Diagnostic Value of Stationary Potential Measurement in Head Injuries

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Summary

Traumatic brain injuries were analysed clinically and experimentally by recording stationary potential (SP) which is a very stable component in the electrical phenomena of the brain. SP value is considered to change positively or negatively only when there are organic lesions, severe metabolic changes of the brain, or epileptic seizures.

Clinically, we have measured the SP of 103 head-injured patients. When an organic lesion was located in the cerebral hemisphere, the SP showed various changes according to the location of the lesion.

Positive SP changes were usually seen in the cases of mild cortical compression and subcortical lesions. Clinically, these lesions included epidural hematoma, subdural hematoma without contusion, chronic subdural hematoma, simple depressed fracture and intracerebral hematoma without cortical damage.

Whereas, almost all of the cortical lesions or extensive brain lesions showed negative changes of SP. They were such as cerebral contusion, intracerebral hematoma with vascular lesion, depressed fracture with cortical damage and traumatic epilepsy.

Subsequently, to support these clinical data, experimental cortical compression with clot inserted into subdural and extradural space, subcortical lesion by artificial intracerebral hematoma, cerebral contusion by mechanical destruction were investigated in cats. The experimental results showed the same tendency as the clinical data.

In another experimental study, clarification of the possibility of SP change in concussion was attempted.

Key words: Head Injury, Stationary Potential, Intracranial Hematoma, Contusion, Concussion.

Introduction

The electrical activities of the brain recorded with macroelectrodes can be classified into three main categories as illustrated in Fig. 1: (1) what is called EEG, or the potential fluctuations with a frequency range of 0.5 to 70 Hz or more and with the amplitude of the order of microvolts; (2) the slowly changing potential (SCP) or the sustained potential which is of the order of microvolts (ranging from 50 µV to several millivolts) and is slower than 0.5 Hz; and (3) the stationary potential (SP) which is of the order of millivolts and is very stable as the name implies. The SP may be compared to the depth of water, the SCP to large waves, and EEG to ripples on the surface.

We have hitherto measured SP-changes of many organic brain damages clinically and experimentally and have discussed the origin of SP or clinical application of SP recording. As we have already reported, in the cases of brain tumor and cerebral vascular disease cortical compression without its damage indicated positive shift of SP, cortical compression with its damage and/or cortical lesion itself made negative changes, subcortical and/or deep seated space occupying lesions showed positive changes, and extensive brain lesion exhibited...
negative change$^{9,11-13,23}$.

The present communication deals with the SP change in head-injured patients and experimental animals. We would like to show the same clinical validity of the SP in case of head injury as in other organic lesions.

**Method**

Stationary potential requires a special recording system which consists of calomel half-cell electrodes, salt bridges and a high impedance (30,000 Megohm) DC amplifier$^9$. In the experimental study, a 2-channel pen recorder was used (Fig. 2). In clinical application in acute cases, about 30 scalp points were selected and measured manually and each SP value was read from the scale of the high impedance DC amplifier. SP-Encephalograph adopted in chronic cases was the equipment that recorded the SP change on the scalp automatically. The potential on the scalp was recorded by the 90 units of agar bridges and calomel half-cells, which were led to the switches exchanged by the scanner in order. The scanner controled the recording mechanism. The amplified potentials were classified into six groups by the comparator and were displayed as colored dots on a circular chart by dotting mechanism. Brain lesions were diagnosed by this chart as abnormalities of the SP pattern. (Fig. 3)

Fig. 2. Block diagram of recording system in experimental study and acute stage of clinical case.

Fig. 3. SP-Encephalograph and normal isopotential chart.

