Destruction of the "Pyramidal Tract" in Man*

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Opportunities to observe the results of the isolated destruction of the "pyramidal tract" in man are rare. There are but few places in the nervous system where the corticospinal fibers are separated sufficiently from other systems to permit of their isolated destruction, either surgically or by disease. Exirpation of the pre- and postcentral cerebral cortex results in a destruction of the "pyramidal tract" but also destroys other fibers, both ascending and descending, thus producing a complex picture. Lesions of the internal capsule frequently destroy the corticospinal system but they also destroy descending pathways to the basal ganglia, the thalamus, the brain stem, the cerebellum, etc., as well as many ascending fibers. Lesions in the spinal cord, likewise, cannot be confined to the corticospinal fibers. Only in the pyramids of the medulla oblongata can truly isolated lesions of the "pyramidal tract" be made. Although division of the medullary pyramids has been made in animals (cat and monkey),26,27 it has not been carried out in man and at the moment no occasion to do so seems likely to arise. In the cerebral peduncle the corticospinal fibers are found segregated from all ascending fibers24 and from all descending pathways except those passing from the cerebral cortex to the brain stem and cerebellum. The corticospinal fibers from the precentral cortex (the classical "pyramidal tract") occupy the central portion of the cerebral peduncle (Fig. 1) as Levin15,19 has shown, while the corticospinal fibers from the postcentral region lie immediately lateral to these (Fig. 2).1 In the most medial part of the peduncle are found the frontopontine fibers.18,19 The exact nature of the fibers in the most lateral part of the cerebral peduncle is less well known. Marin et al.,20 from a study of human material concluded that the lateral segment of the peduncle is composed of corticopontine fibers from the parietal, occipital and temporal lobes. However, their evidence as to occipitopontine fibers is not conclusive, and that dealing with a temporopontine component indicates only a very few such fibers. In fact, in their 2 cases of isolated lesions of the temporal lobe they stated that in 1 (Case 3) there was "no clear-cut evidence of degenerating fibers in the basis pedunculi," and in the other (Case 4) in which the anterior part of the temporal lobe was amputated surgically a "band of less dense glosis (i.e., less dense than in the white matter at a higher level) with no associated pallor of myelin occupies the most lateral part of the lateral segment" (of the peduncle). On the other hand, in those cases in which the parietal cortex was involved (in addition to the temporal and occipital cortices) the degeneration in the lateral segment of the peduncle was intense. Their material provided no evidence on the question of corticospinal fibers from the parietal area.

In recent years several surgeons have divided various parts of the human cerebral peduncle, usually for the relief of abnormal involuntary movements.3,4,8,11,21—23,25,29,30—32,34 To our knowledge no one has had the opportunity previously to study the clinical results of such a lesion in man and then to examine

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Presented with motion pictures before a special meeting of the Dutch Neurological Society in Utrecht on October 4, 1962, a joint meeting of the Czechoslovakian Neurological Society and the Neurosurgical Section of the Czechoslovakian Surgical Society in Prague on June 17, 1963 and at the Medical School of the Free University of Berlin on June 24, 1963.
the location and extent of the surgical lesion and of the resulting degeneration in the human nervous system. The present report concerns such an instance.

In 1961 Bucy and Keplinger found the preliminary report of the case of a man in whom the central portion of the cerebral peduncle was divided in order to relieve a severe hemiballismus. Clinically the operation was completely successful. The abnormal movements were abolished and never returned. The left extremities which were completely disabled prior to the operation were returned to usefulness with almost normal volitional control. The operation was designed to divide the corticospinal fibers from the precentral region, or the classical "pyramidal tract." As this study will show, it was highly successful in that regard also.

Walker and Guiot and Pecker have demonstrated that destruction of part of the cerebral peduncle could relieve permanently the abnormal involuntary movements of hemiballismus and of parkinsonism. Experience had led one of us to believe that the surgical lesion to be effective must include the central portion of the cerebral peduncle. This belief rested partly on the fact that Meyers showed that the medial sector of the peduncle did not relieve such movements, and that Bucy demonstrated that section of the lateral sector was likewise ineffective. Meyers had reached a similar conclusion.

