Overnight Recordings of Intracranial Pressure and Electroencephalography in Neurosurgical Patients

Part II : Changes in Intracranial Pressure during Sleep

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Abstract : Changes in intracranial pressure (ICP) during sleep were investigated in 37 patients with chronic intracranial hypertension or chronic hydrocephalus, in whom episodic pressure waves characterized by A-waves or episodic B-waves were seen in Part I of this paper. The patients were conscious, and sleep stages including REM sleep were observed in all of them. During non-REM sleep, ICP significantly rose in Stage II on many occasions, and was always lower in Stage IV than in other sleep stages. A marked elevation of ICP was seen in REM sleep. ICP changes during REM sleep were characterized by frequent appearances of the pressure waves; 88.9% of A-waves and 95.1% of episodic B-waves appeared during REM sleep. The initiation of REM sleep scored on EEG began 1 to 2 minutes prior to or at the onset of the episodic pressure waves. The episodic pressure waves are assumed to be induced by the intracranial conditions in REM sleep, when increased brain activity and reduced sympathetic tone are known to occur simultaneously.

Key words : ICP, pressure wave, REM sleep.

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Introduction

The evidence that intracranial pressure (ICP) rises during sleep has been well documented in both clinical (Di Rocco *et al.*, 1975; Pierre-Kahn *et al.*, 1976) and experimental (Gücer & Viernstein, 1979; Hirsch *et al.*, 1978) studies, since Cooper and Hulme first demonstrated it polygraphically in 1966. This evidence, particularly the episodic appearance of pressure waves called A- and B-waves during rapid eye movement (REM) sleep, has been applied to reveal occult intracranial hypertension in normal pressure hydrocephalus and to select candidates for shunting operations (Di Rocco *et al.*, 1976; Tamaki *et al.*, 1983). However, no study has been made regarding the extent of the correlation of pressure waves with REM sleep, and only a few papers have referred to the time relation between pressure waves and REM sleep (Gücer & Viernstein, 1979; Munari & Calbucci, 1981; Pierre-Kahn *et al.*, 1976). The question whether REM sleep precedes the pressure waves is considered to be important in determining the pathogenesis of the episodic pressure waves. In our study, quantitative as well as temporal correlations between epi-

sodic pressure waves and REM sleep were investigated to answer the question whether these two episodic phenomena have a causal relationship. This study was also performed to determine ICP changes during non-REM sleep stages. A few investigators have mentioned these changes in their papers but with discordant results (Cooper & Hulme, 1966; Di Rocco *et al.*, 1975; Munari & Calbucci, 1981).

Clinical Materials and Method

Among 85 neurosurgical patients with intracranial hypertension and/or ventricular dilation in whom overnight recording of ICP and electroencephalography (EEG) was simultaneously performed and explained in Part I of this paper (Yokota *et al.*, 1989), 37 patients, in whom episodic pressure waves characterized by A-waves and episodic B-waves were observed, were investigated further to analyze ICP changes during sleep. Patients with A-waves consisted of 15 patients ranging in age from 3 months to 65 years, 10 with brain tumors (7 children and 3 adults), 3 with infant or child non-tumorous hydrocephalus, one with adult hydrocephalus developed in the subacute phase of subarachnoid hemorrhage (SAH), and one adult with benign intracranial hypertension. The average age of these patients was 20.6 years. The consciousness of the patients at the time of recording ranged from alert to slightly drowsy.

Patients with episodic B-waves consisted of 22 patients ranging in age from 8 to 75 years, 9 with postoperative brain tumors, 12 with chronic communicating hydrocephalus after SAH or of idiopathic origin, and one with Chiari type I malformation. The patients were all conscious. Their average age was 47.8 years.

Methods of recording ICP and EEG were described in Part I.

Overnight profiles of ICP and sleep stages were diagrammed for each patient. Sleep stages were scored visually using the criteria of Rechtschaffen & Kales (1968). Correlations between changes in ICP and sleep stages were based on the ICP-sleep stage diagrams. Time relations concerning the initiation of REM sleep and episodic pressure waves were investigated using data replayed from a magnetic tape.