**Materials and Results of Clinical Cases**

One hundred and three cases were measured and investigated, including 21 acute intracranial hematomas, 25 chronic subdural hematomas, 20 cerebral contusions, 20 depressed fractures and 17 traumatic epilepsies. Eight cases of non-traumatic hematomas were also included as a control. The results of SP change were summarized according to the sub-type of lesions in Table 1. The details were as follows. (Table 1)

1) **Intracranial Hematomas (46 cases) (Table 2)**

a) **Acute Subdural Hematoma (SDH)**

All the cases were adult and were accompanied with cerebral contusion. In the two cases
that showed negative SP change, severe cortical contusions with subdural hematoma were disclosed at the operations. In another case with positive SP change, subacute subdural hygroma with little cerebral contusion was found. (Fig. 5-a)

In the other two cases of non-traumatic subdural hematoma, the SP change was negative and other organic lesions on the cortex were found. One was the bleeding from diffuse meningeal carcinomatosis, and the other was subdural empyema. (Fig. 4)

b) Acute Epidural Hematoma (EDH) (Fig. 5-b)

Epidural hematoma always showed positive SP change. The type of these lesions was usually the cortical compression without its damage. Once the compression was released by surgical removal of the hematoma, recovery came about immediately.

These facts suggested that positive change of SP indicated good prognosis of the disease such as chronic subdural hematoma, simple depressed fracture and so on.

c) Intra-cerebral Hematoma (ICH) (Fig. 5-d)

Traumatic intracerebral hematoma consisted of only one case as in the table. The SP change was positive. There was no cerebral contusion at all. Non-traumatic intracerebral hematomas originated from the diffuse cerebral vascular disease such as hypertensive vascular bleeding or arterio-venous malformation showed usually negative SP change.

d) Chronic Subdural Hematoma (CSH) (Fig. 5-c)

The SP change of chronic subdural hematoma was usually positive as the result of cortical compression without its damage (70%), but negative SP changes were also seen in 30% of the cases.

This dissociation of SP is of interest and requires more investigation into the size and the content of hematoma and the patient's clinical symptoms. Many reports of EEG of chronic subdural hematoma indicated similar discrepancies. Some cases show local high voltage foci in the side of the lesion and others show low voltage in the routine EEG records$^{4,18,25}$.

Table 1. SP changes of 103 head injured patients.

<table>
<thead>
<tr>
<th>Type</th>
<th>Low</th>
<th>High</th>
<th>Irreg</th>
<th>No change</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>SDH traumatic</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>SDH non-traumatic</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>EDH traumatic</td>
<td>1</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>EDH non-traumatic</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>ICH traumatic</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>ICH non-traumatic</td>
<td>7</td>
<td>7</td>
<td>1</td>
<td>0</td>
<td>25</td>
</tr>
<tr>
<td>CSH</td>
<td>16</td>
<td>26</td>
<td>2</td>
<td>2</td>
<td>46</td>
</tr>
</tbody>
</table>

*AVM = 2, Hypertensive ICH = 2

SDH: subdural hematoma,
EDH: epidural hematoma,
ICH: intracerebral hematoma,
CSH: chronic subdural hematoma

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<table>
<thead>
<tr>
<th>Type</th>
<th>Low</th>
<th>High</th>
<th>Irreg</th>
<th>No change</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subdural</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>3</td>
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<tr>
<td>Hematoma</td>
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<td>2</td>
<td>3</td>
<td>11</td>
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<td>Intracereb</td>
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<td>0</td>
<td>1</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Contusion acute</td>
<td>7</td>
<td>12</td>
<td>2</td>
<td>3</td>
<td>20</td>
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<tr>
<td>Chronic</td>
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<td>13</td>
<td>2</td>
<td>3</td>
<td>20</td>
</tr>
<tr>
<td>C S H</td>
<td>7</td>
<td>17</td>
<td>2</td>
<td>3</td>
<td>31</td>
</tr>
<tr>
<td>Depressed Fr</td>
<td>8</td>
<td>10</td>
<td>1</td>
<td>1</td>
<td>20</td>
</tr>
<tr>
<td>Traumatic Ed</td>
<td>10</td>
<td>3</td>
<td>4</td>
<td>1</td>
<td>18</td>
</tr>
<tr>
<td>Hematoma non-traumatic</td>
<td>4</td>
<td>12</td>
<td>2</td>
<td>1</td>
<td>18</td>
</tr>
</tbody>
</table>

"Low" means low voltage in the lesion, "High" means high voltage, and "Irregular" means the irregular change in SP value respectively.

