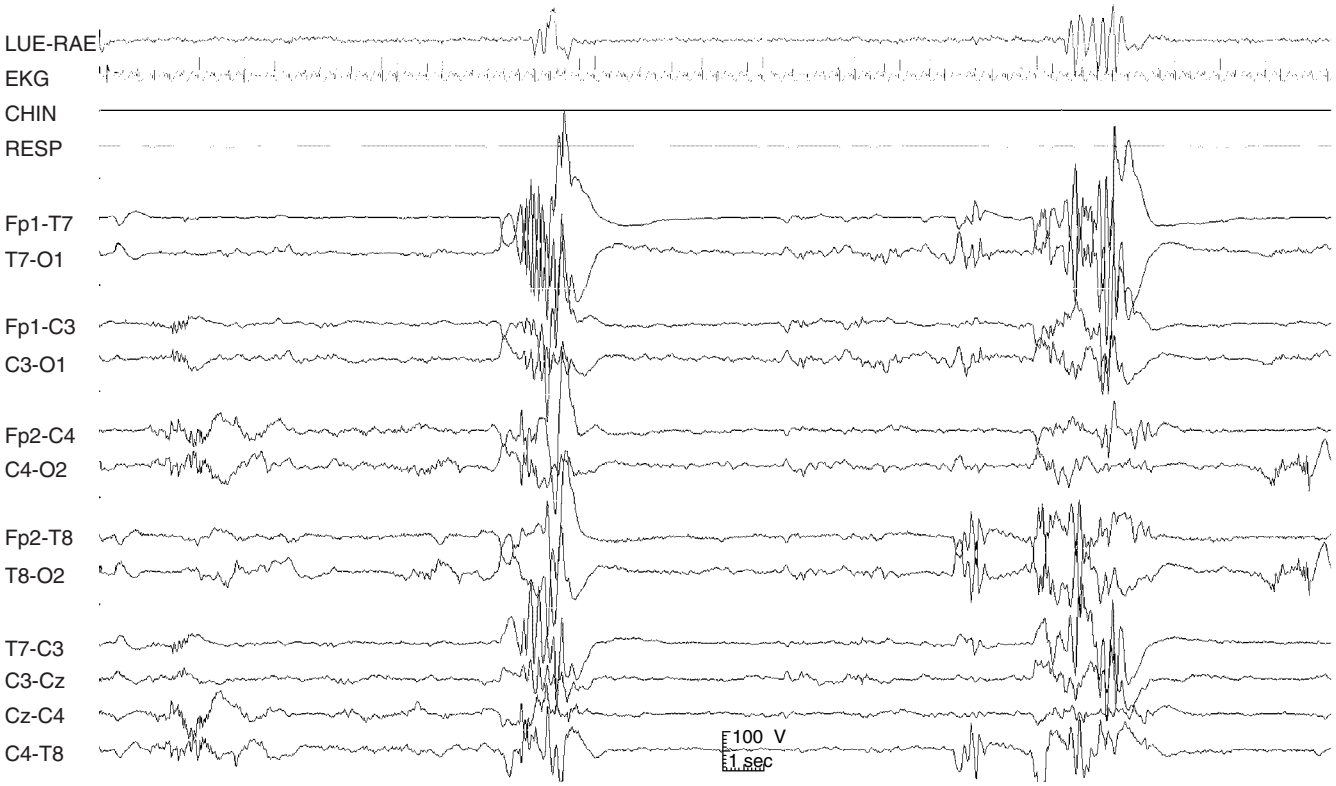

Figure 13-8 When first encountered, the tracé discontinu pattern may appear highly abnormal to the reader accustomed to interpreting adult EEGs. High-voltage bursts containing large amounts of polymorphic activity, often very sharp as in this example, are seen synchronously in both hemispheres. The bursts are separated by quiet periods of varying duration. LUE, left under eye; RAE, right above eye.

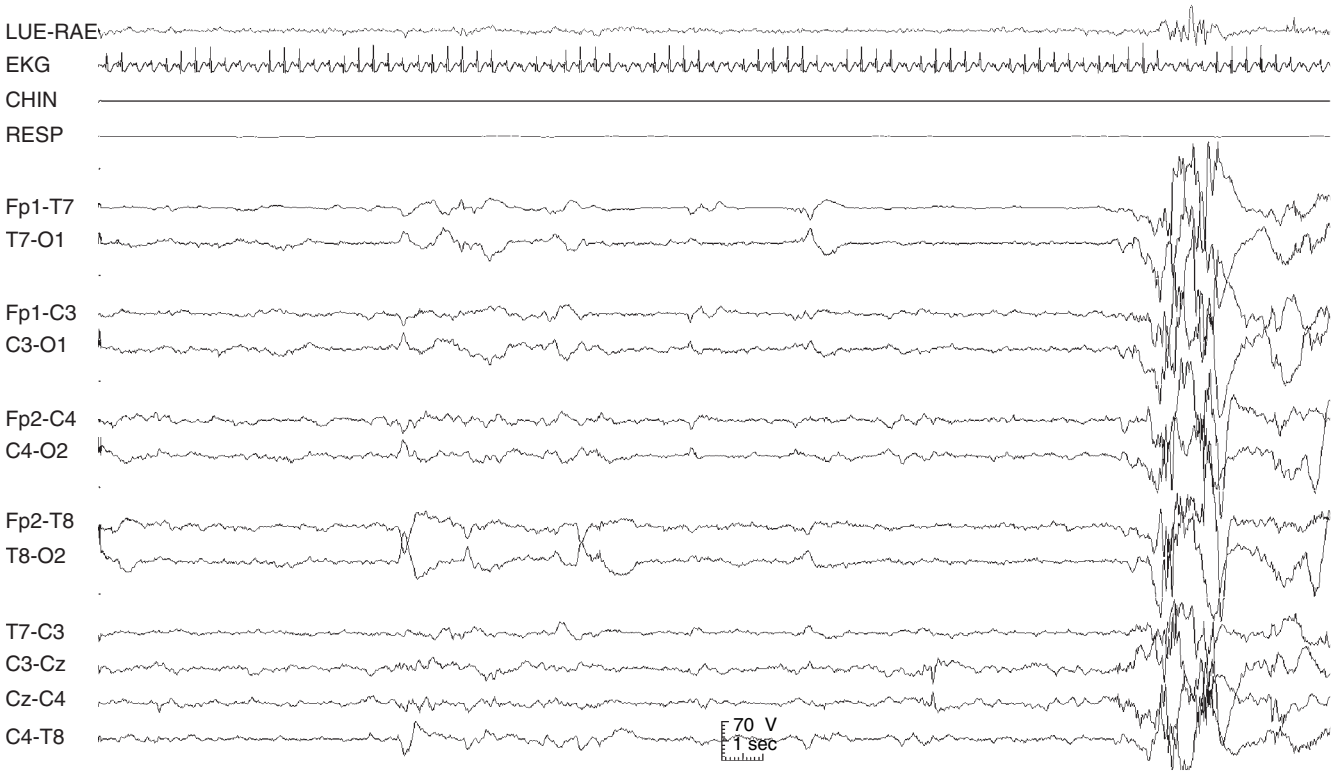

Figure 13-9 In this example of tracé discontinu the interburst periods are particularly lengthy; note the time scale at the bottom of the figure. The plentiful spikes seen within the bisynchronous bursts are considered a normal feature of the tracé discontinu pattern. At the earliest CAs, the flat periods between bursts can be quite lengthy, sometimes exceeding 20 seconds. LUE, left under eye; RAE, right above eye.

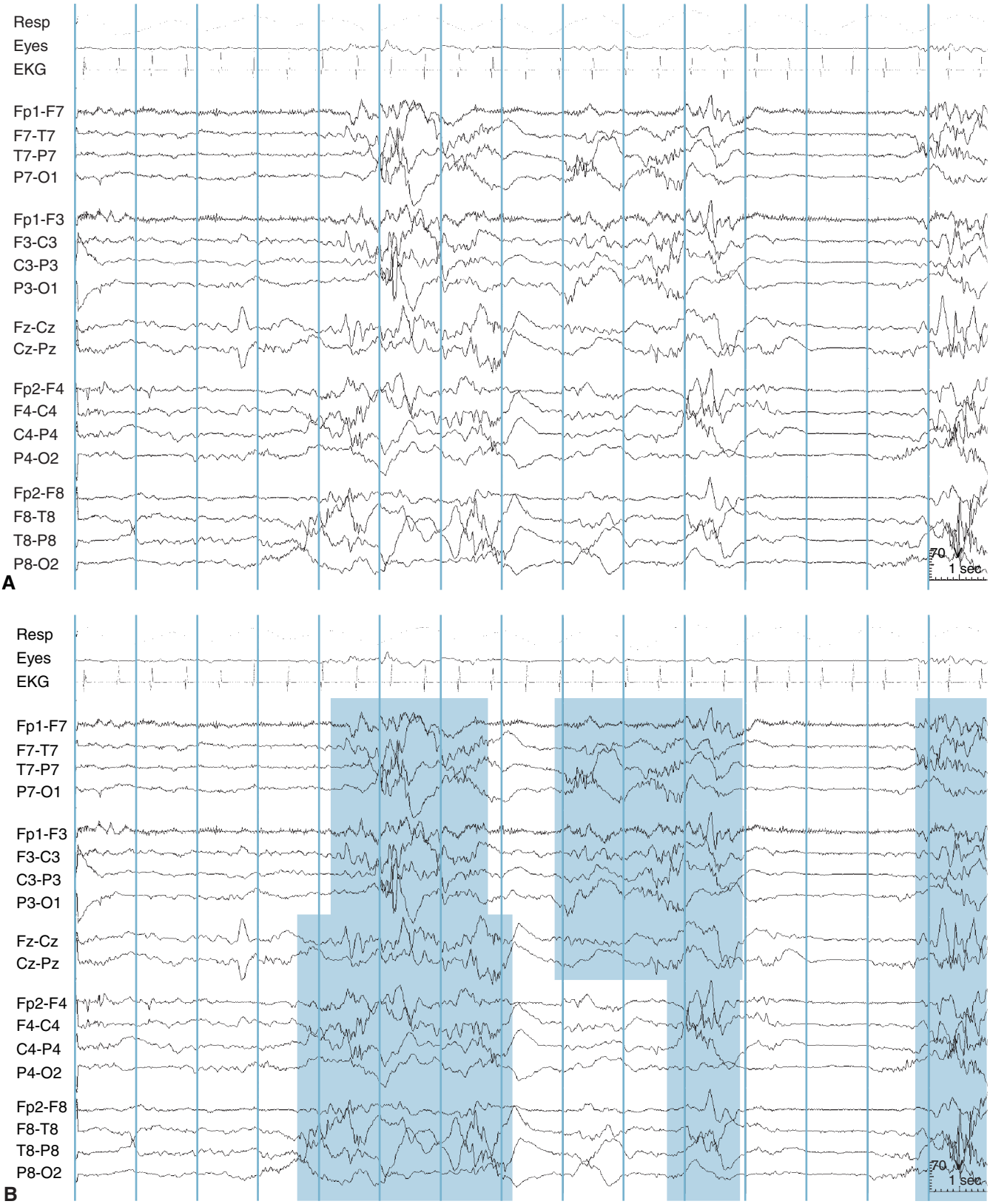

Figure 13-10 The distinctive, discontinuous pattern of tracé alternant is shown. In this example **(A),** most of the bursts are bisynchronous with bursting activity and suppressions occurring in each hemisphere more or less at the same time. Some amount of asynchrony is noted, however. The lower panel **(B)** shows the same page of EEG as was shown in Panel A, now with shading marking the approximate beginning and end of each burst. Note each burst contains a fair amount of sharp activity. The regular respirations and lack of eye movements confirm that this is an example of normal quiet sleep.

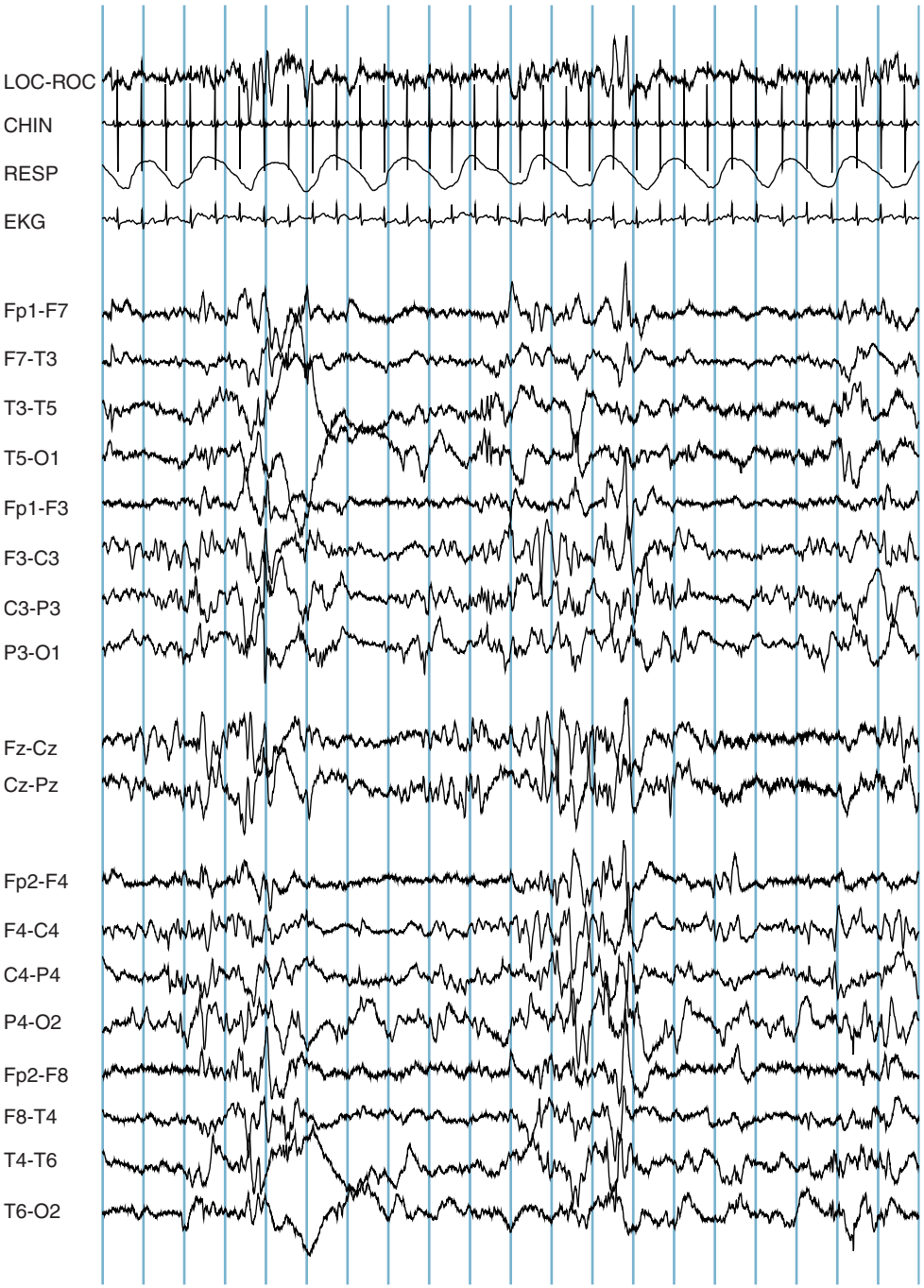

Figure 13-11 In this example of tracé alternant the bursts and suppressions are completely synchronous. The time scale has been compressed to make the discontinuous pattern easier to appreciate. Although clearly of lower voltage than the bursts, the periods of suppression contain a fair amount of activity. This and the high degree of synchrony suggest that the baby is near term.

