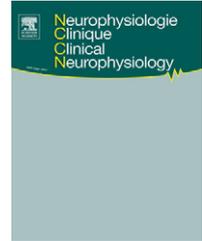




Disponible en ligne sur
SciVerse ScienceDirect
www.sciencedirect.com

Elsevier Masson France
EM|consulte
www.em-consulte.com/en



REVIEW/MISE AU POINT

Normal EEG in childhood: From neonates to adolescents

L'EEG normal chez l'enfant : du nouveau-né à l'adolescent

M. Eisermann^{a,b,*}, A. Kaminska^{a,b}, M.-L. Moutard^c, C. Soufflet^{a,b}, P. Plouin^{a,b}

^a Service des explorations fonctionnelles, neurophysiologie clinique, hôpital Necker–Enfants-Malades, 149, rue de Sèvres, 75743 Paris cedex 15, France

^b Inserm, U663, université Paris Descartes, Paris, France

^c Service de neuropédiatrie, hôpital Trousseau, 26, avenue du Docteur-Arnold-Netter, 75012 Paris, France

Received 11 January 2012; accepted 30 September 2012

Available online 30 October 2012

KEYWORDS

EEG;
Electroencephalogram;
Maturation;
Normal variants;
Neonatology

MOTS CLÉS

EEG ;
Électroencéphalo-
gramme ;
Maturation ;
Variantes normales ;
Néonatalogie

Summary The important EEG changes that occur throughout childhood are a major challenge for the neurophysiologist. These reflect brain maturation, which is especially fast during the first year of life. This article describes normal EEG features and variants, characteristic patterns of development, as well as some patterns that are unusual for age, from the neonatal period to adolescence. We also describe how to adapt techniques and prepare patients in order to get interpretable records of appropriate duration, in neonates, infants, and young children.
© 2012 Elsevier Masson SAS. All rights reserved.

Résumé L'organisation spatio-temporelle et les grapho-éléments physiologiques de l'EEG évoluent parallèlement à la maturation cérébrale, particulièrement rapide dans la première année de vie. Cet article décrit l'EEG normal avec ses variantes et aspects maturatifs caractéristiques en fonction de l'âge et de l'EEG, qui doivent être connus pour interpréter l'EEG de l'enfant. Nous décrivons également les techniques d'enregistrement adaptées au nouveau-né, au nourrisson et à l'enfant, indispensables pour obtenir des tracés de bonne qualité.
© 2012 Elsevier Masson SAS. Tous droits réservés.

Introduction: technical aspects

Because these are two major determinants of EEG features in childhood, both age and level of vigilance should be taken

into consideration (in addition to clinical information) for reliable EEG interpretation.

The evaluation of premature, neonatal, and infantile EEG is based on the conceptional age, which is defined as the sum of gestational age (number of complete weeks and days from the first day of the last menstrual period) at birth and chronological age (number of weeks post-partum).

In newborns and young children, the EEG provides more information when it is also recorded during sleep, for two

* Corresponding author.

E-mail address: monika.eisermann@nck.aphp.fr (M. Eisermann).

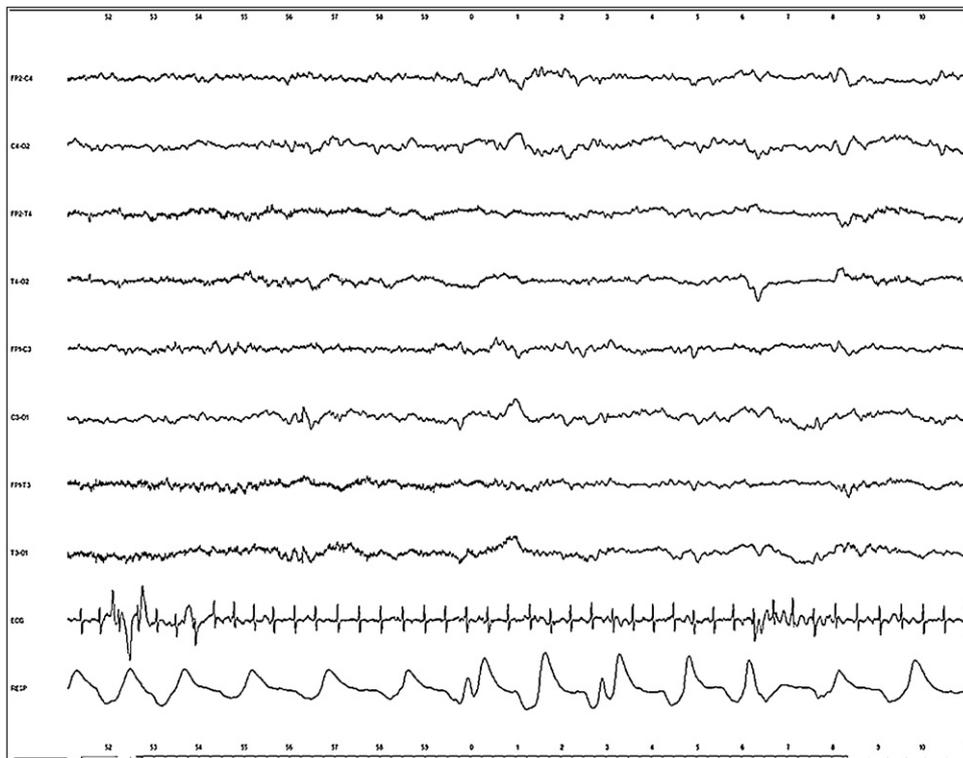


Figure 1 Full-term newborn. Quiet wakefulness. Continuous irregular theta activity with some occipital delta waves.

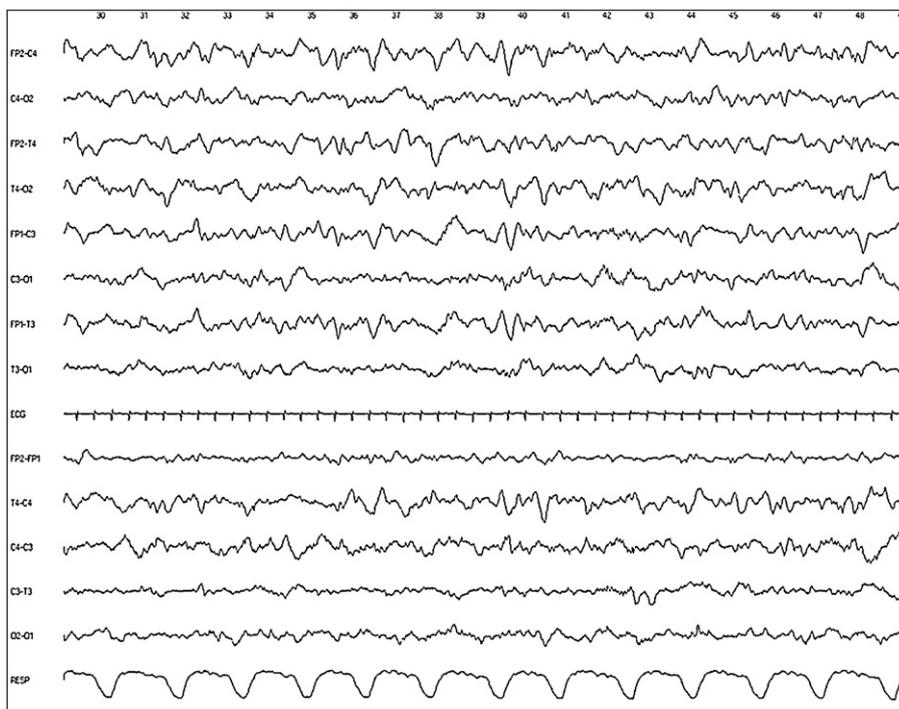


Figure 2 Full-term newborn. Quiet sleep. Slow continuous activity.

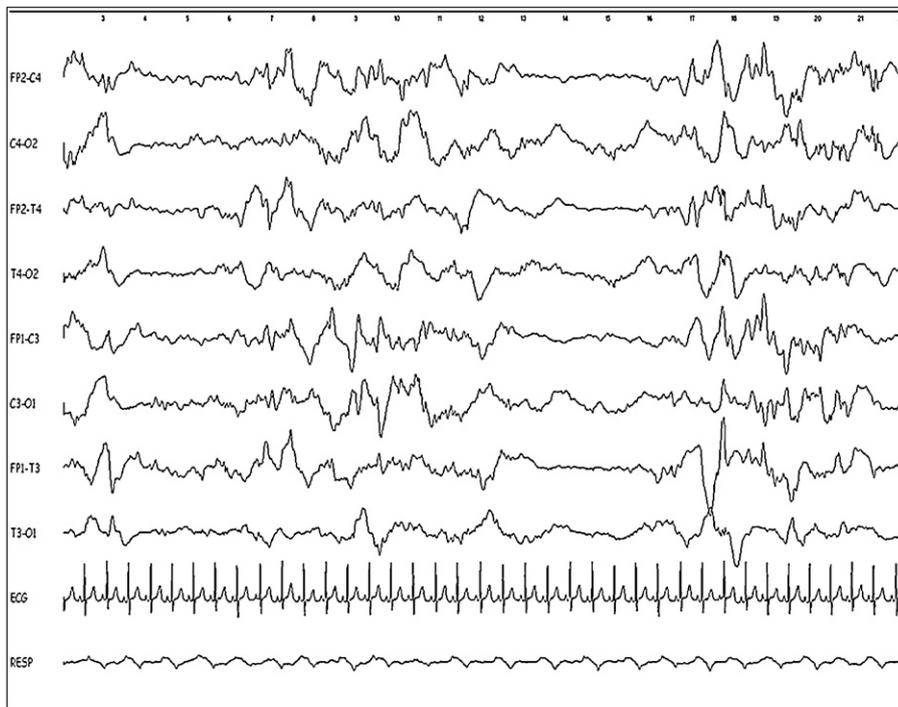


Figure 3 Full-term newborn. Quiet sleep. *Tracé alternant*.

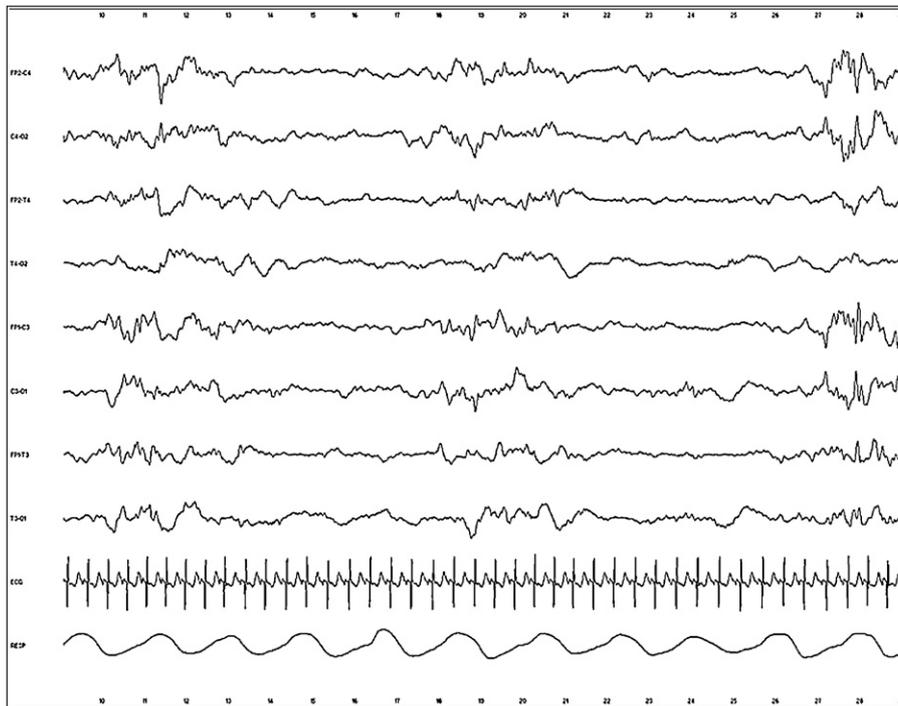


Figure 4 Full-term newborn. Quiet sleep. *Tracé alternant*.

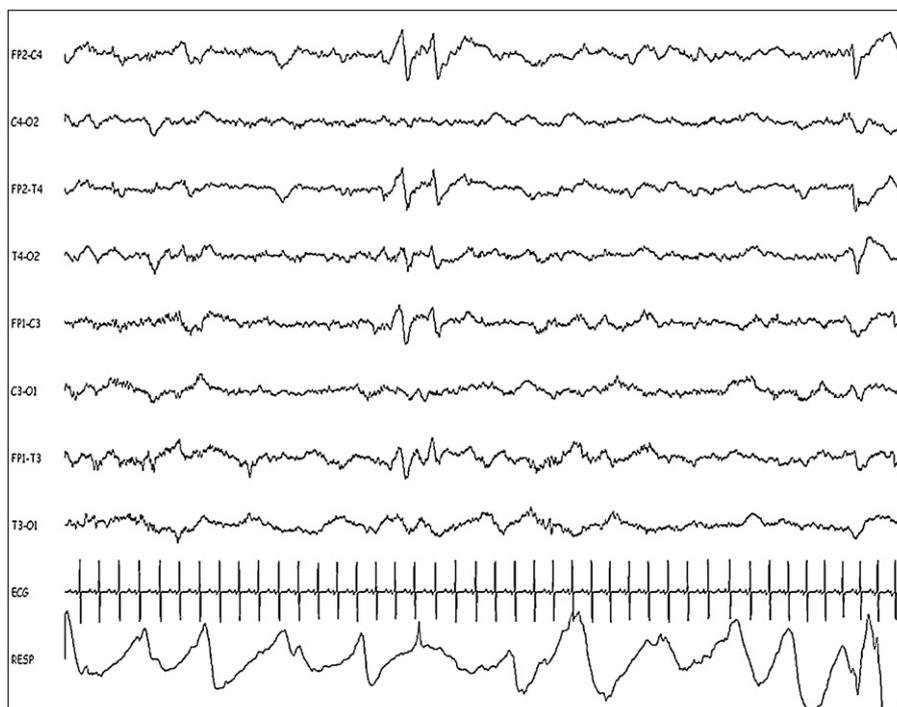


Figure 5 Full-term newborn. Active sleep. Frontal sharp transients.

reasons: firstly, EEG interpretation may be hindered in a crying and excessively moving patient, as it is usually the case when the baby is awake; and, secondly, maturational aspects of sleep can provide additional information. Optimal sleeping conditions are most easily reached when

the recording is performed shortly after feeding or at the usual time of sleeping, possibly after partial sleep deprivation. Oral melatonin can be useful to obtain sleep [6]. These prerequisites also imply that the moment of recording should be planned by the neurophysiological

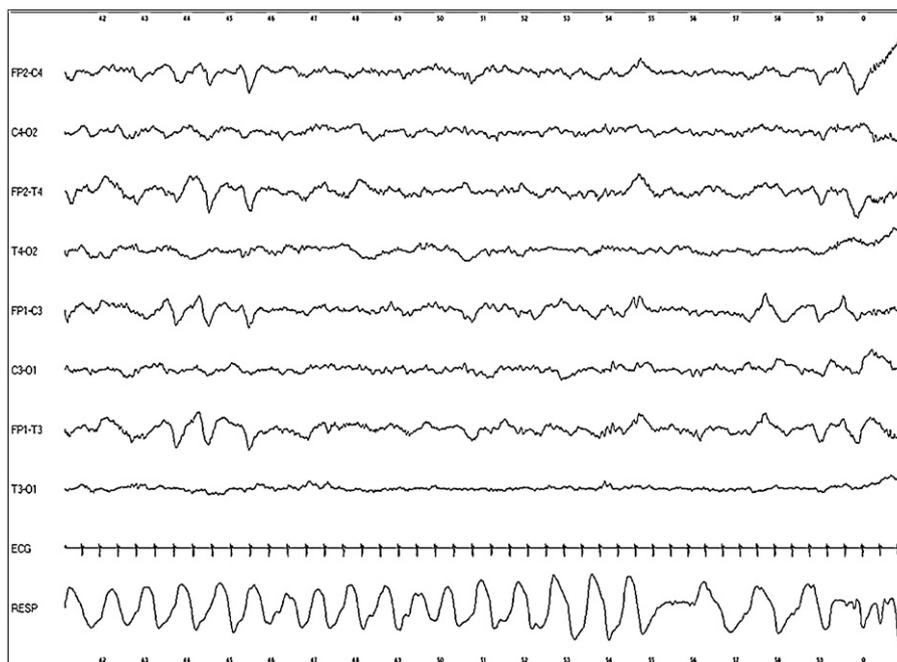


Figure 6 Full-term newborn. Active sleep. Anterior slow dysrhythmia.

