The Interplay Between Cerebral Hemispheres and Cerebellum in Relation to Tonus and Movements*

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There is a system of checks and balances at various levels of the brain and spinal cord which is responsible for the well modulated tonus and the coordinated movements of the head, trunk, and extremities that characterize normal behavior. The balance between the discharges from the cerebral cortex at one end of the arc, and the cerebellum at the opposite end is demonstrated in the present paper. The material is the result of studies of elective lesions made in over 300 operations on the macaque and 4 surgical procedures on man. The conclusions drawn from this study are illustrated best by the presentation of 2 selected cases, 1 in the monkey and the other in man.

Experimental Case

On June 10, 1959 a left posterior parietal and suboccipital craniectomy was performed on a macaque (Monkey 4711). The left occipital lobe was elevated, the tentorium was incised, and, with cautious retraction of the cerebellar hemisphere, the left superior cerebellar peduncle and adjoining areas were sectioned. Postoperatively the animal’s head deviated toward the left side. The left pupil was smaller than the right and there was a tendency for the eyes to drift upward. Hypotonicity was more marked in the left upper than in the left lower extremity.

For 6½ months the animal could not stand but lay at the bottom of the cage. The head was tilted with the chin to the left. Both lower extremities were hypotonic and the left upper extremity displayed less tonus than the right upper one. When the animal was placed on her left side on the bottom of the cage she would immediately roll over to the right side and use the left extremities to push herself about in a circle. During this interval the animal never was able to regain a standing position or to walk.

A right frontoparietal craniectomy with subtotal hemispheric cortectomy was performed on Jan. 7, 1960. Because of the animal’s precarious condition, parts of the amygdala, the hippocampus and the medial surface of the temporal lobe were not excised, but the operation was terminated rapidly to save the animal. She is pictured 3 weeks postoperatively still in essentially the same condition as prior to the second operation (Fig. 1). However, she improved gradually over a period of 4 months and tonus was regained in the left upper extremity and the left lower extremity. The animal regained her feet, walked, and tried to use the wall as a crutch by supporting her right side against the wall (Fig. 2).

When the animal was sacrificed, the gross specimen exhibited the right cortectomy with the residual cortex which included part of occipitoparietal cortex and part of the temporal lobe, the

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Fig. 1. Monkey 4711. 7 months after left superior cerebellar pedunculotomy and 3 weeks after the second procedure, right hemispheric cortectomy. The animal still was unable to stand or walk but lay on her right side using her left extremities to push herself about in a circle on the floor.
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Fig. 2. Monkey 4711 demonstrated ability to walk 4 months following subtotal right hemispheric cortectomy. Although increased tonus of the left extremities made walking possible the gait was awkward because of the sensory loss in these extremities.

Fig. 3. Monkey 4711. Right lateral view of brain showing residual right cortical and subcortical tissue.

Fig. 4. Monkey 4711. A posterior view of the 4th ventricle and brain stem is shown with pointer indicating the site of incision in the left superior cerebellar peduncle.

amygdala, the hippocampus and a small veil over the temporal horn of the right lateral ventricle (Fig. 3). Inspection of the lateral wall of the 4th ventricle demonstrated that the left superior cerebellar peduncle had been sectioned (Fig. 4).

Selected microscopic sections demonstrate the location and extent of the lesions responsible for the alteration in the animal's tonus and movement. The plane through the abducens-facial level of the pons reveals the most extensive part of the left cerebellar lesion (Fig. 5) and shows the degree of destruction of the brachium conjunctivum and the other homolateral cerebellar peduncles. On the left, the spinal tract of V is involved particularly, as is also the major portion of the medial lemniscus. It should be emphasized that the sensory loss, including the proprioception from the body by the lesion in the medial lemniscus, will appear contralateral to the cerebellar lesion and therefore will not increase the ataxia on the side of the latter involvement. The degenerated right corticospinal tract is readily visible.

A microscopic section at the level of the N. XII shows the degenerated right corticospinal tract
and degenerated left spinal tract of N. V (Fig. 6). It demonstrates that there are no other regions of destruction below this level.

The third section taken at midbrain levels, at the transition between planes through the inferior and superior colliculi, contrasts the unpaired paths of discharge from the right side of the cerebellum with the degenerated fibers of the brachium conjunctivum from the injury on the left side of the cerebellum and the cerebellar peduncles (Fig. 7). The degeneration of the capsular fibers provided by the cerebellar peduncle for the caudal end of the red nucleus on the side contralateral to the cerebellar injury can be seen. The light areas in the tegmentum on both sides are caused by degeneration of the cerebellotegmental and corticotegmental pathways.

