9. The Normal EEG of the Waking Adult

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The presentation of the normal mature human electroencephalogram (EEG) and its basic features has a dual purpose. These are to promote knowledge of the boundaries between normality and abnormality in clinical EEG work and to enhance the understanding of the EEG as a phenomenon with important psychophysiological implications.

Both purposes are evident in Hans Berger's pioneering work, from the first report with its strong emphasis on psychophysiology and the discovery of the alpha rhythm (Berger, 1929) to his 14th report (Berger, 1938) of predominantly clinical interest. The question of how the EEG works remains to be answered by the experimental neurophysiologist. The nature of the normal EEG, how it reflects human behavior and mental functions, and the boundaries of normality are most legitimate guidelines for a chapter on the normal adult EEG.

EEG Frequencies

The EEG, as the continuous "roar" or "noise" of the brain, contains a fairly wide frequency spectrum, but it is not simply a hodgepodge of frequencies. Rhythmicity seems to create some law and order among waves of various lengths and amplitudes. The impression of prevailing rhythmicity and organization, however, is not a yardstick for the normality of an EEG. Pronounced rhythmicity may be a sign of abnormality, and a prima vista anarchic appearance does not necessarily imply abnormality. Reactivity may be the magic word in such cases; an EEG of mixed frequencies may be quite responsive to certain stimuli.

The *frequency range* of the EEG has a fuzzy lower and upper limit. There are ultraslow and ultrafast frequency components that play no significant role in the clinical EEG, with the exception of ultraslow activity in profound coma and near-terminal states. For these reasons, the frequency-response curve of an EEG apparatus concentrates on the clinically relevant frequency range, which is also the most important from the psychophysiological viewpoint. This range lies between 0.1/sec [or cycles per second (cps) or Hz] and 100/sec and, in a more restricted sense, between 0.3/sec and 70/sec. In the normal adult, the slow ranges (0.3–7/sec) and the very fast range (above 30/sec) are sparsely represented; medium (8–13/sec) and fast (14–30/sec) ranges predominate.

These frequencies are broken down into the following bands or ranges:

Delta below 3.5/sec (usually 0.1–3.5/sec)
Theta 4–7.5/sec
Alpha 8–13/sec
Beta 14–30/sec
Gamma above 30/sec (unlimited in upper range)

This is the "old-fashioned" breakdown of the EEG frequencies. The reader will find here and in several other chapters of this book discussions on ultraslow activity [below 0.1/sec to near 0 direct current (DC) potentials] and ultrafast activity (Curio, 2000a; also see Chapter 26). The exact width of the gamma range is still debated and may lie between 30 and 60/sec. There has been a proposal by Curio (2000b) for the following designations: omega range 60 to 120 Hz, rho range 120 to 500 Hz, and sigma range 500 to 1,000 Hz. This will need further clarifications. The reader will find below that the letters rho and sigma have been used for EEG phenomena occurring in sleep. Let us wait and see which of these terms will find general and lasting acceptance!

The sequence of these Greek letters is not logical and can be understood only in the historical view. The terms *alpha* and *beta* rhythm or waves were introduced by Berger (1929); the term *gamma* rhythm was subsequently used by Jasper and Andrews (1938) to designate frequencies above 30 or 35/sec; these were essentially 35 to 45/sec and superimposed on the occipital alpha rhythm (see Dutertre, 1977). This term was temporarily abandoned, and *gamma* frequencies became a part of the beta range.

The use of the term *gamma rhythm* or *gamma frequency range* has made an impressive comeback during the 1990s. The use of a *fast* beta range and a *very fast* gamma range might have been convenient for those who utilize frequency analysis with power spectra. Furthermore, modern EEG rhythm research in the 1990s has unearthed the all-but-forgotten term *gamma rhythm* (Basar, 1992; Bullock, 1992; Eckhorn et al., 1992; Gray et al., 1992). In this "new wave" of EEG rhythm research, rhythmic activities of the brain are conceived mainly as induced rather than as spontaneous rhythms.

The term *delta rhythm* was introduced by Walter (1936) to designate all frequencies below the alpha range. Walter himself, however, found a need to introduce a special designation for the 4 to 7.5/sec range and used the letter *theta*. He thus bypassed the Greek letters *epsilon, zeta,* and *eta*; he chose *theta* to stand for thalamus because he presumed a thalamic origin of these waves (also see Knott, 1976b).

The term *pi rhythm* has been used for the designation of posterior slow rhythms (3–4/sec) without harmonious relationship to the posterior alpha rhythm according to Dutertre (1977) who recommended the preferable (although certainly less precise) term *posterior slow rhythms*. Hardly anyone has used the term *pi rhythm* since the 1990s.

The term *phi rhythm* was suggested by D. Daly (according to Silbert et al., 1995) for the designation of monorhythmic posterior delta waves (less than 4/sec), distinct from the background and occurring within 2 sec of eye closure. This rhythm was also described by Belsh et al. (1983) as "posterior rhythmic slow activity after eye closure."

The term *occipital intermittent rhythmical delta activity (OIRDA)* (see Chapter 12) refers to a pattern of childhood that is usually found in epileptic patients (Gullapalli and Fountain, 2003). One may still wonder if the term *OIRDA* is really needed. On the other hand, FIRDA and TIRDA, for frontal and temporal rhythmical delta, are powerful and clinically informative patterns (see Chapters 12 and 27).

The alpha-like anterior temporal kappa rhythm (Laugier and Liberson, 1937) is a controversial pattern that is discussed later in this chapter. Kugler (1981) has been using the term *sigma activity* instead of *sleep spindles* and, furthermore, the term *sigma rhythm* for activity in the 11 to 15/sec range. The term *rho waves* was used for the activity known as POSTS (positive occipital sharp transients of sleep) (Kugler and Laub, 1971), but it has disappeared.

Other Greek letters have been proposed for the designation of distinct EEG activities. Mu rhythm and lambda waves are discussed in this chapter. The term *tau rhythm* is mentioned in Chapter 57, "Magnetoencephalography as a Tool of Clinical Neurophysiology," and it denotes a physiological alpha rhythm of the temporal region (in the author's opinion identical with the "third rhythm" discussed in this chapter). "Zeta wave" simply denotes a certain type of delta wave (rather than a rhythm) with some sharp configuration (Magnus and Van der Holst, 1987; Siepman et al., 2004).

Thus, 13 of the 23 letters of the Greek alphabet are being used in the EEG terminology, and this number could be even higher. In my opinion, it might be better to limit the Greek terms to the classical EEG frequency ranges retaining solely the letters *alpha, beta, gamma, delta,* and *theta*.

EEG Amplitudes

The EEG denotes voltage plotted against time. The voltage of the EEG signal determines its amplitude. The passage of the cortical EEG signal through leptomeninges, cerebrospinal fluid, dura mater, bone, galea, and scalp has a strongly attenuating effect on the original signal (Cooper et al., 1965); this is discussed in the section on the depth EEG. Corticographic discharges show amplitudes of 500–1.500 μ (0.5–1.5 mV) and several millivolts in prominent spiking. The amplitudes of the scalp EEG are markedly reduced and lie between 10 and 100 μV (in adults, more commonly between 10 and 50 μV).

The EEG amplitudes are measured from peak to peak. Precise determination of the voltage of each wave is unnecessary and should be discouraged as pseudoaccuracy; too many variables are involved (above all, the interelectrode distance and the type of montage, whether bipolar or referential recording). Electroencephalographers may indicate in their reports a certain amplitude range, such as "alpha rhythm from 20–30 μV," or, even better, limit themselves to statements such as "of medium voltage" or "of low to medium voltage."

A given frequency can be rendered abnormal by excessive voltage. This is true for all frequencies, and it is particularly important for the fast (beta) band. The problem of low voltage will be thoroughly discussed, because low amplitudes can indicate a life-threatening decline of cerebral voltage output, whereas the vast majority of low-voltage records are "desynchronized" (discussed later) and a variant of normalcy.

Alpha Rhythm

Definition

The International Federation of Societies for Electroencephalography and Clinical Neurophysiology (IFSECN) (committee chaired by G. E. Chatrian; see IFSECN, 1974) proposed the following definition of alpha rhythm:

> Rhythm at 8–13 Hz occurring during wakefulness over the posterior regions of the head, generally with higher voltage over the occipital areas. Amplitude is variable but is mostly below 50 μV in adults. Best seen with eyes closed and under conditions of physical relaxation and relative mental inactivity. Blocked or attenuated by attention, especially visual, and mental effort (IFSECN, 1974).

This committee also has pointed out that the term *alpha rhythm* must be restricted to rhythms fulfilling all of the above criteria. Rolandic mu rhythm may have the same frequency range, but its topography and reactivity are different (also see Markand, 1990).

Frequency

The chapter on EEG maturation shows the gradual frequency increase of a posterior basic rhythm that is detectable around the age of 4 months with a frequency of approximately 4/sec. This posterior basic rhythm shows a progressive frequency increase with average values of around 6/sec at age 12 months and 8/sec at age 3 years. At that time, the alpha frequency band is reached, and there is justification for the use of the term *alpha rhythm*. The frequency reaches a mean of about 10/sec at age 10 years. This is essentially the mean alpha frequency of adulthood; in other words, the progressive alpha rhythm acceleration usually ends around the age of 10 years, but the second decade of life (and to some degree also the third decade) features a constant decline of intermixed posterior slow activity that is usually present in considerable quantity at age 10.

The frequency of the alpha rhythm tends to decline in elderly individuals. This decline apparently reflects some degree of cerebral pathology, which is vascular or fibrillary degenerative, in most instances. Healthy and vigorous elderly people may show little or no alpha frequency decline, even in the ninth decade. An alpha rhythm with a consistent 8/sec frequency ought to be regarded as a mild abnormality.

The figure of 10.2 ± 0.9/sec has been indicated as the mean adult alpha frequency (Petersén and Eeg-Olofsson, 1971). An element of instability of the alpha frequency must be taken into consideration; according to Townsend et al. (1975), the alpha rhythm frequency can be stabilized by sinusoidally modulated light. Extreme upward gaze tends to facilitate the posterior alpha rhythm (Mulholland, 1969; Mulholland and Evans, 1965). Lateral eye deviations may have similar effects (Fenwick and Walker, 1969).

An alpha rhythm frequency shift to the faster portion of the band is not uncommon and is essentially within normal limits, as will be discussed later. The similarities between the frequencies of alpha rhythm and the physiological finger tremor have been discussed by Isokawa and Komisaruk (1983). Immediately after eye closure, the alpha frequency may be accelerated for a moment ("squeak effect," after Storm van Leeuwen and Bekkering, 1958).

Amplitude

Alpha rhythm amplitudes vary considerably from individual to individual and, in a given person, from moment to moment. The electroencephalographer, therefore, should look for stretches of optimal alpha output. A referential montage to the ipsilateral ear is usually most suitable for the determination of the alpha rhythm amplitude, but the interelectrode distances must always be considered. The maximum alpha voltage is usually over the occipital region as such, but a bipolar montage with a parasagittal array may obscure rather than reveal the true alpha maximum. The alpha amplitude may be quite small in the channels displaying P_3–O_1 and P_4–O_2 because of massive homophasic activity, which results in canceling out.

The alpha amplitudes tend to show constant waxing and waning. For this reason, trains of alpha waves show a typical spindle shape with a belly and a thin portion. However, the term *spindles* has been reserved for a classical pattern of sleep (see Chapter 10, "Sleep and EEG") and should not be used in this context.

Berger (1929) found alpha rhythm voltages of 15–20 μV; these are small values when one considers his fronto-occipital recording technique; their smallness was probably due to the instrument limitations of his Edelmann string galvanometer. According to Cobb (1963), the alpha rhythm voltage fluctuates between 0 and 40–50 μV in the individual record; values above 100 μV are uncommon in the adult, whereas maximums of 5–10 μV are frequently seen (Cobb, 1963). The work of Simon (also known as Simonova) and her co-workers has shed more light on this subject; Simonova et al. (1967) found amplitudes between 20 and 60 μV in 66% of their subjects; values below 20 μV were found in 28% and above 60 μV in 6% (also see Simon, 1977).

Higher alpha amplitudes are more likely to be found in association with slower alpha frequencies (Brazier and Finesinger, 1944; Wieneke et al., 1980). There is good evidence of a mild to moderate alpha amplitude asymmetry with higher voltage on the right (Cobb, 1963; Kellaway and Maulsby, 1966; Kiloh et al., 1972; Petersén and Eeg-Olofsson, 1971; Simon, 1977; Wieneke et al., 1980). This seems to indicate that the alpha rhythm is of greater amplitude over the nondominant hemisphere, but no convincing conclusion concerning handedness can be derived from this asymmetry (Petersén and Eeg-Olofsson, 1971). This physiological asymmetry has been confirmed by Matousek et al. (1981), who also found a reversal of this rule (i.e., higher voltage on the left occiput in patients with endogenous depression).

Amplitude asymmetries must be demonstrated in both referential and bipolar montages from two or more posteriorly placed electrodes (such as parietal and posterior temporal) before it is considered significant (Markand, 1990). There may be a fine line between physiological and truly abnormal alpha amplitude asymmetries.

