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An Experimental Study on the Effect of Differential Ventriculomegaly on the Somatosensory Evoked Potential[1]

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= Abstract =Differential ventriculomegaly is encountered after head injury, craniectomy or in some congenital hydrocephalic patients. The evoked potential studies have been widely used for the functional evaluation of the neural tissue.

To evaluate the effect of ventriculomegaly on the somatosensory evoked potentials (SEP's) and the effect of increased intracranial pressure (ICP) on SEP in ventriculomegaly, the authors had recorded SEP in a differential ventriculomegaly model.

To make a differential ventriculomegaly model, twenty-eight cats had kaolin-injections with cisternal puncture and hemicraniectomy. With the SEP studies of the cats in pre-hydrocephalic phase, in post-hydrocephalic phase 4-8 weeks after kaolin-injection, and in increased ICP phase, the effects of differential ventriculomegaly on the electrophysiologic function of the brain were evaluated. The size of the ventricles were evaluated with brain CT scan in 7 cats and with autopsy specimen in all 28 cats.

Intracranial kaolin-injection and hemicraniectomy produced asymmetrical ventriculomegaly with the larger one on the hemicraniectomy side in 68%.

In pre-hydrocephalic and post-hydrocephalic phase studies with left-sided hemicraniectomy, there were no significant side differences in the latencies. With the increased ICP, there was a significant increase in the P1-N1 interpeak latency on the left side ($p<0.01$). Comparing pre- and post-hydrocephalic phases, the latencies of wave II increased significantly in both sides ($p<0.01$), but there was no significant difference between the increases of both sides. There was no significant influence of the differential ventriculomegaly on the central conduction time (II-P1) of the SEP's with or without increased ICP. The differential ventriculomegaly with increased ICP did prolong the early cortical transmission (P1-N1) of the SEP's on the left side ($p<0.01$), which suggested greater vulnerability of cerebral cortex compared with that of the right side.

Key words: ***Hydrocephalus, Differential ventriculomegaly, Somatosensory evoked potential, Increased intracranial pressure, Central conduction time, Vulnerability of cerebral cortex***

INTRODUCTION

Differential ventriculomegaly is encountered after head injury, craniectomy or in some congenital hydrocephalic cases. From an experimental aspect, differential ventriculomegaly provides a good model for studying pathogenesis of hydrocephalus in an identical animal. In their hydrodynamic studies of hydrocephalus, Hochwald *et al.* (1972a), McLone and Naidich (1985) used a differential ventriculomegaly model.

In the present study, the authors had recorded somatosensory evoked potentials (SEP's) for the

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Table 1. Schedule of experiment

Experimental condition	Procedure
Pre-hydrocephalus	1. kaolin injection with cisternal puncture
	2. SEP* study
	3. hemicraniectomy
Post-hydrocephalus	1. tracheostomy and arterial cannulation
	2. ventricular puncture
	3. SEP study
Increased ICP**	1. cord ligation
	2. saline infusion or saline column application if plateau wave (−)
	3. SEP study

Note: *; somatosensory evoked potential
**; intracranial pressure

functional evaluation of brain tissue in a differential ventriculomegaly model.

The effects of hydrocephalus or increased intracranial pressure (ICP) on SEP had been studied by many authors (Ehle and Sklar 1979; Nagao *et al.* 1979; York *et al.* 1981; McPherson *et al.* 1984; Sutton *et al.* 1986), but to our knowledge, there has been no such study in a differential ventriculomegaly with the same conditions in an identical animal.

The purposes of the present study are (1) to evaluate the effect of differential ventriculomegaly on SEP by comparing pre-hydrocephalic and post-hydrocephalic phases, and (2) to evaluate the effect of increased ICP on SEP by comparing post-hydrocephalic and increased ICP phases in cats with differential ventriculomegaly.

