ANATOMICAL AND PHYSIOLOGICAL STUDIES OF THE INTERNAL CAPSULE AND ADJACENT DIENCEPHALIC STRUCTURES IN THE HUMAN DURING STEREOTAXIC SURGERY

Dr. Subramaniam Kalyanaraman, M.S., F.R.C.S., F.R.C.S.Ed.,
Department of Surgical Neurology,
University of Edinburgh.

Thesis submitted for the degree of
Doctor of Philosophy
in the Faculty of Medicine of the
University of Edinburgh
February 1964

"The surgeon must be so fortified by physiological as well as anatomical knowledge that he does not inflict damage in excess of that natural to the lesion; in other words, he must know what is the maximum good that he can expect from each individual intervention. He must see to it that every detail which can be used to build up a picture of the lesion and of its effects in all senses has been extracted beforehand, and that no detail in preparation has been omitted that might lead to defeat. Neurosurgery demands an apprenticeship in which the neophyte learns to regard all lesions as exercises in applied physiology, and all operations as experiments, to be carried out with the greatest caution and solicitude for the welfare of the patient."

.... Sir Geoffrey Jefferson.

CONTENTS

Acknowledgments

The success of this project is in a very great measure due to my supervisor, Professor F.J. Gillingham, whose constant encouragement and wise guidance were invaluable.

I am specially grateful to Professor N.M. Dott for his encouragement and to Dr. A.A. Donaldson and Dr. R.M. Gaze who directed the radiological and electrophysiological parts of the stereotaxic operations.

My special thanks are due to the neurosurgeons and neurologists who received me very kindly in their own departments and were good enough to discuss their stereotaxic procedures with me and show me their operative technique in detail. These discussions greatly clarified my ideas and stimulated my thoughts. Their intellectual impact may be seen in the following pages. Dr. G. Guiot of Paris, Mr. J. Hankinson of Newcastle, Professor R. Hassler of Freiburg, Professor B. Hughes of Birmingham, Mr. G. Knight of London, Professor H. Krayenbuhl of Zurich, Professor L. Leksell of Stockholm, Mr. I. McCaul of London, Professor T. Riechert of Freiburg, Dr. J. Talairach of Paris, Mr. R. Tym of Manchester and Mr. L. Walsh of London should be specially mentioned.

It is a pleasure and a privilege to acknowledge the co-operation of the following members of our own department - Dr. A.S. Brown, Dr. I.M.L. Donaldson, Dr. A. Gordon, Mr. I. Jacobson, Mr. J.J. Maccabe, Dr. A.F.J. Maloney, Dr. R.W. Porter, Dr. R.A. Roy, Mr. J.F. Shaw and Dr. W.S. Watson.

My special thanks are due to Dr. R. Marcal, Dr. I.M.L. Donaldson, Dr. C.F. Gonzales, Dr. D. Doyle and Mr. J. Turner who helped me with the translation of German, French and Spanish literature and to Miss A. Turner, who typed the script.

I am very grateful to Dr. D. Michie of the Department of Surgical Science and Mr. S.A. Sklaroff of the Usher Institute for checking the statistical tests and calculations used in the thesis.

I am happy to acknowledge the kind permission from Messers Thieme, Stuttgart for reproducing photographs from "Introduction to stereotaxis with an atlas of the human brain" by G. Schaltenbrand and P. Bailey.

The co-operation of the staff of the central medical library and the medical photography department was extremely valuable.

I am very grateful to Mr. G. Newell of the Animal Genetics Department who designed and made the stereotaxic instruments used in this project.

My gratitude is due to the resident doctors and other surgical colleagues, the nurses in the theatre and in the ward and the radiographers. They spent long hours on many days patiently with me in this project.

I am very happy to acknowledge the financial support for this work by the Commonwealth Scholarship from the Association of Universities of the British Commonwealth and the Graduate Research Fellowship and Travelling grant from the University of Edinburgh.

I am happy to place on record the assistance given by Miss H. Allison and Miss C. Crawford, Secretaries of our department, Miss D. Wardle, Librarian of the Royal College of Surgeons of Edinburgh and Miss W. Laidlaw of the Usher Institute, in preparing this thesis.

I am grateful to the editor of the British Medical Journal for permission to reproduce a photograph of the stereotaxic instrument.

