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The Relation of Intracranial Pressure B-Waves to Different Sleep Stages in Patients with Suspected Normal Pressure Hydrocephalus

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Summary

The interpretation of data from continuous monitoring of intracranial pressure (ICP) in patients with suspected normal pressure hydrocephalus (NPH) is the subject of controversy. Despite the fact that overnight ICP monitoring is widely used for the diagnosis of NPH, normative criteria are poorly defined. The present study demonstrates that there is a relationship between the relative frequency, the absolute amplitude, the wavelength and the morphology of B-waves and different sleep stages.

Intraventricular intracranial pressure was recorded continuously overnight in 16 patients with suspected normal pressure hydrocephalus. Simultaneous polysomnography was performed to investigate the relation of spontaneous ICP oscillations to different sleep stages. A correlative analysis was done with the data of 13 patients. Three patients were excluded, one who was awake throughout the night and two in whom polysomnography was incomplete due to technical reasons. The mean resting cerebrospinal fluid (CSF) pressure was 12.87 cm CSF. B-waves were observed in the ICP recordings of all patients. They were present for a mean of 72% of the total recording time. The relative frequency of B-waves was higher during REM sleep and sleep stage 2 as compared to wakefulness (87.8% and 83.2% vs. 56, $p < 0.05$). The absolute amplitude was higher during REM sleep than in wakefulness (9.56 vs. 3.44 cm CSF, $p < 0.05$). Wavelengths were longer in REM sleep than in wakefulness and stages 1 and 2 (62.4 vs. 42, 40.7 and 44.8 sec, $p < 0.05$). The morphology of B-waves was also related to different sleep stages. Ramp-type B-waves were associated with REM sleep in six patients, however, were also present in sleep stage 2 in three of them.

Knowledge of the relation of spontaneous ICP oscillations to different sleep stages may help to establish physiological foundations and alterations. Furthermore, polysomnography may be useful to avoid erroneous interpretation of ICP recordings due to sleep stage related variability.

Keywords: B-waves; intracranial pressure; normal pressure hydrocephalus; sleep.

Introduction

The diagnosis of normal pressure hydrocephalus (NPH) will be considered in patients with gait disturbance and/or incontinence and progressive dementia associated with ventricular enlargement. Numerous reports have demonstrated the beneficial effects of cerebrospinal fluid (CSF) shunting in this condition [1, 4, 7, 8, 36]. However, since postoperative success rates varied considerably in different series, various investigations were introduced to find more reliable diagnostic criteria for NPH and to select patients who will respond to subsequent shunt surgery [4, 16, 36, 41, 45]. Nevertheless, to establish the diagnosis of NPH continues to be a challenge for clinical neurology and neurosurgery [39, 47, 48].

Continuous recording of intracranial pressure (ICP) is thought to be one of the most reliable methods for confirming the diagnosis of NPH [6, 7, 16, 39, 41, 44]. Continuous ICP recording provides information on the mean resting ICP and reveals intermittent rises in ICP. Spontaneous rhythmic oscillations of the ICP ranging from 0.5–2/min with variable amplitudes were classified as B-waves by Lundberg [23]. A high relative frequency of B-waves was shown to be a good prognostic indicator for shunt responsiveness [4, 16]. However, it remains unclear, which percentage of time B-waves are present, should be considered as pathological [5, 7, 16, 36, 39, 41]. It has also been proposed that B-waves may be physiological, since they were found in apparently healthy persons [27]. Furthermore, spontaneous rhythmic oscillations of cerebral blood flow velocity resembling B-waves were detected in healthy young persons [11, 13, 28]. There is also disagreement about the diagnostic value of amplitude and morphology [6, 7, 19, 36, 38]. Recently, Raftopoulos *et al.* suggested to shunt only those patients whose ICP recordings show B-waves with an amplitude of more than 9 mmHg [39]. B-

waves with an asymmetrical, gradually increasing and rapidly decreasing, high amplitude have been called ramp waves or intermediate waves and are thought to be of special significance in predicting outcome after CSF diversion [6, 38].

Sleep has been repeatedly reported to induce ICP alterations [9, 34]. Differences in mean ICP, the appearance of plateaus and also of B-waves were found to be associated with different sleep stages in various clinical conditions including NPH [3, 10, 15, 22, 25, 30, 31, 35, 37, 45]. B-waves were shown to occur more frequently in REM sleep [49]. However, to our knowledge no previous study has quantified the variations of B-waves related to different sleep stages. The knowledge of these proportional assessments may be helpful for understanding and interpreting data from continuous ICP monitoring. The present study was designed to investigate the relation of relative frequency, amplitude, wavelength, and morphology of B-waves to different stages of sleep using concomitant ICP monitoring and polysomnography.

Patients and Methods

Patient Selection for ICP Recording and for Shunt Surgery

Several diagnostic tests are performed in our hospital to select patients with a diagnosis of suspected NPH for shunt operations. The patients are referred either from the department of neurology ofr from neurologists in private practice. Those patients from the department of neurology who have shown unequivocal and repeated improvement of clinical symptoms after removal of 30–50 ml CSF after lumbar puncture on several occasions will be scheduled for shunt surgery without pre-operative ICP monitoring. Ancillary investigations include non-invasive techniques like dynamic magnetic resonance imaging and transcranial Doppler monitoring. All other patients who are assumed to be possible candidates for a shunt procedure according to their clinical presentation and neuroradiological findings will undergo continuous ICP monitoring. The ICP recording is thought to be compatible with the diagnosis NPH when the base line ICP is in the upper normal range, B-waves are present throughout most of the recording time (70–100%) and B-waves with an amplitude of 10 cm H_2O are observed at least during 5–10% of the recording time. However, the decision making to schedule a patient for a shunt operation also considers the results of hydrodynamic studies (pressure-volume-index and resistance to outflow) as well as the effect of intraventricular removal of CSF on clinical symptoms. We do not primarily exclude patients with concomitant vascular encephalopathy.