Table 2. SP change in intracranial hematoma.

<table>
<thead>
<tr>
<th>Type</th>
<th>Low</th>
<th>High</th>
<th>Irreg</th>
<th>P</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
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<td>0</td>
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<tr>
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<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>EDH traumatic</td>
<td>1</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>EDH non-traumatic</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>ICH traumatic</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>ICH non-traumatic</td>
<td>7</td>
<td>17</td>
<td>1</td>
<td>0</td>
<td>25</td>
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This dissociation of SP is of interest and requires more investigation into the size and the content of hematoma and the patient’s clinical symptoms. Many reports of EEG of chronic subdural hematoma indicated similar discrepancies. Some cases show local high voltage foci in the side of the lesion and others show low voltage in the routine EEG records$^{4,18,25}$.
2) Cerebral Contusion (C.C.) (20 cases) (Table 3)

Almost all of the SP of cerebral contusions showed negative change. The negative SP change of acute subdural hematoma, which might be theoretically expected to change to positive because of its cortical compression, was fully explained from the fact that traumatic acute subdural hematoma was almost always accompanied with cerebral contusion.

3) The Others

a) Depressed Fracture (DF)

Among twenty cases of depressed fracture SP changes of 10 cases were positive and eight were negative. Cases with positive SP changes had not cortical damage, while cases with negative SP changes had cortical damage. (Fig. 6-b)

b) Traumatic Epilepsy

Seventeen cases were measured. The SP change on the epileptic focus showed tendency to be negative both on the scalp and on the cortical estimation.

Summary of clinical studies. (Fig. 7)

1) The SP of mild cortical compressions showed positive change as we have mentioned before. Clinically, these lesions were epidural hematoma, subdural hematoma without cortical damage, chronic subdural hematoma and simple depressed fracture without cortical damage.

2) Subcortical damages also exhibited positive SP change. They were traumatic intracerebral hematomas without cortical damage (rare) or spontaneous intracerebral hematomas.
3) Negative SP changes occurred in extensive cortical and subcortical damage, or in cerebral contusion and cerebrovascular lesions\(^1,6,7\). The SP shifts toward the negative were also seen in traumatic epilepsy\(^8\) and depressed fracture with cortical damage.

**Materials and Results of Experimental Studies**

In order to confirm the results of clinical data in head-injured patients, experimental studies were carried out. SP was detected in four models simulating epidural hematoma, subdural hematoma, intracerebral hematoma and cerebral contusion. Then, the dynamic test was performed to monitor the SP changes in concussion.

1. **Static study**

Twenty adult cats were used. After the animals were anesthetized with Nembutal, they were immobilized with flaxedil and were mounted on the operating apparatus. Respiration was maintained artificially through the intratracheal canule. The SP monitoring system was as mentioned above (Fig. 2) The indifferent electrode was usually placed on the animal's tongue\(^9\) and the different electrode on the skull or on the dural membrane. EEG and ECG were monitored simultaneously\(^5\).

The results are summarized in Fig. 8 and 9.

The data of one experimental EDH are documented as follows: a burr hole was opened in the left parietal region and 1 ml of autogenous venous blood was injected slowly into the epidural space. The SP changed abruptly to the negative at first, returned to the beginning level of normal.
after five minutes, and shifted gradually to the positive. Finally, the SP reached the level of positive 2.5 mV seven minutes later and was stationary thereafter. No SP change was obtained on the control side. EEG change recovered at least after 5 minutes (Fig. 8-a). The SP were observed at least for 120 minutes in all cases. On the average, SP change was positive 3.6 mV in the experimental EDH, positive 1.8 mV in SDH, and positive 2.0 mV in ICH respectively (Fig. 9). These data suggested that positive SP changes were less in degree when lesions affected the cortex. In cerebral contusions, the SP shifted 4 mV to the negative on the average.