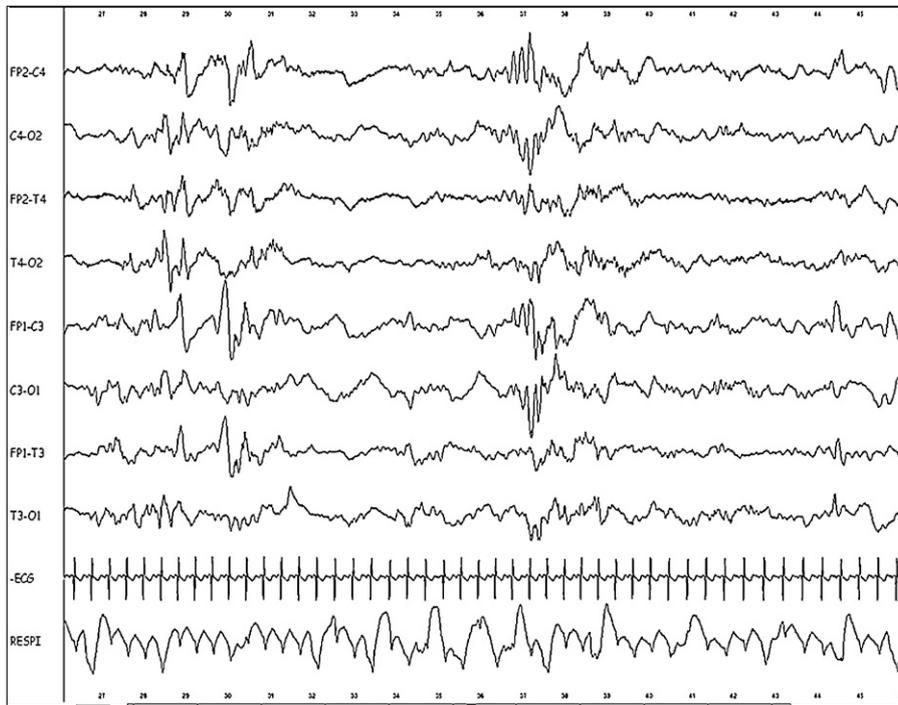


Figure 7 Full-term newborn. Quiet sleep. Bursts of rolandic theta waves.

department in concert with attending nurses and parents.

As the paediatric population encompasses children from a conceptional age of 25 weeks in the incubator until early

adulthood, there is a need to adapt EEG techniques according to these different conditions, i.e., different head size, asepsis, behaviour, etc. The presence of the parents can either be helpful or complicate EEG recording.

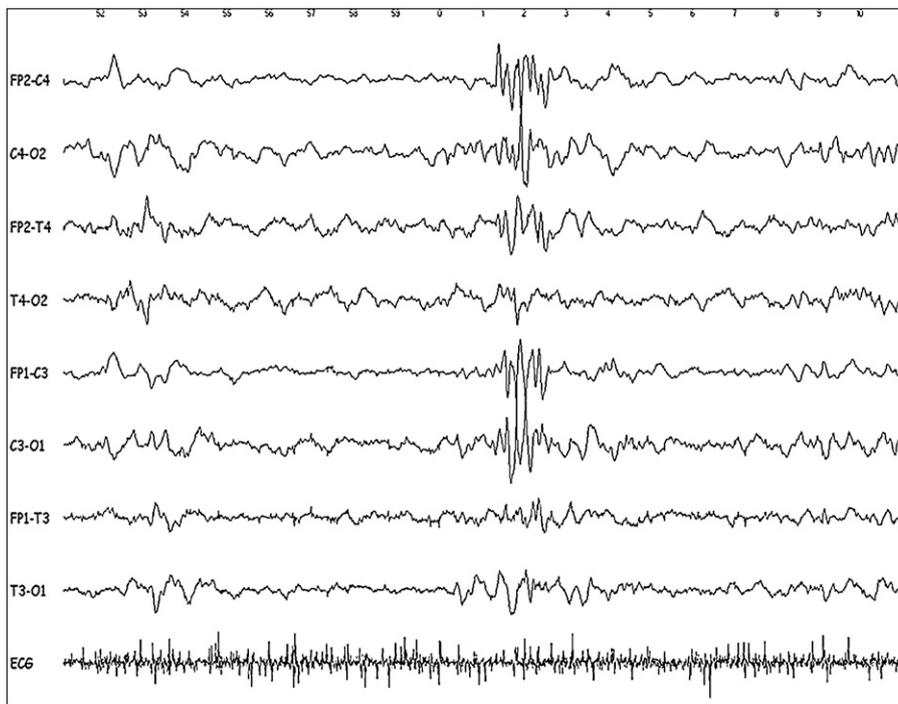


Figure 8 Full-term newborn. Active sleep. Birolandic burst of theta waves.

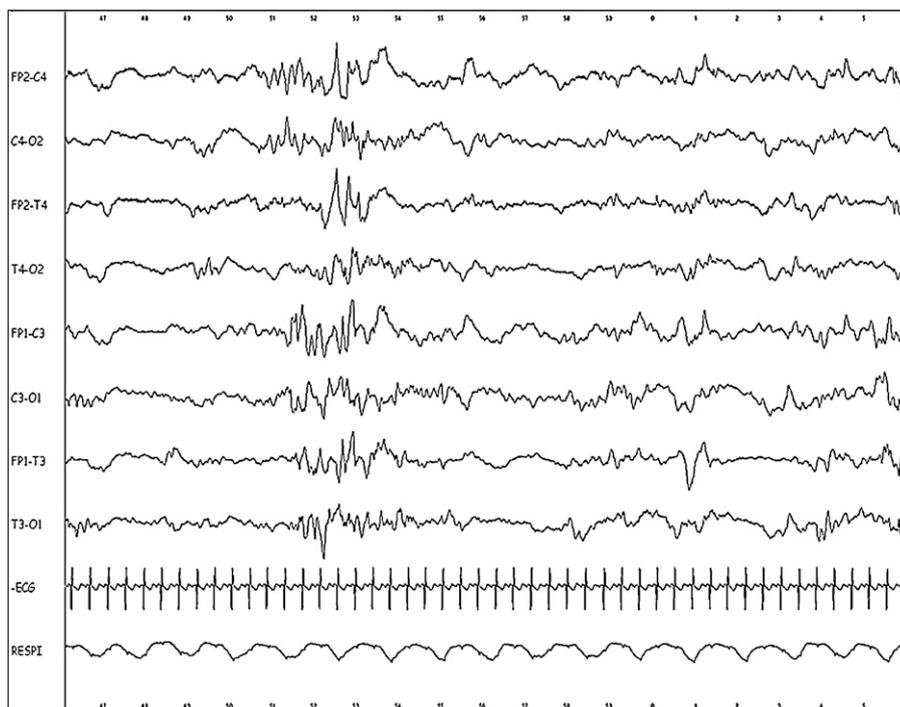


Figure 9 Full-term newborn. Quiet sleep. Bursts of fronto-rolandic theta waves.

The International 10–20 System, which comprises 21 electrodes, can be adapted by using just four electrodes on each hemisphere in the newborn (Fp₁, Fp₂, C₃, C₄, O₁, O₂, T₅, T₆ in addition to a ground electrode), and progressively increasing the number of electrodes with increasing

head size. Adding a C_z electrode in the premature is mandatory for positive rolandic sharp wave detection. ECG and respiration should be systematically recorded. Additional electrooculogram and surface sub-mental EMG recording is mandatory for precise study of the sleep structure.

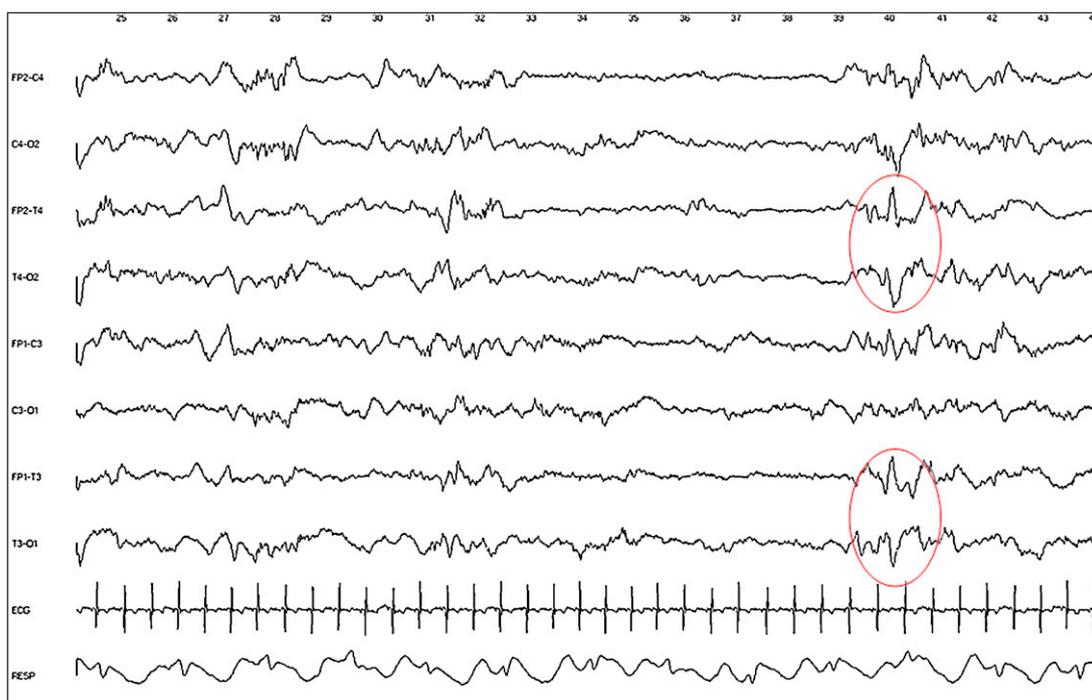


Figure 10 Full-term newborn. Quiet sleep. Positive temporal sharp waves.

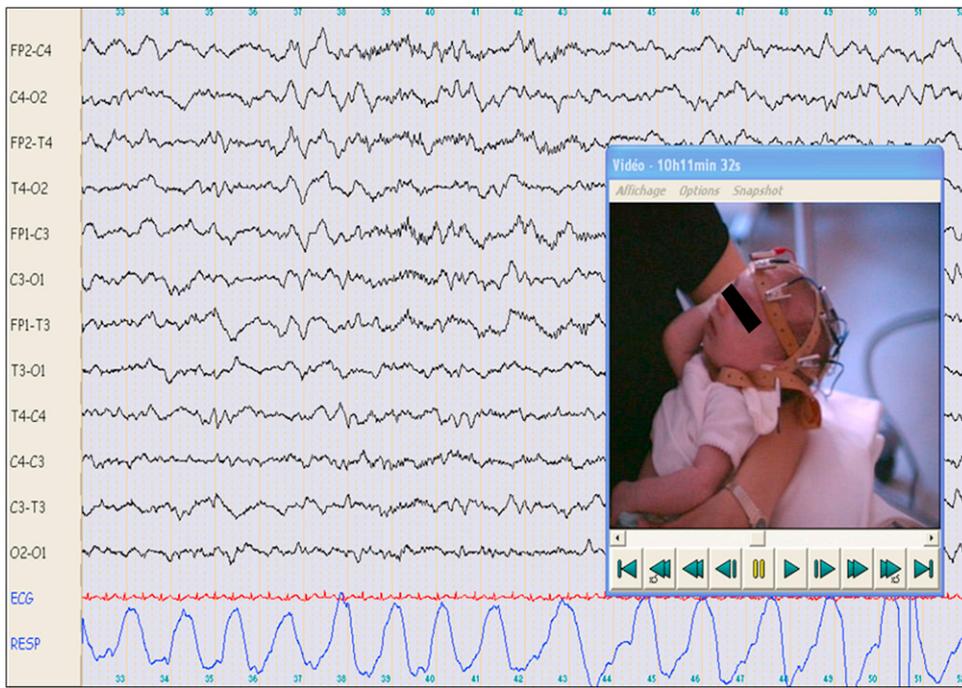


Figure 11 Three-week-old newborn. Transition into quiet sleep.

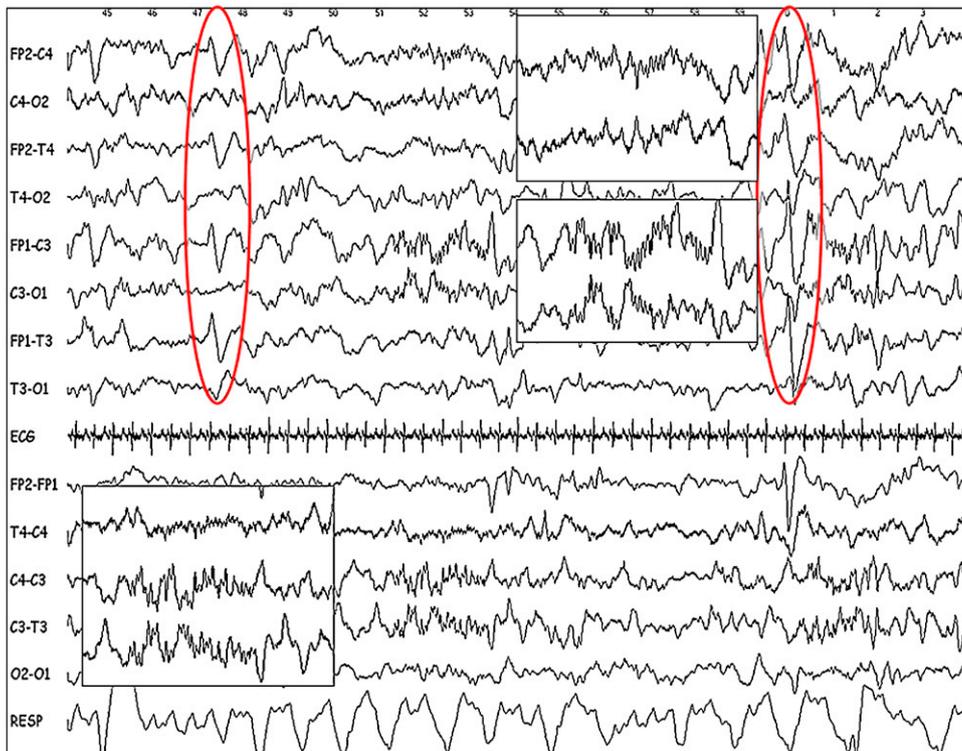


Figure 12 Three-week-old newborn. Frontal sharp transients and spindles.

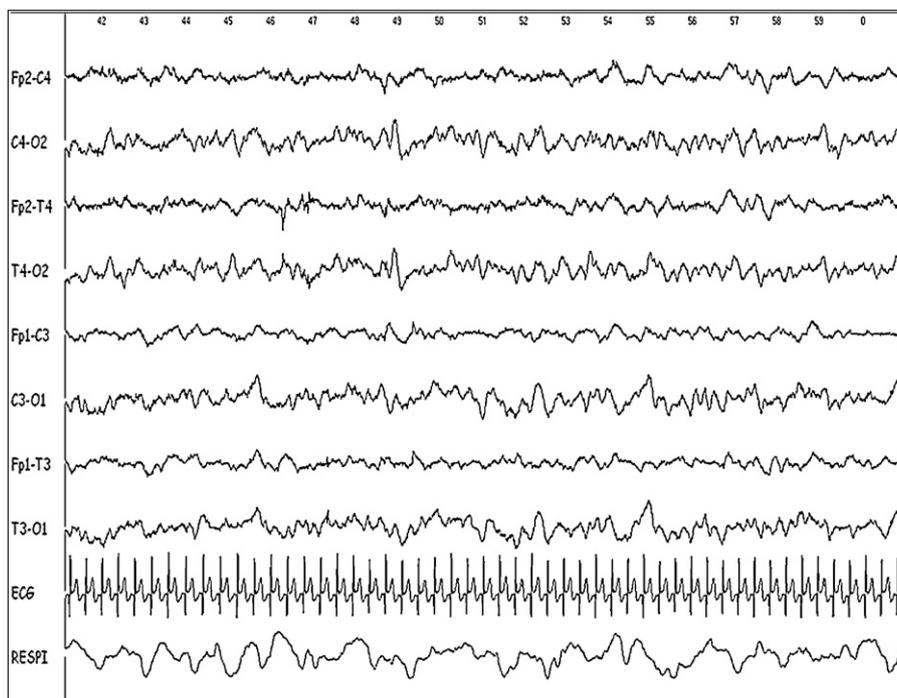


Figure 13 Three-month-old infant. Quiet wakefulness.

Combined recording of upper-airway air exchange, thoracic and abdominal movements, and transcutaneous O_2 saturation are required for the differential diagnosis of sleep apnoeas (central, mixed or obstructive). Surface EMG and synchronous video recording should be added in epileptology, in order to characterize precisely clinical manifestations.

EEG in the neonatal period (normal full-term newborn)

Because of the predictive value of the EEG in the neonate, the limits of normality should be very precisely known in this age group [11].

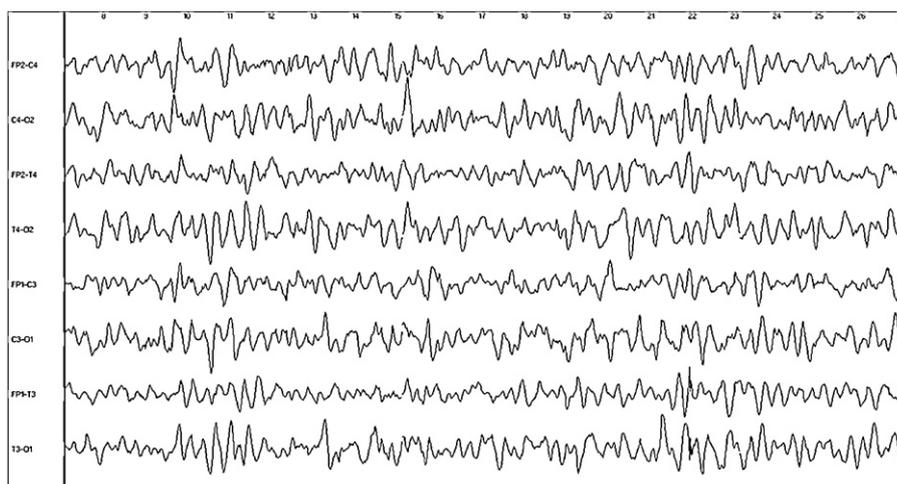


Figure 14 Three-month-old infant. Drowsiness.

