THE SURGICAL TREATMENT
OF
ABNORMAL INVOLUNTARY MOVEMENTS*

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INTRODUCTION

In the past twenty-five years it has been my pleasure to work with many young Japanese surgeons who have visited our clinics and laboratories in Chicago. Naturally I have been delighted to note the progress which each one of these has made after his return home to Japan. Now I find them occupying posts of great importance and responsibility in your hospitals and medical schools. Among these men are Araki, Tanaka, Shimizu, and most recently Handa and Iwata. Of fond memory is Hayashi, Professor of Anatomy in Tokyo, who is no longer with us. Having these and many other friends in Japan that I have made over the years, I was most happy to be able to accept the kind invitation of your President, Dr. Asano, to attend and to address this meeting. It is an honor and a great pleasure to be here. Your hospitality has been overwhelming. Your country is most beautiful, interesting and exciting. With your help I shall make every effort in the short time at my disposal to learn all I can about Japan. My sincere regret is that I cannot speak to you in your own language. However, I am sure that with the aid of your gracious interpreters we shall be able to communicate freely with each other. Again let me thank you for having invited me to meet with you.

PROBLEM

During the almost thirty-five years since I first entered the field of Neurology and Neurological Surgery as a graduate student in Neuropathology in 1924, I have been interested in many things. One of the most important of these has been the neural mechanism responsible for the control and production of normal motor activities and the development of abnormal involuntary movements. It is this latter problem which I wish to discuss with you today. This is an important problem, not only because there are so many people afflicted with these motor disorders but also because these uncontrollable disconcerting movements of any

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or all parts of the body often are so constantly present throughout the patient's every waking hour and have proved so resistant to medical management and so reluctant to respond favorably to surgical interference. It is characteristic of man that he takes great pleasure in his ability to control his own body with great precision. He is proud of his ability to run, to jump, to swim well, to play various musical instruments, to execute beautiful works of art, and amongst ourselves to perform intricate types of surgical operations. It is most distressing to any man not only to lose the ability to perform delicate, well coordinated movements, but to have his body develop grotesque movements which it makes spontaneously over and over again, hour after hour, day after day, and year after year, and which he is unable to stop or control. It is not surprising that the victims of these disorders submit willingly to so many different types of operations almost without regard to the risk involved or the likelihood of improvement. Under these circumstances our responsibilities as physicians are all the greater. With our greater knowledge, wider experience and opportunity for impersonal detachment and objectivity we must protect these patients, take every precaution to avoid unnecessary hazards, and perform only those operations which offer a reasonable hope of improvement. There is another even greater obligation to our patients and to the world at large. The opportunities which these patients offer us to study and investigate these disorders which are peculiar to man are precious. They must not be wasted. Every precaution must be taken to study every case as completely as possible, and with all the controls, anatomical, physiological and clinical, that we can devise. It is unfortunately true that for the most part these disorders cannot at this time be reproduced in experimental animals. They can only be studied in man. Our ignorance regarding them remains enormous. We must still learn the nature and location of the lesions responsible for the release of this abnormal motor activity and the other abnormalities associated with it. This is a complex multiform problem. First we are dealing with various types of abnormal movement. It would appear almost axiomatic that the lesion responsible for the development of the tremor-at-rest so typical of parkinsonism must be different from the lesions which are responsible for the wild flinging movements of hemiballismus, or the violent intention tremor and "wing beating" movements seen in Wilson's hepato-lenticular degeneration, or the jerky restlessness and grimacing of Sydenham's chorea, or the sinuous twisting movements of the athetoid child, or the annoying stereotyped movements of spasmodic torticollis. Although we have some information regarding these important pathological facts, no one would contend that this information is either complete or conclusive. Furthermore, it seems equally obvious that each disorder may well be associated with a multiplicity of lesions. This is particularly apparent with parkinsonism. The manifestations of that condition must be separated into two large categories, the negative and the positive ones. The negative manifestations consist of those functions which are abolished by the disease. In parkinsonism these include
the poverty of movement, the loss of the movements of emotional expression and the loss of associated or automatic movements. Thus we note the expressionless or "mask-like" face, the general immobility of the patient, the failure to swing his arms as he walks, the stooped posture, etc. These are muscular activities which are gone; they are paralytic phenomena and may well result directly from the destructive lesions which give rise to the disease. The positive manifestations on the other hand are not paralytic. On the contrary, they result from definite muscular contraction and neurological activity. They include the characteristic tremor, the rigidity and the oculo-gyric crises. It is obvious at once that these cannot be produced by structures which are destroyed but can only result from the activity of structures which are still present and actively functioning. Here it appears that we are probably dealing with two or more structures, one which has been destroyed or damaged by the disease and which exercised some controlling influence over a second which by virtue of the disease is now released to abnormal or excessive activity. Furthermore, in view of the fact that some patients have rigidity without tremor and others have tremor with little or no rigidity and that only a few have oculo-gyric crises it must be obvious that different lesions release different mechanisms to give rise to these various manifestations of the disease. And still more complicating, it further appears most likely that each of the different types of abnormal involuntary movements occurring in other forms of dyskinesia is associated with a different lesion and a different mechanism for the production of the movement.