MATERIALS AND METHODS

Among 82 mongrel cats weighing 2.4-3.5 kg, 28 cats survived the full schedule of experiment. The experiment had three phases, (1) pre-hydrocephalic, (2) post-hydrocephalic, and (3) increased ICP phases (Table 1).

Preparation of Animals

Pre-hydrocephalic phase : Cats were anesthetized with 25-30 mg/kg i.m. of ketamine and 0.025 mg/kg i.m. of atropine. One cc of cerebrospinal fluid (CSF) was drained through cisternal puncture, and the same volume of kaolin solution (aluminum silicate, 250 mg/cc) was injected. After scalp incision, small burr holes were placed on the bilateral coronal sutures, 12 mm apart from midline, for the insertion of recording electrodes, and another burr hole was made on the midline frontal sinus, 15 mm anterior to the bregma, for the insertion of reference electrode (Fig. 1). SEP was recorded and cranial

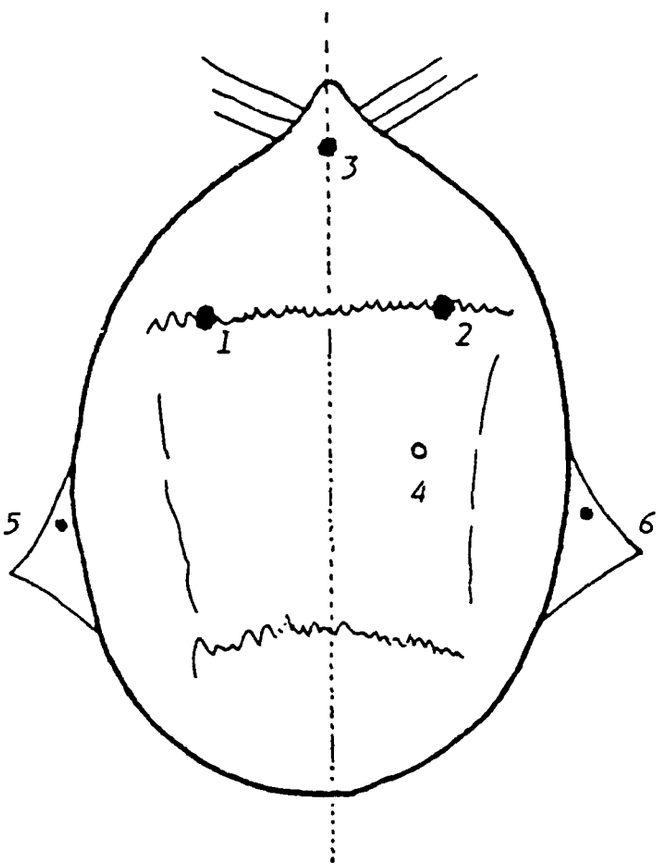

Fig. 1. Sites of electrodes for SEP studies. Recording electrodes (1&2) are placed 12 mm apart from the midline and symmetrically on the coronal suture. Point (3) is over the frontal sinus and 15 mm in front of bregma on the sagittal suture. The hole for ventricular puncture (4) is 8 mm off the midline and 10 mm behind the coronal suture. Ground electrodes (5&6) are over the ears.

bone of the left side was removed widely.

Post-hydrocephalic phase : 4-8 weeks after kaolin injection, cats were anesthetized as in the pre-hydrocephalic phase. After tracheostomy, an endotracheal tube was inserted and connected to the respirator (Baby Bird, Palm Springs Co., FiO_2 21%, 2 l/min, 22-24/min). Solution containing 50 mg% of ketamine and 4 mg% of pancuronium was infused continuously via femoral vein. A catheter was inserted into the femoral artery for monitoring of arterial blood pressure (ABP) and arterial blood gas (ABG). After scalp incision, another small burr hole was trephined on the right parietal bone, 10 mm posterior to the bregma and 8 mm apart from the midline, for intraventricular needle placement. SEP was taken.

