FUNCTIONAL ORGANIZATION OF SLEEP STATES IN NORMAL AND BRAIN-DAMAGED INFANTS

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A polygraphic investigation was made of daytime sleep in healthy infants with analysis of the EEG, oculogram, muscle tone, fluctuations in skin resistance, respiration, ECG, rheoencephalogram, and rheogram of the leg. Infants with cerebral palsy of perinatal genesis were investigated by the same method. The results showed that in early infancy it is possible to distinguish the basic stages of slow sleep and the stage of fast sleep, although the electroencephalographic expression of these stages in infants has special features of its own. The age dynamics of the polygraphic picture of sleep shows that the electroencephalographic, autonomic, and motor components of sleep, although clearly coordinated in the various stages, have at the same time considerably autonomy. This is confirmed by the results obtained by analysis of the cerebral pathology. The need for adequate assessment of this complex function in order to elucidate the physiological nature of sleep is emphasized.

KEY WORDS: human ontogeny; normal and pathological sleep.

The age dynamics of manifestations of sleep in man under normal conditions and in the presence of cerebral pathology could shed light on the mechanism of this phenomenon.

Information in the literature consists chiefly of the electroencephalographic characteristics of sleep in young infants in health and disease, although polygraphic investigations shedding light on the structure of sleep in the early postnatal period and its changes with age and giving the qualitative characteristics of sleep to some degree have also been reported [1, 5, 6, 10, 11, 13, 17, 18, 20]. A neurophysiological analysis of the pathology of sleep has also been undertaken in brain-damaged children during the first year of life [9, 12, 13, 15, 16].

EXPERIMENTAL METHOD

A polygraphic investigation of daytime sleep was carried out on 30 healthy infants aged from 3.5 weeks to 1 year and infants of the same age with brain damage caused by intrauterine pathology, birth trauma, and asphyxia. Records were made of the EEG, is monopolar and bipolar combinations of leads, and also of respiration, the oculogram, the ECG, the psychogalvanic reflex (PGR), the rheoencephalogram, and the rheogram of the limb, on a multichannel electroencephalograph. The tone of the facial and cervical muscles was judged from the muscle potentials superposed on the EEG, and in some of the investigations the electromyogram of the cervical portions of the trapezius muscle was recorded.

EXPERIMENTAL RESULTS AND DISCUSSION

The results showed that in early infancy states of drowsiness, falling asleep, light, medium-deep, and deep slow sleep, and also fast sleep, which normally develops only after slow sleep, can be found on the basis of a combination of manifestations. These states can be defined in accordance with the well-known classification of Loomis et al. with the addition of the stage of fast sleep, although the electroencephalographic expression of the stages of sleep in the child has certain special features of its own.

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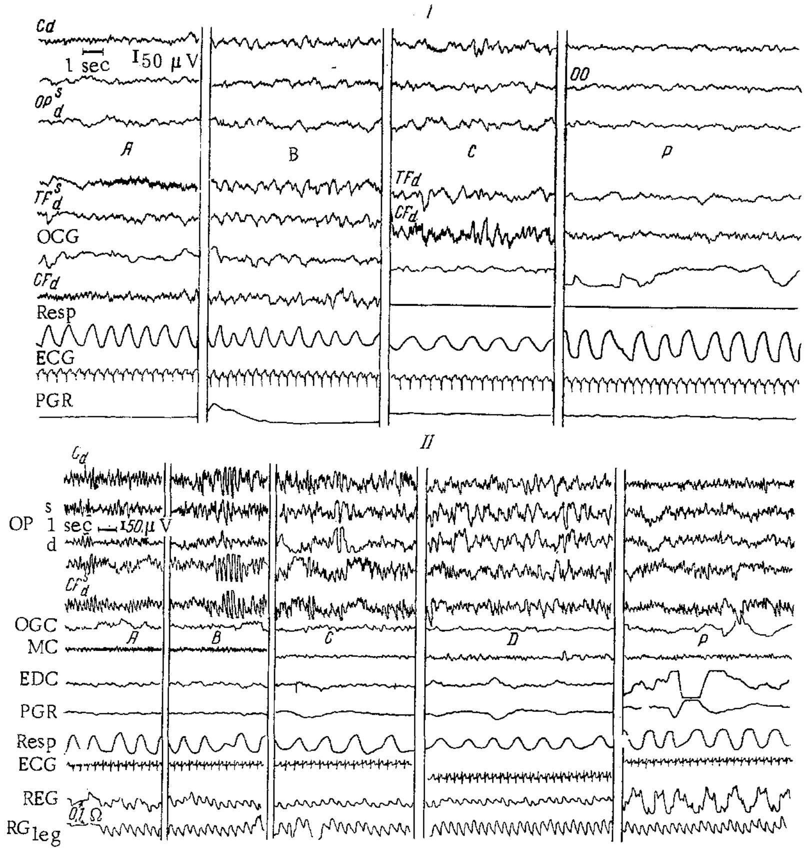