Wave Morphology

The alpha rhythm is usually characterized by rounded or sinusoidal wave forms. However, a sizable minority of individuals have sharp alpha configuration. In such cases, the negative component appears to be sharp and the positive component appears to be rounded, similar to the wave morphology of rolandic mu rhythm.

The sharp configuration of posterior alpha waves is by no means an abnormality. It is a common finding, especially in young adults, adolescents, and older children. An admixture of beta waves is usually the cause of the sharp configuration; drug effects from sedatives or minor tranquilizers must sometimes be suspected in such cases. Unusual morphologies in childhood ("fused forms") are presented in Chapter 11, "Maturation of the EEG: Development of Waking and Sleep Patterns."

Spatial Distribution

The alpha rhythm is clearly a manifestation of the posterior half of the head and is usually found over occipital, parietal, and posterior temporal regions. This observation of Adrian and Matthews (1934) was doubted by Berger (1935), whose concept of alpha rhythm as a global cerebral rhythm was an erroneous conclusion from his fronto-occipital bipolar recording technique. The alpha rhythm may extend into central areas, the vertex, and also the midtemporal region. When the central region is strongly involved, the alpha rhythm must be distinguished from possibly coexisting rolandic mu rhythm. This is usually easily demonstrable with eye opening, blocking alpha rhythm but not blocking mu rhythm.

The alpha rhythm may occasionally extend slightly into the superior frontal leads (F_3, F_4). Extension into the frontopolar region (Fp_1, Fp_2) is practically unheard of. Apparent alpha rhythm in the frontopolar leads may be very prominent in referential (unipolar) montages if the referential ear electrode picks up the posterior alpha rhythm. This is particularly common when the mastoid region is used instead of the ear lobe (the mastoid being a preferred place with paste technique). Another source of confusion is eyelid flutter, with closed eyes giving rise to frontal artifacts in alpha frequency.

In depth electroencephalography and with occipital implants, posterior alpha rhythm can be demonstrated throughout the depth of the occipital lobe and even in the vicinity of the lateral geniculate body. According to Albe-Fessard (1975), alpha rhythm may be recorded from the medial pulvinar but not from the remaining thalamic nuclei in the human (also see Gücer et al., 1978). In the dog, Lopes da Silva et al. (1973a) demonstrated that alpha rhythm of the same peak frequency, bandwidth, and reactivity can be recorded from the visual cortex as well as from the visual thalamus (lateral geniculate body, pulvinar nuclei).

Further inferences on the alpha rhythm distribution can be made from the study of alpha rhythm generation. This topic is discussed later (also see Theories of Neurophysiological Basis of Mu Rhythm, below).

Reactivity

The posterior alpha rhythm is temporarily blocked by an influx of light (eye opening), other afferent stimuli, and mental activities. The degree of reactivity varies; the alpha rhythm may be completely blocked, suppressed, or attenuated with voltage reduction. The alpha blocking response to eye opening was discovered by Berger and described in his first report (1929) on the human EEG; it came as a great surprise for investigators who were searching for action potentials and hence would have expected enhancement of EEG voltage with influx of light (Fig. 9.1). Berger's own explanation was the concept of a zone of inhibition surrounding the area of excitation by the afferent stimulus (Berger, 1933a; also see Gloor, 1971).

It is noteworthy that in some subjects the alpha suppression is more pronounced than in others. These persons may have an alpha-free stretch of desynchronized and chiefly fast EEG activity for the duration of the eye opening, even in rather dim light. In others, the alpha blockage lasts for less than 1 second. According to Gibbs and Gibbs (1950) "after the eyes have been held open for a few minutes, low voltage alpha waves . . . usually reappear, unless the subject continues to look at something which holds his interest."

The amplitude ratio between eyes closed (well-developed alpha) and eyes open (beta of much smaller voltage) declines with advancing age (Könönen and Partanen, 1993).

Alpha attenuation due to auditory, tactile, and other somatosensory stimuli or heightened mental activity (such as solving difficult arithmetical problems) is usually less pronounced than the blocking effect with eye opening.

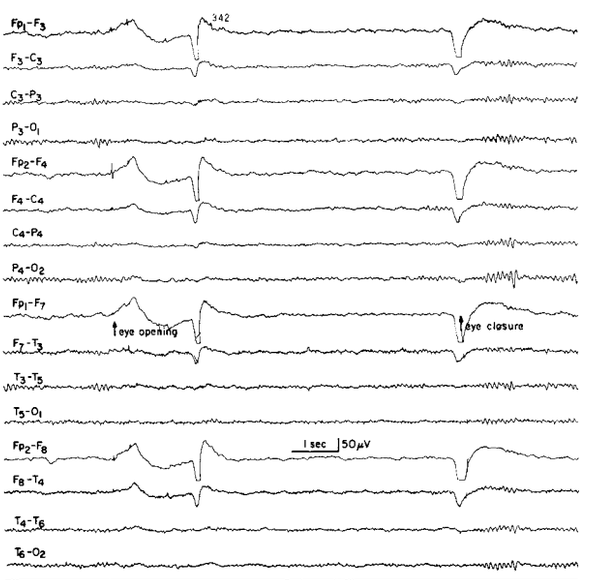

Figure 9.1. Normal record, in a patient age 27 years. Posterior 10–12/sec alpha rhythm. Good blocking response to eye opening and reactivation of alpha rhythm with eye closure. Note low-voltage fast activity and some return of alpha rhythm while eyes are kept open. Also note typical artifacts with eye opening, eye closure, and eye blink (caused by eye potential shifts).

According to Niedermeyer et al. (1989), the blocking or attenuating effect of mental arithmetic on the posterior alpha rhythm is absent in most cases (Niedermeyer et al., 1989). In a study done in 1,280 patients (598 with normal EEG tracings, 682 with various degrees of EEG abnormality), alpha suppression or attenuation with calculation (serial sevens) was noted in only 21 (1.6%) of the patients. On the other hand, Berger (1931, 1932, 1933a,b, 1937) had shown fine examples of arithmetic-induced alpha blocking. We found, however, that even "serial seventeens" (100 minus 17, etc.) are mostly unassociated with any decrease of the posterior alpha rhythm. Does this mean that the venerable discoverer of the human EEG was all wrong as far as arithmetic-induced alpha blocking is concerned? Not at all. The arithmetical tasks given to his two teenage children, Klaus and Ilse, were just a lot more difficult: for Klaus (age 19) 23×43, and for Ilse (age 14) $196 \div 7$. Difficulty of the task is not the only factor. Even simple arithmetic may cause alpha blocking if the subject shows great motivation trying to please the examiner. With a nonchalant approach, there is usually no alpha blocking.

No EEG is complete without certain reactivity tests. Although alpha rhythm disappears with the earliest approach of drowsiness, there are exceptional cases of persisting posterior alpha rhythm in profoundly comatose patients with pontine vascular lesions; their alpha rhythm shows no reactivity, even with strong nociceptive stimuli. According to Kiloh et al. (1972), "complete unresponsiveness of the alpha rhythm to visual stimuli is a rare and unequivocally abnormal finding."

Pfurtscheller (1990) conceives the stimulus-induced alpha blocking or attenuation as an "event-related EEG desynchronization." With the use of averaging techniques, he compared such desynchronizing effects with stimulus-induced evoked potentials and found different topographical patterns and time courses, presumably due to different underlying neuronal processes. This is just a small vignette of the "new wave" of alpha (and other EEG rhythms) research, which will be discussed in the further course of this chapter.

In albinism, misrouting of the optic nerve fibers with decussation anomaly is a frequent finding. Smith et al. (1998) have demonstrated that unilateral alpha activity to eye opening and closure can be found. In other words, there is right-sided alpha rhythm with left eye closed, left-sided alpha rhythm with right eye closed, and alpha blocking with closure of both eyes.

Alpha Rhythm and Vigilance

Alpha rhythm is the classical EEG correlate for a state of relaxed wakefulness best obtained with the eyes closed. A degree of higher alertness attenuates or suppresses the alpha rhythm, which is then supplanted by "desynchronized" low voltage fast activity.

The earliest stage of drowsiness is characterized by "alpha dropout" (Fig. 9.2). The trains of alpha waves become less and less continuous, and the last alpha fragments finally give way to a low-voltage pattern of mixed slow (mostly theta range) and fast frequencies. This type of alpha dropout is a hallmark of a normal adult EEG; in children and infants, various types of slow patterns appear (see Chapter

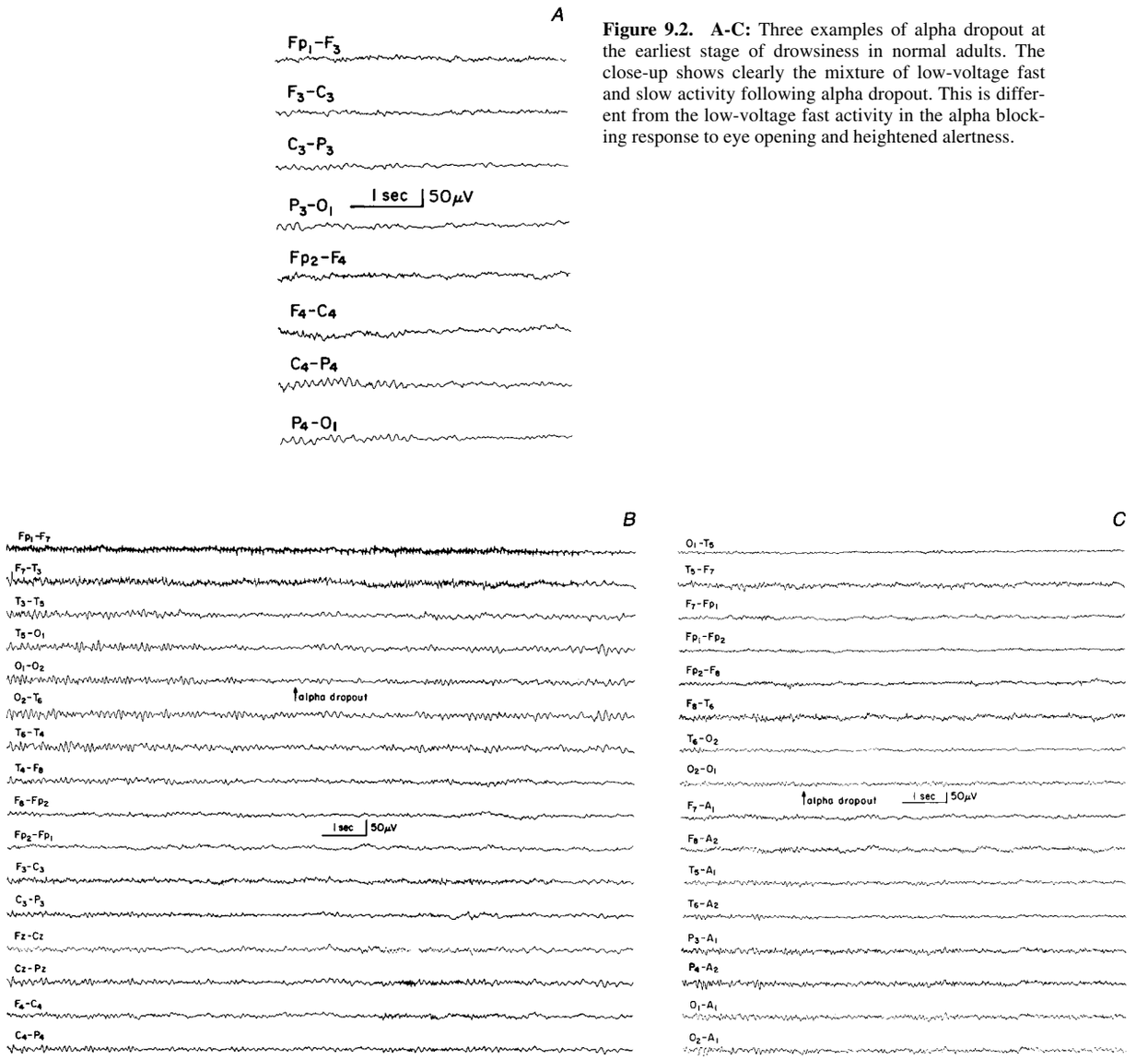

Figure 9.2. A-C: Three examples of alpha dropout at the earliest stage of drowsiness in normal adults. The close-up shows clearly the mixture of low-voltage fast and slow activity following alpha dropout. This is different from the low-voltage fast activity in the alpha blocking response to eye opening and heightened alertness.

11, "Maturation of the EEG: Development of Waking and Sleep Patterns"). In adults with some organic cerebral problems and in old age, a posterior alpha rhythm of normal appearance may be replaced by activity in the theta and delta range; a special pattern with rhythmical widespread (but mainly parietotemporal) 5 to 7/sec activity has been individualized recently by Westmoreland and Klass (1981), who consider this pattern "benign."

Interindividual Differences

When Adrian and Matthews demonstrated their own EEGs on May 12, 1934, at a meeting of the astonished members of the Physiological Society in Cambridge, England, it was found that Adrian's 10/sec alpha rhythm was quite impressive, whereas Matthews produced "no regular waves" (Adrian, 1971). Studies of further subjects showed that Adrian's alpha development was that of the majority, whereas Matthews, the ingenious engineer and designer of Adrian's instrumentation, belonged to a minority of persons with little or no organized alpha rhythm.