Patient Population

All patients were videotaped at the time of admission. For each of the cardinal symptoms the severity was assessed on a four-point scale. Sixteen consecutive patients with suspected NPH according to clinical symptoms and neuroradiological demonstration of hydrocephalus were examined by simultaneous continuous overnight ICP monitoring and polysomnography. Computed tomography was performed in all patients and magnetic resonance (MR) imaging in thirteen.

No changes in the routinely administered medication were made prior and during ICP monitoring. Additional sedation or sleep inducing drugs were given in only one instance.

ICP Monitoring and Polysomnography

The study was approved by the local clinical ethics committee. All patients and/or their relatives gave informed consent. ICP recordings were performed for at least twelve hours overnight using a polyethylene catheter of 2 mm diameter. The tip of the catheter was placed into the frontal horn of the left lateral ventricle under local anaesthesia via a hand-drilled 2 mm burr hole located 2 cm lateral to the midline and 2 cm rostral to the coronal suture. The catheter was secured to the scalp with sutures and directly connected to a pressure transducer (Braun, Germany) fixed over the bregma. The transducer was coupled to an amplifier (Servomed, Hellige, Germany) and a plotter (Recomed, Hellige, Germany), which was operated on line at a paper speed of 30 cm/h. The transducer was calibrated without loss of CSF in each patient. The foramina of Monro were considered to be at 0 cm CSF when the patient was in a supine position with the head horizontal. An observer was present throughout the recording time and noted any movements of the patient. In two patients the monitoring included simultaneous continuous transcranial Doppler (TCD) measurements of middle cerebral artery blood flow velocity as described in detail elsewhere [11–13, 21]. At the end of the night hydrodynamic studies were performed. Before the ventricular catheter was taken out, 20 ml of intraventricular CSF were removed. One patient developed meningitis two days after ICP monitoring, which was treated by antibiotics without residual symptoms. There were no further adverse effects.

Polysomnography was performed with an ambulant monitoring device (Walter, Germany) which allows recording of 8 channels. The recordings included registration of electroencephalogram (C3–A2 and C4–A1), horizontal electro-oculogram (two channels), submental electromyogram (two channels), electrocardiogram and respirogram. The following filter settings were used: electroencephalogram – sensitivity 7 μV/mm, time constant (TC) 0.3 sec, high frequency filter (HI) 70 Hz; electro-oculogram – sensitivity 30 μV/mm, TC 1.0 sec, HI 35 Hz; electromyogram – sensitivity 5 μm/mm, TC 0.03 sec, HI 500 Hz. The data were stored on a cassette recorder. Lights out time ranged from 21 : 30 – 23 : 00 and lights on time from 6 : 30 – 7 : 00.

Temporal compatibility of the sleep recording and the ICP monitoring was obtained by adjusting the exact time of the recordings at the beginning of the measurements.

Evaluation of Data and Statistical Analysis

Sleep recordings were scored by experienced raters not privy to the findings of the ICP recordings according to standard criteria [40]. Assessment of the ICP recordings were also done without knowledge of the sleep stage and the clinical features of the patient. The mean resting ICP in the absence of spontaneous oscillations was measured at the beginning of the night. The relative frequency of B-waves (%) in the overnight ICP recording was assessed off line according to the definition by Lundberg (spontaneous ICP oscillations with a wavelength between 0.5 and 2 min clearly discernible from the baseline recording). To evaluate the proportion of B-waves with respect to different sleep stages the relative frequen-

cy (%) of these oscillations was assessed in the two longest periods of each sleep stage in the individual recordings. Furthermore, in each recording the wavelength (sec) and the amplitude (cm CSF) of two B-waves per sleep stage were determined. For these measurements the first visible oscillations with a wavelength between 0.5 and 2 min next to the centre of the two longest sleep stages were used. For further analysis we connected the corresponding values for absolute amplitude, wavelength and relative frequency of B-waves of identical sleep stages. Furthermore, the complete ICP recordings were reviewed for the presence of ramp-type B-waves again without knowledge of the sleep stage. In the absence of an unanimous definition of what exactly constitutes a ramp-type we used the following criteria: 1. the apex of the oscillation amounts to at least twice as much as the baseline ICP, however, at least 30 cm CSF, and 2. the increase in the ascending slope is slow and the subsequent decrease rapid with the apex located in the last fifth of the wavelength. The presence of ramp-type B-waves was then correlated with the sleep stage.

Differences between the samples with respect to different sleep stages were assessed using Bonferroni-adjusted-t-tests. Statistical significance was declared at the $p < 0.05$ level. To avoid repeated measures we took the mean values of the two longest periods of each sleep stage for the Bonferroni-adjusted t-tests.

Results

Clinical Features

Ten patients were male and six were female. The demographic and clinical data are summarized in Table 1. Gait disturbance had been the primary symptom in nine patients. In all patients but two gait disturbance was the leading symptom at the time of the pressure recording. Signs of vascular encephalopathy were present in the MR scans of 9/13 patients.