Fig. 1. Polygraphic picture of the stages of sleep in healthy infants in the first year of life: I) a child aged 3.5 weeks; II) a child aged 11 months. Stages of slow sleep: A) drowsiness, B) falling asleep, C) light sleep, D) moderately deep sleep. P) Fast sleep. EEG leads: C) central monopolar, OP) occipito-partietal, CG) central-frontal, TF) temporo-frontal, OO) occipito-occipital; s) left, d) right. OCG) Oculogram; MC) electromyogram of cervical muscle; EDC) electromyogram of extensor digitorum communis muscle; PGR) psychogalvanic reflex; Resp) respiration; ECG) electrocardiogram; REG) rheoencephalogram; RG_{leg}) rheogram of leg.

At the earliest age correlation is observed between the dynamics of the EEG and the autonomic and motor manifestations. With the change from falling asleep to established sleep the frequency and usually the amplitude of the respiratory movements decrease, respiration becomes regular, the pulse rate falls, and the spontaneous PGR often decreases as sleep becomes deeper, although this index is very variable. During falling asleep, the apex of the pulse waves on the rheogram is smoothed a little and their amplitude often increases, but with the deepening of sleep the degree of expressiveness of the respiratory waves diminishes. Besides rapid movements of the eyes and EEG changes, fast sleep is characterized by marked irregularity of respiration and changes in the heart rate and the rheographic indices. Generalized phasic changes are often observed against the background of fast sleep; autonomic indices change synchronously with the eye movement and sometimes general tremor arises. In the stage of fast sleep, by contrast with drowsiness and falling asleep, activity of the facial and cervical musculature could not be recorded (Fig. 1).

During the first year of life the electroencephalographic expression of sleep, especially in the initial stages, varies significantly: the local manifestations of the theta-rhythm in the central portions during drowsiness and falling asleep are replaced by a more widespread theta-activity, a high-voltage "falling asleep rhythm," and groups of uniformly shaped waves with a frequency of 2.5-3.5/sec; spindles of a sigma-rhythm (12-16/sec) appearing usually during the first months of life in the central zones) increase considerably in amplitude; pointed waves appear and then increase in strength in the central zones – they are evidently heralds of the vertex-potential; during the second month of life the Δ-activity increases in the

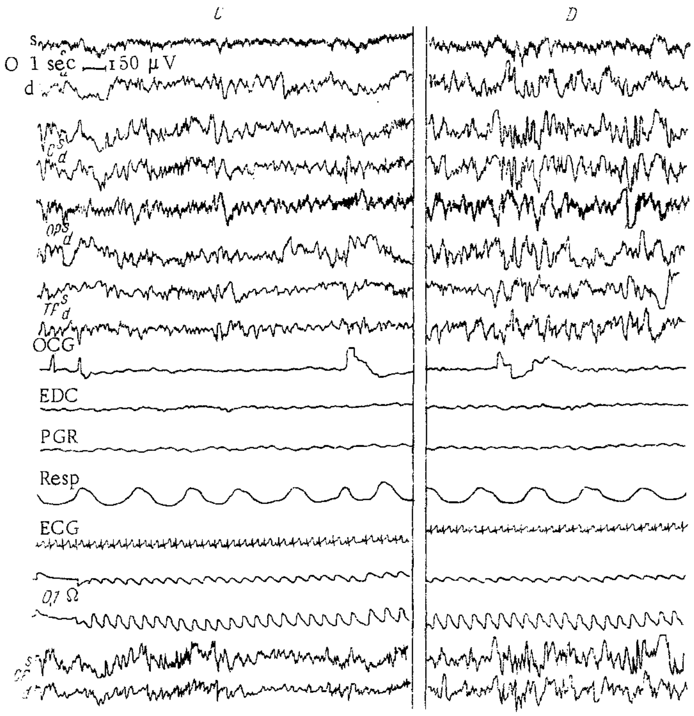

Fig. 2. Pathological dissociation of "behavior" of physiological indices during sleep: fast movements of the eyes are recorded and cardiac arrhythmias observed in the stages of slow sleep (investigation of child aged 6 months with diagnosis of infantile cerebral palsy of the spastic diplegia form). O) Occipital monopolar lead for EEG. Remainder of legend as in Fig. 1.

