HANDBOOK OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY

EDITOR-IN-CHIEF A. REMOND

VOLUME 6

The Normal EEG Throughout Life

EDITOR: G.C. LAIRY

Hôpital Henri-Rousselle, Paris (France)

PART B

The Evolution of the EEG from Birth to Adulthood

EDITOR: G.C. LAIRY

Hôpital Henri-Rousselle, Paris (France)

ELSEVIER

HANDBOOK OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY

Editor-in-Chief: **Antoine Rémond**
Centre National de la Recherche Scientifique, Paris (France)

VOLUME 6

The Normal EEG Throughout Life

Editor: **G. C. Lairy**
Hôpital Henri-Rousselle, Paris (France)

PART B

The Evolution of the EEG from Birth to Adulthood

Editor: **G. C. Lairy**
Hôpital Henri-Rousselle, Paris (France)

Elsevier Scientific Publishing Company – Amsterdam – The Netherlands

International Federation of Societies for EEG and Clinical Neurophysiology

HANDBOOK EDITORIAL COMMITTEE

ISBN 0-444-41390-1

Elsevier Scientific Publishing Company, Jan van Galenstraat 335, Amsterdam
Printed in The Netherlands

Sole distributor for Japan:
Igaku Shoin Ltd.
5-29-11 Hongo Bunkyo-ku
Tokyo

All other countries:
Elsevier Scientific Publishing Company
Amsterdam, The Netherlands

International Federation of Societies for EEG and Clinical Neurophysiology

A great need has long been felt for a Handbook giving a complete picture of the present-day knowledge on the electrical activity of the nervous system.

The International Federation of Societies for EEG and Clinical Neurophysiology is happy to be able to present such a Handbook, of which this is a small part.

The decision to prepare this work was made formally by the Federation at its VIIth International Congress. Since then nearly two hundred specialists from all over the world have collaborated in writing the Handbook, each part being prepared jointly by a team of writers.

The Handbook begins with an appraisal of 40 years of achievements by pioneers in these fields and an evaluation of the current use and future perspectives of EEG and EMG. The work subsequently progresses through a wide variety of topics – for example, an analysis of the basic principles of the electrogenesis of the nervous system; a critical review of techniques and methods, including data processing; a description of the normal EEG from birth to death, with special consideration of the effect of physiological and metabolic variables and of the changes relative to brain function and the individual's behaviour in his environment. Finally, a large clinical section covering the electrical abnormalities in various diseases is introduced by a study of electrographic semeiology and of the rules of diagnostic interpretation.

The Handbook will be published in 16 volumes comprising 40 parts (about 2500 pages altogether). For speed of publication most of the 40 parts will be published separately and in random order.

ISBN 0-444-41390-1

Elsevier Scientific Publishing Company, Jan van Galenstraat 335, Amsterdam
Printed in The Netherlands

Sole distributor for Japan:
Igaku Shoin Ltd.
5-29-11 Hongo Bunkyo-ku
Tokyo

All other countries:
Elsevier Scientific Publishing Company
Amsterdam, The Netherlands

PART B

THE EVOLUTION OF THE EEG FROM BIRTH TO ADULTHOOD

Editor: **G. C. Lairy**
Laboratoire de Neurophysiologie Clinique et de Psychophysiologie Expérimentale, Hôpital Henri-Rousselle, Paris (France)

Collaborators:

L. Curzi-Dascalova, *Hôpital Port-Royal, Paris (France)*

C. Dreyfus-Brisac, *Hôpital Port-Royal, Paris (France)*

O. Eeg-Olofsson, *Ostra Sjukhuset, Goteborg (Sweden)*

G. C. Lairy, *Hôpital Henri-Rousselle, Paris (France)*

N. Monod, *St. Cloud (France)*

S. Netchine, *Laboratoire de Psychobiologie de l'Enfant, Paris (France)*

I. Petersén, *Sahlgrenska Sjukhuset, Goteborg (Sweden)*

U. Selldén, *Sahlgrenska Sjukhuset, Goteborg (Sweden)*

Introduction

To give an exhaustive view of the waking EEG from birth to adulthood in the "normal" subject is not an easy project. There is indeed an ontogenetic evolution of the EEG, that is, a progressive maturation of the electrical activity of the brain, which is now well-known in its general lines. But the complexity of its description, interpretation and relationships with other domains of physical and psychological maturation makes it necessary to raise a number of preliminary comments.