Last but not least my grateful thanks are due to the patients who, in the process of obtaining surgical treatment for their diseases, enriched scientific knowledge.

PUBLICATIONS

The major part of the work reported in this thesis consists of hitherto unpublished material. A small part of the work, however, has been communicated to learned societies or published in scientific journals as shown below. Such publication was felt to be desirable, in view of the importance of the work, both by me and by my supervisor, Professor F.J. Gillingham.

A. Demonstrations

1. Meeting of the Scottish Society of Experimental Medicine, May 1961, Edinburgh.
2. Meeting of the Second International Congress of Neurological Surgery, September 1961, Washington.
3. Meeting of the Surgical Research Society, May 1963, Edinburgh.
4. Meeting of the International Neurosurgical Travellers' Club, July 1963, Edinburgh.
5. Meeting of the Physiological Society, July 1963, Edinburgh.
6. Meeting of the Royal College of Surgeons of Edinburgh, October 1963, Edinburgh.

B. Communications to Learned Societies

1. Meeting of the Department of Surgery, University of Edinburgh, 9th June, 1962. Paper by Gillingham, F.J. and Kalyanaraman, S. on "The Surgeon's Laboratory".
2. Meeting of the Scottish Association of Neurological Sciences, Edinburgh, 22nd June, 1962. Paper by Gillingham, F.J. and Kalyanaraman, S. on "Surgical Exploration of the Internal Capsule".

3. Meeting of the Second European Congress of Neurological Surgery, Rome, 19th April, 1963. Paper by Gillingham, F.J. and Kalyanaraman, S. on "Bilateral Stereotaxic Lesions in the Management of Parkinsonism and the Dyskinesias".

4. Meeting of the Scottish Association of Neurological Sciences, Glasgow, 21st May, 1963. Paper by Gillingham, F.J. and Kalyanaraman, S. on "Bilateral Stereotaxic Lesions in the Treatment of Parkinsonism and the Dyskinesias".

5. Meeting of the Department of Surgical Neurology, University of Edinburgh, 28th October, 1963. Paper by Kalyanaraman, S. on "Stereotaxic Biopsy".

6. Meeting of the Department of Surgical Neurology, University of Edinburgh, 18th November, 1963. Paper by Kalyanaraman, S. on "Transcapsular Lesions".

7. Meeting of the Department of Surgical Neurology, University of Edinburgh, 13th January, 1964. Paper by Kalyanaraman, S. on "The Surgical Treatment of Oculogyric Crises".

8. Due for presentation. Meeting of the Edinburgh Pathological Club, March, 1964. Paper by Kalyanaraman, S. in the "Symposium on Stereotaxic Surgery".

C. Scientific Papers Published

1. Gillingham, F.J. and Kalyanaraman, S. (1963). Bilateral Stereotaxic Lesions in the Management of Parkinsonism and the Dyskinesias. Excerpta Medica International Congress Series No. 60, 115.

2. Kalyanaraman, S. (1963). Stereotaxic Surgery. Madras Clinical Journal, 30, 6, 129.

3. Gillingham, F.J. and Kalyanaraman, S. (1963). Parkinson's Disease. Journal of Physiotherapy, 49, 12, 378.

4. Kalyanaraman, S. and Gillingham, F.J. (1964). Stereotaxic Biopsy. J. Neurosurg. (in print).

5. Kalyanaraman, S. (1964). Stereotaxic Surgery for Intractable Pain. Silver Jubilee Number of the Journal of the Stanley Medical College, Madras (in print).

Arrangement of Material

The method followed in arranging the material in this thesis is slightly unconventional but is intended to facilitate easy reference.

As far as possible the text has been made self-explanatory. Since each chapter relates to one aspect of the study, its summary, explanatory diagrams and references are grouped together and presented at the end of that chapter.

The X-rays and scattergrams for each chapter require comparison with each other. Grouping them together at the end of the chapter is expected to facilitate this.

There is some unavoidable repetition due to the presentation of references with each chapter. This is felt justified because of their easier access.

The nomenclature followed to describe the thalamic nuclei in the text is based on that of Schaltenbrand and Bailey's atlas. This is essential since the scattergrams are superimposed on the plates of that atlas. A note on the nomenclature of thalamic nuclei as well as a list of some of their abbreviations are included in the appendix, for clarification of the atlas plates reproduced in the thesis.