The Evolution of Interhemispheric Synchrony

In contrast to tracé alternant, the tracé discontinu pattern is almost completely synchronous between the hemispheres. This leads to a distinctive sequence in the evolution of interhemispheric synchrony through the weeks of prematurity. In the most premature babies, there is nearly complete interhemispheric synchrony, and the tracé discontinu pattern persists (up to about 30 weeks CA). When the tracé alternant pattern first appears (at approximately 30 weeks CA), the pattern is initially significantly asynchronous. This is followed by a gradual return of interhemispheric synchrony as the tracé alternant matures as the baby approaches term. Therefore the EEG is synchronous in early prematurity, becomes moderately asynchronous in "middle" prematurity, and becomes synchronous again near term.

SLEEP STAGES IN THE NEWBORN NEAR TERM

The three main sleep stages of the newborn near term are *active sleep, quiet sleep,* and *wakefulness.* Fundamentally, the concept of "asleep" is defined by the outward appearance of the baby, with clinical sleep considered a state of persistent eye closure and wakefulness of eyes open.

Active Sleep

During active sleep, the baby is seen to squirm, grimace, and have an agitated appearance, yet the eyes are closed. In fact, the movements may lead an observer to think that the baby is on the verge of waking up. Respirations are irregular, and occasional respiratory pauses may be seen. Rapid eye movements of sleep are seen, both on the eye channels of the EEG and by casual observation of the baby's eyelids; movements of the corneal bulge can be seen through the baby's eyelids. The chin EMG lead picks up phasic bursts of muscle activity that correspond to facial muscle movements, such as grimacing or other movements. However, in between facial movements, chin EMG activity is low. The EEG shows an LVI pattern that is similar to what is seen during wakefulness (see Figure 13-12). Although most active sleep stages are typically associated with an LVI pattern, the first period of active sleep occurring as a baby falls asleep may show a somewhat higher voltage EEG pattern compared with later active sleep stages, such as an M pattern.

Active sleep in neonates is analogous to REM (dream) sleep in children and adults, although there are two interesting distinctions. First, although older subjects experience a form of paralysis during dream sleep, presumably so that dreams are not physically acted out, as the name implies, babies move actively during active sleep. Second, whereas the first REM sleep stages typically start only after a considerable time asleep in children and adults, newborns enter active sleep as their first sleep stage at the time of transition from wakefulness to sleep. REM sleep at sleep onset is not expected in adults, except in patients with narcolepsy in whom this phenomenon is one of the hallmarks of the syndrome.

Quiet Sleep

The term *quiet sleep* derives from the quiet appearance of the baby during this sleep stage. Respirations are deeper and regular, and there are few, if any, limb movements. Outwardly, the baby appears to be in a deep sleep state. REMS are not seen (see Figure 13-9). The chin EMG lead, perhaps surprisingly, shows a *high* level of tonic muscle activity, with comparatively more EMG activity than is seen between body movements in active sleep. After term, quiet sleep evolves into slow-wave sleep.

The EEG pattern seen during quiet sleep before term is the distinctive tracé alternant pattern, a discontinuous pattern over each hemisphere with periods of high-voltage mixed activity followed by periods of relative quiescence. As the baby approaches term, an HVS pattern gradually replaces the tracé alternant pattern during quiet sleep stages. During this transitional period, which occurs during the weeks just before and after term, some babies manifest an HVS pattern at the

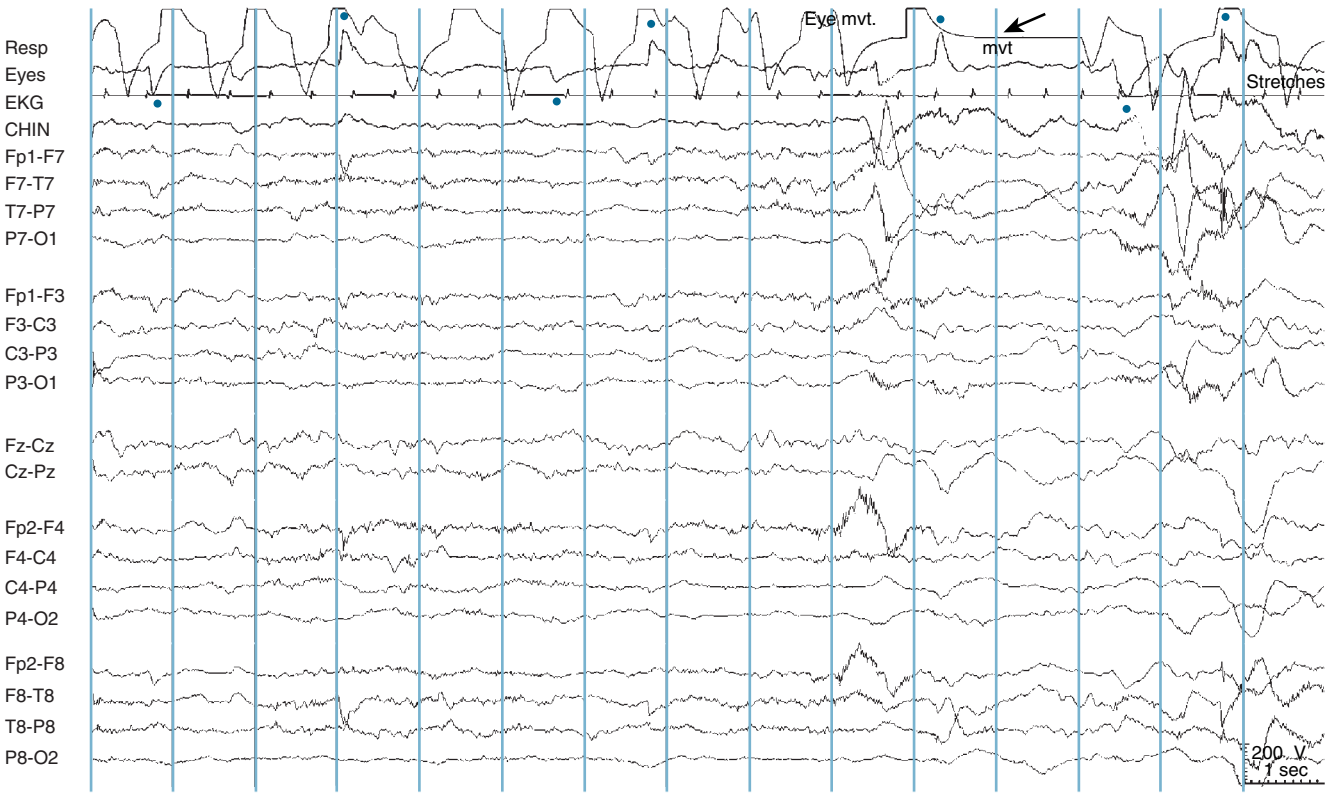

Figure 13-12 An example of active sleep is shown. Note the low-voltage irregular (LVI) pattern in which overall voltages are low save for examples of superimposed motion artifact. The respiratory (top) channel shows irregular respirations and a brief respiratory pause (arrow), consistent with active sleep. The oculogram (second channel) shows sharp deflections representing horizontal rapid eye movement of sleep (dots), the hallmark of active sleep.

beginning of a quiet sleep epoch, which may then transition to a tracé alternant pattern with deepening quiet sleep within the same epoch.

Wakefulness

In wakefulness, the baby's eyes are open and the activity level may vary considerably, from relaxed wakefulness (often seen just after feeding) to states of considerable agitation and crying. Breathing can be mildly irregular when the baby is calm to very irregular when the baby is more active. Recorded eye movements are irregular and include voluntary tracking and searching movements. These searching movements during wakefulness are usually easy to differentiate from REMS of active sleep which are faster and are more prominent to the horizontal plane.

The EEG pattern during wakefulness usually consists of an LVI pattern that may include a large amount of superimposed motion artifact depending on the baby's level of activity (see Figure 13-13). A somewhat higher voltage, M pattern may also be seen. The EEG patterns seen during wakefulness can be quite similar to those seen during active sleep, and at times it can be difficult for the reader to determine whether a page of EEG represents active sleep or wakefulness. This distinction is usually not difficult to make for the EEG technologist who is directly observing the baby and knows whether the baby's eyes are open or closed, the key factor in making the distinction. This situation highlights the importance of the technologist making frequent observational notes while recording newborn EEGs.

Transitional Sleep and Indeterminate Sleep

For completeness's sake, two additional sleep states are defined. The term *transitional sleep* is used for periods when the EEG transitions from one sleep state to another, including elements of both. Some babies spend a considerable amount of time in these transitional states. The term *indeterminate state* is used for stages that cannot be assigned clearly to any of the aforementioned groups.

Table 13-1 summarizes the features of the three main sleep states of the newborn after they have become well differentiated.

TYPICAL EVOLUTION OF NEONATAL SLEEP STAGES NEAR TERM

Sleep State Cycling in the Newborn

Typical sleep state cycling is depicted thus (W = wakefulness, AS = active sleep, QS = quiet sleep):

W→AS→QS→AS→QS→AS→W→W→W→AS→QS→AS→QS→W …

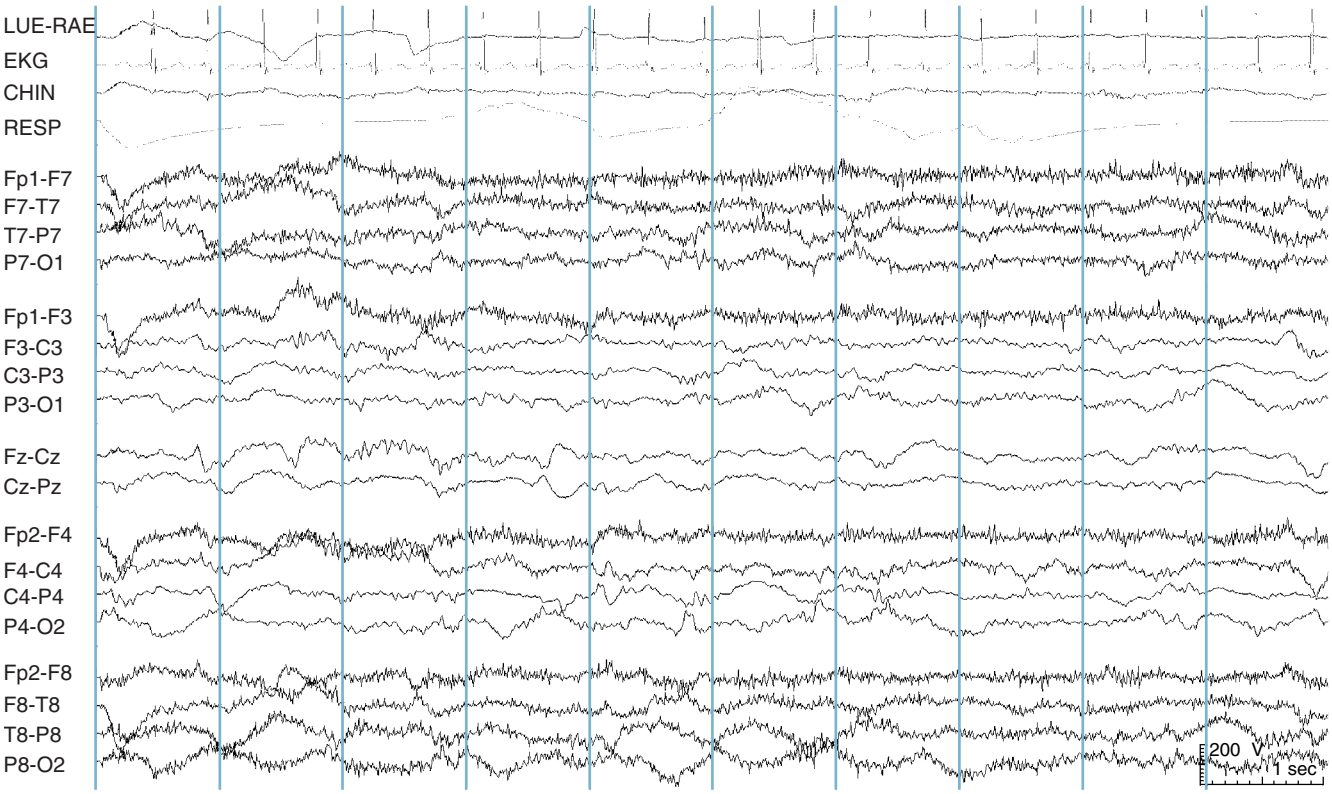

Figure 13-13 A page of wakefulness is shown with the EEG showing an M pattern. The technologist's observation that the baby's eyes are open and the moderate amount of muscle and motion artifact imply wakefulness. Eye movements are seen in the eye channel (labeled "LUE-RAE"); however they do not clearly have the classic "sharp" or triangular morphology of horizontal rapid eye movement sleep and likely represent voluntary or searching eye movements.