Davis and Davis (1936) distinguished four types of records: (a) dominant alpha (found in 20% of healthy adults); (b) subdominant alpha (35%); (c) mixed alpha (20%); and (d) rare alpha (25%). Golla et al. (1943) distinguished three alpha types: M for minus or minimal, P for persistent, and R for responsive. The P type shows no real persistence of alpha, but has a very short blocking response to eye opening (also see Kiloh et al., 1972). Another type of alpha rhythm is the "monotonous high voltage alpha," which shows little or no waxing and waning of the amplitude (Kuhlo, 1976a).

These alpha traits are, to some degree, genetically transmitted (Davis and Davis, 1936; Lennox et al., 1945; Travis and Gottlober, 1936); marked similarities have been re-

ported in identical twins. Here the question arises as to whether these alpha traits are EEG correlates of certain psychological personality traits. Such relationships were suggested by Lemere (1936), who related good alpha development with cyclothymic personalities and poor alpha development with schizothymic personalities (thus using the nomenclature of Kretschmer, 1931). Saul et al. (1937) found high alpha indices (i.e., a large quantity of alpha rhythm) in passive dependent people and low alpha indices in persons with a consistent drive to activity. This could not be confirmed by Lindsley (1938). An assumed correlation with extrovert and introvert personalities was not substantiated by the work of Henry and Knott (1941). Mental work given as tasks revealed a higher alpha percent time in introverts, according to Broadhurst and Glass (1969). Nowak and Marczynski (1981) studied the effects of anxiety traits and stress on the EEG; persons in the high-anxiety group showed stronger and more homogeneous alpha blocking responses, along with "paradoxical" alpha augmentation.

Earlier attempts at correlations between alpha rhythm and intelligence used to be futile or unconvincing (Berger, 1931, 1935, 1938; Gastaut, 1960; Henry, 1944; Kreezer, 1936; Shagass, 1946). More information is found in the handbook article of Knott (1976a). More sophisticated EEG technology appears to have shed more light on this topic. With the use of power spectral analysis, Gasser et al. (1983) found good correlations between alpha rhythm (plus other EEG criteria) and intelligence. Good memory performers are faster in retrieving information from memory and their alpha frequency tends to be higher by about 1/sec (Klimesch et al., 1996).

Intraindividual Differences

A person's EEG traits and alpha rhythm development must not be considered as permanent and unchangeable features like fingerprints. Every EEG tracing of some length gives testimony to a certain degree of variability, even within the state of relaxed vigilance. This is probably the effect of a large number of physiological and psychophysiological variables. To mention just one example, the waxing phase of alpha amplitudes has been related to the waning phase of afterimages (Jasper and Cruikshank, 1937). With advancing age, the alpha frequency tends to decrease (Fisch et al., 1990) but this is probably the effect of cerebral pathology occurring at old age.

Circadian studies of the EEG have been done sparingly (Frank, 1964; Scheich, 1969) and with inconclusive results (for further data, see Harding and Thompson, 1976). The *menstrual cycle* has been thought to influence the EEG and especially the alpha rhythm frequency. According to Dusser de Barenne and Gibbs (1942), a one-half cycle per second drop of the alpha frequency occurs for 1 to 2 days during the ovulation period and a similar decrease of 0.5/sec is also noted on the first day of menstrual flow. Lamb et al. (1953) found a maximum of alpha activity at the time of ovulation and a premenstrual increase of alpha frequency. Pitot and Gastaut (1954) reported alpha slowing (0.3–1/sec) immediately after ovulation and during menstruation. Later work was done with the use of frequency analysis (Harding, 1967; Roubicek et al., 1968) and an integrator (Sugerman et al., 1970). Harding (1967) noted an activity increase in the alpha band during menstruation. Roubicek et al. (1968) observed increasing anterior theta activity at the time of menstruation. Harding and Thompson (1976) have summarized the work in this field as follows:

Preovulatory phase (days 5–14): Alpha frequency increased, amount of beta increased, photic driving reduced.

Postovulation or luteal phase (days 15–23): Alpha slower, amount of alpha increased, less beta and more theta activity, photic driving increased.

Premenstrual phase (days 23–28): Alpha frequency increased, amount of alpha reduced, more beta, less theta activity.

Menstrual phase (days 1–5): Alpha frequency slowed and amount increased, less beta, more theta activity.

According to Harding and Thompson (1976), no consistent changes have been found with the use of oral contraceptives. The study of Creutzfeldt et al. (1976) yielded slightly different results; there was a mild occipital alpha acceleration during the luteal phase (0.3/sec), along with slight decline of cognitive functions. These functions were significantly depressed in the group of women taking oral contraceptives (when compared with the group of women with spontaneous menstrual cycle). Small differences in the EEG of males and females have been reported by Veldhuizen et al. (1993).

Body temperature also modifies the alpha frequency; the alpha rhythm accelerates with increasing body temperature (Gundel, 1984). Therapeutic hyperthermia (up to 41°C) in cancer patients slows down the EEG to the delta range and depresses the overall EEG output (Dubois et al., 1980).

Alpha Conditioning and Alpha Feedback

The method of conditioning reflex formation was introduced in EEG research by Durup and Fessard (1935), who found that normally ineffective auditory stimuli when repeatedly presented in conjunction with visual stimuli were capable of producing alpha blocking. Similar observations had been made in passing by Berger (1930). Further work in this field was done by Cruikshank (1937), Jasper and Cruikshank (1937), Travis and Egan (1938), Knott and Henry (1941), and Jasper and Shagass (1941). Later studies were done by Visser (1961), Hofer and Hinkle (1964), Milstein (1965), and Torres (1968). Knott's (1976a) review of this topic is most illuminating.

Voluntary control of the alpha rhythm and the use of alpha feedback methods have been widely discussed topics since the late 1960s. This work was presumably prompted by the observation of well modulated alpha during meditation practiced by *yogis* (Anand et al., 1961; Bagchi and Wenger, 1958) or *zen buddhists* (Hirai, 1968, and Kasamatsu and Hirai, 1966, both articles cited in Gastaut, 1974). Nowlis and Kamiya (1970) and Brown (1970) associated alpha rhythm enhancement (on a "voluntary" basis) with a pleasant mood. Further work in this field was done by Wallace (1970), Wallace et al. (1971), and Banquet (1973), who

studied states of *transcendental meditation.* Alpha amplitudes increased or decreased; in some subjects there were periods of low voltage theta activity. Knott (1976a) feels that such states are in essence periods of drowsiness and doubts the validity of the view that the high alpha state is a desirable condition. According to Stigsby et al. (1981), there is no consistent EEG pattern associated with successful or unsuccessful transcendental meditation. Gastaut (1974) has sharply criticized all forms of alpha cult. Hypnosis may induce states of relaxed wakefulness or lowered vigilance. The EEG simply reflects the level of vigilance (see Dongier et al., 1976). In the 1980s, the interest in alpha conditioning and alpha feedback started to decline.

Alpha Rhythm, Anxiety, and Emotional Tension

It has been pointed out that a relaxed waking state is the optimal condition for the posterior alpha rhythm. It is hence reasonable to assume that emotional tension attenuates or blocks the alpha rhythm. If this statement is correct, the EEG could serve as a tool for the assessment of emotional tension. This seems to hold true for the state of emotional tension in patients or subjects with pending litigation, after a head injury, or after other forms of physical damage (Scherzer, 1966). In such cases, the tension is, in essence, a state of expectancy pertaining to the outcome of an important test. In other states of tension and, especially, in psychotic individuals described as emotionally very tense, a well-developed alpha rhythm of average amplitude may be present. Catatonic schizophrenics in a state of extreme tension usually show low-voltage fast records.

Alpha Rhythm: Human Versus Animal

The posterior alpha rhythm shows considerable difference from species to species. The alpha frequency usually lies above 10/sec in primates, and the amplitude is surprisingly small in primates (recorded from dural electrodes). In the cat, an 8 to 13/sec rhythm was recorded from the most posterior part of the occipital cortex (Lanoir, 1972). Pampiglione (1963) demonstrated a fairly well developed posterior 6 to 8/sec rhythm in the dog at 1 year of age. This rhythm represents an equivalent of the human alpha rhythm (Storm van Leeuwen et al., 1967).

A 10/sec "alpha-like" rhythm in the visual cortex of the cat was reported by Chatila et al. (1992). Başar and Schürmann (1994) described evoked a alpha responses over the cat's visual cortex, evoked by visual stimuli. These data were obtained with extra- and intracranial recording.

Jurko and Andy (1967) compared the posterior alpha rhythm in three species of macaque monkeys. In rhesus *(Macaca mulatta)* and stumptail *(Rhesus speciosa)* monkeys, fairly well developed alpha rhythms, 10/sec and 12/sec, respectively, were recorded, whereas greater admixture of slow and fast frequencies was found in cynomolgus monkeys *(Macaca ira).* Caveness (1962) found an alpha average frequency of 9.8/sec in *Macaca mulatta.*

The closest resemblance to the human alpha is found in the dog (Storm van Leeuwen et al., 1963, 1967; also see Markand, 1990).

In this context, a study of Götze et al. (1959) on the EEG in healthy and diseased animals ought to be mentioned. This study deals with zoo monkeys and dogs. It is deplorable that collaboration between human electroencephalography and veterinary medicine has not intensified.

Alpha Rhythm and Its Generators

Alpha rhythm is of cortical origin, but the theory of a thalamic pacemaker function has frequently surfaced since the work of Berger (1933a), who presumed cortical genesis but thalamic governance of the alpha phenomenon. Bishop (1936) proposed the concept of corticothalamic reverberating circuits, and Andersen and Andersson (1968) are the proponents of a thalamic theory that is based on presumed similarities between human alpha rhythm and experimental barbiturate spindle activity in animals. According to this theory, the alpha rhythm is driven by presynaptic input to cortical neurons from the thalamic level (also see Andersen and Andersson, 1974; Frost, 1976). This concept has been challenged by Lopes da Silva et al. (1973a,b). Watanabe (1981) has postulated a "somewhat loose but stable oscillator system" subserving the generation of alpha rhythm.

Adrian and Yamagiwa (1935) regarded the alpha rhythm as cortical with maximal involvement of the visual area. Important new vistas were opened with the demonstration of some degree of interhemispheric asynchrony between alpha waves (Aird and Garoutte, 1958). It has been presumed that there is more than one alpha generator within the posterior regions of the cerebrum (Walter et al., 1966); this was further substantiated by depth EEG studies in the human (Perez-Boija et al., 1962). The technique of chronotopography has added further insight into the possibility of multiple sources of alpha generation (Remond, 1968). Further work on interhemispheric phase differences between alpha waves has been carried out with toposcopic analysis (Cooper and Mundy-Castle, 1960) and cross-correlation technique (Liske et al., 1967).

Posterior alpha generation and spread in posteroanterior direction (Walter et al., 1966) appears to be a fact. This concept, however, has been challenged by Inouye et al. (1983), who feel that alpha spread occurs in an anteroposterior direction in the dominant as well as in the nondominant hemisphere. These surprising views are based on the method of entropy analysis.

Our comprehension of alpha-rhythm genesis has not strikingly increased in the course of the 1980s and 1990s. It may be assumed that there are corticocortical and thalamocortical systems that interact in the generation of cortical alpha rhythms (Steriade et al., 1990). From the experimenter's as well as from the clinical EEGer's viewpoint, there is good reason to presume that alpha rhythm is most definitely a cortical phenomenon but there has been, thus far, no evidence of a synchronizing mechanism in cortical level.

The alpha rhythm caught the fancy of some of the most illustrious geniuses of the 20th century. In a book review of *Norbert Wiener, 1894–1964,* by P. R. Masani (Boston: Birkhauser, 1990), Barlow (1991) beautifully pointed out the alpha rhythm theories of Wiener and McCulloch and proceeded as follows:

In the next few years after 1953, Wiener evolved the concept of a stable component of the alpha rhythm (Wiener's interest in the EEG was essentially limited to the alpha rhythm) as a brain clock, to serve a gating function. (In *Cybernetics,* 1948), Wiener cites Pitts and McCulloch's 1947 idea of the alpha rhythm as a scanning rhythm; McCulloch, on the other hand, in one of the Macy Cybernetics Conference volumes, attributes the idea to Wiener's 1940 work on digital computer design.) Wiener added a mathematical basis, entailing nonlinear mutual entrainment (as an example of "self-organizing systems") a few years later (1958) and discussed the general concept at the 1963 Kershman Lecture (for which he had been suggested by John Hughes), under the title, "The Harmonic Analysis of Physiological Phenomena with Special Reference to Electroencephalography." Commentaries on Wiener's concept of the alpha rhythm as a brain clock, by Grey Walter and by this reviewer, can be found in *Survey of Cybernetics* (J. Rose [Ed.], 1969) and in Vol. 4 of *Collected Works* respectively.

If the alpha rhythm were to function as a brain clock, it would be a miserable time keeper. Virtually every EEG record with a well-formed posterior alpha rhythm shows minor or even more prominent fluctuations of its frequency. Such unrealistic alpha theories are simply not compatible with the clinical EEG practice (Niedermeyer, 1997).