To define what is "normal" and to draw the limits of normalcy is difficult, although indispensible for using the EEG as a diagnostic tool. Methodological remarks already made on "normalcy" in the adult EEG (see Volume 6A of this Handbook) are also valuable here. But, in addition, one has to keep in mind a series of general and important points: in any study of development "adultomorphism" must be avoided. A child is not, or not only, a small unachieved adult; he is a different organization. So the EEG in children must not be judged in regard to the adult EEG but in regard to specific child organization or successive organizations.

Variability in children is much greater than in adults and expresses itself as marked intra-individual and extra-individual variability of the EEG. Cross-sectional age studies define statistical means but also deviations from the mean. These deviations are an intrinsic part of the "normal" range and must not be interpreted (as they often are) as abnormalities. Reactivity (this term being taken in its wider sense) of the physiologically immature organism and of course of its EEG, is always greater than in the mature organism. The range of plasticity in reaction to external and internal factors, which may be of short or of very long duration, is a fundamental component of the child organization to be taken into consideration.

This Part was intended to treat only the waking EEG of the normal child. Obviously, the waking EEG can only be clearly delineated when the waking state is clearly differentiated from sleep. At the very beginning of human life and especially if this beginning is precocious as in premature infants, sleep and waking are too intertwined to allow such a differentiation, which only obtains its full significance when the adult-like sleep-waking circadian rhythm is complete. For this reason descriptions of the EEG during sleep have been given in the first Sections.

EEG descriptions are usually made in terms of frequencies mainly defined as patterns constituted by more or less rhythmically grouped frequencies. Implicitly, it is thought that these different patterns originate from different anatomo-physiological structures, giving each of them a specific significance. In fact, if individuation of these frequencies or patterns is indispensible or unavoidable and if progressive temporal changes in these frequencies or patterns are one of the main characteristics of EEG maturation, it is only a segmentary aspect of the whole problem, which is the dynamic organization of brain activity. From a descriptive point of view this concept of organization implies both relationships of electrical activities from the different areas of the brain (spatial parameters) and their variations with time (temporal parameters), the latter having to take into consideration short term and long term variations.

Whether EEG maturation relates to neuronal, biological or psychological maturation is of crucial importance for interpreting the EEG and delineating steps, phases or stages of evolution. During the first weeks or months of life, one may say that all these domains are indistinguishable, and that each of them depends on the other. At this stage, the EEG can be safely related to the physical maturation of the central nervous structures. But myelinization, development of dendritic connections, etc., reach their final state long before the EEG reaches its definite mature aspect. EEG maturation must then be related to "development" as a whole, this term including different fields (somatic, psychological, ...). Determining relationships between EEG and development is made difficult by two main factors: first, development is never a linear process. It proceeds by successive steps linked by phases of slowing, arrest or even regression of one or another field of evolution; second, there is no strict correspondence between physical, biological and psychological development, each of which follows specific curves of evolution that are never superimposed or even parallel.

Reference to chronological age is necessary in such a study but is valuable only with respect to statistical data. For individual cases, it is too ambiguous a reference to define development, except for very young ages (premature infants, new born babies). Later, development must be appreciated at different levels, taken into consideration together, to be reliably related to EEG maturation, especially when a distinction between normal and abnormal must be made.

An important point for EEG interpretation, especially before adulthood, is to determine the respective importance of internal and environmental factors. Most commonly a quasi-absolute primacy is given to internal factors: the EEG is thought to reflect the functioning of the brain, this functioning being responsible for biological homeostasis, psychological structuring, etc. Any deviation of the EEG from the "normal" is interpreted as the consequence of primary dysfunction of the brain or as an "abnormality" or impairment of the cerebral structures, which in turn will be taken as responsible for any deviation in development or behavior. The role of external or environmental factors is denied, ignored or markedly under-estimated and considered only in terms of immediate reactivity to exteroceptive stimuli.

Greater importance should be given to environmental factors and especially long term ones in the dynamic organization of the EEG during brain maturation and development. Clinically it is well known that the handling of a baby influences the quality of his feeding and of his sleep-waking balance. In the extreme, hospitalism or abandonism can lead to impairment of physical and mental health, and sometimes to death. At a later age, harmful environmental factors may induce stress, developmental disturbances, cognitive or behavior problems, etc. It is logical to think that they also have an influence on EEG organization, or successive organizations during maturation. Conversely, possible reverberant consequences on the immature EEG of an improved environment should not be ignored.

