SPECIAL REPORT

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PERS 340   THE ANALYSIS OF SLEEP EEGs IN INFANTS
            AND YOUNG CHILDREN

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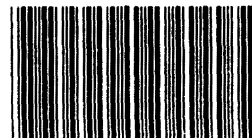
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J INFANTS

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ACKNOWLEDGEMENTS

SUMMARY

The following are discussed in relation to the sleep EEGs of infants and young children:

1. the normal patterns of sleep and changes in them, during the first few months of life;

2. the characteristics of normal paroxysmal (arousal) phenomena, drowsy hypersynchrony and encoches frontales;

3. criteria for abnormality;

4. some recording considerations.

OPSOMMING

Die volgende is bespreek ten opsigte van die slaap-EEGs van babas en jong kinders:

1. die normale slaap patrone en veranderinge gedurende die eerste paar maande;

2. die eienskappe van normale paroksismale tekens van opwekking, hipersinchronie gedurende duiseligheid en encoches frontales;

3. kriteria om abnormaliteit te bepaal;

4. oorwegings in verband met EEG-opname.

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There is general agreement that the analysis of the EEGs of sleeping infants and young children is fraught with hazards, particularly for the unwary and inexperienced. Many factors contribute to the difficulties encountered. One, for example, is the immaturity of the central nervous system. The rapid development of the cortex is reflected in the marked changes that occur in the electrical activity recorded by the EEG, particularly during the first few months of life. By its nature, also, the recording situation adds to the complexities of analysis. Children and infants who are unable or unwilling to comprehend what is required must be sedated. The alternative to this is to wait until natural sleep supervenes. However, in a busy clinical practice this is not always feasible, and some form of sedation is usually necessary. Many of the commonly used sedatives produce beta (fast) activity in the EEG which is generalised and usually maximal in the fronto-centro-temporal area. (Figure 1). Although this can be predicted and allowed for during EEG analysis, very often the combination of drug-induced beta activity, and the fast (spindle) activity normally occurring during sleep leads to the production of activity of sharp-tipped (spike) appearance. (Figure 2). Differentiation between the latter and epileptiform activity is not always possible. Further, sedating the child to induce sleep which is rapid may suppress epileptiform discharges. It is not always possible to judge the optimal sedative dosage with precision. These factors excepted, however, the use of sedation to induce sleep in children in the clinical EEG laboratory is an accepted and widespread practice, based on the demonstrated lack of significant differences in EEG patterns resulting from this practice and those during natural daytime sleep. A prime difficulty lies in distinguishing between the normal phenomena (paroxysmal) of sleep, and epileptogenic activity. This implies that some sort of knowledge of what is normal in the sleep EEGs of children is necessary.

W H A T   I S   N O R M A L ?

A.   SLEEP PATTERNS

FIGURE 1   Very considerable beta activity induced in a
                   sedated male, aged 14 months, referred for EEG
                   examination.   Probably Stage 1 sleep.
                   Patient was not on medication.   Sedation was
                   induced by 1 Vesparaxette suppository.

FIGURE 1

TC O,

CAPD

FO

100μV

1 sec

FIGURE 2   The combination of beta activity (high voltage)
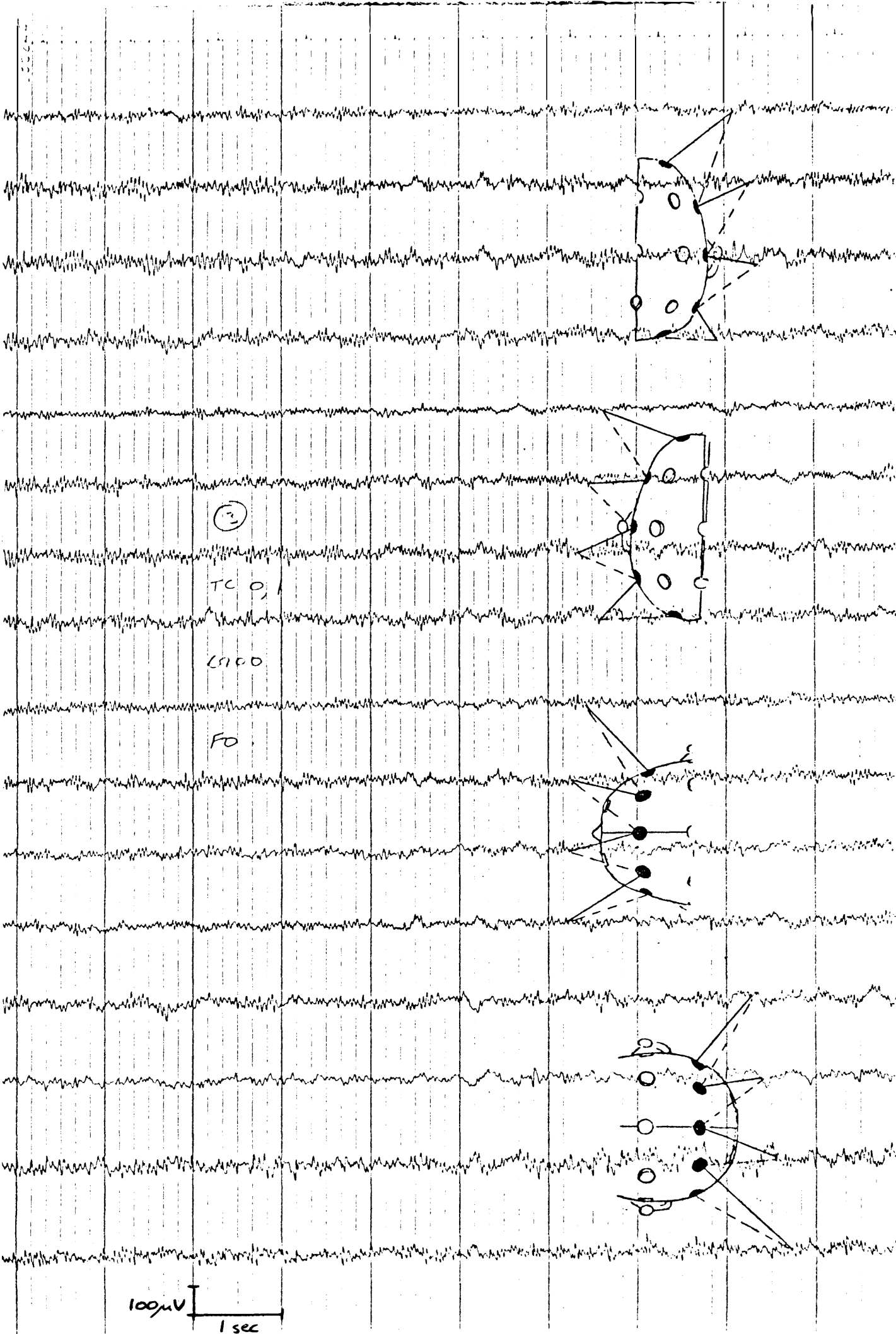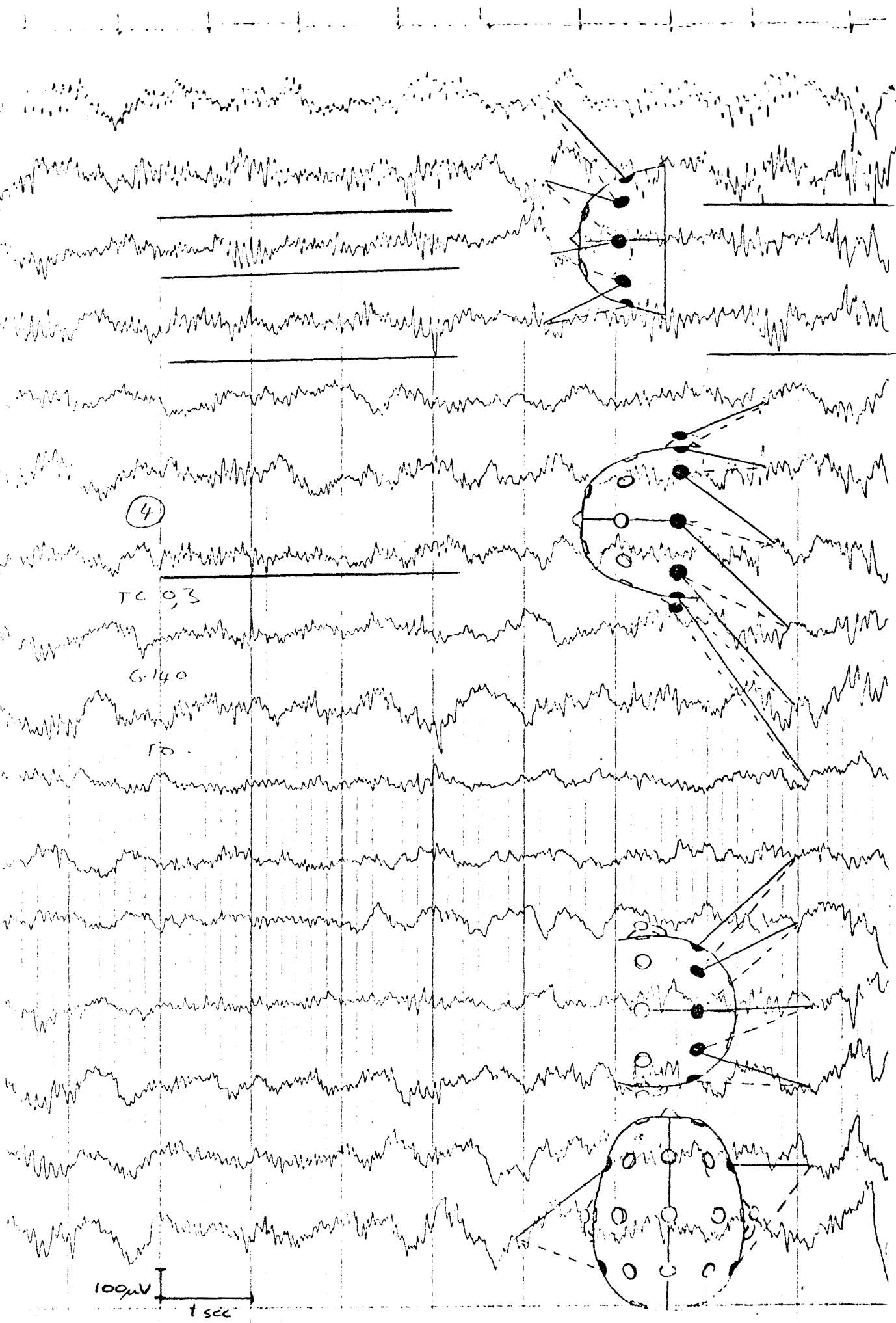   induced by sedation and sleep spindles particularly
                    in frontal areas produces a spike-like effect.
                    The patient was a male, 21 months, sedated by a
                    Vesparaxette suppository.
                    Normal EEG.

FIGURE   2   5.

FIGURE 2

By the 8th month of intra-uterine life the EEG reflects changes related to the sleep/wakefulness cycle. Increases in amplitude of the slowest waves of the EEG, particularly in the posterior cortical areas, occur during sleep. At term, Anders and his coworkers (1971)[1] suggest that 3 sleep states are distinguishable, and have provided criteria for their scoring. Briefly, these are as follows:

1. ACTIVE SLEEP (AS). This corresponds to REM sleep in the adult. Activity prevails during this state. The infant's respiration is irregular, his facial expressions change, and digit, limb and body movements occur. Chin EMG is absent. REMs are present. The EEG shows low voltage irregular fast theta activity (dominant frequency of 5 - 8 Hz). This may be superimposed on high voltage slow activity, or the latter may rarely be seen alone.

2. QUIET SLEEP (QS). The adult equivalent of this is slow wave sleep (SWS). Behavioural quiescence with no body movements is typical. Chin EMG is increased. Respiration is regular and REMs are absent. The EEG shows trace alternant or high voltage slow activity (0,5-3 Hz). (Figure 3). Trace alternant is characterised by bursts of HVS, at times intermixed with sharp waves, with periods of intervening relative quiescence.

3. INTERMEDIATE SLEEP (IS). The characteristics of this state do not permit classification as either AS or QS. This is also termed transitional sleep, and occurs at sleep onset, when states are changing, or during arousal.