Increased ICP phase : ICP was raised to 40-100 torr with the ligation of lumbar spinal cord, intraventricular saline injection or application of fluid column to the needle placed in the right lateral ventricle. Cerebral perfusion pressure was maintained above 40 torr which is adequate for the preservation of cerebral autoregulation. SEP was taken.

Monitoring

ABP, ABG, and body temperature were monitored to be within physiological range.

SEP

SEP was taken at burr holes on the bilateral coronal sutures with the electrodes contacting the dura. Reference electrodes were inserted into the frontal sinus and ground electrodes to both ear lobes. The SEP corresponds to C4'-Fz or C3'-Fz of the human electode montage. the needle electrodes for stimulation were inserted to the distal part of the forepaw of both sides. Bilateral median nerves were stimulated (rectangle-shaped direct current, 0.1 msec duration, 5/sec, total 256-512 times, the stimulator was madé by the Department of Medical Engineering, Seoul National University Hospital). The signals were processed according to the method described by Sutton *et al.* (1984) with an A-D converter (1 μsec conversion time, 12 bit resolution) and an amplifier (Electronics for Medicine, Inc., V 1205 A, filter 1/500 Hz, 0.02 mV/cm). The duration of signal sampliing after each stimuli was 30 msec. The waves were named as Fig. 2 (Sutton *et al.* 1982). Small upward re-

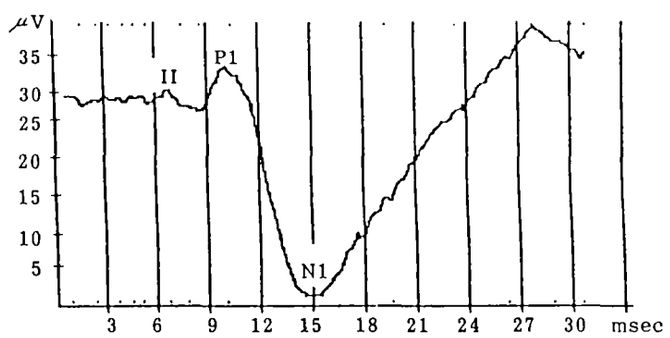

Fig. 2. A representative SEP curve and definition of the wave and interpeak latencies.