PART I : I N T R O D U C T I O N

Chapter I: Object of Study

Chapter 2: Methods and Material

Chapter 3: Individual Variations

CHAPTER I

OBJECT OF STUDY

"There is perhaps no subject in physiology of greater importance and general interest than the functions of the brain, and there are few which present to experimental investigation conditions of greater intricacy and complexity. No one who has attentively studied the results of the labours of the numerous investigators in this field can help being struck by the want of harmony, and even positive contradictions, among the conclusions which apparently the same experiments and the same facts have led to in different hands. And when seemingly well established facts of experimentation on the brains of lower animals are compared with those of clinical observation and morbid anatomy in man, the discord between them is so great as to lead many to the opinion that physiological investigation is little calculated to throw true light on the functions of the human brain."

.... Ferrier (1886)

Ferrier's classical assessment of the difficulties of interpretation of neurophysiological experiments is very evident in the study of the internal capsule and adjacent diencephalic structures.

The neurophysiologist and clinical neurologist are faced with several problems while trying to equate animal experiments with human phenomena. Most animals have nervous systems which are in no way comparable to the human in complexity of structure and function. Even animals like the monkey and the chimpanzee which are nearer to man than other laboratory animals, are still different from him for any close similarity to be assumed with reference to complex neurological functions.

These comparisons become specially difficult in structures like the basal ganglia and the thalamus which have probably taken on 'new' functions in the course of evolution as well as 'lost' some of their 'old' functions.

Again the progressive diffuse neurological disorders loosely referred to as 'diseases of the extrapyramidal system' cannot be reproduced in the nervous system of lower animals. These difficulties have been well summarised by Ward (1958).

It has been impossible, until very recently, to explore the interior of the living human brain with neurophysiological tools. The advent of stereotaxic surgery during the past fifteen years has however opened up new opportunities to do so.

When such explorations were carried out, it was seen that the phenomena observed in the human were contradictory to previously held assumptions as a result of animal work. Numerous examples of such contradictions can be quoted.

For example, Kennard (1944) observed that monkeys with bilateral isolated stereotaxic lesions in the pallidum did not show any abnormalities of behaviour. When the lesions were larger, however, a definite action tremor with hypertonia was seen. The observations of Krayenbuhl, Wyss and Yasargil (1961) as well as those of Gillingham and Kalyanaraman, (1963) in the human on bilateral pallidal stereotaxic lesions were quite different from these.

Hunter and Jasper (1949) observed as a result of thalamic stimulation in cats, arrest reaction, petit mal and grand mal attacks. Stimulation of comparable areas of the human thalamus by Hassler (1960) and Hughes (1963) produced phenomena which were different from those observed in animals.

Lassek (1954) summarised the work up to date on the pyramidal tract in man and in subhuman animals. It is surprising how little work had been done on the disposition

of the fibers in the internal capsule.

The studies of Levin (1936) and more recently of Barnard and Woolsey (1956) in animals led us to believe that the anterior part of the posterior limb of the internal capsule contained the corticobulbar and corticospinal tracts. This view has been handed down from textbook to textbook and from generation to generation of medical students. It was accepted as a corollary that any encroachment surgically on this area carried with it the risk of production of a 'capsular hemiplegia'. Smith (1962) however showed on autopsy material that stereotaxic lesions encroaching on the internal capsule did not in fact produce such a syndrome clinically when the patient was alive. Guiot (1959) reported an arrangement of fibers in the internal capsule different from the classically held concept.

The method of stereotaxic surgery practised by Gillingham and his colleagues (1960) afforded opportunities to study for the first time in the living human brain the effects of creation of reversible and irreversible lesions in the sensory relay nucleus of the thalamus, the thalamoparietal projection, the oral ventral nucleus of the thalamus, the posterior limb of the internal capsule and the globus pallidus, all in the same electrode track. This posterior approach as well as the deliberate production of capsular lesions by Gillingham (1961) provided an opportunity not available in other methods of stereotaxy to study the internal capsule and adjacent diencephalic structures in the living human brain.

The introduction of two new methods not used before in human stereotaxic surgery facilitated these studies and provided valuable information. The first was depth recording using unipolar microelectrodes and the second was the technique of stereotaxic biopsy.

The anatomical and physiological studies presented in this thesis are the results of these unique opportunities and methods.

