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CRITERIA FOR THE ASSESSMENT OF ABNORMALITY IN THE SLEEP ELECTROENCEPHALOGRAMS OF INFANTS AND YOUNG CHILDREN

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SUMMARY

Available literature was surveyed and criteria compiled for the assessment of abnormality, particularly as regards paroxysmal activity, in the sleep electroencephalograms (EEGs) of infants and young children.

OPSOMMING

Beskikbare bronne is nagegaan en kriteria saamgestel vir die beoordeling van abnormaliteit, veral met betrekking tot paroksismale aktiwiteit, in die slaap elektroënkefalogramme (EEGs) van babas en jong kinders.

CONTENTS

INTRODUCTION

The task of deciding whether activity seen in the sleep EEGs of infants and young children, particularly paroxysmal activity, is abnormal is a daunting one. Many of the accepted criteria for the judgement of abnormality in adults' EEGs can not be applied to children's sleep EEGs. Criteria which may be used for determining the abnormality of the latter are scattered throughout the literature and, in many cases, not easily available. However, the need for a compilation of these criteria is dictated by the fact that in any clinical EEG service or practice, most recordings made from infants and young children are performed while the patient sleeps. Sleep may be spontaneous or induced and is necessary as young patients are unable otherwise to co-operate so that a satisfactory recording may be obtained. This report provides a compilation from the literature of the criteria for the assessment of abnormality in children's sleep EEGs.

TERMINOLOGICAL CONSIDERATIONS

Terminology used has obfuscated the issue. Arousal phenomena have been given many names: (1) Vertex sharp waves (transients), (2) biparietal humps, (3) bicentral transients, (4) v waves, (5) evoked negative sharp waves on the vertex, (6) startle waves, (7) sigma waves (activity), (8) spindles, (9) K-waves and (10) K-complexes. (1) to (6) appear to be, roughly, descriptions of the same phenomenon. (7) and (8) likewise describe the same thing. (9) and (10) are, it appears, combinations of both types of previously-mentioned activity, and are equivalent terms. However, attempting to pinpoint exactly what various authors have meant by each term is not a fruitful exercise. Overlap occurs as a matter of historical inexactitude. Of relevance to the clinical electroencephalographer is that all types of arousal phenomena are normal. (1) to (6), referred to on aggregate as vertex sharp waves, appear with all degrees of sharpness in both normal and epileptic groups (Kiloh et al, 1953)[1].

They may, in young children, be of such high amplitude and so spikelike, that the temptation to describe them as epileptogenic is difficult to resist (Kiloh et al, 1972)[2] although mistaken. Spindles, (7) and (8), may show asynchrony even into adulthood. However, marked asynchrony after the age of 2 years is regarded as abnormal (Silverman, 1966)[3]. Further, as the frequency characteristics and paroxysmal morphology of K-complexes may closely approximate those of wave and spike activity, caution should be exercised in describing them as abnormal (Monod and Ducas, 1968)[4].

1. VERTEX SHARP WAVES

These are monophasic (Kiloh et al, 1953)[5] or triphasic (Duterte, 1977)[6] sharp waves. If monophasic, they are electronegative. If triphasic, the initial component is a small spike, followed by a sharp wave lasting 50 - 250 ms (that is, 4-20 Hz in frequency) followed by a small spike slower than the first component. They occur spontaneously during sleep, or are stimulus-evoked during sleep or wakefulness (Duterte, 1977)[7]. If during sleep, they are maximal during light sleep. Kiloh et al (1953[8], 1972[9]) and Rechtshaffen and Kales (1968)[10] see a maximal incidence of vertex sharp waves during stage 1 sleep - when alpha activity, if present, has disappeared, and low voltage, mixed frequency activity predominates. Duterte (1977)[11] regards this activity as being a good index of stage 2 sleep (in adults at any rate). Vertex sharp waves are phase-reversed about the midline, of maximal amplitude in adults at the vertex (that is, in the region of the Rolandic fissure), and, in young children, in the frontal areas. They are seen in the quiet sleep of newborn infants (Anders et al, 1971)[12], and appear clearly at the age of 5 or 6 months (Silverman, 1966)[13], (Samson-Dollfus, et al 1964)[14]. Thereafter, the maturational pattern is author-dependent. Silverman (1966)[15] has a maximal abundance at 3 years of age, with a decline thereafter to 13 years and maintenance at this level into adulthood. Duterte (1977)[16] proposes maximal amplitude and abundance from 2 to 12 years. In adults, the amplitude appears to be somewhat less than 200 - 250 μv, (Rechtschaffen and Kales, 1966[17], Duterte, 1977[18]), in children often much more (Kiloh et al, 1972)[19]. Vertex sharp waves occur in isolated waves or in bursts, in a paroxysmal and aperiodic manner.