Ellingson and Peters (1980a)[2] suggest that in normal infants the following maturational changes occur during daytime sleep in the first few weeks of post-term existence.

1. Trace alternant activity disappears. This pattern is seen in all of the EEGs of newborn infants, but disappears entirely by 6 weeks.

FIGURE   3   High voltage generalised slow activity typical of
                      quiet sleep in a female aged 8 weeks (sedation
                      by means of Vesparaxette suppository).
                      AS is an abbreviation for auditory stimulus.

FIGURE 3

100μV

1 sec
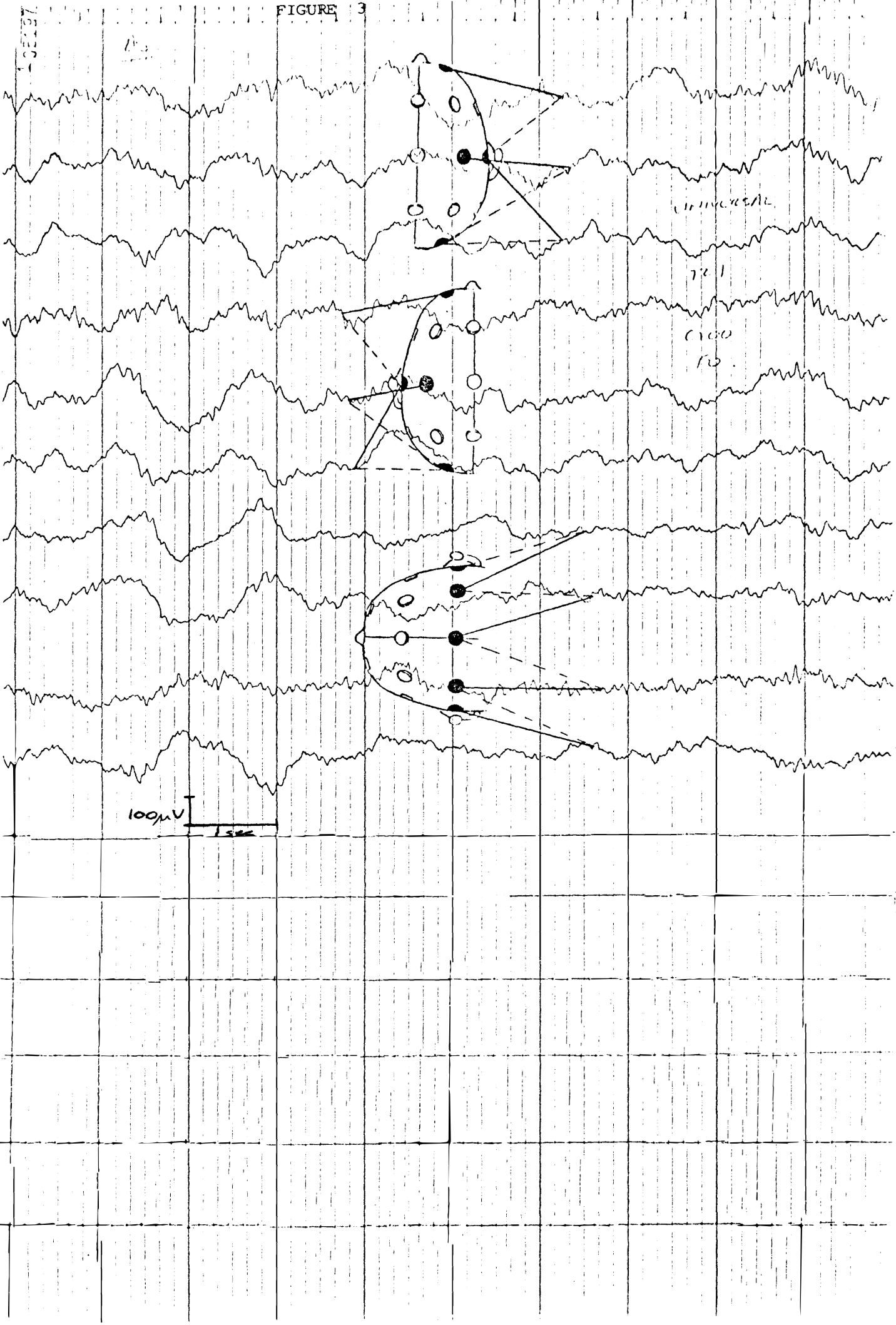
2. <u>AS onset is replaced by QS onset.</u>   AS is the first sleep stage
    in about 80% of episodes in the first 3 weeks of life, but in
    only 5 - 10% by the 3rd month of life.

3. <u>Decrease in amount of AS.</u>   During the first week of life, AS
    occupies about 50% of total sleep time, QS less than 50% and IS
    about 13%.   By 8 weeks, AS has decreased to 20%, and QS has
    increased proportionately.   Adults likewise spend about 20% of
    TST in REM sleep.

These findings emphasise the importance of the first two months of life
as a period of rapid maturational flux in brain function.   The
importance of this period is further underscored by the results of a
recent study by Crowell and his coworkers[3], published at the beginning
of this year.   They found that infants at 3 months post-term have EEG
features in daytime sleep, which together with EOG and EMG activity,
signify the presence of sleep stages and their organisation resembling
those in adults.   Sleep regulatory mechanisms thus approach a level
of functional maturity at 3 months in the human infant.   This has
implications for the clinical electroencephalographer, as a mature
sleep pattern has usually been considered to emerge only at a much
later age.

B.   <u>SPECIFIC SLEEP PHENOMENA (INCLUDING AROUSAL RESPONSES)</u>

The characteristics of normal sleep phenomena are shown in Table 1.
It must be emphasised that the specific phenomena of sleep spindles,
vertex sharp transients, and K-complexes, the latter 2 referred to as
arousal responses, are all normal.   (Figure 4).   Of relevance to the
clinical electroencephalographer is that sleep spindles may show
asymmetry and asynchrony between hemispheres well into childhood and
still be normal.   The development of sleep spindles can be seen in
Figures 5 - 8.   Vertex sharp transients appear with all degrees of
sharpness in both normal and epileptic groups.   Thus, even if their
presence is characterised by high amplitude and spike-like format,

<u>TABLE 1</u>

<u>CHARACTERISTICS OF NORMAL SLEEP PHENOMENA</u>

	SLEEP SPINDLE	VERTEX SHARP TRANSIENT	K-COMPLEX
Alternative Names	Sigma activity	Vertex Sharp wave Biparietal hump Bicentral transient V wave Evoked negative sharp waves on the vertex Startle wave	K wave
Description	Run/group of rhythmic waves of progressively increasing and then decreasing amplitude. Waves are monomorphic and symmetrical with respect to baseline	Monophasic/triphasic sharp waves	(1) Sharp wave, followed by (2) slow wave, followed by (3) spindle burst
Frequency	12 - 14 Hz	4 - 20 Hz	(1) 3 - 8 Hz (2) 2 - 2,5 Hz (3) 12 - 14 Hz
Amplitude	'low voltage'	250μV	(1) 50 - 150μV (2) 100 - 250μV (3) 25 - 100μV
Total Duration	0,5 - 1,8s	Transients, some bursts	0,5 - 4s
Location	F, C or P Midline phasereversal	Vx F (children) Phasereversal about Vx	Vx Midline phasereversal
Stage of Sleep	'Light sleep' QS	Stage 1 or 2 'Light sleep' QS	Stage 2 and deeper
Stimulus Relation	Attenuated by auditory stimuli	Elicited by stimuli or spontaneous	Elicited by stimuli or spontaneous. Not sensory specific. Evoked indefinitely if interstimulus interval + 3s. All or none. Better if stimulus meaningful
Appearance	Birth - weak 3 - 9wks - clear 3mo - well-established	Birth 5 - 6mo - well established	5 - 6mo
Symmetry/ Synchrony	At first asymmetrical. By 3mo 49% of bursts symmetrical & synchron. Some asymmetry to 4yrs.	Symmetrical	
Developmental Tendency	Maximal abundance at 4-6mo, minimal 27mo, constant to 54mo, incr. to 60mo. Duration of bursts & frequency decrease with age	Maximal amplitude and abundance 2 - 12 yrs	Sharpness of (1) increases with age. Vx dominance establ at 1,5 yrs. Spread to surrounding areas decreases

FIGURE 4   Stylized representation of EEG waveforms
                   specific to sleep.

FIGURE 4   12.
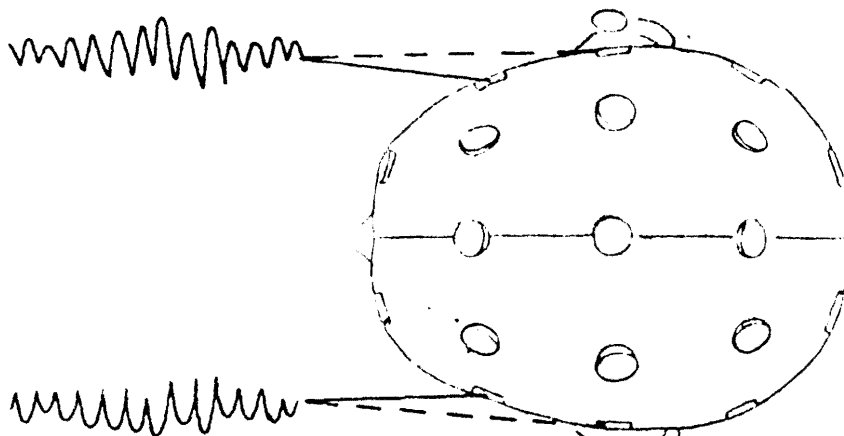
SLEEP SPINDLES

VERTEX SHARP TRANSIENTS

K-COMPLEXES

100μV

1 sec
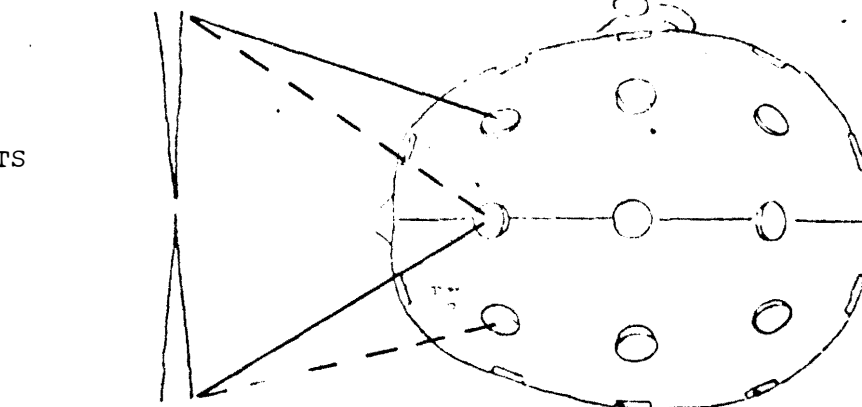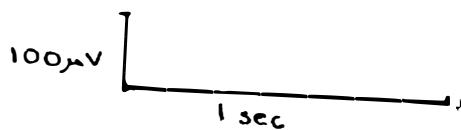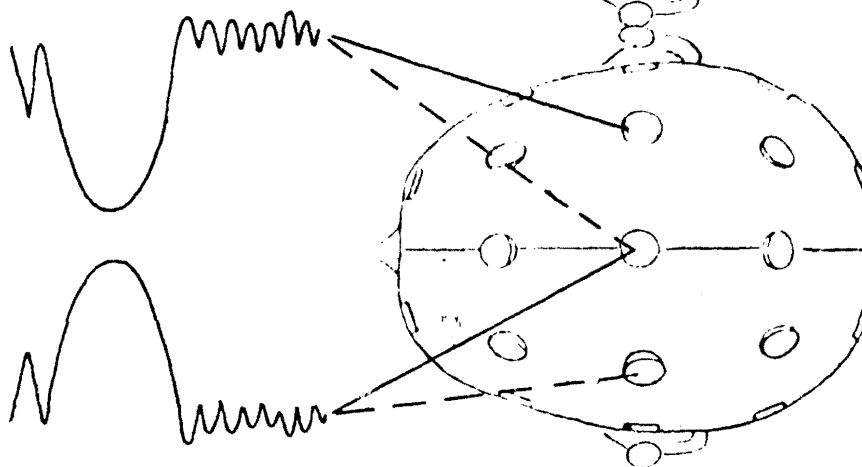
FIGURE   5   Male, aged 8 weeks, sedated with Trichloryl.