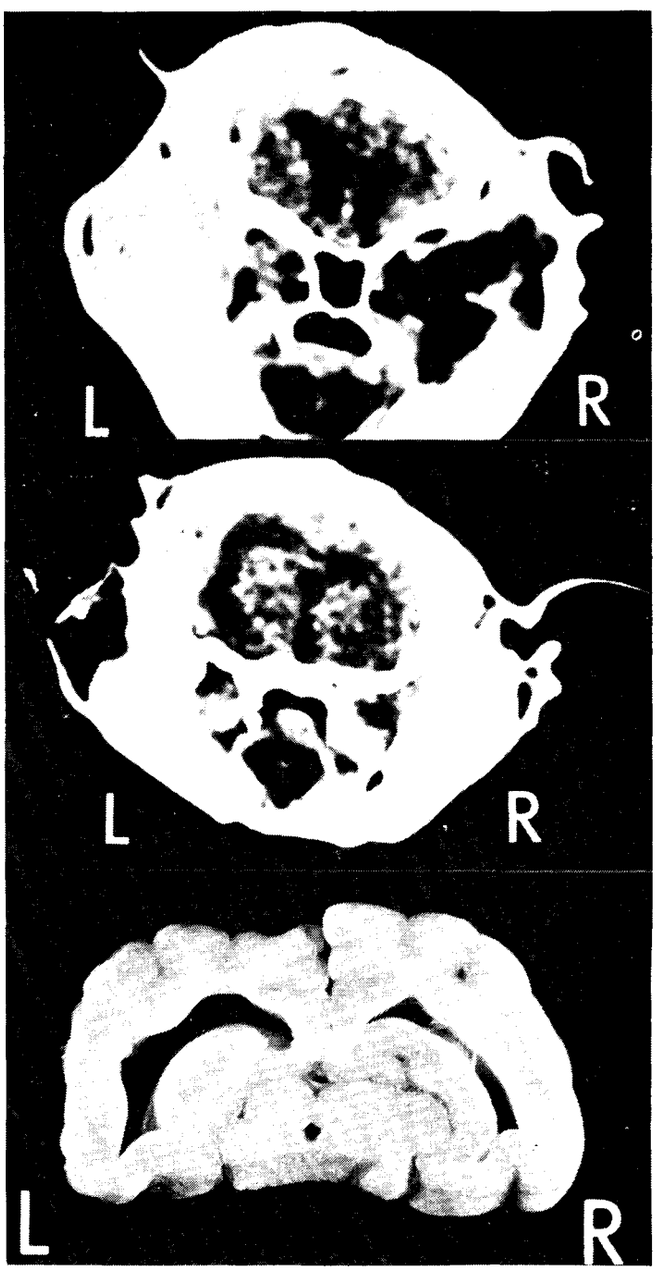

Fig. 3. Photographs of representative brain CT scans and cut surface of the brain autopsy specimen R; right, L; left.

Table 2. Absolute wave or interpeak latencies of SEP studies in three experimental phases

Wave	Side*	Latency (msec, mean±SD**, n***=28)		
		Pre-hydrocedphalic	Post-hydrocephalic	Increased ICP+
II	R	7.21±0.10	7.85±0.13	7.96±0.19
	L	7.37±0.11	7.94±0.14	8.29±0.19
P1	R	10.65±0.17	11.25±0.22	11.90±0.31
	L	10.53±0.16	11.67±0.24	12.30±0.28
N1	R	15.45±0.26	16.25±0.29	17.44±0.45
	L	15.09±0.27	16.77±0.35	18.80±0.55
II-P1	R	3.44±0.15	3.43±0.17	3.94±0.28
	L	3.16±0.12	3.73±0.24	3.99±0.33
P1-N1	R	4.86±0.16	5.05±0.18	5.55±0.29
	L	4.56±0.16	5.10±0.18	6.50±0.33
II-N1	R	8.25±0.22	8.39±0.28	9.51±0.43
	L	7.72±0.23	8.83±0.36	10.49±0.59

Note: *; R=right, L=left, **; SD=standard deviation, ***; number of materials, +ICP=intracranial pressure

flections before the large upward wave P1, were named wave I-IV. Of the 4 small waves, only the wave II could be reproduced constantly. The major downward reflection was named wave N1. With the help of the software of the Apple II microcomputer, made by the Department of Medical Engineering, Seoul National University Hospital, the absolute latencies of wave II, P1, N1 were measured and II-P1, P1-N1 interpeak latencies were calculated.

Observation of differential ventriculomegaly

CT scan (GE 9800, GE) was taken in 7 cats and autopsy was performed in all 28 cats.

Statistical analysis

The basic components of SEP, i.e., absolute latency of wave II, interpeak latencies of II-P1 and P1-N1, were tested for side to side differences in each of three experimental phases. Each of those three latencies was tested for difference between pre- and post-hydrocephalic phases, and between post-hydrocephalic and increased ICP phases. The test was performed with 2-tailed probability of paired t-test.

RESULTS

Observation of differential ventriculomegaly

In all the CT scans taken later than 5 days after kaolin injection, there was evident differential venticulomegaly with the left lateral ventricle being larger. On autopsy examination, 19 brains (68%) showed evident differential ventriculomegaly (Fig. 3).

SEP of pre-hydrocephalic phase

There were no statistically significant side to side differences in the wave latencies (Table 2).

SEP of post-hydrocephalic phase

Opening ICP was 3-20 torr (mean; 8 torr). there were no significant side to side differences in the wave latencies (Table 2).

SEP of increased ICP phase

Only in one cat (4%), did ICP rise up to 40 torr and appeared plateau wave with the lumbar cord ligation. Fluid column was applied in the other 27 cats. Induced ICP was 40-100 torr (mean; 55 torr) and cerebral perfusion pressure was 40-120 torr (mean; 62 torr). There was a significant increase in the P1-N1 interpeak latency on the left side ($p<0.01$, Table 2).