REFERENCES

1. Barnard, J.W. and Woolsey, C.N. (1956). J. Comp. Neurol. 105, 25.

2. Ferrier, D. (1886). Introduction to 'The Functions of the Brain'. Second Edition. Quoted by Walshe, F.M.R. (1942). Brain, 65, 409.

3. Gillingham, F.J., Watson, W.S., Donaldson, A.A. and Naughton, J.A.L. (1960). Brit. med. J., 2, 1395.

4. Gillingham, F.J. (1961). First International Symposium on Stereo-encephalotomy, Philadelphia, 1961. Confin. Neurol., 22, 385. (1962).

5. Gillingham, F.J. and Kalyanaraman, S. (1963). Second European Congress of Neurological Surgery, Rome, 1963. Excerpta Medica International Congress Series No. 60, 115.

6. Guiot, G. (1959). Neuro-chirurgie, 5, 17.

7. Hassler, R. (1960). In Handbook of Physiology, Section I, Neurophysiology. Ed. by Field, J., Magoun, H.W. and Hall, V.E., American Physiological Society, 2, 863.

8. Hughes, B. (1963). Second European Congress of Neurological Surgery, Rome, 1963. Excerpta Medica International Congress Series No. 60, 124.

9. Hunter, J. and Jasper, H.H. (1949). Electroencephalog. & Cli. Neurophysiol., I, 305.

10. Kennard, M.A. (1944). J. Neurophysiol., 7, 127.

11. Krayenbuhl, H., Wyss, O.A.M. and Yasargil, M.G. (1961). J. Neurosurg., 18, 429.

12. Lassek, A.M. (1954). The Pyramidal Tract. Its Status in Medicine. C.C. Thomas, Springfield, Illinois, 108.

13. Levin, P.M. (1936). J. Comp. Neurol, 63, 369.

14. Smith, M.C. (1962). Brit. med. J., I, 900.

15. Ward, A.A. (1958). In Pathogenesis and Treatment of Parkinsonism. Ed. by Fields, W.S., C.C. Thomas, Springfield, Illinois, 106.

CHAPTER 2

METHODS AND MATERIAL

"All true scientific research in medicine stems from the bedside"

.... Barney Sachs.

"Clinical experiment need not mean the subjection of patients to uncomfortable procedures of doubtful value or benefit. It means the planning of a line of action and the recording of observations designed to withstand critical analysis and give the answer to a clinical problem. It is an attitude of mind."

.... Sir Robert Platt.

(a) Evolution of Stereotaxic Surgery

Although the principle of stereotaxy had been used in animals towards the end of the last century by several workers, to Horsley and Clarke (1908) is due the credit of building a properly designed stereotaxic instrument for animals, the basic principles of which were subsequently used in the human machines.

Spiegel, Wycis, Marks and Lee (1947) described the first stereotaxic apparatus for operations on the human brain. During the next few years, a number of stereotaxic instruments and operative procedures were described. Among these, the methods of Bailey and Stein (1951), Bertrand (1958), Cooper (1955), Guiot (1958), Hughes (1961), Leksell (1949), Mark et al (1954), McCaul (1959), Narabayashi (1956), Riechert and Mundinger (1959), Schaltenbrand (1959) and Talairach et al (1952) are well known.

Each method when properly carried out, as practised by the original authors, was capable of accuracy within a mm. of the desired target.

(b) The Guiot-Gillingham Method of Stereotaxy

All the operations from which material for this work was

drawn were carried out by this method. It is a modification of the original method described by Guiot. It has been described in detail by Gillingham et al (1960).

The first part of this procedure was designed to determine the midline of the brain in relation to the ventricular system with reference to the outer table of the skull.

In the majority of the procedures, local infiltration of lignocaine hydrochloride with adrenaline was used for anaesthesia. In a very small number of cases, general endotracheal anaesthesia was required. In the last hundred operations, the local anaesthesia was supplemented by intravenous neuroleptanalgesia using dehydrobenzperidol and phenopyridine. A few cases were done under local anaesthesia supplemented by intravenous methohexitone drip turned on and off several times during the operation depending on whether the patient was required to be unconscious or awake and co-operative.

