THE EFFECTS OF CEREBRAL SENSORY CORTICAL ABLATION ON INSTRUMENTAL CONDITIONED REFLEX IN CATS WITH CEREBELLAR LESIONS

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Abstract. Cats were trained to reach with their fore limbs for food placed in horizontally mounted cylinders of various diameters. The chain instrumental reflex (entering cylinder, reaching for, grasping and bringing food toward the mouth) survived cerebellar paravermal cortical ablation or interposite-dentate nuclear lesion, with some exaggerated flexor or extensor responses respectively. Movements improved with time. Cats were then subjected to ablation of cerebral sensory cortex, SI and SII. SI or combined SI and SII lesion resulted in an initial period of decompensation of cerebellar symptoms. Sensory loss was also noted for 30–40 days after SI lesion and throughout the observation period up to 53 days after combined SI and SII lesion. The conditioned responses were soon accomplished at preoperative level. Furthermore, visual occlusion did not change the performance in conditioning situation. The result indicates that the compensation of cerebellar symptoms does not depend on an intact cerebral sensory cortex. It also suggests that functional accomplishment can be obtained through training despite neurological deficits following certain brain damage.

INTRODUCTION

In a previous investigation (1), cats were trained to reach with their fore limbs for food placed in horizontally mounted cylinders of various diameters. The chain instrumental reflex (entering cylinder, reaching
for, grasping and bringing food toward the mouth) survived cerebellar interposi
tive-dentate nuclear lesion or paravermal cortical ablation. Inter
posite animals displayed big oscillatory movements around the entrance
to the cylinder and overshooting in reaching for food with occasional
forced grasping. Paravermal cats displayed overflexion in entering the
cylinder and wild batting movements in the large cylinder.

According to Konorski (2), the feedback controlling skilled movements
is generated either by articular receptors, or by stretch receptors in
muscles and tendons, or by both. Articular receptors inform the brain
through the lemniscal afferent system about positions of the limbs and
their changes, while muscle-tendon receptors inform the brain through
cerebellum about movements. He postulated that these two systems could
to some extent substitute for one another and accordingly, if both were
destroyed, the animal would be strongly incapacitated in its motor be-
havior as revealed in their experiments by serial removals of the cerebel-
lum (or its paravermal part) and the somatosensory area of the cerebral
cortex.

In this study, those cats described above were subjected to cerebral
sensory cortical ablation. We attempted to determine the role of cerebral
sensory cortex on the conditioned and unconditioned behaviors of cats
with cerebellar lesions. We observed again that the functional accomplish-
ment can be obtained with training despite neurological deficits.

MATERIALS AND METHODS

The eight adult cats used in the previous investigation (1) were em-
ployed for this study. They were trained in a test chamber to reach
with their fore limbs for food (small pieces of bovine spleen) placed in
horizontally mounted transparent leucite cylinders with inside diameters
of 10.1, 7.6, 5.1 and 3.9 cm and length of 19–21.6 cm. Four cats (IP1–4)
had cerebellar interposi
tive nuclear lesions, two (IP5 and 6) with inter-
posite and dentate nuclear lesion and the other two (PV1 and 2) with
paravermal cortical ablation. The detail methods of training, surgery and
histology were described in the previous paper (1). In this study, all
eight cats were operated with cerebral sensory cortical lesions, SI or
SI and SII, as shown in Table I. The lesion was made aseptically by
gentle aspiration after craniotomy and opening of the dura mater under
Nembutal anesthesia. They were examined neurologically and tested in
the same test chamber throughout the survival period. IP1–4 and 6 were
also tested with visual occlusion to determine the effect of vision on the
conditioned responses and to confirm the tactile impairment. Visual occlu-
liders were black plastic cup-shaped with a short holder on the center of
the convex side and were applied to eyes like contact lenses.
Lesions and durations of observation. Abbreviations: B, bilateral; L, left; R, right; IP, interposite nucleus; D, dentate nucleus; PV, paravermal cortex; SI, primary sensory cortex; SII, secondary sensory cortex. In brackets, days in operative stages.