**Comment.** In this instance the left cerebellar lesion was compensated for by the right subtotal cortectomy so that the animal attained sufficient tonus to walk. The sensory loss in the right upper and lower extremities also was definitely responsible for a failure of the monkey to assume a more normal type of gait.

### Human Case

An 8-year-old boy, G.I., who had suffered from erythroblastosis fetalis with severe residual choreo-athetosis, was admitted to University Hospital on Sept. 8, 1958. The patient was the youngest of eight children. He had definite mental retardation with an IQ of 51 and was unable to communicate by speech. There was a marked degree of athetoid movement and spasticity with more involvement in the right arm than in the left. He was almost totally incapacitated but had the ability to grasp a toy awkwardly with the left hand. On the slightest stimulus the lower extremities became extended at the knee with a 90-degree flexion at the hip (Fig. 8). The upper extremities tended to extend at the elbows and the hands closed with moderate spastic flexion of the fingers. The left arm most frequently was thrown upward to the height of the shoulder or above the head.

![Fig. 8. Preoperative photograph illustrates the marked degree of spastic involuntary movements. At the slightest stimulus the legs were thrown upward into marked extension, the arms were extended, with fingers flexed and facial grimacing obliterated all normal or voluntary expression.](image-url)
Marked facial grimacing occurred so that the face became contorted and the spasms even seemed to involve the muscles of the neck, the tongue, and the throat.

Roentgenograms of his skull and chest, pneumoencephalogram, lumbar puncture and cerebrospinal-fluid studies all gave normal findings. The electroencephalogram was borderline with a mild diffuse dysrhythmia questionably more focal in the right posterior parieto-occipital region.

Since there had been little or no medical control of his symptoms, the possibilities of surgical treatment were discussed with the family. The father and the mother, who was a nurse, were told that cerebellectomy, which was proposed, was strictly a research approach to the problem. The dangers related to such a procedure were impressed upon them. They agreed to the operation, for they had cared for him in the home with the seven other children since birth and were reluctant to have him placed in an institution.

On Sept. 18, 1958, under local anesthesia supplemented by small amounts of Surital and Tri-lene, a complete suboccipital craniotomy was performed with exposure of the vermis and both cerebellar hemispheres. Repeated electroencephalographic recordings were made prior to, and following, stimulation of the hemispheres, vermis and tonsils, at 40 c./sec., 1.5 amperes, and voltages varying from 4 to 9 V, without significant changes in the electroencephalogram. Approximately 3 X 3 X 4 cm. of the inferior medial half of the right cerebellar hemisphere were excised, sparing the dentate nucleus. The cerebellar tonsil was removed bilaterally and nodule, uvula, lingula, pyramid (and presumably the caudal part of the fastigial nucleus) also were excised but during this period the patient, who had been responding readily by moving about, suddenly became unresponsive without any alteration occurring in his vital signs and without change in his anesthesia. The cause for this change in the state of consciousness could not be determined. The wound was closed and the operation was concluded.

The patient was discharged on Oct. 7, 1958, 3 weeks after operation. He moved his head in all directions, but did not respond to any simple commands. Rather he had a negative attitude, constantly turning away from the examiner. He made gutteral sounds with a cry and had smacking movements of the lips with associated movements of the tongue. The pupils were equal and reacted to light. There was no nasal rigidity but his head offered resistance to flexion and he tended to hold it in extension. There was marked spasticity in all extremities, but the left arm was much looser than the right one. He was unable to grip with the right hand and had no prehensile movements with the left hand and fingers. There was a coarse kinetic tremor bilaterally, more on the right than the left. No true athetoid movements were present. The deep tendon reflexes and Hoffmann’s signs could not be checked because of the degree of spasticity. The legs were held drawn up close to the trunk with the thigh and the leg in flexion and marked adductor spasticity. On May 25, 1959 the patient was seen as an outpatient. There had been considerable improvement. The spasticity had decreased, but he still had athetoid movements in each extremity. Nine months after operation the boy could smile, and had less movement of the head, loss of severe spasms in the lower extremities and better control of the left arm. His finger-to-nose test was performed reasonably well, considering his preoperative status. He could drink a glass of milk and feed himself with some difficulty (Fig. 9). The lower extremities were hypotonic (Fig. 10) and he could flex voluntarily his lower extremities at the knee with only slight spasm (Fig. 11). Three and one-half years after operation the movement of the head had become even less noticeable; his finger-to-nose test had improved. After a prolonged period he could pound out words on a typewriter. He usually used an electric one at school and the portable one pictured here provided a much sterner test (Fig. 12).

