in Parkinsonism would seem to warrant more critical measures for evaluation of these symptoms, not only for the purpose of assessment of surgical results but also to elucidate the underlying neurologic mechanisms. A total of 197 Parkinsonians were investigated for rigidity by clinical examinations and marked exaggeration of rigidity was observed in 82%.

113 Parkinsonians and 20 normal controls were investigated electromyographically. In normal persons or postoperative Parkinsonians, who had complete alleviation of rigidity, no stretch discharge is elicited with or without this maneuver. All preoperative and postoperative patients who had incomplete alleviation of rigidity, stretch discharges were readily demonstrated by the contralateral movement. Electromyographical studies further substantiate this maneuver both for confirmation of the diagnosis as well as the evaluation of surgical results.

Pathophysiological mechanism of this phenomenon would seem to be related with the central activation to gamma system, not due to proprioceptive input from the contralateral limbs. However, the precise role at the gamma system in reinforcement of rigidity by the contralateral maneuver is not yet certain.

57. Supplemental Findings on Huntington’s Chorea

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In one group of patients with Huntington’s Chorea, chorea movements are localized to a certain part of the body and do not spread to the entire body during the remainder of their lives. This type of Huntington’s Chorea is called limited type (type L), and was first reported by Davenport. Recently, we experienced several cases of type L which appeared in the same family. We had questions such as the following.

1) What is the incidence of type L, compared with general type (type G) in which chorea movements spread to entire body?
2) What type of heredity does type L show?

A statistical observation was made by 311 cases with Huntington’s Chorea reported in Japan. The results were obtained as following;

1) The general incidence of Huntington’s Chorea is obviously lower in Japan than in other countries. However, the age of onset, the duration of the disease, and the average age of death due to the disease are same both in Japan and other countries.
2) If we examine the deceased cases in Japan, as in shown in the table, there is no difference in the age of onset, duration of the disease and the
average age of death due to the disease, between type G and type L.

<table>
<thead>
<tr>
<th></th>
<th>No. of family</th>
<th>No. of case</th>
<th>age of onset</th>
<th>duration of the disease</th>
<th>average age of death</th>
</tr>
</thead>
<tbody>
<tr>
<td>type G</td>
<td>20</td>
<td>43</td>
<td>37.7</td>
<td>13.6</td>
<td>51.4</td>
</tr>
<tr>
<td>type L</td>
<td>14</td>
<td>26</td>
<td>38.6</td>
<td>13.5</td>
<td>53.4</td>
</tr>
</tbody>
</table>

3) We examined 50 type L cases in 14 families, in which the same muscles were affected by the disease in each member of the same family. We found that occurrence of type L cases were only sporadic among siblings or between parents and children. There was no tendency that type L cases appear persistently in several generations. Thus, it became clear that type L is not a biotype which shows particular type of heredity but rather a particular form of expression (expressivity).

4) There are only 15-25% of L type among all Huntington's Chorea cases.

58. Pathological Study of Hemispherectomized Brains in Infantile Cerebral Palsy

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Infantile cerebral palsy associated with hemiplegia and uncontrollable convulsive seizure was operated on in five cases and the removed hemispheres investigated histopathologically.

In all five cases, the lesions are more or less circumscribed scarring softenings and, from their distributions and features, presumed to be resulted from insufficiency of the brain arteries. The middle and anterior cerebral artery should be most responsible for the lesion which tends to avoid the occipital lobe, medial aspect of the temporal lobe and hippocampus.

In the pathological features of the lesions, the cases with high fever at the onset (Case 1 & 2) do not differ essentially from those without fever (Case 3 & 4), while the cases with acute onset of hemiplegia followed by epileptic seizures (Case 1, 2, 3 & 4) are apparently different from that which started with convulsive seizure and developed hemiplegia afterward (Case 5). In the cases with acute onset of hemiplegia the feeding arteries were presumably affected near their proximal trunk, and in the other case (Case 5) the circulatory insufficiency took place probably in the peripheral arterioles.