An extracerebral artifact theory of alpha rhythm has been proposed by Lippold (1970, 1973), who has presumed that the alpha rhythm is caused by extraocular motor activity ("translational eye tremor modulating the position of the corneo-retinal potential"). There is little to substantiate this hypothesis. Some authors have taken pains to invalidate Lippold's theory; the work of Tait and Pavlovski (1978) has provided convincing evidence of its erroneous premises. Hess (1980) has further corroborated the cerebral genesis of alpha rhythm. The origin of the canine alpha was found in the cortex (phase reversal at laminae IV/V) (Lopes da Silva and Storm van Leeuwen, 1978; also see Steriade et al., 1990). Hogan and Fitzpatrick (1987) observed occipital alpha rhythm in the isolated canine brain after removal of mandible, orbit, and snout.

No neurophysiological or psychophysiological alpha rhythm theory has yet found general acceptance, and there are still uncertainties about the origin and psychophysiological significance of this remarkable phenomenon. And yet, our insights into the nature of the alpha rhythm (and other EEG rhythms) have been deepening. The ceaseless work of experimental neurophysiologists, psychoneurophysiologists, and neurotheoreticians has provided us with a better view of the nature of the alpha rhythm. Lopes da Silva (1991) has pointed out that "EEG signals can reflect functional states of neuronal networks." Accordingly, oscillatory mechanisms allow for changes between different behavioral modes. As in Wagnerian music, each oscillatory state may serve as a leitmotiv evoking different sets of mental associations and emotional states.

Rolandic (Central) Mu Rhythm

Rolandic (central) mu rhythm is in frequency and amplitude related to the posterior alpha rhythm, but its topography and physiological significance are quite different. Historically, the existence of a special central rhythm was presumed by Jasper and Andrews (1938) ("precentral alpha rhythm"), Maddocks et al. (1951) ("alphoid activity"), and Schütz and Müller (1951) ("high voltage rolandic alpha"). The features of mu rhythm were first described in detail by Gastaut et al. (1952) and Gastaut (1952), who also included electrocorticographic tracings; these authors introduced the term *rhythme rolandique en arceau*. The epithet *en arceau* alludes to the arch-shaped wave morphology, which has also prompted the term *wicket rhythm* (Gastaut et al., 1954). Magnus (1954) used the term *central alpha* (see also Jasper and Andrews, 1938). Other terms are *arcade rhythm* (Van der Drift and Magnus, 1961), *comb rhythm* (Cobb, 1963), and *somatosensory alpha rhythm* (Kuhlman, 1978a).

Mu rhythm is not detectable in every mature subject; as will be discussed later, its prevalence is limited unless the "hidden" mu rhythm is visualized with special methods. Mu stands for motor; this rhythm is strongly related to functions of the motor cortex, but the contribution of the adjacent somatosensory cortex must not be ignored.

Mu rhythm and associated beta activity over the sensorimotor cortex have become a topic of special interest during the 1990s. As a matter of fact, mu rhythm as an interesting clinical EEG phenomenon has transcended into a powerful contributor to the understanding of motor activity in general. This development started with the work of Pfurtscheller on event-related desynchronization (ERD) (Pfurtscheller and Aranibar, 1978a,b)—a phenomenon briefly mentioned in our discussion of posterior alpha rhythm reactivity. The ERD pertains even more strongly to the rolandic mu rhythm (and furthermore to various cognitive tasks and their cortical EEG accompaniment) (Pfurtscheller, 1981, 1990, 1992; Pfurtscheller and Klimesch, 1992; Pfurtscheller and Neuper, 1992; Pfurtscheller et al., 1994). In other words, the ERD has become a new criterion for the assessment of cortical functioning, over the motor cortex but also over various cortical regions of strong afferent input related to neurocognitive activities. Further detail on the ERD is found in the work of Pfurtscheller and Lopes da Silva (1999a,b) and Chapter 51 of this book, also written by Pfurtscheller and Lopes da Silva.

Age and Prevalence

Central mu rhythm used to be considered scarce. The introduction of the International Electrode System (10–20 system) has contributed to a much greater awareness of this pattern. The C_3 and C_4 electrodes are located over the precentral gyrus in an optimal location for picking up central mu rhythm. Large material tested with a different electrode system showed a prevalence of only 3.2% (Klass and Bickford, 1957) and 2.9% (Schnell and Klass, 1966). This lies well below the figures found in other studies.

Gastaut et al. (1954) found mu rhythm in 10% of their adult patients; Beek (1958) observed this rhythm in 13% of a predominantly psychiatric patient population. In 500 essentially healthy young male adults, Gastaut et al. (1959a,b) found mu rhythm in 14.4%; the mu rhythm was often found in persons with certain psychopathic personality traits as evidenced by the Minnesota Multiphasic Personality Inventory (MMPI) test. Dongier and Dongier (1958) noted mu rhythm in 18% of a population of neurotic patients. Figures of 12% were reported by Picard et al. (1955) and Simonova et al. (1966).

In the patients of Niedermeyer and Koshino (1975), the prevalence of mu rhythm was 8.1% (182 of 2,248); broken down into age ranges, there were 9.0% between ages 0 and 10 years, 13.8% between 11 and 20 years, 8.4% between 21 and 40 years, and 4.5% above 41 years. These authors dem-

onstrated rolandic mu rhythm in a 20-month-old child; this was thought to be an exceptionally early manifestation of mu rhythm. But was it really exceptionally early? Not when one considers the data of Stroganova et al. (1999), who demonstrated central mu rhythm (during a state of attention) in the tracing of an 8-month-old baby with a frequency of about 6 to 8.8/sec. The authors contend that mu rhythm tends to appear before the occipital alpha-equivalent because, unlike visual stimulation, somatosensory stimulation is present within the uterus.

Familial occurrence of mu rhythm has been reported by Koshino and Isaki (1986). With the use of frequency analysis, the prevalence of mu rhythm reaches values close to 100% (Schoppenhorst et al., 1980).

Wave Morphology, Frequency, and Spatial Distribution

Older synonyms such as *rhythme en arceau* or *wicket rhythm* pertain to the wave morphology; mu rhythm shows in most instances a sharp (or spiky) negative and a rounded positive phase. There are a few patterns with a monophasic spiky appearance. Posterior alpha rhythm may have a similar configuration: "wicket spikes" (Fig. 9.3) have a similar appearance. The same is true for 14 and 6/sec positive spikes, except for the spike positive and rounded negative compounded. (As to negativity and positivity, see Chapter 8, "The EEG Signal: Polarity and Field Determination.") All these patterns show a spatial distribution that is different from the mu rhythm or occurs at levels of vigilance that are usually incompatible with mu rhythm. The amplitudes are comparable to those of the posterior alpha rhythm (Fig. 9.3).

The most common frequency of the mu rhythm is 10/sec. Frequencies may lie below 9/sec and above 11/sec; a mu rhythm of less than 8/sec is probably a mild abnormality. Mu rhythm is often mixed with local activity around 20/sec (Fig. 9.4). According to Storm van Leeuwen et al. (1978), mu frequencies are slightly higher than alpha frequencies. The spatial distribution is essentially confined to the precentral-postcentral region; some spread into parietal leads is not uncommon. The C_3 and C_4 electrodes are mostly involved; occasionally, a vertex (C_z) maximum is noted. A special

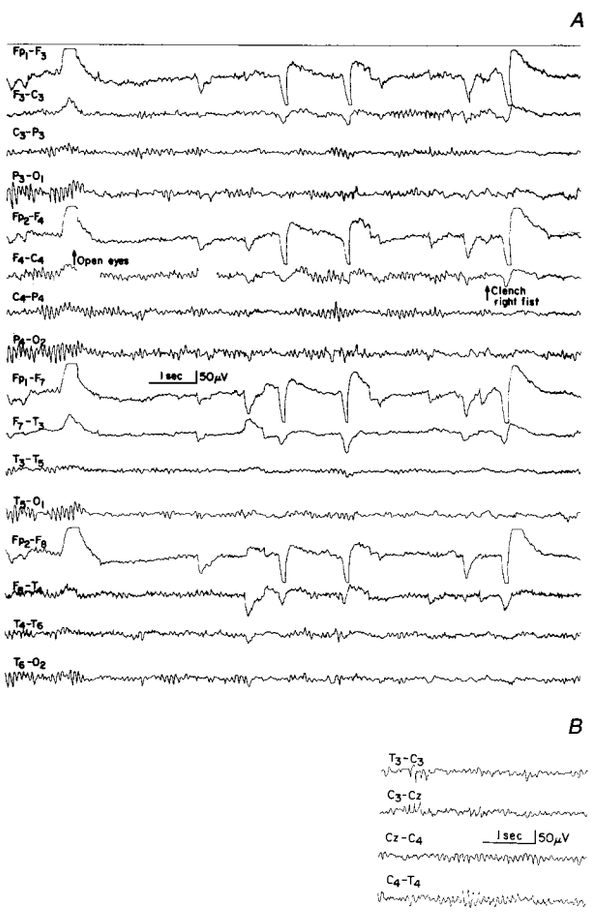

Figure 9.3. **A:** Rolandic mu rhythm, in a patient age 13 years. Stretches of central mu rhythm while eyes are kept open. These runs occur asynchronously on both sides and are blocked with clenching of right fist. Some posterior alpha rhythm persists after eye opening. Possible spread of mu rhythm into the parietal area makes differentiation of alpha and mu difficult in right parieto-occipital lead (eighth channel from the top). **B:** The close-up view of the same patient shows independent bilateral central mu rhythm.

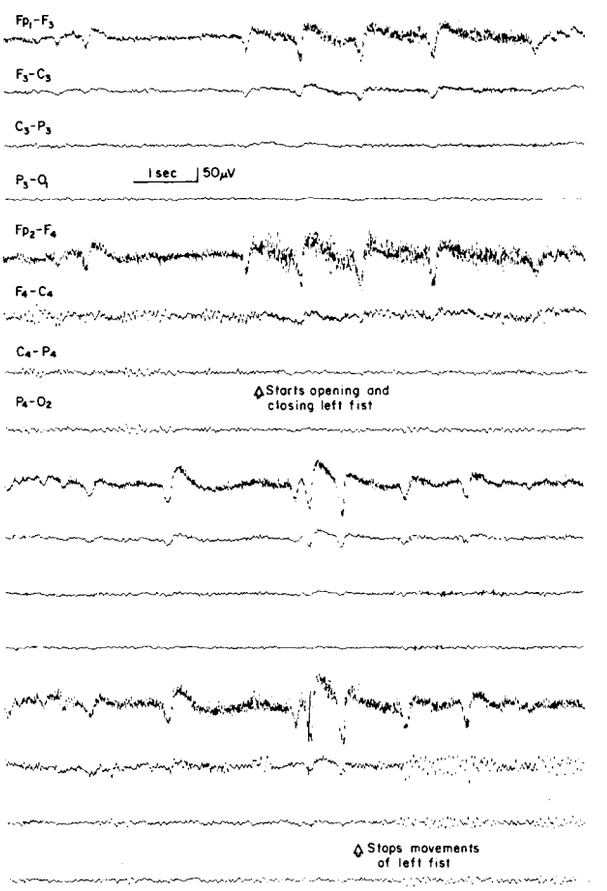

Figure 9.4. Patient aged 51, 2 years after evacuation of a right-sided subdural hematoma. A right-sided central mu rhythm shows admixture of fast frequencies and spiky potentials. All components of this rhythm show a prominent and persistent blocking response to contralateral hand movements. (From Niedermeyer, E., and Koshino, Y. 1975. My-Rhythmus: Vorkommen und Klinische Bedeutung. *Z. EEG-EMG.* 6:69–78.)

vertex mu rhythm with special reactivity (lower limb) was described in aged patients by Farnarier et al. (1981). When recorded through bone defects, the mu rhythm is more widely distributed. According to Kuhlman (1978a), the maximum lies over the postcentral rather than the precentral cortex.

The alphoid (around 10/sec) and the fast (around 20/sec) component of the mu rhythm seem to be inseparably intermingled but, with more advanced technology, spatial separation is possible. According to Salmelin and Hari (1994a,b) the beta component arises from the motor cortex and the alphoid component from the sensory cortex. These magnetoencephalographic findings have been confirmed by Nashmi et al. (1994).

Mu rhythm usually occurs in short stretches. Persons with pronounced mu rhythm show long trains of mu rhythm.

In most persons with mu rhythm, this activity is bilateral but tends to shift from side to side. Coherence function studies have shown lack of bilateral coherence of mu rhythm in normal subjects (Storm van Leeuwen et al., 1978). Strictly unilateral mu rhythm must be scrutinized for the possibility of an ipsilateral rolandic disturbance as, for instance, an early stage of parasagittal meningioma, an arteriovenous malformation, or other types of neoplasm (Tannier et al., 1983). In such cases, even the possibility of a contralateral rolandic lesion must be taken into consideration (Gastaut, 1952; Hess, 1975; Kubicki et al., 1973). Above all, the possibility of a local cranial bone defect, surgical or traumatic, must be ruled out; a single burr hole in the rolandic region enables otherwise hidden mu rhythm to become manifest on the scalp (Fig. 9.4). Thus, mu rhythm represents at least a portion of rhythmical activity beneath bone defects ["breach rhythm," (Cobb et al., 1979)]; also see The "Third Rhythm" (Independent Temporal Alphoid Rhythm), below.