The patients were under treatment with a variety of drugs including inotropic, anti-arrhythmic, antihypertensive, diuretic, antiplatelet, antithyroid, hypolipaemic and hypoglycaemic agents, antacids, anticoagulants, anticonvulsants and muscle relaxants. Three patients were under treatment with psychotherapeutic drugs; one each received flunitrazepame, amitriptyline, and dibenzepine and pipamperone. One patient who became anxious and restless during the recording was sedated with promethazine and levomepromazine.

Table 1. *Clinical and Demographic Data of 16 Patients with Suspected Normal Pressure Hydrocephalus*

Age at investigation (years)		
Mean	63	
Range	23–80	
Mode (decade)	70–80	
Aetiology of suspected NPH		
Idiopathic	13/16	(81%)
Meningitis	1/16	(6%)
Head trauma	1/16	(6%)
Cerebral radiation therapy	1/16	(6%)
Clinical symptoms		
Gait disturbance	16/16	(100%)
Urinary incontinence	13/16	(81%)
Cognitive deficits	15/16	(94%)
Complete triad	12/16	(75%)

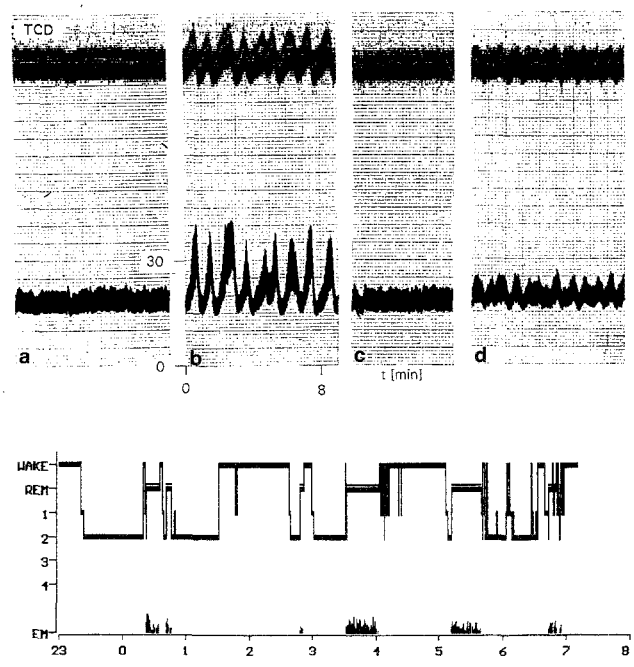

Fig. 1. Sectors of simultaneous ICP and TCD recordings during different sleep stages (top) and sleep stage diagram (bottom) of a 73-year-old male patient with suspected NPH. *a* wakefulness, *b* REM, *c* sleep stage 1, *d* sleep stage 2. *EM* eye movements

After completing the investigations the diagnosis was confirmed in ten patients. Seven patients were submitted to subsequent shunt operations. One patient refused shunt surgery. Ventriculo-peritoneal or ventriculo-atrial shunts were used. Clinical symptoms were improved in all patients except one on postoperative follow-up examinations ranging from four to six months. No complications occurred.

Polysomnography

Complete polysomnographic recordings were obtained in 14 patients. Examples are demonstrated in Figs. 1 and 2. The recordings of two patients were incomplete due to technical problems during the recording period and their data were excluded from further analysis. One patient was awake throughout the recording time. Thus, thirteen polysomnographic recordings were available for correlative analysis

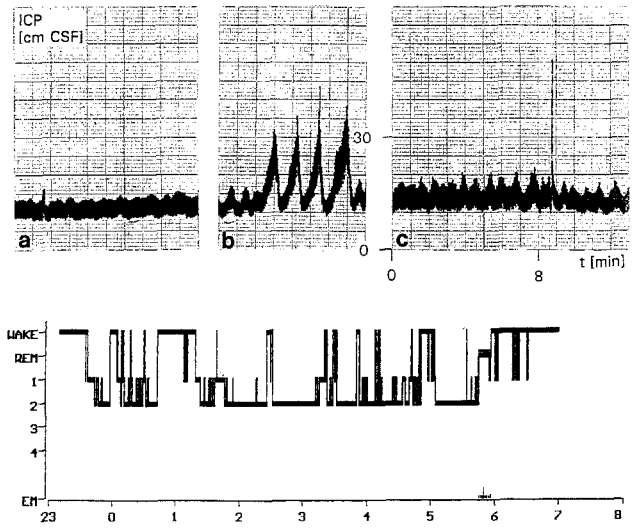

Fig. 2. Sectors of ICP recordings during different sleep stages (top) and sleep stage diagram (bottom) of a 74-year-old male patient with suspected NPH. *a* wakefulness, *b* REM, *c* sleep stage 2. *EM* eye movements

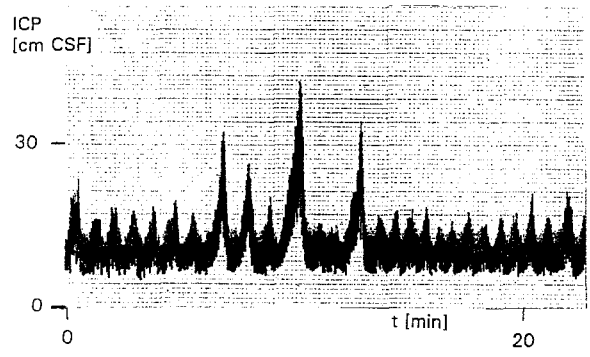

Fig. 3. Sector of the ICP recording of a 79-year-old male patient with suspected NPH. The trace shows the transition from sleep stage 2 to REM which is followed by the awakening of the patient. The large ramp-type B-waves are related to REM sleep. The morphology of the B-waves of sleep stage 2 (on the left side) is different from that of the waking state (on the right side)

with the data of the ICP measurements. All these patients had episodes of varying time of sleep stages 1 and 2. Short intervals of sleep stage 3 were present in six patients, however, sleep stage 4 in only two. These two patients were 28 and 44 years old, respectively. REM sleep appeared in nine patients with one to five episodes per person. Episodes of REM sleep varied from 1 to 30 minutes.

