PARADOXICAL IMPROVEMENT IN HEMIPLEGIA FOLLOWING CORTICAL EXCISION

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Improvement in longstanding hemiparesis has resulted from complete ablation of the previously injured motor cortex. This unexpected result followed the operative removal of epileptogenic lesions in 3 cases of focal epilepsy.

David Ferrier removed the Rolandic cortex of monkeys and proved that here resided an area of motor control. The experiment had been well conceived and its significance was clear. However, observations such as we are about to record show that, although there is abundant clinical evidence to identify the deficit of the hemiplegic patient with that seen following removal of inactivation of the motor area, additional and perhaps more subtle disorder might operate as well during the course of human disease.

Improvement in the control of hemiparetic limbs as the result of excision of sensorimotor cortex seems to constitute a clinical paradox. In each of the patients to be reported, sensorimotor convolutions were excised in order to free the patients of epileptic attacks. In each case the hemiparesis was not made worse, but was actually improved. The observations that we have made are presented in the hope that they may cast light upon the neural mechanisms involved in hemiplegia, spasticity and recovery of motor function in man.

OBSERVATIONS

Before summarizing our observations, abstracts from the records of the 3 patients will be presented.

Case 1. L.W. A 22-year-old girl, was first seen Oct. 12, 1946. She complained of weakness of the right arm and leg and of seizures which were ushered in by sensation in the right arm and movement of the arm.

The weakness of the right arm and leg was first noted when she was a baby of 1 month and epileptiform attacks made their appearance at 11 years. In an attempt to stop the seizures she was operated upon that year by Dr. Charles Frazier. He discovered in the left hemisphere a porencephalic cyst which he unroofed. Attacks became less frequent for a year, stopped for 4 years and then recurred.

Examination in Montreal showed the patient to be a tall left-handed young woman. The right arm and leg were small by comparison with the other side. Movement of these extremities was limited. Her chief difficulty in using them was the stiffness produced by spasticity. She walked with a pronounced limp and used the hand for nothing more than the simplest actions.

The tendon reflexes were exaggerated on the right side and the plantar response was extensor. Two-point discrimination and position sense were severely impaired.
on that side and examination of the visual fields showed that she had a right inferior quadratic anopsia.

EEGs gave evidence of a localized epileptogenic area in the left frontal region. "Spikes" and "sharp wave" variations in potential were recorded consistently from this area.

1st Operation. Left craniotomy and partial removal of meningocerebral cicatrix. On Oct. 16, 1946, the left hemisphere was exposed through the cranial defect left at the time of the previous operation. The scalp was found to be adherent to dura and the dura to brain. Anteriorly the frontal lobe appeared quite normal but for a distance of 3 or 4 cm. from the edge of the former opening backward there were dense adhesions.

The patient had two spontaneous seizures during the early stages of the operation. The operation was carried out under local anesthesia but stimulation produced no response from the exposed cortex, possibly because of the attacks. A large excision of abnormal cortex was made in the precentral region (Fig. 1).

Course. The attacks continued to occur, though with decreased frequency. Consequently 8 months later a second operation was undertaken.

2nd Operation. Left craniotomy and removal of meningocerebral cicatrix including the remainder of central area. The brain was exposed a second time using local analgesia. Stimulation now produced both sensory and motor responses in arm and leg as shown in Fig. 1. It was thought that this responsive area was part of the postcentral gyrus. There was some cerebral tissue anterior to this gyrus, but no response was obtained upon stimulating this. In the central portion of the hemisphere there was a large fluid-filled cavity. No other responses were obtained by stimulation of its banks.

It was decided to complete the removal of all cortex surrounding the central area of destruction. The patient's strength of voluntary movement of hand and foot was tested. She was warned that in order to stop the attacks we would have to increase the weakness of hand and foot. The responsive convolution and all other convolutions above the fissure of Sylvius which formed the posterior border of the area of destruction were then removed.

The anesthetist, Dr. Nitikman, then tested strength in the opposite hand and foot and found no decrease whatever!

Postoperative Course. Careful study showed that there was an increase in the two-point and position sense deficit but no increased paresis.

When seen 2 years after operation, she had had no postoperative convulsive

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**Fig. 1. Case 1.** Interrupted line = extent of first excision. Crossed line = 2nd excision. The stimulation results were obtained at the 2nd operation, utilizing the Rahm stimulator, frequency 60, 3 V. Point 1: Movement of 3rd, 4th, and 5th fingers of right hand. Point 1: (Patient was asked to keep her hand still.) Tingling of ulnar side of hand. Point 3: Sensation in right arm followed by movement of this arm. Point 4: Sudden movement of right arm and a feeling like that which habitually occurred before an attack, 1½ V., Point 10: Tingling in right leg.
seizures. However, it was clear that there was a remarkable change in the patient's hemiparesis. Instead of carrying the paretic upper extremity in a flexed and spastic manner as she had done before, she now kept her arm extended by her side (see Fig. 2). The muscles were plastic. She was beginning to use the hand for eating which had never been possible before. In walking she could swing her leg without the former spastic stiffness so that her hemiplegic limp had actually disappeared. She had spent the year doing satisfactory university work.