Table 13-1 Summary of Most Common Features of the Major Neonatal Sleep States

	Quiet Sleep	Active Sleep	Wakefulness
EEG Pattern	Tracé alternant (or high voltage slow)	Low voltage irregular	Low voltage irregular or mixed
Breathing	Very regular, deeper, slower	Irregular, with some pauses	Irregular, variable
Body Movements	Few movements, peaceful appearance	Squirming, sucking, grimacing	Calm or active, eyes open
Eye Movements	Little or none	Horizontal REMS	Consistent with having eyes open
Chin EMG	High tonic activity	Low tonic activity (measured in between movements)	Usually phasic

REMS, rapid eye movements of sleep; EMG, electromyogram.

Active sleep is usually the first sleep stage on falling asleep followed by quiet sleep. Periods of active sleep and quiet sleep then alternate until the next waking period. Periods of transitional sleep that may include the features of both sleep states may be interposed between well-defined active sleep and quiet sleep epochs. Newborns normally sleep for as many as 20 hours out of a 24-hour day. Each complete sleep cycle lasts approximately 60 minutes but with wide variation. Typically, newborns spend about half of their sleep time in active sleep and half in quiet sleep. The fraction of sleep time spent in dream sleep decreases with age; adults spend only about 20% of the night in REM sleep.

After remaining in active sleep for approximately 20 to 25 minutes, if the baby remains asleep, a transition to quiet sleep is expected. The changes associated with the transition from active sleep to quiet sleep are more dramatic than those associated with transition from wakefulness to active sleep. First, as the baby's sleep quiets, muscle and motion artifact disappear from the record. The respiratory pattern becomes very regular, and eye movements are rare. A tracé alternant pattern then appears (or HVS in infants closer to term). The baby thereafter alternates between quiet sleep and active sleep until arousal. Between sleep epochs, brief transitional states may be seen with elements of both types of sleep present at the same time (e.g., irregular breathing accompanying a tracé alternant pattern).

EEG Architecture in More Premature Infants

The orderly sleep structure described in the previous section is a characteristic of infants nearing term. Very premature newborns, however, lack this sleep structure. The earliest EEGs in clinical practice are recorded in babies at 23 to 24 weeks gestational age, which is considered near the limit of viability. In fact, EEGs are only occasionally obtained in such premature infants, partly because of the extreme fragility of their skin but also because seizures are believed to be uncommon at these very early CAs. At these early gestations, the predominant EEG background is tracé discontinu, consisting of bilateral complex bursts separated by prolonged periods of electrical quiescence. The period of electrical quiet may last longer than 20 seconds. Between 24 and 30 weeks CA, there is a tendency for the quiet periods to become shorter and the amount of activity during the interburst to increase. It may come as a surprise to learn that there is no reliable relationship between sleep state and EEG appearance before 30 weeks CA; the tracé discontinu pattern is seen during both wakefulness and sleep, even though periods of wakefulness and sleep are clinically distinguishable.

The Emergence of Continuity

The first state seen with continuous activity in the premature appears at approximately 30 weeks CA. At that time, the continuous LVI or M patterns (with REMs) are first seen during active sleep. Continuous activity during wakefulness first becomes well established at approximately 34 weeks CA. The final sleep stage to manifest continuous activity is quiet sleep. As described earlier, the discontinuous tracé alternant pattern is typically seen during quiet sleep, but at approximately 38 weeks CA the continuous HVS pattern makes its first appearance. During this transition, both tracé alternant and HVS patterns may be seen during quiet sleep at different times in the same baby. Even though the HVS pattern predominates during quiet sleep after term, fragments of the discontinuity related to the tracé alternant pattern may be seen during quiet sleep up to 46 to 48 weeks CA. After 48 weeks CA, any discontinuity in the EEG is considered abnormal. In summary, the EEG first becomes continuous at 30 weeks CA in active sleep, at 34 weeks CA in wakefulness, and at 38 weeks CA in quiet sleep.

AN ORDERLY APPROACH TO NEONATAL EEG INTERPRETATION

The first step in visual analysis of a page of newborn EEG is asking the question, what state is this? As the EEG is sequentially examined from beginning to end, the reader attempts to identify the various sleep states described in Table 13-1: wakefulness, active sleep, and quiet sleep. Do the sleep stages have the expected structure according to the baby's reported CA? For example, in a baby nearer to term, during wakefulness is there an LVI or M pattern? Often the presence of motion artifact in the tracing and technologist comments confirm that the child is awake and active. As the baby falls asleep, the reader will expect to see a first sleep stage, most likely

active sleep. Because wakefulness and active sleep are both associated with either an LVI or M pattern, the transition may not be obvious based on the EEG alone. Active sleep is marked by closed eyes and the appearance of REMs. After a period of active sleep, does the EEG become discontinuous (tracé alternant)? Does the baby quiet, and do respirations become regular with disappearance of REMS, marking the onset of quiet sleep? The presence of this type of normal sleep architecture is considered a positive clinical sign. Although the absence of expected sleep architecture may be related to CNS pathology, it is important to keep in mind that there are many other possible explanations for disrupted sleep architecture (noises, medications, procedures), especially on hospital inpatient units. In the absence of other explanatory circumstances, the fewer features of normal sleep stage structure noted, the greater the worry of brain pathology.

EEG GRAPHOELEMENTS

During the late 1950s and early 1960s, in addition to describing neonatal sleep states, the "French school" of electroencephalography described certain specific waveforms in the normal newborn EEG that appeared and disappeared at specific CAs, calling these features *graphoéléments*. The concept of EEG graphoelements remains useful. Familiarity with the different neonatal EEG graphoelements and the CAs and sleep states during which they are expected to appear helps the reader "date" the EEG in terms of apparent CA. It also helps to avoid labeling normal elements as abnormal.

Temporal Sawtooth Waves

Temporal sawtooth waves are seen in the EEG most prominently between 26 and 32 weeks CA, declining thereafter. They are a hallmark finding of the EEG between 28 and 30 weeks in particular. Temporal sawtooth waves appear as 4- to 6-Hz sharply contoured theta waves of varying voltage seen in each midtemporal area (see Figure 13-14). Because sleep states are not yet well defined at these CAs, they are not particularly associated with a specific state.

Delta Brushes

The delta brush is one of the most distinctive waves of prematurity. Delta brushes have also been called *beta-delta complexes* and *ripples of prematurity*. They consist of a delta wave with superimposed fast activity that may have a wide range of frequencies, from 8 to 22 Hz (see Figure 13-15). Delta brushes show a predilection for the posterior quadrants (central, temporal, parietal, and occipital areas) and are not often seen frontally.

Delta brushes make their first appearance at 26 to 28 weeks CA at a time when sleep stages are not well differentiated. They are initially seen in the central areas. They reach their peak density in the EEG at about 34 weeks CA, by which time they are also prominent in the occipital areas. When quiet sleep becomes a distinct sleep stage, delta brushes are particularly seen as a part of the tracé alternant pattern of quiet sleep; they are only rarely seen during well-established active sleep or wakefulness. By 39 to 40 weeks CA delta brushes have all but disappeared and should be absent

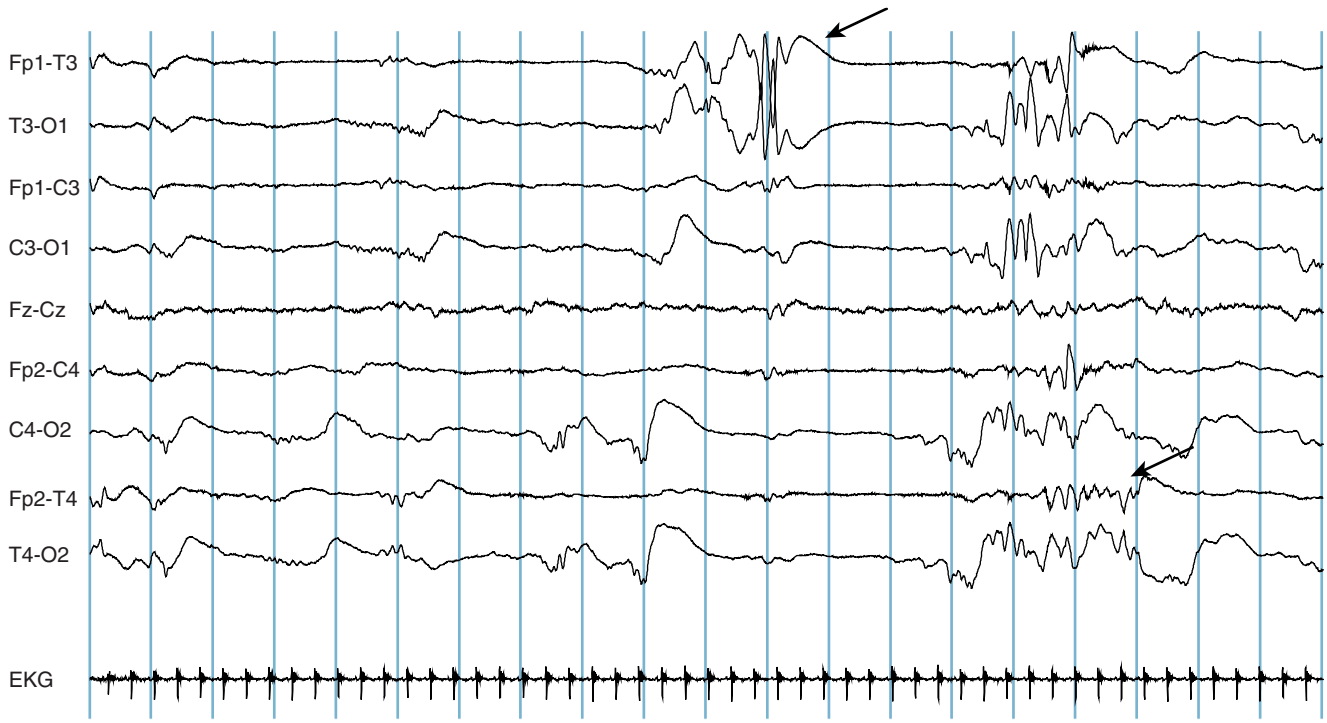

Figure 13-14 Temporal sawtooth waves are among the most characteristic waves seen in the EEG between 27 and 28 weeks CA. Sawtooth waves consist of brief runs of sharp theta activity seen over each temporal lobe (arrows).

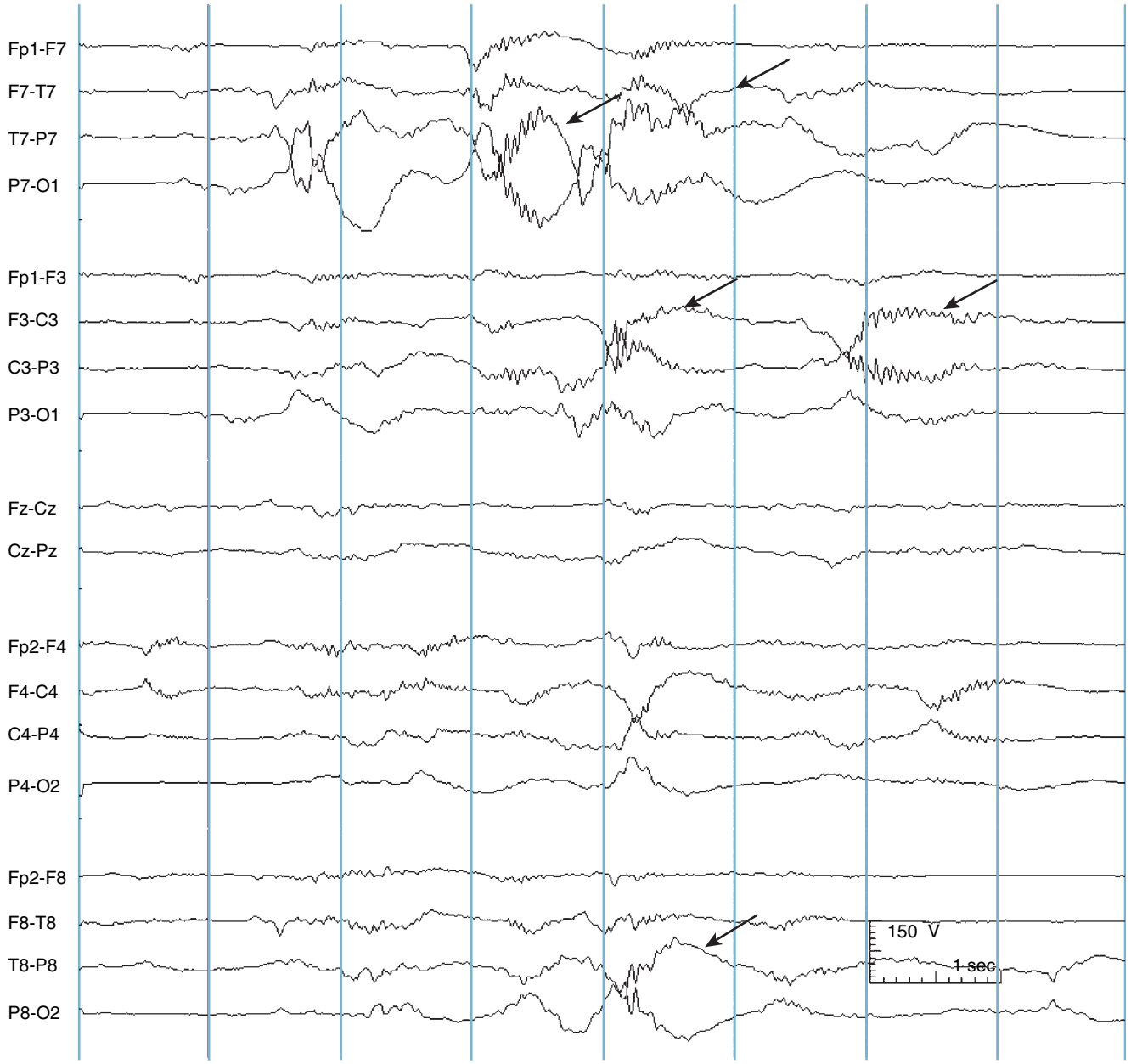

Figure 13-15 Delta brushes consists of a "brush" or "ripple" of activity riding on a delta wave (arrows). Delta brush activity peaks at 34 weeks CA. The amplitude of both the fast activity and the slow component vary in different examples. Delta brush activity tends to be most prominent in the posterior quadrants, as seen in the segment.

by 44 weeks CA. Similarly, by this CA the tracé alternant pattern has been replaced by the HVS pattern during quiet sleep.