Levin (Fig. 1) and Barnard and Woolsey (Fig. 2) showed that this central portion of the cerebral peduncle contains the corticospinal fibers arising from the precentral re-

![Diagram of cerebral peduncle](image_url)
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...region, i.e., the classical “pyramidal tract.” It is accordingly our opinion that the abolition of abnormal involuntary movements seen in this case, and in other similar cases, resulted from the destruction of these corticospinal fibers of precentral origin—the “pyramidal tract.”

Barnard and Woolsey also showed that although there is considerable overlap in the disposition in the cerebral peduncle of the corticospinal fibers arising from the “face,” “arm” and “leg” areas of the precentral region, those from the “face” area tend to lie medially, those from the “leg” area more laterally and those from the “arm” area occupy an intermediate position in this central segment of the cerebral peduncle (Fig. 2).

For an understanding of the problem under discussion here it is important that the reader bear in mind the fact that in addition to the corticospinal fibers of precentral origin there are in the cerebral peduncle other fibers. These include the corticospinal fibers which arise in the parietal cortex and fibers from the cerebral cortex to the mesencephalon, to the bulbar nuclei and fibers of cortical origin which conduct impulses to the cerebellum via the pontine nuclei. In addition, Barnard and Woolsey and Hines have shown that at least on occasion there exist in the monkey fibers connecting the cerebral cortex and the cerebellum directly.

In the case reported here, as the section of the peduncle was just above the pons, the fibers to the mesencephalon were not greatly affected. Thus only those fibers to the bulbar and pontine nuclei and to the cerebellum, in addition to the corticospinal fibers, could possibly have been concerned with the relief of the abnormal movements of hemiballismus in this case.

Although both the patient and we were gratified with the permanent relief from the violent involuntary movements which was obtained, that is not our principal concern here. In this report we shall devote our attention to the effect on normal muscular activity of the destruction of the corticospinal fibers—the “pyramidal tract”—on one side in man. We shall point out that discrete, well-controlled and well-coordinated useful activity is still possible in the hand and fingers, the foot and toes, as well as in the rest of the left extremities in the absence of the related “pyramidal tract,” and that so-called “pyramidal syndrome” (spastic paralysis with hyperactive tendon reflexes and the sign of Babinski) develops only incompletely and to a mild degree.

It has been said that one of us (Bucy) is trying to deny the existence of the “pyramidal tract.” Walshe has so misread previous publications as to say that Bucy asked the question, “Is there a pyramidal tract?” and answered it “in a negative sense.” This is, of course, a gross distortion of what was written. Bucy said “there is no such single entity as the ‘Pyramidal Tract,’” as the corticospinal fibers which occupy the medullary pyramids do not all have a common origin, a common termination or a common function. Bucy has also said of the “pyramidal syndrome”:

“The symptom complex of a spastic paralysis without atrophy, with hyperactive tendon reflexes, absent abdominal reflexes and the sign of Babinski simply . . . does not result from interruption of those corticospinal fibers which arise from the precentral gyrus and pass downward through the cerebral peduncle.”

At the time that these earlier remarks were made in 1956 and 1957 we knew less about the corticospinal fibers than we know today. Since then Russell and DeMyer in a study of the corticospinal system in the monkey have shown that “all or virtually all descending pyramidal axons arise in area 6, area 4 and the parietal lobe . . . approximately 50% . . . in area 6, 31% in area 4, and 40% from the parietal lobe.” In addition, Kuypers has shown, in the monkey, that many of the corticospinal fibers from the precentral gyrus terminate on the anterior horn cells themselves rather than on interneural neurons in the spinal grey matter as in some other animals, such as the cat. He has also shown that in both man and monkey descending neurons arising in the central cortical areas terminate in various sensory nuclei—the spinal root of the 5th cranial nerve, the nuclei gracilis and cuneatus and
the posterior grey horn of the spinal cord. It is not unlikely that these descending corticobulbar and corticospinal neurons which end in sensory nuclei are concerned with modulating incoming sensory impulses, with the control of sensory threshold, with the sensitization of attention. In spite of these possible sensory functions of the corticospinal and corticobulbar fibers, it is still our opinion that the corticospinal fibers are probably the most important neural mechanism having to do with muscular movement—but they are not the only such mechanism. Good useful movement is possible without the "pyramidal tract." Furthermore, it is still our opinion that destruction of the corticospinal system does not give rise to the so-called "pyramidal syndrome." We are not trying to deny the existence of the corticospinal tract but merely to understand it so that neurology may progress in an intelligent and scientific fashion. Blind adherence to erroneous concepts can only retard such progress, regardless of what "authority" promulgated them.