Poorly-developed sleep spindles are shown.

Poor development of this waveform is seen in

the excessive length of the spindles(upto 3,5

seconds), a pronounced interhemispheric

asymmetry with activity greater on the left and

a relatively high frequency (14 Hz).

FIGURE 5   14.

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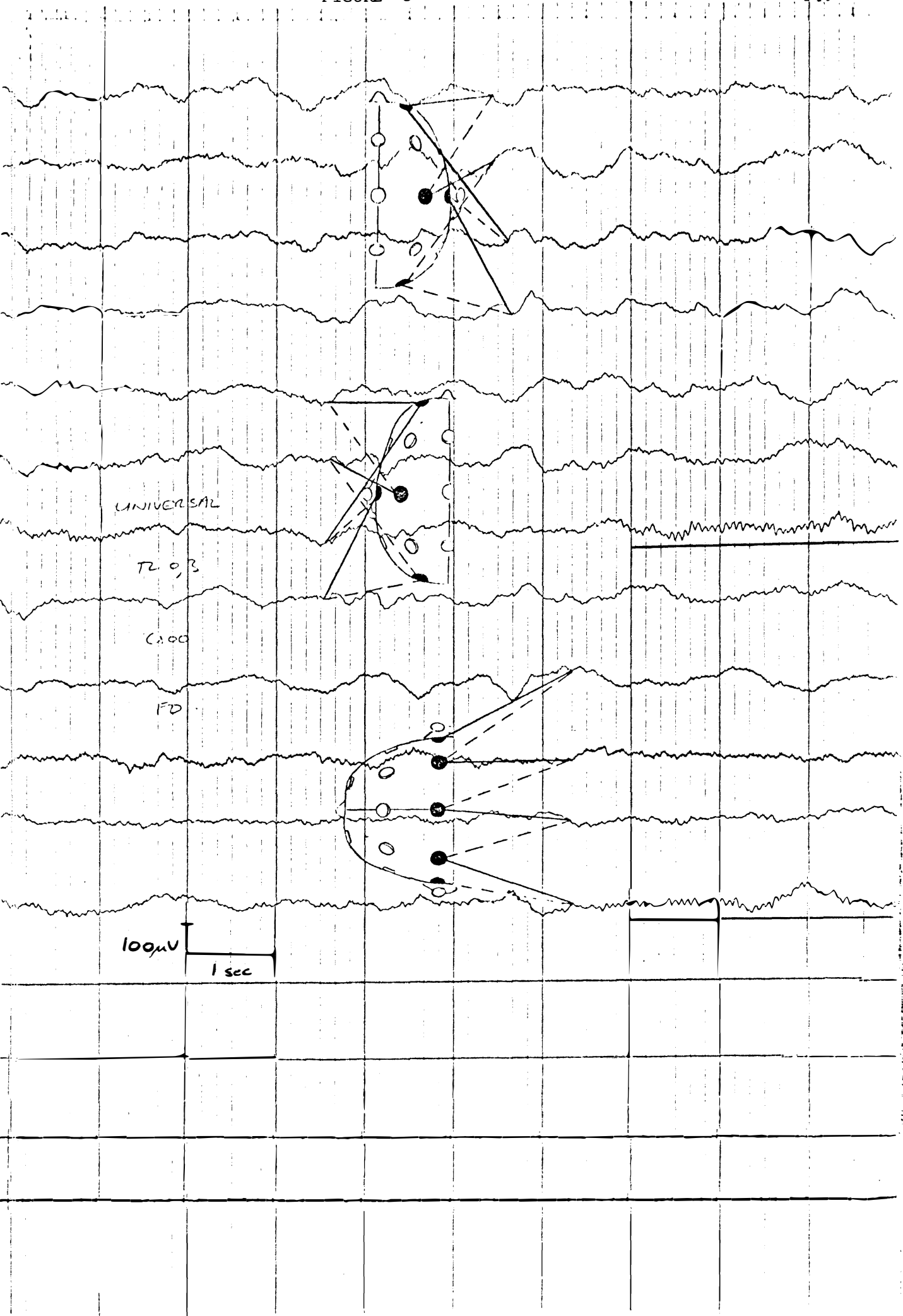
FIGURE 6   Spindle activity in a male, aged 8 months.
A more mature spindle form is shown with a
relative symmetry of activity between hemis-
pheres, in greater amplitude, shorter burst
duration and a frequency of 13 Hz.
Compare these parameters with those of
Figure 5.

FIGURE 6

16.

100μV

1sec
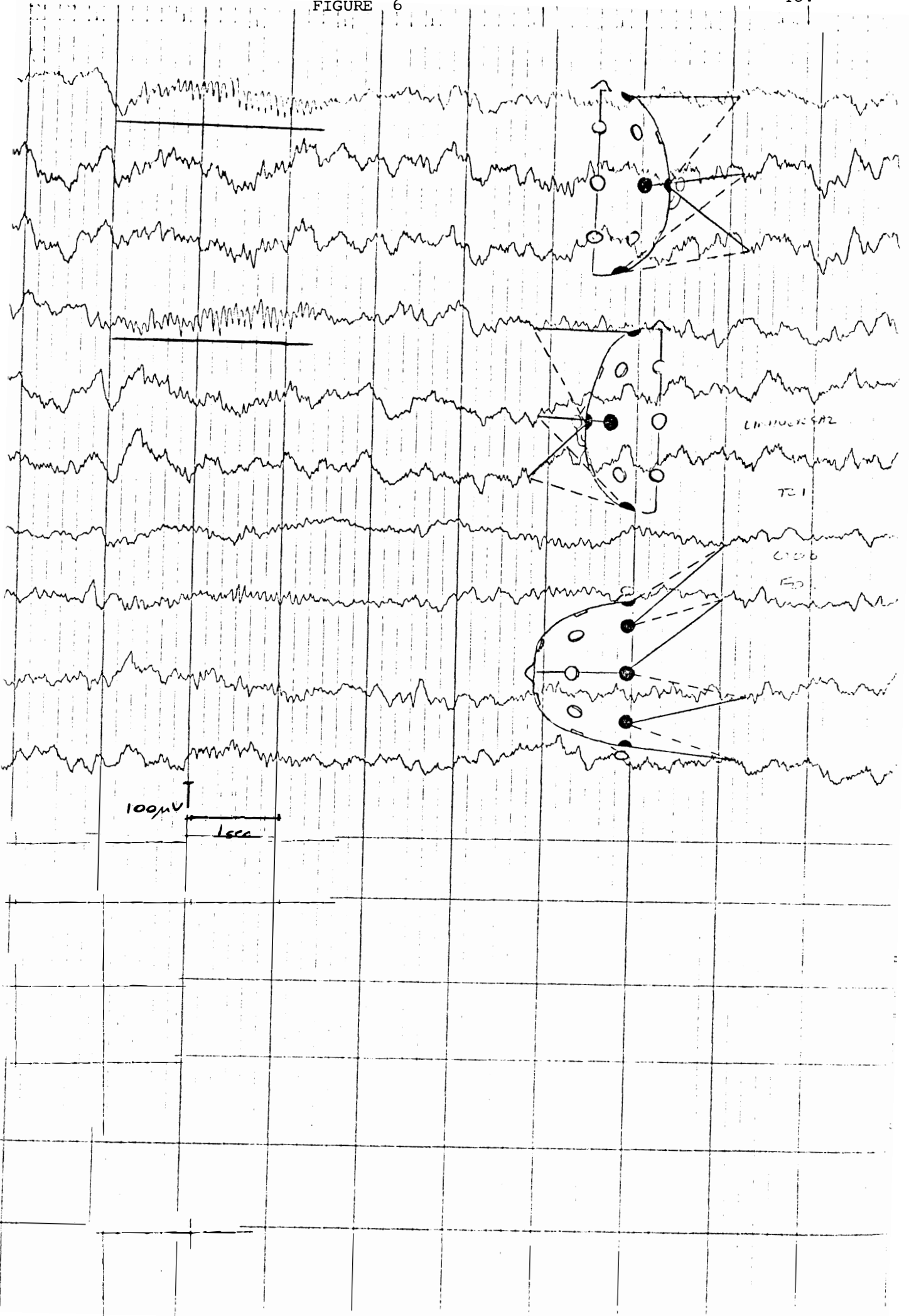
FIGURE   7   Spindle activity in a male, aged 17 months.
Well developed spindles of short duration,
frequency of 13 Hz, high degree of symmetry
and relatively high voltage are shown.
Compare with Figures 5 and 6.

FIGURE 7

100µV

1 sec
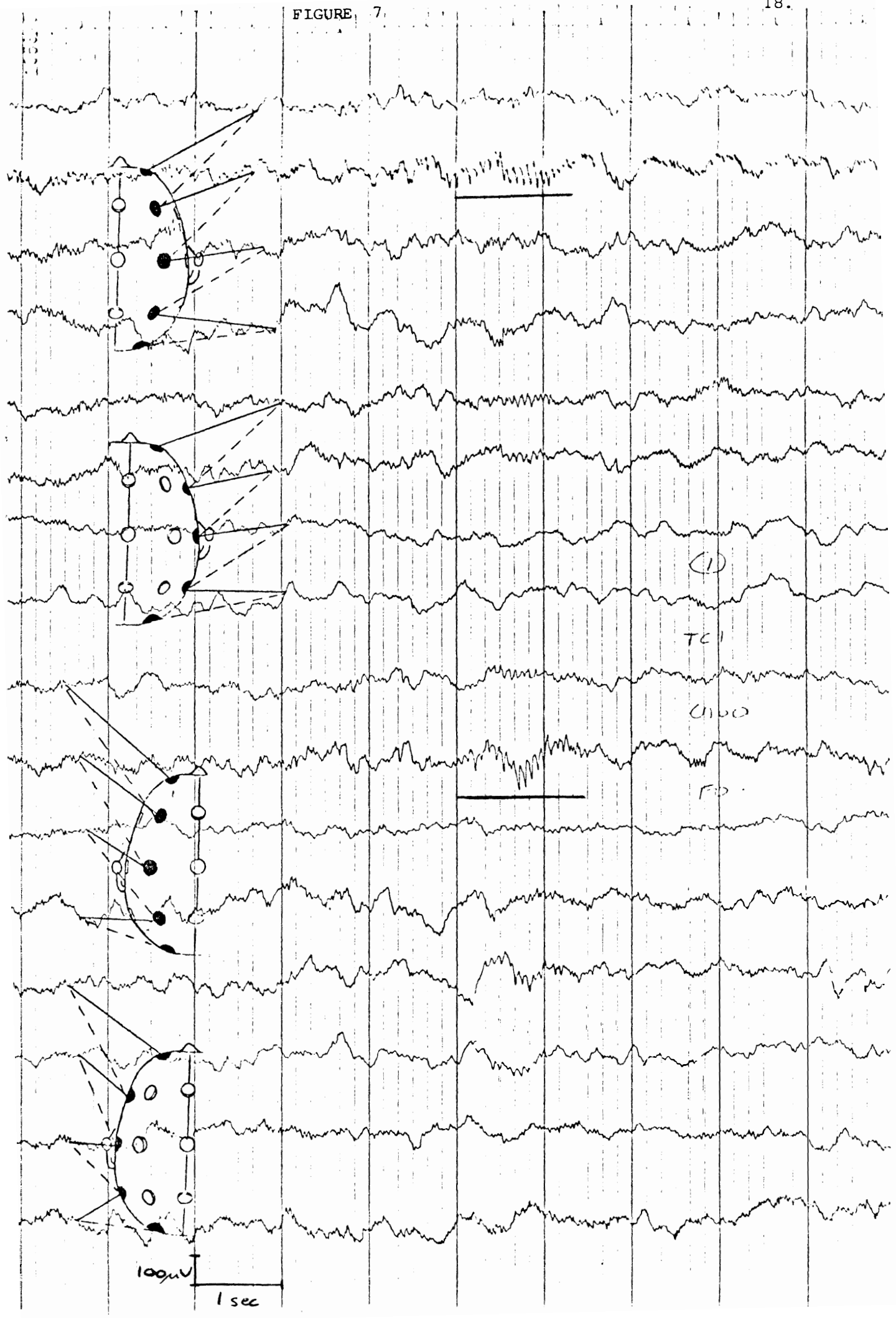
FIGURE 8   Natural sleep in a male, aged 3 years 6 months. Short spindle bursts, symmetrical, with a high voltage and a frequency of 11 Hz occur. (Regular medication - Epanutin).