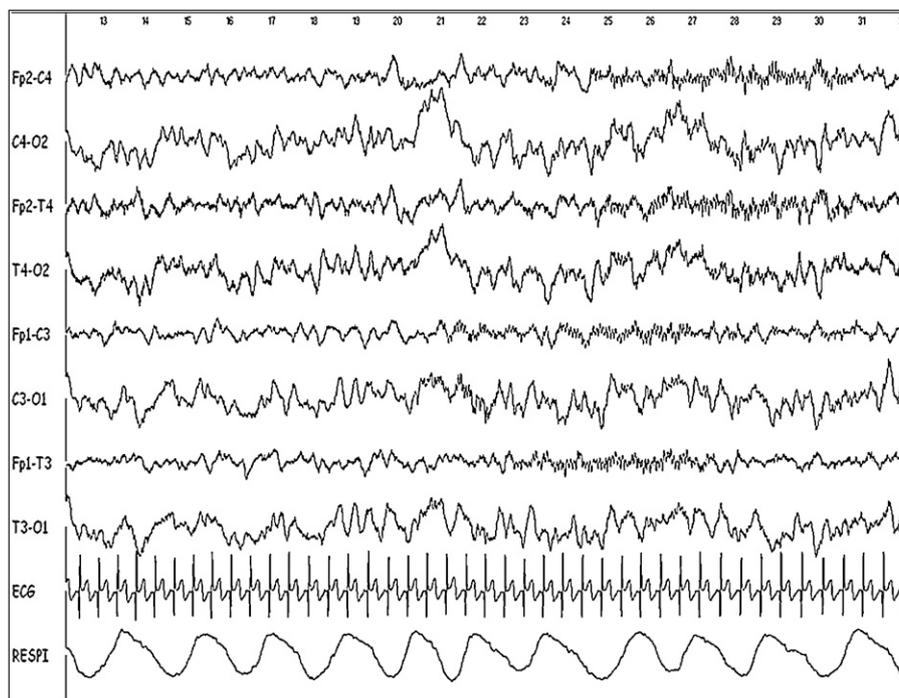


Figure 15 Three-month-old infant. Quiet sleep. Spindles.

Temporal organisation

Three behavioural states are distinguished: wakefulness (active and quiet), active sleep (precursor of the later rapid eye movement [REM] sleep), and quiet sleep (precursor of the later slow sleep).

Active sleep is characterized by the presence of rapid eye movements, axial muscular atonia, phasic movements, and irregular respiration and cardiac rhythm. EEG of active sleep preceding quiet sleep (Type 1) can be different from EEG of active sleep following quiet sleep (Type 2). The newborn falls asleep first in active sleep then in quiet sleep, this structure persisting until the age of 2–3 months. From this age on until adulthood wakefulness/drowsiness is followed by slow sleep. Quiet sleep is characterized by the absence of rapid eye movements, presence of axial tonic muscular activity, and regular respiration and cardiac rhythm. If observation criteria are not congruent, the term “indeterminate” or “transitional” sleep is used.

In the newborn baby, 50% of the sleeping time consists of active sleep in contrast to adults who spend only 20% in REM sleep. The duration of every sleeping state is about 20 minutes, but active sleep may be very long lasting in some neonates. Therefore, the standard recording duration should be at least one hour in order to get a complete cycle [13].

Background activity

The following background activities can be observed:

- “mixed frequencies”, which corresponds to the characteristic background activity of wakefulness and active sleep: continuous, irregular, diffuse activity, with a rolandic predominance, mainly in the theta band (4–7 Hz), sometimes mixed up with some occipital delta activities, with a voltage of 25–50 μV (Fig. 1);
- “slow continuous high-voltage activity”, which is a characteristic background activity in quiet sleep, often preceding “tracé alternant”: Continuous diffuse delta wave activity (1–3 Hz) with central or occipital predominance, of variable voltage (50–150 μV) (Fig. 2) [5];
- “tracé alternant”, which is a characteristic background activity of quiet sleep from 37 to 44 weeks conceptional age: bilateral bursts of delta waves (1–3 Hz) of high amplitude (50–150 μV) superimposing on continuous theta activity (4–7 Hz) of lower amplitude (25–50 μV). Bursts may present with sharper morphology over frontal regions and smoother morphology over occipital regions. These last 3–8 seconds. Their aspect may present high inter-individual variability. Inter-burst intervals are generally of the same duration as the slow wave bursts (Figs. 3 and 4).

The background activity is symmetrical and synchronous in full-term newborns. Only during transition from active to quiet sleep temporary asynchrony can be observed for up to several minutes without any pathological significance.

Physiological EEG patterns

The following patterns are physiological:

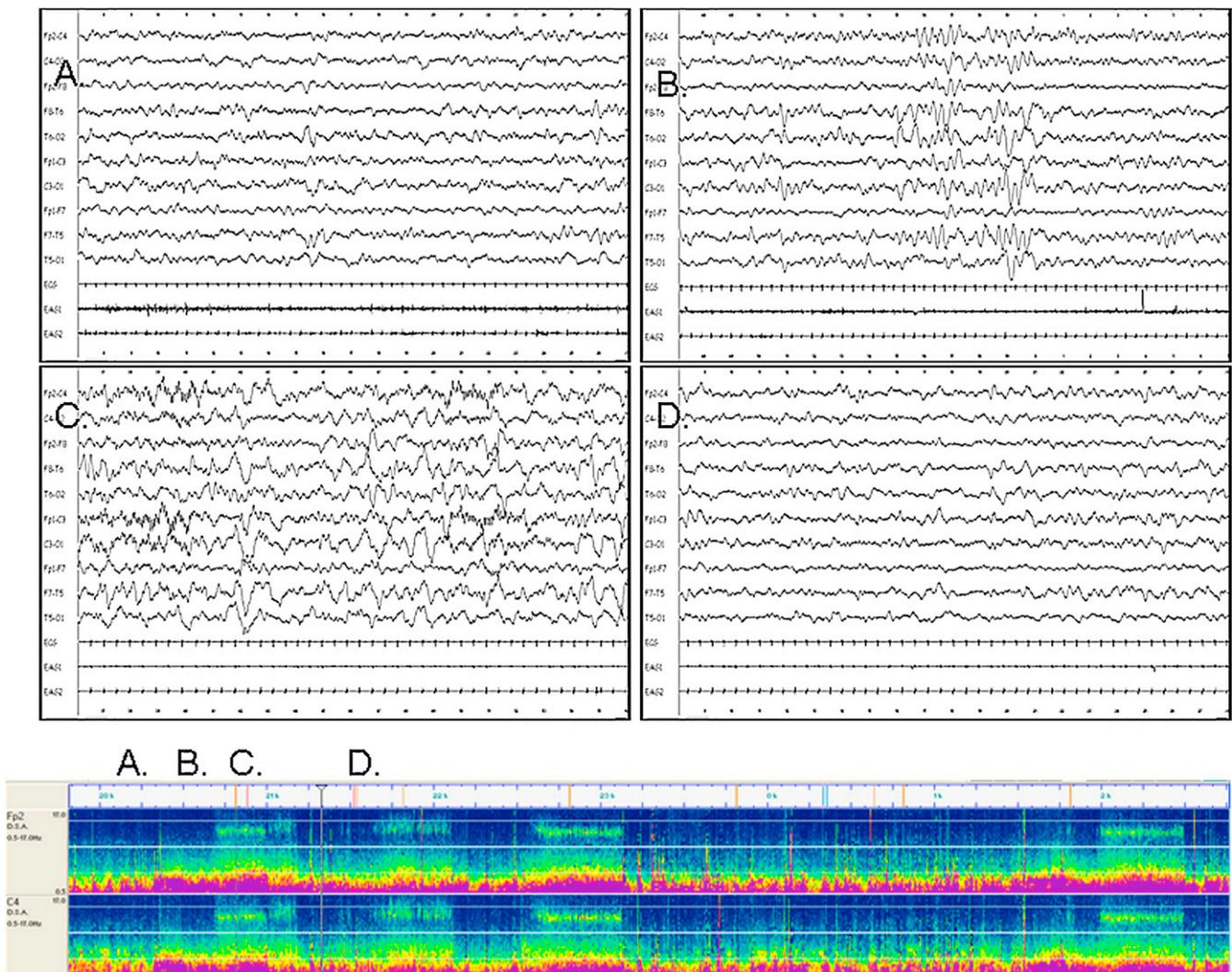


Figure 16 Four-month-old infant. Seven hours nocturnal recording presented as DSA (density spectral array) of derivations Fp2 and C4. Periods of wakefulness (A), drowsiness (B), slow sleep (C) and REM sleep (D). Note the absence of sleep stages III and IV.

- frontal sharp transient (“*encoche frontale*”): specific EEG feature of the newborn from 35 to 44 weeks conceptional age consisting of a diphasic complex, typically with initial negative deflection followed by a larger positive deflexion, uni- or bilateral, sometimes sharp, with a voltage of 50–200 μV and a duration of 0.5–0.75 seconds. These are located in frontal regions, may occur isolated or in repeated bursts. Frontal sharp transients may be absent in strictly normal babies. Although these can be present both in wakefulness and active sleep, these are usually more numerous and of higher amplitude in slow sleep. Differentiation from eye movements can be difficult (Fig. 5) [2];
- anterior slow dysrhythmia (“*dysrythmie lente antérieure*”): another EEG feature, which is specific of the newborn from 36 to 44 weeks conceptional age, consisting of a burst or a short sequence of rhythmic monomorphic delta waves (1–3 Hz, 50–100 μV) located

over frontal regions. It is essentially present in active sleep Type 1 (Fig. 6).

Other EEG patterns and figures

The following patterns can also be observed:

- alpha and theta activity: bursts of alpha and theta waves, of short duration (< 5 seconds) and variable voltage, located in rolandic or fronto-rolandic regions, which can be observed in active sleep Type 1 or quiet sleep (Figs. 7–9);
- positive temporal sharp waves: these positive diphasic sharp waves are located in T₃ or T₄, can be isolated or occur in short bursts, last less than 400 ms and do not have any proven pathological significance (Fig. 10);

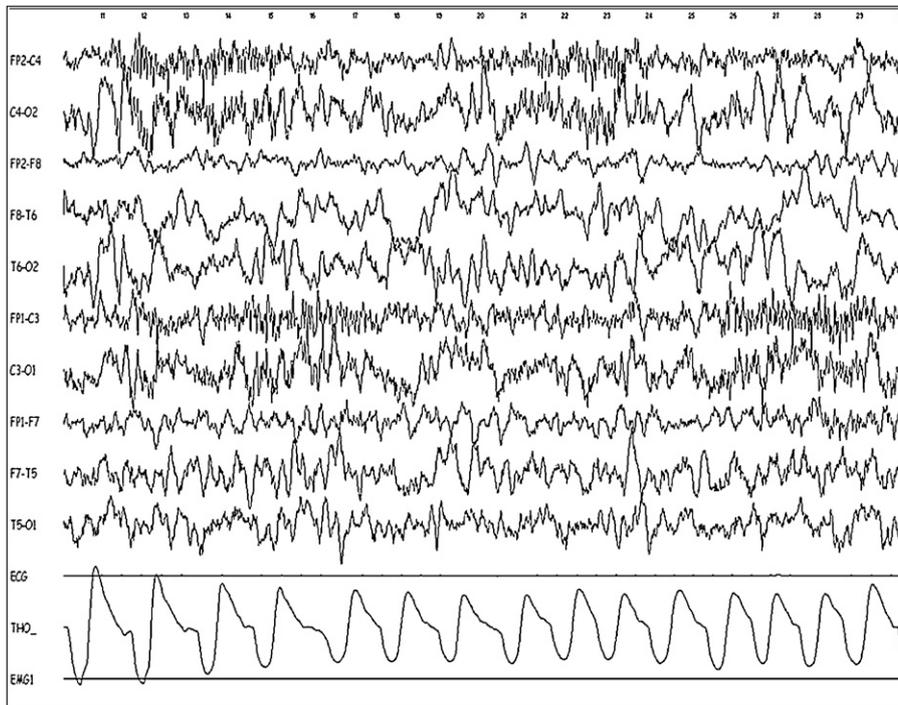


Figure 17 Six-month-old infant. Slow sleep stage II. Asynchronous sleep spindles.

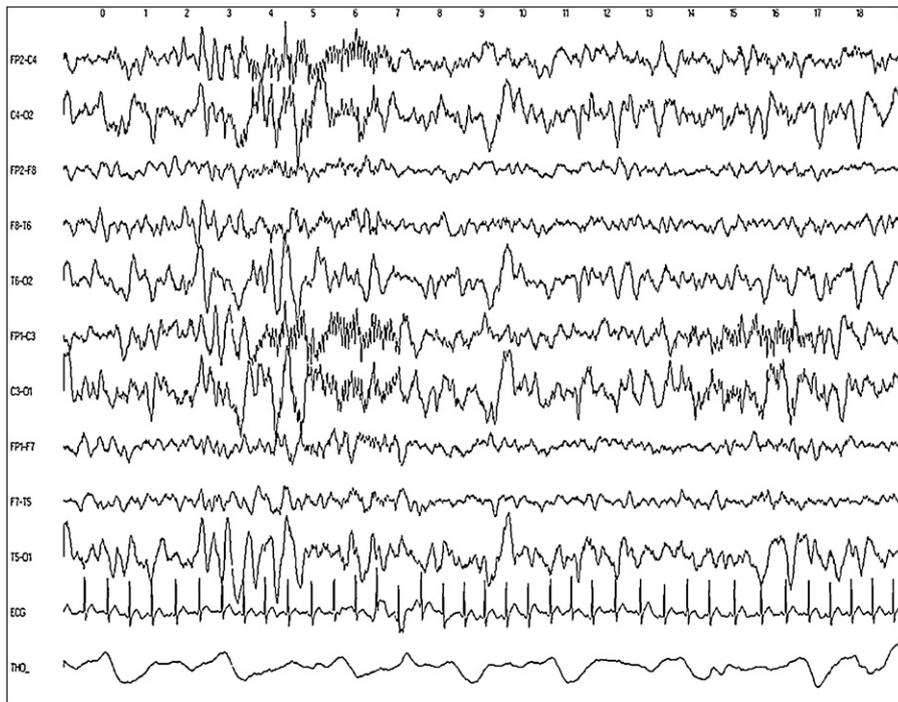


Figure 18 Six-month-old infant. Slow sleep stage II. Synchronous and asynchronous sleep spindles.

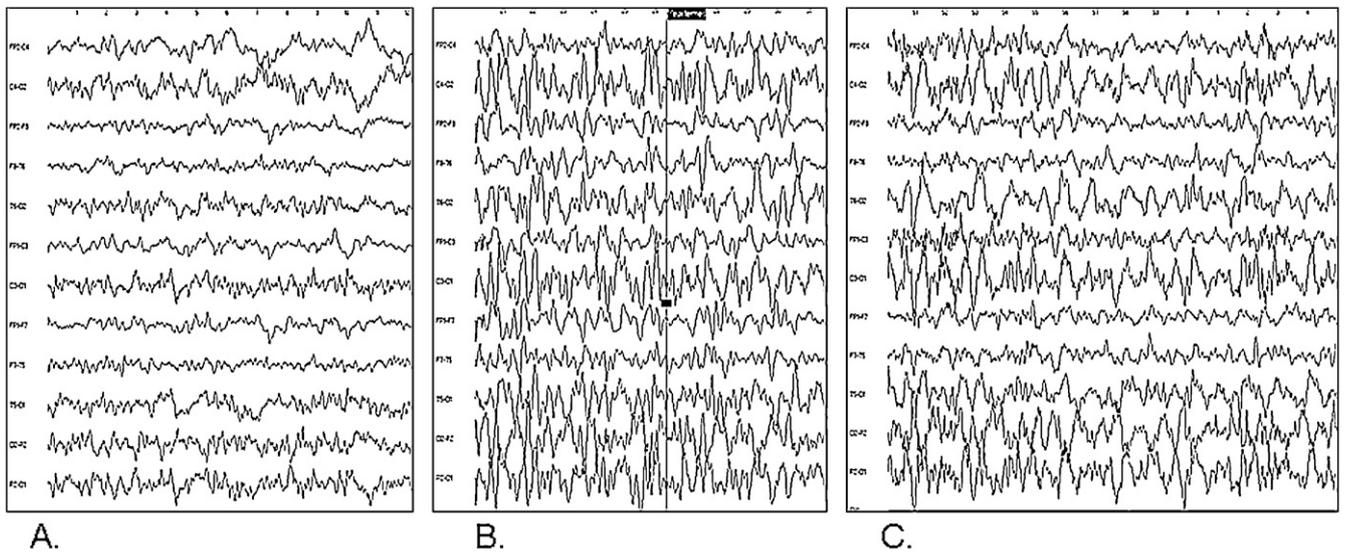


Figure 19 Nine-month-old infant. A: quiet wakefulness. B: drowsiness. C: sleep stage II.