The actual removal of tissue in the two operations was confined to cortex. In Fig. 1, the extent of removal by both operations is indicated. Some improvement in function may have followed the first operation but it was certainly accelerated by the second procedure.

Case 2. A.A., a 12-year-old girl, was first admitted to the Montreal Neurological Institute June 21, 1947, complaining of seizures.

She was well until she was 2 1/2 years of age when she had a severe febrile illness with a temperature which is said to have reached 107° F. A generalized seizure occurred at that time and for 6 days afterward there were numerous bouts of muscular twitching. Then another severe seizure involving the left arm, face and leg occurred and following this attack the left arm and leg were paralyzed.

For 2 years preceding her admission the child had seizures characterized in onset by a flexion and elevation of the left arm, and for 1 year she had in addition small attacks in which she was unable to speak.

On examination it was found that the left arm and leg were smaller than those on the right. The patient walked with a spastic gait, the left arm held rigidly flexed and adducted. There was left spastic hemiplegia with hyperactive tendon reflexes and plantar extension on that side.

X-rays of the skull showed a small right hemicranium. Pneumoencephalogram revealed cerebral atrophy most pronounced on the right, and lowering of the floor of the right lateral ventricle suggested that this process involved the basal nuclei as well.

EEGs revealed high voltage "sharp waves" and rhythmic disturbances at 2 to 3/sec., maximal from the right frontal region.

After a trial of medical management, she was readmitted Jan. 5, 1948, because attacks had continued.

1st Operation, Jan. 10, 1948. Right osteoplastic craniotomy and removal of atrophic area of brain. There were adhesions between brain and dura, particularly in the region of the Sylvian fissure. This fissure was wide due to underlying atrophy, and there was an area of small, leathery gyri posterior to the central sulcus and above the fissure of Sylvius. A very small gyrus, which joined both the pre- and postcentral gyri above, was the downward continuation of both.

Stimulation produced movements in the paretic extremities though at somewhat higher threshold than average (Fig. 3).

Excision included the pre- and postcentral gyri of the arm subdivision and below,
down to the fissure of Sylvius as well as the shrivelled parietal gyri (Fig. 3). When the excision was completed she could move her arm as well as before.

**Course.** There was never any increased motor disability postoperatively. The patient was readmitted March 10, 1948, because attacks had continued. Examination disclosed that her spasticity was less severe than before in both the arm and leg on the left side. The decrease in spasticity was most marked in the elbow, although her gait had improved as well.

**2nd Operation. April 3, 1948.** Right osteoplastic craniotomy and removal of atrophic area of brain. There were adhesions beneath the dura. The banks of the previous removal were satisfactory. The patient’s attacks could be reproduced by stimulation anterior to the upper part of the precentral gyrus and a large removal was carried out (Fig. 3).

**Postoperative Course.** There was no increase in paresis in the contralateral extremities, and spasticity in both the arm and the leg was less pronounced than before.

**Case 3.** A.M., a 25-year-old woman, was admitted to the Montreal Neurological Institute, Feb. 14, 1937, complaining of seizures for 15 years.

The patient had been born spontaneously after transverse presentation. At the age of 1 week her mother noticed that her right arm and leg were weak, and walking was delayed until the age of 3 years on this account.

Fifteen years before her admission she began to have seizures characterized, in
onset, by a pounding sensation at the top of the head followed by shaking of the right arm. These attacks became generalized.

*Examination disclosed* spastic hemiplegia with exaggerated deep tendon reflexes and plantar extension on the right side. There was impairment of position sense and stereognosis on the right, and smallness of the right extremities.

*Operation. Feb. 18, 1937. Left osteoplastic craniotomy and removal of cerebral cicatrix.* The brain was seen to have undergone atrophy in the region of the Sylvian fissure and extending upward and backward beyond the limits of this fissure. The precentral region was atrophic up to the midline.

The stimulation results identified the central sulcus. A large excision including pre- and postcentral gyri was carried out (Fig. 4.)

*Postoperative Course.* There was no increased weakness of the paretic extremities. There was an homonymous hemianopsia.

*Summary of Results.* Large removals of sensorimotor cortex were made in 3 patients who had been hemiplegic since infancy and who exhibited focal cerebral seizures. No increased paresis was seen in the contralateral extremities following operation although the excised cortex had been electrically excitable. There was decreased spasticity in the paretic extremities after operation, and as a result, motor performance was improved.