FIGURE 8   20.

100μV
1 sec
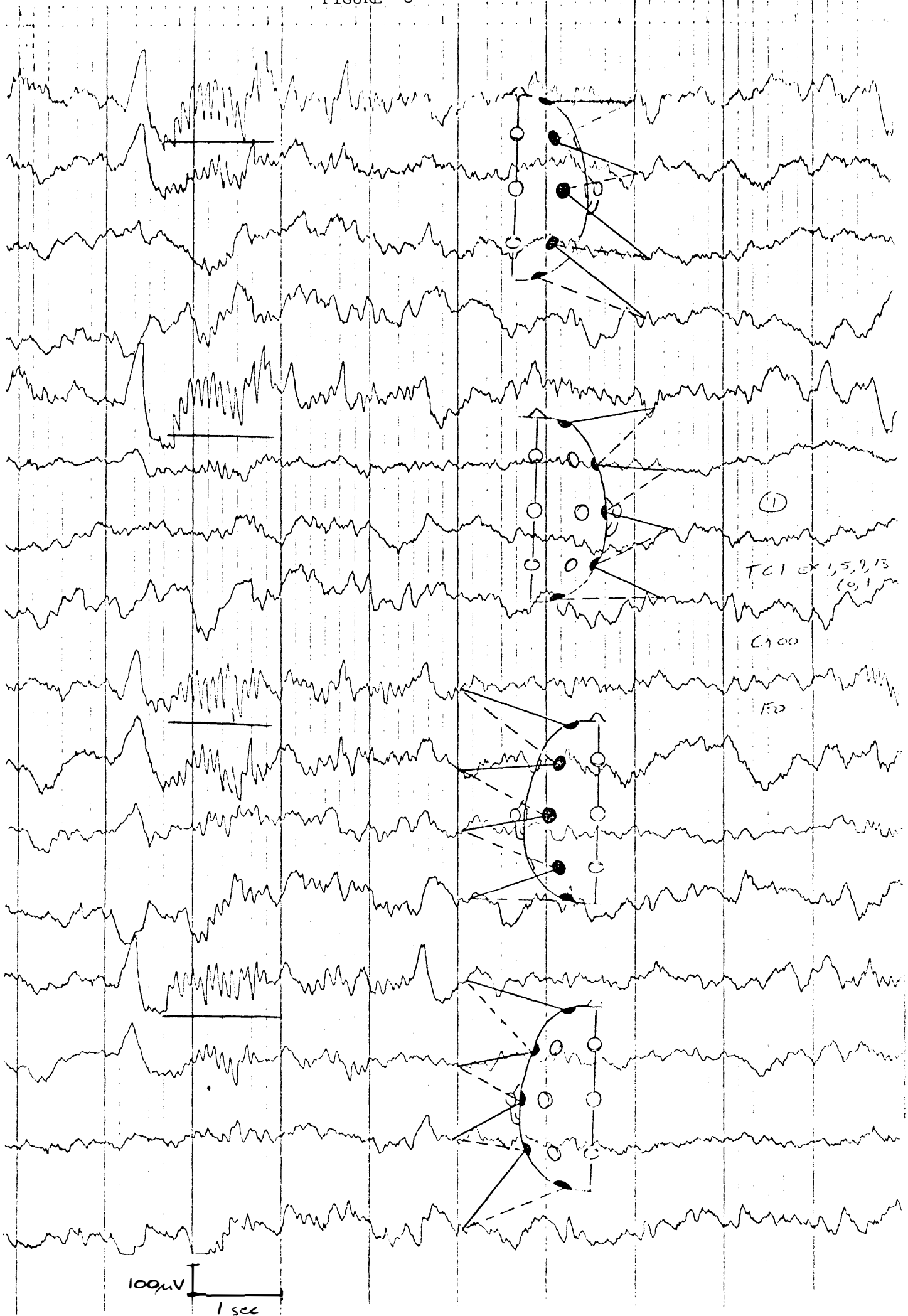
they should not be described as abnormal. Further, as the frequency,
amplitude and paroxysmal morphology of K-complexes may easily be
confounded with those of spike and wave activity, caution should be
exercised in describing paroxysmal activity of this type as abnormal.
(Figures 9 - 14). Of relevance to the decision as to whether burst
or paroxysmal activity during sleep is abnormal, is the midline phase-
reversal, and, in most cases, maximal amplitude affinity for the vertex,
that these phenomena share. Thus, if a midline phase-reversal and/or
vertex origin can be demonstrated for EEG activity of this type, a
valid rule of thumb would appear to be to regard it as normal. This
is demonstrated in Table 1 under location.

C.   DROWSY HYPERSYNCHRONY

This, high voltage slow activity, often paroxysmal, may be associated
with either pre- or post-sleep drowsiness.   (Figure 15).   It occurs
in children up to the age of 5-6 years, and is maximal at the age of
1-2 years.   It may be seen in children who, from all outward signs,
appear awake.   This is a potential error in interpretation, as
paroxysmal activity in an awake child is usually regarded as epileptogenic.
This type of activity should therefore be considered as normal unless
definite spikes appear in association with it.

D.   ENCOCHES FRONTALES

It has been shown (Ellingson and Peters, 1980a)[4] that random focal and
multifocal sharp waves and spikes, often involving the frontal areas
(the so-called 'encoches frontales') are frequently seen in the EEGs of
full-term newborns.   (Figure 16).   This form of activity should
therefore be interpreted conservatively.   It should only be regarded
as abnormal if it is highly repetitive or consistently focal.   If
this is so, this may signify an epileptogenic process.

FIGURE 9   Sleep in a female aged 7 weeks.   A weak
                   (poorly developed) vertex sharp transient
                   occurs.   Note the midline phase reversal
                   and the relatively slow frequency of the
                   transient.

FIGURE   10   Male, aged 8 weeks, sedated with Trichloryl.
                     Two poorly-developed vertex sharp transients
                     occur after auditory stimulation (clapping of
                     hands).

FIGURE 10   25.

CLAP
HANDS

UNIVERSAL

Tc 9,3

(100

TO

100μV
1 sec
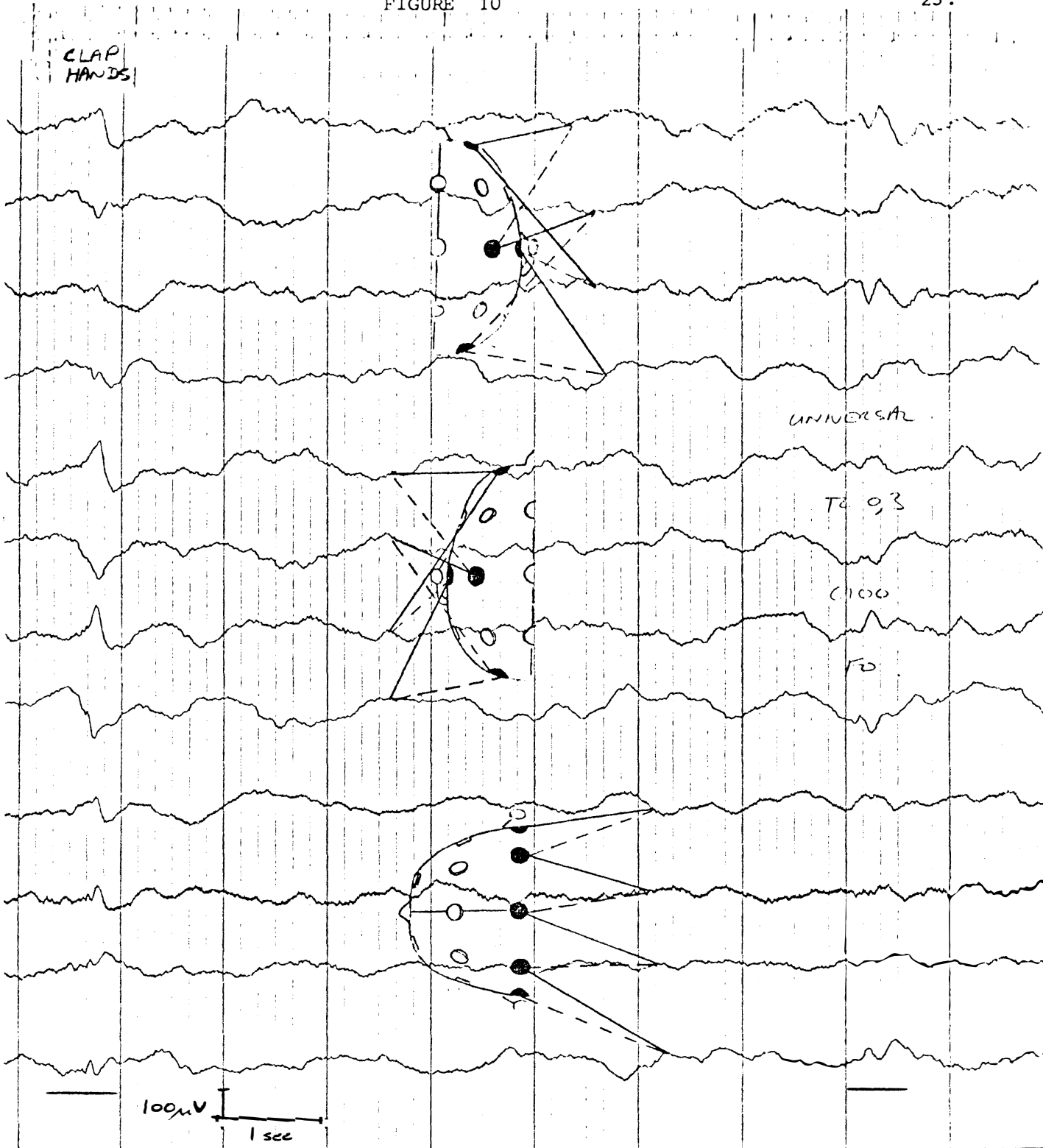
FIGURE 11   Sleep EEG in a male aged 16 months.   A normal
                    well-developed vertex sharp transient maximal
                    in the fronto-central areas.