ICP Recordings

The mean baseline resting pressure was 12.87 ±/– 4.48 cm CSF during wakefulness with a

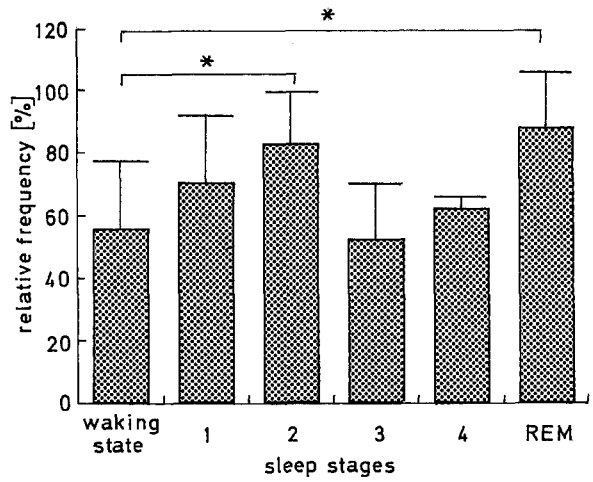

Fig. 4. Relative frequency of B-waves in different sleep stages. Mean values and standard deviations. * Indicates a statistically significant difference ($p < 0.05$)

range from 9 to 18 cm CSF. B-waves were present in the recordings of all patients (Figs. 1–3). Sleep related variations of B-waves were observed in all recordings, both in patients in whom the diagnosis of NPH was confirmed and in patients in whom it was not. The ICP pulsatility index and the sleep related variations of the amplitudes of B-waves tended to be higher in those patients who were scheduled for shunt operations. The relative frequency of B-waves of the total recording time varied from 20% to 96%, with a mean of 72%. Only one patient had less than 50% B-waves. The mean ICP in different sleep stages in the absence of spontaneous oscillations was 13.31 ± 4.44 cm CSF in sleep stage 1, 12.64 ± 3.71 cm CSF in sleep stage 2, 13.25 ± 3.18 cm CSF in sleep stage 3, 10.75 ± 3.18 cm CSF in sleep stage 4, and 12.56 ± 2.89 cm CSF during REM sleep. These differences were not significant ($p > 0.05$).

Relative Frequency of B-Waves in Sleep Stages

The relative frequency of B-waves was related to different sleep stages. Figure 4 shows the connected mean values and standard deviations in different sleep stages. The relative frequency of B-waves was significantly higher during REM sleep (87.8%) and sleep stage 2 (83.2%) as compared to wakefulness (56%) according to Bonferroni-adjusted t-tests ($p < 0.05$). The greatest fluctuations of the relative frequency from patient to patient were found during wakefulness and sleep stage 1 varying from 24% to 90% and from 26% to 100%, respectively. The relative frequency ranged from 50% to 100% in sleep stage 2, and from 48% to 100% in REM sleep.

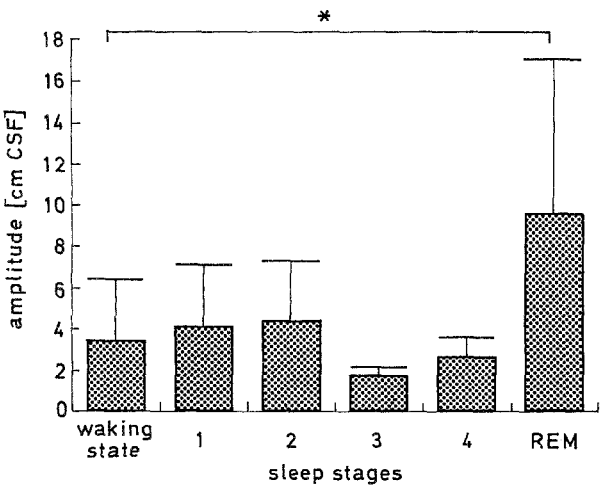

Fig. 5. Absolute amplitude of B-waves in different sleep stages. Mean values and standard deviations. * Indicates a statistically significant difference ($p < 0.05$)

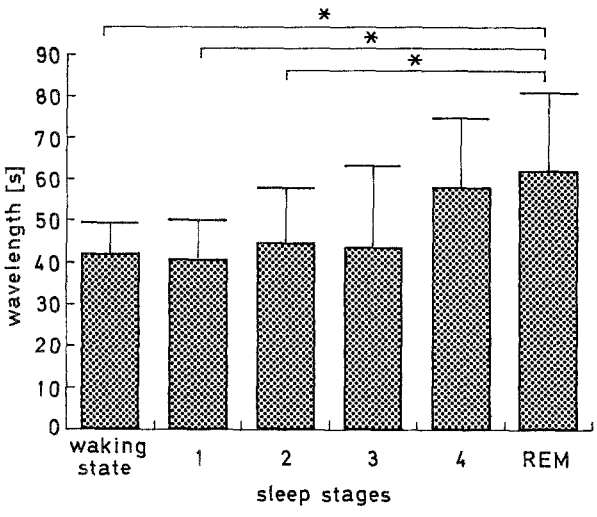

Fig. 6. Wavelength of B-waves in different sleep stages. Mean values and standard deviations. * Indicates a statistically significant difference ($p < 0.05$)