Comment. This case demonstrates considerable improvement in the choreo-athetotic patient following cerebellectomy with the realization that the results are far from excellent. The discharge and progress notes are included to indicate how remarkably little change there had been in the patient’s condition immediately postoperatively, and demonstrate that balancing of tone and reduction of involuntary movements occurred gradually over a period of months or years. This case therefore sounds a note of caution that such surgical procedures should be undertaken with care for there may be an overswing from spasticity on the one side of the arc to too great hypotonicity on the other. In this instance the child might be better able to stand and even walk if he were not quite so hypotonic.

The case also demonstrates how the excision of part of the vermis and of one cerebellar hemisphere may have a marked bilateral effect on the lower extremities. If the procedure were to be repeated, it would be better to be a little less radical in excision...
Richard C. Schneider and Elizabeth C. Crosby

Fig. 9 (left). The patient could now haltingly raise a glass of milk to his mouth and drink it. This was facilitated to some degree by the loss of the severe extensor spasm of the legs as well as diminished tone in the arms.

Fig. 10 (right). A year and a half postoperatively there was hypotonia in all four extremities which was more noticeable in the lower than upper extremities.

of the lower part of the cerebellar hemisphere and to excise more of the upper part of its lower medial half or third to have a greater influence on the upper extremities.

The advantage of the surgical attack on the cerebellum with its modification of tonus and movement as compared with a lesion in the globus pallidus or a thalamotomy is that it presents less danger of altering the mental-ity or changing the personality. If the surgical excision of the cerebellum could be regulated properly it might permit bilateral relief of symptoms by a single operation.

Anatomical Considerations and Conclusions

The cortical areas along the central fissure which, on stimulation, yield motor responses

Fig. 11 (left). The degree of voluntary movement of the lower extremities is demonstrated 2½ years postoperatively.

Fig. 12 (right). Control of the right hand was far from perfect, but 3½ years after his operation, he could type very slowly using one finger.
in the well recognized sequence of movements of body and face, are well known. Various other cortical areas in parietal, temporal, occipital, preoccipital, frontal, and cingulate cortices (as well as in hippocampus and portions of the amygdaloid complex) from which movements of the face and body likewise can be elicited on stimulation, have been termed additional, supplementary, second motor or extrapyramidal areas. The responses from the supplementary motor areas may be homolateral, contralateral or bilateral for extremities and/or the face. Sometimes, on strong stimulation, a generalized response occurs with bilateral turning of the head, body, and sometimes the eyes, away from the side of irritation—the advesive response of Foerster.

In the normally functioning individual, the supplementary motor areas serve several functions, some of which have been discussed by various observers who have considered the motor patterns elicitable and, in some cases, the pertinent connections of these areas. The present paper deals with the interplay between the supplementary motor areas of the cerebral cortex on the one hand and the posterior lobe of the cerebellum and the deep cerebellar nuclei on the other, in the regulation of tonus and of involuntary movements. For those interested some details of the pertinent connections follow:

The supplementary motor areas discharge to brain-stem centers by various paths (Figs. 13 and 14). Corticostriate fibers have been demonstrated by various observers from supplementary

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![Diagram of brain structures showing the interplay between cerebrum and cerebellum](image-url)
motor areas (such as area 6,1,3 parietal,1 temporal,1,6,20,21 insular1,22 and cingulate22 regions) to the putamen and directly, or after relay over intrinsic neurons, to the globus pallidus.9 Some of these corticostriate fibers pass directly into the putamen and the globus pallidus, others reach these areas by way of the internal or the external capsule (Fig. 14). Two major paths of discharge from the lenticular nucleus are the lenticular fasciculus and the ansa lenticularis.