<table>
<thead>
<tr>
<th>Cat</th>
<th>1st operation</th>
<th>2nd operation</th>
<th>3rd operation</th>
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<tr>
<td>IP1</td>
<td>B. IP (22)</td>
<td>R. SI (25)</td>
<td>L. SI + SII (35)</td>
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<tr>
<td>IP2</td>
<td>B. IP (22)</td>
<td>R. SI (27)</td>
<td>L. SI + SII (40)</td>
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<td>B. IP (16)</td>
<td>R. SI + SII (38)</td>
<td>L. SI + SII (40)</td>
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<td>IP4</td>
<td>B. IP (34)</td>
<td>B. SI + SII (53)</td>
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<td>IP5</td>
<td>B. IP + D (58)</td>
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<td>IP6</td>
<td>B. IP + D (60)</td>
<td>R. SI + L. SI + SII (40)</td>
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<tr>
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<td>B. SI (38)</td>
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<td>PV2</td>
<td>L. PV (24)</td>
<td>B. SI (45)</td>
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RESULTS

Extensive but subtotal ablation of cerebral sensory cortex, SI or SI and SII (Fig. 1), in cats with cerebellar lesions resulted mainly in transient or permanent sensory loss and an initial period of decompensation of

Fig. 1. Lateral views of cerebral sensory cortical lesions (dotted area) plotted by gross inspection in four cats (IP2, IP3, IP5 and PV2). Dashed area indicates SI (upper) and SII (lower) according to Rose and Woolsey (5).
cerebellar symptoms. The conditioned responses (entering cylinder, reaching for, grasping and bringing food toward mouth with fore limbs) were soon accomplished at the preoperative level.

Loss of tactile sensation was noted for 30 to 40 days after SI lesion and throughout the survival period (up to 53 days) after combined SI and SII lesions. Severe impairment of placing responses (proprioceptive, visual, abduction and chin) and hopping reactions were also found initially after cerebral lesions and at least part of them may be manifestations of decompensation of cerebellar symptoms.

Following cerebral sensory cortical ablation, all cats with previous cerebellar lesions could walk in 1 to 4 days. Cats with interposite nuclear lesions (IP1–4) showed decompensation of cerebellar symptoms for 2 to 6 days, such as increased extensor tone, more marked goose stepping in walking and more ataxia in entering the cylinders and overshooting in reaching for food in the test chamber. The cats with interposite and dentate nuclear lesion (IP5 and 6) were noted to have a longer period of decompensation. IP5 was most severely impaired after cerebellar lesion and could not enter all the cylinders with its fore limbs until the 54th postoperative day. After bilateral SI and SII ablation, IP5 could not enter all the cylinders for 13 days. The cats with paravermal cortical ablation (PV1 and 2) were more ataxic with exaggerated flexor responses initially after bilateral SI lesion. However, entering all the cylinders with their fore limbs was accomplished by the fourth postoperative day. All cerebellar cats preferred to use the limb uninvolved or less involved by the cerebral lesion, but they could be trained to use either limb in the test chamber with very little difficulty.

Visual occlusion did not change the performance in the test chamber. Movements were usually slower and often slightly less ataxic when the animal was properly aligned with the opening of the cylinder. Sensory loss was clearly revealed in cats with visual occluders. The impaired limb could enter the cylinder, making movements of reaching for and grasping at food and then withdrawing to floor or bringing paw to mouth no matter whether food was in the paw or not.

In marked contrast to the performance in the test chamber (correct projection into cylinders and taking food placed inside with the fore limb) was the ataxia of the limb in reaching for food in a free situation. Throughout the observation period following cerebellar lesion alone or also with subsequent cerebral lesion, the animal used one limb exclusively and the other only when the preferred limb was restrained. When the cat tried with its involved fore limb to bat a piece of food held in air by forceps, the movements were always ataxic, undirected coarse swipes and food was obtained only by chance.
DISCUSSION

Subsequent extensive ablation of cerebral sensory cortex in cats with cerebellar lesions resulted in only a brief period of decompensation of cerebellar symptoms in walking and the performance in the test chamber. This indicates that the compensation of both unconditioned and conditioned behaviors in animals with cerebellar lesions does not depend on an intact cerebral sensory cortex. Our observations on compensation of ipsilateral limb hyperflexion after paravermal cortical ablation in an earlier study (4) also support this finding. Neodecortication in a cat recovering from paravermal cortical ablation brought back the limb hyperflexion but compensation occurred again with time. It is clear that recovery or compensation of cerebellar symptoms involves not only cerebral cortex but also lower centers.

Cats after cerebellar or cerebellar and cerebral sensory cortical lesions could perform the instrumental task even without vision and tactile sensation while they were ataxic and undirected in batting food in free situation. Thus, we observed again the marked difference of functional level in training and free situations as we did in the other investigations. For example, the monkey (3) or the cat (unpublished observation) with bulbar pyramid section showed permanent loss of tactile placing but could be trained to place following tactile stimuli in the conditioning situation. Results suggest that training might activate a functionally independent and parallel system essential to conditioned responses.

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REFERENCES


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