FIGURE   11

100μV

1 sec
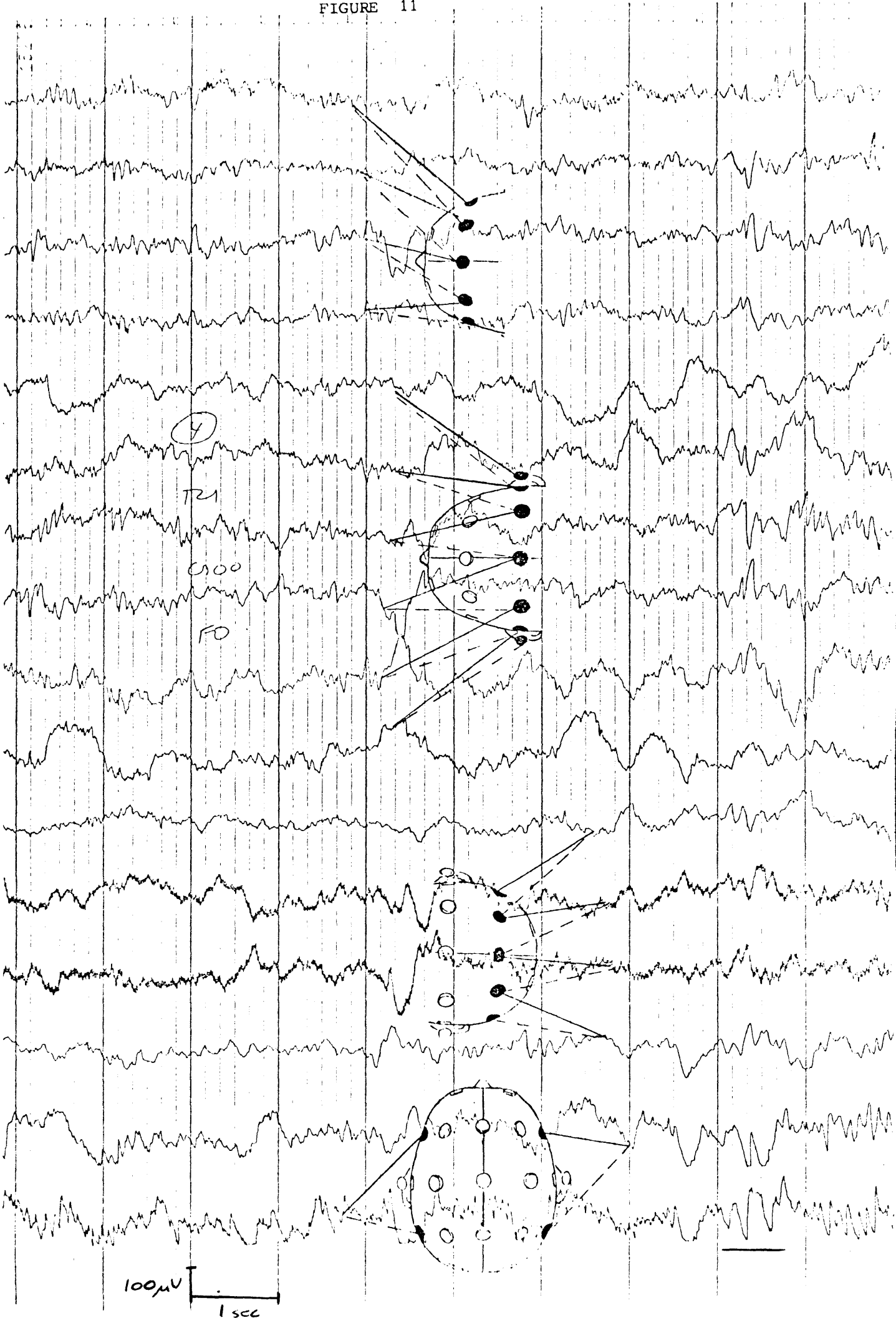
FIGURE 12   Sleep EEG in a male aged 18 months.   A well-
                    developed vertex sharp transient is shown phase
                    reversed over the midline in the frontal areas.

FIGURE 12

TC 1

C100

FO
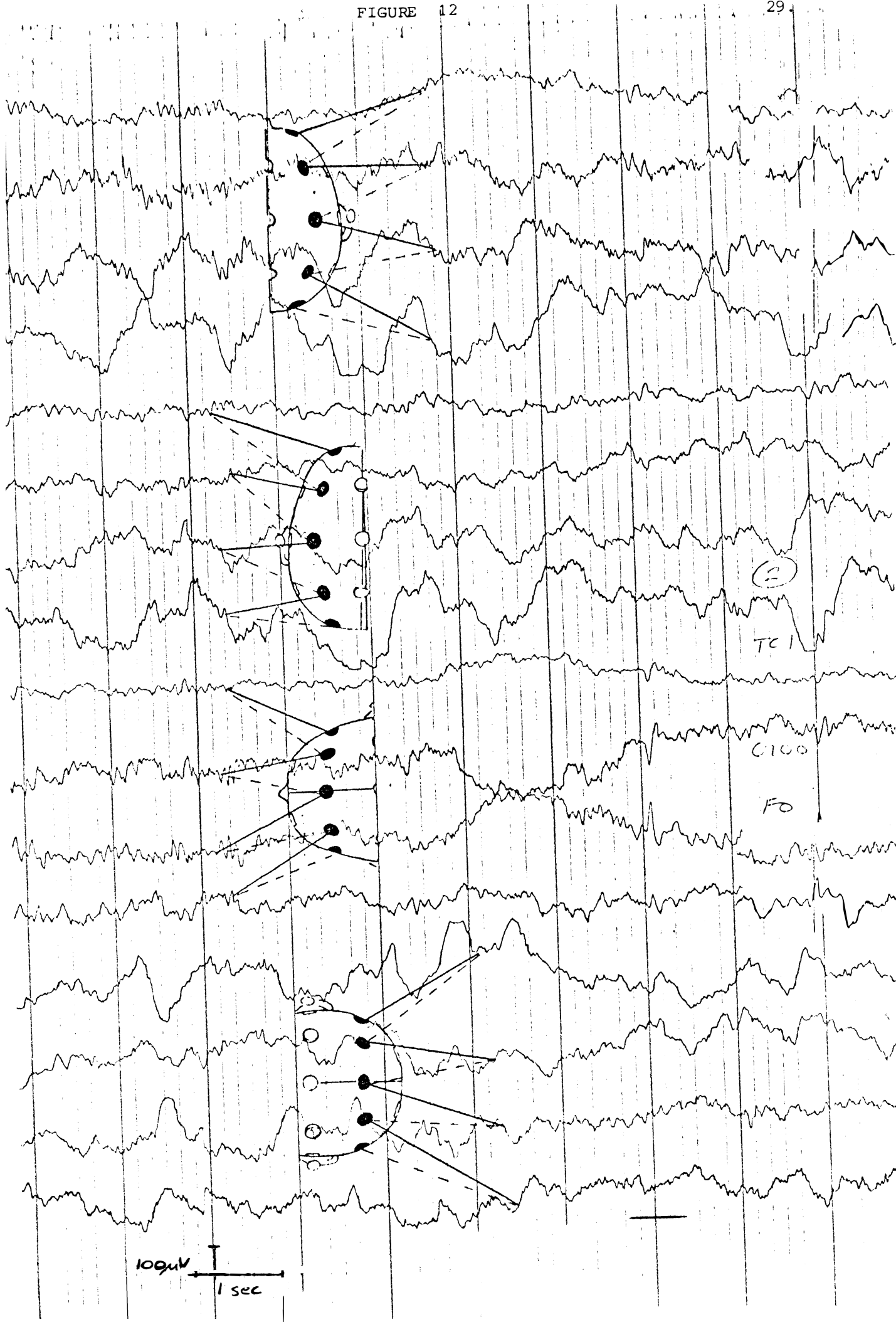
100μV

1 sec

FIGURE 13   Sleep EEG (Vesparaxette) in a male, aged 16
                    months.   A distinct K-complex in response
                    to calling the patient's name is shown.
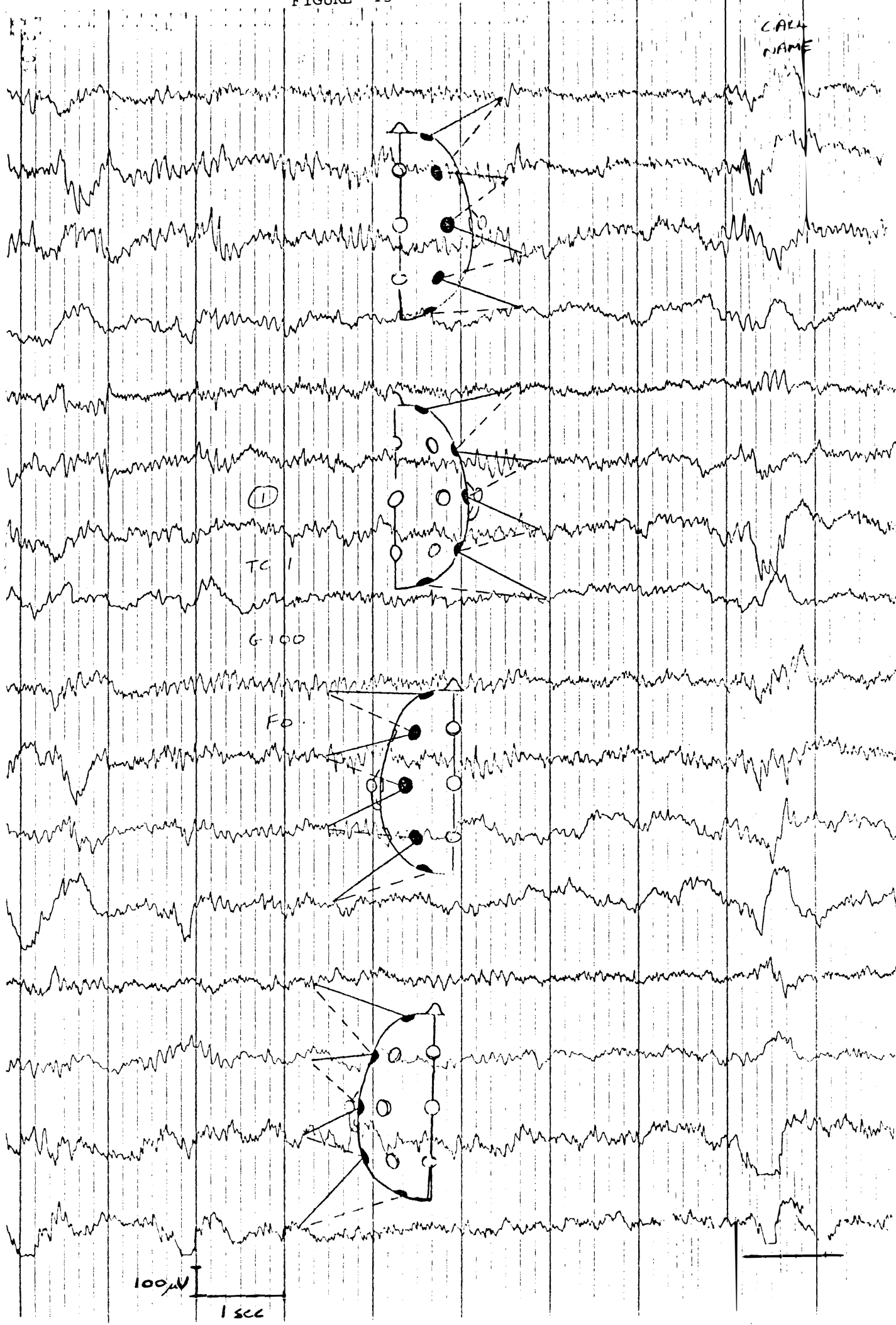
FIGURE   13

100 μV

1 sec

FIGURE 14   Sleep EEG (Trichloryl) in a male, aged 3 years
                6 months.   A K-complex in response to hand
                clapping is shown.   Note the distinctiveness of
                this phenomenon in relation to the surrounding
                activity and the sharpness of the initial
                component.

FIGURE   14   33.