Reactivity

Mu rhythm is blocked by movements. These movements may be active (voluntary), passive, or reflexive (Chatrian, 1964, 1976a; Chatrian et al., 1959; Gastaut, 1952; Gastaut et al., 1952). The blocking effect is bilateral but more pronounced on the rolandic region contralateral to the site of movement; the effect appears prior to the onset of muscular contraction (Chatrian, 1976a; Chatrian et al., 1959; Gastaut, 1952; Gastaut et al., 1952; Klass and Bickford, 1957). According to Chatrian et at. (1959), there are delays of 50 msec to 7.5 seconds (average, 1.5 seconds) at the onset of the mu blocking effect after the initiation of the spontaneous flexion of the contralateral thumb, with the ipsilateral lagging behind the contralateral response. Gastaut et al. (1952) and Ciganek (1959) demonstrated a somatotopic distribution of the blocking effect (according to the functional anatomy of the rolandic cortex), but this observation could not be confirmed by Chatrian et al. (1959).

With the use of a gamma band (38–40/sec) frequency analysis focusing on the event-related synchronization (ERS) of the gamma component, Pfurtscheller et al. (1994) beautifully demonstrated the locus for right and left index finger movement, right toes, and the rather broad and bilateral area for tongue movements.

The mu blocking response is likely to be related to the conceptual design of the movement to be executed. Mere thoughts about performing movements and readiness to move block the mu rhythm (Chatrian, 1964, 1976a; Chatrian et al., 1959; Gastaut, 1952; Gastaut et al., 1952; Klass and Bickford, 1957). Mu blocking responses have also been demonstrated in persons with amputations of extremities; these mental activities concern movement of a phantom limb (Gastaut et al., 1965; Klass and Bickford, 1957).

Light tactile stimuli also produced a mu rhythm blocking effect that is most evident over the contralateral rolandic region (Chatrian, 1964, 1976a; Chatrian et al., 1959; Gastaut, 1952; Gastaut et al., 1952; Magnus, 1954). Kuhlman (1978a) has placed even greater emphasis on the sensory component and regards mu rhythm as the "idling" of the cortical sensory region, in analogy to the alpha rhythm as "idling" of the visual cortex and its vicinity. His work was carried out with power spectral analysis. This technique shows that central mu rhythm is a more common phenomenon than one would expect from the visual analysis of the scalp EEG (also see the power spectra study of Pfurtscheller and Aranibar, 1978a). These authors also showed simultaneous posterior alpha desynchronization (Pfurtscheller and Aranibar, 1978a,b). Coherence functions have also been used for the demonstration of rolandic mu rhythm (Schoppenhorst et al., 1980; Storm van Leeuwen et al., 1978).

It has been noted that rolandic mu rhythm is enhanced during intermittent photic stimulation (Brechet and Lecasble, 1965) and pattern vision (Koshino and Niedermeyer, 1975). The latter observation would contradict the basic law of mu rhythm as a rhythm of immobility; one is tempted to perceive the extraocular movements involved in pattern vision as a different ("extrarolandic") system in the motor organization.

Mu Rhythm, the (Slower) 20/Sec Beta and the (Faster) 40/Sec Beta (or Gamma) Components

The separation of the mu rhythm proper (around 10/sec) and the 20/sec beta component have been mentioned earlier. The 40/sec gamma component is not a spontaneous rhythm; rather, it constitutes the fast activity associated with the enhancement (event-related synchronization, ERS) of small preexisting activity with movement according to Pfurtscheller and his co-workers.

Cortical Rolandic Basis of Mu Rhythm

Jasper and Penfield (1949) found evidence of a strictly localized beta (around 20/sec) over the human motor cortex with electrocorticographic recording technique in the locally anesthetized patient. This rhythm could be blocked like mu rhythm with movement, especially contralateral movement, and also with thinking about the execution of movement. This rhythm is obviously the cortical equivalent of rolandic mu rhythm on the scalp. The mu waves on the scalp often show some notching or are imbedded in local fast activity, which is also blocked by movement. Gastaut (1952) confirmed the cortical precentral fast activity in electrocorticographic tracings. These data conflict with Kuhlman's (1978a) observation of a postcentral maximum of rolandic mu rhythm (scalp recordings with power spectral analysis). On the other hand, Graf et al. (1984) demonstrated precentral 8 to 10/sec mu rhythm in the electrocorticogram of an adult epileptic.

As mentioned earlier, mu rhythm around 10/sec on the scalp and cortical rolandic 20/sec activity appear to be harmonically related; such a relationship, however, does not exist between mu rhythm and rolandic beta activity on the scalp according to Pfurtscheller (1981) who used power spectral analysis. Quantitative EEG studies have also established a clean separating line between mu rhythm and posterior alpha rhythm (Van Huffelen et al., 1984).

A study of the functional significance of the mu rhythm from the human cortex was carried out with subdural electrodes by Arroyo et al. (1993). This work also showed the relationship of the cortical mu rhythm to the well-known somatotopic arrangement of the sensorimotor cortex.

Mu Rhythm-Like Activity in the Experimental Animal

A sensorimotor 7 to 14/sec rhythm has been demonstrated in quiet cats trained to obtain a reward (Chase and Harper, 1971; Sterman et al., 1969, 1970; Wyrwicka and Sterman, 1968). A similar rhythm of immobility was shown by Rougeul et al. (1972) in the cat; this is a 14/sec rhythm presumed to originate in the nucleus ventralis posterior of the thalamus (Bouyer et al., 1983).

Mu Rhythm Conditioning and Feedback Training

An impressive international study on mu rhythm and the phasic evolution of conditioning was presented by Gastaut et al. (1957). Mu rhythm feedback training has been used for various purposes, including the treatment of epileptic seizure disorders (Finley et al., 1975; Kuhlman, 1978b,c; Sterman et al., 1974). Roldan et al. (1981) observed the development of mu rhythm-like activity in the course of Hatha yoga exercises. This activity was called chi rhythm. It was believed to be a specific rolandic pattern of 11 to 17/sec waves and theta frequencies.

Evidence of More than One Type of Rolandic Mu Rhythm

This question has been raised by Covello et al. (1975), who feel that several entities of mu rhythm exist, even in one subject, and that their underlying physiological mechanisms may differ. Personal impressions lend some support to this idea. Earlier observations (Niedermeyer and Koshino, 1975) led to the conclusion that even the slightest degree of lessened vigilance (i.e., earliest drowsiness) was incompatible with mu rhythm; this view has been supported by Schoppenhorst et al. (1980). However, there have been some personal observations of patients demonstrating their best stretches of central mu rhythm in the light drowsy state after posterior alpha dropout. Yamada and Kooi (1975) even described mu rhythm in sleep [stages 1 and 2, as well as rapid-eye-movement (REM) sleep] in 20 individuals (19 patients with various complaints). The concept of Covello et al. (1975) is certainly in need of further study.

Mu Rhythm Status

The word *status* is not necessarily reserved for sustained epileptic activity (e.g., status migrainosus, status asthmaticus). Unusually sustained mu rhythm activity ("mu rhythm status") has been observed by Niedermeyer et al., 2004, in the setting of frontal lobe impairment and also in the absence of pathology (perhaps a side effect of levetiracetam).

Clinical Significance of Mu Rhythm

Rolandic mu rhythm as such is categorically within normal limits. In some cases, unusually spiky discharges may be present and even single spikes may stand out; such tracings are slightly abnormal. Children with benign rolandic epilepsy (see Chapter 27, "Epileptic Seizure Disorders") may gradually develop mu rhythm when rolandic spike activity subsides and the seizures appear to be under control. In such patients, grand mal or focal motor attack seizures may return later in adult life under unusual circumstances, such as severe stress with insomnia or infections. There are indeed a certain number of adults with rare grand mal attacks and normal EEG tracings featuring some central mu rhythm. This could be merely a coincidence, but there is reason to presume that mu rhythm may have a certain relationship to epileptic seizure disorders. Interestingly, in some children with benign rolandic epilepsy, cerebral spikes can be blocked by contralateral movements in the same manner as mu rhythm is blocked (Niedermeyer, 1972a,b). This response is not present in all children with rolandic spikes; this explains negative responses to motor activity reported by Isch-Treussard (1972).

Patients or control subjects with rolandic mu rhythm appear to be more prone to headaches than the average population (Schnell and Klass, 1966). Gastaut et al. (1954) and Bostem et al. (1964) emphasized relationships between mu rhythm and a variety of dysfunctions such as migraine, bronchial asthma, peptic ulcer eczema, tinnitus, arterial hypertension, and hyperthyroid states. Mu rhythm has been thought to be related to a low pain threshold (Niedermeyer et al., 1982).

According to Dongier and Dongier (1958), mu rhythm is common in patients with mild to moderate psychiatric disorders; it is associated with anxiety, aggressiveness, emotional instability, and psychosomatic disorders. Netchine et al. (1964) presented a psychodynamic theory of mu rhythm that is thought to be enhanced with repressed aggression and absent in patients with overt aggressiveness.

Relationships to other EEG patterns of marginal or mildly abnormal character have been pointed out (14 and 6/sec positive spikes, 6/sec spike waves; see Chapter 13). These patterns are often found in association with mild to moderate psychiatric disturbances and autonomic nervous system dysfunction.

Does this mean that rolandic mu rhythm—generally conceived as a physiological pattern—is, in reality, a mild abnormality? Let us reflect for a moment on the earlier statement that every healthy adolescent and adult harbors central mu rhythm when power spectra and coherence tests are utilized (see the aforementioned work of Schoppenhorst et al., 1980). Why then is mu rhythm evident in the conventional EEG in just a minority of persons? The answer is quite logical: their mu rhythm has to be more powerful than that of average persons in order to be picked up from the scalp. Thus, an individual with mu rhythm in the conventional EEG (and optimally placed electrodes) might already

have a certain "excess" of mu rhythm. Such a presumed excess could be an indicator of local hyperexcitability. From here, the road could lead to autonomic (vegetative) dysfunctions, emotional dyscontrol and even to epileptic activities. Thus, life without a (conventionally recordable) mu rhythm could be easier than life with excessive mu rhythm activity.

In cases of strictly unilateral mu rhythm, a careful search for bone defects (bur holes) and/or local pathology is indicated. In the wake of ischemic hemiplegic insults, central mu rhythm may disappear or its reactivity may vanish (Pfurtscheller, 1986).

Theories of Neurophysiological Basis of Mu Rhythm

There are in essence three theories to be dealt with: (a) the hypothesis of "neuronal hyperexcitability" ("hyperexcitabilité neuronique"), either diffuse or locally restricted to the rolandic cortex (Beek, 1958; Gastaut, 1952; Gastaut et al., 1952; Van der Drift and Magnus, 1961); (b) the hypothesis of superficial cortical inhibition ("inhibition corticale superficielle"), comparable to aforementioned theories of "cortical idling"; this theory would explain the suppression of mu rhythm with motor activity (Bostem et al., 1964, 1965; Gastaut et al., 1964, 1965); and (c) the hypothesis of somatosensory "cortical idling," turning mu rhythm into an afference-dependent phenomenon (Kuhlman, 1978a).

In the discussion of central mu rhythm, simple mechanical factors must never be forgotten, not only the well-known facilitation of mu rhythm by local defects of bone, but even the possibility of an unusually thin scalp locally over the rolandic region.

Breach Rhythm (Rhythms Over Skull Defects)

The presence of cranial bone defects has considerable effect on the EEG frequency spectrum recorded from the overlying scalp. Fischgold et al. (1952) focused attention on this subject. In the following, this topic is discussed chiefly in connection with rolandic mu rhythm.

As has been pointed out, rolandic mu rhythm may become very prominent beneath a bone defect; this EEG pattern is naturally suppressed by motor activity. There is, however, a rhythmical activity in the 6 to 11/sec range that does not respond to movements and is found mainly over the midtemporal region (electrodes T_3 or T_4). This activity often shows spike character. The work of Cobb et al. (1979) has beautifully demonstrated the characteristics of this type of "breach rhythm" as a special entity among the EEG patterns. Bone replacement may or may not lead to some reduction of the midtemporal rhythm. Breach rhythm of the midtemporal variety is possibly an abnormal rhythm related to cerebral pathology; for this reason, its discussion in this chapter on the normal EEG may not be appropriate. In addition to surgical and traumatic skull lesions, an osteolytic skull metastasis can also give rise to local breach rhythm (Radhakrishnan et al., 1994).

The "Third Rhythm" (Independent Temporal Alphoid Rhythm)

Rhythmical activity in the alpha and upper theta range can be picked up from epidural electrodes over the midtemporal region (Niedermeyer, 1990a-c, 1991, 1997). This rhythm is not detectable in the scalp EEG unless there is a local bone defect in the midtemporal region where it has been termed (together with coexisting central mu rhythm in the vicinity) "breach rhythm" by Cobb et al. (1979), who regarded the temporal activity as an abnormal rhythm.

There is now good reason to presume that the rhythmical temporal lobe activity, which is clearly independent from the posterior alpha and rolandic mu rhythm, constitutes a physiological rhythm.