Results

ICP changes during sleep. ICP changes in accordance with sleep stages were clearly observed in patients with A-waves (Fig. 1a, b) and in some patients with episodic B-waves whose resting pressure was higher than normal (Figs. 2, 3a, b). On many occasions ICP in the light sleep stages (Stage I, II) was higher than that in the deep sleep stages (Stage III, IV), but ICP in Stage III remained as high as that in Stages I and II in some cases (Fig. 3b). However, ICP in Stage IV was always lower than that in the other sleep stages (Fig. 3b), except when ICP subsequent to the episodic pressure waves transiently dropped below the resting pressure. Depending on the case, ICP in non-REM sleep was both

Fig. 1a. Sleep stage diagram (upper column) and overnight ICP profile (lower column) in a 4-year-old boy with optic-hypothalamic glioma. Episodic pressure waves (A-waves) repeatedly appear coinciding with REM sleep stage.

higher and lower than ICP in the waking state (Figs. 1a, 2, 3a). Thus no definite conclusion concerning changes in ICP according to waking and sleep stage could be reached. In REM sleep, however, ICP rose abruptly, forming A-waves or episodic B-waves in the majority of cases (Figs. 1a, 2, 3a).

Correlation between pressure waves and REM sleep. In patients with A-waves, the average frequency of A-waves and REM stages per night per person were 5.73 and 5.55, respectively. Further, 88.9% of the total numbers of A-waves appeared in accordance with REM sleep, 7.9% occurred during waking and 3.2% occurred in Stage II. A-waves never occurred in Stage III or IV (Fig. 1a, b). On the other hand, REM were associated with A-waves at a rate of 91.8%, and the other REM were associated with B-waves in this group. In patients with episodic B-waves, episodic B-waves and REM stages appeared 3.15 and 4.0 times on average, respectively, per night per person. Ninety-five point one percent of episodic B-waves occurred during REM sleep, and only 4.9% of episodic B-waves appeared in Stage II and during waking (Figs. 2, 3a). REM accompanied episodic B-waves at a rate of 75.0%, while other REM occurred without any pressure changes.

Time relation between the initiation of REM and pressure waves. In the subjects investigated above, the interrelation between REM and pressure waves was based on data replayed from the data recorder which were stored on magnetic tapes. The initiation of REM on EEG usually occurred 1 to 2 minutes prior to the initiation of A-waves (Fig. 1b) or just at the ascending phase of the plateau. Episodic B-waves also occurred 1 to 2 minutes after the initiation of REM (Fig. 3b).

Fig. 1b. Magnified recording of ICP and real-time electroencephalogram (EEG) recordings at the point of time indicated by an asterisk in Fig. 1a. ICP recording shows the initiation of A-(plateau) wave. REM stage characterized by electrooculogram (EOG) discharges (3) appear markedly at the plateau of A-wave, however, REM stage scored on EEG (2) begins about 2 minutes prior to the ascending slope of A-wave (EMG: electromyogram).

Fig. 2. Sleep stage diagram (upper column) and overnight ICP profile (lower column) in 45-year-old woman who had been operated on for acoustic tumor 6 days before this recording. Episodic B-waves appear in REM sleep stage and in Stage II sleep. ICP in sleep stages is higher than that in waking.

Fig. 3a. Sleep stage diagram (upper column) and overnight ICP profile (lower column) in a 10-year-old boy with a pineal tumor. Episodic B-waves appear in REM sleep and in light sleep. ICP in waking is as high as in sleep stages in this patient.

The end of REM did not necessarily match the completion of the plateau waves; REM remained after plateau waves had been transformed into the resting pressure level on some occasions or terminated at the plateau on other occasions. When episodic B-waves had almost disappeared, REM remained.

Discussion

ICP has been found to be higher during sleep than waking (Cooper & Hulme, 1966; Di Rocco *et al.*, 1975; Gücer & Viernstein, 1979), subject to some considerations. ICP is definitely higher in sleep than waking in the REM stage of patients with episodic pressure waves (Munari & Calbucci, 1981; Renier *et al.*, 1982) and in patients with continuous high pressure B-waves in whom alternating light sleep and waking EEG were observed in accordance with periodic respiration (Ingvar & Lundberg, 1961; Yokota *et al.*, 1989). However, no elevation of ICP has been observed during sleep in patients with low resting pressure (Munari & Calbucci, 1981; Yokota *et al.*, 1989) and the elevation of ICP during non-REM sleep was not significantly higher than that in waking in the present study. Concerning ICP during non-REM sleep, Cooper & Hulme (1966) and Munari & Calbucci (1981) observed that ICP rose in Stage II sleep in patients with chronic intracranial hypertension or chronic hydrocephalus, but Di Rocco *et al.* (1975) reported that ICP rose more obviously in slow wave sleep than in light sleep in infantile hydrocephalic patients. In the present study the authors observed that ICP in Stage II sleep rose significantly compared with other non-REM sleep stages on many occasions. However, on some occasions ICP in