In the first one hundred and seventy two cases lumbar air encephalography was used in the determination of the midline and the electrode track. Positive contrast (Ethyl-Iodophenylundecylate - "myodil") ventriculography was subsequently used to align the lateral sights under fluroscopy during the second part of the operation. In the other cases, however, positive contrast ventriculography with 3 c.c. of contrast and 3 c.c. of air was used for the first part of the procedure also. The radiological part of the operation has been described by Gillingham et al (1960) and Donaldson and Gillingham (1960).

With the patient in the semisitting position, three incisions were made above the frontal sinus, behind the coronal suture and below the lambda. The outer table of the skull was exposed at each site and a number of small dental drill holes, straddling the midline, were made. Into each hole was introduced an opaque lead marker which was fixed in position with bone wax. After polybactrin spray, the incisions were closed. A rubber catheter was tied into the frontal horn of the right lateral ventricle by a frontal burrhole.

The patient was now transferred to the X-ray department and 3 c.c. of air and 3 c.c. of myodil were introduced through the catheter. The head was manipulated in such a way that the contrast flowed into the third ventricle. By proper positioning of the head, the posterior border of the anterior commissure and the anterior border of the posterior commissure could be easily outlined on two separate but identically superimposable lateral films. The films were superimposed and a line was drawn joining the posterior border of the anterior commissure and the anterior border of the posterior commissure. This formed the horizontal axis of the three Cartesian co-ordinate axes on which all subsequent calaulations, measurements and research were based.

Two points were now marked on the X-ray. These were called the thalamic and pallidal target points. It should be emphasised that these were radiological points and did not necessarily represent the thalamus and pallidum in the subsequent surgical procedure. Depending on the lateral distance of the track used, as well as individual anatomical

variation, either of them could be in the posterior limb of the internal capsule.

The pallidal target was a point 3 mm. behind the posterior border of the anterior commissure in the direction of the intercommissural line and one mm. below the intercommissural line. The thalamic target was a point on the midcommissural line usually 5 mm. above the intercommissural line. (The midcommissural line was taken as the perpendicular bisector of the previously drawn intercommissural line which is also referred to subsequently as the CA-CP line. These two lines in the midsagittal plane of the brain represented two axes while the third axis was taken as a line perpendicular to both of them through their point of intersection.) In some of the early cases, the thalamic target was taken 4 mm. above the intercommissural line. In a few cases of depth electrical recording for evoked potentials, it was taken at 3 mm. above the intercommissural line.

The pallidal target was now joined to the thalamic target on the lateral X-ray film to meet the outer table of the skull in the parieto-occipital region a few cms. above the posterior row of lead markers. If, as was usually done, the posterior row of markers was put in at 6 cm. above the external occipital protruberance, the line joining the targets intersected the outer table of the skull between 2 and 4 cm. above the markers.

The Guiot-Gillingham stereotaxic machine had been so designed that the fixation of the cross-bar at elevation mark 0 on the sagittal bar would correspond to the entry of the electrode at 2 cm. above the posterior row of markers. The

elevation of the cross-bar for any given electrode track therefore could be very easily calculated on the lateral films by projecting the line joining the targets backwards, measuring the height of intersection of this line on the outer table of the skull above the posterior row of markers and subtracting 2 cm. from it. Using the principle of similar triangles, in comparing the elevation of the cross-bar on the sagittal bar with the height of the thalamic target above the intercommissural line, the ratio of proportion was easily worked out. For routine X-rays this was 1 in 10. Therefore if an error of 2 mm. was made on the machine, the error at the thalamic target would be 0.2 mm. Since the machine was accurate to 0.5 mm. any error at the thalamic level was negligible.

In addition to the lateral films, three AP films were taken. One was taken in the position judged to be strictly AP by naked eye inspection and the other two films were taken at half a degree tilt of the tube to either side. On inspection of the films it was seen that one of the three films was a strict AP as regards the ventricular system of the brain. From this film, the midline of the brain was marked out with reference to the septum pellucidum and the third ventricle (also taking into account, where suitable, the aqueduct, the fourth ventricle and the falx cerebri). In each of the three rows of lead markers, the marker corresponding to the midline of the brain was identified.

The patient was taken back to theatre either on the same day or after an interval of two days if it was felt that the patient was too tired after the preliminary procedure. With

the patient in the prone position, the midline incisions were now reopened and the midline marker in each row was identified. A large dental drillhole was made corresponding to the midline at each row and a screw with an inset conical head was fixed into it. The patient thus had a 'built-in' midsagittal plane with reference to his ventricular system marked in the outer table of his skull by three stainless steel screws.