Case Report*

Antonio Y. Lopez was a 70-year-old man who had been born in Mexico. He was admitted to the Chicago Wesley Memorial Hospital on Sept. 30, 1958 because of urinary retention. He had a benign hypertrophy of the prostate, diabetes melitus, generalized arteriosclerosis and a blood pressure of 190/90. Shortly after admission a left hemiballismus developed. Various medications resulted in only temporary amelioration of the abnormal involuntary movements. On October 30 a suprapubic prostatectomy was made. Because of the increasing severity of the involuntary movements he was referred to the neurosurgical service for treatment in December.

Examination disclosed severe involuntary movements of the left side of the face and of his left arm and leg. These movements involved all parts of the extremities. They were present constantly while the patient was awake. They were aggravated by voluntary effort. He could not execute sustained or useful movements with the left extremities. Neither could he stand or walk. He could sit on a chair, but when doing so he would attempt to restrain the left extremities by holding the left hand or wrist with the right hand, and by hooking the left foot back of the right ankle. The tendon reflexes on the left side could not be evaluated because of the severe continuous involuntary movements. The tendon reflexes on the right side were normal. The abdominal reflexes were all absent and Babinski's sign was not elicited. Various forms of sensation (tactile, painful and vibratory) were intact everywhere except that sense of position could not be tested satisfactorily in the left extremities because of the abnormal movements.

Operation. On Dec. 19, 1958, about 2½ months after the onset of the hemiballismus, the central portion of the right cerebral peduncle was divided (Fig. 3). The incision measured 10 mm. on the surface of the peduncle and was 7 mm. deep. (The average normal cerebral peduncle in an adult brain has a width of about 17 mm. and a thickness of about 7 mm.) It was our intent to divide the entire corticospinal tract arising from the precentral region. The operation was performed through a vertical linear incision in the right temporal region. The temporal lobe was lifted off the floor of the middle cranial fossa intradurally. The exposure of the peduncle was good. The operation was performed without unusual incident.

Course. The abnormal involuntary hemiballistic movements were abolished completely following the operation and never returned. Immediately after operation he had a complete flaccid left hemiplegia. Within 24 hours slight voluntary movements returned to the paralyzed extremities and there was continuous improvement in strength and coordination thereafter. We were surprised to note that with the initial return of movement he was able to grasp with his left hand and could move his left toes, as with the ordinary capsular or cortical hemiplegia these parts are the last to recover, if they ever do.

On the 3rd postoperative day a weak Babinski's sign was elicited on the left side. Strength and

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* A preliminary report of this case was made by Bucy and Keplinger in 1961 and was presented with motion pictures to a meeting of the American Neurological Association on June 13, 1960.
range of voluntary movement returned more rapidly in the hand and fingers, and in the foot and toes than at the more proximal joints. Recovery was also slightly more rapid in the lower than in the upper extremity. On the 10th postoperative day he was able to stand alone and to walk with assistance. He could lift his left arm above his head. Strength in his leg was good but the movements were performed in an awkward and unsteady manner. There was no increase in resistance to passive manipulation. In fact, muscular tone on the left side was slightly less than on the right. There was no appreciable difference in the tendon reflexes on the two sides. By the 24th postoperative day the strength and control of his movements were further improved. He could walk with the aid of a “walker” and could get in and out of a chair unassisted. There was a slight increase in tone in the left leg and the tendon reflexes were a little more active in the left extremities. Babinski’s sign was easily elicited on the left side. By the 29th postoperative day he could walk unaided for short distances, although he dragged his left foot a little. When he was discharged from the hospital on Jan. 20, 1959—32 days after the operation—he had no abnormal involuntary movements. He had fairly good use of his left hand and upper extremity. He could execute fine movements of individual fingers fairly well. Voluntary movements of his left leg were quite good, and he could move his left foot and toes well. He could walk unaided but favored his left leg a little as he did so. The tendon reflexes were moderately hyperactive in the left extremities, but resistance to passive manipulation was increased little if at all. Babinski’s sign was present. There was a moderate left lower facial weakness. Various forms of sensibility (light touch, pain, vibration, sense of position and localization of points stimulated) were intact everywhere.