These data corresponded closely with the clinical records

2. Dynamic Study

Twenty-three adult dogs were used. After the animal was lightly anesthetized with nembutal, a 10 kg iron ball was dropped on the semi-free movable head which was covered with a direct-bioresin plate so that the ball would hit the animal precisely on the occipital region. Shock absorbing materials were patched on the plate to control the impact duration. Kinetic energy and linear acceleration with its duration were recorded and the SP change, EEG and ECG were monitored. Concussion phenomena were observed and estimated at the moment of the impact. The cerebral contusion was ascertained in the autopsy.

The results are shown in Fig. 10(a). There was a correlation between the SP change and the kinetic energy which was estimated from the height and the weight of the iron ball. The larger the energy was, the more negative change of SP was obtained and the more frequently cerebral contusion was seen in the autopsy.

Definite correlations could not be observed between the linear acceleration and the change of SP, probably due to some artificial factors.

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![Fig. 9. Mean SP value in experimental brain injury.](image)

![Fig. 10. a) SP change and kinetic energy, b) SP change in concussion and contusion.](image)
such as excess movements of the impacted animal's head in recording linear acceleration.

In (Fig. 10-b) the SP changes were compared between the concussed and non-concussed groups, and between the contused and non-contused groups, respectively. More negative changes of SP was observed in the contused group than that of the non-contused group, and in the concussed group than that of the non-concussed group.

In this blow method, the jolt of the animal's head at the moment of impact was so large that the SP system could not be stabilized to the head and accordingly the SP could not be recorded during the impact. Thus any definite comment on the SP change during and immediately after the impact cannot be made, but it is conceivable that the negative SP change occurred in concussive condition.

Discussion

1. Injuries and SP changes

Through clinical applications and animal experiments in head injury the hypothesis of SP change in brain damage was confirmed. The SP of the brain shifted to the positive both in mild cortical compression and subcortical lesion, and changed toward the negative in cortical lesion or extensive injury. There was no contradiction as to the type of etiology. Bearing these inclinations in mind, we can obtain valuable information by evaluating SP changes in head injury. For example, in the case shown in Fig. 4, the SP change shifted toward the negative though no cerebral contusion was expected from the clinical course. Cerebral angiography revealed that a thin avascular area in the right frontotemporoparietal region and mild cortical compression might change SP toward the positive. But this dissociation of SP change and cerebral angiography suggested the existence of some factors other than the cortical compression alone. In fact, diffuse meningeal carciomatosis was found in the operation and the autopsy.

In the SP of head injury, the cerebral contusion often plays an important part as one of "the other factors". A simple depressed fracture usually shows a positive SP change as the result of mild cortical compression, but when there is some dural and cerebral injury, or infection in case of an open depressed fracture, the SP may shift toward the negative. A depressed fracture with negative SP change is more likely to be a focus and the chance of epilepsy increases. The negative change of SP is valuable not only to estimate the long term prognosis but also to presume the mortality and morbidity in acute or subacute stages of head injury. The more extensive the negative SP, the more severe irreversible cortical damage is assumed.

Undoubtedly the diagnosis of intracranial hematoma is possible by detecting the asymmetry of SP values. In an acute stage of head injury, especially in front emergency hospitals, it is common that no clinical signs nor findings on cerebral angiography for intracranial hematomas are present soon after the injury (lucid intervals). If continuous SP monitoring is applied to these patients during the observation period, the side, the extension and the nature of a gradually growing hematoma or the cerebral edema can be found even before symptoms and signs of compression or other hazards develop. A warning apparatus by SP recording should be set up in the near future.