stage of slow sleep. In the stage of fast sleep, the theta-rhythm becomes more regular with age. In all age groups the special role of the central zones of the cortex, evidently proving a sort of "window" into the subcortical brain regions, becomes apparent on the EEG [2-4, 8-19].

The age dynamics of the EEG gives evidence of the functional and morphogenetic maturation of the synchronizing mechanisms of the brain, whereas the "behavior" of the autonomic components is basically similar in infants aged 1 month and 1 year; only quantitative differences are observed (respiration, for example, becomes slower in the corresponding stages of sleep with age). Consequently, the reticular mechanisms controlling autonomic manifestation in sleep are already sufficiently mature in early infancy and their activity is organized to fit in regularly with the integration that forms the electroencephalographic picture of sleep. The degree of maturity of the mechanisms determining the electroencephalographic and autonomic manifestation in the dynamics of sleep thus undoubtedly differs, although these mechanisms function in harmony.

With the repetition of the manifestation of a certain stage, for example, stage C, the EEG-characteristics of that stage and its characteristics as reflected in autonomic indices may vary somewhat in the different cycles of sleep; sometimes changes in the EEG (in the degree of expressiveness of the slow wave and brightness of the "sleep spindles") are unaccompanied by changes in the respiration and pulse rate, whereas changes in the last indices are not always accompanied by changes in the EEG. Consequently, coordinated operation of the mechanisms determining the "behavior" of the autonomic components and dynamics of the EEG may permit some degree of mismatching, which again points to a certain measure of autonomy.

In the presence of brain damage the qualitative characteristics of the young infant's sleep are often altered. Forms of pathology are found in which, although the normal EEG of sleep is preserved, coordination is disturbed between the EEG, autonomic, and, sometimes, the motor manifestations of sleep (Fig. 2). In other forms of pathology of sleep the EEG picture may be so severely disturbed, yet at the same time stable, that it is impossible to identify the cyclic structure of sleep, whereas the autonomic and motor indices clearly reveal this cyclic pattern (Fig. 3). In severe brain damage the autonomic and motor manifestations