Thereafter he was seen frequently both as an out-patient and in the hospital. Treatment in the hospital for his diabetes and for increasing cardiac disturbances was required from time to time. The abnormal involuntary movements never returned. For some time he increased steadily in strength and in a sense of well-being. Within 7 months after the operation the maximum recovery seemed to have been reached. When he was examined on Aug. 4, 1959 he was mentally alert. There were no involuntary movements. The cranial nerves were intact. He was blind in his left eye because of an injury early in life. The left pupil was small and fixed. There was no longer any facial weakness. Sensation in various forms was intact everywhere. He had a very mild left hemiparesis. The grasp of his left hand was slightly less than that of the right (he was right-handed). Fine, individual movements of the left fingers were only slightly less well executed than on the right side. He could move his left toes but not quite as extensively as the right ones. There was no increase in resistance to passive manipulation of the left extremities as compared to the normal right ones. The tendon reflexes were a little more active in the left extremities. The abdominal reflexes were all absent, as before operation. Babinski’s sign was present on the left. He favored his left leg slightly as he walked. He could hop well on either foot alone, but did so a little better on the right foot than on the left.

Thereafter the neurological picture remained stationary, but increasing evidence of cardiac failure and of malignancy developed. He died on July 3, 1961 at the age of 78 years, 2½ years after section of the central portion of the right cerebral peduncle. He had very thoughtfully arranged with his family for a complete postmortem examination.

Autopsy Findings. Apart from the nervous system there was an acute bronchopneumonia; rheumatic heart disease; lymphosarcoma with involvement of mediastinal and retroperitoneal lymph nodes, parietal pleura, peritoneum, liver, pancreas and left adrenal gland; papillary carcinoma of the right lobe of the thyroid gland with metastases to the cervical lymph nodes; and a carcinoma of the right kidney.

The brain and spinal cord were fixed in 10 per cent formalin. There were some atheromatous plaques involving the vessels of the circle of Willis. The brain appeared normal externally, except for some meningeal thickening over the right cerebral peduncle. The actual incision in the peduncle was not clearly visible from the surface. The right half of the pons was obviously shrunken and its surface was flattened. The right medullary pyramid was definitely smaller than the left.

The midbrain, including the subthalamic nuclei, was sectioned serially, as was the right central cerebral cortical area. Representative sections were made from the left central cortical area, the basal ganglia, and the thalami, the pons, medulla oblongata, the cerebellum and the spinal cord. Sections were stained with Nissl’s method, Weil’s, Kultschitsky’s and Kliter and Barrera’s method for myelin sheaths, and DeMyer’s7 method for axis cylinders.

There were no grossly infarcted areas and no neoplastic lesions in any part of the brain. Neither were any microscopic areas of infarction seen. The entire subthalamic nuclei were studied in serial sections. Sections at intervals of 0.1 mm. were stained with thionine. No abnormality was seen. There was neither neuronal loss nor degeneration. The subthalamic nuclei on the two sides did not appear different in size or in cellular content. Microscopic examination of the caudate nuclei and of the lenticular nuclei was unrevealing.
Fig. 4. The lesion actually produced in the right cerebral peduncle. (Weil's stain for myelin sheaths)

There was a distinct segmental loss of cells in Sommer's sector of the hippocampal formation. This loss was moderate and was bilateral, but was more marked on the right side. We believe that this loss has no relationship to the patient's motor disease, or to the operation. It is most likely related to his age, or to the episodes of hypoxia resulting from his cardiac failure. The dorsal thalami, caudate nuclei and lenticular nuclei disclosed no pathological changes on microscopic examination.

The surgical lesion involved the middle three-fifths of the right cerebral peduncle at its caudal extremity just above the pons (Fig. 4). It extended deeper medially than laterally. The cut entered the central segment of the peduncle but divided only the ventral fibers laterally. As it extended deeper into the peduncle it passed medially, sparing the most medial and ventral fibers of the medial segment. The largest bundle of spared fibers lay laterally. There was a very slight involvement of the medial portion of the substantia nigra (Fig. 5). The most anterior part of the medial pontine nuclei was also involved to a very slight degree. The lesion itself was characterized by a loss of nerve fibers, both axis cylinders and myelin sheaths, and by the existence of microcysts, macrophages and reactive astrocytes. Descending and retrograde degeneration of nerve fibers could be seen below and immediately above the lesion respectively.

