AN ELECTROENCEPHALOGRAPHIC STUDY FOR CHRONIC NITROGLYCEROL POISONING

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To clarify chronic nitroglycerol intoxication upon the cerebral activity, electroencephalographic examinations of nine subjects working in a dynamite factory were conducted.

There were only slight and little positive findings in the workers on the subjective symptoms and objective examinations except for EEG. Seven subjects out of nine indicated abnormal EEG records both in cases experienced anginal attack and not yet experienced such sign. It can be seen from the EEG findings in workers exposed to nitroglycerol that fast activity in all areas was dominant particularly.

Based on these findings, it may be concluded that chronic nitroglycerol exposure seemed to influence upon the cerebral activity of factory workers.

Sudden unexpected death and anginal attack have been observed in cases of severe nitroglycerol poisoning, since the first report of Symanski. The mechanism of this attack is commonly regarded to be due to the cardiac causes associated with disorders of autonomic nervous system. From the author's experience, disorders of pituitary-adrenocortical functions seem to be connected with the outcome of this attack likewise. Judging from these reports, it may be supposed that the cerebral activity of the workers exposed to nitroglycerol for many years is affected as well. To ascertain this, electroencephalography was undertaken for workers exposed to nitroglycerol.

MATERIALS AND METHODS

Cases investigated

The subjects for this study were nine workers exposed to nitroglycerol in a dynamite factory. Four of them had been experienced anginal attacks in the past at dynamite working, and then they were transposed personally to other workrooms. Five cases were the subjects random sampled from the workers now working in dynamite workroom for many years and not yet experienced anginal attack which had been assumed to be due to severe nitroglycerol intoxication. The outline of the cases are as shown in Table 1. There were no remarkable personal history and family history.
The concentration of nitroglycol vapor in working rooms of the investigated dynamite factory decreased notably year after year, and for these several years it had been below 0.2 ppm, TLV adopted in Japan.

**Electroencephalography and other clinical examinations**

Electroencephalograms were recorded formally using an electroencephalograph of type ME–132B (Nihonkoden Co.). The recording included 3 min of hyperventilation and activation by 10 ml of Megimide (0.5%) intravenous injection.

To avoid effects by drugs to the EEG findings, the subjects were prohibited any medication for at least a week before electroencephalography were undertaken. The cases now working in the dynamite workroom were kept off the shop for leastwise two days before EEG examination to avert the effects of acute nitroglycol exposure.

Subjective symptoms and some physical examinations, such as blood pressure, ECG, hematological examination, urinalysis, neurological examination and so forth, were checked up.

**RESULTS**

**Subjective symptoms and physical examinations**

Subjective symptoms and objective findings of the workers investigated are summarized as Table 2. Subjective symptoms in the transposed workers were none and or few, and a few but not so hard in cases now working in dynamite workroom. Except for EEG finding, there was no notable abnormal finding other than No. 9 in physical examinations.
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Table 2. Subjective symptoms and objective findings of the subjects.

<table>
<thead>
<tr>
<th>No.</th>
<th>Subjective symptoms</th>
<th>Abnormal clinical findings except EEG findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Palpitation at times</td>
<td>Nothing particularly</td>
</tr>
<tr>
<td>2</td>
<td>Nothing</td>
<td>Nothing particularly</td>
</tr>
<tr>
<td>3</td>
<td>Nothing</td>
<td>Nothing particularly</td>
</tr>
<tr>
<td>4</td>
<td>Paresthesia in the upper limbs</td>
<td>Nothing particularly</td>
</tr>
<tr>
<td>5</td>
<td>Paresthesia in the left wrist, headache and fatigue at times</td>
<td>Nothing particularly</td>
</tr>
<tr>
<td>6</td>
<td>Headache at times</td>
<td>Nothing particularly</td>
</tr>
<tr>
<td>7</td>
<td>Paresthesia in the left fingers, paltitation and feeling heavy in the head at times</td>
<td>Nothing particularly</td>
</tr>
<tr>
<td>8</td>
<td>Anorexia, stiffness of the shoulders, palpitation at times</td>
<td>Nothing particularly</td>
</tr>
<tr>
<td>9</td>
<td>Feeling heavy in the head and lumbago at times</td>
<td>Kestner's examination(+)</td>
</tr>
</tbody>
</table>

Notes: 1) Objective findings were tested by various examinations such as blood pressure, ECG, hematological examinations, urinalysis, neurological examination and so forth.
2) There was no episode in their past history and family history particularly.

Electroencephalogram

The EEG diagnosis on the subjects examined are summarized as Table 3. Seven cases out of nine indicated abnormal EEG record both in workers experienced anginal attack and not yet experienced such sign. The degree of the disorders diagnosed by electroencephalograph was not so severe but slight. There were some recording suspected epileptic disturbance and autonomic seizure, characteristic feature in EEG findings of the subjects exposed to nitroglycol seemed to be fast activity comparatively. The following are representative cases for EEG findings.