To appreciate at every moment the respective importance of internal and environmental factors on the EEG is most often left to the subjective appreciation of the EEGer and depends on his own theoretical attitude towards the significance attributed to this apparently "objective" trace of the brain. Consequently, the different Sections of this Part are unavoidably tainted with the theoretical position of their authors, resulting in possible discrepancies particularly concerning the limits to be given to "normal" as opposed to "abnormal". These discrepancies exist between laboratories and must not be underestimated: however precise the description of an EEG, however complete the statistical scales, it is the interpretation of the EEG that counts in its handling for diagnosis and treatment of the individual subject. Taking into account the extreme plasticity of the physiologically immature organism, to know not only the statistical means but also the ranges of possible normal variations under the influences of multiple and constantly intermingled factors is indispensible to any practitioner. Otherwise, the EEG could be misused and lead to conclusions prejudicial to the subject.

Section I. The Electroencephalogram of Full-Term New Borns and Premature Infants

A. INTRODUCTION

The EEG provides very good information on the development of the central nervous system.

The bioelectrical activity of the central nervous system may be recorded through scalp electrodes, if the survival of the early premature new born is possible for at least a few hours, at the conceptional age of 24 weeks (calculated from the first day of last menses). At this early age, the state of the new born cannot be identified as wakefulness or sleep—some weeks later, at about 32 weeks, wakefulness and sleep are two different states, but their differentiation is still very imprecise—crying and movements with eyes open are the main distinctive characteristics. As recording is generally impossible in wakefulness, we mainly record sleeping infants.

The EEG of the full-term new born was recorded first by Loomis *et al.* (1938), Lindsley (1938) and Smith (1938a, c). Later, main contributions to the descriptions of the EEG of new borns and prematures were given by Hughes *et al.* (1948a, b, 1949), Ellingson and Lindsley (1949), Ellingson (1958), Arfel-Capdevielle (1950), Gibbs and Gibbs (1950), Mai *et al.* (1951, 1953), Janzen *et al.* (1952), Mai and Schaper (1953), Melin (1953), Samson-Dollfus (1955), Dreyfus-Brisac and Monod (1956a), Kellaway (1957).

Records of premature infants in incubators were first described by Hughes *et al.* (1951) and Mai and Schaper (1953). The first systematic study of maturation was given by Samson-Dollfus (1955). Since that time the main studies have been those of Dreyfus-Brisac *et al.* (1956b, 1962, 1964), Ellingson (1964a) and Polikanina (1966).

Polygraphic studies of sleep of premature new borns have been mainly performed by Monod and Dreyfus-Brisac (1965a), Weitzman *et al.* (1965, 1968), Dreyfus-Brisac (1966a, 1970), and Parmelee *et al.* (1967, 1968a, b).

B. TECHNIQUE

One of the main problems is to record safely, without any risk for the baby. It is necessary to be aware of the fragility of the prematures and new borns, to reduce the displacements of the neck and of the head of the baby, to avoid any tightness of the head rubber bands, and to place the electrodes as rapidly as possible, after having carefully cleaned the skin.

Recording in incubator, even with assisted ventilation or during an epicranial infusion, is possible. Good earthing helps to get rid of the alternating current artifacts. Heat and oxygen concentration must be watched carefully if the baby is in incubator. All the manipulations must be done through the openings, and the incubator must not be fully open. Transistorized portable apparatus are now available which can be used in the ward so that the baby may remain in his normal environment.

For research studies, recording on a magnetic tape will allow delayed treatment of information. Distant transmission through cables is also possible from the ward to the laboratory, where the magnetic tape and other equipment are available. In such conditions, observation of the baby is made through closed circuit T.V.

The technician must be aware of different dangers: infection (electrodes must be sterilized after each recording, and cables must be kept in a sterile atmosphere); high concentration of volatile products in the incubator; fire (which may occur when volatile liquids are used with a high oxygen concentration and with many electrical apparatus).

Apneas are sometimes caused by prolonged crying, but occur often spontaneously. Monitoring respiration rate (with a strain gauge or with a thermistor, or with impedance measurement) helps detect prolonged apnea, classify states of sleep and study reactivity.

The montage and electrodes vary from one laboratory to another. The number of active electrodes varies from 9 to 12. Different methods are used (see recent reviews by Dreyfus-Brisac 1966b; Ellingson 1967).

1. *EEG patterns*

(a) *The EEG of the full-term new born*

Two main EEG patterns may be described during the first week of life of a full-term new born (Fig. 1, *A* and *D*):

a. Low voltage EEG pattern, which consists of continuous more or less rhythmic activity, with a dominant frequency below 7 c/sec, mainly in theta range, the voltage being inferior to 50 μV. This activity has been described by Samson-Dollfus as *activité moyenne*, and is diffuse all over the scalp.

b. *Tracé alternant* with bursts of slow waves (1–3 c/sec, 50–100 μV) lasting 4–5 sec, synchronously over the two hemispheres. The interburst activity is similar to the low voltage pattern and has the same duration as the bursts.