The sagittal bar of the machine was fixed to the screws and the cross-bar was attached at the height previously calculated from the lateral X-rays. The lateral sights of the cross-bar were now aligned against the pallidal target using positive contrast ventriculography (usually 3 to 5 c.c. more of myodil were needed), the final position being guided by the radiologist by fluroscopic control with the image intensifier as the surgeon manipulated the instrument. By keeping the target in the centre of the viewing field, X-ray distortion was negligible when the alignment was made on a target 3 mm. behind and 1 mm. below the anterior commissure outlined by contrast. The stereotaxic machine had been designed in such a way that when the centres of the two rings in the lateral sights coincided with the pallidal target, the electrode tip would reach the target inside the brain on the line joining the centres of these two rings. For purposes of permanent record, a lateral X-ray was now taken of the alignment of the sights with the image intensifier in position.

The entry burr-hole for the electrode was now made at 12 to 18 mm. from the midline depending on the lateral distance

chosen by the surgeon for that particular case. The lateral distance was decided on clinical grounds rather than for radiological reasons. However, if the ventricular system was markedly dilated a more lateral track was chosen. The usual lateral distance was 15 or 16 mm. from the midline.

All the 'electrodes' used traversed the brain in a parasagittal plane parallel to the midsagittal plane. In the following order, one or more of the instruments were introduced along the same track-depth recording - electrode, biopsy cannula, combined stimulating and coagulating electrode and opaque marker carrier. All these instruments were of the same length, and diameter, so that any one of them was replaceable by any other without alteration of the track. The data obtained from the use of any of them could be referred to the postoperative charts on the stereotaxic atlases, without error.

(c) Use of Stereotaxic Anatomical Atlases

When it became obvious that stereotaxic surgery had a large and permanent place in neurosurgery, efforts were made by several workers to secure a reliable guide from autopsy material for accurate anatomical localisation of stereotaxic lesions. Several stereotaxic atlases and guides have been published. That of Schaltenbrand and Bailey (1959) is probably the most widely used, as well as the most detailed and accurate. Those of Spiegel and Wycis (1952) and of Talairach and his colleagues (1957) are less widely used. The atlas of Guiot and his coworkers (1961) is useful only if their method if followed. Other atlases include those of Delmas and Pertuiset (1959) and Van Buren and Maccubbin (1962).

In the present work, the atlas of Schaltenbrand and Bailey was used. The 'microscopic' plates at four times magnification were found to be the most useful.

At the end of the operative procedure by the Gillingham technique, an opaque steel marker 1.5 mm. in diameter was left at the most anterior point of coagulation. The patient was kept in the prone position and was transferred to the X-ray department where strict PA and lateral films were taken.

By superimposition of these films on the previous ones, the electrode track could easily be drawn from the available data on the X-ray. All measurements were corrected for X-ray distortion by 10%. Again, since the marker was usually left at the pallidal target, close to the anterior commissure, minor variations in X-ray distortion due to wrong centering of tube or different sized heads would not matter. Even if the error in calculation of distortion were 50% (for the sake of argument) the actual distortion would be from 10 to 15% and this in terms of the distance from the anterior commissure would be 2.55 mm. instead of 2.7 mm. Since the accuracy of measurement on the X-ray was only of the order of 0.5 mm., such minor errors did not amount to significant proportions. Finally, as would be seen later, the conclusions were drawn from scattergram studies and electrical studies both of which eliminated errors in diametrically opposed, but equally effective, methods. It was felt, therefore, that problems of X-ray distortion did not arise in this procedure either for

therapeutic or for research purposes.

Five hundred lesions were charted on graph paper and cellophane for superimposition on the stereotaxic atlas. Each stereotaxic lesion was drawn on a graph to correspond to the four times magnification of the 'microscopic' series of the atlas. The sagittal plane was first drawn since this corresponded to the plane of the electrode track and the entire lesion could be visualised in one plane. From this, horizontal and frontal sections could be drawn. In addition to superimposition of these charts on the atlas figures, they were also treated as mathematical points in relation to the Cartesian system of co-ordinates without any reference to the atlas and statistical tests were applied to them. This procedure cancelled out the error due to individual variations and the criticism that the atlas brain was not an 'ideal' brain for comparison. The details of the methods of charting lesions from post-operative X-rays to anatomical atlases are given in Appendix (A).