The presence of such an independent temporal alpha rhythm has also been demonstrated with the use of magnetoencephalography in healthy adults by Hari (personal communication, 1990, 1991), who feels that this rhythm is strongly related to the cortical auditory function ("auditory alpha rhythm") (see Hari's Chapter 57, "Magnetoencephalography as a Tool of Clinical Neurophysiology"). Personal data (Niedermeyer, 1990a-c, 1991) do not provide good evidence for a relationship to auditory function. In some individuals, cognitive activities result in blocking responses of the third rhythm. It seems that the function of this rhythm is still debatable.

In the wake of the usual partial temporal lobectomy for the treatment of temporal lobe epilepsy, the third rhythm cannot be recorded despite the presence of surgical bone defects. Naturally, the auditory cortex remains preserved in this procedure. If the concept of an "auditory alpha rhythm" were correct, one would expect a particularly strong third rhythm after removal of overlying neocortical temporal lobe tissue. This is one more reason for disagreement with Hari's view.

Rhythmical activity in the alpha and upper theta range over the anterior temporal–midtemporal region in conventional scalp EEGs can be found in patients with cerebrovascular problems (Kendel and Koufen, 1970) and other types of temporal lobe changes but this abnormal rhythm can be differentiated from the physiological "third rhythm" (Niedermeyer, 1991).

Over the ensuing years, there has been almost no discussion concerning the nature of the third rhythm/tau rhythm. Bastiaansen et al. (1999) were unable to reach conclusions about these rhythms. Shinomiya et al. (1999) studied the third rhythm in eight patients with local bone defects. These authors carefully distinguished the third rhythm from the rhythmical form of temporal minor slow and sharp activity in cerebrovascular disorder.

Kappa Rhythm

This anterior temporal rhythm in alpha frequency was first reported by Laugier and Liberson (1937) and subsequently studied by Kennedy et al. (1948). Chatrian (1976b) has presented a lucid review of the literature on this phenomenon, which is unlikely to be an authentic cerebral rhythm. It is possibly an ocular artifact caused by discrete lateral oscillations of the eyeballs. Kappa rhythm has become all but forgotten and may be regarded as a nonissue in the field of EEG.

Beta Rhythms

The Greek letter *beta* stands for the frequency band above 13/sec. In customary EEG recording, frequencies

above 70/sec are considerably attenuated due to filter effects. This establishes a natural upper limit of the beta band somewhere between 50 and 100/sec; otherwise, the upper limit of the beta band is open-ended. Faster chart speed or even special technical equipment is needed for studies of the fast frequency band. Any rhythmical EEG activity above 13/sec may be regarded as beta rhythm. Rhythmical beta activity is encountered chiefly over the frontal and central regions; it usually does not exceed 35/sec. A central beta rhythm is related to the rolandic mu rhythm and can be blocked by motor activity or tactile stimulation [see Rolandic (Central) Mu Rhythm, above]. The amplitude of beta activity seldom exceeds 30 μV. Beta activity may be locally enhanced over bone defects and shows considerable increase (in quantity and voltage) after the administration of barbiturates, some nonbarbituric sedatives, and minor tranquilizers. With very large amounts and high amplitudes of beta activity, abnormal proportions may be reached; in general, however, fast activity alone is seldom a cogent reason for calling a tracing abnormal.

Historically, the discovery of beta activity is closely linked to the first description of the alpha rhythm by Berger (1929). His first report on the human EEG of 1929 contains the following passage: "The electroencephalogram represents a continuous curve with continuous oscillations in which . . . one can distinguish larger first order waves with an average duration of 90 sigma (msec) and smaller second order waves of an average duration of 35 sigma (msec)" (after Gloor, 1971). In his second report, Berger (1930) pointed out: "For the sake of brevity I shall subsequently designate the waves of first order as alpha waves, the waves of second order as beta waves." Berger (1938) reversed earlier views and pointed out "that the beta waves and not the alpha waves of the EEG are the concomitant phenomena of mental activity." This concept is now provoking renewed interest.

As to differences between beta and gamma (around or above 40/sec) activity, the reader should return to the beginning of this chapter. The old term *gamma* for the designation of faster beta activity used to be all but forgotten until its resurrection around 1990.

Prevalence of Beta Activity

Beta activity is found in almost every healthy adult. Simon's (1977) statement that beta frequencies have been found in 22% of normal adults probably applies to more conspicuous forms of beta activity. Depth EEG studies and electrocorticograms show a remarkably large share of the beta band; such recordings have been obtained mostly in epileptics or in patients with neurological or psychiatric problems. Although obtained in patients and not in healthy persons, such depth records are quite informative concerning the spatial distribution of fast frequencies. Every scalp recording performed over a bone defect gives testimony to the wealth of cortical beta activity.

The prevalence of beta activity in the normal person can also be derived from various studies of normal volunteers (Brazier and Finesinger, 1944; Finley, 1944; Gallais et al., 1957; Gibbs et al., 1943; Mundy-Castle, 1951, 1953; Obrist, 1954; Picard et al., 1957; Roger and Bert, 1959; Vogel and Fujiya, 1969). The results of these studies have been reviewed by Kuhlo (1976b) in his thorough EEG handbook article on beta rhythms. Later work has been presented by Fortuin and Künkel (1983) and Kozelak and Pedley (1990).

The Gibbsian Approach: Distinction According to Beta Quantity

Gibbs and Gibbs (1950) distinguished two types of predominantly fast records. A moderate increase of fast activity was termed "F1" and a marked increase, "F2." Records of the F1 type were regarded as abnormal until the 40th year; F2 type records were regarded as abnormal at any age (Gibbs and Gibbs, 1950). Even an F3 type ("exceedingly fast") was introduced by Gibbs and Gibbs (1950).

Electroencephalographers have shown a more lenient philosophy toward fast tracings over the ensuing years. Beta activity must be particularly abundant in quantity and of rather high voltage to be termed abnormal. Even such fast records are categorically but slightly abnormal unless they occur in unresponsive patients, where such fast activity may represent a severe abnormality. This view is essentially congruent with the study of Drake (1984).

Further Attempts at a Differentiation of Beta Activities

Spatial Characteristics

In certain individuals, the posterior alpha rhythm is unusually fast and its frequency exceeds the upper limit of the alpha rhythm (13/sec). Such a 14 to 15/sec rhythm shows a good blocking response to eye opening and enhancement with eye closure; it may be considered a fast equivalent of the alpha rhythm. Gradual acceleration of the alpha frequency may be caused by hyperthyroidism (see Chapter 22, "Metabolic Central Nervous System Disorders," Fig. 22.9). Vogel and Götze (1962) separated the occipital 14 to 19/sec rhythm from the rest of beta activities, which were divided into frontocentral bursts of 20 to 30/sec waves and normal occipital alpha rhythm and diffuse beta activity generally mixed with alpha frequencies. Vogel (1962, 1966, 1970) subsequently divided the anterior beta activity into the 25 to 30/sec activity of the frontal region, occurring in brief trains, and the 20 to 25/sec activity of comparatively larger amplitudes occurring for variable periods.

Frontal runs of beta activity often exceed 30/sec and reach 35 to 40/sec when the patient is allowed to fall asleep. This activity may be found in unmedicated persons; sleep-inducing medication, which increases the activity in the 18 to 25/sec band, also augments activities in the faster beta range.

The region of fastest EEG frequencies and highest levels of cerebral blood flow (as well as oxygen and glucose uptake) is located in the frontocentral area. This observation has prompted the concept of "cerebral hyperfrontality" (Ingvar, 1987). This investigator has pointed out that (a) prefrontal activity constitutes an integral part of consciousness, and (b) abnormal prefrontal activity is accompanied by various forms of altered consciousness.

The physiological beta frequencies may be broken down as follows:

1. Frontal beta: fairly common, may be very fast, no relationship to physiological rhythm.

2. Central beta: partly but not generally the basis of rolandic mu rhythm often mixed with mu rhythm.
3. Posterior beta: often a fast alpha equivalent, reactive like alpha rhythm.
4. Diffuse beta: no linkage with any special physiological rhythm.

By contrast, "vertex beta" or "fast central midline rhythm" (Veilleux et al., 1988)—vertex activity in the 10 to 25/sec range—appears to be the expression of polyetiological focal abnormality in the frontocentral region.

Relationship to Personality Traits

Beta activity over the central region reportedly is absent in emotionally stable persons (Gallais et al., 1957; Picard et al., 1957; Remond and Lesèvre, 1957). Gastaut (1957) pointed out that the central fast rhythm and the closely related mu rhythm correlate with aggressive, domineering, and dynamic personality traits. Gastaut et al. (1960), however, felt that more detailed statistical analysis could not corroborate the aforementioned correlations.

In the older literature, fast activity was thought to be more common in patients with psychiatric problems (Finley, 1944). Cohn (1946) noted frontal beta activity (18–22/sec, 10–50 μV) in patients suffering from anxiety states. Gibbs and Gibbs (1950) reported a high incidence of very fast activity (above 30/sec) ("F3 type") in "dull psychopaths." Giannitrappani and Kayton (1974) demonstrated a prominent peak of 29/sec activity with the use of power spectral analysis in schizophrenic patients. A discussion of correlations between fast activity and neurological disorders would be inappropriate in a chapter dealing with the normal EEG. During the entire maturational period (including early adulthood), beta activity has been found to be relatively more prominent in females than in males (Matsuura et al., 1985).

Beta Activity and Drugs

This subject is discussed in Chapter 34, "EEG, Drug Effects, and Central Nervous System Poisoning."

Beta Activity at the Onset of Movements

Rhythmical 40/sec activity has been demonstrated over the left central region (C3 electrode) at the onset of self-paced voluntary right finger movements (Pfurtscheller and Neuper, 1992). With "event-related desynchronization" of local rhythmical activity around 10/sec in the movement-programming stage, a burst of 40/sec activity ("event-related synchronization") coincides with the initiation of movement. Naturally, the 40/sec activity had to be carefully separated from muscle potentials.

"Ultra-fast" EEG Activity

Interest in the ultrafast range from 80 to 1,000/sec has been surging over the past few years. The availability of digital EEG recording has flung open the gate to this new domain and it is just a matter of a few years and is going to stimulate new and very promising work. This range will provide us with new epileptological insights (Draguhn et al., 2000; Niedermeyer and Sherman, 2001a,b; Schiff et al., 2000) but also, above all, with a new understanding of cortical perception, motor activity, and, in particular, neurocognitive processes (Curio, 2000 a,b; Traub et al., 1999; Ziemann and Rothwell, 2000). A new chapter on ultrafast activity by Curio has been added to this volume (see Chapter 26).

Figure 9.5. The EEG of the 21st century. (See Color Figure 9.5.)

Parallel with this new wave, there has been also renewed interest in ultraslow activity, from near zero (DC) up to 0.3/sec available nowadays. This range can improve focal epilepsy diagnosis (Vanhatolo et al., 2003, who have also contributed a new chapter to this volume; see Chapter 25). This range will also be fruitful for neurocognitive research and motor initiation not to leave aside sleep studies in prematurity (Vanhatolo et al., 2002).

Figure 9.5 illustrates these new research direction as the basis for the EEG of the 21st century. Interestingly, the very optimistic study on the future of EEG in assessing neurocognitive functions (Gevins, 1998) does not explicitly mention the possibilities provided by and enlarged frequency band but speaks of "fine-grain temporal resolution."

Very fast activity (beyond the conventional EEG range) has been found in cerebellar structures of animals, mostly in the range of 200 to 300/sec (Adrian, 1934; Bremer, 1958; Dow, 1938; Snider, 1950). Trabka (1963) found similar high EEG frequencies in the cerebrum of cats.

The Low-Voltage Record

A below-average voltage output is compatible with perfect central nervous system (CNS) functioning. In such individuals, the small amplitudes are the result of a lesser degree of synchronization of electrical activity in the neuronal level. Brainstem reticular formation systems strongly influence the degree of cortical neuronal synchronization; arousal mediated through the ascending mesodiencephalic reticular for-

mation has a desynchronizing effect (Moruzzi and Magoun, 1949), whereas synchronizing effects are presumed to originate from the pontine portion of the ascending reticular formation (Magnes et al., 1961; Magni et al., 1959).

This type of brainstem regulation of the cortical EEG synchronization is likely to account for numerous fluctuations in the EEG amplitudes, especially those related to the level of vigilance. It is not clear whether these reticular effects account for a low-voltage EEG as a personal trait (with genetic determination, as will be pointed out later). Under pathological conditions, low-voltage tracings in vertebrobasilar artery insufficiency (to be discussed in Chapter 17) are probably due to some degree of pontine ischemia acting on the reticular formation. This could also be true for the frequently encountered low-voltage tracings of chronic alcoholics in advanced stages, with the possibility of early stages of pontine myelinolysis; this explanation, however, is but a working hypothesis (Lawson and Niedermeyer, 2002). True decline of the cerebral EEG voltage output (i.e., low-voltage activity independent of desynchronizing mechanisms) is a grave danger sign of a preterminal state unless it occurs for a few seconds only, as in certain syncopal attacks. It may also indicate diffuse chronic cortical degeneration (as described by Rosas et al., 2003).