The accumulation of the information essential to answer these problems is largely dependent upon the study of man. It seems most unlikely that we will achieve anything like a solution of this problem until we have a much better understanding of the neural mechanism responsible for the control of normal muscular activity, and most unlikely that we will develop the optimum treatment for these abnormal involuntary movements until we have in hand a far better comprehension of the pathology and pathol-physiology of these disorders than we have at present. Yet because our ignorance is great and these problems difficult of solution we must not despair. Much progress has been made in this past quarter of a century and there have been ample encouraging facts to give us the courage and persistence to continue.

**ABOLITION OF ABNORMAL INVOLUNTARY MOVEMENTS**

The first indication that these annoying movements might be abolished was given by Parkinson in his original paper in 1817 in which he so well described the disease which still bears his name. The patient described in his case 6 suffered from a generalized tremor-at-rest. He developed an apoplectic seizure resulting in a right hemiplegia which lasted for two weeks and then completely disappeared. Parkinson stated, "During the time of their having remained in this state, neither the arm nor the leg of the paralytic side was in the least affected.
with the tremulous agitation; but as their paralyzed state was removed, the shaking returned.” In later years others confirmed this observation (Patrick and Levy). This type of observation was not confined to parkinsonism. Jakob reported the case of a patient with a luetic infection who had a severe athetosis. He developed a hemiplegia which lasted but a very few days and then almost completely disappeared. The athetosis vanished with the onset of the hemiplegia and never recurred during the patient’s remaining eighteen months of life. Here was the first evidence that a suitably placed lesion in the central nervous system could abolish abnormal involuntary movements without producing a permanent paralysis.

Attempts to accomplish this same result surgically had of necessity to await the development of the modern era of neurological surgery. This was first undertaken by Sir Victor Horsley, the great English pioneer, and reported by him in 1909. He demonstrated that the abnormal involuntary movements of athetosis could be abolished by extirpation of the precentral gyrus. However, this observation excited little interest, except for a very few isolated attempts by continental surgeons (Anschutz, 1910; Payr, 1921; Nazaroff, 1927; Polenow, 1929) to duplicate what Horsley had done. Somewhat later Kinnier Wilson (1929) postulated that the cortico-spinal pathway from the precentral gyrus is probably the central pathway which conducts the impulses which are responsible for the production of the abnormal movements seen in choreo-athetosis. However, these observations were largely ignored until our observations made with Buchanan and published in 1932 re-awakened interest in this field. Subsequent observations by ourselves and others (Bucy; Sachs; Alajouanine, LeBeau and Houdart; DeLisi, Perria and Sacchi; Reid; Verbiest; Chorobski; etc.) have left no doubt of the fact that extirpation of the precentral region, when adequately and accurately done, will completely abolish abnormal involuntary movements. (Fig. 1) Although this observation was originally applied to choreo-athetoid movements it was soon