Fig. 3b. Magnified recordings of ICP and real-time EEG recordings at three points indicated by the asterisks in Fig. 3a. The upper two columns (*) show that ICP in Stage III sleep (second arrow) remains as high as that in Stage II sleep (first arrow). The middle two columns (**) show that REM sleep stage appears at the initiation of the cluster of episodic B-waves (second arrow) and an arousal pattern on EEG appears when episodic B-waves continue (third arrow). The lower two columns (***) show that ICP in Stage IV (third arrow) is lower than that in Stage II (first arrow) and in Stage III (second arrow).

Stage III remained as high as that in Stage II. ICP in Stage IV was always lower than in the other sleep stages as has been previously described (Rossi *et al.*, 1975). The most significant elevation of ICP is seen in REM sleep (Cooper & Hulme, 1966; Di Rocco *et al.*, 1975; Munari & Calbucci, 1981).

ICP changes during REM sleep are characterized by the frequent appearance of episodic pressure waves, rather than by gradual elevation of the resting pressure. The present study showed that 88.9% of A-waves and 95.1% of episodic B-waves were observed in REM sleep during overnight recording. This high incidence of episodic pressure waves in REM sleep in clinical cases coincides well with the study in experimental animals conducted by Hirsch *et al.* (1978), in which 88% of short-lasting oscillations of ICP were seen in REM sleep. Such high correlation between REM sleep and episodic pressure waves suggests a causal relationship between them. REM sleep has been reported to begin at the onset of pressure waves or 40 to 60 seconds later at the ascending slope of ICP (Gücer & Viernstein, 1979; Munari & Calbucci, 1981; Pierre-Kahn *et al.*, 1976). These reports on the time relation between the initiation of episodic pressure waves and REM sleep are in close accord with our observation that REM sleep as scored on EEG started 1 to 2 minutes prior to or at the beginning of episodic pressure waves, since electro-oculographic discharges characteristic of REM usually lag 1 to 2 minutes behind the initiation of the REM stage on EEG.

The clinical observations stated above suggest that the episodic pressure waves might be induced by intracranial conditions in the REM stage, when increased brain activity and reduced sympathetic tone are known to occur simultaneously (Katayama *et al.*, 1984). The mechanism of ICP increase in the REM stage is assumed to be caused by the increase of the cerebral blood volume associated with vasodilatation, since the speed and magnitude of the pressure change in episodic pressure waves are so pronounced (Gücer & Viernstein, 1979). In fact, a rapid and marked augmentation of the cerebral blood volume has been demonstrated in REM sleep (Risberg & Ingvar, 1973; Seylaz *et al.*, 1975). Increase of the cerebral blood volume is postulated due to an increase in cerebral metabolism (Reivich *et al.*, 1968), which has been confirmed to occur in REM sleep (Cooper & Hulme, 1966; Meyer & Toyoda, 1971). In addition to this augmented cerebral metabolic activity, changes in the nervous control of the tone of the cerebral vessels such as reduced sympathetic tone (Futuro-Note & Coote, 1982) are also considered to play an important role in the increase of cerebral blood volume in the REM stage (Hirsch *et al.*, 1978; Katayama *et al.*, 1984).

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脳神経外科疾患における睡眠時頭蓋内圧波の分析 そのⅡ．睡眠時の頭蓋内圧の変動

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要 旨： 睡眠時の頭蓋内圧の変動を詳細に検討するために，本研究そのⅠで突発性圧波がみられた37人を対象とした．これらの患者では睡眠周期および圧波の変動がよく観察された．Non-REM 睡眠期では，多くの場合Ⅱ期に有意な頭蓋内圧の上昇がみられたが，Ⅲ期においてもⅠ期，Ⅱ期と同様の頭蓋内圧の上昇をみることもあった．しかし，Ⅳ期では常に頭蓋内圧は他の睡眠段階よりも低値であった．最も頭蓋内圧が上昇するのはREM 睡眠期で，この時期の頭蓋内圧の上昇は突発性に出現する圧波の形成によって特徴づけられる．今回の記録で出現したA波の88.9%およびepisodic B波の95.1%がこのREM 期に一致して出現した．また，これらの突発性圧波とREM 睡眠期の開始時間を検討すると，REM 期の脳波所見が圧波の始まりに先行することが分かり，圧波形成にREM 期の頭蓋内環境が関与するものと推定された．

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