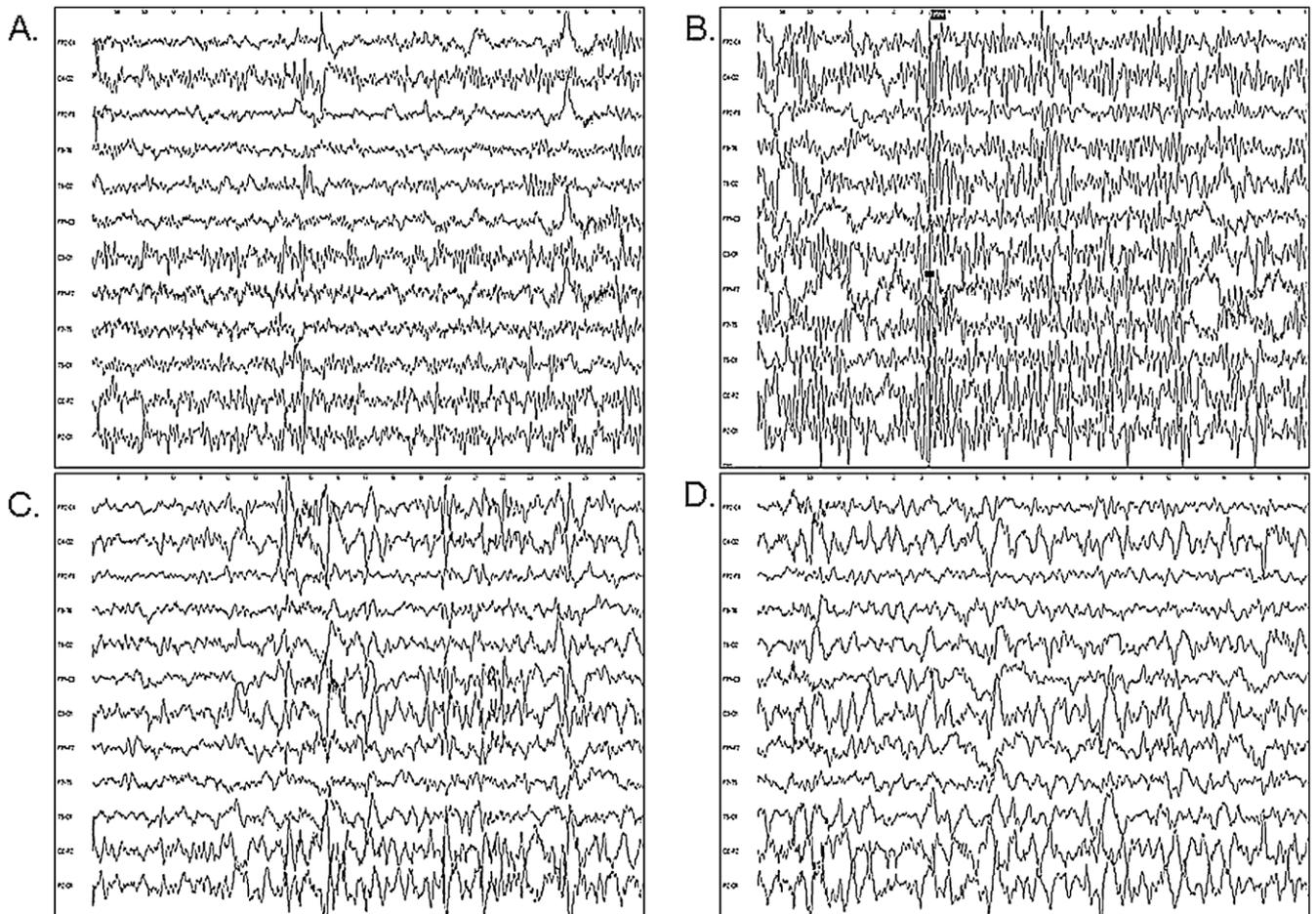


Figure 20 One-year-old infant. A: quiet wakefulness. B: drowsiness. C and D: sleep stage II.

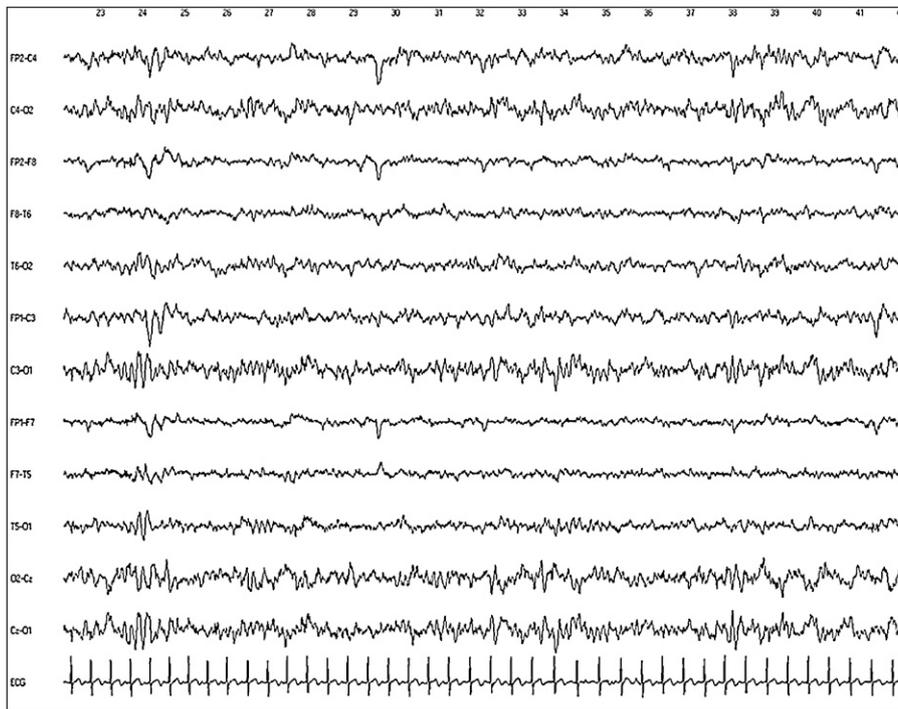


Figure 21 Two and a half-year-old child. Quiet wakefulness. Eyes closed. Posterior basic rhythm in the theta and lower alpha range.

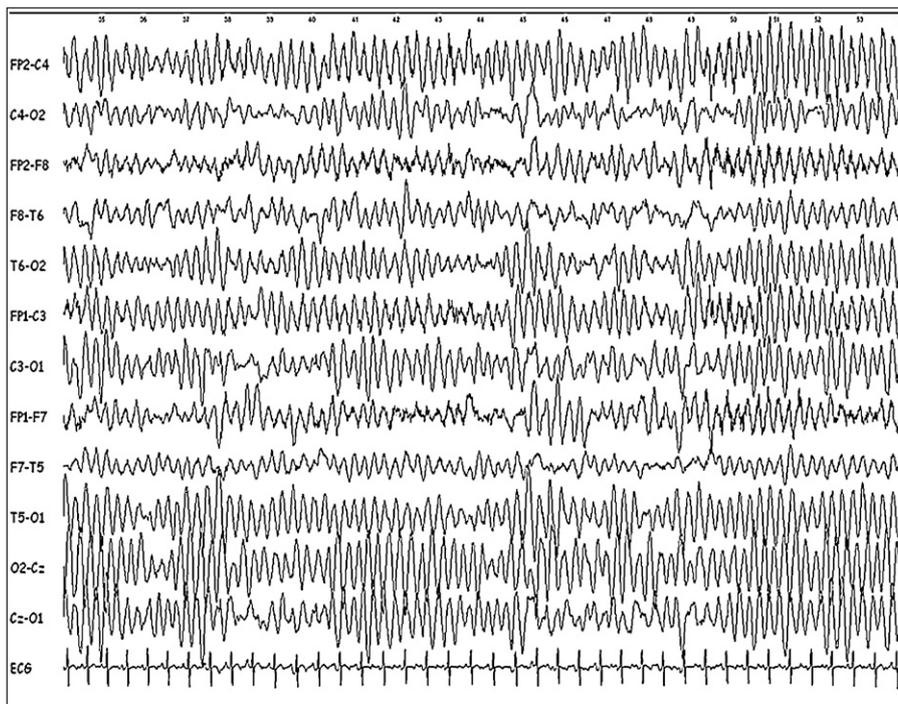


Figure 22 Fifteen-month-old child. Drowsiness. Hypnagogic hypersynchrony.

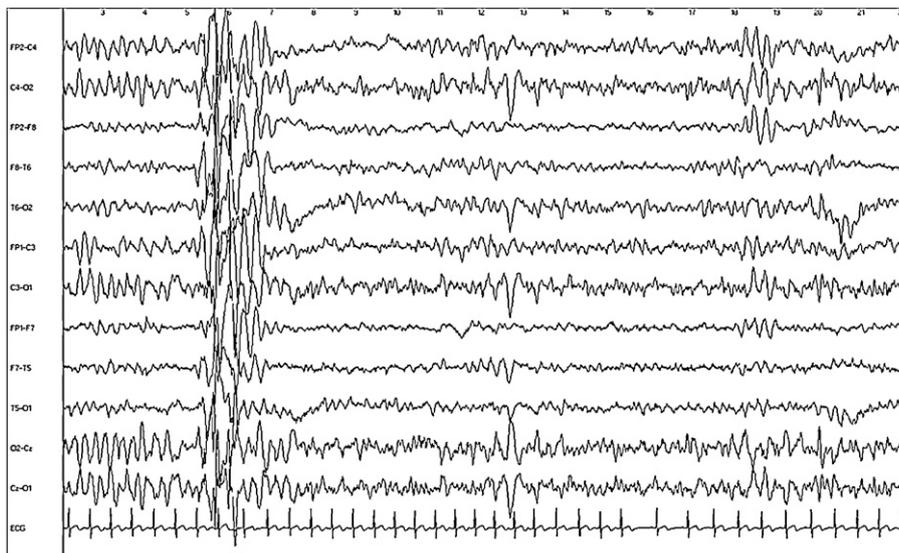


Figure 23 Two and a half-year-old child. Drowsiness. Burst of High voltage slow activity with interspersed spikes.

- rudimentary sleep spindles or pre-spindles: diffuse low-amplitude fast activity predominating over frontal or rolandic regions may be seen in full-term newborns. Their amplitude is much lower and these occur in longer runs as compared to “mature” sleep spindles (Fig. 11);
- frontal sharp transients (neonatal EEG pattern) and sleep spindles (infantile EEG pattern) can co-exist (Fig. 12).

EEG in infancy (1–12 months) [14]

The first 3 months of life are characterized by a gradual transition from neonatal to infantile EEG patterns.

Waking: the medium voltage irregular diffuse activity present in the neonatal period is gradually replaced

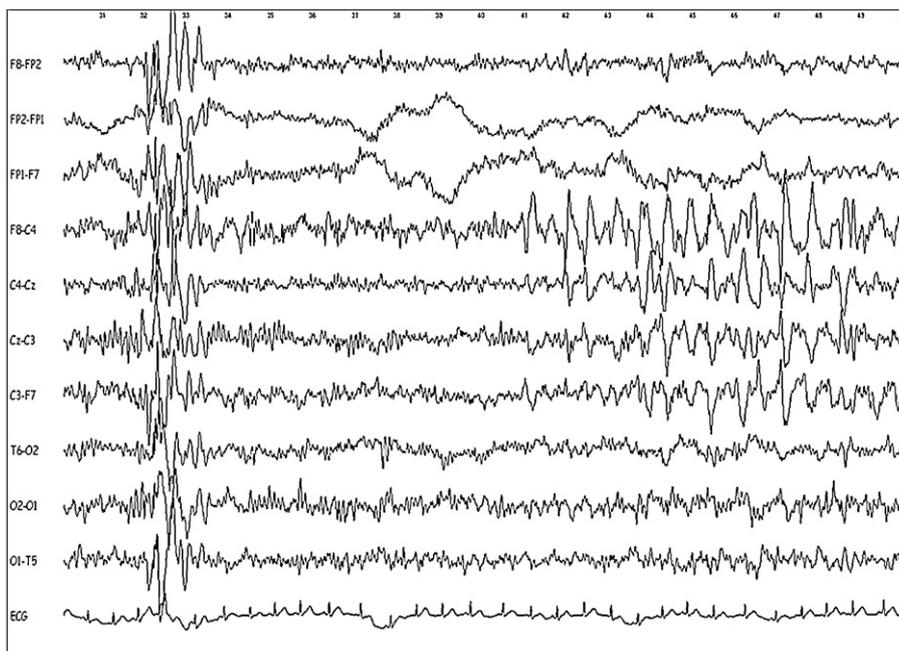


Figure 24 Four-year-old child. Drowsiness. Burst of high voltage slow waves with interspersed spikes (left side) and vertex sharp waves (right side).

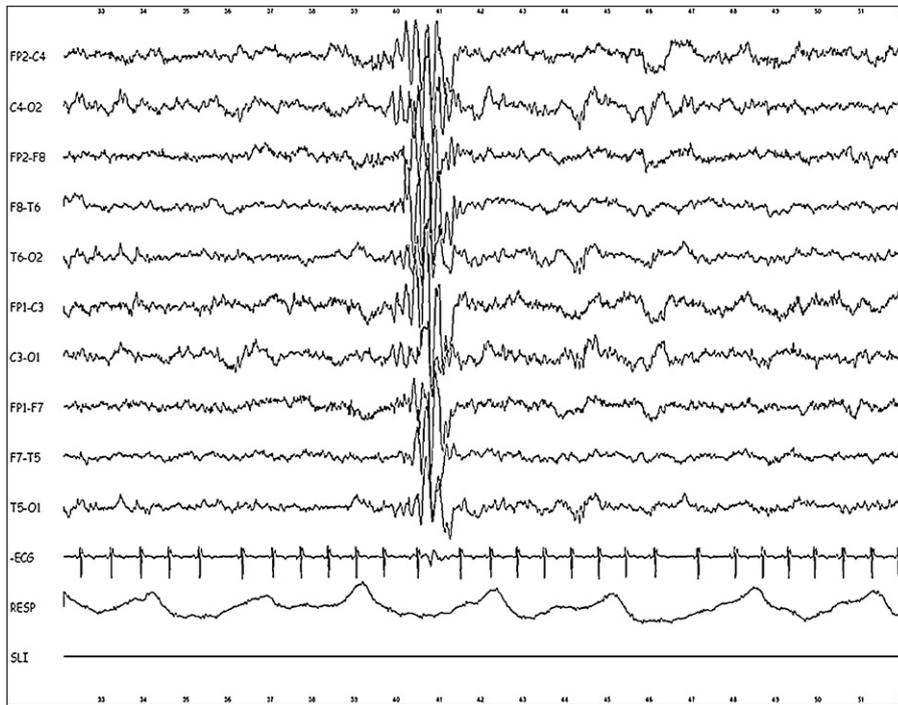


Figure 25 Four-year-old child. Drowsiness. Bursts of diffuse high voltage slow waves.

by more rhythmical theta waves with increasing frequency from 3–4 Hz at 3 months, to 5 Hz at 5 months, and 6–7 Hz at the end of the first year of life. These rhythms, which precede the occipital alpha rhythm,

are initially located over rolandic-occipital regions and can reach a voltage of 75 μ V. A visual blocking response is usually present at the age of 3–4 months (Fig. 13).

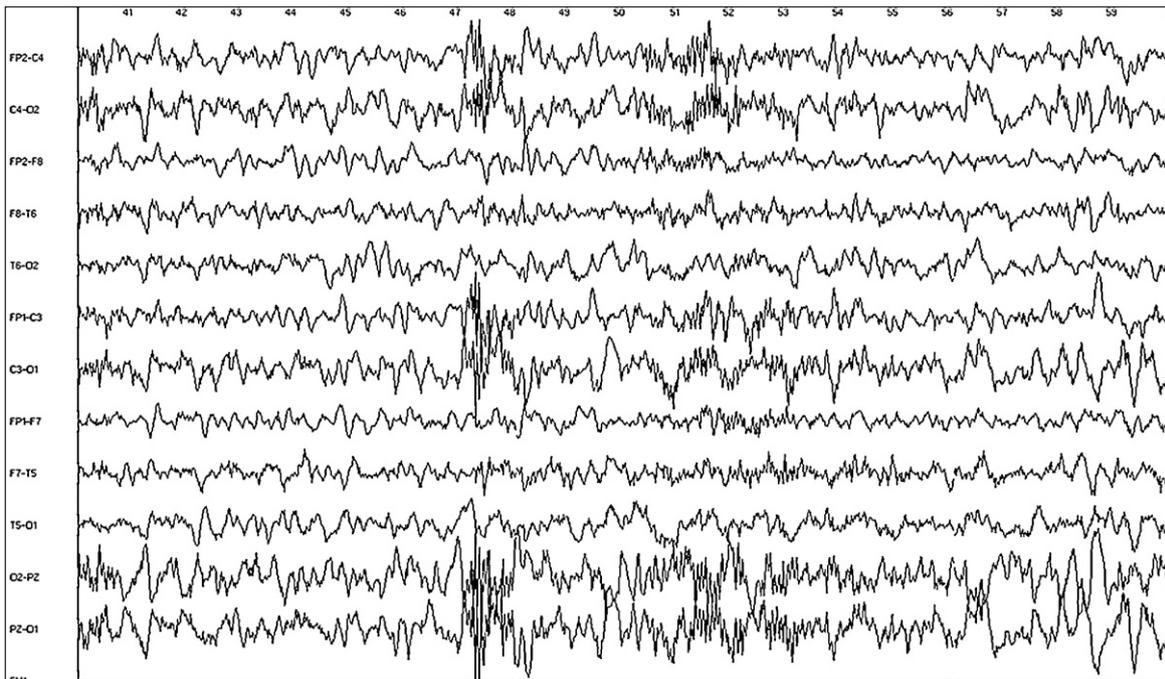


Figure 26 Seventeen-month-old child. Slow sleep. Vertex sharp waves and sleep spindles.