Fig. 1. Diagramatic sketch of cerebral cortex showing the extirpation of the precentral motor cortex (areas 4 and 6) which will abolish choreo-athetoid movements when the representation of the involved extremities is accurately removed.
shown that a similar operation would abolish tremor (Fig. 2) (Bucy and Case 1939; Bucy 1940; Putnam 1940) and hemiballismus (Bucy 1949, Case 2). Such operations have the very considerable disadvantage that they not only abolished the abnormal movements but also produced a contralateral spastic hemiparesis. In cases of athetosis where a hemiplegia often is already present this is not a great disadvantage but in cases of tremor or hemiballismus where no paralysis exists the production of such an added disability is unacceptable except in the most unusual cases.

Attempts were made early to abolish the abnormal involuntary movements without producing a paralysis. As we had pointed out that it seemed likely that descending motor pathways other than the cortico-spinal tract from the precentral gyrus were responsible for the production of choreo-athetosis Putnam (1933, 1938) attempted to relieve the condition by sectioning the anterior white columns of the spinal cord. (Fig. 3) Unfortunately this procedure was only partially successful. It did not produce paralysis but neither did it result in a complete and enduring abolition of the choreo-athetoid movements. In 1937 we demonstrated for the first time that tremor could be abolished by a surgical procedure upon the human brain (Bucy and Case 1939; Bucy 1949, Case 3). In this and a few subsequent cases the precentral region was excised and both tremor-at-rest and intention tremor were completely or almost completely abolished. Our observations at that time, in contradistinction to those on choreo-athetosis, led us to conclude that tremor disappears when the cortico-spinal pathway from the precentral gyrus is destroyed. This observation was promptly communicated to Putnam. He was thus stimulated to section the lateral cortico-spinal tract in the spinal cord. (Fig. 4) Earlier he had demonstrated that section of the anterior
white column of the spinal cord did not affect the tremor of parkinsonism although it reduced the movements of athetosis (Putnam, 1938). Foerster and Gagel (1932) in Germany had made a somewhat similar observation as regards tremor. However, attempts to abolish tremor by sectioning the lateral cortico-spinal tracts in the spinal cord left much to be desired. Although the paralysis which was produced was not always as severe as was expected, the effect upon the tremor was incomplete and often temporary. Eben (1949) and Oliver (1949, 1950, 1953) endeavored to improve the results by more extensive section of the spinal cord, but the results still were more or less unsatisfactory.

Meanwhile others by different means were seeking to abolish abnormal involuntary movements without producing the undesired paralysis. Klemme removed the cortex anterior to the excitable precentral cortex but no one else was able to achieve the favorable results which he claimed. Meyers (1941) and Takebayashi (1951) made less extensive lesions in the electrically excitable precentral gyrus but the results were unsatisfactory. Walker (1949, 1952, 1955), and Guiot and Pecker (1949) demonstrated that section of the cerebral peduncle is capable of decreasing or abolishing various types of abnormal involuntary movements. This was subsequently confirmed by White (1950), Bucy (1957), Meyers (1956) and Hamby (1953). Bucy and Meyers produced evidence that abolition
or diminution of the abnormal movements occur when the central portion of the peduncle which contains the cortico-spinal fibers is divided and not when the lateral part containing the so-called temporopontine bundle or the medial portion containing the fronto-pontine bundle is sectioned. (Figs. 5, 6 & 7) These observations, thus, again indicated that the abnormal involuntary movements are decreased or abolished when the descending motor pathways from the precentral gyrus, particularly the cortico-spinal fibers, are divided. Furthermore, although some of these patients developed a hemiparesis, some did not. These latter clearly demonstrated that it was possible to abolish these abnormal involuntary move-
ments without producing a paralysis. I had made a similar observation in 1948 under quite different circumstances. A young man suffering from postencephalitic parkinsonism also had a most severe behavior disorder for which a bilateral frontal lobotomy was made. For three months thereafter he had no tremor, although there was no paralysis whatsoever. Then the tremor returned although in a milder form in which it has been readily controllable with medication.