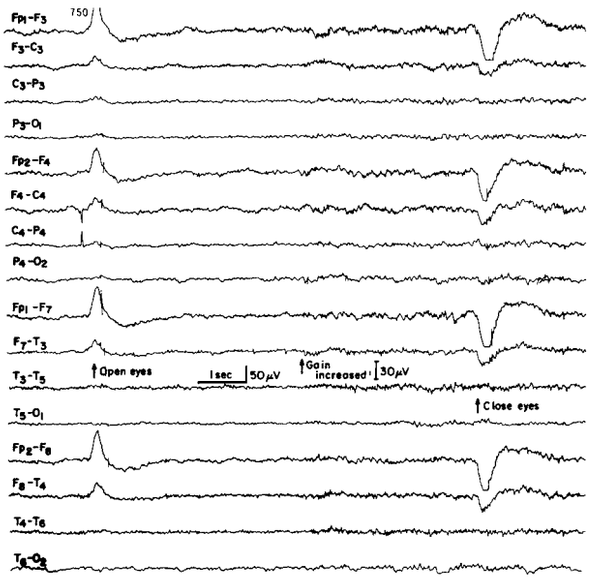

Figure 9.6. Low-voltage record with poor alpha organization, in a patient age 31 years. Note that recording was done with above-standard gain. Finer details demonstrable with further gain increase.

Definition and Categories of Low-Voltage Records

The term *low-voltage EEG* has always proved to be quite flexible; it is in need of a strict definition. It was pointed out earlier in this chapter that precise figures concerning amplitudes are not truly accurate, because they depend on interelectrode distances and the montage; even an ear electrode may be slightly contaminated with EEG activity. The definition of Chatrian et al. (IFSECN, 1974) reads as follows:

> Low voltage record. A waking record characterized by activity of amplitudes not greater than 20 μV over all head regions. With appropriate instrumental sensitivities this activity can be shown to be composed primarily of beta, theta and, to a lesser degree, delta waves, with or without alpha activity, over the posterior areas.

This definition serves as a solid guideline. One could subdivide low-voltage records into two or three degrees; occasionally, one may see tracings in which 10 μV are not exceeded. Even such "very low voltage records" may be due to desynchronization and thus occur in a waking and alert patient or even in a control subject (Fig. 9.6). Low-voltage records are categorically within broad normal limits of variability and do not represent an abnormality unless the frequency spectrum shows abnormal local or diffuse slowing, asymmetries, or paroxysmal events. In a comatose patient, one must infer that the low voltage is due to true decline of cerebral activity and not merely caused by desynchronization. Such records must be regarded as extremely abnormal. This indicates that the record reader should depend on behavior observation (patient comatose, on respirator, etc.). On the other hand, the EEG as such reveals signs of telltale character for the experienced reader, such as presence or absence of eye blinks, types of artifacts, etc., even when he or she must read without any behavioral information.

The IFSECN (1974) definition does not elaborate on sleep and response to hyperventilation. It also omits the question of low-voltage alpha versus low-voltage fast records. One could add further categories of low-voltage records in the following manner:

1. Low-voltage fast record awake but voltage increase in non-REM sleep
   (a) with alpha improvement and voltage increase during hyperventilation (provided that the patient is cooperative in the test),
   (b) without alpha improvement and voltage increase during a well-performed hyperventilation;
2. Low-voltage fast record awake with persisting low voltage in non-REM sleep;
3. Low-voltage record awake with good alpha organization and voltage increase in sleep
   (a) with voltage increase during hyperventilation,
   (b) without voltage increase during hyperventilation;
4. Low-voltage record awake with good alpha organization and persisting low voltage in sleep.

The distinction between low voltage fast records and those with good alpha rhythm was suggested by Gastaut et al. (1957).

Prevalence and Electroclinical Correlations

Early studies of low-voltage tracings were carried out by Davis and Davis (1936), Jasper et al. (1939), and Finley (1944). Jasper et al. (1939) used the term *flat EEG*, which is not reserved for truly isoelectric records.

Gibbs et al. (1943) (also see Gibbs and Gibbs, 1950, 1964) found low-voltage fast records in 11.6% of 1,000 normal adult subjects. The study of Adams (1959) revealed a prevalence of 8% (in 427 normal individuals); the breakdown into age ranges is most important, because low-volt-

age tracings were found in 1% between ages 0 and 20 years, 7% between 20 and 39 years, and 11% between 40 and 69 years. Pine and Pine (1953) reported 7.25% low voltage records in 2,000 neurological and psychiatric patients between 17 and 70 years. Vogel and Götze (1959) found low-voltage tracings in 7.09% of 1,540 patients. In 7,000 patients including a large pediatric segment, only 3.6% were found to have a low-voltage tracing (Niedermeyer, 1963).

According to Gibbs and Gibbs (1950), the prevalence of low-voltage tracings increases sharply after age 13. This is congruent with the observations of Panzani and Turner (1952), Adams (1959), and Lucioni and Penati (1966). Low-voltage fast records in children below age 10 are suspect and clearly abnormal if neither hyperventilation nor non-REM sleep changes the low-voltage character of the tracing. In adults, persistence of low-voltage activity with hyperventilation and sleep is not categorically abnormal, although this lends more support to the presumption that such patients have an organic cerebral disorder. Chatrian (1976c) stressed the high incidence of low-voltage tracings in postconcussion syndrome and posttraumatic neurosis.

Posttraumatic records have been studied by Duensing (1948), Meyer-Mickeleit (1953), Vogel et al. (1961), -Courjon (1962), Mifka and Scherzer (1962), Arfel et al. (1963), Lorenzoni (1963), and Scherzer (1966). Low-voltage records show a high incidence in posttraumatic patients, and "psychogenic alpha suppression" often accounts for the small amplitudes (Scherzer, 1966). Hyperventilation quickly leads to alpha development and voltage increase. Not all posttraumatic low-voltage records should be attributed to such psychological mechanisms of "posttraumatic neurosis" or "litigation neurosis"; there seems to be a niche for an organic posttraumatic syndrome with low-voltage EEG tracings (Chatrian, 1976c). Radermecker (1961) and Kugler (1964) have minimized the value of EEG as a method of "objectivation" of the patient's posttraumatic complaints.

Basal ganglia diseases show a somewhat higher incidence of low-voltage records. In the case of Huntington's chorea, however, progressive voltage decline could perhaps indicate "true" loss of cortical activity rather than desynchronization; a view supported by the work of Rosas et al. (2003). There has been some suggestion that endocrine and psychiatric disorders are associated with a greater incidence of low-voltage tracing (Adams, 1959).

There is some evidence that genetic factors play an important role in determining a low-voltage tracing in a healthy person (Vogel and Götze, 1959). This genetic predisposition, however, obviously does not manifest itself in infancy or childhood, a time when almost all low-voltage records bear the mark of abnormality. With adolescence (age 13, according to Gibbs and Gibbs [1950]), an unknown maturational change apparently starts to desynchronize the cortical EEG activity. Adams (1968) has contended that, in the presence of CNS pathology, the low-voltage fast character tends to change and the record shows local or diffuse slowing. The evolution of an epileptic seizure disorder is also likely to interfere with the previous low-voltage character of a tracing. The statement that the epileptic seizure disorders are very seldom associated with a low-voltage EEG (Adams, 1959) is certainly correct. However, a low-voltage record does not rule out an epileptic seizure disorder, even when one leaves aside the group of chronic alcoholics with withdrawal seizures and predominantly low-voltage tracings. This view has been supported by the work of Synek (1983).

According to this author's experience, two major groups of adult neurological patients are characterized by the frequent occurrence of low-voltage tracings: (a) patients with chronic vertebrobasilar artery insufficiency (even though this view has been criticized by Chatrian [1976c]), and (b) patients with chronic alcoholism (Krauss and Niedermeyer, 1991). Further discussion is found in Chapter 17, "Cerebrovascular Disorders and EEG," and Chapter 34, "EEG, Drug Effects, and CNS Poisoning."

Theta Rhythms

The term *theta* was introduced by Walter and Dovey (1944) and denotes the frequency range of 4 to 7/sec or 4 to 7.5/sec. This frequency band was a part of the delta range until Walter and Dovey felt that an intermediate band should be established. The term *theta* was chosen in order to allude to its presumed thalamic origin. The intermediate character of the theta band has been stressed in the German terminology *Zwischenwellen*, meaning intermediate waves. According to the international nomenclature, the theta band is the "frequency band from 4 to under 8 Hz" and the theta rhythm is the "rhythm with a frequency of 4 to under 8 Hz" (IFSECN, 1974).

Theta Activity in the Waking Adult

The normal adult waking record contains but a small amount of theta frequencies and no organized theta rhythm. Theta frequencies and theta rhythms, however, play an important role in infancy and childhood, as well as in states of drowsiness and sleep. The most important aspect is the maturational one; the reader will find more pertinent information in Chapter 11, "Maturation of the EEG: Development of Waking and Sleep Patterns." Larger contingents of theta activity in the waking adult are abnormal and are caused by various forms of pathology.

Studies of young adults such as army personnel and navy pilots have shown a sizable amount of theta activity (Gallais et al., 1957; Picard et al., 1957) because these slow frequencies tend to linger on through the third decade of life; the completely mature aspect of the human EEG cannot be expected before the age of 25 to 30 years.

On the basis of power spectra from normal adults, Rugg and Dickens (1982) presume that alpha and theta activity is generated by separate mechanisms.

Slow (theta and delta) EEG activity has been correlated with cholinergic activities and central cholinergic pathways (Steriade et al., 1990). The observation of slowing induced by the action of cholinergic substances on the ascending brainstem reticular formation dates back to Rinaldi and Himwich (1955). Further information is found in the overview of Riekkinen et al. (1991).

Hedonic Theta Rhythms

W. Grey Walter (1959) has associated theta activity with emotional processes, and thought that this activity might be

a sign of "relative maturity of the mechanisms linking the cortex, the thalamus and the hypothalamus" (also see Knott, 1976b). He also attributed runs of theta waves to the emotional correlates of disappointment and frustration because of its appearance at the conclusion or interruption of a pleasurable stimulus. Similar views were expressed by Garsche (1956) and Lairy (1956). Maulsby (1971) presented a very impressive combined EEG and photographic demonstration of a 9-month-old girl and her EEG response to pleasurable stimuli; the most effective stimulus was being kissed by her mother. The response consisted of very pronounced rhythmical 4/sec activity of posterior accentuation, strongly spreading into central areas. This rhythm was different from the rhythmical 5 to 6/sec activity seen in this child's drowsy state. The 4/sec activity appeared to be pleasure-related rather than caused by the termination of a pleasurable stimulus. Similar rhythmical 4/sec activities were described by Kugler and Laub (1971) in children between the ages of 6 months and 6 years, elicited by watching puppets, moving objects, toys, and picture books ("puppet show theta rhythm"). Such hedonic EEG responses have not been observed in the adult EEG. According to Futagi et al. (1998), infantile theta rhythm associated with sucking, crying, gazing, and handling (including the hedonic type) is due to cortical activation driven by the limbic system.

From the Hedonic Theta Rhythm to EEG Findings During Sexual Activities

It may be interesting to note in this context that Mosovich and Tallaferro (1954) obtained EEG tracings from volunteers during coitus and masturbation. Further studies of human orgasm were carried out by Sem-Jacobsen (1968) and Heath (1972) with the use of depth electrodes in chronic psychiatric patients. Pre- and intraorgasmic paroxysmal discharges were reported by these authors (septal spiking; Heath, 1972), but the tracings of Heath show no special thalamic participation and no theta rhythm in the depth. A thorough EEG and polygraphic study of ejaculation and orgasm was carried out in young healthy males (Graber et al., 1985). The results turned out to be quite meager. "We have failed to demonstrate any significant and specific EEG changes during masturbation, ejaculation and the subjective experience of orgasm." This is probably a barren field for EEG research.

Other Physiological or Marginal Theta Rhythms

The 6 to 7/sec rhythm over the frontal midline is discussed in Chapter 11, "Maturation of the EEG: Development of Waking and Sleep Patterns." This rhythm usually disappears in adolescence. The theta activities described in children with primary generalized epilepsy and in their healthy siblings ("rhythmical monomorphic" 4 to 7/sec activity of parietal accentuation in children from 2 to 7 years) are discussed in Chapter 27, "Epileptic Seizure Disorders" and Chapter 11, "Maturation of the EEG: Development of Waking and Sleep Patterns."

With the use of a stress paradigm, Nowak and Marczynski (1981) produced in six of 24 healthy volunteers a rhythmical 5 to 6/sec theta response originating from either the occipital region or vertex during a state of maximal alertness.

A rhythmical 4/sec pattern occurring over the vertex solely in the waking state has been described as "4/sec vertex spindles" by Van Huffelen and Magnus (1973). This very rare pattern is likely to represent a mild abnormality. It is found mainly in adolescents with syncopal attacks and other signs of vasomotor instability. The use of the term *spindles* appears to be somewhat out of place when one considers the slow frequency and the occurrence in the waking state. Hence, Daoust-Roy (1989) proposed the term *4/sec vertex rhythm.*

Frontal Midline 6–7/Sec Theta Rhythm and Thinking

Rhythmical theta activity in the 6 to 7/sec range over the frontal midline region has been correlated with mental activities such as problem solving (Arellano and Schwab, 1950; Brazier and Casby, 1952; Ishihara and Yoshii, 1972; Mizuki, 1982, 1987; Mizuki et al., 1980, 1983). In personal studies of mental EEG activation, we were unable to produce this pattern (Niedermeyer et al., 1989); this indicates the technical difficulties in the demonstration of the task-related frontal theta rhythm.