2. SPINDLES

These comprise runs or groups of rhythmic waves of progressively and gradually increasing and then decreasing amplitude. The waves are monomorphic, diphasic and symmetrical with respect to the baseline. A frequency of 12-14 Hz for the individual wave components of spindles appears generally accepted (for example, by Rechtschaffen and Kales, 1968[20]; Metcalf and Jordan, 1972[21]; Duterte, 1977[22]). Silverman (1966)[23] states, however, that a spindle component frequency of 14 Hz is present initially, and that 12 Hz components only appear at 4 years of age. Spindles are phase-reversed about the midline. They are maximal in the prerolandic (frontal) or central areas, or in the postrolandic (parietal) areas (Gibbs and Gibbs, 1950)[24]. Asynchrony of spindles is common in young infants, particularly before the age of 4 weeks (Metcalf and Jordan, 1972)[25], but is reduced after 4 months (Silverman, 1966)[26] and is, apparently, rare after 6 - 8 months (Dreyfus-Brisac and Curzi-Dascalova, 1975)[27]. Marked asynchrony after 2 years may be abnormal (Silverman, 1966)[28]. Asymmetry of spindles in young infants is also common (Dreyfus-Brisac and Curzi-Dascalova, 1975)[29], but is reduced after 5 - 6 months (Metcalf and Jordan, 1972)[30]. A degree of asymmetry may persist to 4 years of age, and is only considered abnormal if extreme (Dreyfus-Brisac and Curzi-Dascalova, 1975)[31]. Spindles are regarded as appearing only in quiet sleep (Parmelee et al, 1968)[32] or high voltage slow wave sleep (Metcalf and Jordan, 1972)[33]. However, a measure of confusion exists here, as Duterte (1977)[34], Samson-Dollfus et al (1964)[35] and Gibbs and Gibbs (1950)[36] suggest that spindles are characteristic of light sleep. In any event, spindles respond to auditory stimuli by attenuation (Duterte, 1977)[37]. They are rare in full-term infants before the age of 4 weeks, but are well-established by 7 - 8 weeks (Dreyfus-Brisac and Curzi-Dascalova, 1975[38]; Metcalf and Jordan, 1972[39]). Initially, spindle episodes are long (8 - 10 seconds) but at the age of 3 - 5 months have decreased to 1 - 3 seconds (Metcalf and Jordan, 1972)[40]. Parmelee et al (1968)[41] and Dreyfus-Brisac and Curzi-Dascalova (1975)[42] regard the absence of spindles in the sleep of infants aged 3 to 8 months as abnormal. Monod and Ducas (1968)[43] state that the appearance of sleep spindles is rarely delayed beyond the age of 8 months in normal infants.