Absolute Amplitude and Wavelength of B-Waves in Sleep Stages

The height of the absolute amplitude differed with sleep stages. Figure 5 shows the connected mean values and standard deviations for the absolute amplitude in different sleep stages. Amplitudes of B-waves were significantly higher during REM sleep (9.56 cm CSF) as compared to wakefulness (3.44 cm CSF) according to the Bonferroni-adjusted t-tests ($p < 0.05$). Absolute amplitudes during wakefulness varied from 1 to 12 cm CSF. The greatest variations of the absolute amplitude ranging from 3 to 41 cm CSF were found in REM sleep. This is explained by different heights of amplitudes from patient to patient, as well as by the finding that both high and low amplitude B-waves could be present in the individual patient during REM sleep.

The wavelength of B-waves was also related to different sleep stages. Figure 6 demonstrates the connected mean values and standard deviations of the wavelength in different sleep stages. B-waves were significantly longer during REM sleep (62.4 sec) than during wakefulness (42 sec) and sleep stages 1 (40.7 sec) and 2 (44.8 sec) according to the Bonferroni-adjusted t-tests ($p < 0.05$). Wavelength varied from 32 to 55 sec during wakefulness. Again, as with the absolute amplitude the greatest differences of wavelength were seen in REM sleep with a range from 33 to 100 sec.

Morphology of B-Waves in Sleep Stages

Sleep stage had an effect on the morphology of B-waves (Figs. 1–3). There was a marked difference of the morphology of B-waves in different sleep stages in individual patients. In many instances transition from one sleep stage to another resulted in prompt changes in the appearance of B-waves (Fig. 3). While the absolute amplitude could not be predicted from the parallel TCD recordings, the ramp-type B-wave equivalents could be easily identified (Fig. 1). Ramp-type B-waves were present in six patients associated with REM sleep. They were present at the start of REM sleep in three patients, while they appeared after a delay of several seconds in two patients and after a delay of 60 sec in one. During REM sleep, ramp-type B-waves occurred in sequences consisting of two to ten waves, which were occasionally interrupted, however, by smaller, more symmetrical waves. Four patients had runs of ramp-type B-waves during REM sleep with a build-up of subsequent increasing amplitudes (Fig. 2 b). Two patients had single, interspersed ramp-type B-waves during sleep stage 2. Another patient had episodic groups of subsequent ramp-type B-waves while sleep fluctuated between sleep stage 1 and 2, however, without a build-up phenomenon of the amplitude. In those patients who had ramp-type B-waves during REM sleep and during stage 2 as well the maximal amplitude of ramp-type B-waves was lower in sleep stage 2 as compared with REM sleep with ratios (REM/stage 2) of 2.2, 1.6, and 1.2 for the relative amplitude.

Discussion

Optimistic views on the outcome of patients with suspected NPH after shunting operations have been challenged by the recent reports of Vanneste and colleagues [47, 48]. On the other hand it has been shown repeatedly that a high clinical improvement rate can be expected with adequate pre-operative selection of these patients [4, 7, 16, 38]. The need to improve the pre-operative assessment of these patients has led to a "reappraisal" of continuous ICP monitoring [39, 41]. Although the method is widely accepted as a valuable tool in selecting patients suitable for shunt operations, knowledge about the physiological mechanisms which are involved in the generation of spontaneous ICP oscillations is limited and normative criteria still remain to be defined.

Our study demonstrates that there is a relationship between the relative frequency, the amplitude, the wavelength and the morphology of B-waves and different sleep stages. The relative frequency of B-waves is clearly higher in REM sleep, and to a lesser degree in sleep stage 2, than during wakefulness. The amplitudes are significantly higher in REM sleep than during wakefulness, and the oscillations have a longer wavelength during REM than during wakefulness and sleep stages 1 and 2. However, we did not detect significant changes in the baseline ICP during different sleep stages.

It is not known whether and to what extent sleep in patients with suspected NPH is disturbed or not. There are conflicting data concerning sleep patterns. Munari and Calbucci also observed that slow wave sleep was almost absent [31]. Furthermore, periods of REM sleep were extremely rare in their series of 14 patients. Paucity of REM sleep was also found by Ogashiwa and Takeuchi, who did not detect episodes of REM sleep in five out of seven patients with suspected NPH [35]. In contrast, Yokota *et al.* reported an average of four episodes of REM sleep per patient [49]. Kuchiwaki and colleagues on the other hand, described that sleep stage 2 was extremely rare in their patients [22]. Our data show that sleep stages 3 and 4 are rare in the majority of patients. This, however, may not be specific for NPH, since sleep stages 3 and 4 are generally reduced in elderly people [29]. Furthermore, the disturbance caused by the recording itself has to be taken into account in these patients who are investigated with concomittant ICP recording. Further studies are needed to evaluate characteristics of sleep profiles in patients with suspected NPH.