Sharp Transients

In the wider world of electroencephalography, spikes and sharp waves have the connotation of both being abnormal and associated with seizures. In neonatal electroencephalography, both of these biases must be reversed. First, not only may sharp activity in the EEG be normal, certain sharp discharges are actually expected in newborn EEGs as normal graphoelements. Second, when *abnormal* sharp activity is seen in newborns, such discharges may be associated with brain injury but not specifically with seizures as they are in older individuals. Therefore even abnormal sharp waves in babies are not considered an epileptiform abnormality. To avoid the negative connotation of the terms "spike" and "sharp wave," such discharges in the newborn EEG are generally referred to as *sharp transients*.

Frontal Sharp Transients

Frontal sharp transients, also referred to by the original French term *encoches frontales*, consist of high-voltage, usually bilateral, frontal sharp waves (see Figure 13-16). They may have a biphasic morphology, and the primary phase

has negative more often than positive polarity. Occasionally unilateral examples are seen, but when one-sided, the number of discharges seen on each side should remain similar. Encoches frontales are seen most often in quiet sleep and are only rarely seen in active sleep or wakefulness; large numbers of frontal sharp transients during wakefulness or active sleep are considered abnormal. These transients first appear at about 34 weeks CA and become less common after term. They should not be seen at all after 48 weeks CA.

Temporal Sharp Transients

Temporal, central, or centrotemporal sharp transients with negative polarity occur in normal newborn EEGs and may be seen during both the LVI and higher voltage M, tracé alternant, or HVS portions of the tracing (see Figure 13-17). In fact, such sharp activity is an expected feature of the discontinuous neonatal EEG patterns, tracé alternant and tracé discontinu; sharp activity may be especially plentiful within the bursts themselves. The sharp transients described here are those that occur outside the context of the bursts.

There is probably no lower CA limit of normal for temporal sharp transients. Temporal sharp transients are a particularly common feature of the newborn EEG between 35 and 42 weeks CA. After 44 weeks CA, they are uncommon, and they are considered abnormal after 48 weeks CA.

The Emergence of Vertex Waves and Spindles

Sleep spindles appear in the EEG between 6 and 8 weeks after term (44–46 weeks CA), and vertex waves of sleep follow soon after at 8 weeks or thereafter. During infancy and up to 18 to 24 months of age, there is a tendency for sleep spindles to occur asynchronously over each hemisphere. After 2 years of age, sleep spindles occur synchronously over both hemispheres. During the transitional stage between spindle asynchrony and synchrony during the second year of life, the spindles may be asynchronous in light sleep but become more synchronous as Stage II sleep deepens. Spindle duration tends to be longer in infants (several seconds) and shortens toward adulthood (approximately 2 seconds).

When Are Sharp Transients in the Temporal, Frontal, and Other Areas Considered Abnormal?

Establishing strict criteria of normality for neonatal sharp transients that occur outside of tracé alternant bursts has not been easy. It has already been stated that some amount of frontal and temporal sharp transient activity should not just be "passed" as normal but is actually an expected feature of the newborn EEG. However, it has been shown statistically that abnormal babies manifest higher numbers of sharp transients than do normal babies. This pair of facts suggests that there may be some specific upper limit to the number of sharp transient activity a baby may have beyond which a tracing should be considered abnormal. An exact limit, however, has been difficult to define. As readers of neonatal EEGs gain experience, they become accustomed to the number of sharp transients that are usually seen in a newborn record, giving a benchmark against which to decide how many is "too many." One sharp transient per minute has generally been considered to be clearly within the normal range, though exceeding this frequency by some amount is not necessarily considered abnormal. When it is felt that a tracing clearly shows too much sharp transient activity, the abnormality is referred to as "excess neonatal sharp transients." The reader must take care not to be overly aggressive in calling neonatal EEG tracings abnormal solely on the basis of the abundance of these discharges, keeping in mind that a baseline number of these transients is considered completely normal.

Normal neonatal sharp transients can probably be seen in any brain area, although there is a tendency for transients to be more widely distributed at earlier CAs and to concentrate in the central, temporal, and frontal areas nearer term. Transients seen in the midline electrodes are more often associated with abnormality.

Other Features of Abnormal Sharp Transients

Apart from the frequency of their appearance, certain other features are felt to mark sharp transients as abnormal. These include *very high voltage* (>150 μV), *asymmetrical appearance* (considerably more on one side than the other), *polyphasic* rather than the usual monophasic or diphasic morphology, *repetitive* discharges in one location, and, sometimes, *positive polarity* (discussed later). Certain locations are felt to have a higher association with abnormality than others. Sharp transients occurring in the *midline,* such as at the Cz electrode, are more often seen in abnormal babies. Some authors feel occipital sharp transients are abnormal, but others do not. When a sharp transient is deemed abnormal, it is felt to mark a nonspecific brain injury rather than an epileptiform abnormality. Figure 13-18 shows excessively repetitive sharp transients in the left temporal area of a baby who experienced a stroke in that area.

Central and Temporal Positive Sharp Waves

When first described, central *positive* sharp waves in newborns were felt to be strongly associated with intraventricular hemorrhage (IVH). These central positive sharp waves are now understood to be associated with multiple disorders; however, they are particularly characteristic of injury to the deep white matter structures in premature infants, including IVH and periventricular leukomalacia. The positive polarity of such sharp waves is indicated by the distinctive phase reversal seen in bipolar montages in which the peaks of the sharp wave point away from (rather than toward) each other (see Figure 13-19). Temporal positive sharp waves have

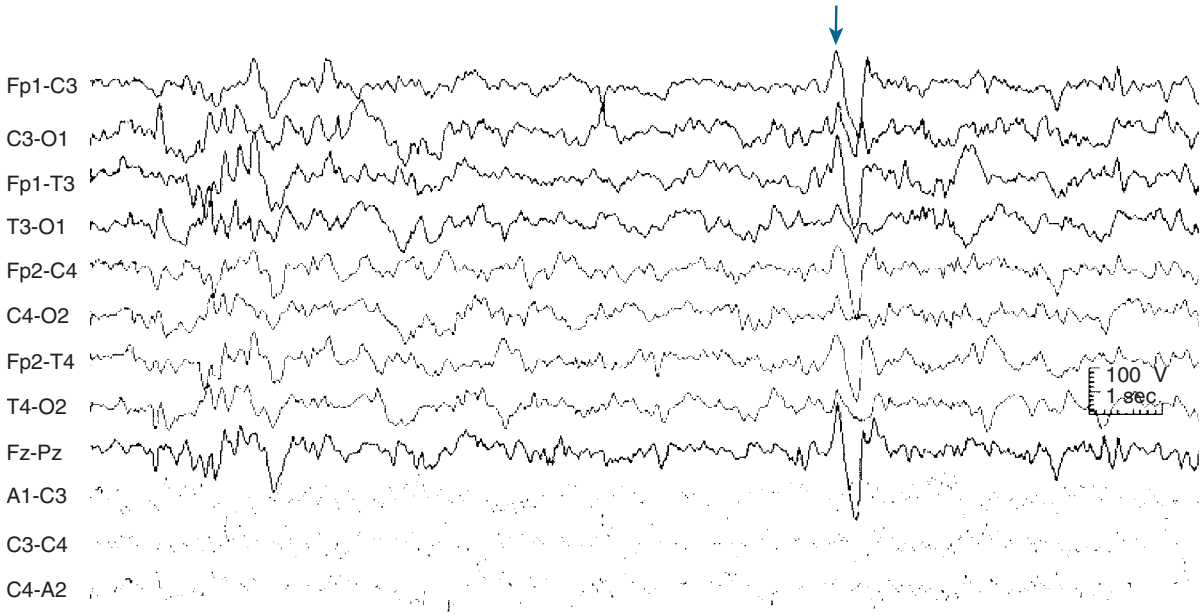

Figure 13-16 A single frontal sharp transient is seen (arrow) in this double-distance montage.

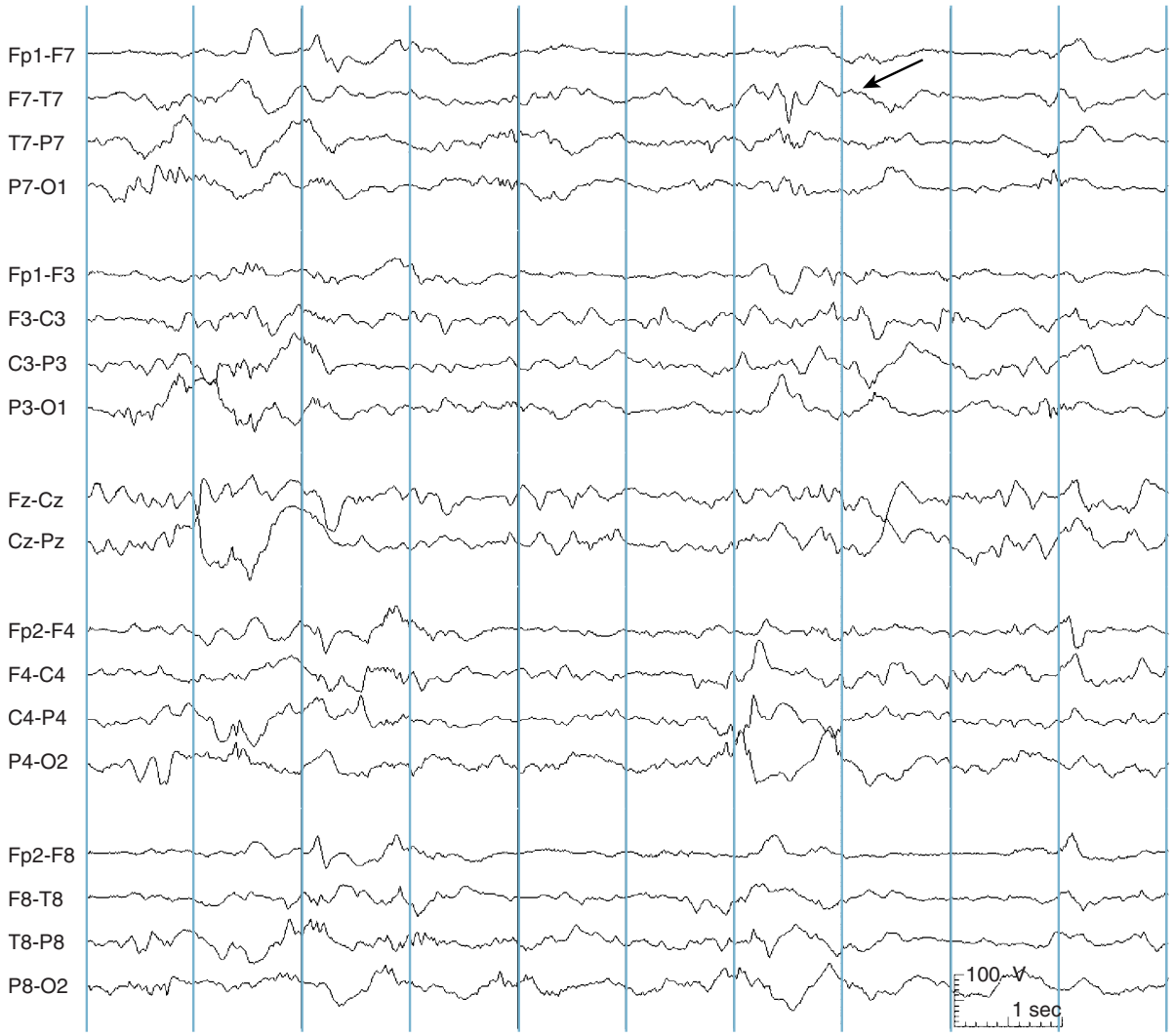

Figure 13-17 A temporal sharp transient is seen, maximum in the T7 electrode (arrow). Normal temporal sharp transients usually occur singly, are of low to moderate voltage and should be seen to the same extent over each temporal lobe throughout the record. *(Image courtesy of Dr. Sanjeev Kothare.)*