Absolute latency of wave II

Comparing pre- and post-hydrocephalic phases, the absolute latencies of wave II increased significantly on both sides ($p<0.01$), but there was no significant difference between the increases of both sides. There was no significant difference of the absolute latency of wave II between post-hydrocephalic and increased ICP phases (Fig. 3).

II-P1 interpeak latency

There were no significant influences of the differential ventriculomegaly on the central conduc-

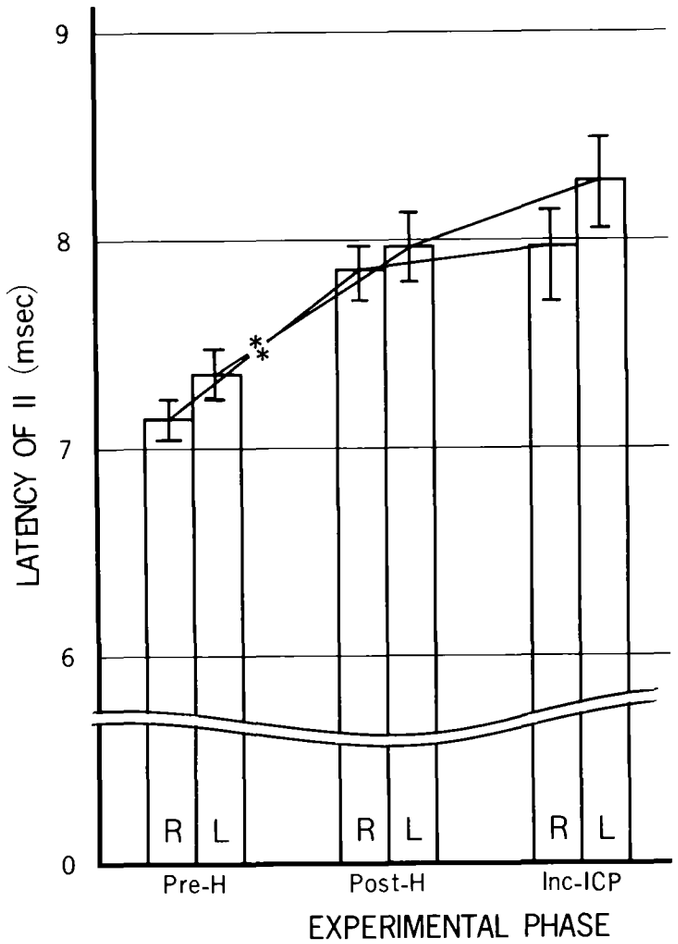

Fig. 4. Absolute latencies of wave II in the three experimental conditions.
*There are significant differences between the absolute latencies of wave II in pre-hydrocephalus and post-hydrocephalus phases at both sides ($p<0.01$ by the paired t-test). Pre-H; Pre-hydrocephalus, Post-H; post-hydrocephalus, Inc-ICP: increased intracranial pressure, R; right, L; left.