Raised ICP during REM sleep was detected in experimental animals [17]. Changes in ICP related to sleep have also been described in various clinical settings since Cooper and Hulme demonstrated intermittent ICP rises in REM and sleep stage 2 by polysomnography in 1966 [9]. They showed that plateau waves occurred most frequently during REM sleep and to a lesser degree in sleep stage 2 in patients with intracranial hypertension mainly due to cerebral tumours. Intermittent rises of ICP during REM sleep were also described in hydrocephalic children [10, 37].

After Symon and Dorsch had demonstrated spontaneous oscillations in overnight ICP recordings of patients with suspected NPH in 1972, ICP recordings during sleep were introduced in routine clinical practice [43]. However, concomitant polysomnography was performed only rarely in these patients. As in other clinical conditions intermittent rises in ICP were described in patients with suspected NPH during REM sleep [3, 25, 31, 35] and sleep stage 2 [31]. These studies, however, did not evaluate the appearance of B-waves. Yokota *et al.* demonstrated that the elevation of ICP during REM sleep is due to the frequent appearance of spontaneous oscillations rather than to gradual elevation of the baseline ICP [49]. These authors also investigated the correlation of B-waves and sleep in a heterogeneous group of patients with brain tumours, communicating hydrocephalus and Chiari malformation. They reported that 95.1% of B-waves occurred during REM sleep and 4.9% during sleep stage 2 and wakefulness. However, they conceivably concentrated their analysis to B-waves with higher amplitudes. Such B-waves accompanied 75% of the time during REM sleep.

Frequent B-waves in overnight ICP recordings are thought to indicate the diagnosis of NPH. However, in the absence of normative criteria and the lack of standardized evaluation the interpretation of the occurrence of B-waves in ICP recordings is controversial. The original description of Lundberg defined B-waves as "smaller, sharper waves (usual ranges: frequency 1/2–2 minutes, amplitude: from discernibility to 50 mm Hg)" [23]. According to this definition, spontaneous ICP oscillations with different amplitudes, wavelengths and morphology will constitute B-waves. However, it seems that not all authors strictly adhere to Lundberg's original definition of what should be considered to represent a B-wave. This renders interpretation and comparison of data from dif-

ferent reports even more difficult. Suggestions as to which proportional frequency of B-waves should be regarded as a pathological finding differ widely. Occasional B-wave activity has been thought to be not necessarily indicative of pathological CSF absorption [45]. Opinions are also divided as regards the amplitude and morphology of B-waves [38, 39]. Some authors have suggested that particularly ramp-type B-waves indicate NPH [6].

As demonstrated in our study, different types of B-waves occur in the same patient, dependent on the stage of sleep. The morphology of these oscillations is, at least partially, a function of amplitude and wavelength. Furthermore, ramp-type B-waves are largely associated with REM sleep.

The clinical relevance of these findings is evident. Despite the uncertainty as to what should be considered pathological or not, knowledge of the relations of ICP oscillations to different sleep stages may help to avoid erroneous or misleading interpretations of ICP recordings. Intra-individual and interindividual variability of the sleep pattern may result in quite distinct ICP recordings with other proportions and characteristics of B-waves. Disregarding these aspects, some patients who might benefit from a shunt operation will not be offered surgery. Ramp-type and other high amplitude B-waves, for example, may not be seen in patients without REM sleep during the investigation. Furthermore, patients with NPH might not be shunted because they have a "sound" sleep with predominant sleep stages 3 and 4 or because they did not sleep at all during the ICP monitoring. On the other hand, the investigator will be re-assured in his decision to refuse shunt surgery to a patient who did not show a high relative frequency of B-waves or high amplitude B-waves despite the fact he or she had an unremarkable sleep pattern with several cycles of REM sleep.

B-waves most probably are secondary to rhythmic alterations of cerebral blood flow [2, 12, 32]. Relative changes in cerebral blood flow velocities as measured by TCD are considered to reflect cerebral perfusion [19]. We have shown previously that B-waves are accompanied by spontaneous rhythmic oscillations of blood flow velocity in the middle cerebral artery in TCD recordings in patients with suspected NPH (B-wave equivalents) [12, 13, 21]. A nonlinear relationship between ICP and blood flow velocity is found [13]. As demonstrated in Fig. 1 of the present study, the morphology of ICP B-waves of different sleep stages can be mirrored in the TCD recordings. We have demonstrated the occurrence of B-wave equivalents also in healthy young adults [11]. It is remarkable that B-wave equivalents in healthy young adults are more frequent, are higher and longer during REM sleep [13]. However, it remains unclear whether B-wave equivalents in healthy persons are accompanied by concomitant ICP oscillations and whether there might also be alterations of amplitudes in different sleep stages. At least, it is conceivable that in patients with NPH reduced compliance may amplify transmission of periodic changes in cerebral blood flow into more marked ICP oscillations.

Nocturnal increase in B-waves and B-wave equivalents might be linked to altered cerebral blood flow and metabolism during sleep. Cerebral blood flow, cerebral glucose metabolism and the average cerebral metabolic rate based on oxygen uptake are reduced in synchronized sleep, while they increase during REM sleep [14, 20, 24, 26, 42, 46]. Furthermore, it has been demonstrated by MR with interleaved gradient-echo pulse sequences that the net flow of CSF through the cerebral aqueduct is higher at night in normal volunteers [33]. This probably indicates increased nocturnal production of CSF, which also may be associated with spontaneous oscillations of cerebral blood flow.