Because abnormal involuntary movements have long been classified as "extra-pyramidal" diseases, and as the basal ganglia were long regarded as practically synonymous with the "extra-pyramidal" system and as some earlier workers, notably Foerster (1921) and Jakob (1923), believe that the abnormal movements had their genesis in the globus pallidus, it was natural that lesions should be made surgically in the basal ganglia. This was first undertaken by Meyers in 1939. He extirpated the head of the caudate nucleus. Because of the favorable result in this case he subsequently carried out numerous other operations upon other structures in this general area. These included the anterior limb of the internal capsule, the putamen, the globus pallidus, the pallidofugal fibers. Some patients were definitely improved but because of the mortality (12%) and the uncertainty of the results he concluded that such operations were of very limited applicability and they have been discarded. There was, however, one important fact again demonstrated by these procedures; to wit, that under proper circumstances it is possible to abolish abnormal involuntary movements without producing a paralysis, Browder (1948). However, we must not lose sight of the fact that we do not have accurate anatomical information as to what was done in these operations and what was responsible for the improvement which was observed. It was determined early that the extirpation of the caudate nucleus was not responsible for the cessation of abnormal movements and the operation was extended to include division of the anterior limb of the internal capsule (Browder). This operation is a blind procedure and the section of fibers in the region of the internal capsule was extended posteriorly until weakness was produced in the contralateral extremities. This fact raises a serious question as to whether damage to the cortico-spinal fibers in the posterior limb of the internal capsule was not the effective part of the procedure. The fact that Meyers demonstrated that section of the fronto-pontine fibers in the medial part of the cerebral peduncle does not affect either abnormal involuntary or voluntary movements is further evidence that section of the anterior limb of the internal capsule is not the important part of the procedure.

Spiegel and Wycis using stereotactic methods produced electrolytic lesions which they believe are located in the midbrain, the thalamus and the ansa lenticularis in an effort to stop various abnormal movements. Lesions believed to have damaged the ansa lenticularis were the most effective but even these produced results which were inconstant, often temporary, and even in the best cases the tremor was not completely abolished. Narabayashi and his associates
injected a mixture of procaine in oil and wax into the globus pallidus in cases of parkinsonism. The rigidity in particular was reduced. The diminution of tremor was less striking and less long lasting than was the effect upon rigidity. Nevertheless, this pioneer work here in Japan is of the greatest importance in having for the first time demonstrated that lesions in the neighborhood of the globus pallidus produced by injection could decrease the rigidity and tremor of paralysis agitans without producing serious paralysis of the involved extremities. Guiot attacked the region of the ansa lenticularis and the globus pallidus by an operative approach beneath frontal lobe. Although many favorable results were obtained his procedure has been discarded because of the undesirable effects upon the patient's personality and behavior, and Guiot now is producing lesions in the region of the globus pallidus by a stereotactic technic. Cooper produced lesions in this area by obstructing the anterior choroidal artery, by injecting the region of the globus pallidus (Fig. 8) and the anterior part of the thalamus. Riechert and Hessler were among the first to assert the value of lesions in the anterior part of the thalamus in the treatment of parkinsonism. Following the lead of these pioneers many surgeons are now producing lesions in this general area by injecting alcohol or some other material, by electrolysis, by supersonic methods, or by mechanical means. It is noteworthy that the desirable results are obtained from lesions presumed to be in a variety of structures in this general area (Ansa lenticularis, Globus pallidus, Thalamus, etc.), and that the results are inconstant in that only some of the patients operated upon with these various technics are improved and that the beneficial results are often incomplete and temporary.