CLAP
HANDS

100μV

1 sec
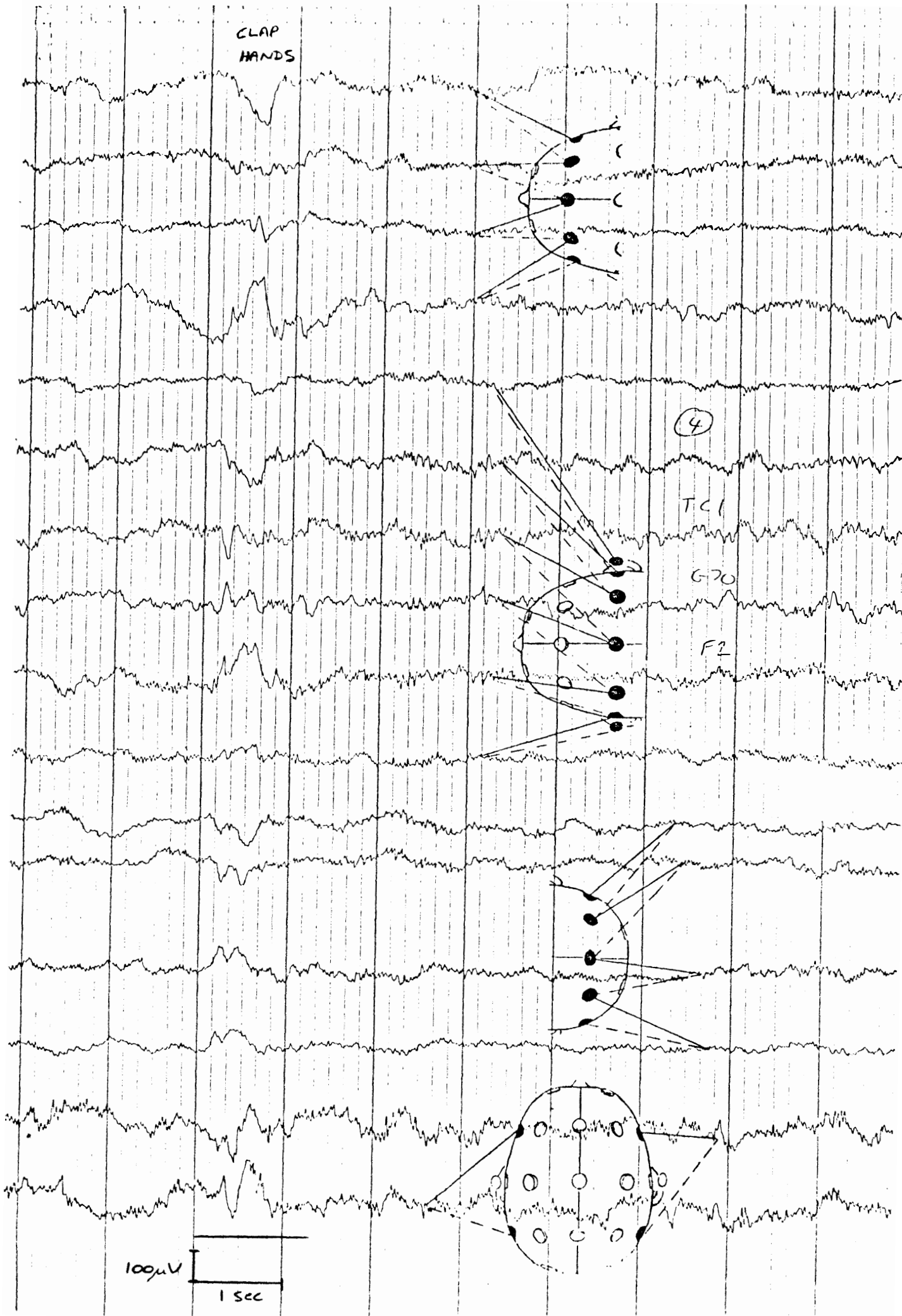
FIGURE   15   Female, aged 15 months, immediately after arousal

from sleep induced by Vesparaxette.

Hypersynchronous high voltage delta activity is

shown while the patient is drinking milk.

Normal.

FIGURE 15   35.

DRINKING MILK - DROWSY

100μV

1 sec

FIGURE 16   Photic stimulation in a sleeping male, aged 17
                    months.   Frontal sharp transient (Encoches
                    Frontales) of higher voltage on the left occur.
                    Normal.

FIGURE   16   37.

PHOTIC

(I)

TG I

(100)

F0

100μV

1 sec

W H A T   I S   A B N O R M A L ?

Dreyfus-Brisac and Curzi-Dascalova (1975)[5] state, as a general orientation: "From 3 to 12 months the presence of abnormal figures (focal spikes of any location, paroxysmal patterns of any kind) is very rare". (p6B - 27B). In spite of this, however, certain EEG patterns have been described as abnormal. A list of these is offered.
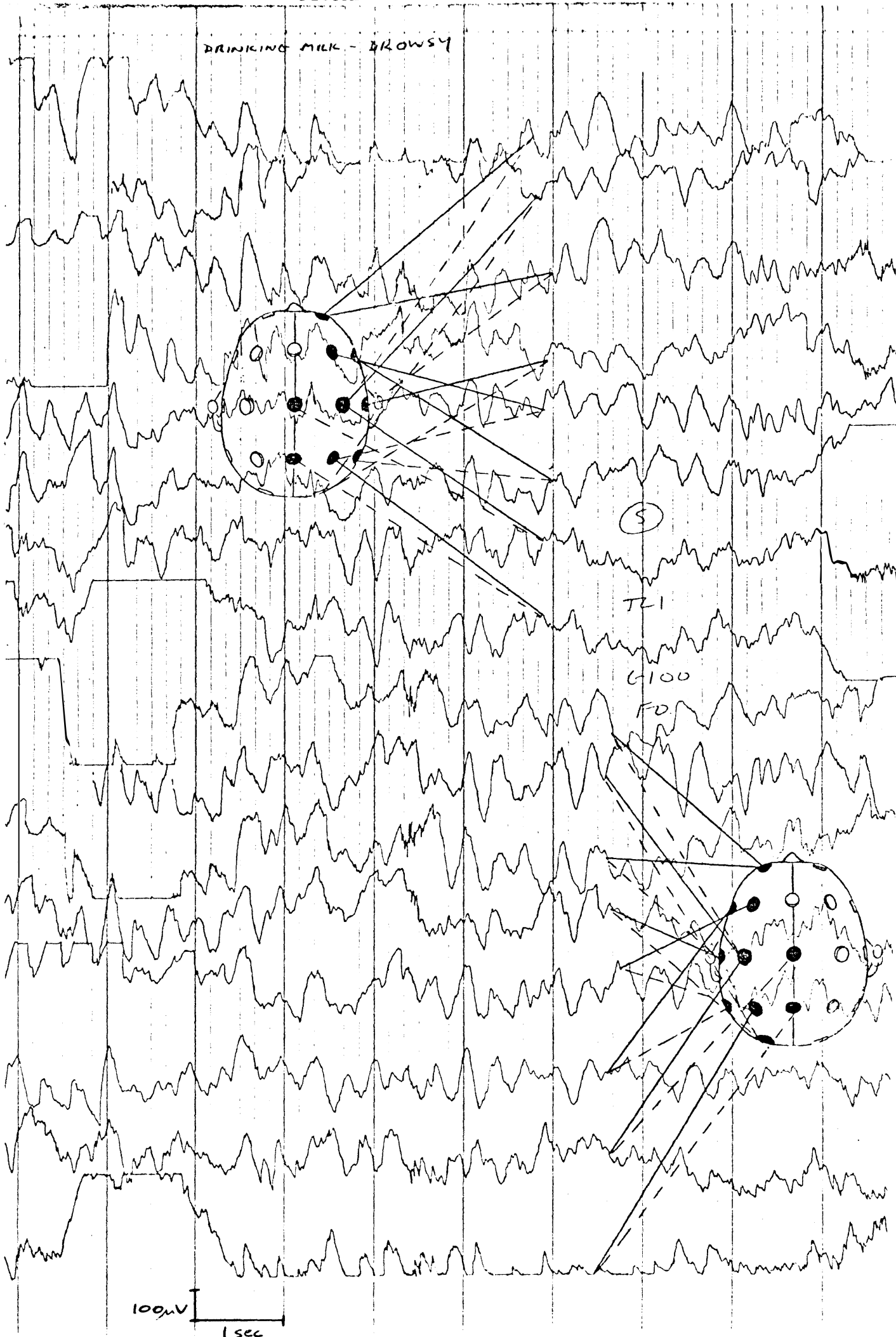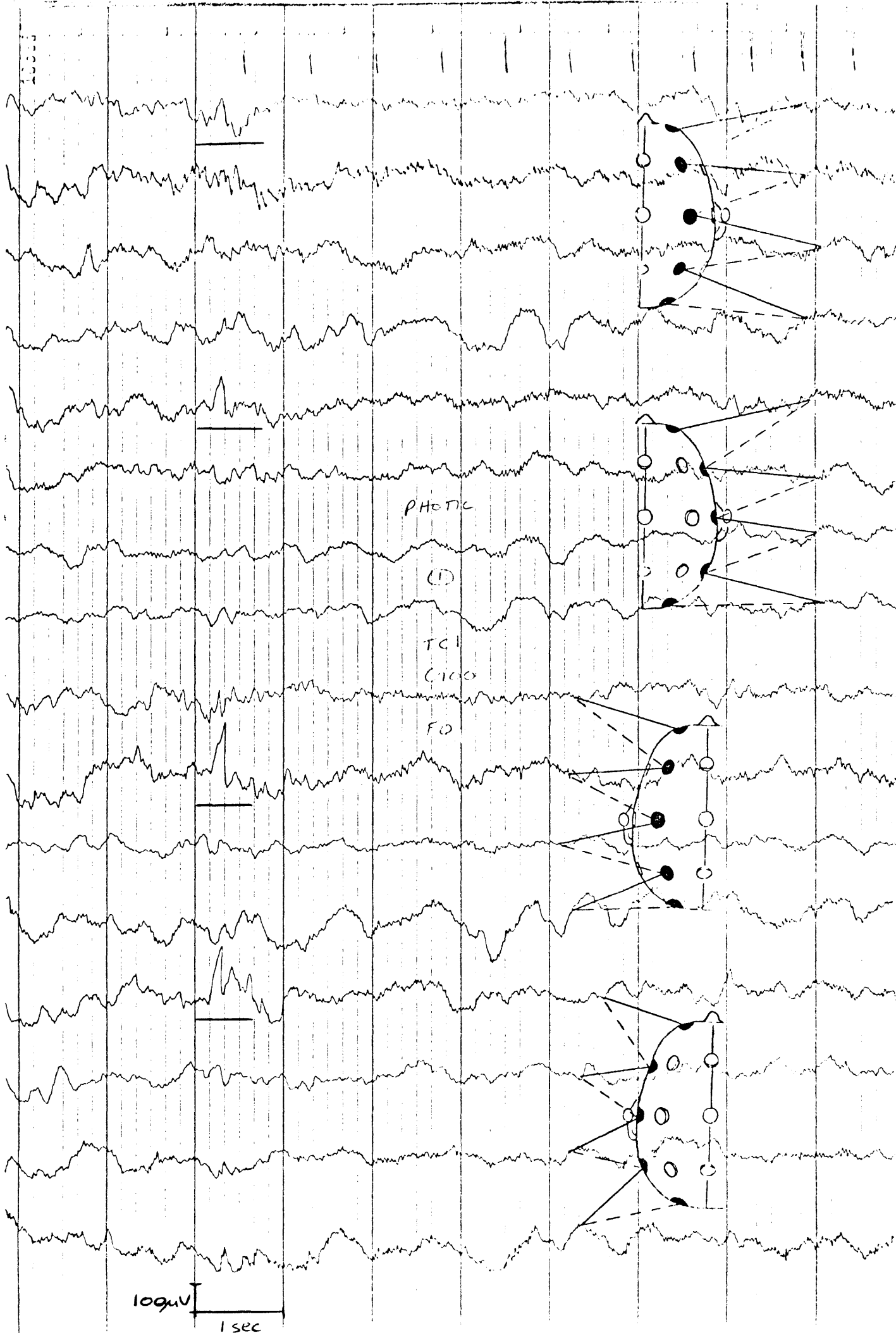
1. Lack of differentiation in the EEG between behavioural sleep and wakefulness. EEG changes from wakefulness to sleep have been noted in the 8 month (pre-term) foetus. Thus, if these changes are absent in the EEG of a full-term neonate, this is regarded as abnormal. Continuous slow (7 Hz) diffuse EEG activity during both sleep and wakefulness has been seen in neonates subjected to prolonged anoxia, and in microcephalic infants.

2. Disappearance of 'trace alternant'. Trace alternant activity has been described previously. This activity disappears in the EEGs of full-term noenates by 6 weeks of age. Once it is absent in the EEG of the individual case, it is not normally seen again. Persistence of this pattern beyond 6 weeks may signify a disturbance of cortical/subcortical relations. Ellingson and Peters (1980b)[6] found that trace alternant disappeared later in Trisomy 21 infants (mean of 57 days) than in normals (mean 33 days).

3. Asymmetry and asynchrony of sleep spindles. Due to considerable inter-individual variation in the symmetry and synchrony of sleep spindle bursts, Ellingson and Peters (1980a)[7] consider that only a complete unilateral or bilateral absence of spindles after the age of 3 months should be regarded as abnormal. Dreyfus-Brisac and Curzi-Dascalova (1975)[8] likewise feel that the absence of spindles between 3 and 8 months is a severe abnormality. Hypo-thyroid and hypoxic infants (Schultz et al (1968)[9], for example,

showed delayed development of sleep spindles. It has been postulated that this is due to insufficient axon branching and synaptic contact. Sleep spindles also appeared significantly asymmetric and asynchronous as late as 12 months in Trisomy 21 infants, whereas normal full-term infants showed reasonable symmetry and synchrony as early as 3 months.