More studies are required to approach normative criteria for the interpretation of ICP recordings. Further studies should also evaluate the relationship between the sleep-ICP interaction and the outcome of surgery. Polysomnography may prove to be a useful tool to ascertain the interpretation of overnight ICP recordings. Knowledge of the relationships of sleep related ICP variations may help to avoid faulty analysis of ICP tracings secondary to disturbed sleep or variabilities in sleep patterns. We suggest that future ICP recordings should be accompanied by polysomnography.

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References

1. Adams RD, Fisher CM, Hakim S, Ojemann RG, Sweet WH (1965) Symptomatic occult hydrocephalus with "normal" cerebrospinal fluid pressure. A treatable syndrome. N Engl J Med 273: 117–126
2. Auer LM, Sayama I (1983) Intracranial pressure oscillations (B-waves) caused by oscillations in cerebrovascular volume. Acta Neurochir (Wien) 68: 93–100
3. Belloni G, Di Rocco C, Focacci C, Galli G, Maira G, Rossi GF

(1976) Surgical indications in normotensive hydrocephalus. A retrospective analysis of the relations of some diagnostic findings to the results of surgical treatment. Acta Neurochir (Wien) 33: 1–21
4. Black P McL, Ojemann RG, Tzouras A (1985) CSF shunts for dementia, incontinence, and gait disturbance. Clin Neurosurg 32: 632–651
5. Boergesen SE, Gjerris F, Soerensen SC (1979) Intracranial pressure and conductance to outflow of cerebrospinal fluid in normal-pressure hydrocephalus. J Neurosurg 50: 489–493
6. Brock M (1977) Klinik und Therapie des intermittierend normotensiven Hydrocephalus. Radiologe 17: 460–465
7. Cardoso ER, Piatek D, Del Bigio MR, Stambrook M, Sutherland JB (1989) Quantification of abnormal intracranial pressure waves and isotope cisternography for diagnosis of occult communicating hydrocephalus. Surg Neurol 31: 20–27
8. Chawla JC, Hulme A, Cooper R (1974) Intracranial pressure in patients with dementia and communicating hydrocephalus. J Neurosurg 40: 376–380
9. Cooper R, Hulme A (1966) Intracranial pressure and related phenomena during sleep. J Neurol Neurosurg Psychiatry 29: 564–570
10. Di Rocco C, McLone DG, Shimoji T, Raimondi AJ (1975) Continuous intraventricular cerebrospinal fluid pressure recording in hydrocephalic children during wakefulness and sleep. J Neurosurg 42: 683–689
11. Droste DW, Berger W, Schuler E, Krauss JK (1993) Middle cerebral artery blood flow velocity in healthy persons during wakefulness and sleep: a transcranial Doppler study. Sleep 16: 603–609
12. Droste DW, Krauss JK (1993) Simultaneous recording of cerebrospinal fluid pressure and middle cerebral artery blood flow velocity in patients with suspected symptomatic normal pressure hydrocephalus. J Neurol Neurosurg Psychiatry 56: 75–79
13. Droste DW, Krauss JK, Berger W, Schuler E, Brown MM (1994) Rhythmic oscillations with a wavelength of 0.5–2 min in transcranial Doppler recordings. Acta Neurol Scand 90: 99–104
14. Franzini C (1992) Brain metabolism and blood flow during sleep. J Sleep Res 1: 3–16
15. Furuse M, Kuchiwaki H, Nakaya T, Toyama K, Teraoka M, Hasuo M, Ikeyama A, Kageyama N (1980) Relationship between rapid ICP variation, respiratory pattern and wakefulness in neurological patients. In: Shulman K, Marmarou A, Miller JD, Becker DP, Hochwalt GH, Brock M (eds) Intracranial pressure IV. Springer, Berlin Heidelberg New York, pp 584–587
16. Graff-Radford NR, Godersky JC, Jones MP (1989) Variables predicting surgical outcome in symptomatic hydrocephalus in the elderly. Neurology 39: 1601–1604
17. Gücer G, Viernstein L (1979) Intracranial pressure in the normal monkey while awake and asleep. J Neurosurg 51: 206–210
18. Hajak G, Klingelhöfer J, Schulz-Varszegi M, Matzander G, Saunder D, Conrad B, Rüther E (1994) Relationship between cerebral blood flow velocities and cerebral electrical activity in sleep. Sleep 17: 11–19
19. Hamer J, Kühner A (1979) Mean intracranial resting pressure, episodic pressure fluctuations, and intracranial volume/pressure response in patients with subarachnoid hemorrhage. In: Beks JWF, Bosch DA, Brock M (eds) Intracranial pressure III. Springer, Berlin Heidelberg New York, pp 157–161
20. Heiss WD, Pawlik G, Herholz K, Wagner R, Wienhard K (1985) Regional cerebral glucose metabolism in man during wakefulness, sleep, and dreaming. Brain Res 327: 362–366
21. Krauss JK, Droste DW (1994) Predictability of intracranial pressure oscillations in patients with suspected normal pressure hydrocephalus by transcranial Doppler ultrasound. Neurol Res 16: 398–402
22. Kuchiwaki H, Kageyama N, Hirai N, Takada S, Inao S, Terashima M, Sugiura M (1984) A biological rhythm in a patient with normal pressure hydrocephalus – comparative studies in pre- and postoperative patients by a polygraphy. No To Shinkei 36: 911–916 (Jpn)
23. Lundberg N (1960) Continuous recording and control of ventricular fluid pressure in neurosurgical practice. Acta Psychiatr Neurol Scand [Suppl] 149: 1–193
24. Madsen PL, Schmidt JF, Wildschiodtz G, Friberg L, Holm S, Vorstrup S, Lassen NA (1991) Cerebral O_2 metabolism and cerebral blood flow in humans during deep and rapid-eye-movement sleep. J Appl Physiol 70: 2597–2601
25. Maira G, Di Rocco C, Rossi GF (1974) Pression intraventriculaire dans l'hydrocephalie à pression "normale" pendant la veille et le sommeil. Neurochirurgie 20: 462–467
26. Maquet P, Dive D, Salmon E, Sadzot B, Franco G, Poirrier R, von Frenckell R, Frank G (1990) Cerebral glucose utilization during sleep-wake cycle in man determined by positron emission tomography and 18F 2-Fluoro-2-deoxy-D-glucose method. Brain Res 513: 136–143
27. Martin G (1978) Lundberg's B-waves as a feature of normal intracranial pressure. Surg Neurol 9: 347–348
28. Mautner-Huppert D, Haberl RL, Dirnagl U, Villringer A, Schmiedek P, Einhäupl K (1989) B-waves in healthy persons. Neurol Res 11: 194–196
29. Miles LE, Dement WC (1980) Sleep and aging. Sleep 3: 119–121
30. Moszyniski K, Darwaj B, Kozniewska H, Czochra T, Smyk K, Lewelt W (1980) The results of ICP and EEG monitoring of sleep in cases of patients with supratentorial brain tumors. In: Shulman K, Marmarou A, Miller JD, Becker DP, Hochwalt GH, Brock M (eds) Intracranial pressure IV. Springer, Berlin Heidelberg New York, pp 579–583
31. Munari C, Calbucci F (1976) Organisation du sommeil et variations de la pression intracranienne chez les malades ayant une hydrocéphalie a pression normale. Rev EEG Neurophysiol 6: 497–501
32. Newell DW, Aaslid R, Stooss R, Reulen HJ (1992) The relationship of blood flow velocity fluctuations to intracranial pressure B-waves. J Neurosurg 76: 415–421
33. Nilsson C, Stahlberg F, Thomsen C, Henriksen O, Herning M, Owman C (1992) Circadian variation in human cerebrospinal fluid production measured by magnetic resonance imaging. Am J Physiol 262 (Regulatory Integrative Comp Physiol 31): R20–R24
34. Nornes H, Rootwelt K, Sjaastad O (1973) Normal pressure hydrocephalus. Long-term intracranial pressure recording. Eur Neurol 9: 261–274
35. Ogashiwa M, Takeuchi K (1983) Intracranial pressure changes during sleep in man. No To Shinkei 35: 123–129