Besides these two main patterns, two variants can be seen (Fig. 1, *B* and *C*):

c. Low voltage EEG pattern with superimposed slow waves at 2–4 c/sec with an amplitude of less than 100 μV. These slow waves may be continuous or discontinuous.

d. Continuous slow wave EEG pattern, with a frequency of 0.5–2 c/sec and an amplitude higher than 50 μV.

These 4 EEG patterns are recorded in different states. The low voltage or *activité moyenne* EEG being recorded in wakefulness and in active or light sleep; its rhythmicity is somewhat more visible on central areas in active sleep than in wakefulness. Low voltage EEG patterns with superimposed slow waves can be also recorded in active sleep and wakefulness.

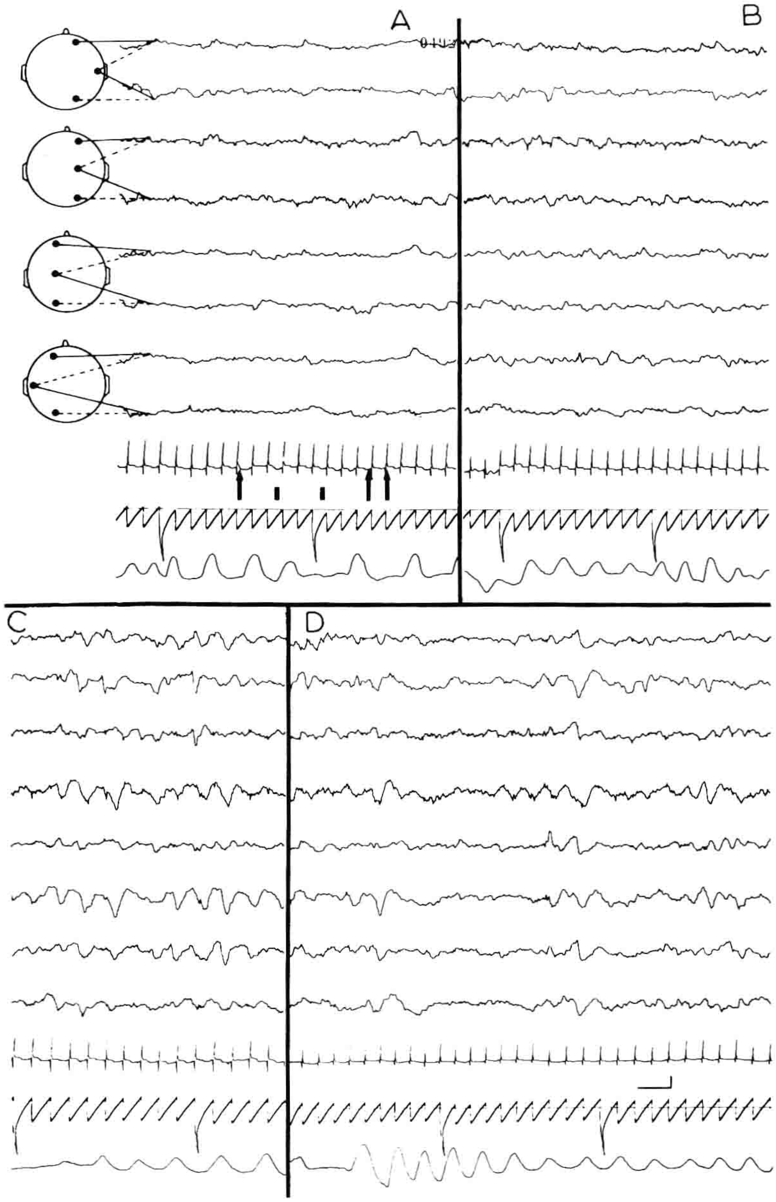

Fig. 1. The four EEG patterns of a normal full-term newborn, 6 days. Term (T): 40 weeks; birth weight (B.W.): 3630 g; height (H): 49 cm; cephalic circumference (C.C.): 36 cm. *A*: Low voltage or *activité moyenne* EEG pattern. *B*: Low voltage EEG pattern with superimposed slow waves. *A* and *B* are recorded in active sleep, with irregular respiratory rate. *C*: Continuous slow wave EEG pattern. *D*: "Tracé alternant". *C* and *D* are recorded in quiet sleep, the respiratory rate is less regular than normal. Black rectangles: Rem; arrows: mouthing. In this figure and Fig. 4, 5 and 6: channel 9 = electrocardiogram; channel 10 = cardiotachygraph, channel 11 = respiration. Calibrations for all figures: 50 μV; 1 sec.