The lenticular fasciculus and the ansa lenticularis have both cortical and lenticular components (Figs. 13 and 14). The cortical component from more dorsal regions of the cortex (as frontal, parietal and parts of the insular cortex) joins the lenticular component after coursing through the external capsule.1,3 The fascicles from the temporal cortex reach the ansa lenticularis and the lenticular fasciculus by a sublenticular course. The lenticular fasciculus17 crosses from its origin in the globus pallidus through the posterior limb of the internal capsule and continues directly medialward, above the subthalamic nucleus, to the field of Forel. The ansa lenticularis accumulates sublenticularly after its origin from the putamen and the globus pallidus, then crosses into the diencephalon rostral and ventral to the posterior limb of the internal capsule. It loops dorsalward, ventromedially to the subthalamic nucleus, to reach the nucleus of the field of Forel. Apparently all of the lenticular components of the ansa lenticularis and many, but not all, of those in the lenticular fasciculus11 relay in this nucleus of the field of Forel but part of the cortical components in each bundle, joined by fascicles arising from the nucleus itself, proceed caudalward to the midbrain. The lenticular fasciculus and its accompanying fascicles end in the red nucleus and the tegmentum of the midbrain, particularly the parts lateral and dorsolateral to the red nucleus (including the interstitial nucleus of the medial longitudinal fasciculus and the nucleus of Darkschewitsch, which are not illustrated). The augmented ansa lenticularis discharges to the tegmentum of the midbrain ventral and ventrolateral to, and behind, the red nucleus.

![Fig. 14.](image)
Direct pathways also connect at least many of the supplementary motor areas with the tegmentum of the midbrain. From the frontal cortex corticorubral and corticogranular paths have been demonstrated. They reach their destination by the posterior limb of the internal capsule. Other corticofugal parts pass from parietal, temporal, and occipital areas (area 19) by postlenticular pathways. Corticofugal connections from parieto-temporal (including pyriform cortex) have been demonstrated by physiological methods. For lack of space most of the direct corticofugal pathways have not been illustrated. There are still other pathways of discharge from supplementary motor areas to the midbrain which cannot be considered here.

In the tegmentum of the midbrain the descending systems just described from the supplementary motor areas synapse with various other bundles of fibers (Figs. 13 and 14); among such bundles are the crossed and the uncrossed ascending fascicles from the cerebellum and the homolateral and the contralateral bundles of fibers of the multisynaptic ascending reticular system.

The cerebellum is concerned with the maintenance of equilibrium and with the preservation of normal posture at rest and in motion. It is, then, related to the production of tonus and the stabilization of movements which make such normal posture possible. Most of the paths of discharge of the cerebellum arise in the deep cerebellar nuclei (Fig. 13). Cerebellothalamic fibers arising from the dentate nucleus (Fig. 13), cerebellorubral fibers from the dentate and emboliform nuclei (Fig. 13) and the cerebellotegmental fascicles having origin largely in the emboliform and the globose nuclei (Figs. 13 and 14) leave the cerebellum through the brachium conjunctivum. The cerebellorubral and the cerebellothalamic bundles decussate at inferior collicular levels (Fig. 13) and then proceed rostrally to the red nucleus and to the nucleus ventralis lateralis of the dorsal thalamus, respectively. The cerebellotegmental fibers (Figs. 13 and 14) terminate, in part homolaterally and in part contralaterally, in the tegmentum of the midbrain through an area extending rostrally into planes cutting the rostral one-third of the superior colliculus and caudally to planes behind the level of decussation of the brachium conjunctivum. From the tegmental gray of the midbrain, which receives impulses bilaterally from both the ascending multisynaptic reticular system and the cerebellotegmental fascicles, relay is made to the intralaminar nuclei of the dorsal thalamus. It seems probable that some fibers accompanying the cerebellotegmental paths pass through the midbrain to project directly upon the intralaminar nuclei (Fig. 14). These nuclei connect with the surrounding thalamic nuclei (such as the dorso medial and the lateral) which, in turn, discharge impulses reaching them in this fashion from the midbrain to the supplementary areas of the cerebral cortex.

Impulses projected over cerebellotegmental paths to the more ventral portion of the tegmentum of the midbrain are relayed by short tegmentonigral fascicles to the substantia nigra (Fig. 13). The substantia nigra projects to the lenticular nucleus over the well known nigrostriatal path.