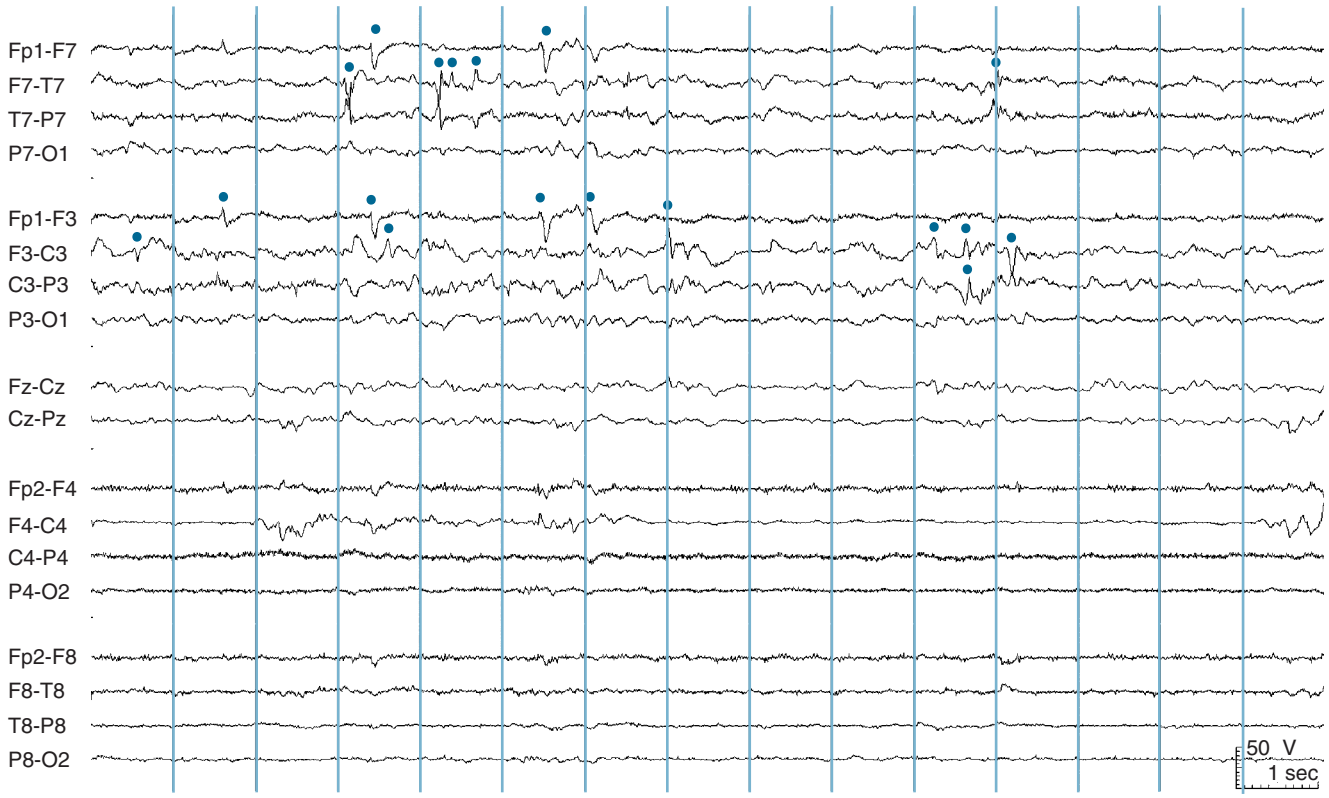

Figure 13-18 Multiple sharp transients are seen over the left hemisphere (dots) but not over the right. Although not of particularly high voltage, the repetitive and asymmetric nature of these transients marks them as abnormal. This baby experienced a stroke in the left hemisphere.

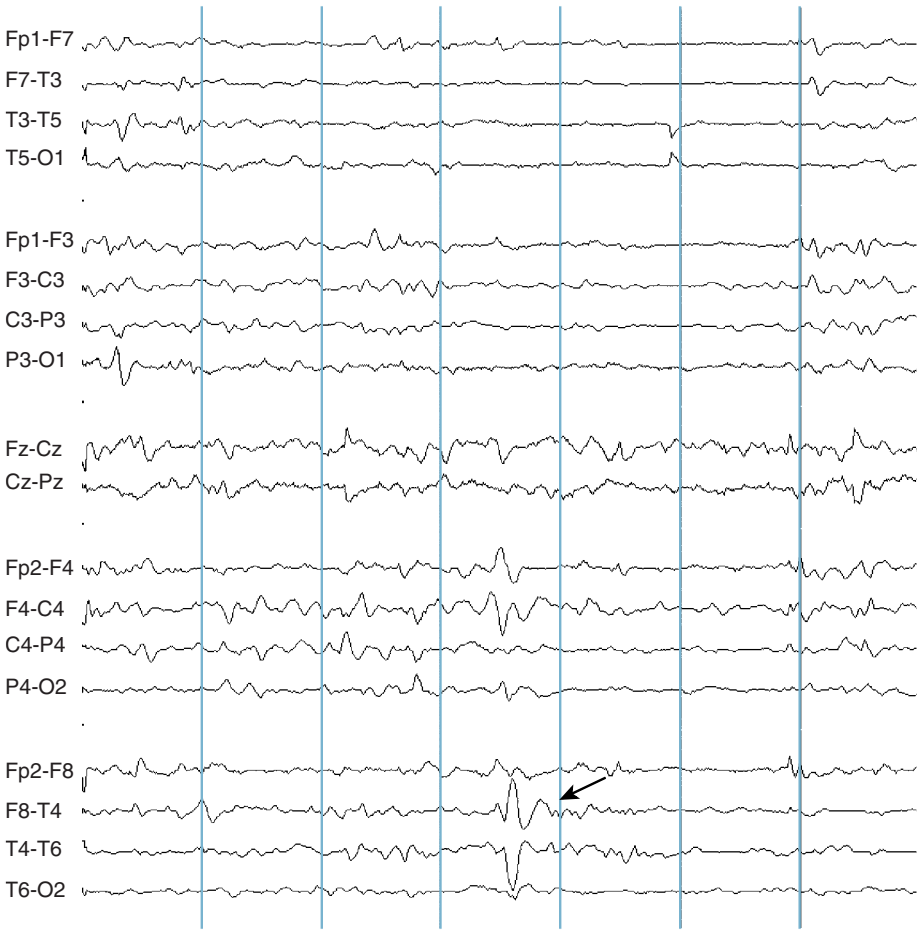

Figure 13-19 A positive sharp wave is noted in the right midtemporal area with a positive phase reversal seen in T4 (arrow). Neonatal positive sharp waves have been associated with deep white matter lesions.

also been associated with IVH and hypoxic-ischemic injury in the past; however, they have also been described in normal infants, possibly related to the sharp temporal theta discharges (sawtooth waves) that have already been described.

BACKGROUND ABNORMALITIES OF THE NEONATAL EEG

Of all the parameters that can be examined with regard to the neonatal EEG, the feature most predictive of a baby's final developmental outcome is the EEG background. The topic of neonatal EEG background was comprehensively reviewed by Holmes and Lombroso (1993). Various background abnormalities encountered in the neonatal EEG are described next, generally in order of decreasing severity.

Electrocerebral Inactivity and Voltage Depression

Electrocerebral inactivity (ECI) implies a complete lack of electrical activity recorded from the brain. The term should be reserved for recordings that have been performed with stringent technique (The proper recording techniques for possible ECI tracings and the concept of brain death are discussed in more detail in Chapter 12, "EEG Patterns in Stupor and Coma," but are summarized here as they pertain to newborns). Recording technique includes the use of double-distance electrodes, proper electrode impedances, confirmation that the EEG apparatus is connected properly by tapping the individual electrodes, and that the baby has received noxious stimulation to try to elicit EEG activity. The tracing must be of adequate duration and the cutoff frequency for the low filter should be set adequately low (≤1 Hz). Sensitivities of 2 μV/mm should be used, at least for a portion of the tracing. At such high amplifier gains, it can sometimes be difficult to distinguish true brain wave activity from electrical artifact. Synonyms for ECI include electrocerebral silence and "isoelectric" EEG (see Figure 13-20).

Note that the presence of an ECI tracing is not equivalent to the diagnosis of brain death. The diagnosis of brain death must be backed up by multiple elements of the neurologic examination and sometimes by specific types of neuroimaging; the specific legal definition used to declare brain death depends on the jurisdiction and is particularly difficult to apply in the case of newborns. It should not be surprising that an EEG showing ECI is not equivalent to brain death. The scalp-recorded electroencephalogram records electrical activity from the cortical surface, but the diagnosis of brain death implies complete inactivity of both the brain surface and deeper brain structures, such as the brainstem (medulla, pons, and midbrain), structures that are distant from scalp EEG electrodes. In fact, in most settings, an EEG recording is not absolutely required to establish the diagnosis of brain death.

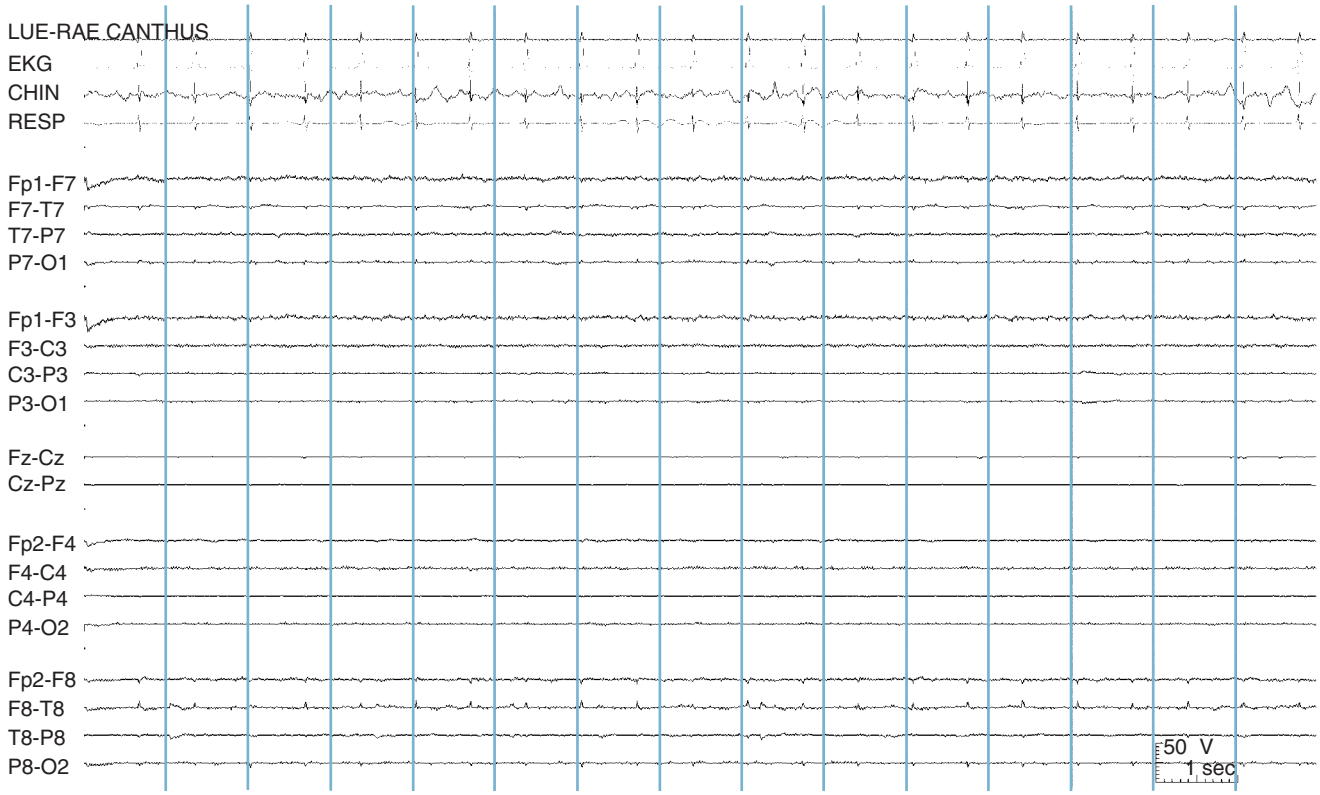

Figure 13-20 This page of EEG shows absence of any definite electrocerebral activity in an asphyxiated newborn. At the high amplifier gains used for such recordings, artifacts may become prominent in the tracing. In this example, the low-voltage electrocardiogram artifact noted in several channels would not be mistaken for brain waves because of its rhythmic, monomorphic features.

Not infrequently, the reader will encounter an EEG in which no definite electrocerebral activity can be identified but that has not been recorded with the strict technique described here and in Chapter 12, "EEG Patterns in Stupor and Coma." Such tracings may be described as "flat" or as showing "no definite electrocerebral activity," but in these cases, the more absolute designation of ECI should not be used. In such cases, the report should also indicate that the strict technique necessary to make a diagnosis of ECI was not used for the recording.

Of course, when the diagnosis of ECI is made, the lack of electrocerebral activity must persist throughout the whole tracing, and the recording must be of sufficient duration. It is important to note that some babies experience a complete but transient suppression of EEG voltages after a seizure. The EEG tracing may similarly flatten and recover after a transient hypoxic-ischemic insult. In such cases of transient flattening, however, the EEG only remains flat for several minutes, and activity in such cases should be seen to recover soon after. Occasionally, a seizure may have occurred by chance just before a recording was started, and the fact that observed voltage suppression is a postictal change is not obvious. In such cases, some electrical activity should return after several minutes. Rarely, high levels of sedative agents may also flatten the EEG.

The large majority of babies with an ECI pattern have a poor neurologic outcome, including death and severe disability. The longer the pattern persists, the more certain the poor prognosis.

Voltage depression in the form of low voltage, invariant patterns are also associated with a poor neurologic prognosis, but the outcome is somewhat more variable. In such records, voltages are persistently less than 20 μV and sleep cycling cannot be discerned (see Figure 13-21). When serial EEG studies document improvement in the pattern, the prognosis may also be improved.

Apart from suspecting widespread cortical damage as the explanation for low-voltage records, the electroencephalographer must consider other possible explanations. These include causes of diffuse scalp swelling, such as caput succedaneum or extensive subgaleal hematomas and extra-axial fluid collections, such as subdural hematomas or hygromas or pus collections.