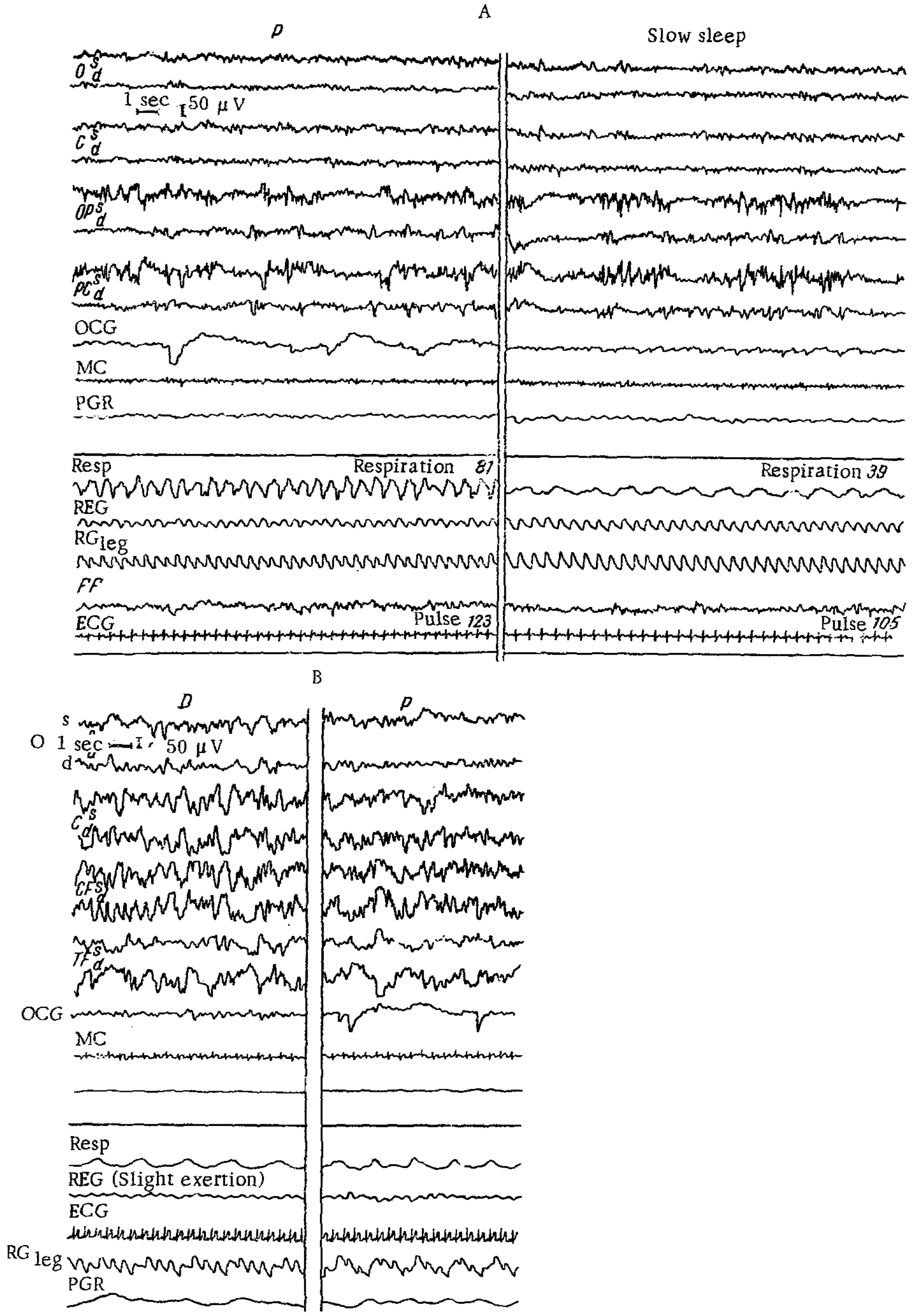

Fig. 3. Polygraphic picture of slow and fast sleep in a child aged 3.5 months with hydrocephalus and infantile cerebral palsy manifested as severe spastic diplegia (A) and in healthy infants of the same age (B). A) Pathology of sleep preventing identification of its stages of the EEG. Differentiation of slow and fast sleep is possible only from the oculogram, respiration, and rheographic indices; B) slow and fast sleep of a healthy infant aged 3.5 months. Remainder of legend as in Figs. 1 and 2.

of sleep, moreover, are more stable than the EEG. This agrees to some extent with the observations of Harmon and Emde [7], who found an intact cyclic organization of sleep in a microcephalic. It is important to note that in those rare cases of pathology when in general it was impossible to judge the structure of sleep even from a combination of indices, the behavioral pattern of sleep was still preserved.

The different components of the functional complex characterizing the infant's sleep may thus be affected in isolation. This confirms the hypothesis of their relative autonomy and is in agreement with data in the literature [9, 12, 13, 15].

The facts described above show that the various functional changes regularly manifested in the dynamics of sleep and analyzed during polygraphic investigation are only special manifestations of it. Changes in the EEG, despite all their importance and their evident connection with emotional and psychological activity (as a rule defects of prespeech and mental development were observed in infants with the severest distrubances of their sleep EEG), are no exception in this respect. They reflect symbolically as it were the events taking place on a certain "effector field," meaning the cerebral cortex in this case, and they enable the state of some of the deep brain formations to be judged indirectly. Like the dynamics of the autonomic and motor indices, they do not shed light on the mechanism that is the prime mover, determining the dynamics of sleep from its beginning to its end and responsible for the qualitatively unique integration of functions of the whole body, i.e., they do not shed light on the physiological essence of sleep. Comparison of the various physiological manifestations of sleep during its course must improve the chances of assessing the role of particular brain structures in the formation of the stages and the differentiation of these stages, but it does not solve the fundamental problem of the nature of the phenomenon.

A correct idea of the biological function of sleep, on the basis of which a hypothetical model of the functional system responsible for sleep, with the aid of physiological mechanisms that have largely been studied already, can be created must offer some guidance to the way ahead.

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