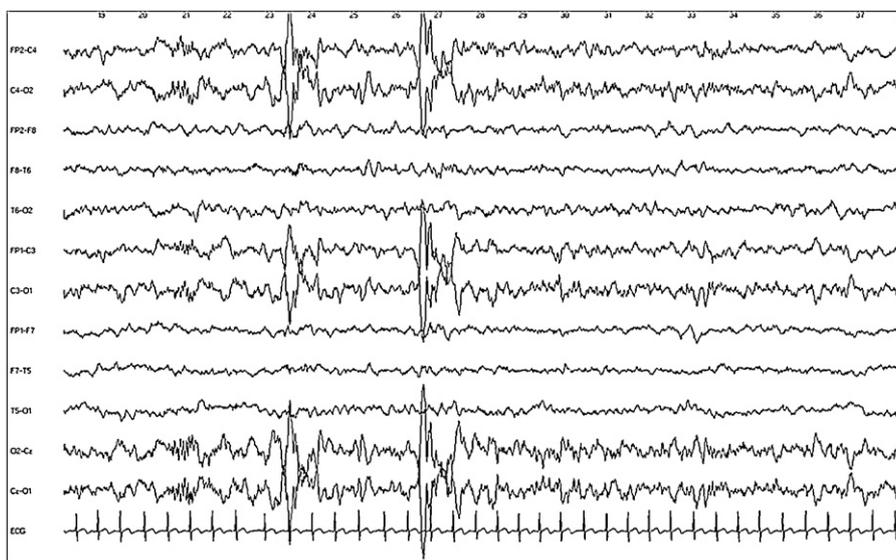


Figure 27 Two-year-old child. Slow sleep. Vertex sharp waves and sleep spindles.

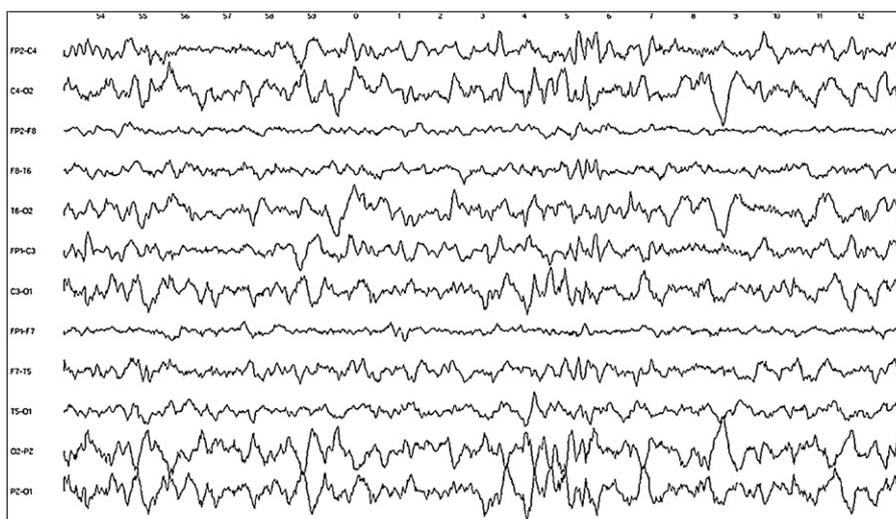


Figure 28 Two-year-old child. Deep slow sleep.

Drowsiness: the transition from waking to sleep is characterized by a progressive slowing into the delta frequency range. From the age of 6–8 months on, a strong rhythmicity predominating in centro-parietal or occipital regions is usually observed. It initially corresponds to the lower theta range around 4 Hz, with a gradual acceleration to 5–6 Hz over the following months. It is known as “hypnagogic hypersynchrony” (Fig. 14) [15].

Sleep: the following “key stages” characterize sleep organisation during this period of life:

- “*tracé alternant*” disappears at about 44 weeks conceptional age and is replaced by a diffuse polymorphic delta activity with maximal amplitude (150–200 μ V) over the occipital areas;
- sleep spindles are bursts of rapid frequencies of 12–15 Hz maximal over central or centro-parietal regions, possibly asynchronous until the age of 6 months, lasting up to 10 seconds. These characterize the onset of stage II slow sleep. These usually appear during the second month of life and persist onto adulthood. Their complete absence

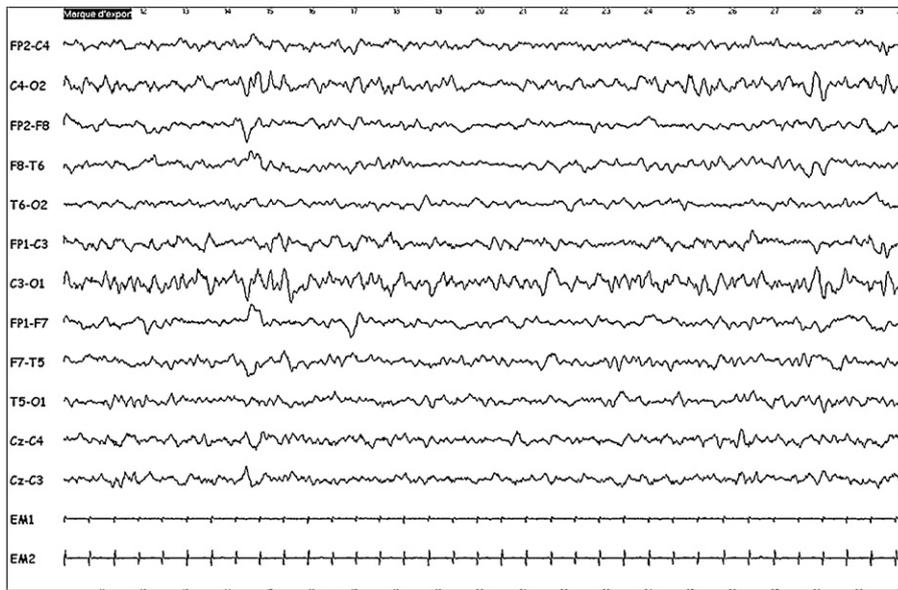


Figure 29 Three-year-old child. REM sleep.

at the age of 3 months represents a severe abnormality (Fig. 15);

- vertex sharp waves and K-complexes usually appear at the age of 5–6 months.

A gradual decrease of REM sleep occurs during the first year of life from around 50% at birth, to 40% at 3–5 months and 30% between 1 and 2 years. REM sleep can present with two aspects: diffuse high-voltage delta activity around 2Hz or diffuse theta activity associated to occipital sharp transients (Fig. 16).

Arousal: after the age of 5 months the activity is comparable to that in drowsiness and consists of diffuse hypersynchrony.

Reactivity: the blocking response of the posterior basic rhythm is present from age 3–4 months on.

Activation: the usefulness of intermittent photic stimulation has not been proven at this age. Although hyperventilation is impossible to achieve because of non cooperation, crying episodes can lead to diffuse slowing of background activity.

By 6 months of age spindles are bilaterally present but may be still asynchronous. Beyond this age synchrony is the rule (Figs. 17 and 18). At 9 months of age drowsiness

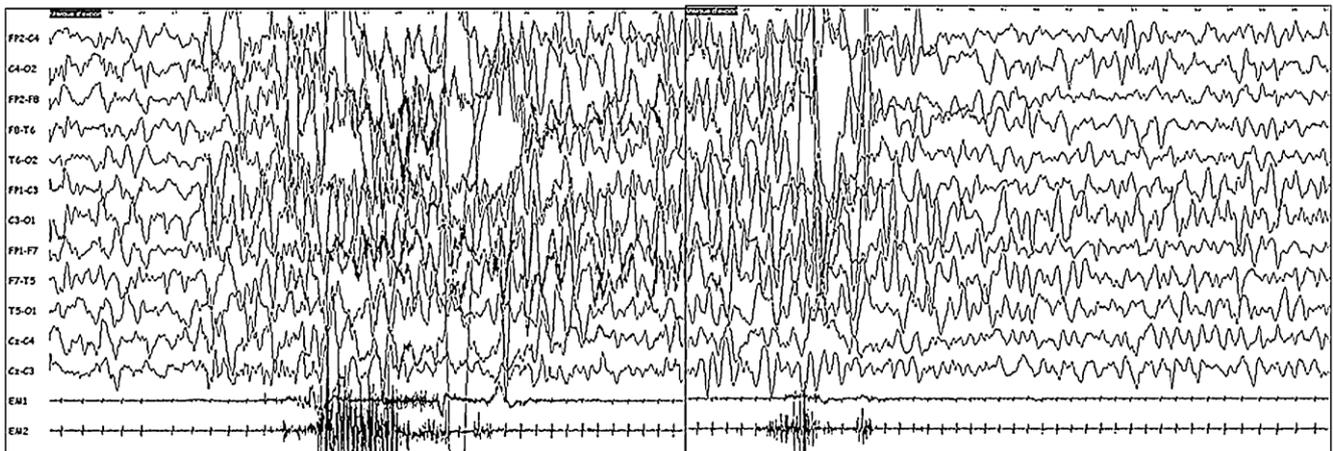


Figure 30 Three years old child. Awakening. High-voltage slow activity 3–4Hz, hypersynchrony during 40seconds.

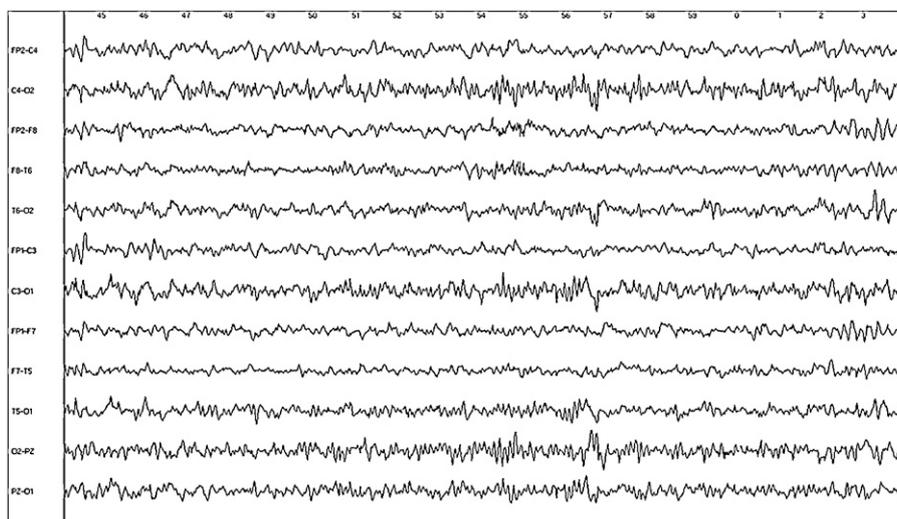


Figure 31 Four-year-old child. Quiet wakefulness. Posterior alpha rhythm, symmetrical, low voltage.

is clearly defined between wakefulness and slow sleep (Fig. 19). At one year of age, slow sleep can be divided into two different states according to the different patterns: spindles followed by diffuse delta waves (Fig. 20).

EEG in early childhood (1–3 years)

Waking (Fig. 13): readable waking records are not always easily obtained at this age. The posterior basic rhythm increases from theta frequencies to lower alpha range

(6–7 Hz in 2nd year, 7–9 Hz in 3rd year) with high interindividual variability (Fig. 21).

Drowsiness: hypnagogic hypersynchrony progressively diminishes (75% at age 1–2 years, 57% at age 2–3 years) (Fig. 22). Other patterns can be obtained in drowsiness as i.e. the ‘anterior theta aspect’ with monomorphic fronto-central theta activity. In a large number of children bursts of diffuse irregular high-voltage slow activity with interspersed spikes can be found without representing a definite abnormality (Figs. 23–25) [12].

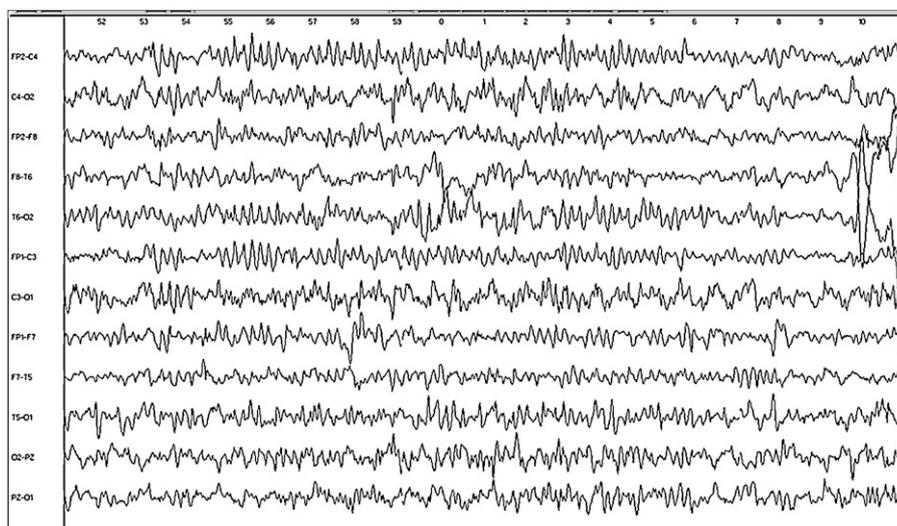


Figure 32 Four-year-old child. Drowsiness. Diffuse theta activity.

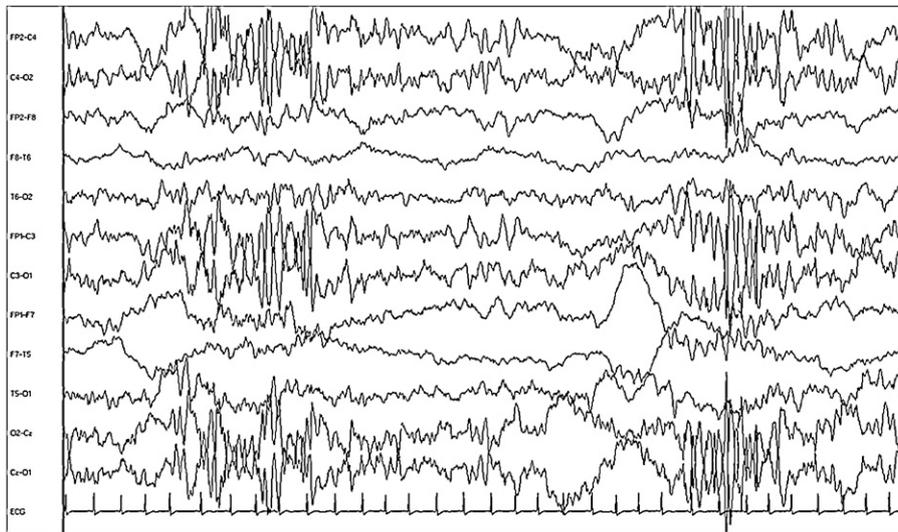


Figure 33 Four-year-old child. Slow sleep stage II. Vertex sharp waves and sleep spindles.

Sleep: stage II sleep is characterized by sleep spindles, which predominate over central regions with maximum over the vertex. These are symmetrical and synchronous with a frequency range of 12–14Hz. In deeper sleep these diffuse onto frontal areas. Vertex waves are very prominent at this age, sometimes presenting with impressive amplitudes and appearing in repetitive bursts (Fig. 26).

K-complexes are diffuse high-voltage slow waves with an initial sharp component followed by fast activity; occurring either spontaneously or after auditory stimulation. These can be observed from the age of 6 months on with a less specific morphology as compared to those obtained in older children; their absence at this age is not pathological (Figs. 27 and 28). Stage III–IV slow sleep is characterized

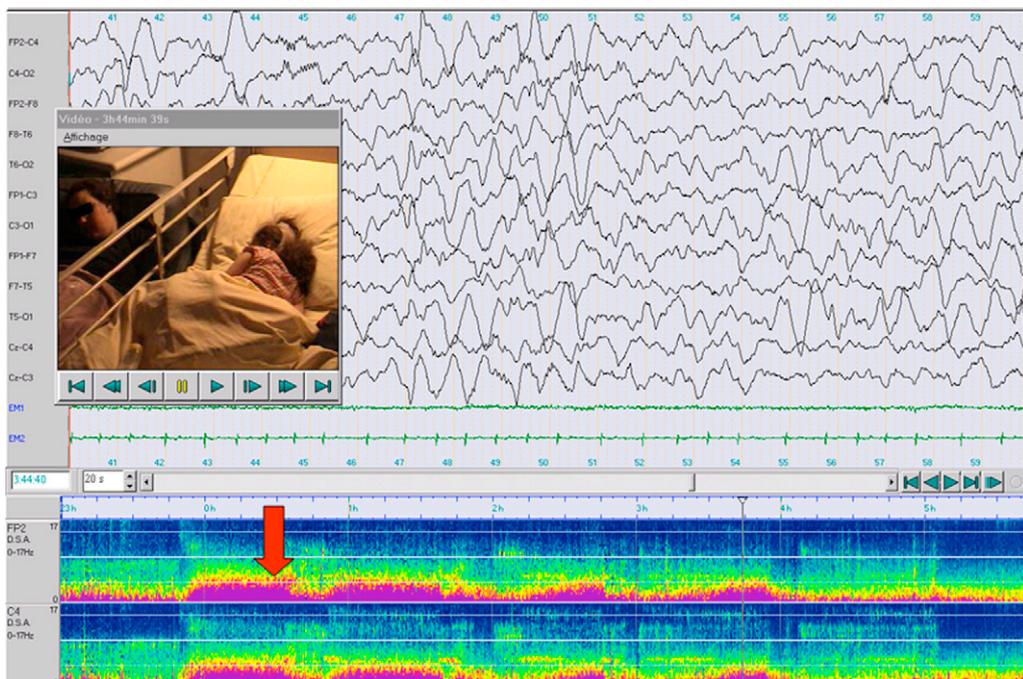


Figure 34 Four-year-old child. Slow sleep stage IV. The arrow indicates SS identified on DSA (rhythms < 5 Hz).