Burst-Suppression Patterns

Burst-suppression patterns consist of high-voltage bursts across the brain, in some patients bilaterally synchronous and in others interhemispherically asynchronous. The bursts are separated by flat periods (see Figures 13-22, 13-23, and 13-24). Generally, burst-suppression patterns are persistent (i.e., they are not governed by a sleep-wake cycle), and they are not responsive to outside stimuli. The diagnosis of burst-suppression in the neonate is made considerably more difficult because the normal discontinuous patterns in very premature newborns, tracé discontinu and tracé alternant, bear some resemblance to burst-suppression. It is usually not difficult to distinguish burst-suppression from tracé

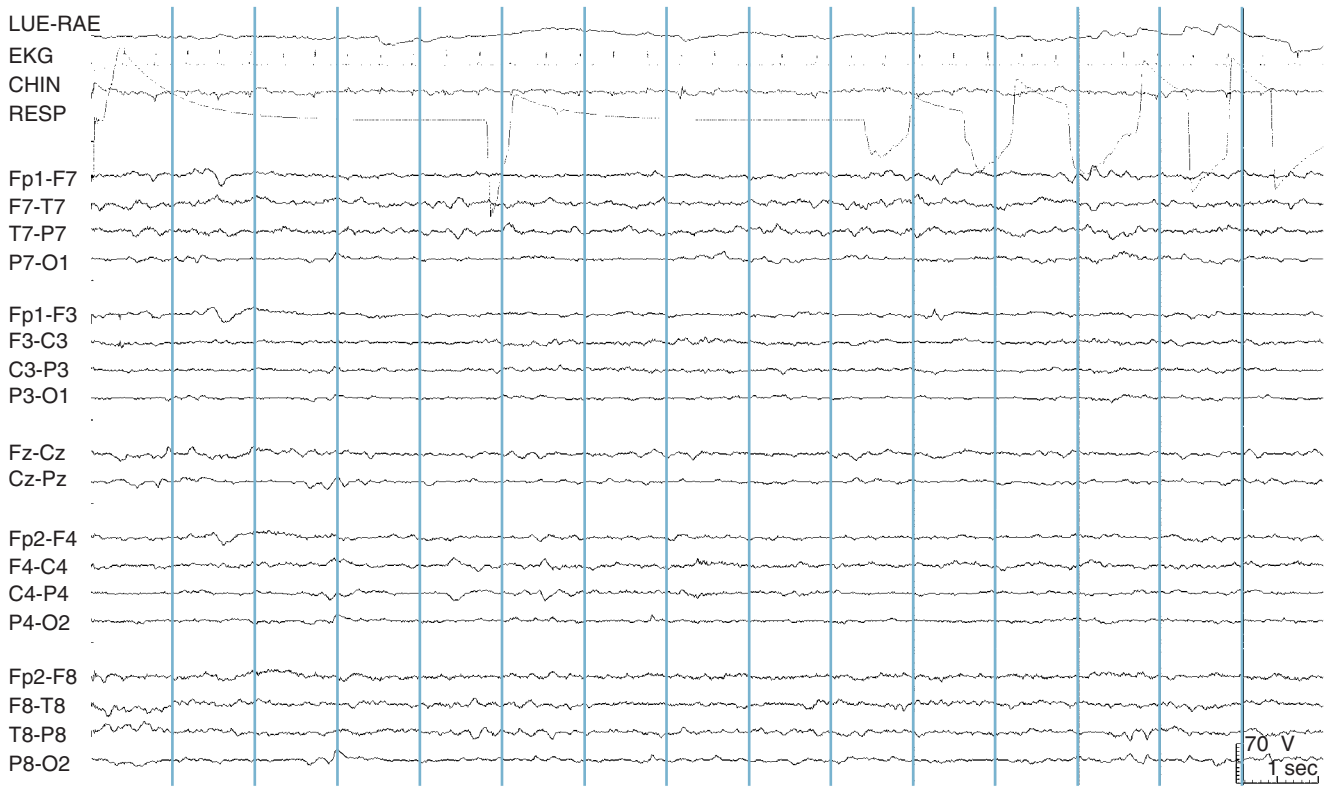

Figure 13-21 Although small amounts of electrocerebral activity are detected, nearly all activity in this tracing is below 20 μV, signifying voltage depression. Note the high amplifier gains used as suggested by the scale in the lower right-hand corner. When persistent and if not explained by other causes, voltage depression is often associated with a poor neurologic prognosis.

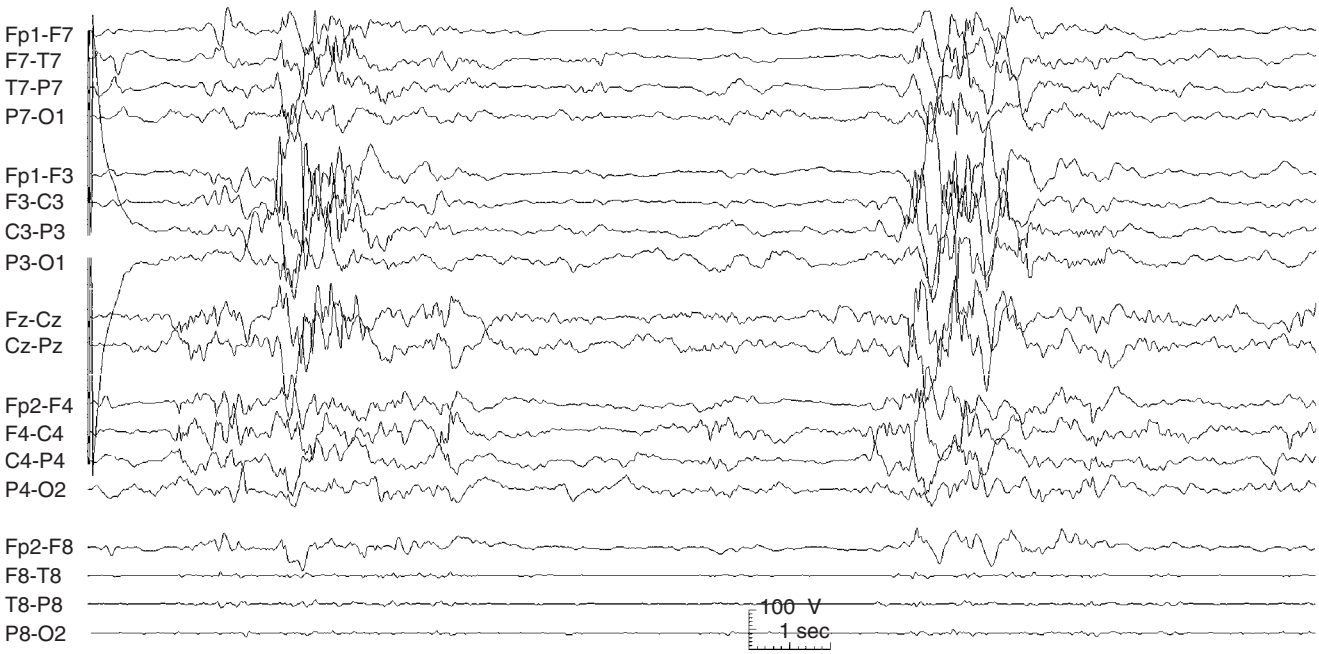

Figure 13-22 The burst-suppression pattern shown in this segment appears as "angry" polymorphic bursts of activity across both hemispheres separated by flatter periods. This pattern continued monotonously throughout the tracing. Note the lower voltages in the right temporal chain (bottom four channels) related to a subdural hematoma over the right temporal lobe.

alternant, a pattern that has more interburst activity and that may include normal forms such as delta brushes. Tracé alternant should not be invariant. In fact, any pattern that cycles on and off should not be labeled burst-suppression, which is typically a monotonously persistent pattern. Tracé discontinu can be more difficult to distinguish from burst-suppression in very preterm infants. The diagnosis of burst-suppression in this conceptional age group should only be made with hesitation and should be confirmed by serial recordings. So-called "permanently discontinuous" patterns probably represent a variant of burst-suppression.

With some exceptions, burst-suppression is associated with a dismal neurologic outcome. Babies who show improvement on a 1-week follow-up EEG tend to do considerably better than those who do not. Aside from being associated with hypoxic-ischemic or hemorrhagic injury, a burst-suppression pattern is also the hallmark finding in several epileptic syndromes of infancy, including early myoclonic encephalopathy, early infantile epileptic encephalopathy, and pyridoxine-dependent seizures (these disorders are discussed in more detail in Chapter 10 "The EEG in Epilepsy"). High therapeutic levels of barbiturates may also contribute to the appearance of a burst-suppression pattern in newborns, but this effect is probably not common.

Electrographic Seizure Activity

The presence of electrographic seizure discharges in the EEG background is generally considered an ominous prognostic sign; however, newborns who manifest seizure activity can be divided into different prognostic groups. The most distinctive of these groups is the newborn with repetitive seizures arising from the same location. Unilateral seizures are highly suggestive of a focal lesion, such as neonatal stroke or cerebral dysgenesis. The prognosis in these babies depends on the nature of the underlying lesion.

Among those children with seizures arising from both hemispheres, children who only manifest sporadic seizure activity, as a group, have a better prognosis than those who have unremitting electrographic seizure activity (electrographic status epilepticus). In severely asphyxiated babies, continuous electrographic seizure activity may even progress to an ECI pattern, although some babies may show considerable improvement. Most babies with continuous seizures on EEG do not survive or do poorly.

Amplitude Asymmetries

When a persistent amplitude asymmetry is seen, the extra-axial causes of voltage asymmetry listed earlier (such as fluid collections in the various extra-axial spaces) should be excluded by the clinician. Asymmetries of 25% or more are generally considered significant. It is good practice to confirm a potential voltage asymmetry with a referential montage. Large voltage asymmetries may be caused by strokes, hemorrhages, or cerebral dysgenesis. Marked asymmetries may be associated with a poor prognosis, which is usually a function of the underlying lesion.

Generalized slowing and focal slowing are uncommon findings in newborn EEGs. The impression of focal slowing over one hemisphere may actually represent a voltage asymmetry, and the possibility that it is

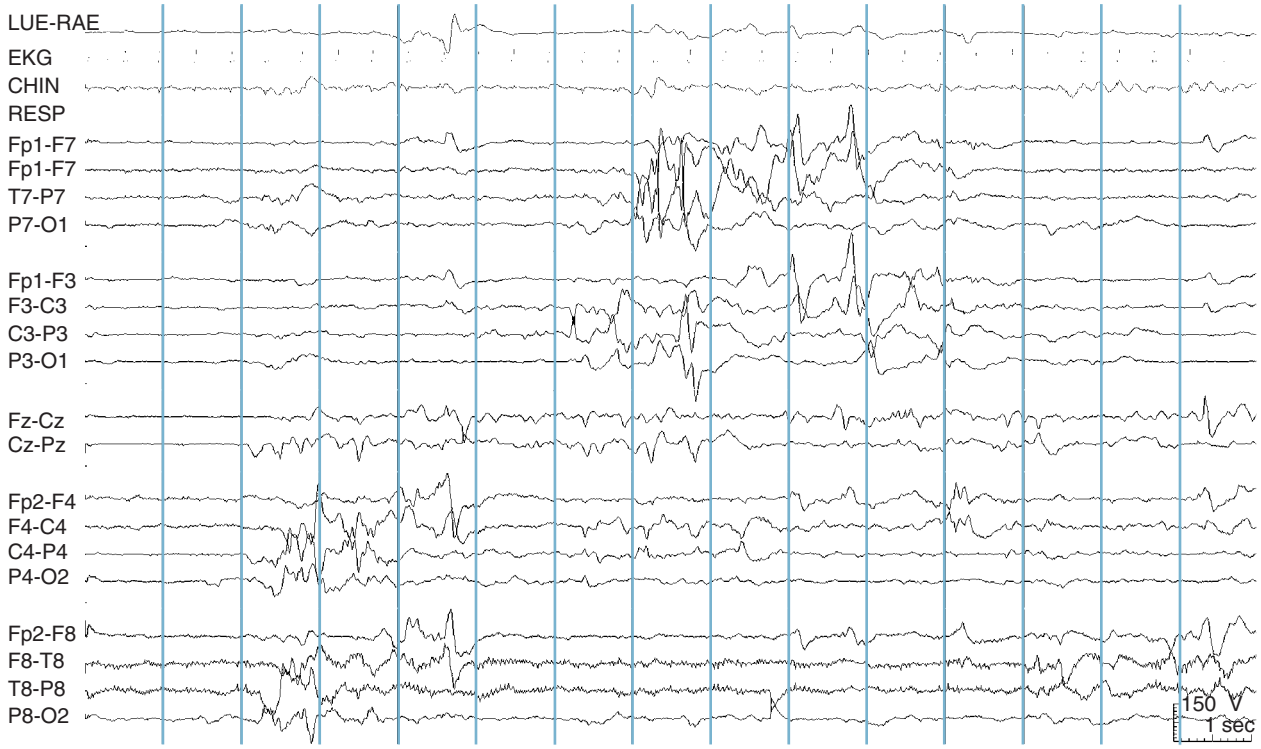

Figure 13-23 This unremitting burst-suppression pattern differs from the previous example in that the bursts occur asynchronously over each hemisphere. The lack of sleep cycling, either clinical or electrographic, during the rest of this recording confirms that this is a burst-suppression pattern rather than a normal sleep pattern.