3. K-COMPLEXES

A degree of inexactitude prevails regarding the number of components constituting a K-complex. Some authors, for example Gibbs and Gibbs (1950)[44], Rechtschaffen and Kales (1968)[45], Kiloh et al (1972)[46] and Duterte (1977)[47], regard the K-complex as having 2 components. Others (Roth et al, 1953[48]; 1956[49]) identify 3 components. The following morphology appears to encompass most of the variations described, but some degree of laxity should be allowed in the individual case. The first component is a surface negative sharp wave, either di- (bi-) or tri-phasic, with a frequency between 3 and 8 Hz and amplitude between 50 and 150 µv. The second is surface positive, and slower than the first, appearing with a frequency of about 2 - 2,5 Hz. Amplitude is 100 - 250 µv. Finally, the K-complex is constituted by a burst of spindles at 12 - 14 Hz with a regular amplitude of 25 - 100 µv. The total duration of the K-complex is 0,5 to 4 seconds. Its onset is paroxysmal (Duterte, 1977)[50]. K-complexes are phase-reversed about the midline, and of maximal amplitude over the vertex (Rechtschaffen and Kales, 1968)[51]. The latter authors and Roth et al (1956)[52] restrict the occurrence of K-complexes to stage 2 sleep. However, Kiloh et al (1972)[53] and Metcalf and Jordan (1972)[54] suggest that K-complexes are elicited also in deeper sleep stages, although their morphology may not be as well-differentiated as in stage 2. K-complexes occur spontaneously or in response to sudden stimuli. They may be evoked indefinitely in suitable subjects if the interstimulus interval is of sufficient duration (3 seconds or more; Roth et al, 1956[55]). They are all-or-none, and are not sensory-specific, (that is, they occur to visual, auditory and tactile stimuli). A more prominent K-complex is evoked in response to meaningful (for example, calling the subject's name) than non-meaningful stimuli (Metcalf et al, 1971)[56]. Roth et al (1956)[57] regard the K-complex as signalling both a crude perception of the stimulus and an abortive arousal. K-complexes appear to emerge at 5 - 6 months of age (Gibbs and Gibbs, 1950[58]; Metcalf and Jordan, 1972[59]) and their emergence may relate to the onset of dreams (Metcalf et al, 1971)[60]. They develope further to the age of 2 - 3 years (Metcalf et al, 1971)[61] and are well-differentiated by the latter age (Metcalf and Jordan, 1972)[62]. There may be further development until 12 years of age (Metcalf et al, 1971)[63], and some reduction in amplitude in adulthood (Gibbs and Gibbs, 1950)[64].

WHEN IS A K-COMPLEX A K-COMPLEX?

Metcalf et al (1971)[65] give a number of parameters in terms of which a K-complex may be identified. These are as follows:

1. INDIVIDUATION (The degree to which the complex is distinguished from background activity)

As the process of maturation procedes, K-complexes become isolated, more regular and better-differentiated from background activity.

2. VERTEX DOMINANCE (The degree to which voltage dominance is maximal at the vertex)

At the age of 2,5 to 3 months a tendency is noted for vertex activity during quiet sleep to be of higher voltage and more regular than in other areas. At 5 - 6 months, vertex activity assumes the typical K-complex morphology, although vertex voltage dominance is incomplete. Vertex dominance is established at 1,5 years and complete at 2 years.

3. REPETITION

Initially, K-complexes occur in bursts with a relatively long duration (4 - 6 seconds) and poorly defined initial sharp component. Maturation brings differentiation of this component and reduction in the amount of sleep time occupied by repeating K-complexes. From age 3 to 9 years the initial sharp component may repeat so rapidly (3 to 9 times in a 1 to 3 second period) that the slow wave segment of the K-complex does not have a chance to occur. In adolescence and young adulthood the rate of repetition abates and the slow wave component is seen. (1 complex per second or 1 every 2 - 3 seconds may be seen).

4. SPREAD (The appearance of the complex simultaneously in areas other than the true vertex)

At an early stage of development (6 months to 12 months) the K-complex

appears diffusely (with no vertex localisation). Vertex dominance is established at 1,5 years, and spread diminishes until about 10 years. At 14 to 16 years of age spread is virtually non-existent. Metcalf and Jordan (1972)[66] indicate that in addition to the above, the sharpness of the initial component of the complex is an additional descriptive parameter. In the infant, the first component is not sharp, but this feature emerges as an aspect of development. In adulthood, however, sharpness abates.

In summary, therefore, the immature K-complex is characterised by poor vertex dominance, large spread, and an initial component which is not sharp. Further, it is intermingled with the ongoing EEG activity. The mature K-complex is vertex dominant, well-differentiated from the background EEG and shows little spread. Single complexes, not bursts of mixed activity, and a sharp primary component, are present.