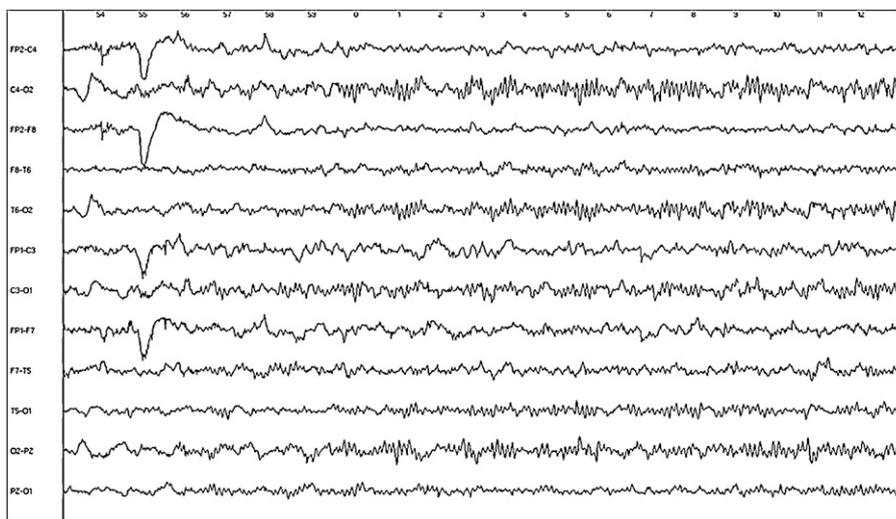


Figure 35 Eight-year-old child. Wakefulness.

by diffuse delta waves predominating on posterior areas (Fig. 28). REM sleep is characterized by diffuse medium-voltage theta waves (Fig. 29).

Arousal: arousal is most often characterized by a prolonged diffuse high-voltage slow activity predominating over frontal areas (Fig. 30).

EEG in preschool children (3–5 years)

Waking: although the posterior basic rhythm has reached the alpha range, it is frequently interrupted by intermingled

theta frequencies predominating over posterior areas. Its amplitude is almost always higher as in adolescence or adulthood (Fig. 31).

Drowsiness: hypnagogic hypersynchrony progressively disappears from the age of 3 years on. Monomorphic fronto-central theta activity increases in early drowsiness and decreases in deep drowsiness (Fig. 32).

Sleep: stage II sleep is characterized by vertex waves, sleep spindles, and K-complexes (Fig. 33) The predominance of slow delta waves over occipital areas is less prominent in comparison to infancy and early childhood. Sleep stages III

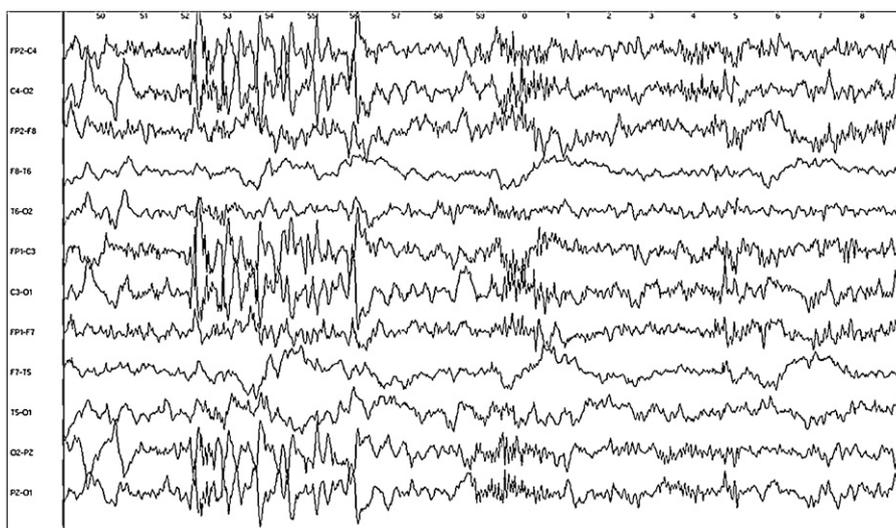


Figure 36 Seven-year-old child. Slow sleep stage II. Vertex waves and sleep spindles.

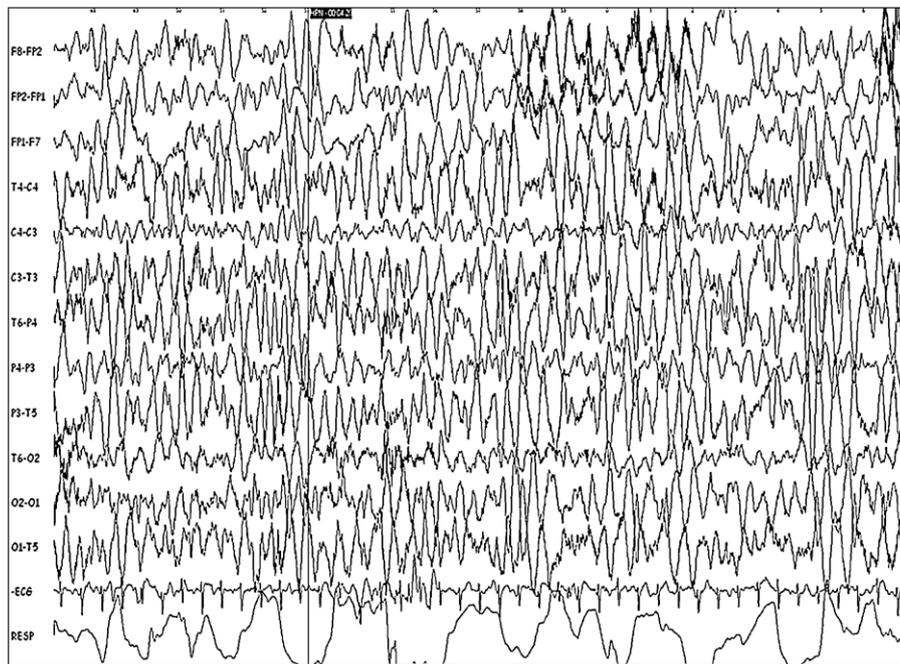


Figure 37 Nine-year-old child. Hyperventilation. Diffuse high voltage delta-theta activity predominating over fronto-central regions.

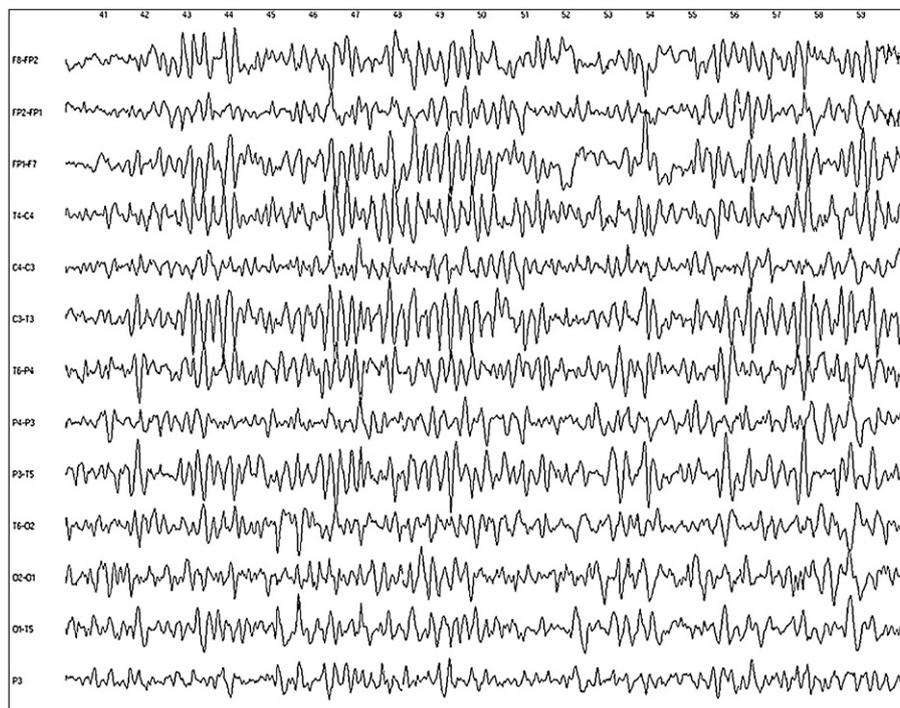


Figure 38 Eleven-year-old child. Hyperventilation. Diffuse monomorphic high voltage theta activity predominating over fronto-central regions.

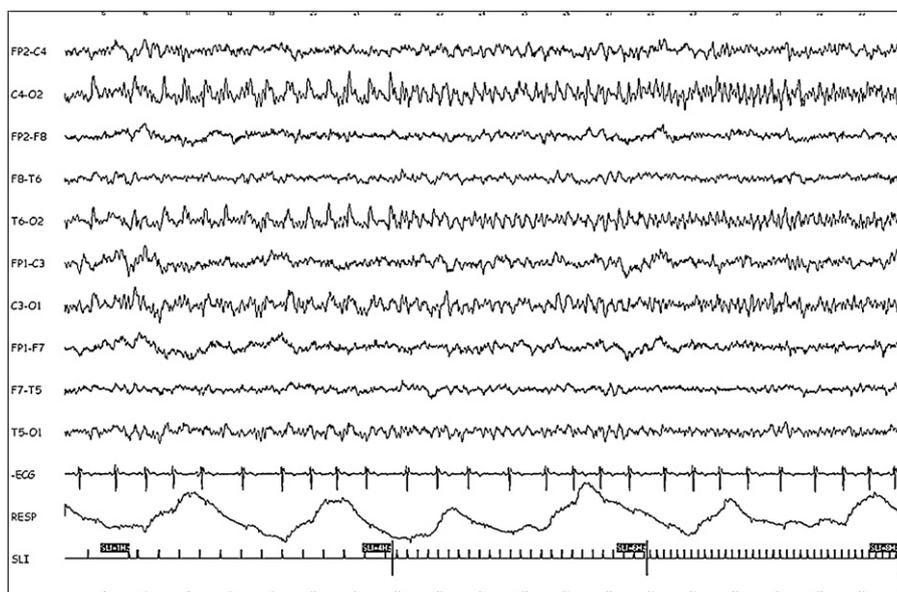


Figure 39 Sixteen-year-old adolescent. Occipital driving response.

and IV are seen from age 3 years on and characterized by increasing diffuse high voltage delta activity (Fig. 34). In REM sleep EEG activity shows low-voltage theta activity.

Arousal: records are comparable to those of younger children.

Activation: from the age of 4 years on, children usually become cooperative for hyperventilation, which can give rise to very pronounced high-voltage slow responses.

Only epileptic discharges or marked asymmetry can be interpreted as pathological.

EEG in school age children (6–12 years)

Waking: the posterior basic rhythm progressively increases up to 11 Hz at age 10–11 years. Its amplitude is generally

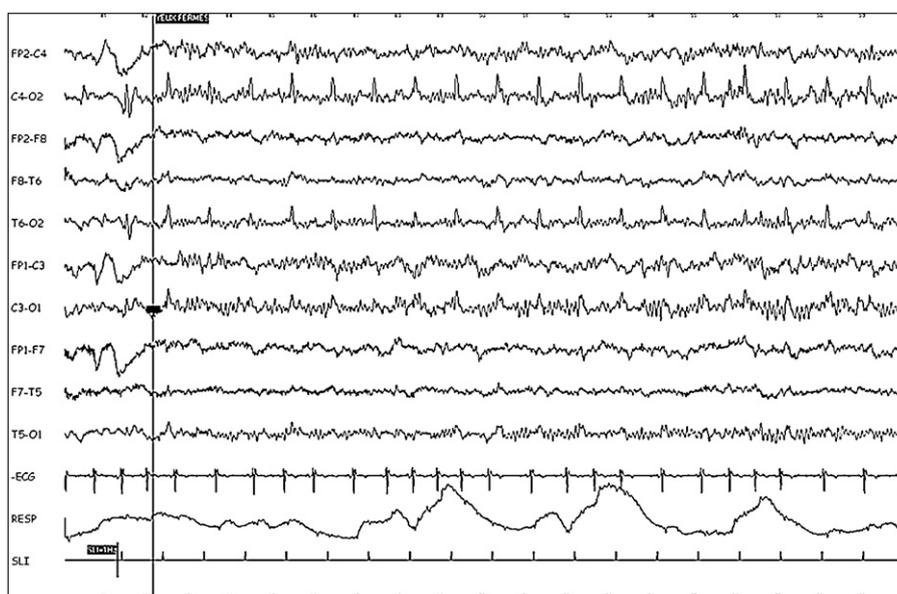


Figure 40 Six-year-old child. Unilateral photic driving at 1 Hz; giant visual potentials.

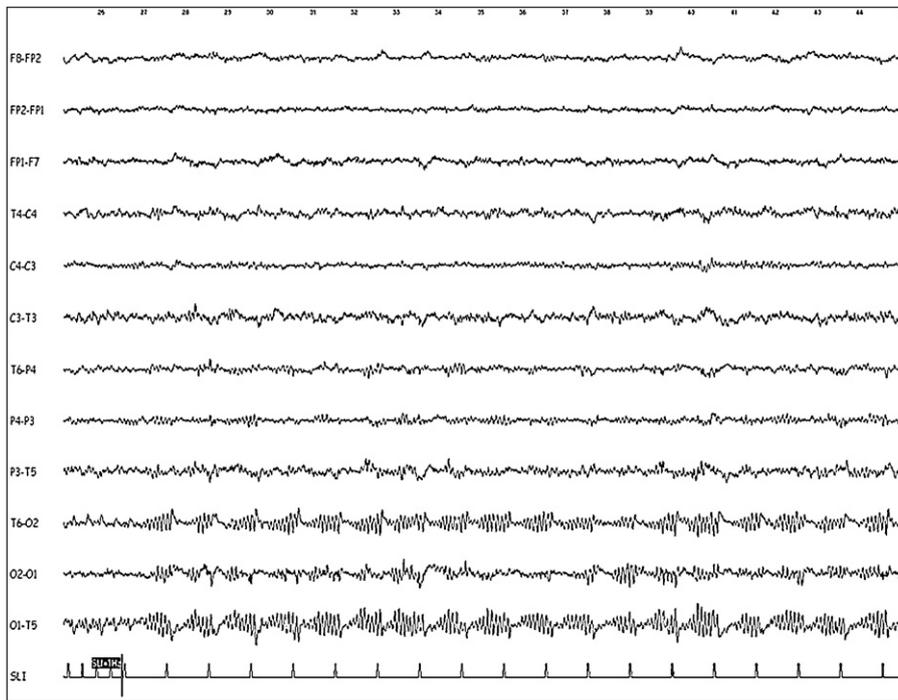


Figure 41 Fourteen-year-old child. Bilateral sinusoidal occipital driving response at 1 Hz.

higher over the non-dominant hemisphere. This asymmetry rarely exceeds $20\mu\text{V}$. Intermixed posterior slow activity is still present at the age of 6 years and clearly diminishes after the age of 12 years. On eye closure a transitory disorganisation with occipital sharp theta activity can be observed (Fig. 35).

Drowsiness: the mature type of drowsiness onset with alpha dropout and mixed low-voltage slow and fast activity

is usually not present before early adolescence. Hypnagogic hypersynchrony disappears at age 6 years, and drowsiness at this age is characterized by increasing slow activities.

Sleep (Fig. 36): vertex waves are often of high amplitude, these occur in bursts, and can be slightly asymmetrical. Sleep spindles have vertex maximum and are usually shorter than 1 second. K-complexes are often combined

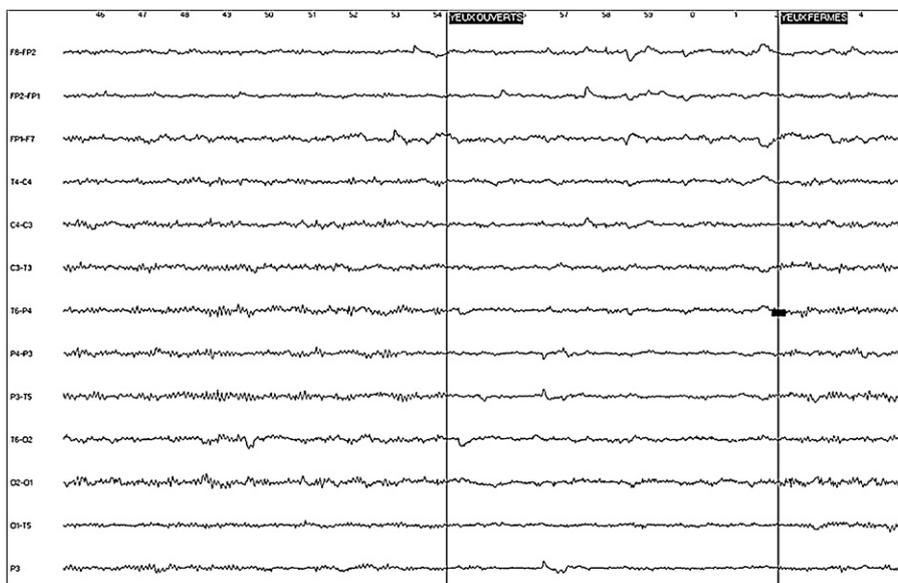


Figure 42 Twenty-year-old. Waking. Blocking response of posterior basic rhythm at eyes opening.