The *tracé alternant* is recorded in quiet or deep sleep, and can be replaced by a continuous slow wave EEG even in normal infants.

Limits of normality are difficult to establish. No clear limits of the normal range of variations in new borns have been described so far. We can nevertheless reach some conclusions (Monod *et al.* 1960; Kellaway and Crawley 1964; Dreyfus-Brisac 1966b; Ellingson 1967). The frontal activity (Fig. 2) occurring synchronously on both

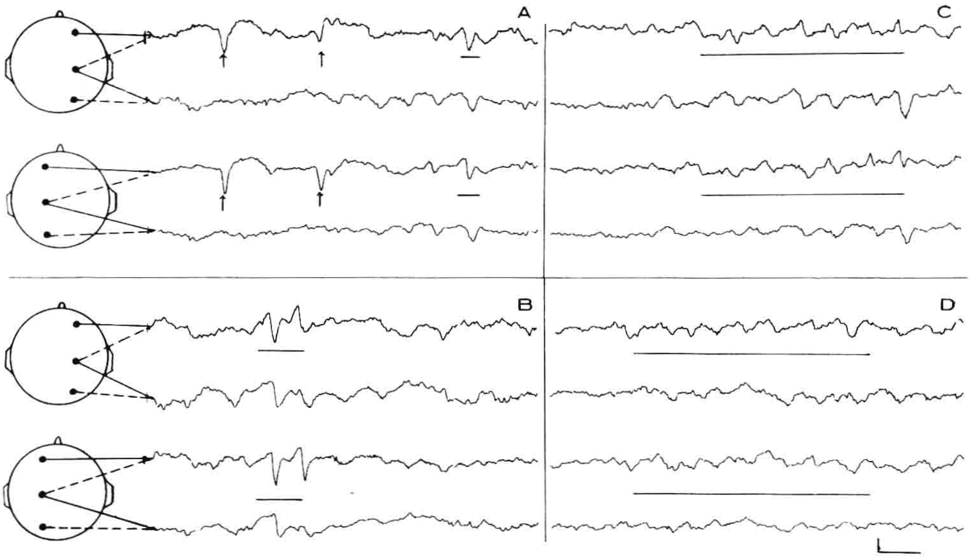

Fig. 2. Slow anterior dysrhythmia (underlined). *A*: Awake, eyes open, one slow diphasic spike (sharp frontal transient), which must be distinguished from eye movement artifacts (arrows). *B*: Repeated sharp frontal transients. *C*: Association of monomorphic delta and sharp frontal transients. *D*: Monomorphic delta waves. (From Dreyfus-Brisac 1966)

hemispheres (either sharp diphasic transients, or monomorphic or polymorphic slow waves occurring either in bursts or continuously) is considered normal in drowsiness or sleep records, but abnormal when occurring in wakefulness. This activity is to be distinguished from ocular movement artifacts (Fig. 2). Low voltage records may be found for one or two days in normal and subnormal babies.

Rhythmicity may also vary and a high degree of rhythmicity in the theta frequency (not in alpha frequency) band in wakefulness is not necessarily abnormal.

In sleep, low voltage, poorly defined vertex spindles (Kellaway 1957; Petre-Quadens 1964; Metcalf 1969), rolandic humps and sporadic slow waves, as well as rapid low voltage occipital rhythms may occur without clear relation to pathological findings (Kellaway and Crawley 1964; Dubois-Dalcq 1966). Transitory asymmetry in wakefulness or sleep may also occur.

Bursts of rhythmic alpha-like activity on parietal or or central leads, occurring during a few (less than 5) seconds are considered normal, and perhaps related to the position of the head during delivery (Churchill 1966).

(b) *The EEG in premature infants*

Rapid development of cerebral hemispheres between 24 and 41 weeks of gestational age is parallel to rapid variations of cerebral electrogenesis. At this period of life, these EEG patterns vary so quickly that an electroencephalographic age can be evaluated within an approximation of 2 weeks.