Several points are apparent from these observations. First, rigidity and tremor are not dependent upon the integrity of the same structures. Many

Fig. 8. Lesion produced in the globus pallidus in a cadaver following the technic used in patients treated for parkinsonism. (after Cooper).
observers have noted that lesions in the region of the globus pallidus abolish rigidity more readily and in a more lasting fashion than they do tremor. Others have noted that tremor is reduced by lesions which lie posterior to those which affect rigidity (Guiot). Second, it is obvious that the lesions are not placed with any considerable degree of accuracy. This is not surprising. Blundell has pointed out that the anatomical variation in this region is considerable and that it is not possible to place an instrument with strict accuracy in this area. Furthermore, the inability of the operator to control the flow of any destructive fluid which he may inject or of any electrolytic current which he may use increases the error of the method. That these errors are real is indicated by the inconstancy of the results. If the surgeon always destroyed the same neural structure it would be anticipated that he would always obtain the same results. However, he does not do so. Abolition or reduction of the tremor is obtained in between 25 to 60% of the patients operated upon and the favorable results vary in permanency from one patient to another. Obviously the surgeon is not always destroying the same neural structure and neither is he able to determine with accuracy what he will destroy or has destroyed. Third, the fact that the surgeons are able to obtain similar results from lesions supposed to be in the globus pallidus or in the antero-lateral part of the thalamus raises serious question as to where the effective lesion lies. It would seem not unreasonable to conclude that the effective lesions lie in the most central portion of the general area in which these various lesions are placed. The structure which lies in the center of this area, between the globus pallidus and the thalamus is the anterior part of the posterior limb of the internal capsule. (Fig. 9) It seems obvious that mere chance would determine that destructive lesions inaccurately placed in this general area would involve the internal capsule more often than any other structure. That this is the important structure which is destroyed in these operations is further supported by the fact that the internal capsule lies posterior to the globus pallidus and would be the structure involved by lesions placed more posteriorly so as to control tremor more effectively. Furthermore, this would agree with the fact that this is the part of the internal capsule containing the cortico-spinal fibers from the precentral gyrus and that destruction of these fibers at other points in the central nervous system (precentral motor cortex, cerebral peduncle, or spinal cord) also reduces or abolishes

Fig. 9. Diagramatic sketch of the area of the basal ganglia, thalamus and internal capsule. The location of the anterior part of the posterior limb of the internal capsule (the portion containing the cortico-spinal fibers to the arm and leg) between the internal segment of the globus pallidus and the anterior part of the thalamus is noteworthy.
parkinsonian tremor. The arguments of some that they have not destroyed the cortico-spinal fibers because they have not produced a hemiplegia or the sign of Babinski are without merit. The old concept that destruction of the "pyramidal" tract gives rise to a spastic paralysis appears to be in error. It has been shown that the cortico-spinal tract as it courses through the cerebral peduncle can be destroyed without producing a serious paralysis or spasticity while at the same time abolishing abnormal involuntary movements (Walker; Bucy). It is true that destruction of that portion of the cortico-spinal tract which arises from the uppermost part of the precentral gyrus and is related to the foot will permit Babinski's sign to be elicited, but the cortico-spinal fibers concerned with the remainder of the lower extremity, with the trunk and with the upper extremity can be destroyed without Babinski's sign appearing. It so happens that the organization of the cortico-spinal fibers in the internal capsule places the fibers to the great toe most posteriorly and in a position where they are less likely to be damaged by a lesion directed toward the globus pallidus than are cortico-spinal fibers concerned with those parts of the body lying above the foot.