Destruction of one dentate nucleus produces homolateral hypotonicity and an action tremor. Section of the brachium conjunctivum, in its course from its origin to its decussation, has a like effect. Bilateral lesions in this peduncle more than double the effect. Destruction of the decussation of the brachia conjunctiva gives bilateral hypotonicity and action tremor. Rostral to the decussation, as the brachium fibers end in the red nucleus or run in its capsule, a unilateral involvement produces a hypotonicity and an action tremor contralateral to the lesion. Hemorrhages into the nucleus ventralis lateralis sometimes give a like result.

Lesions in the substantia nigra also produce hypotonicity. Either irritative or destructive lesions in the tegmentum of the midbrain may be indicated by tremor. Action tremor is obtained from regions lateral to, or dorsolateral to, the red nucleus. Postural tremor has been elicited in the macaque from lesions of the tegmentum of the midbrain just dorsal to the substantia nigra and has been abolished by bilateral gross destruction of the substantia nigra in some human patients.

If the midbrain is cut off from the areas in front of it or if a bilateral cortectomy is carried out, so that, in either case, the supplementary motor discharges and the motor discharge to brain stem and spinal-cord centers are disrupted, a marked hypertonicity or opisthotonus appears. As the present experiments have shown, this can be somewhat reduced by large lesions in the posterior lobe of the cerebellum.

**Discussion**

It would appear, then, that impulses from the cerebellum are balanced against those carried over pathways from the supplementary motor areas of the cortex at cortical, basal ganglionic, thalamic, and midbrain levels and also at the motor levels in brain stem and spinal cord. This balancing serves to give adequate tonus and stabilized (smooth, efficient) responses for voluntary and associated automatic movements. When, as in the experiment on the monkey, there is destruction of the cerebellar peduncles (in-
cluding especially the superior cerebellar peduncle) and of portions of the posterior lobe of the cerebellum on one side, an imbalance is set up between the efferent discharges of the cerebellum, on the one hand, and the efferent discharges of the cerebral cortex (especially the contralateral cortex) on the other hand. A handicapping hypotonicity results on the side of the cerebellar lesion. The experiment here reported, and others like it in which the results have been documented, show that this imbalance can be made somewhat less, with some improvement in the motor performance of the animal, by removal of the cerebral cortex contralateral to the original cerebellar lesion. Although the lesions were increased, the motor behavior of the animal improved.

A reverse process is illustrated in the case of the child with marked hypertonia and many involuntary movements caused by involvement of motor and supplementary motor areas of the cerebral cortex. In this case when the discharges from the posterior lobe of the cerebellum to higher levels were unregulated largely by the efferent cerebral cortex centers again a handicapping imbalance had been set up. Removal of considerable portions of the posterior lobe of the cerebellum lessened the cerebellar discharge to midbrain and thalamic regions (and so indirectly to cerebral cortex) and thus established a smoother motor performance, with more satisfactory tonus, although the brain lesions had been increased. The various other surgical procedures, such as pallidectomies, thalamotomies, lesions in substantia nigra and certain cortical destructions in the brain-damaged individual likewise establish a better balance between cerebral hemisphere and cerebellar discharges by placing lesions at some one of the various levels where these systems balance.

Summary

Destruction of the motor areas of one cerebral hemisphere does not prohibit cortical regulation of movement contralaterally (or bilaterally) although fine movements are lost contralaterally.

Both tonus and movement may be altered by a lesion anywhere along the pathway from the cortex at one end of the arc through the basal ganglia, thalamus, and midbrain levels to the cerebellum at the opposite end of the complex. The balancing of the cerebral cortex against the contralateral cerebellum occurs at various levels of the brain.

Selection of an operative site for alteration of tonus and prevention of involuntary movements in the brain-damaged individual will depend upon the demonstrable neurologic deficits and will also be governed by the intention of obtaining as few undesirable side effects postoperatively as possible.

A monkey is presented, in which a left superior cerebellar pedunculotomy (with destruction of adjoining cerebellar and brain-stem areas) had caused marked hypotonicity and ataxia. The animal received benefit from a compensatory lesion—a contralateral or right-sided cortectomy.

A boy with severe choreo-a-thetosis had an amelioration of symptoms after a unilateral partial posterior cerebellectomy. The advantages of such a lesion over a more centrally placed subcortical one are: 1) a bilateral effect from a unilateral cerebellar lesion, and 2) less danger of injuring the sensorium of an individual with an already impaired mental status.

A warning is issued against too radical cerebellar excision in such individuals since adjustments of tonus and movement may continue even 2 or 3 years after operation.