36. Pickard JD (1982) Adult communicating hydrocephalus. Br J Hosp Med 27: 35–44
37. Pierre-Kahn A, Gabersek V, Hirsch JF (1976) Intracranial pressure and rapid eye movement sleep in hydrocephalus. Childs Brain 2: 156–166
38. Raftopoulos C, Chaskis C, Delecluse F, Cantraine F, Bidaut L, Brotchi J (1992) Morphological quantitative analysis of intracranial pressure waves in normal pressure hydrocephalus. Neurol Res 14: 389–396
39. Raftopoulos C, Deleval J, Chaskis C, Leonard A, Cantraine F, Desmyttere F, Clarysse S, Brotchi J (1994) Cognitive recovery in idiopathic normal pressure hydrocephalus: a prospective study. Neurosurgery 35: 397–405
40. Rechtschaffen A, Kales A (1968) A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. Public Health Service, U.S. Government Printing Office, Washington
41. Sahuquillo J, Rubio E, Codina A, Molins A, Giutart JM, Poca MA, Chasampi A (1991) Reappraisal of the intracranial pressure and cerebrospinal fluid dynamics in patients with the so-called "normal pressure hydrocephalus" syndrome. Acta Neurochir (Wien) 112: 50–61
42. Sakai F, Meyer JS, Karacan I, Derman S, Yamamoto M (1980) Normal human sleep: regional cerebral hemodynamics. Ann Neurol 7: 471–478
43. Symon L, Dorsch NWC, Stephens RJ (1972) Pressure waves in so-called low pressure hydrocephalus. Lancet 2: 1291–1292
44. Symon L, Dorsch NWC (1975) Use of long-term intracranial pressure measurement to assess hydrocephalic patients prior to shunt surgery. J Neurosurg 42: 258–273
45. Symon L, Hinzpeter T (1977) The enigma of normal pressure hydrocephalus: tests to select patients for surgery and to predict shunt function. Clin Neurosurg 24: 285–315
46. Townsend RE, Prinz PN, Obrist WD (1973) Human cerebral blood flow during sleep and waking. J Appl Physiol 35: 620–625
47. Vanneste JAL (1994) Three decades of normal pressure hydrocephalus: are we wiser now? J Neurol Neurosurg Psychiatry 57: 1021–1025
48. Vanneste JAL, Augustijn P, Dirven C, Tan WF, Goedhart ZD (1992) Shunting normal-pressure hydrocephalus: do the benefits outweigh the risks? Neurology 42: 54–59
49. Yokota A, Matsuoka S, Ishikawa T, Kohshi K, Kajiwara H (1989) Overnight recordings of intracranial pressure and electroencephalography in neurosurgical patients. Part II: changes in intracranial pressure during sleep. Sangyo Ika Daigaku Zasshi 11: 383–391

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