Descending Degeneration. The descending degeneration was massive. It was limited to the right side in the brain stem and involved the corticospinal fibers. In the pons almost all of the descending fibers were destroyed (Fig. 6). The majority of the spared fibers lay dorsolaterally. Most of these remaining fibers probably represent frontal and parietal corticopontine fibers and corticospinal fibers of parietal origin which were left intact in the medial and lateral parts of the cerebral peduncle. The transverse pontine fibers appeared to be intact. The pontine nuclei situated among the degenerated descending tracts showed no evidence of neuronal loss. The degenerated

Fig. 5. Higher magnification of the lesion in the right cerebral peduncle, showing the very slight extension of the lesion into the region of the substantia nigra in the upper left-hand corner. (Weil's method)
areas were characterized by a loss of axons as well as by a reactive gliosis. There were also numerous macrophages in the perivascular areas. These were laden with debris. There was a striking decrease in the size of the area normally occupied by the corticospinal tract. An even more striking shrinkage of the right medullary pyramid was apparent (Fig. 7). There was a marked demyelination of the pyramid but a few scattered myelinated fibers still persisted (Fig. 8). The sections of the medullary pyramid stained by DeMyer’s method for axis cylinders showed a marked condensation of the supporting tissues on the right side but the number of axis cylinders was greatly reduced (Fig. 9). Dr. William DeMyer of Indiana University, who has had an extensive experience in counting the fibers in degenerated medullary pyramids, very kindly examined these specimens. By actual count Dr. DeMyer found that 88 per cent of the axis cylinders in the right medullary pyramid have disappeared.

In the spinal cord the degenerated uncrossed ventral corticospinal tract can be followed down to the mid-thoracic region. The degenerated crossed corticospinal tract can be followed down to the conus medullaris. The spinal cord, otherwise, appeared normal. Specifically no evidence of change, transneuronal or otherwise, could be detected in the anterior horn cells.

Retrograde Degeneration. Above the lesion there is a decrease in the number of fibers in the right internal capsule, but it is difficult to make a quantitative estimate of the axonal loss. The reactive gliosis is slight.

Central Cortex. Serial sections were made of the right pre- and postcentral cortex, and random sections of the left. Comparing the two central areas there was an estimated loss of at least 90 per cent of the gigantic cells of Betz in the precentral cortex on the right side. There was no apparent decrease in the number of other pyramidal cells but no detailed cellular counts were made. It is presumed that this cellular loss was caused by retrograde degeneration resulting from section of the right cerebral peduncle, and that it was complete at the time of the patient’s death.

Discussion

The studies of the brain of this man confirm the surgical observations. The central portion of the right cerebral peduncle was divided and almost all of the corticospinal tract—both from the precentral and postcentral cortices—was degenerated. Those corticospinal fibers which remained (17 per cent of the total) were either the fibers of precentral origin which are related to the lower extremity as they lie more laterally than the fibers to the arm or, more likely, corticospinal fibers of parietal origin. In this connection it is significant that Russell and DeMyer found that 40 per cent of the corticospinal fibers in the macaque monkey arise from the parietal lobe. Barnard and Woolsey have shown that these fibers lie lateral to the corticospinal fibers of precentral origin in the cerebral peduncle (Fig. 2). Further confirmation of this opinion that the corticospinal

Fig. 7. Section through the medulla oblongata, showing the almost complete degeneration of the right pyramid. Note the great reduction in size of the right pyramid as compared with the left. (Well’s method)