WHAT IS ABNORMAL IN SLEEP EEGs OF INFANTS?

1. BURSTS

Metcalf and Jordan (1972)[67] suggest that bursts are adjudged abnormal if: (a) there is a loss of vertex dominance. The more regions associated simultaneously with a burst, the more abnormal it is considered. Thus, a burst involving only the vertex and temporal areas is more acceptable than one simultaneously involving the vertex, frontal, temporal and occipital regions; (b) of long duration. As the duration of a burst increases, the probability of it falling within normal limits decreases. The critical duration appears to be 5 - 6 seconds. If longer than this, the burst is regarded as abnormal by these authors. Dreyfus-Brisac et al (1956)[68] agree with these criteria of assessing burst abnormality. However, they suggest that a duration of 10 to 60 seconds renders a burst abnormal. They also indicate that abnormal bursts are likely to be: (a) of high amplitude (300 - 500 μv); (b) very slow (1 Hz); (c) monomorphous; (d) repetitive or self-perpetuating in a manner reminiscent of epileptic after discharges. Bursts meeting these criteria are encountered, according to Dreyfus-Brisac et al (1956)[69], in infants aged 10 months to 2 years after encephalopathy or meningitis. Waking EEGs in these infants may be normal. Metcalf and Jordan (1972)[70]

indicate that distorted burst patterns are extremely rare in normals under 2 years of age and in adolescents, and are seen most commonly between 3 and 7 years. The younger the child with this type of pattern, therefore, the more likely it is that significant central nervous system pathology is present. Dreyfus-Brisac and Curzi-Dascalova (1975)[71] state, "From 3 to 12 months the presence of abnormal figures (focal spikes of any location, paroxysmal patterns of any kind) is very rare." (p.6B - 27). Further, the relatively high incidence of distorted bursts in normals aged 3 to 7 years makes a diagnosis of epilepsy based solely on this characteristic unreliable in children between these ages.

2. OTHER ACTIVITY

(a) Trace alternant. Prematures born at 32 weeks (full-term 40 weeks) show 70% active sleep and 30% quiet sleep. The latter comprises bursts of high voltage slow wave (0,5 - 3 Hz) activity, sometimes with superimposed sharp waves and low voltage activity, interspersed with flat periods lasting 8 - 15 seconds, (the so-called 'trace alternant'). As the infant matures the flat periods shorten in duration and are replaced by slow wave activity. At full-term, quiet sleep comprises virtually continuous slow wave activity - however, some trace alternant may still be present until 4 - 5 weeks post term. Rarely, however, is this seen in normals after the age of 6 weeks. If a clear trace alternant persists after this age, a cortical/subcortical disturbance, or panencephalitic dysfunction, may be indicated (Metcalf and Jordan, 1972)[72]. (b) Lack of differentiation between sleep and wakefulness. In the normal foetus, a differentiation between EEG patterns during wakefulness and sleep is established by 8 months (pre-term). If the full-term neonate shows no wake/sleep differentiation in EEG terms, this is regarded as abnormal. Slow (7 Hz) continuous, diffuse EEG activity appears during both sleep and wakefulness. This pattern may occur in cases of prolonged exposure to anoxia (Dreyfus-Brisac, 1964)[73] or in microcephaly (Samson-Dollfus et al, 1964)[74]. Prognostically, this pattern is ominous. (c) Prolongation of immature sleep pattern. Before 3 months of age, the infant goes direct into active sleep (the forerunner of REM sleep), and then into quiet sleep. After 3 months, quiet sleep is entered directly, preceeded by high voltage slow waves which are the precursors of later drowsy hypersynchrony. If the change

is prolonged to 4 - 5 months of age, this is regarded as abnormal (Metcalf and Jordan, 1972)[75]. (d) Hypsarrhythmia. The EEG in this diagnosis is marked by poorly organised, high voltage patterns, in both sleep and wakefulness, with generalised wave and spike or sharp wave activity. Usually, this is seen after 4 months of age, and rarely after 4 years. Structural brain damage is frequently indicated (Samson-Dollfus et al, 1964[76]; Kiloh et al, 1972[77]). (e) Abnormally slow EEGs. Here, 2 - 3 Hz activity in all cortical areas is seen during wakefulness, accentuated by drowsiness. This is usually seen in the first year of life (Samson-Dollfus et al, 1964)[78]. (f) Generalised depression. EEGs during both sleep and wakefulness show a generalised depression of activity. (Samson-Dollfus et al 1964)[79].