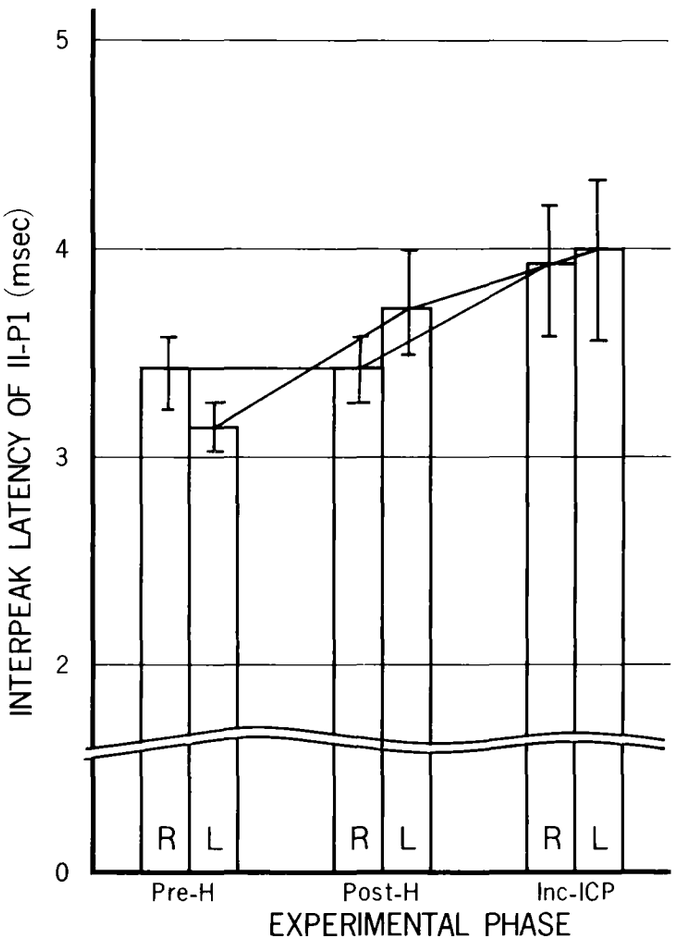

Fig. 5. II-P1 interpeak latencies (central conduction time) in the three experimental conditions.

tion time (II-P1) with or without increased ICP (Fig. 4).

P1-N1 interpeak latency

There were no significant differences between pre- and post-hydrocephalic phases. However, comparing post-hydrocephalic and increased ICP phases, the P1-N1 interpeak latency revealed significant increase on the left side ($p<0.01$, Fig. 5).

DISCUSSION

Differential ventriculomegaly model

A differential ventriculomegaly model is very useful in the research of hydrocephalus not only because neural morphology and function of both sides can be compared in an identical animal with an identical condition, but because there are many patients whose ventricles demonstrate asymmetrical dilatation, such as hydrocephalus with skull defect or encephalomalacia, asymmetrical craniosynostosis, and obstructed foramen of Monro.

In the present study, 68% of autopsied brain showed evident differential ventriculomegaly. However, the higher detection rate in CT scans suggested it is possible that the CSF leakage during autopsy procedure made the difference of ventricular size equivocal.

Hochwald *et al.* (1976) induced increased ICP with the ligation of lumbar spinal cord and Sutton *et al.* (1986) could have plateau waves in 100% of cases. But in the present study, only one (4%) showed a plateau wave with the ligation of the lumbar spinal cord. Hayashi *et al.* (1982) and Hayashi *et al.* (1984) explained the formation of plateau wave in two aspects: (1) vasomotor activity as a part of autoregulation, and (2) intracranial rigid condition brought by decreased amount of CSF and disturbed absorption