(d) Depth Electrical Recording and Audiomonitoring

This was the most reliable and precise among the methods available for eliciting anatomical and physiological information during stereotaxic operations.

The electrophysiological part of the procedure described below was carried out by Dr. R.M.Gaze and the surgical part as well as the charting of the lesion postoperatively on stereotaxic anatomical atlases was done by the author. The entire procedure is described as a combined team work. The

information and conclusions in one of the subsequent chapters is based partly on scattergram studies of the sites, where evoked potentials were elicited, as charted on stereotaxic anatomical atlases.

Stereotaxic unipolar depth electrical recording has been done in animals by several workers. The work done by Gaze and Gordon (1952, 1954, 1955) in the thalamus of the cat was the method on which the present human work was based. Work on the human thalamus using bipolar microelectrodes during stereotaxic surgery has been done by Guiot (1962), Albe-Fessard (1962) and their colleagues. Vourc'h et al (1963) showed that depth recording with microelectrodes was unaffected by hydroxydione (viadril), an experience similar to that of Brown (1963) with intravenous neuroleptanalgesia.

Using intravenous methohexitone drip anaesthesia Hider and Kalyanaraman (1963) found that depth electrical recording was relatively unaffected. When the electrode picked up thalamic rhythms synchronous with clinical tremor, this was abolished when the patient was rendered unconscious and the tremor abolished by methohexitone. When the drip was stopped and the patient awakened, the rhythm returned with the reappearance of clinical tremor. Spontaneous thalamic background activity and evoked potentials were not affected by methohexitone. During the stage of fasciculation during induction with or recovery from methohexitone, a deep rhythm synchronous with fasciculation was observed.

Ervin and Mark (1960) reported on stereotaxic thalamotomy

in the human where they used audiomonitoring to define the sensory relay nucleus. They were also able to obtain in some cases evoked potentials in the thalamus from peripheral stimulation, although they used macroelectrodes. Hankinson, Pearce and Rowbotham (1960) reported a few cases where they recorded spontaneous activity and evoked potentials in a similar manner. Spiegel and Wycis (1961) recorded from the spinothalamic tract below the thalamus, peripherally evoked potentials, using a concentric needle electrode.

The recording electrode used in the present study was a concentric needle electrode consisting of a stainless steel tube of external diameter of 2 mm. which carried within it a retractable tungsten wire 0.5 mm. in diameter, sharpened at the point to a tip diameter of 10 - 20 microns. The tip was sharpened by grinding initially and electroplated finally in saturated sodium nitrite solution, using a carbon indifferent electrode and a voltage of 3-8 volts from a bell transformer. The sharpened electrode was then insulated with 5-10 coatings of lacquer. After each coating, the electrode was baked in dry heat to about 200°C.. Such electrodes were normally ready for use without the necessity of removing the insulation from the tip.

The outer tube which was uninsulated, had, at its distal end, a nylon collar with a small hole in it through which the tip of the tungsten recording electrode could be protruded. At the proximal end of the electrode there was a sliding collar arrangement which allowed the tungsten wire either to be completely withdrawn within the outer sheath

or to be protruded to a predetermined distance, usually 5 mm. beyond the nylon tip. The uninsulated outer sheath in contact with the brain along the electrode track as well as the stereotaxic instrument fixed to the patient's head, acted as the indifferent electrode for recording purposes. There were two movable collars along the shaft of the recording electrode. The upper collar served to set the total length of the instrument so that the length of the electrode coincided accurately with those of the other instruments used along the electrode track. The lower collar was used to clamp the electrode rigidly to the movable bar of the stereotaxic machine so that it could be driven forward using the micrometer screw. This forward movement could be adjusted to be as little as 10 microns at a time. (See photographs at the end of chapter 2)

The tungsten recording electrode was connected through a cathode follower of standard type to a Tektronix 122 preamplifier in the operating theatre. The preamplifier was used at a gain of 1000, with high frequency cut-off at 10 KC and coupling time-control of 2 msec. The signal from the preamplifier was then transmitted through coaxial cable to a gallery above the theatre where it was split into two parts. The first was fed into a Tektronix 502 double beam oscilloscope with photographic facilities and the second into an audioamplifier. Time marks from a signal generator (Tektronix Type 180A) were put on the other beam of the oscilloscope. The output of the audioamplifier

was then sent both to a loudspeaker in the gallery above the theatre as well as to a loudspeaker in the operation theatre. Thus both the surgeon and the physiologist could hear the electrical responses as the electrode was moved. Permanent records of the depth electrical activity were produced by photographing the oscilloscope trace on moving film (Shackman AC 2/810 camera) and/or by feeding the output of the preamplifier on to a tape recorder.