4. Hypsarrhythmia. The EEG is marked by high voltage poorly organised, multifocal or generalised spike/sharp wave and slow wave discharges. (Figure 17). The latter may occur with periods of intervening relative electrical silence (the burst - suppression pattern), or against a diffusely abnormal, slow background. (Figure 18). This type of activity is seen after the age of 4 months, and occurs up to the age of 4 years. It is associated with any diffuse insult to the maturing brain and suggests structural damage.

5. Focal spikes and spike and wave. Focal activity (phase reversed) is abnormal at any age. The problem for the electroencephalo-grapher is whether phase reversed activity appears consistently enough to be described as focal. (Figures 19 - 21).

In general, Ellingson's (in Klass and Daly) (1979)[10] observations regarding neonatal EEGs appears to have general validity for those of sleeping infants as a whole: "They are difficult to evaluate, and there is no disgrace in sometimes having to equivocate. Less harm is done in confessing ignorance or indecision than in jumping to wrong conslusions".

SOME RECORDING CONSIDERATIONS

1. In very young infants, the head may be too small to accommodate a full electrode array. A limited number of electrodes must then be used and the question arises as to what array is most suitable. An important consideration is that at least one transverse series of electrodes should be used to assist the electroencephalographer to detect activity arising at the midline. This aids in deter-mining whether paroxysmal activity may be classified as a specific sleep phenomenon or as epileptogenic.

FIGURE 17   Female, aged 11 months who presented with
                    Salaam attacks.   The sleep EEG showed high
                    voltage poorly-organised multifocal spike and
                    wave and spike and slow wave and sharp and slow
                    wave discharges typical of hypsarrhythmia.
                    Severely abnormal.

FIGURE 17 41.

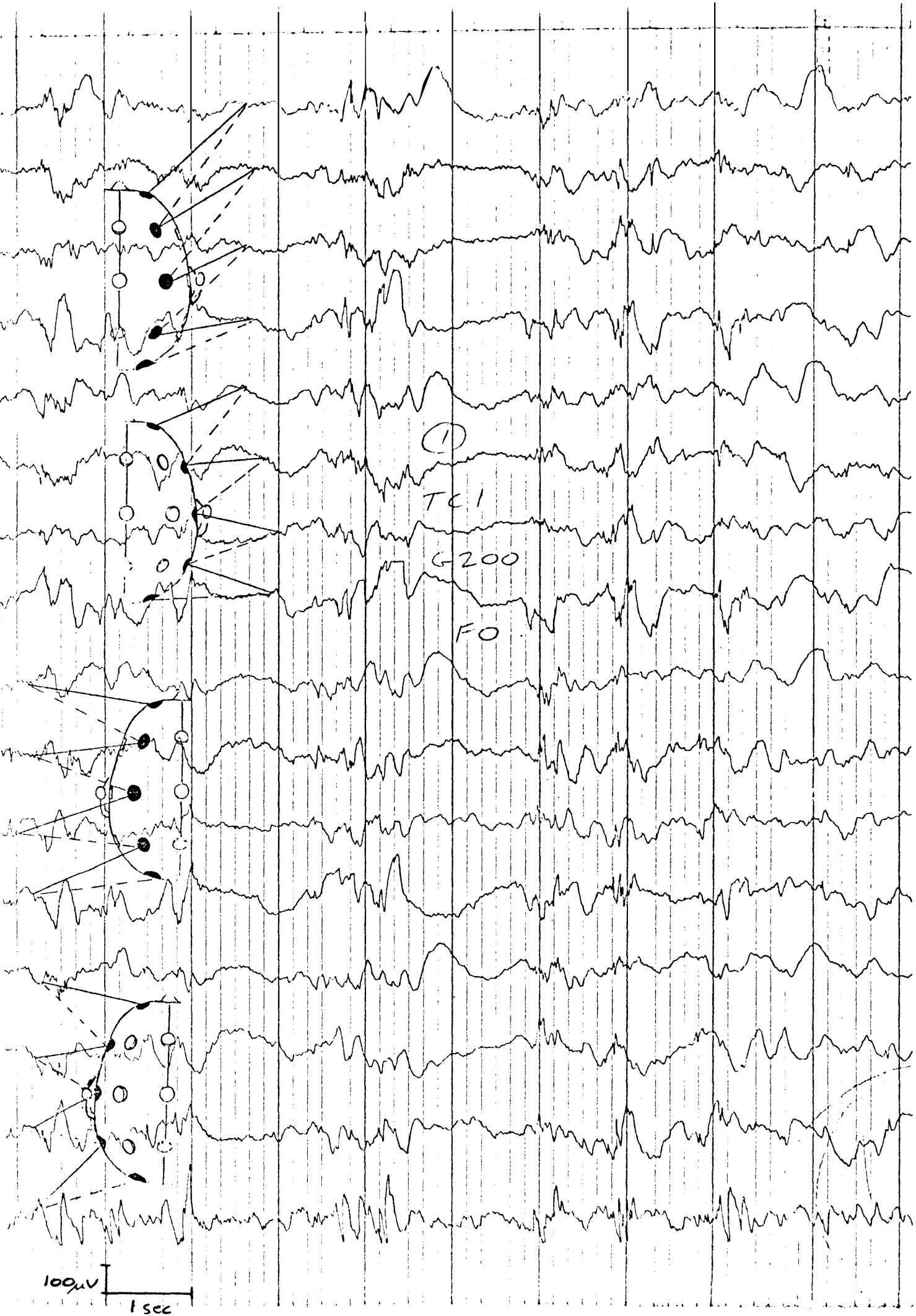FIGURE   18   Sleep EEG in a male, aged 13 months, with
                     cerebral palsy after possible foetal anoxia.
                     The EEG shows a burst suppression pattern
                     frequently seen in hypsarrhythmia
                     Severely abnormal.

FIGURE 18 43.

100µV
1 sec.
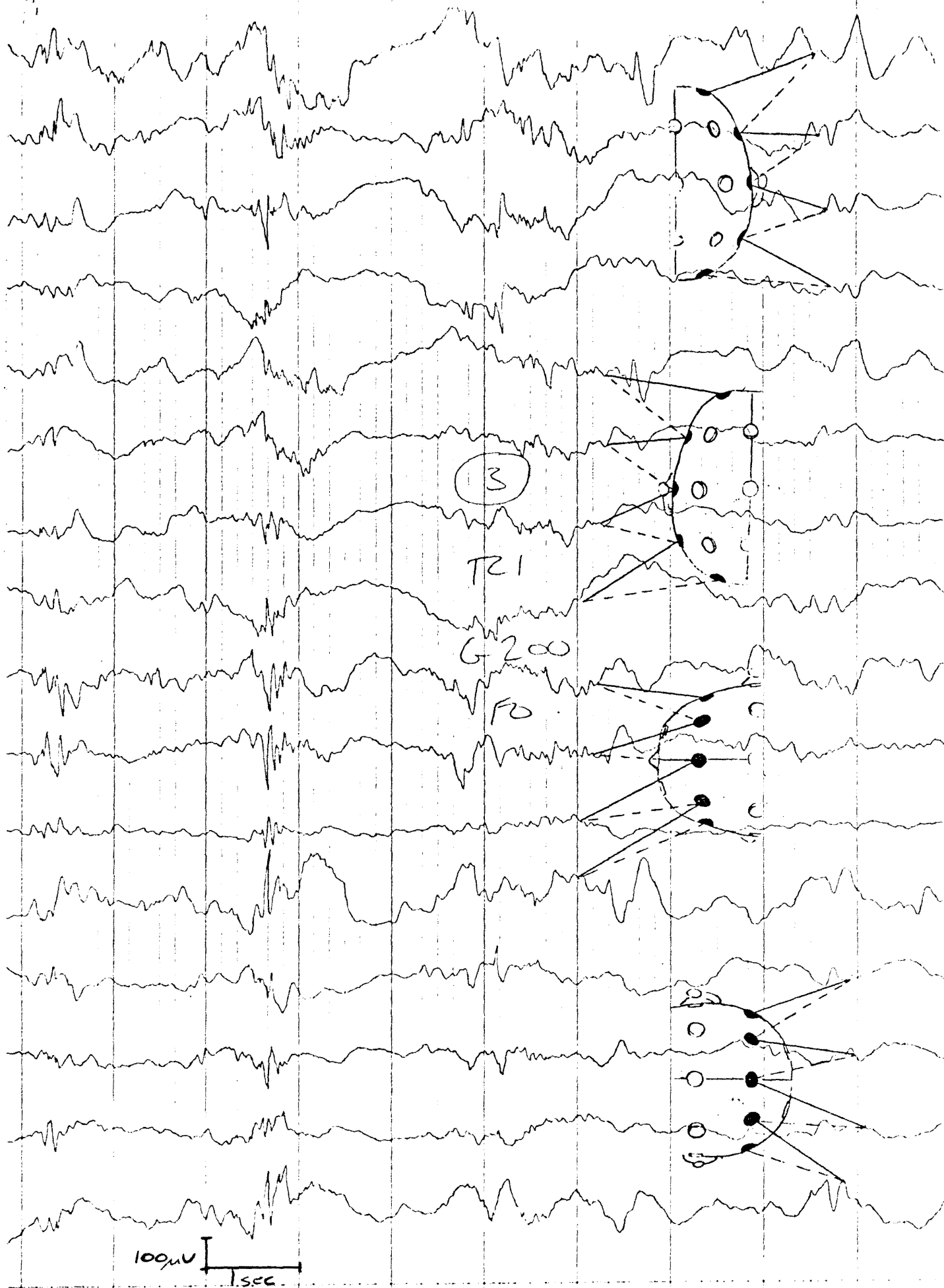
FIGURE   19   Sleep EEG (trichloryl) in a male, aged 2 years
                   5 months with a history of tick bite fever.
                   Focal left temporal spike discharges occur.
                   Markedly abnormal.

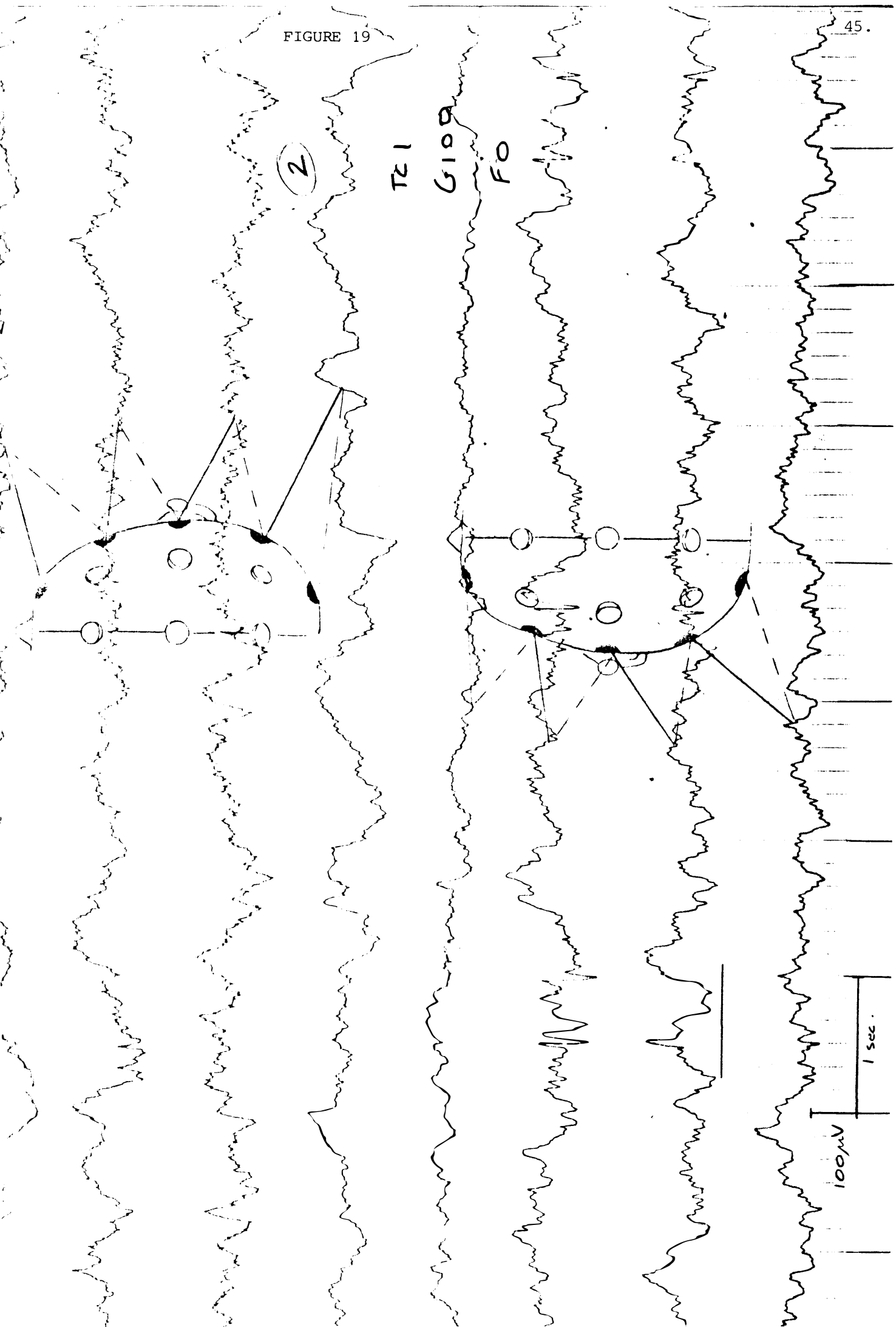FIGURE 19

FIGURE 20   Sleep EEG (Vesparaxette) in a female, aged 3
              years 5 months with a provisional diagnosis of
              epilepsy. Frequent spike discharges focal in
              the right centro-temporal area are shown.
              Markedly abnormal upholding diagnosis of
              epilepsy.