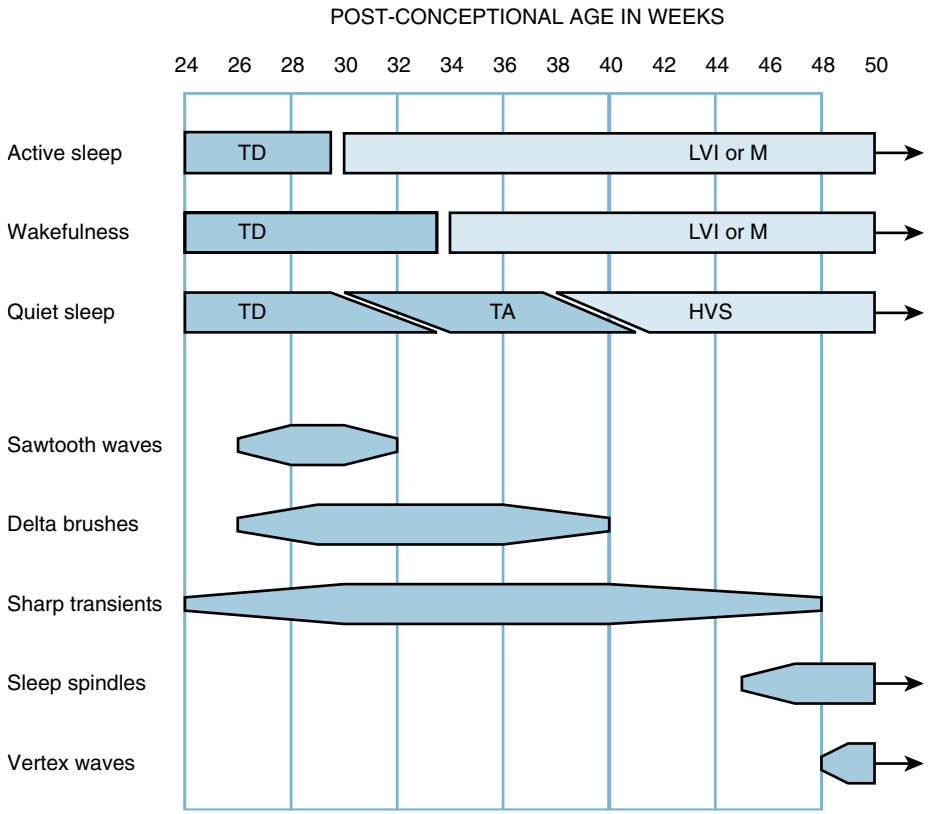

Figure 13-24 A summary of the background patterns and EEG graphoelements is shown according to CA. Note that the tracé discontinu pattern is seen in all sleep stages before 30 weeks CA, before which differentiation between wakefulness and sleep can only be made on the basis of the baby's clinical appearance and specific sleep stages are hard to differentiate. The continuous background patterns, LVI, M, and HVS, are highlighted in lighter blue to emphasize the stages during which continuous activity is seen. HVS, high voltage slow; LVI, low voltage irregular; M, mixed pattern; TA, tracé alternant; TD, tracé discontinu.

the opposite side that is abnormal because of low voltage should also be considered.

When amplitude asymmetries are transient, they may not be clinically significant. An unusual example of asymmetry is seen in prematures in whom one hemisphere may appear to "fall asleep" by attenuating before the other. This relatively uncommon phenomenon should only be seen once in a recording, in which case it is not considered abnormal.

Abnormal Sleep Architecture

It is difficult to appreciate sleep architecture abnormalities until 34 weeks CA. Sleep structure abnormalities range from a complete lack of sleep cycling to more subtle disruptions in expected sleep patterns. In many EEG records with abnormal sleep structure, the abnormal sleep architecture may be only one of many abnormalities present, and the coexisting abnormalities may have more prognostic significance. Abnormal sleep structure as a sole finding may have other possible explanations, such as sleep interruptions related to the intensive care unit environment, administered medications, and so forth. Furthermore, lack of normal sleep cycling as a sole abnormal finding will often improve by the time of a follow-up tracing. Therefore this is considered one of the milder background abnormalities, and many babies whose only EEG abnormality is abnormal sleep structure will eventually do well. Conversely, in a baby for whom there is worry of significant neurologic injury, the presence of normal sleep cycling can be considered a reassuring sign.

Immature Patterns and EEG Dysmaturity

The array of EEG sleep patterns and graphoelements described earlier allows the reader to estimate the CA of a baby to within approximately two weeks. Note is made of which sleep states are continuous or discontinuous, the presence of various EEG graphoelements, and the degree of interhemispheric synchrony, as described earlier. The duration of interburst intervals and the amount of activity present during interbursts may also provide clues as to CA. When the CA estimate based on the history and examination does not match the CA suggested by the EEG, it is worthwhile to reexamine the baby's clinical gestational age assessment. When the clinically derived CA seems secure but significantly exceeds the CA suggested by the EEG, this is referred to as EEG dysmaturity or simply as an *immature pattern.*

Immature patterns represent a nonspecific abnormality and are considered to be among the mildest background abnormalities. The EEG should be more than 2 weeks "dysmature" before clinical significance should be presumed. Examples of EEG dysmaturity could be a tracing of a 38-week CA baby that still shows copious delta brush activity (as might be seen at 34 weeks) or excessively asynchronous tracé alternant patterns near term (a stage by which the pattern should have become nearly completely synchronous). Babies whose only EEG abnormality is an immature pattern are presumed to have sustained a mild and possibly reversible neurologic insult, and a large number of such babies are neurologically normal at follow-up. A summary of sleep stage patterns and EEG graphoelements is shown in Figure 13-25.

ELECTROGRAPHIC SEIZURE DISCHARGES

Because many newborn EEGs are obtained with the goal of excluding or confirming the presence of seizures, the EEG reader is frequently called on to assess the EEG tracing for seizure activity. In contrast to the EEGs of older children and adults, the background of the normal newborn EEG contains little rhythmic activity. Familiar rhythmic forms such as the posterior rhythm and sleep spindles seen in the EEGs of older individuals have not yet appeared in the newborn EEG. The main continuous EEG background patterns of newborns, LVI, M, and HVS, consist of irregular activity; the discontinuous patterns of tracé discontinu and tracé alternant have even less of a tendency toward rhythmicity. Except in rare examples, as discussed subsequently, electrographic seizure activity in newborns consists of *rhythmic* activity. This fact makes identification of neonatal seizures considerably easier. Any rhythmic activity of sufficient duration in the newborn EEG is suspect. Although the discharge duration required by others to declare a rhythmic discharge an electrographic seizure has varied, an 8-second lower limit for ictal rhythmic discharges in neonates is reasonable.

Typically, neonatal seizure discharges consist of rhythmic waves in a focal distribution, sometimes with sharp features and sometimes not. The morphology of the discharge can range from repetitive sharp waves, spikes, or even polyspikes, to a rhythmic sinusoidal pattern (see Figure 13-25). In some cases the discharge can be so focal as to be confined to a single electrode (see Figure 13-26) but even the most focal discharge will usually affect other electrode contacts throughout its course. During its evolution, a neonatal seizure discharge may migrate from one location to another in the involved hemisphere and may also appear to "spread" to the opposite hemisphere as in Figure 13-27, although an independent, simultaneous onset in the opposite hemisphere may also explain this appearance. Neonatal seizure discharges generally fire at a rate of 1 to 3 Hz and occasionally faster, although it is more common for seizure discharges to fire at the lower end of this range. Especially when the discharge is accompanied by clonic activity, the rate of the jerking is usually nearer one per second than the top end of this range.

It is said that newborns cannot have truly generalized seizures, and this is probably the case. For instance, generalized spike-wave discharges are not seen in the newborn. Presumably the cortical circuitry of very young

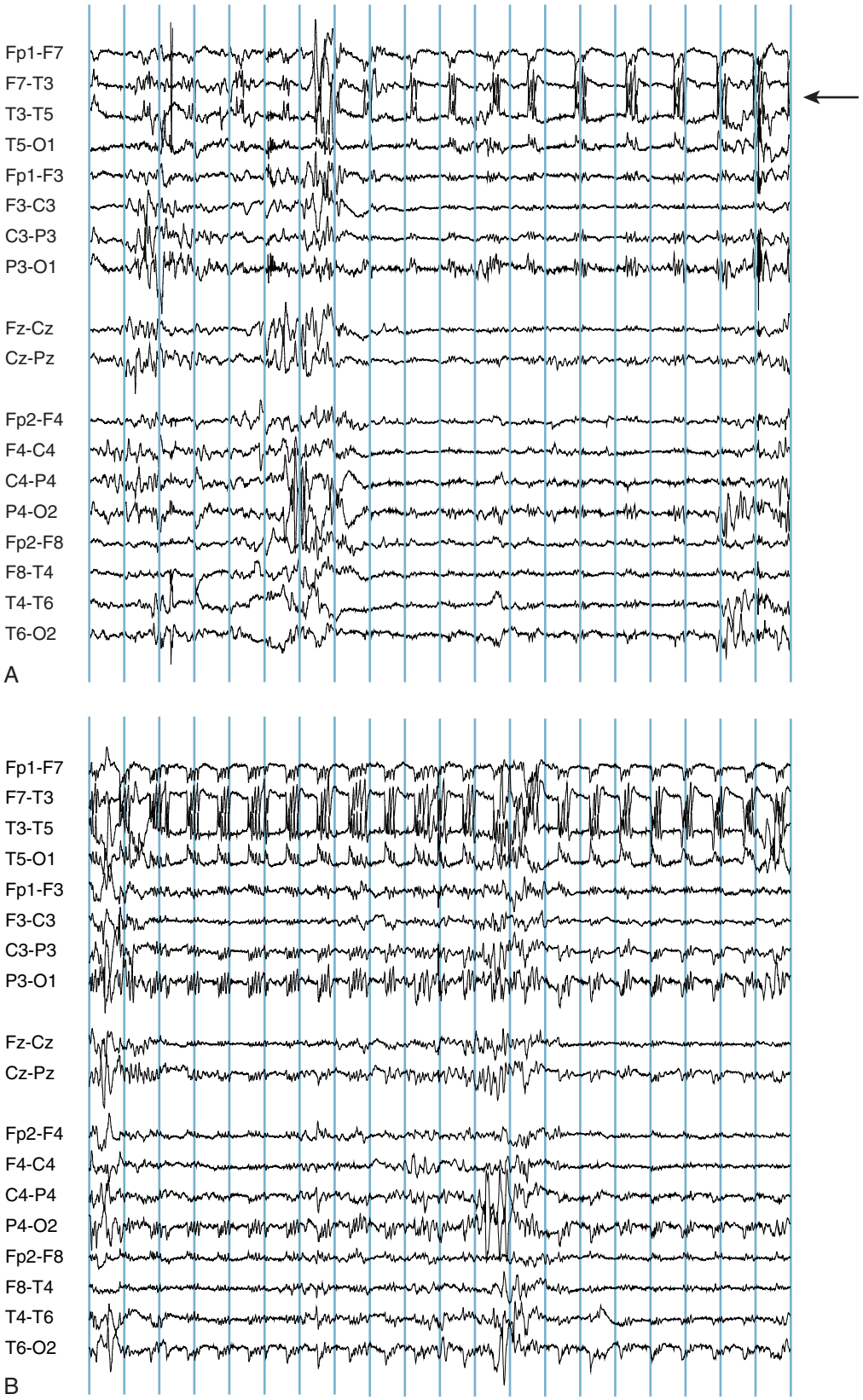

Figure 13-25 An electrographic seizure discharge is seen in the left temporal area, maximum in the left midtemporal electrode (T3). In this example, the discharge consists of a repetitive polyspike rather than a rhythmic sharp wave or slow wave. The discharge begins at the beginning of the first page **(A)** and increases in amplitude and complexity throughout its course **(B).**

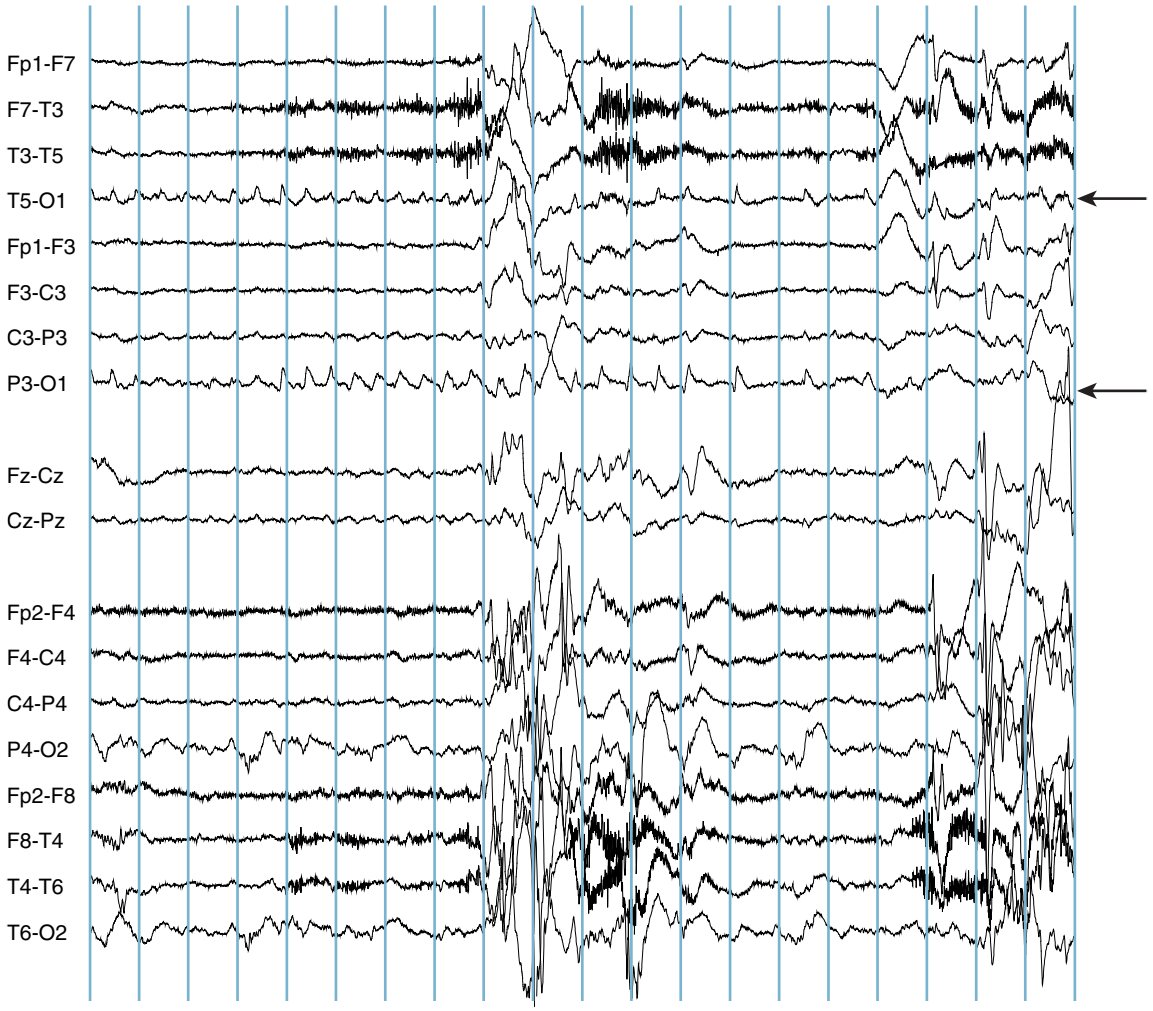

Figure 13-26 This seizure discharge begins as a run of rhythmic sharp waves recorded exclusively from the left occipital (O1) electrode. In some cases, a seizure discharge may start and stop remaining localized to a small area. During the last 3 seconds of the page, however, the discharge can be seen to have rapidly spread to involve a broader area over the left hemisphere. The evolution of this discharge is shown in the next figure.