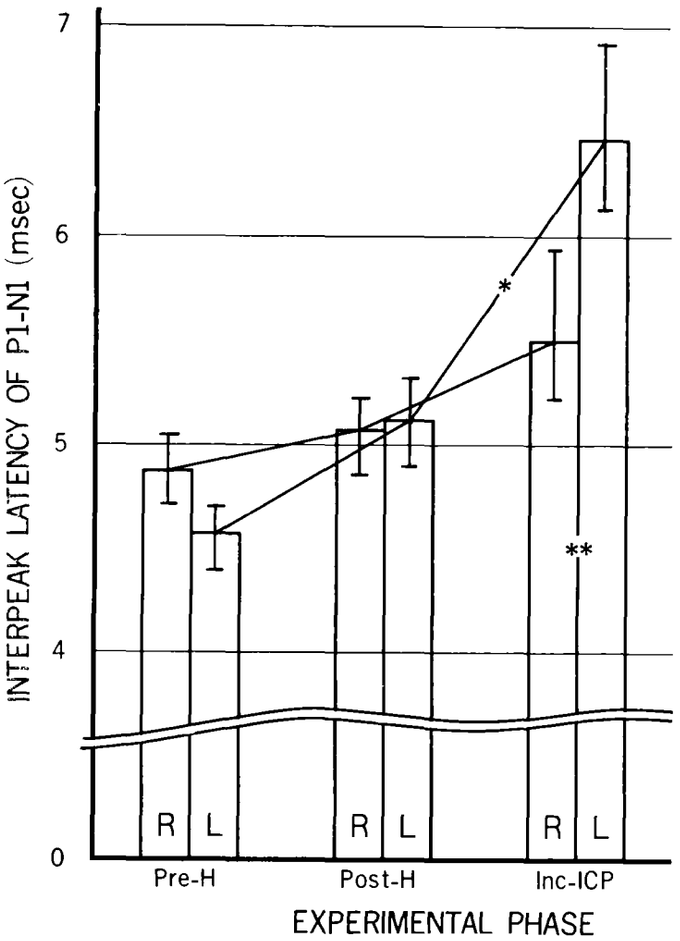

Fig. 6. P1-N1 interpeak latencies in the three experimental conditions.
*There is a significant difference between the P1-N1 interpeak latencies in post-hydrocephalus and in increased ICP phases.
**There is a significant difference between the increases of P1-N1 interpeak latencies of both sides in increased ICP phase.

of CSF. The wide removal of cranial bone in our study might have made the intracranial condition, "less rigid". Another explanation of ours is CSF drainage via "alternative pathways.". In some cats in the present study, Evans Blue was injected into the ventricle before the sacrifice of animals. Autopsy revealed dense discoloration of paranasal sinuses, lymphatics and lymph nodes of the neck. However, the role of such drainage should be evaluated quantitatively taking normal amount of drainage into consideration (Bradbury and Cole 1980; Bradbury *et al.* 1981).

Name of SEP waves and their origins

Many authors presented different opinions on the naming of the feline SEP waves and their origins. In the present study, we accepted those of Sutton *et al.* (1982) which were very close to the pattern of curves in our study. the wave II is considered to originate from medial lemniscus or dorsal column nuclei, and the wave P1 to be the first cortical wave. Wave N1 seems to originate from somatosensory association area. Central conduction time was defined as II-P1 interpeak latency.

Effects of differential ventriculomegaly on SEP

Bilateral increase of absolute latency of wave II in post-hydrocephalic phase : The increases were of the same degree on both sides and not influenced by increased ICP. The origin of wave II, medial lemniscus or dorsal column nuclei, suggested a pathology below the medulla oblongata with bilateral diffuse involvement, such as impaired blood flow, mechanical compression, and inflammation. Hochwald *et al.* (1972b) had reported quadriparesis in kaolin-injected cats and explained it with kaolin-induced meningitis and vasculitis, which supports our assumption. Further study will be needed to clarify the exact mechanism of wave II prolongation.

Left-sided increase of P1-N1 interpeak latency in increased ICP phase: The increase of P1-N1 interpeak latency on the side of the larger ventricle suggested vulnerability of cerebral cortex to increased ICP. Greitz (1969), Symon *et al.* (1974), Sohmer *et al.* (1983), Hayashi *et al.* (1984), and Hayashi *et al.* (1986) demonstrated decreased global or periventricular blood flow in hydrocephalus or increased ICP, and Lesnick *et al.* (1984), Izumi *et al.* (1986), and Lewelt *et al.* (1986) related decreased cerebral blood flow (CBF) to the decrease of amplitudes or increased latencies in evoked potential studies. Cohen *et al.* (1967), and Robinson (1967) showed disturbed cerebral metabolism in increased ICP and McPherson *et al.* (1982), and Koeher *et al.* (1986) demonstrated a causal relationship between the changes of SEP and metabolic derangement in increased ICP or hypoxia. However, Ladds *et al.* (1986) reported higher threshold of CBF for SEP changes in increased ICP than that in vascular occlusion and suggested the presence of other factors except CBF in changes of SEP caused by increased ICP.