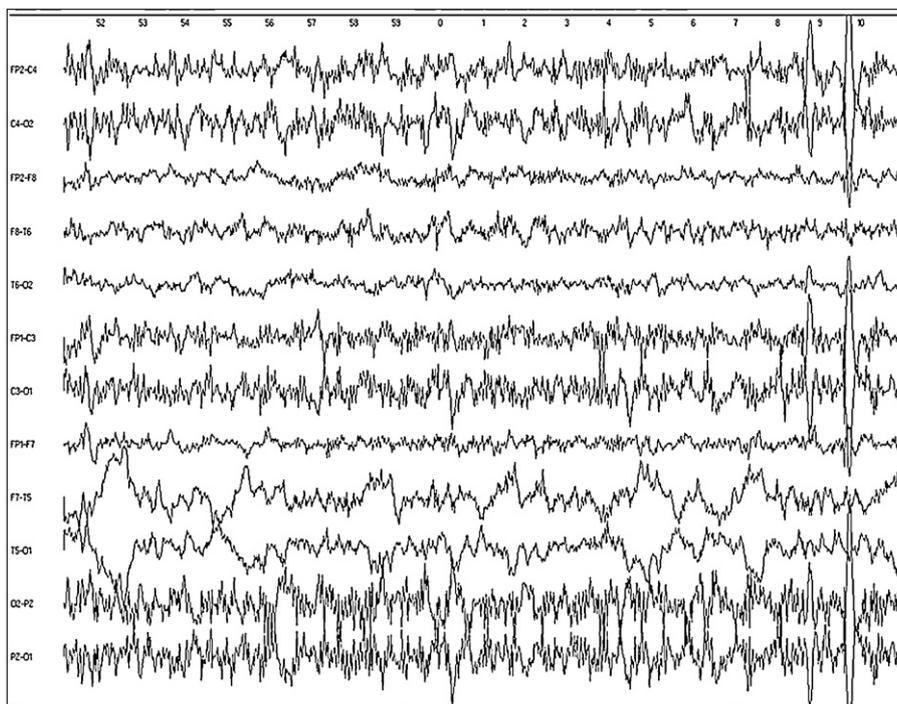


Figure 43 Three-year-old child. Slow sleep stage II. Extreme spindles associated with vertex sharp waves.

with spindle activity. Deep slow sleep contains diffuse high voltage delta activity. REM sleep shows a desynchronized low-voltage mixed activity with theta, alpha, and beta frequencies.

Arousal: arousal is characterized by increasingly shorter transitions from sleep to waking and decreasing length of high-voltage theta frequencies.

Activation: in this age range, hyperventilation discloses a particularly pronounced high-voltage EEG slowing with

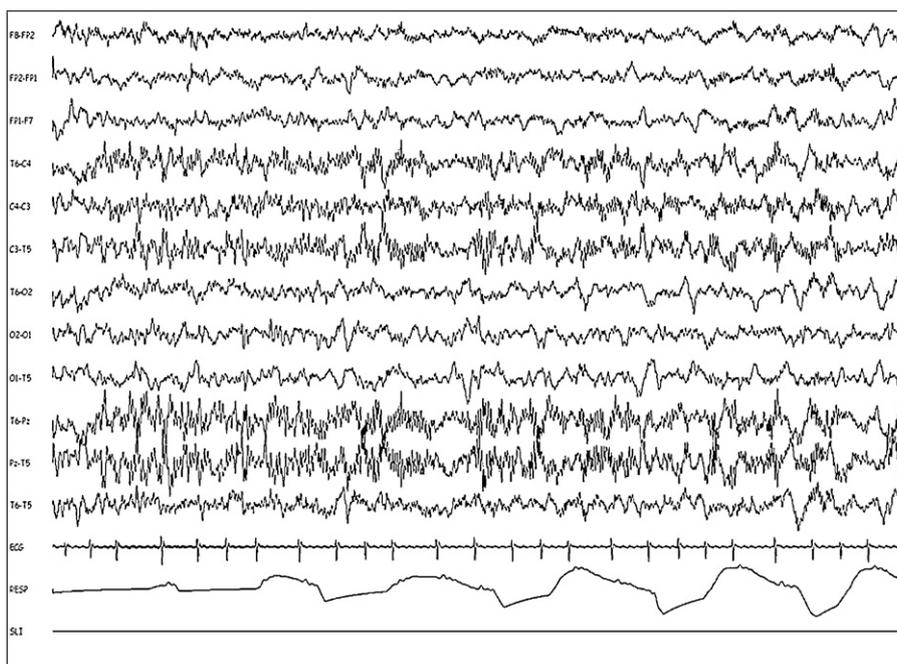


Figure 44 Five-year-old child. Slow sleep stage II. « Extreme spindles ».

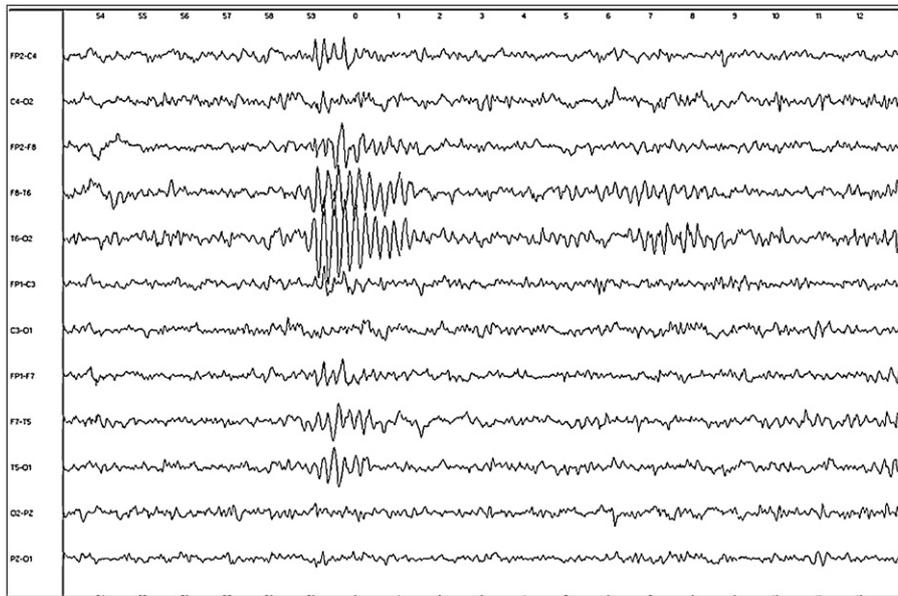


Figure 45 Three-year-old child. Drowsiness. Burst of theta waves predominant on temporal regions more marked on the right hemisphere.

appearance of rhythmical monomorphic theta or delta waves predominating over anterior or posterior areas (Fig. 37). This physiological reaction increases between 7 and 10 years, decreases thereafter, and disappears at about 15 years of age. There is a wide interindividual variability. These EEG changes may last over several seconds after the

end of the hyperventilation before disappearing (Fig. 38) [17].

Intermittent photic stimulation may give rise to an occipital driving response that is less prominent at low flash rates and more important at higher flash rates (Fig. 39). An asymmetrical occipital driving response is not pathological

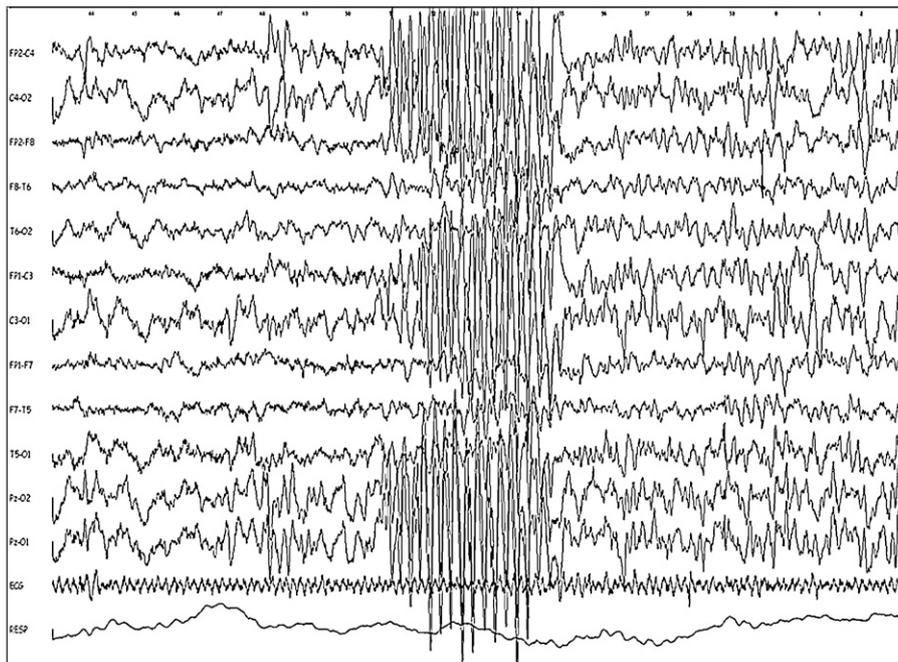


Figure 46 Four-year-old child. Slow sleep stage II. Vertex sharp waves of very high voltage.

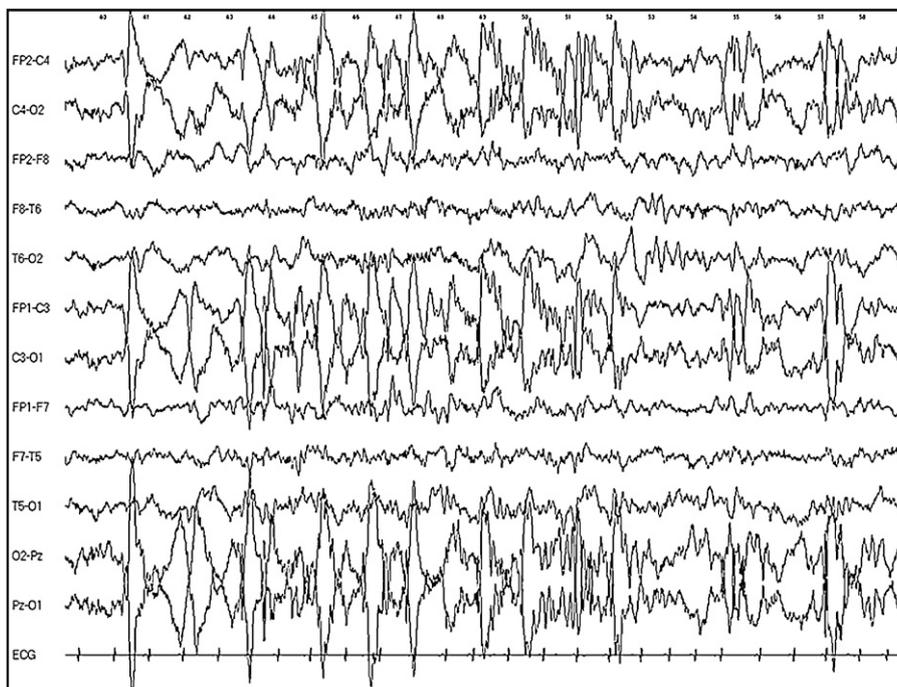


Figure 47 Four-year-old child. Slow sleep. Vertex sharp waves occurring in bursts or "trains".

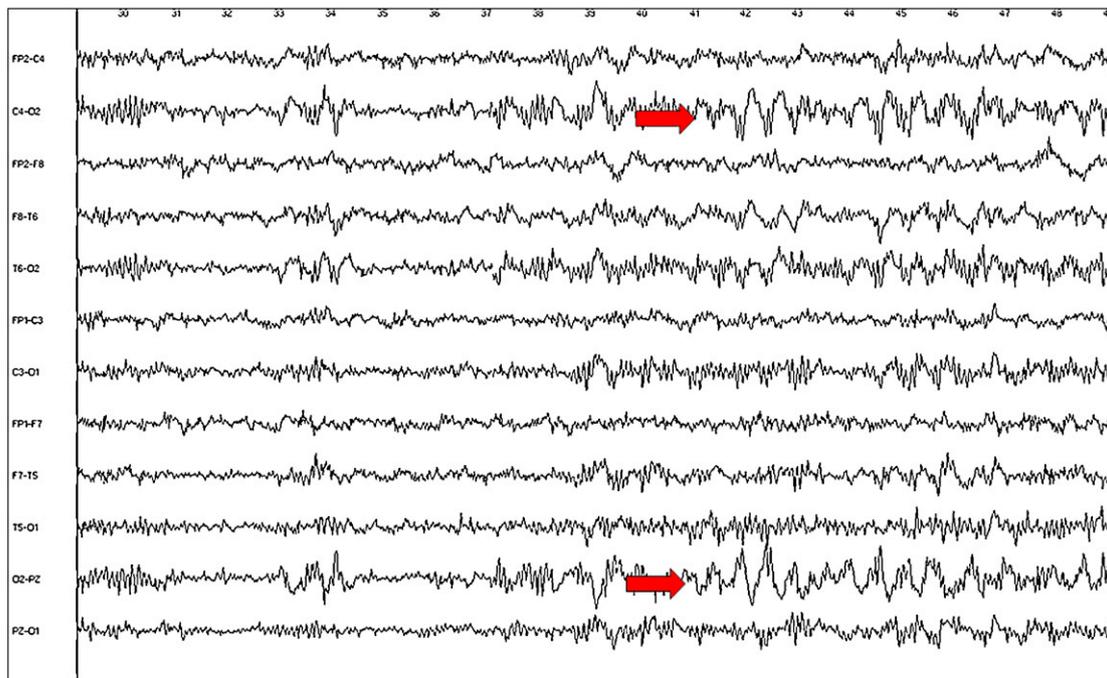


Figure 48 Nine-year-old. Awake state. Bilateral occipital slow waves predominating on the right side with superimposed alpha rhythm.

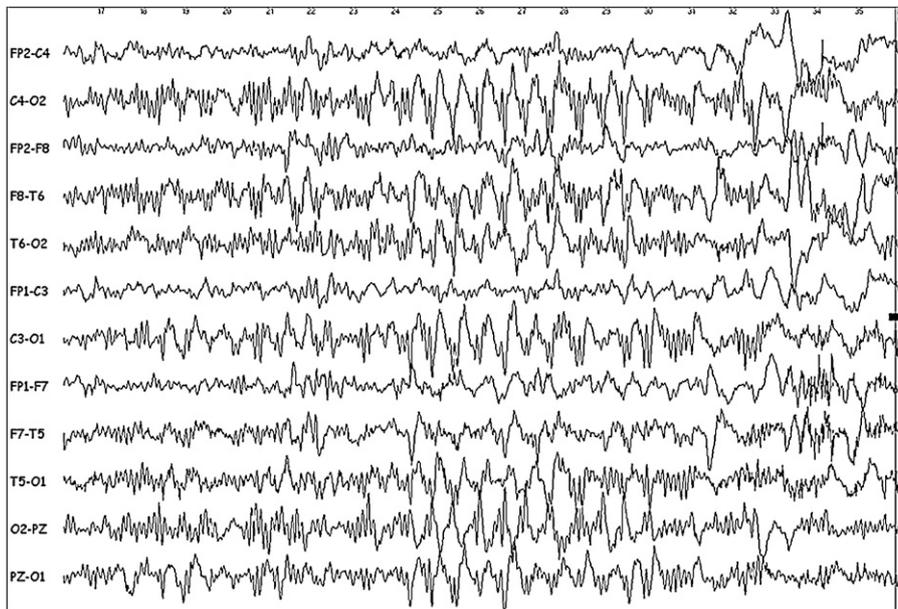


Figure 49 Nine-year-old child. Awake state. Intermixed bilateral occipital slow waves preceded by a sharp wave.

(Fig. 40), neither the bilateral sinusoidal occipital driving aspect at 1 Hz (Fig. 41).

(Fig. 42). Amplitude asymmetry with higher voltage over the non-dominant hemisphere does not exceed 20%. Posterior slow activities decrease during adolescence. Beta frequencies can be observed in frontal regions. Reactions to hyperventilation are found in about 20% of the adolescents. Frontal high-voltage slow waves, sometimes with a rather rhythmic aspect, can be observed in slow sleep and are often often impressively activated at arousal.

EEG in adolescents (13–20 years)

There are no striking changes during this period. The occipital basic alpha rhythm shows a medium frequency of 10 Hz with lower amplitude compared to younger children

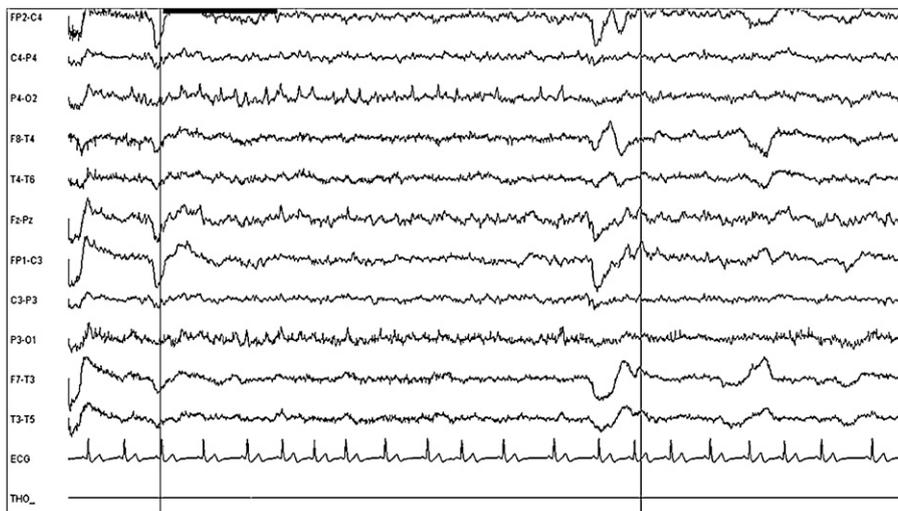


Figure 50 Twelve-year-old child. Awake state. Eyes opened. Lambda waves.