**DISCUSSION**

*The Nature of the Deficit.* Since the results of these excisions are clearly unusual* it is well to examine the pathological state of the sensorimotor areas in these cases from anatomical and functional viewpoints. Anatomically each of these patients showed an objective lesion involving the Rolandoic region and other portions of the exposed cortex as well. A large porencephalic cyst with surrounding atrophic gyri was found in Case 1. This must have been present from birth and resulted from birth injury or some ante-natal cause. The cerebral atrophy seen in Case 2 followed an infection at the age of 2 years, and the mechanism of injury was probably thrombosis of cerebral veins. In Case 3, the atrophy was due to obstruction of the middle cerebral artery.†

Each of these patients showed a functional deficit which indicated damage to both pre- and postcentral areas. In each, the cortex was, however, electrically excitable in that movements of the paretic extremities were produced by stimulation of the sensorimotor cortex. The responses were elicited at higher threshold than usual, however. Following the cortical excisions which have been outlined, no increased deficit in posture or movement could be found.

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* It should be emphasized that the usual result of removal of the precentral gyrus even in hemiplegic patients is an increased paralysis. The cortical excisions reported here were done with the full expectation that this would occur, but with the hope that this would be less of a handicap than the seizures. In the present state of knowledge it is not possible to predict in advance which patients will show no increased deficit following such an excision and we do not advocate this approach for the treatment of spasticity.

† This case was reported, together with several similar ones, by Evans and McEachern. Patient No. 2566 in their report is the patient under discussion here.
It can be concluded, therefore, that the activity of these atrophic sensorimotor areas was not essential for the performance of movements, impaired as they were in these patients. That the Rolandoic cortex had retained a capacity to influence anterior horn cells is demonstrated by the responses to electrical stimulation. This suggests that the clinical deficit was partly due to isolation or de-afferentiation of the damaged but electrically excitable cortex or, in other words, that the paralysis was, in part, of supracortical origin.

Reorganization of Function. The recovery of function that occurs after injury to the motor area has been the subject of considerable interest, especially during the past 15 years. As yet, however, the factors and mechanisms involved in recovery are not clear, the experimental data from laboratory animals are conflicting, and criteria for acceptance of recovery as reorganization of function are not apparent.

Few facts pertaining to the portion of the nervous system responsible for recovery have stood the test of repeated experiment. The data that lent strong support to the idea that the ipsilateral motor area takes over motor function in monkeys\(^2,5\) have not been entirely substantiated.\(^3,8,10\) Kennard\(^6\) has adduced evidence in monkeys for the importance of the postcentral and frontal association areas (on the side of the motor area lesion) in recovery, but it is questionable that this represents true reorganization in the sense that a new or vicarious function has been assumed by these cortical areas. The evidence for the participation of subcortical structures in recovery of function is entirely based upon exclusion of participation of other parts.\(^8\)

The present observations lend no support to the concept of participation of the postcentral or intermediate precentral region in motor recovery in man as our excisions did not increase paresis. In each of the patients, however, there was evidence that the original lesion involved the postcentral gyrus as there were impaired stereognosis, two-point discrimination and position sense as well as smallness of the contralateral extremities before operation was carried out.

If true reorganization of function occurs, the sites where motor function becomes secondarily localized must depend upon the nature and extent of the original injury. The gross forces of birth injury, obstruction of a middle cerebral artery and thrombosis of cerebral veins which operated in our cases did not respect the boundaries of cortical areas and certainly involved subcortical structures as well as cortex. It is not surprising, therefore, that our observations are not in accord with those obtained in animal experimentation where the extent of the original lesion can be accurately determined and where the remainder of the brain is uninjured.

Spasticity. Spasticity has not been increased in these patients following operation. Indeed in \(2\) the change has been in the opposite direction and has allowed improved motor performance. Browder\(^4\) reported decreased spasticity and consequent improvement in motor ability following large removals of parietal cortex in patients showing spastic hemiplegia. It is, of course,
impossible to attribute the decreased spasticity that we have observed to any one part of the ablation—precentral or postcentral. In any event, Browder’s observations and ours show an important species difference between man and other primates. In monkeys and chimpanzees existing spasticity is increased by postcentral excision and a flaccid paralysis can be converted into a spastic one by removing the postcentral gyrus.  

Evidence from many sources, summarized by Magoun and Rhines, indicates that, in addition to being a release phenomenon depending upon the removal of inhibitory influences, spasticity depends upon intact and functioning facilitatory systems. In our cases the damaged sensorimotor cortex seems to have reinforced the spinal stretch reflexes so that when its influence was removed depression rather than release resulted.

**SUMMARY AND CONCLUSIONS**

1. Injured cortex, capable of producing movement in paretic limbs when stimulated and of causing movement during epileptic discharge, has been removed without producing increase of functional disability in the extremities.

2. Excision of these areas has resulted in decreased spasticity and consequently produced paradoxical improvement in the use of the limbs.

3. The postcentral gyrus and the intermediate precentral area did not take part in reorganization of function after the original injury in these cases.

4. Sensorimotor cortex which retains the capacity to influence spinal motor mechanisms, but which plays no essential role in the voluntary control of these mechanisms, seems to have exerted a specific augmenting influence upon the spinal reflexes of the involved parts. Decrease of spasticity and increase of normal function resulted from ablation of abnormal sensorimotor gyri.

**REFERENCES**