**SUMMARY**

Complete understanding of the anatomy and physiology of normal muscular activity and of the pathological anatomy and physiology of abnormal involuntary muscular activity is not yet available. Although experiments upon laboratory animals have been and will continue to be of assistance in answering these problems the final solution must come from observations made upon man. The scientific controls exercised in the study of laboratory animals are difficult to apply in human cases but this does not make such controls any less essential if we are to learn the truth.

The formulation of hypotheses which will guide our therapeutic and experimental efforts must take into consideration all of the known facts. Important among these are the following:

*First:* It is possible consistently to abolish the tremor of parkinsonism, the intention tremor characteristic of lesions of the efferent cerebellar pathways, the abnormal involuntary movements of choreo-athetosis and of hemiballismus by extirpation of that portion of the precentral motor cortex in which the affected extremities are represented. From my own experience I have not the slightest doubt but what one can with certainty obtain such results in every case. Such an operation of necessity produces a partial paralysis of the involved extremities.

*Second:* It is possible to abolish or diminish the tremor of parkinsonism by lesions which sever the cortico-spinal fibers descending from the precentral gyrus either in the cerebral peduncle or in the spinal cord. It is possible to diminish the abnormal involuntary movements of choreo-athetosis and hemiballismus by lesions of descending pathways in the anterior white columns of the spinal cord or by section of the central portion of the cerebral peduncle.
Third: Destruction of the cortico-spinal fibers, whether carried out at cortical, midbrain or spinal levels has not reduced rigidity even though it has abolished the tremor of parkinsonism. This may be because other fibers have also been divided but in any event there is a striking difference so far as rigidity is concerned between the operations directed at the cortico-spinal fibers and those aimed at the globus pallidus.

Fourth: It is possible to abolish these various types of abnormal involuntary movements without producing paralysis. This has been accomplished by operations at several levels in the central nervous system.

Fifth: Destruction of the cortico-spinal fibers arising from the precentral gyrus apparently can be carried out without producing any considerable degree of paralysis.

Sixth: Lesions in the general region of the globus pallidus and the anterior part of the thalamus appear to be the most effective in abolishing parkinsonian tremor without producing paralysis and also in reducing the rigidity characteristic of this disease.

Although this sixth point has been interpreted as indicating that the nervous impulses responsible for the production of the parkinsonian tremor arise from the globus pallidus (Cooper), such an explanation seems unlikely. It does not explain why tremor is abolished or diminished by lesions directed toward the thalamus, or by destruction of the cortico-spinal fibers at the cortical, peduncular, or spinal level. On the other hand, it appears not unlikely that the beneficial effects upon the tremor are achieved by damaging the corticospinal fibers from the precentral gyrus in the anterior part of the posterior limb of the internal capsule. This structure lies at the center of the area toward which the attacks upon the globus pallidus and the thalamus have been directed. It has been shown that this pathway can be destroyed at other levels abolishing or reducing tremor and other abnormal movements without producing a serious paralysis of the extremities. It still appears reasonable that the development of tremor is dependent upon the existence of an intact cortico-spinal pathway from the precentral gyrus and that destruction of this pathway will abolish tremor. At one time I was of the opinion that the tremor was produced by impulses traveling over the cortico-spinal tract from the precentral gyrus. Such may be the case, but I agree with Ward that the movements may be produced by some more peripheral mechanism, perhaps in the brain stem, possibly in the anterior horn cells of the spinal cord. If such is the case the cortico-spinal tract may be important in this connection by maintaining a state of excitation in this more peripheral mechanism, without which it cannot produce the abnormal involuntary movements.

It is obvious that no one knows the neural mechanism by which abnormal involuntary movements are produced. Neither do we know which is the optimum surgical procedure for the abolition of such movements. The elicitation of such knowledge must be our goals. Carefully planned and carefully executed opera-
tions upon man are the means by which we must proceed. While continuing with our operations to help the victims of these disorders and to gain further knowledge, we must maintain complete objectivity, we must keep in mind all of the known facts and we must be satisfied by nothing less than the most perfectly controlled observations which are possible.

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