THE EMERGENCE OF WAVE AND SPIKE ACTIVITY DURING SLEEP

Sleep activation (spontaneous and induced) is a procedure adopted in cases of suspected epilepsy where the wake EEG is non-specific, equivocal or normal. Drug-induced daytime sleep does not differ from spontaneous sleep (Kiloh et al, 1972)[80]. As an aid in deciding whether paroxysmal EEG activity in sleep is epileptogenic or an arousal phenomenon, it is instructive to consider which sleep stages are most productive of wave and spike activity. This issue has been reviewed by Daly (1973)[81]. He indicated that NREM sleep activates the generalised 3 Hz wave and spike activity seen in patients with absences while REM sleep diminishes these discharges. He cites a study which showed (using 13 subjects) the following (all discharges per minute): 0,24 during the daytime waking state; 0,08 in the period immediately before sleep; 0,46 at the onset of NREM sleep; 1,09 during stage 4 sleep and 0,07 during REM sleep. Focal wave and spike activity, however, appears augmented by both NREM and REM sleep, although the morphology of discharges may differ in each. During NREM sleep the bursts are longer in duration, higher in amplitude, the focus may expand in size and activity may be induced contralaterally and homologously. During REM sleep the focus is equivalent in size to that of the waking state and spikes resemble those during the waking state.

GUIDELINES FOR CLINICAL ELECTROENCEPHALOGRAPHERS

This section summarises what has been detailed previously, stressing the normal arousal response or activity.

1. VERTEX SHARP WAVES

An arousal response (ie. a normal phenomenon): may be extremely sharp (spikelike); is evoked most readily in sleep stages 1 or 2, or in the quiet sleep of newborns; is phase-reversed about the midline and maximal in amplitude at the vertex or its immediate vicinity; emerges clearly at 5 - 6 months of age, and, although its subsequent development is controversial, may be expected to be seen up to about 13 years of age with some abundance, and to a lesser extent into adulthood; may, in children, be of high amplitude.

2. SPINDLES

Spindles which may be considered normal: are phase-reversed about the midline and maximal in the frontal, central or parietal cortical areas; may show a reasonable degree of asynchrony and asymmetry before the ages of 5 - 8 months, with asynchrony reduced after 2, and asymmetry after 4, years; may appear in both light and deeper sleep stages; are rare before 4 weeks of age but common by 7 - 8 weeks; are long in duration (8 - 10 s) before 3 - 5 months of age, but short (1 - 3 s) in duration thereafter; are established by the age of 8 months.

3. K-COMPLEXES

A normal arousal phenomenon: may have 2 or 3 components; is phase-reversed about the midline and maximal at the vertex by 5 - 6 months and vertex dominant by 1,5 years; may occur in sleep stage 2 or deeper sleep stages; emerges at 5 - 6 months of age, is well-differentiated by 2 - 3 years and seen prominently to 12 years with some amplitude reduction in adulthood; is better differentiated and more isolated from background activity with increasing maturation; is maximally repetitive from 3 - 9 years and less so in adolescence and young adulthood; is well localised to the vertex by 1,5 years with little involvement of other areas after 10 years; is of short duration (less than 10 s); is of relatively low amplitude ($< 300\ \mu v$); is non-perpetuating; may be distorted between 3 and 7 years of age although this is rarely so under 2 years or in adolescents.

4. OTHER ACTIVITY

In normals: trace alternant is rarely seen after 6 weeks; differentiation between sleep and wakefulness is seen in the EEG at full-term birth; a mature sleep pattern has emerged by 3 months.

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