Fig. 6. Section through the pons showing the extensive degeneration of the corticospinal fibers on the right side. Some descending fibers—corticospinal, corticopontine and corticobulbar—are seen in the upper lateral part of the pons. This is the area where descending fibers of parietal origin are to be found. (Kulchitsky’s method for myelin sheaths)
Fig. 8. Higher magnification of comparable portions of the right and left medullary pyramids. (Weil's method) (Above) Left pyramid. All of the myelin sheaths are intact. (Below) Right pyramid. A scattered few myelinated fibers remain intact.
Fig. 9. Comparable areas of the right and left pyramids impregnated by DeMyer's method for axis cylinders. The interstitial supporting tissues are also shown by this method. (Above) Left pyramid. It is normal and the impregnated axis cylinders can be seen scattered diffusely throughout the pyramid. (Below) Right pyramid. The interstitial supporting tissues are markedly condensed, as compared with the normal left pyramid. This is the result of the degeneration of almost all of the nerve fibers in the pyramid. A few remaining axis cylinders can be seen here and there.
fibers which remain intact in this case are of parietal origin is offered by the fact that the intact corticospinal fibers occupy a dorsolateral position in the pons (Fig. 6) and this is exactly the position occupied by the corticospinal fibers of postcentral origin in the monkey as shown by Barnard and Woolsey¹ (see their Fig. 5a). (In the medullary pyramid, according to these authors, the corticospinal fibers of parietal origin are scattered throughout the pyramid but they show a greater concentration in the more lateral parts of the bundle—see their Fig. 6b.) The intact fibers lying in the lateral part of the right cerebral peduncle must have contained corticopontine fibers of temporal or parietal origin in addition to these corticospinal fibers of parietal origin.¹,¹⁶ The intact fibers in the medial portion of the cerebral peduncle must have been only frontopontine fibers.

To state the anatomical situation differently, the corticospinal fibers from the precentral region appear to have been completely destroyed, while something less than half of the corticospinal fibers from the postcentral region remained intact (17 per cent of the corticospinal fibers left intact, as compared with 40 per cent which arise from the parietal cortex). In addition, many of the fibers from the frontal lobe to the pons, and all of the corticopontine fibers from the temporal or the parietal lobe, remained intact. It should also be noted that most or all of the fibers from the precentral region to the pontine nuclei must have been destroyed. These fibers serve the important function of conducting impulses from the precentral motor cortex to the cerebellum. It is believed that they are concerned in the mechanism of cerebellar coordination. The corticobulbar fibers to the nuclei of various cranial nerves must also have been largely destroyed in the right peduncle.

In view of this almost complete destruction of the corticospinal fibers from the right central cerebral cortex this man's clinical neurological status is truly remarkable. It is not surprising that he had some weakness and impairment of usefulness of his left extremities, but it is surprising that he had so little disturbance and that that which he had did not follow the pattern that we had come to expect from destruction of the "pyramidal tract." As we pointed out earlier, this is not the only patient who has had a surgical lesion made in his cerebral peduncle and abnormal involuntary movements relieved without the production of a spastic paralysis. There have been a number of similar cases in which the operations have been performed by us and by other neurological surgeons. This case is unique only in that it is the only one in which there has been a pathological study carried out following the operation.

For a long time it has been believed that destruction of the "pyramidal tract" will result in a paralysis of the opposite side of the body, including the face, arm and leg, as well as the trunk. It has been known that removal of the cerebral cortex from which the corticospinal fibers arise does not produce a complete paralysis.² Some movement remains, notably in the upper part of the face and in the proximal parts of the extremities, the shoulder and elbow, and the hip and knee. It has also been recognized that this persisting movement is in general more extensive and more useful in the lower than in the upper extremity. However, the distal parts of the extremities, the wrist, hand and fingers, the ankle, foot and toes have usually been completely paralyzed or nearly so. This paralysis is such as to render the upper extremity completely useless, while most patients with extensive central cerebral cortical lesions or lesions in the internal capsule become able to use the lower extremity in walking. Their gait is hemiplegic, and the spastic extremity is advanced by circumduction.² Most observers attribute this very limited movement which persists to innervation from the ipsilateral cerebral cortex.⁵ Others believe the basal ganglia or both the basal ganglia and the ipsilateral cortex to be responsible. It has also been generally believed that the severe motor loss which these people suffer after a cortical or a capsular lesion, or after decortication of a cerebral hemisphere, is the result of destruction of the "pyramidal tract." It is now obvious that this latter opinion must be
revised. As is shown by the case recorded here, the section of the corticospinal tract in the cerebral peduncle produces a loss which is far less severe than that resulting from extensive lesions of the cerebral cortex or of the internal capsule, and, furthermore, the loss is not greater in the distal parts of the extremity. This man had as good movement of his hand and fingers, foot and toes, as he did at his more proximal joints. There can thus be no doubt but that the loss produced by extensive resection of the precentral cortex in man results from the destruction of some descending fiber system from the central cortex in addition to the corticospinal fibers. As there are no other single-fiber systems from the cortex to the spinal cord this must be a multineuronal pathway and it must pass through some part of the midbrain other than the cerebral peduncle.