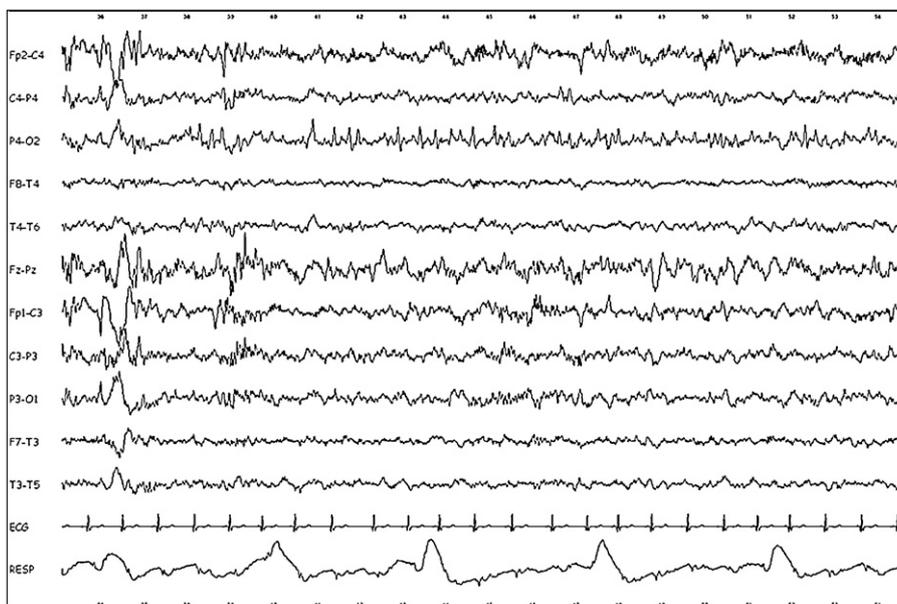


Figure 51 Ten-year-old child. Positive occipital sharp transients in sleep.

Unusual EEG patterns

Most importantly, the following unusual EEG patterns should not be misinterpreted.

Extreme spindles: this unusual variant of sleep spindle activity can be found in 0.05% of normal children. Spindles

are of high voltage with a wide frequency range (6–18 Hz) and occasionally paroxysmal traits, with a possible duration of up to 20 seconds (Figs. 43 and 44).

Theta activity: bursts of high-amplitude theta waves predominating over temporal regions can be observed during drowsiness from age of 3 years



Figure 52 Twelve-year-old child. Rolandic mu rhythm.

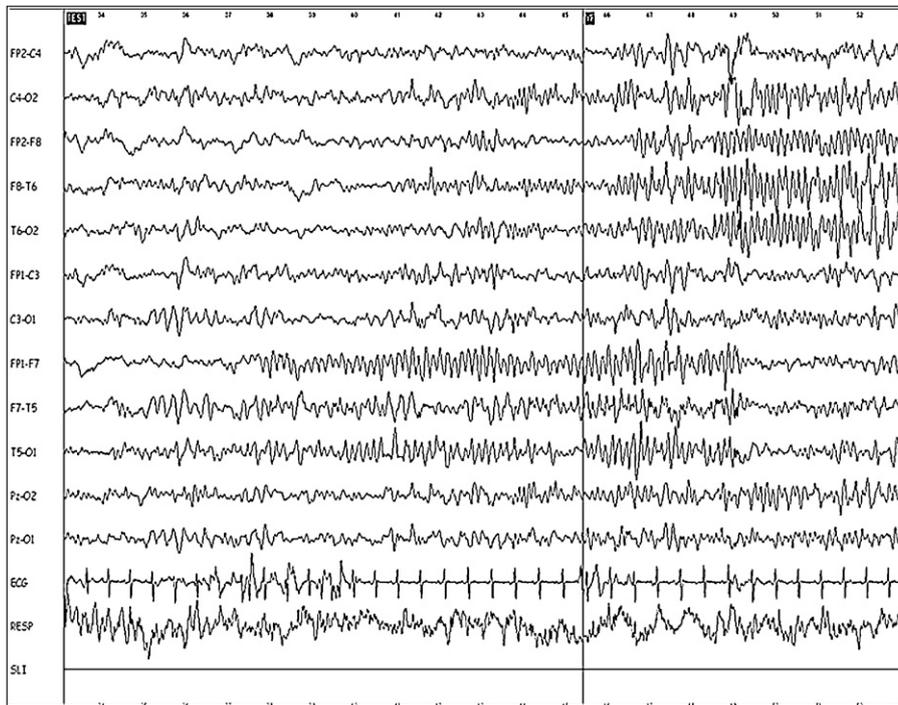


Figure 53 Eight-year-old child. Drowsiness. Psychomotor variant pattern. Bilateral asynchronous rhythmic theta activity over temporal regions.

on: these are vestiges of hypnagogic hypersynchrony (Fig. 45).

Vertex waves: these are present in slow sleep and localized over centro-parietal regions. They can be of very high amplitude, with nearly paroxysmal aspects and occur in bursts or “trains” (Figs. 46 and 47). This aspect is frequent between ages 3 and 5 years. The distinction between vertex waves and epileptic spikes may be difficult [8,10].

Posterior slow waves: these are of variable frequency (delta or theta), are sometimes preceded by a sharp wave, and may be predominant over one hemisphere. In adolescence they are intermixed to the posterior alpha rhythm. The distinction between these “delta waves of adolescence” and posterior spike wave activity can be difficult (Figs. 48 and 49) [1].

Lambda waves: these can be observed from age 3 years on. Lambda waves are sharp transients occurring over the occipital region of waking subjects during visual exploration and disappear at eye closure. These present as biphasic or triphasic waveform of low voltage (20–50 μ V) and a duration of 200–300 ms. These can repeat themselves at intervals from 200–500 ms (Fig. 50) [4].

Positive occipital sharp transients of sleep also called lambda waves of sleep: although well-described in adult patients, this pattern is rather uncommon in children

but important to know. These share the same characteristics as these of lambda waves in the waking state: biphasic waveforms of low amplitude over occipital areas, usually bilateral but possibly unilateral (Fig. 51) [7].

Rolandic mu rhythm also called arcade rhythm or comb rhythm: this activity has been described from the second year of life on. Mu rhythm is a rolandic 10-Hz activity occurring in short stretches and most frequently bilateral but sometimes shifting from side to side. This rhythm persists on eye opening and is partially or totally blocked by movements (active, passive or reflexive). The blocking effect is bilateral but predominates on the rolandic regions contralateral to the side of the movement (Fig. 52) [9,16].

“Psychomotor variant pattern” or rhythmic midtemporal discharge: it is predominantly seen in adolescents but can be observed in younger children. It consists of a rhythmic sharp theta activity of about 5 Hz that is localized over midtemporal regions, mostly diffusing onto anterior or posterior temporal regions, either unilateral or bilateral, synchronous or asynchronous over both hemispheres, often shifting from one hemisphere to the other, possibly activated by hyperventilation or occurring during drowsiness. It is mostly unaffected by eye opening but can be interrupted by auditory stimuli or by simple orders as counting or speaking (Figs. 53 and 54) [3].

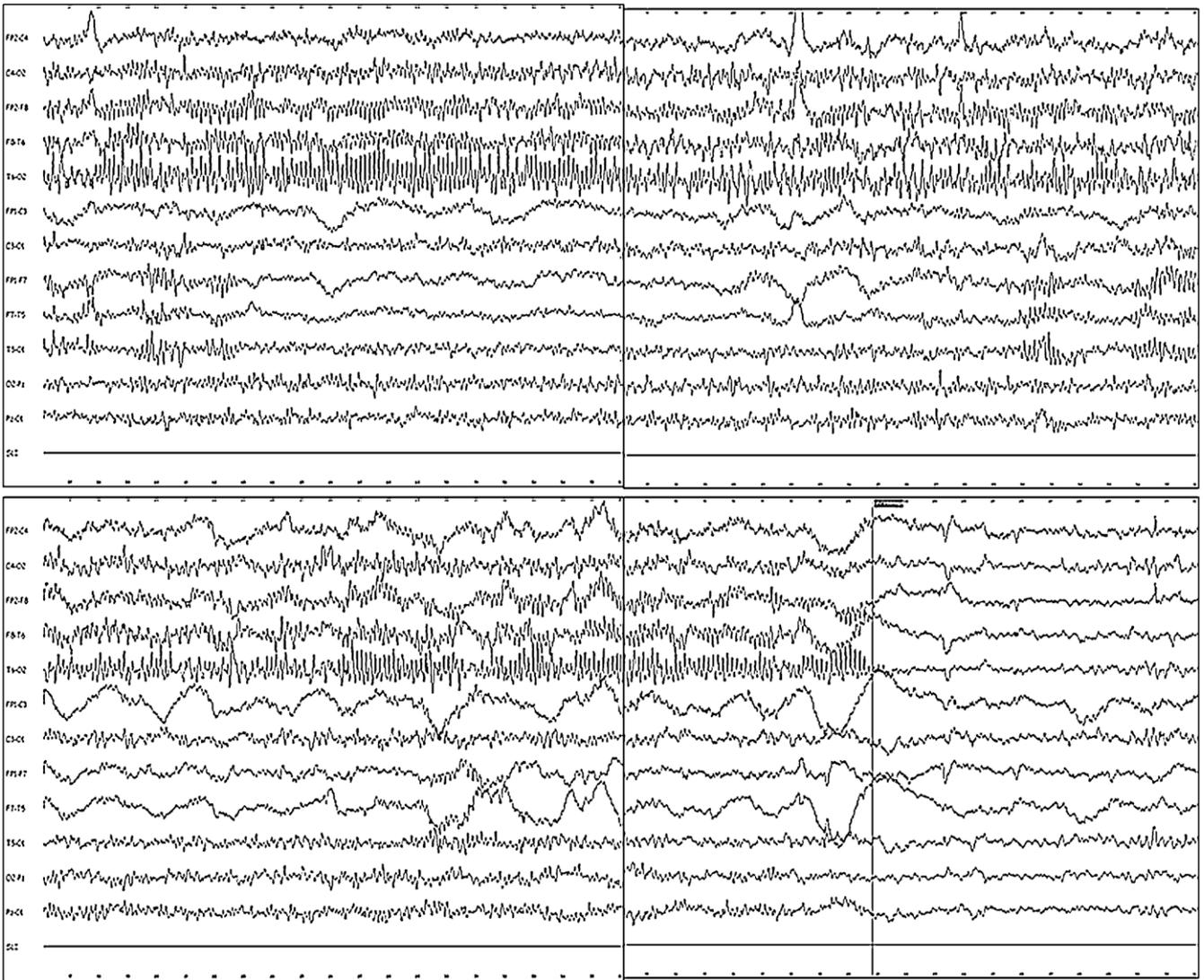


Figure 54 Eight-year-old child. Drowsiness. Psychomotor variant pattern. Bilateral asynchronous rhythmic theta activity over temporal regions.

EEG maturational stages

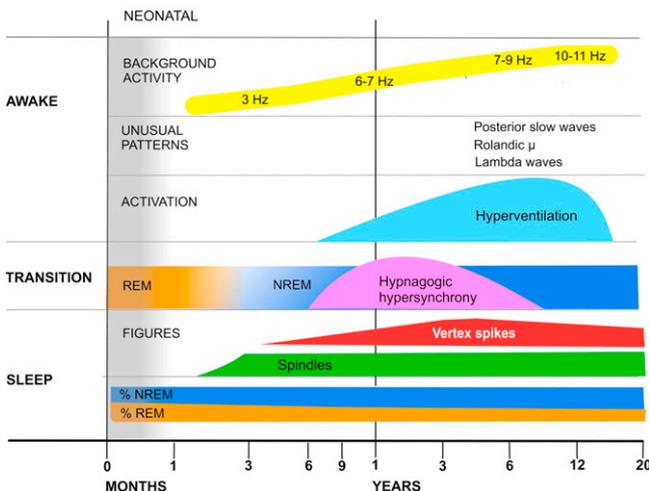


Figure 55 EEG maturational stages.

Conclusions

Fig. 55 summarizes the main features of EEG ontogenesis from birth to early adulthood. It is of utmost importance to know the boundaries between normal and abnormal EEG tracing, according to age and to level of consciousness. Indeed, it may be more dangerous to conclude in a ‘‘pathological EEG’’ when actually facing a normal one, than missing slightly abnormal transients.

Even if there has not been any important study of the normal EEG during infancy and childhood since 1971, many books and atlas have been published on this topic, and a list of the most important ones has been appended to the bibliography.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References

- [1] Aird RB, Gastaut Y. Occipital and posterior electroencephalographic rhythms. *Electroencephalogr Clin Neurophysiol* 1959;11:637–56.
- [2] Andre M, Lamblin MD, d'Allest AM, Curzi-Dascalova L, Moussalli-Salefranque F, Nguyen The S, et al. Electroencephalography in premature and full-term infants. Developmental features and glossary. *Neurophysiol Clin* 2010;40:59–124.
- [3] Arfel G, Leonardon N, Bureau M, Isman ML, Naquet R. Unexplained electrographic temporal discharges. *Rev Electroencephalogr Neurophysiol Clin* 1978;8:335–40.
- [4] Chatrian GE. The lamda waves. In: Rémond A, editor. *Handbook of electroencephalography and clinical neurophysiology*, vol. 6A. Amsterdam: Elsevier; 1976. p. 123–49.
- [5] Dreyfus-Brisac C. Neurophysiological studies in human premature and full-term newborns. *Biol Psychiatry* 1975;10:485–96.
- [6] Eisermann M, Kaminska A, Berdougou B, Brunet ML. Melatonin: experience in its use for recording sleep EEG in children and review of the literature. *Neuropediatrics* 2010;41(4):163–6.
- [7] Gastaut Y. Un signe électroencéphalographique peu connu : les pointes occipitales survenant pendant l'ouverture des yeux. *Rev Neurol* 1951;84:640–3.
- [8] Gastaut H. Étude électrocorticographique de la réactivité des rythmes rolandiques. *Revue Neurol (Paris)* 1952;87:176–82.
- [9] Koshino Y, Niedermeyer. Enhancement of Rolandic mu-rhythm by pattern vision. *Electroencephalogr Clin Neurophysiol* 1975;38:535–8.
- [10] Kuhlo W, Heintel H, Vogel F. The 4-5 c-sec rhythm. *Electroencephalogr Clin Neurophysiol* 1969;26:613–8.
- [11] Lamblin MD, André M, Auzoux M, Bednarek N, Bour F, Charollais A, et al. Indications of electroencephalogram in the newborn. *Arch Pediatr* 2004;11:829–33.
- [12] Lericque-Koechlin A, Mises J, Arnaud MB. Les figures paroxysmiques de l'EEG au cours de la somnolence et du sommeil chez le jeune enfant. *Rev Neurol* 1966;115:497–8.
- [13] Lombroso CT, Matsumiya Y. Stability in waking-sleep states in neonates as a predictor of long-term neurologic outcome. *Pediatrics* 1985;76:52–63.
- [14] Petersen I, Eeg-Olofsson O. The development of the electroencephalogram in normal children from the age of 1 through 15 years: non-paroxysmal activity. *Neuropädiatrie* 1971;3:247–304.
- [15] Plouin P, D'Allest AM, Bour F, Lericque A, Mises J, Moussalli-Salefranque F, et al. Aspects EEG de l'endormissement et du réveil chez l'enfant de 1 à 4 ans (pathologie lourde exclue). *Rev EEG Neurophysiol* 1981;11:45–50.
- [16] Storm van Leeuwen W, Wienecke G, Spoelstra P, Versteeg W. Lack of bilateral coherence of mu-rhythm. *Electroenceph Clin Neurophysiol* 1978;44:140–6.
- [17] Yamatani M, Konishi T, Murakami M, Okuda T. Hyperventilation activation on EEG recording in childhood. *Epilepsia* 1994;35:1199–203.

Further readings

- Blume WT, Kaibara M, editors. *Atlas of Pediatric Electroencephalography*; 1999.
- Crespel A, Gelisse P, editors. *Atlas of Electroencephalography. EEG Awake and sleep EEG*, vol. 1. John Libbey Eurotext; 2005.
- Deuschl G, Eisen A, editors. *Recommendation for the practice of clinical neurophysiology: guidelines of the International Federation of Clinical Neurophysiology*. Elsevier Science; 1999, 320 p.
- Gibbs FA, Gibbs EL. *Atlas of electroencephalography*, vol. 1. Cambridge, MA: Addison-Wesley; 1950.
- Gibbs FA, Gibbs EL. *Atlas of electroencephalography*, vol. 2. Reading (Mass): Addison-Wesley; 1952.
- Gibbs FA, Gibbs EL. *Atlas of electroencephalography*, vol. 3. Reading (Mass): Addison-Wesley; 1964.
- Holmes GL, Moshe SL, Jones HR. *Clinical neurophysiology of infancy, childhood, and adolescence*. Butterworth-Heinemann Ltd; 2005.
- Mizrahi EM, Hrachovy RA, Kellaway P, editors. *Atlas of neonatal electroencephalography*; 2003.
- Niedermeyer E. In: Niedermeyer E, Lopes da Silva F, editors. *Electroencephalography, basic principles, clinical applications, and related fields*. 4th ed. Lippincott Williams&Wilkins; p. 189–214.
- Plouin P, Kaminska A, Moutard ML, Soufflet C, editors. *L'EEG en pédiatrie*. John Libbey Eurotext; 2006, 185 p.
- Pressler R, Colin D, Binnie CD, Cooper R, Robinson R, editors. *Neonatal and paediatric clinical neurophysiology*. Samson-Dollfus D, editors. *Électroencéphalographie de l'enfant*. Paris: Masson; 1998, 136 p.
- Stockard-Pope JE, Werner SS, Bickford RG. *Atlas of neonatal electroencephalography*, 2nd ed. New-York: Raven Press; 1992.