Our inability to demonstrate frontal midline 6 to 7/sec activity as a response to mental tasks has been a nagging problem for the writer of this chapter. I asked Japanese and Chinese co-workers to solve mental tasks during EEG recordings (in hopes of finding a certain racial or cultural element specific to individuals from the Far East) with completely negative results. This vexing problem has been clarified by the work of Takahashi et al. (1997), who found that individuals with rhythmical frontal midline 6 to 7/sec activity in light drowsiness also had the same type of activity during mental tasks: "These two frontal theta rhythms closely resembled each other in frequency (94.6%) and distribution (83.8%)" (Takahashi et al., 1997). One wonders if these tasks become boring enough to produce light drowsiness rather than increased alertness (one is even tempted to utilize the old Pavlovian term *internal inhibition*, i.e., sleep induction in a certain repetitious experimental setting).

Macrosmatic mammals (rats, etc.) show a very powerful limbic and especially hippocampic rhythm from 3 to 12/sec (mainly 4 to 7/sec), which is further activated by arousal (Green and Arduini, 1954). An overview of limbic rhythmic theta activity has been presented by Lopes da Silva (1992).

Posterior Slow Activity

Posterior slow activities constitute a very important aspect of the EEG maturation in childhood, adolescence, and early adulthood (the third decade of life). All of these slow posterior activities in the theta and delta range are discussed in Chapter 11, "Maturation of the EEG: Development of Waking and Sleep Patterns." The mature adult waking record contains only traces of posterior slowing.

Lambda Waves

Definition and Historical Aspects

Lambda waves are "sharp transients occurring over the occipital region of the head of waking subjects during visual

exploration. Mainly positive relative to other areas. Time-locked to saccadic eye movements. Amplitude varies, but is generally below 50 μV" (IFSECN, 1974).

This visually induced occipital activity was described by Y. Gastaut (1951); there was an earlier oral presentation by Evans (EEG Society in London, 1949). Subsequent EEG studies on lambda waves were presented by Cobb and Pampiglione (1952), Evans (1952, 1953), and Roth and Green (1953). A very extensive review of this subject is found in the handbook article of Chatrian (1976d).

Prevalence and Further Characteristics

Lambda waves are unmistakably present in some records and are not readily demonstrable in others. They are most prominent in waking patients intently viewing an illuminated visual field (Chatrian, 1976d). The prevalence essentially depends on the thoroughness of the EEG evaluation and the emphasis placed on the demonstration of this special phenomenon. According to Tsai and Liu (1965; quoted in Chatrian, 1976d), lambda waves are most frequent between ages 3 and 12 years (82.3%); their prevalence declines to 72% between 18 and 30 years and to 36.4% between ages 31 and 50 years.

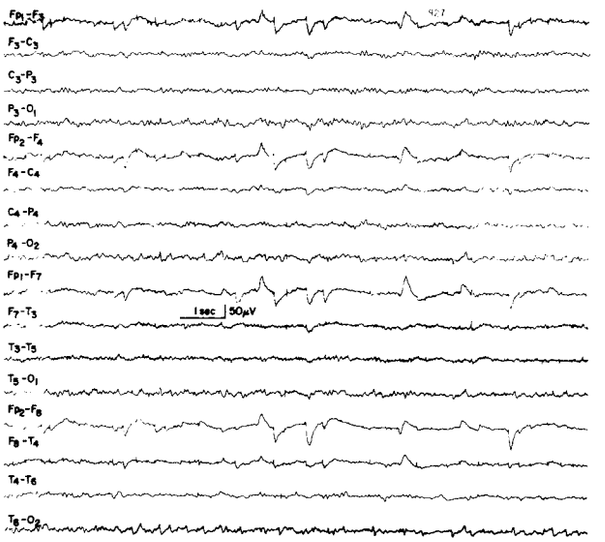

Figure 9.7. Occipital lambda waves while viewing pictures, in a patient age 15 years. Note ocular artifacts caused by scanning eye movements.

Wave Morphology

Lambda waves have been described as biphasic or triphasic; the most prominent phase is positive. Their form has been described as triangular or saw-toothed shaped. The amplitude is usually below 20 μV and may exceed 50 μV in some persons. The overall duration of lambda waves lies between 200 and 300 msec. These waves repeat themselves, usually at intervals from 200 to 500 msec. Marton et al. (1982) have demonstrated the complexities of lambda waves; their predominant positive component is preceded and followed by a negative component.

Spatial Distribution

Lambda waves are most prominent in (or confined to) the occipital leads. Spread into parietal and posterotemporal areas is common and, in certain cases, the maximum may be found in areas adjacent to the occipital lobe (Evans, 1953; Roth and Green, 1953). Lambda activity is strictly bilateral synchronous. With the use of depth electrodes, Perez-Borja et al. (1962) demonstrated multiple foci of lambda waves, either in or near the calcarine region or more laterally in the occipital lobes.

Precipitating Factors

Voluntary scanning eye movements (exploratory saccades) play a very important role. It was found that most lambda waves follow an exploratory eye movement with a latency of 67 to 85 msec (mean, 78 msec) (Green, 1957). The preceding eye movement has been subsequently used as a trigger for computer techniques (Barlow, 1963, 1964; Barlow and Ciganek, 1969; Chatrian, 1964; Remond and Lesèvre, 1971; Remond et al., 1965; Scott and Bickford, 1967). Further interesting details on this subject are found in the handbook article of Chatrian (1976d) (Figs. 9.7 and 9.8).

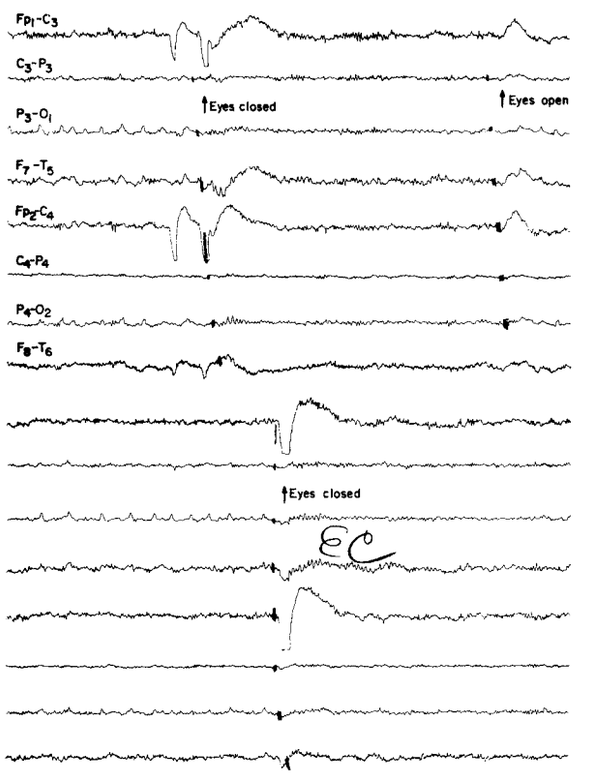

Figure 9.8. Prominent occipital lambda waves during eye opening. Patient, age 54 years, looking ahead in the dimly lit laboratory. There is no evidence of scanning ocular artifacts. Note instability and fast character of alpha rhythm (1–13/second and even faster). The *bottom part* shows continuation of the *upper part*.

Lambda waves are best found in brightly lit laboratories and cannot be elicited in darkness. The size of a given pattern and its distance and color are further variables. Binocular viewing of a picture may or may not produce larger lambda waves than monocular viewing (Bickford and Klass, 1964; Scott et al., 1967).

The role of psychological factors has also been pondered. Green (1954) felt that nervous tension would diminish lambda activity. Presentation of a new picture enhances lambda production (Chatrian, 1976d; Green, 1954). Lambda activity does not seem to be related to recognition of objects (Green, 1957). Hypnotic suggestion of a test picture failed to produce a lambda response (Scott et al., 1967).

Relationships to Alpha Rhythm and Visual Evoked Responses

There appears to be considerable independence between posterior alpha rhythm (with eyes closed) and lambda waves (with eyes open); no interaction or complementary action of these two activities has been found. Marked similarities between lambda waves and visual evoked responses have been reported by Remond et al. (1965) and Lesèvre (1967). Relationships between lambda waves and occipital photic driving were originally presumed by Y. Gastaut (1951) and Evans (1953) but could not be confirmed by Chatrian et al. (1960), Perez-Borja et al. (1962), and Scott et al. (1967).

Correlations with Neurological and Psychiatric Diseases

No such correlation has been demonstrated. There are also no correlations with certain personality traits.

Theories on the Basic Neurophysiological Mechanism

Lambda waves have been thought to be related to (a) visual evoked responses, (b) oculomotor-visual integration, (c) oculomotor potentials, or (d) arousal mechanisms. These theories have been thoroughly discussed by Chatrian (1976d).

Billings (1989) has proposed that there are two different occipital lambda waves in the human. The first one is the result of the release of peripheral visual inhibition during the braking phase of the primary saccade. This wave is transmitted via the faster conducting Y-type fibers of the optic nerve (subserving peripheral vision). The second wave is elicited by the return of normal central vision, during or slightly before the braking phase of the secondary corrective saccade. This wave is transmitted via the slower conducting X-type fibers of the optic nerve (subserving central vision). According to Billings, "lambda waves, long considered to be of rather trivial import by clinical electroencephalographers, are of considerable functional significance in the visual system."

Relationship to "Lambdoid" Activity or Positive Occipital Sharp Transients of Sleep (POSTS)

These occipital positive sharp discharges are discussed in the section on sleep. A relationship to lambda waves of the waking state appears to be quite obvious, but Chatrian (1976d) feels that the similarities between the patterns are only superficial.

Lambda Wave-Like Occipital Slow Discharges during Eye Blinks in Children

Large slow transients of a duration of 200 to 400 msec over the occipital region bisynchronously occurring after an eye blink with a latency of 100 to 200 msec have been described by Westmoreland and Sharbrough (1975). These waves were found mainly from ages 1 to 3 years. It has been thought that these discharges represent a variant of lambda waves. Pattern and picture scanning also produced these potentials.

EEG Rhythms: Are They Spontaneous or Induced?

The "new wave of EEG research" (initiated by Başar and Bullock) has emphasized the induced nature of EEG rhythms. The old adage of spontaneous EEG activity of either rhythmical or nonrhythmical character has been strongly challenged by the "new wave." This new line of research also has adopted a view in which there is hardly any gap left between EEG rhythms and evoked potentials.

In the old days of evoked potential work, it was shown that flash-evoked visual potentials are followed by a series of alphoid rhythmical waves (also called "ringing"). Thus, relationships between EEG rhythms and evoked potentials are not altogether new, though almost forgotten. Hence their revival in the 1990s must be warmly welcomed.

Imagine a jam-packed stadium with spectators watching an important ball game. There is noise—a gentle hum or single shouts or even tremendous outbursts of almost synchronously emitted vocal energies. Certainly, the most powerful activities are induced by the events on the field (analogy to "event-related potentials") but the constant hum cannot be attributed to any special outside event and may be called spontaneous. When, in less exciting phases of the game, the spectators' attention dwindles, then people start to talk to each other thus generating the hum that had prevailed when the crowd gathered prior to the game. It is reasonable to assume that, in periods of little action, cerebral neurons simply "talk to each other." Under the influence of synchronizing structures, this "hum" or "neuronal conversation" then turns into a spontaneous rhythm.

Concluding Remarks: Background Versus Foreground EEG Interpretation

This chapter presented the frequencies and patterns of the normal adult waking record. The reader may have searched in vain for a discussion of the background activity of the EEG. But what is background and what is foreground? This question touches problems of EEG interpretation that cannot be adequately taught in a textbook. The eye must be trained for the recognition of patterns that may be normal, abnormal-nonparoxysmal, or abnormal-paroxysmal. Above all, however, the interpreter must understand the gestalt of the record; this depends on a formidable number of variables. In this process of interpretation of EEG phenomena, the so-called background may have to be moved into the foreground and vice versa, depending on the momentary focus of emphasis.

This shows clearly where the limitations of an EEG textbook lie. There is simply no firm rule concerning the manner

in which the record reader's eyes and brain have to operate in this process. There is simply no substitute for an adequate training period with regular joint reading sessions.

Every experienced electroencephalographer has his or her personal approach to EEG interpretation. This is also true for the manner in which the EEG report is written. Panel discussions on the EEG report and the proper way of writing reports consistently reveal striking differences among the panelists. Although standardization is an important goal in many areas of EEG technology, experienced electroencephalographers should not abandon a certain individualistic spirit, notwithstanding the fact that the eminent features of each tracing should be easily gleaned from the report. EEG reports should be sound and lucid, but the format does not have to be standardized. Why not? Because there is an element of science and an element of art in a good EEG interpretation; it is the latter that defies standardization.

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