The movement which remained in this case was doubtless, in part, to be attributed to innervation from the ipsilateral (left) cerebral hemisphere. But a comparison of this man with any patient with a capsular hemiplegia, or an excision of the precentral motor cortex, or a hemidecortication leaves no doubt but that he was receiving far more cerebral innervation to his left extremities than could be accounted for by the ipsilateral cerebral hemisphere. What then produced this remarkably fine, detailed, well-coordinated, useful movement of all parts of his left upper and lower extremities? Whatever descending innervation was responsible must of necessity have passed downward through his midbrain, either through the cerebral peduncle or the mesencephalic tegmentum. Naturally it must be asked, "Could this movement have been produced by those 17 per cent of the corticospinal fibers that remained intact?" As was pointed out above, these fibers must have been largely or exclusively corticospinal fibers of parietal origin. Travis and Woolsey have expressed the opinion that the parietal cortex is capable of producing some useful movement, but the amount, as compared with that produced by the precentral cortex, is little. In this same connection it should be noted that in Walker's cases in which the cerebral peduncle was incised and extensive, useful movements of the contralateral extremities persisted, the section was believed to have included the lateral as well as the central portion of the peduncle and thus should have destroyed corticospinal fibers both from the precentral and the postcentral cortices.

It is also possible that a few corticospinal fibers of precentral origin may have survived and been included in that 17 per cent of intact fibers. In support of that contention is the fact that a few gigantic cells of Betz survived in the right precentral gyrus. However, the number of such fibers must have been very small indeed. If any such survived all of them must have been concerned with the activity of the left lower extremity as they lay in the lateral part of the peduncle. If such corticospinal fibers from the precentral region were responsible for the excellent motor control of the left side which this man retained, the motor activity should then have been much better in the leg than in the arm. Such was not the case.

It appears far more likely that, although some of the movement retained should be attributed to ipsilateral innervation and perhaps to the remaining corticospinal fibers, most of the movement of this man's left extremities was produced by multineuronal pathways descending through the mesencephalic tegmentum. Whether this multineuronal path is a single pathway with a common origin and termination and a common course for all of its fibers; where it lies in the mesencephalic tegmentum; what the subcortical nuclear structures (thalamus, basal ganglia, reticular formation, other brainstem nuclei, the cerebellum) are with which this pathway is involved, are all unknown at this time.

In the past it has also been believed that destruction of the pyramidal tract gives rise to spasticity and increased reflexes. During the past quarter of a century evidence has been accumulating steadily, showing that this is not the case. The control of postural and stretch reflexes is exercised by a different neural system extending from the cerebral
cortex, through subcortical nuclei, particularly the reticular formation of the brain stem, to the gamma system of the spinal cord. This mechanism is well known and generally accepted. It need not be discussed further here except to note that the findings in this case are in complete accord with the modern understanding of this reflex mechanism. This man had his "pyramidal tract" (the corticospinal fibers from the precentral region) destroyed. He did not have any increased resistance to passive manipulation of his extremities. There was no clasp-knife phenomenon, no lengthening or shortening reaction. It is true that he had some increase of the tendon reflexes in his left extremities as compared with the right. But this was hardly enough to enable one to describe his condition as a spastic paralysis as he had little spasticity or paralysis. The cause of the moderately increased tendon reflexes is conjectural. It was impossible, because of the abnormal involuntary movements, to evaluate these reflexes before the operation. Were they increased then, or was the moderate hyperreflexia found subsequently the result of the operation? In this connection it is to be noted that Tower\(^{37}\) noted an increase in the tendon reflexes following division of the medullary pyramids in the monkey. Perhaps this means that these reflexes are subject to control by a multiplicity of neural mechanisms, and that they may not be under the exclusive control of the reticular formation. Certainly the observations in this case are not conclusive on this point.

It has long been believed that the sign of Babinski can be elicited when the "pyramidal tract," the corticospinal fibers from the "foot" area of the precentral gyrus, has been destroyed. Our observations support this belief. It is, of course, obvious that this reflex will develop only when the corticospinal fibers specifically concerned with the musculature of the great toe are involved. It is not to be expected that involvement of just any fibers of the "pyramidal tract" would give rise to this reflex. Furthermore, the reflex can be elicited only if the peripheral neuromuscular mechanism and the skeletal components of the foot are intact and functioning properly.