FIGURE 20 47.
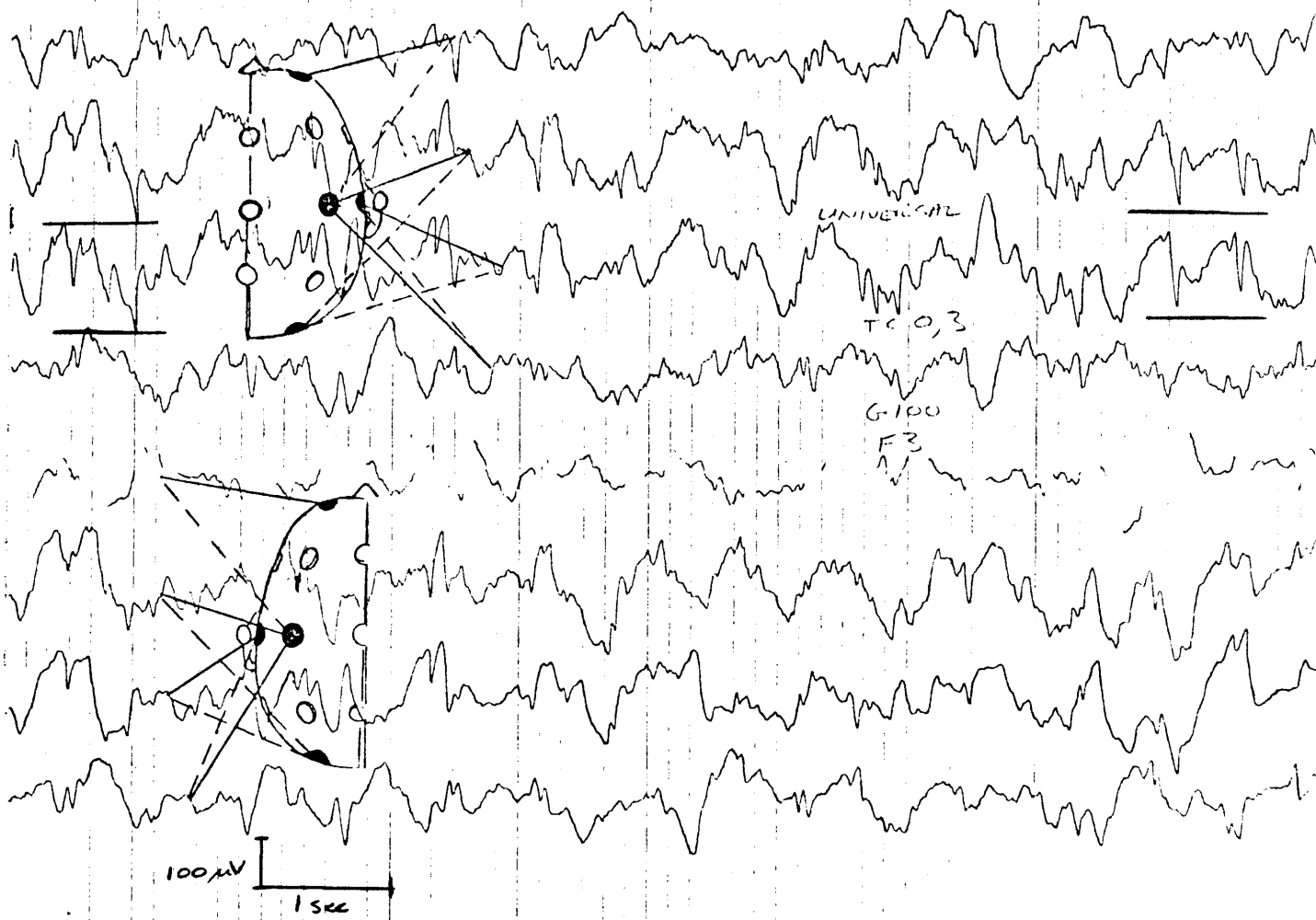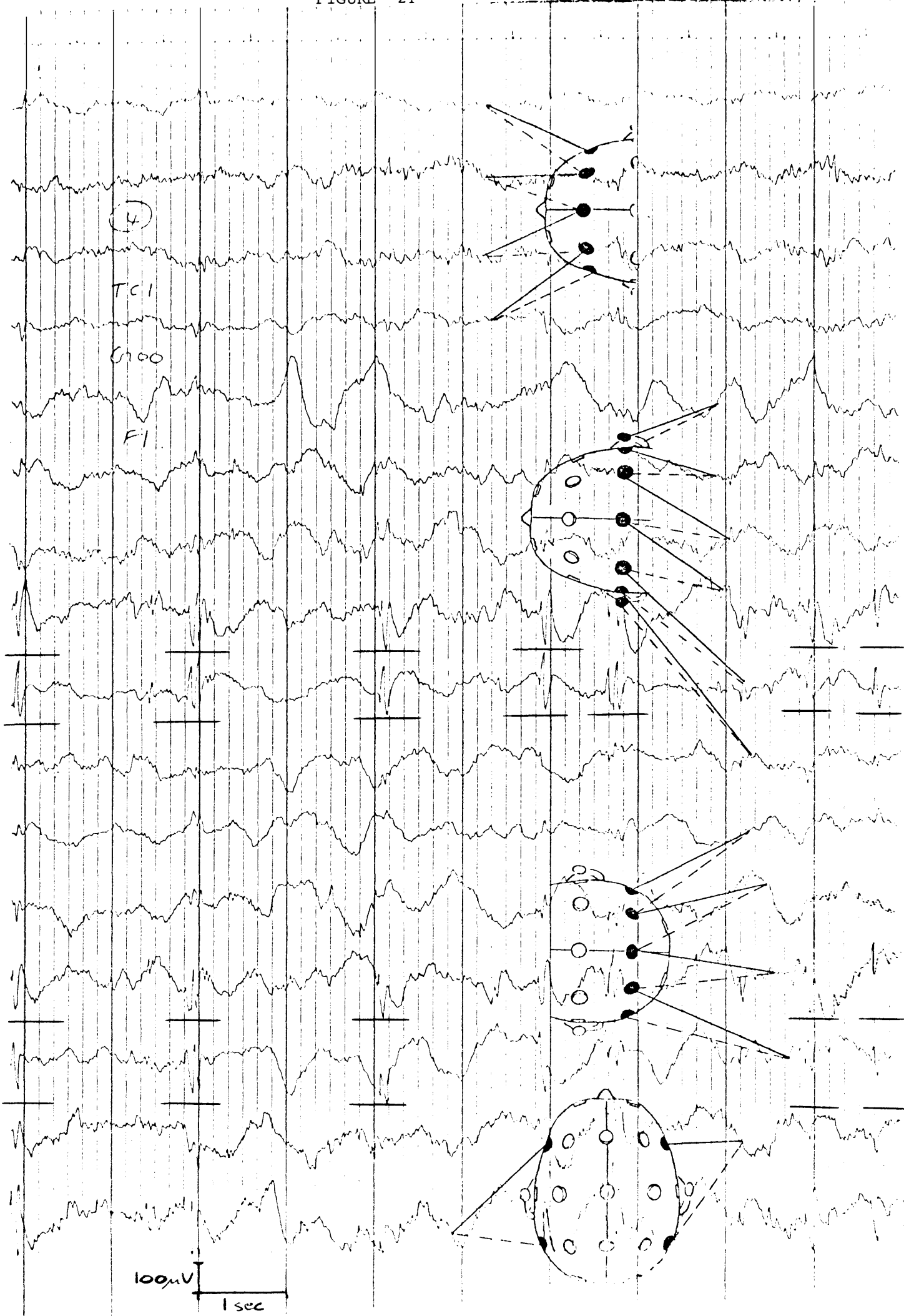
FIGURE 21   Sleep EEG (Vesparaxette) in a female, 4 years and
                    4 months old.   History of screaming episodes.
                    Markedly abnormal EEG with spike and wave and
                    multiple spike and wave activity appearing
                    focally in the temporal and post-temporal areas.

FIGURE 21

100μV

1 sec

2. The technician should be alert to, and note, any movements or
    activity on the part of the child. Especially important is to
    note any auditory stimuli in the environment. These in close
    association with paroxysmal acitivty in the EEG may prove
    crucial in the decision as to whether this is normal or epilep-
    togenic. Further, the technician should attempt to rate the
    depth of sleep of the child during the course of the EEG.

3. If different electrode montages are employed, the technician
    should apply a standardised auditory stimulus (perhaps by tapping
    against the side of the EEG machine) during each. This allows
    later comparison of specific sleep phenomena obtained under
    standardised conditions.

4. A mundane consideration - allow extra time for sleep EEGs when
    making appointments, so that recordings of adequate length for
    the detection of abnormalities can be obtained.

51.

REFERENCES   Order of appearance

ANDERS T, EMDE R and PARMELEE A (Eds.) (1971).   1
A manual of standardized terminology, techniques
and criteria for scoring of states of sleep and
wakefulness in newborn infants.   Los Angeles;
Brain Information Service, UCLA.

CROWELL D H, KAPUNIAI L E, BOYCHUK R B, LIGHT M J   3
and HODGMAN J E (1982).   Daytime sleep stage
organization in 3 month old infants. Electroenceph.
clin. Neurophysiol. 53: 36-47.

DREYFUS-BRISAC C and CURZI-DASCALOVA L (1975).   5
EEG during first year of life.   In: REMOND A (Ed.)
Handbook of Electroenceph. clin. Neurophysiol. Vol. 6B:
24-30. Amsterdam; Elsevier.

ELLINGSON R J (1979).   EEGs of premature and full-term   10
newborns.   In: KLASS D and DALY D (Eds.) Current
Practice of Clinical Electroencephalography. New York:Raven.

ELLINGSON R J (1980a).   Development of EEG and daytime   2   4   7
sleep patterns in normal full-term infants during the
first 3 months of life : longitudinal observations.
Electroenceph. clin. Neurophysiol. 49: 112-124.

ELLINGSON R J (1980b).   Development of EEG and daytime   6
sleep patterns in trisomy-21 infants during the first
year of life : longitudinal observations.
Electroenceph. clin. Neurophysiol. 50: 457-466.

SCHULTZ M A, SCHULTE F J, AKIYAMA Y and PARMALEE A H   9
(1968).   Development of electroencephalographic sleep
phenomena in hypothyroid infants.   Electroenceph.
clin. Neurophysiol. 25: 351-358.

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