Case 1 (No. 1). Basic rhythm was 10~11 per sec diffuse activity with voltages between 30 and 60 microvolts in all leads. 25~30 per sec fast activity with some α-wave suppressed in all areas were remarkable in awake state, and shown 18~20 per sec fast wave in drowsy state. After activation by hyperventilation and intravenous injec-

Table 3. E. E. G. diagnosis in the subjects examined.

<table>
<thead>
<tr>
<th>No.</th>
<th>E. E. G. diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Abnormal record (fast wave, sharp &amp; wave)</td>
</tr>
<tr>
<td>2</td>
<td>Abnormal record (fast wave)</td>
</tr>
<tr>
<td>3</td>
<td>Slightly abnormal (epileptic wave)</td>
</tr>
<tr>
<td>4</td>
<td>Almost normal</td>
</tr>
<tr>
<td>5</td>
<td>Abnormal record (fast wave)</td>
</tr>
<tr>
<td>6</td>
<td>Abnormal record (fast wave &amp; autonomic seizure)</td>
</tr>
<tr>
<td>7</td>
<td>Slightly abnormal (sharp wave)</td>
</tr>
<tr>
<td>8</td>
<td>Almost normal</td>
</tr>
<tr>
<td>9</td>
<td>Abnormal record (fast wave, sharp &amp; wave)</td>
</tr>
</tbody>
</table>

Note: Electroencephalograms were recorded including sleep, hyperventilation and Megimide activation.
tion of Megimide, slow wave mixed with sharp wave, sharp and wave complex appeared in no localized areas sporadically.

Case 2 (No. 5). Basic rhythm was 10–11 per sec diffuse activity with voltages between 40 and 50 microvolts. In awake state, EEG showed 20–30 per sec α-wave with fast activity in all areas. 25–30 per sec much fast wave with about 30 microvolts were recorded especially in the central and frontal areas in drowsy state. By activation of hyperventilation and Megimide administration, these waves were slightly build up, but no spike and sharp wave appeared.

Case 3 (No. 6). Basic rhythm was 7–11 per sec activity with voltages between 30 and 60 microvolts. In awake state, 18–20 per sec fast waves were dominant in all areas. Both 25–30 per sec fast wave in all areas and episodical 6–7 and 14 per sec single positive and or negative spikes of no clear localization were recorded in drowsy state. No remarkable change appeared by activation.

DISCUSSION

There are many reports explaining the outcome of anginoid attack due to chronic nitroglycol poisoning. We have scarcely any report concerning EEG findings of the workers exposed to nitroglycol, notwithstanding affection of cerebral activity from nitroglycol exposure to the outcome are supposed as well. To clarify the affection of chronic nitroglycol exposure to cerebral activity, nine subjects, i.e. four of them had been experienced anginal attack in the past and were transposed personally to other from dynamite workroom and five wave random sampled from the workers now working in dynamite workroom for many years, were investigated.

Notwithstanding remarkable findings on the subjective symptoms and objective findings being poor, it was found that the incidence of abnormal electroencephalograms was markedly high in the former and the latter groups. From Gibbs' experience, the incidence of non-specific abnormal EEG in healthy adult was about 18%. In our cases seven out of nine showed abnormal EEG findings surprisingly. There were some recordings suspected epileptic disturbance and autonomic seizure, characteristic feature in EEG findings of the subjects seemed to be fast activity comparatively. It was of some interest that these findings had been indicated in the subjects who were transposed personally over two years to other from dynamite workroom before this examination. As for epileptic wave, according to Mabuchi, there is a case who had been working in a dynamite factory for many years and experienced anginal attack. EEG of this case showed spike waves in the temporal areas, and his symptom of the attack disappeared by administration of antiepileptic drug.

Toxicologic EEG findings give no evidence for a special focal discharge generally. The fast activity in our cases appeared in the diffuse areas. Appearance of the fast activity has been considered a pattern of slight disorders in a part of the brain and it was observed in some of the prisoners, patients of essential hypotension and chronic
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cerebrovascular disease,\textsuperscript{11}) and so forth. Remarkable fast activity were also observed in
the cases of the administration of such drugs as barbiturates\textsuperscript{12,13}) and reserpin\textsuperscript{14}), and
intoxication due to cycloserin\textsuperscript{15}) and others.

As known well, there are some disagreements between EEG findings and clinical
manifestations and or histological pattern, therefore, we must aware of limitations for
electroencephalography to appreciate cerebral lesion.

Though the degree of the disorders in our cases diagnosed by electroencephalography
was not so severe but slight, long term exposure to nitroglycol seemed to influence upon
the cerebral activity and cause some disorders. The clear significance of these EEG
findings in the course of chronic nitroglycol poisoning remains obscure. Further studies
into this problem are necessary.

Acknowledgement

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References

9) Mabuchi, C.: Personal communication.
physiol., 7, 1.
enceph. (Osaka), 10, 456. (in Japanese)