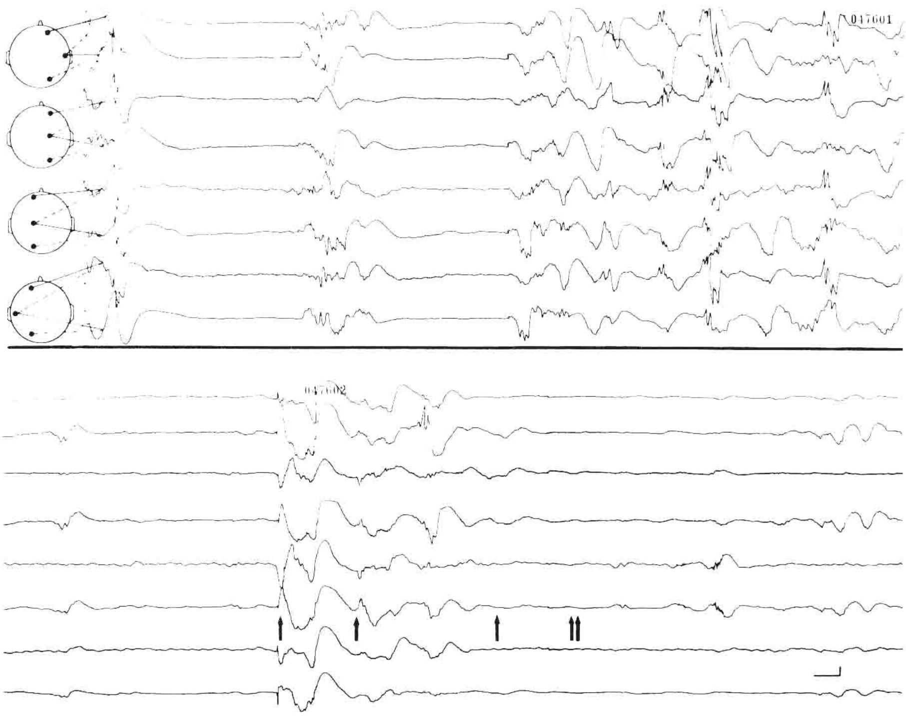

Fig. 3. R ... Premature 20 hours. T: 27 weeks; B.W.: 810 g; H: 32.5 cm; C.C.: 23 cm. Irregular bursts of activity, of variable length and shape, spiky waves mostly on the frontal leads, rapid waves on rolandic leads, slow waves mostly on occipital leads. Period of silence of variable duration. Pulse artifact is seen on leads 5 and 7. Arrows: limb movements.

(i) *The EEG of the early premature infant (24–27 weeks of conceptional age)* (Fig. 3). Only one EEG pattern is present at this early age. Slow waves from 0.3 to 1 c/sec with a voltage of 300 μV occur in bursts lasting 3–20 sec. They are diffuse with occipital predominance. Rhythmic activity in the alpha band between 8 and 14 c/sec appears in bursts of smaller amplitude (25–30 μV) lasting 1–2 sec predominant on the rolandic leads, more rarely on the occipital leads. Bursts of 5–6 c/sec rhythm, lasting 1–2 sec, often predominantly occipital, and spikes of high voltage (200–300 μV) of variable topography, more often frontal, are also seen.

These different types of activity are discontinuous and without regular succession. Bursts may last from 1 to 20 sec. Periods of quiescence between the bursts may last from 2–3 min or only 5–10 sec. There is some disorganization in the timing as well as in the sites of the bursts. In no case were we able to detect isosynchrony between two points in the same hemisphere and the bursts of activity seemed generally to occur simultaneously over the two hemispheres.

Wakefulness and sleep do not seem to be differentiated at this age, though the infant is sometimes quieter or more active, without a total interruption of body mobility.

(ii) *The EEG between 28 and 31 weeks of conceptional age* (Fig. 4). An important modification takes place at or around 28 weeks of conceptional age, when the premature becomes viable. The EEG patterns found before 28 weeks are replaced by much simpler ones. They are composed of bursts of regular rhythmic theta waves (4–6 c/sec), lasting 1 or 2 sec, with voltage between 25 and 100 μV; between bursts there are long quiescent periods.

During a short period of development, at around 28 and 29 weeks of gestation, this theta activity is synchronous at all points on the same hemicranium and cancels itself when electrodes are paired on right or on left side leads. The bursts appear only in recordings made between two electrodes placed on opposite sides of the midline. After 29 weeks of gestation, this intrahemispheric isosynchrony disappears. Until 30 or 31 weeks the bursts of theta rhythms remain the predominant activity. In some cases, slow waves of 0.3–1 c/sec are picked up by the occipital leads from such infants.

Patterns characteristic of 7 months of fetal life (32–36 weeks) appear around 30 weeks. The typical pattern is composed of slow activity (1 c/sec at 25–100 μV) with superimposed rapid rhythms (10–14 c/sec, 10–20 μV): these slow waves appear in bursts lasting 3–10 sec, still mixed with the theta bursts characteristic of the preceding period.

At 30–31 weeks, the EEG patterns are similar in wakefulness and sleep. Wakefulness is not clearly established, and states of sleep are not differentiated (Monod and Garma 1971): there is still more than 60% of discontinuous recording: relationships between EEG patterns and changes in behavior are not yet well established.