Yakovlev (1947) speculated that stretching of periventricular neural fibers brings about the neurological deficits in hydrocephalus and Engel (1975) and McPherson *et al.* (1984) related this

stretching to the changes of SEP. Trojaborg and Petersen (1979) explained the changes of the SEP in hydrocephalus by periventricular edema, gliosis, and demyelination.

In the present study, the fact that the increase of P1-N1 interpeak latency on the left side in increased ICP phase was normalized after reduction of ICP, suggested that the changes of SEP were due to functional changes such as CBF or metabolism rather than to morphological changes.

In the study of the SEP changes in reduced CBF, Lesnick *et al.* (1984) stressed the vulnerability of the cerebral cortex and white matter to ischemia compared with deep gray matter. Ladds *et al.* (1986), and Takeuchi *et al.* (1986) reported similar results in the study of evoked potential changes in increased ICP. Our results, which showed no significant change in wave latencies except that of cortical origin, corresponded well with those of these authors.

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= 국문초록 =

차등적 뇌실 확장이 체성감각 유발전위에 미치는 영향에 관한 실험적 연구

서울대학교 의과대학 신경외과학교실

조병규 · 왕규창 · 이용진 · 김현집 · 최길수

차등적 뇌실확장은 두부외상후, 두개골 절제술후 또는 선천성 수두증예의 일부 등에서 관찰된다. 저자들은 유발전위 측정이 신경조직의 기능적 평가에 유용함을 이용하여 차등적 뇌실확장 모델에서 체성감각유발전위(이하 SEP라함)를 측정함으로써 뇌실확장이 SEP에 미치는 영향과 뇌실확장시 뇌압상승이 SEP에 미치는 영향을 고찰하였다.

성숙한 잡종 고양이 28마리를 대상으로 대조(cisterna magna)에 kaolin용액을 주입한 직후 "수두증전 SEP"를 측정하고 좌측 두개골을 제거하여 차등적 뇌실확장을 만든 실험모델에서, kaolin용액 주입후 4-8주가 경과한 뒤 "수두증후 SEP"를 측정하고 경막외 척수 결찰, 생리적 식염수의 뇌실내 주입 또는 생리적 식염수의 용액주(fluid column)를 이용하여 뇌압 상승을 유발한 후 "뇌압상승후 SEP"를 측정하였다.

그리고 이들의 뇌전산화단층촬영소견과 부검소견을 통하여 실험모델의 뇌실확장 정도를 관찰하였다.

대조에 kaolin용액을 주입하고 한쪽 두개골을 제거하여, 28마리 중 19마리(68%)에서 두개골 제거측 뇌실이 더 큰 차등적 뇌실확장을 관찰할 수 있었다.

수두증전 SEP 검사 및 좌측 두개골 제거후의 수두증후 SEP 검사상, II, II-P1, P1-N1파의 잠복 시간은 좌우의 유의한 차이가 없었다.

뇌압상승후 SEP 검사상 좌측에서 P1-N1파간 잠복시간이 유의하게 증가되었다 ($p<0.01$).

수두증 전후의 SEP를 비교하면 양측 II파의 잠복시간이 모두 유의하게 증가되었으나 ($p<0.01$) 증가된 정도는 좌우에서 유의한 차이가 없었으며, 뇌압상승 전후에는 유의한 변화를 보이지 않았다.

중추전달시간(II-P1파간 잠복시간)은 수두증 전후와 뇌압상승 전후 모두 유의한 변화를 보이지 않았다.

P1-N1파간 잠복시간은 수두증 전후 유의한 변화를 보이지 않았으나 뇌압상승후 좌측에서 유의한 증가를 보여($p<0.01$) 더 확장된 뇌실측의 대뇌피질이 반대측보다 뇌압상승에 의하여 먼저 기능장애를 보임을 알 수 있었다.