2. SP change in concussion.

There have been a few reports concerning the SP change in concussion. Among them important are the studies of Walker et al (1944) and of Meyer and Denny-Brown et al (1955). Meyer et al recorded the SP change of animal brain by producing concussion on cats and monkeys with the methods of 1) acceleration concussion 2) percussion concussion 3) compression concussion. According to their study, usually the SP of the moderately impacted part changed to the negative (1–7 mV) transiently, but when the impact was strong enough to produce severe concussion, the duration of negative SP change is not transient but continuous for several minutes or more.

In the present study the SP was not recorded immediately after the impact, but the impact was strong enough to produce severe and continuous concussion, and the SP value of concussion was reliable because it was detected during the course of concussion. Furthermore, it is greatly interesting that Meyer et al considered concussion to be produced even by the local cortical damage. Namely, concussion can
be elicidated not only at the dysfunction of brain stem or reticular activating system but also at the focal cortical damage independently (local percussion paralysis). Walker et al mentioned in their important paper, that the physiological basis of concussion consists of the traumatic discharge of the polarized cell membranes of the neurons of the central nervous system by shaking up or commotion of the brain and subsequent course of events is that which would be expected when large masses of nerve cells discharge, such as is seen in the spontaneous or electrically induced convulsive seizure. A local concussion, according to their paper, may on occasion a brief dazed feeling compatible to a petit mal attack.\(^{27}\)

As to the origin of the SP, we have stressed the nerve cells\(^{9,11,15,17,20-24}\). Their consideration of the origin of SP in their experimental studies are substantially the same as ours. Clinically we did not have sufficient time to measure the SP of concussed patient due to the fact that the clinical course of concussion actually so brief. It may, however, be possible to suppose that local negative SP change could occur in some concussed patients. In such a case, we can reasonably explain that SP changes not only in long-standing organic brain damages but also in these momental phenomena-concussion from the SP neuron hypothesis.

Ommaya et al, as Tschirgi suggested, considered that the change of SP developed from the destruction of blood brain barrier\(^{9,26}\).

There were studies of SP changes in various brain injuries such as in experimental cerebrovascular lesion\(^{4,6,7}\) or brain death\(^{10}\), but no study of SP on head injuries was found in any references except in experimental concussions as mentioned above. This is, so far, a rare report in which the SP is applied to head-injured patients clinically. The SP detection is simple and is an easy method to verify the nature and the extent of the brain damage in a short time without harmful stimuli to patients. This system could be utilized more in clinical practices a useful diagnostic aid.

Conclusion

The SP change of 103 clinical head-injured patients and of 21 cats and 23 dogs experimented were analysed. Results were as follows.

1) Positive SP changes were usually seen in the cases of mild cortical compression and subcortical lesions. Clinically, these lesions were equivalent to acute epidural hematoma, acute subdural hematoma without contusion, chronic subdural hematoma, simple depressed fracture and intracerebral hematoma without cortical damage respectively.

Almost all of the cortical lesion or extensive brain lesions showed negative changes of SP. Such cases were cerebral contusion, intracerebral hematoma with vascular lesion, depressed fracture with cortical damage and traumatic epilepsy. These inclinations of SP changes was confirmed by experimental study.

2) The group with positive SP change showed a tendency to have good prognosis in mortality and morbidity.

3) In experimental study, the more severe the brain injury, the more to the negative the SP value shifted. Furthermore, the SP changes in cerebral concussion was observed to shift toward the negative.

4) The authors are of opinion that SP recording, especially in acute stage of head injury, is one of the most useful diagnostic methods because SP change is very simple to measure without any complication and from the distribution of SP change, the nature and extension of the lesion can easily and precisely be predicted.

Acknowledgment

The authors wish to thank Dr. Yahagi, Y., the chief director, and the members of the department of neurosurgery, Tokyo Toshima Municipal Hospital for their assistance during the study.

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