babies is not organized in a fashion capable of mounting generalized discharges. Mimics of generalized discharges can occur, however. In some cases, a baby may have a seizure discharge ongoing in both hemispheres at once. In many such cases, the discharges are not truly synchronous and the discharges are actually bilateral but independent (see Figure 13-28). Clinically, a seizure is occasionally seen with bilateral extremity jerking, suggesting a generalized discharge. In some such cases, the EEG may show a unilateral discharge in which the outflow causes bilateral synchronous limb movement, although the movements may be of different intensity on each side. In other cases, close inspection reveals that the jerking movements are not synchronous but bilaterally independent.

Neonatal seizures that consist of nonrhythmic discharges occur more rarely. For instance, high-voltage single generalized polyspikes may result in whole body myoclonic jerks. Tonic seizures and clonic seizures in newborns are usually associated with rhythmic seizure discharges.

CLASSIFICATION OF NEONATAL SEIZURES

The neonatal seizure classification system in most common use today was described by Volpe (see Table 13-2). This 1989 classification includes subtle seizures, clonic seizures (focal and multifocal), tonic seizures (focal and generalized), and myoclonic seizures (focal, multifocal, and generalized).

Types of Clinical Seizure Activity and Their Relationship to EEG Seizure Discharges

The clinical diagnosis of seizure activity in newborns is not straightforward, especially for the "subtle" category of seizure manifestations because not all apparent clinical seizure behaviors described in the classification are consistently accompanied by an electrographic seizure discharge. The odds that an apparent clinical seizure event is associated with an EEG seizure discharge depends on the type of behavior observed.

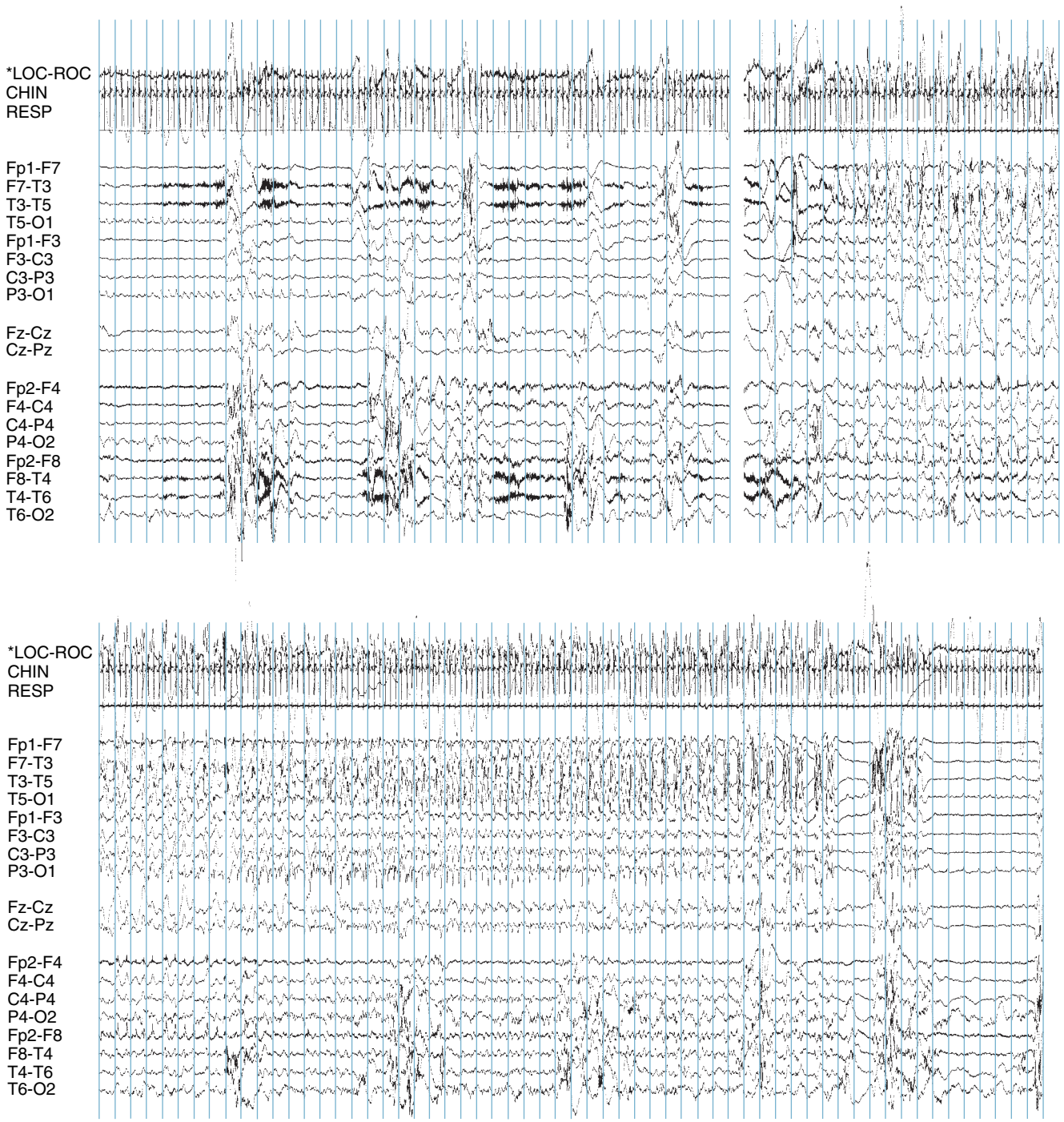

Figure 13-27 The seizure for which the onset is shown in the previous figure is displayed in compressed form to demonstrate its propagation. Each vertical division represents 1 second. The vertical gray bar denotes an area in which several seconds of the tracing have been removed to aid in the display. The seizure begins in the left occipital area and spreads more widely in the left hemisphere. Soon after the vertical gray bar, an independent discharge begins in the right hemisphere.

Not surprisingly, simultaneous video/EEG recordings of babies at risk for seizures have shown that clonic activity is highly correlated to electrographic seizure activity. In contrast, whole body tonic stiffening, a dramatic clinical behavior that resembles the tonic phase of tonic-clonic seizures in older subjects, does not have a strong association with EEG seizure discharges in newborns. Whole body stiffening may actually represent nonepileptic posturing in the setting of CNS dysfunction or perhaps a brainstem release phenomenon in a baby in whom cerebral cortex is not able to carry out its usual role in suppressing such behaviors. Focal limb tonic stiffening, however, is highly correlated with EEG seizure activity. Focal and multifocal myoclonic jerks are usually nonepileptic, but generalized myoclonic jerks have a higher rate of association with seizure discharges on the EEG (see Mizrahi and Kellaway).

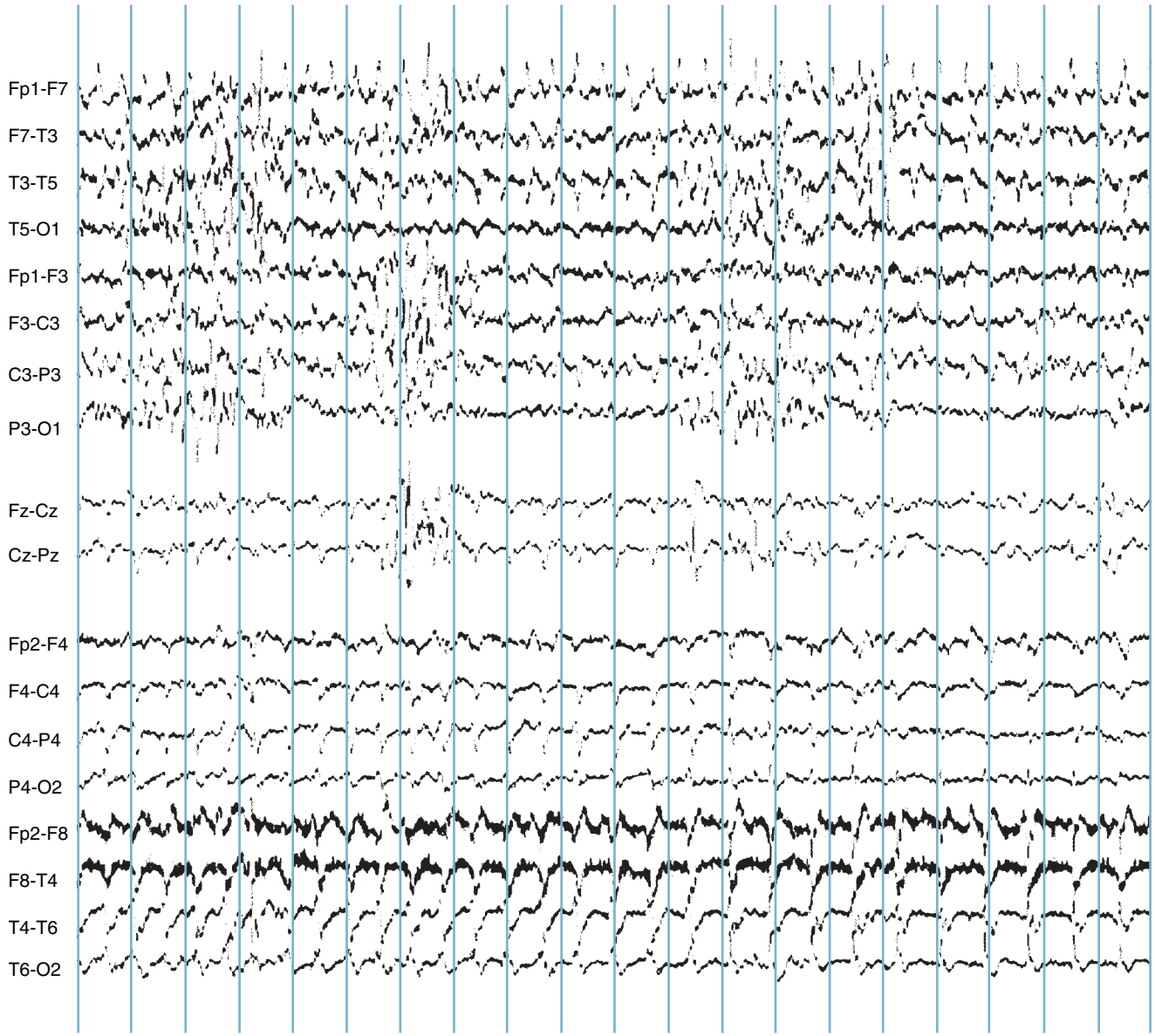

Figure 13-28 An electrographic seizure is seen to engulf both hemispheres in this term newborn. Close examination shows that the discharges in each hemisphere are bilateral but independent rather than synchronous.

Table 13-2 Neonatal Seizure Classification

Clinical Seizure	Association With Electrographic Seizures
Subtle	Variable
Clonic	
Focal	Common
Multifocal	Common
Tonic	
Focal	Common
Generalized	Uncommon
Myoclonic	
Focal, Multifocal	Uncommon
Generalized	Common

Adapted from Volpe, 1989.

The most important difference between the 1989 neonatal seizure classification and the International League Against Epilepsy (ILAE) seizure classification used for older children and adults is the addition of the somewhat controversial category of "subtle seizures." Subtle seizures described in neonates include movements such as "swimming," "boxing," or "hooking" movements of the upper extremities; "bicycling" movements of the lower extremities; orobuccolingual movements such as sucking or lipsmacking, eye opening, or complex eye movements; and pure apneas. Controversy has arisen regarding this family of behaviors as they are frequently not associated with a concurrent EEG seizure discharge. Therefore, the epileptic nature of these "subtle seizure" behaviors has not been firmly established, and many may represent the

unmasking of automatic reflex behaviors at times when the cerebral cortex is unable to suppress these reflex sequences. This question is of considerable importance because if these behaviors are nonepileptic, they would not require specific treatment with antiseizure medications. The clinical picture of newborns with subtle seizures becomes even more complicated considering that, even if nonepileptic, these behaviors may be the result of cortical injury and may coexist with true EEG seizure discharges occurring at other times in the same baby.

Among these subtle behaviors, apneas are perhaps the most difficult behavior to diagnose correctly. The great majority of apneas in newborns are not related to seizure activity. Occasionally, however, an epileptic seizure may manifest as a pure apnea in a newborn unaccompanied by clear motor or other behavioral change. Whether apneas in the newborn require EEG investigation depends on the clinical context.

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