(iii) *The EEG between 32 and 35 weeks of conceptional age* (Fig. 5). After 32 weeks, two different EEG patterns are present for long periods.

Slow waves (1–2 c/sec, with superimposed rapid rhythms which were already seen for short periods at 30 weeks) are found on occipital, temporal and also rolandic leads. The bioccipital record is often of low voltage, due to interhemispheric isosynchrony of the occipital areas.

In continuous or discontinuous EEG patterns, wave form is grossly similar, though discontinuous activity is frequently superimposed with sharper waves than continuous activity.

It is important to stress that these different patterns are not correlated with wakefulness and sleep. Continuous activity is present in wakefulness and active (or light) sleep, and discontinuous activity appears only in quiet sleep, though the differentiation between quiet and active sleep is still not fully complete at this age.

(iv) *The EEG of the premature infant between 36 and 41 weeks.* From this age, three different EEG patterns are present: diffuse low voltage EEG patterns, similar to that observed in the full-term new born: continuous slow wave EEG patterns,

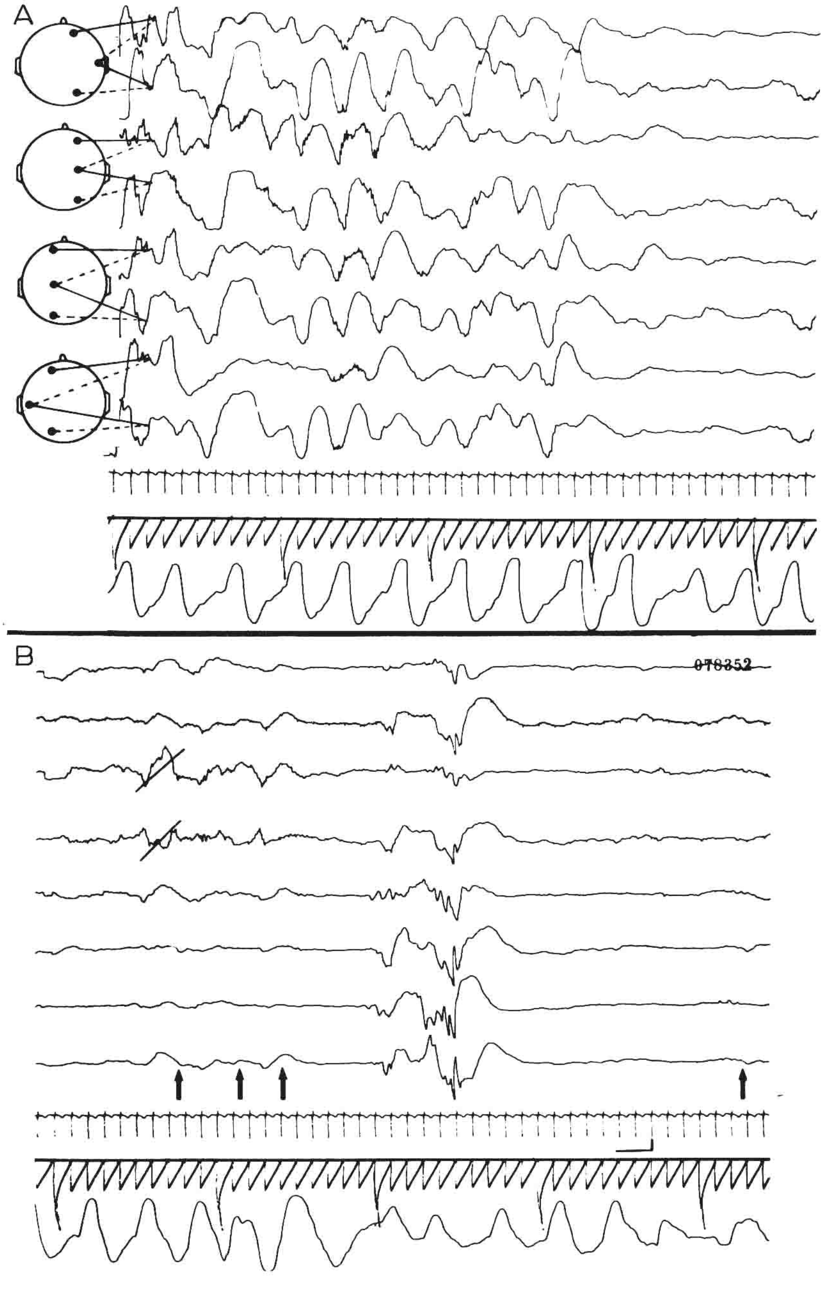

Fig. 4. B ... C ... Premature. T: 29 weeks; B.W.: 1120 g; H: 37 cm; C.C.: 26 cm. First EEG recorded at 5 days, *i.e.*, 30 weeks of conceptional age. Two discontinuous EEG patterns are present at this age. *A*: Slow waves with superimposed rapid rhythms. *B*: Bursts of theta waves, which were already present at 28 weeks. Limb movements (arrows) and irregular respiration are present with a discontinuous EEG. Active and quiet sleep are not differentiated.