Lastly, it should be noted that this case supports the demonstration of Walker\(^{20}\) that the abnormal involuntary movements of hemiballismus can be abolished permanently by sectioning the cerebral peduncle without producing a seriously disabling paralysis. However, that point is not the real subject of this paper and requires no further comment here.

**Animal Investigations**

Obviously opportunities to make observations on man similar to those recorded here are rare indeed. We are, therefore, attempting to extend our investigations by studying similar and more extensive lesions in the cerebral peduncles of monkeys. These are not yet completed.

In our preliminary report in 1961\(^{6}\) we described a monkey in which the central portions of both cerebral peduncles had been divided. This animal had good control over all four extremities. He could walk, run, and climb the walls of his cage easily. He could pick up small articles with either hand. His movements were a little slower than those of a normal animal and he appeared to tire more quickly.

Since then we have divided the cerebral peduncles in many monkeys on one or both sides. These operations have been more extensive than those reported previously and have produced a complete degeneration of the medullary pyramids bilaterally. These animals, too, are remarkably agile but have somewhat greater deficits than the animal (No. 54) previously reported upon. Because of the long periods required for recovery, rehabilitation and testing after and between operations, and the long period required for complete degeneration of the severed axis cylinders\(^{24}\) these experiments are not yet ready for detailed report.

**Summary**

The case is reported of a 70-year-old man in whom a severe left hemiballismus developed. The abnormal involuntary movements
Destruction of “Pyramidal Tract” in Man

continued and increased in severity for 2½ months in spite of medical treatment. They were then permanently abolished by surgical division of the central half of the right cerebral peduncle.

Following the operation he had a complete left hemiplegia. Within a few days this began to recover and continued to do so for the next 7 or 8 months. When the condition became stationary the patient had no facial weakness and the movements of his left extremities were strong, useful and well-coordinated. These movements included fine movements of his hand and individual digits, and good movements of his foot and toes. There was no spasticity in the sense of resistance to passive manipulation. There was a moderate increase in the tendon reflexes in his left extremities. Babinski’s sign appeared soon after the operation and persisted thereafter.

Detailed microscopic study revealed a destruction of the central portion of the right cerebral peduncle, extending somewhat more medially than laterally. Almost all of the gigantic cells of Betz of the right precentral gyrus had disappeared. Most of the fibers in the medullary pyramid were completely degenerated. The degenerated fibers extended all the way down the spinal cord to the conus medullaris. Only 17 per cent of the corticospinal fibers remained intact. It is believed that these remaining corticospinal fibers were of parietal origin. Even so, at least half of the corticospinal fibers of parietal origin were also destroyed. Some corticopontine and corticobulbar fibers were also destroyed, but many were preserved.

On the basis of these observations the following conclusions are drawn:

1) The “pyramidal tract” (the corticospinal fibers arising from the precentral region) is not essential to useful control of the skeletal musculature. Nevertheless, it is believed that the corticospinal system is important in the control of the skeletal musculature.

2) In the absence of the corticospinal fibers other fiber systems, particularly some as yet undefined multineuronal mechanism passing downward through the mesencephalic tegmentum, are capable of producing useful, well-coordinated, strong and delicate movements of the extremities. Innervation from the ipsilateral cerebral hemisphere plays a minor role in the control of this musculature. And the partially preserved corticospinal fibers of parietal origin may have played a small role in this case.

3) Destruction of the corticospinal fibers does not produce a motor deficit which is greater in the distal parts of the extremities (hand and fingers, foot and toes).

4) Destruction of the corticospinal system does not result in an increase in resistance to passive manipulation, the clasp-knife phenomenon, or the lengthening or shortening reactions; to wit, it does not cause spasticity. It may be associated with some increase in the tendon reflexes.

5) Babinski’s sign can be elicited following the destruction of that part of the corticospinal tract concerned with the innervation of the foot, providing the peripheral neuroskeletomuscular mechanism is intact.

6) Section of the central portion of the cerebral peduncle will permanently abolish hemiballismus in the contralateral half of the body.

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