The anatomical pathways for arriving at these conclusions are presented for those readers who may be interested.

References

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Discussion

Dr. Claude Bertrand: Immediate reduction of cerebral rigidity obtained with chlorpromazine is indisputable; its marked diminution with lesions of the globus pallidus also is quite evident.

The following remarks are taken from our presentation to the Cushing Society in 1957: "During these studies it has been demonstrated that destruction of a majority of the efferent fibers from the lentiform nucleus produces a marked reduction in muscle tone in cases of Parkinson’s disease. It is known that most of these fibers terminate in the region of the red nucleus and in the reticular substance of the midbrain. The role of the rubro- and reticulospinal tracts in the maintenance of muscle tone through their influence on the gamma fibers, is well substantiated."—that is, prior to that presentation of ours.

Dr. Stern and Dr. Ward are to be complimented on their lucid analysis of the relationship between the gamma inhibitory and alpha facilitatory centers.

As further support, it might be mentioned that following localization of V.L fibers and their section, stimulation of the corticospinal tract will give a greater motor response than before the lesion has been made. This is only a working hypothesis; the actual mechanism involved may well be much more complex, as Dr. Schneider has pointed out.

Dr. Stern logically relates tremor initiated in monkeys by lesions of the medial ventral reticular formation to their role in gamma facilitation.

[Film strip] This patient was brought in in decerebrate rigidity 2 years prior to this film. He has a dilated right pupil and has a large-amplitude tremor which, although larger than that seen in most cases of Parkinson’s is somewhat similar to that produced in monkeys. This was relieved totally by a lesion in the vicinity of and immediately below VPL.

Also, it is difficult for me to explain the existence in certain cases of Parkinson’s disease of a profound ataxia with normal tone and, in others, of fairly marked rigidity with satisfactory movements, simply on the assumption that, in one instance gamma inhibition predominates and, in the other, it is caused by alpha facilitation. I think this is probably oversimplification, although the basic mechanism may well be something of the sort.

The paper of Dr. Schneider and Dr. Crosby is a very
patient and most beautiful study of the delicate balance necessary for normal movements. The efficacy of lesions of fibers going to ventralis lateralis and temporary diminution of awareness of muscles following these lesions (the day after a lesion the patient seems not to be very well aware of where he must put his foot or his hand, although sense of position seems to be intact) support their contention of the role played by the cerebellum. The consistently deleterious effects of cerebellar involvement following corticospinal lesions, or in multiple sclerosis when thalamic lesions have been shown to be effective in relieving tremor, suggest that cerebellar ablations of this type, as Dr. Schneider has pointed out, probably should be graded very carefully and used only when the patient might be unable to compensate for even a very small loss of reticular substance as is produced in lesions of the basal ganglia. Otherwise, it would seem that the latter, done where the pathways are more concentrated, have a better chance of being effective.

**Dr. Jack Stern:** Doctors Shelden and Pudenz brought out the height of gamma hyperactivity based on passive flexion of the limbs, and showing increased muscular discharge. They postulated that this is the result of failure of the Golgi tendon organ and its effect on motoneurons. It could be argued the other way that this hyperreflexia is because of failure of inhibition of the antagonist stimulation of muscle spindle acting to inhibit.

Rushworth's conclusion that gamma hyperactivity is responsible for rigidity is based on the chemical blockade of the peripheral nerve, and I do not have time to discuss this. We do not believe the spindle is knocked out completely. We feel that perhaps there is a residual decreased spindle afferent input into the spinal cord that allows sufficient background for this activity to occur. We still feel that we have good grounds for this simple, perhaps naive, approach.

**Dr. Richard C. Schneider:** I would like to thank the discussers for their comments. I think their warnings are justified and important, particularly with regard to the cerebellectomy. I should like to have shown you the rest of the movie on the boy. It is amazing. Immediately after the initial operation on this youngster, the boy still had quite a number of abnormal movements, quite a bit of spasm. Really, he obtained his stabilization, as much as is shown here, only about 3 years after the initial operation.

One other point I neglected to mention is extremely important, and that is, the judicious sparing of certain extrapyramidal areas, the location of which we showed on the diagram. Since the paths from these areas in part pass through the external capsule or discharge directly to the midbrain and so by-pass the internal capsule, a fair amount of control of movements may be left even though there is a lesion in the internal capsule itself.