A

B

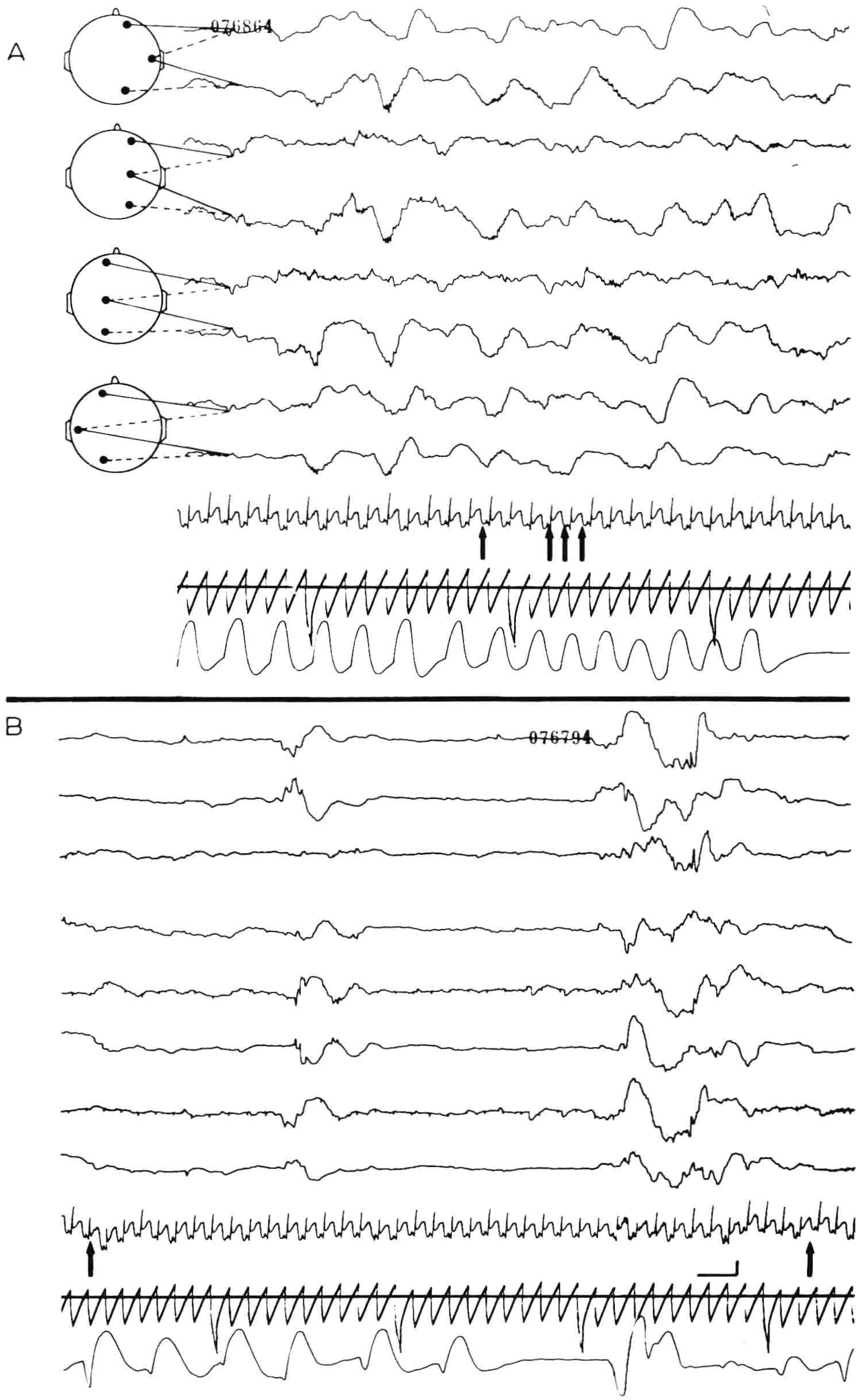

Fig. 5. 33 weeks. Same infant as Fig. 4. *A*: Continuous posterior slow waves in active sleep, with limb motility indicated by arrows. *B*: Discontinuous EEG in quiet sleep. Respiration is still irregular in quiet sleep.