The recording tip was protruded beyond the blunt tip of the electrode carrier after the latter had penetrated the pulvinar of the thalamus. By means of this arrangement any possible laceration of the choroid plexus by the sharp recording tip was avoided. This ensured that there was no added risk to the patient by using depth electrical recording.

The distinction between grey and white matter was relatively easy. The oscilloscope records as well as the sound in the loudspeaker were characteristic and distinct. It was thus possible to define the thalamocapsular junction and the pallidocapsular junction with extreme accuracy, usually of a fraction of a millimeter. The definition of the various nuclei traversed in the thalamus was not so precise. If the electrode track was low, it usually passed through the sensory relay nucleus of the thalamus or the thalamoparietal projection in front of it. In such a case, distinct evoked potentials were obtained from the face or upper limb from exteroceptive or proprioceptive stimuli or both. Both in the sensory relay nucleus as well as in

the region above it (nucleus lateralis posterior of Guiot or nucleus zentralis of Hassler) which was traversed by a higher electrode track, the background noise of spontaneous cellular activity was much louder than in the pulvinar. The entry from the pulvinar into these structures was therefore usually recognisable. The junction however was not as sharp as the border between white and grey matter. Guiot (1963) recognised the pulvinar by its characteristic spindle activity in the depth E.E.G.. This was probably due to the bipolar nature of his electrode and the different filters used for the E.E.G. machine. Using the present technique of unipolar recording, no spindle activity was noted.

One other nucleus was sometimes distinguishable. When the recording tip left the anterior aspect of the thalamic target site (nucleus ventralis lateralis of Guiot or nucleus ventralis oralis of Hassler) there was a short zone of silence after which cellular activity was again heard for a millimeter or less. The sound corresponded to that heard in the reticular nucleus of animals and was taken to represent the reticular nucleus between the thalamus and capsule. After this, there was great paucity of spike discharges in the oscilloscope with silence in the loudspeaker as the posterior limb of the internal capsule was traversed by the electrode. The entry into pallidum was again characterised by cellular activity shown by high amplitude spikes in the oscilloscope and corresponding sound in the loudspeaker.

It was thus possible to define along the electrode track, the positions of the pulvinar, the sensory or the central nucleus, the complex of ventral intermediate and ventral oral nucleus, the reticular nucleus, the posterior limb of the internal capsule and the globus pallidus.

(e) Stereotaxic Biopsy

Since the introduction of therapeutic methods to create small localised lesions in the deep structures of the brain for the treatment of different kinds of neurological disorders, interest has been stimulated in obtaining small biopsies of these regions. Such biopsies are useful to verify by histological methods the accuracy of radiological localisation used for stereotaxic procedures. They also serve to study by histological and histochemical methods the pathology of disease processes.

Housepian and Pool (1960) reported on thirty-four biopsies from twenty-eight patients, using a leucotome technique. The biopsies obtained were approximately 500 mgrms. specimens. Five of these biopsies were described as negative, being from white matter. The same authors (1962) later reported further studies with stereotaxic biopsies from the thalamus. Heath, John and Foss (1961) reported biopsies from the cortex of three human subjects, obtained by stereotaxic methods. The amount of tissue removed was somewhat smaller. Jinnai and his colleagues (1961) referred to stereotaxic biopsies of the thalamocapsular junction carried out by them. They did

not however give further details.

In the present study, 155 biopsies were taken from 62 patients during 65 operations. Three of these patients had biopsies from both hemispheres on different occasions. Fifty-eight of these patients suffered from Parkinsonism, one from disseminated sclerosis and two from choreoathetosis. In one, the pathology causing involuntary movements was uncertain.

The biopsy cannula was devised by Mr. G. Newell of the University of Edinburgh. (See figures at the end of chapter)

It was introduced to the required depth, the inner piston was withdrawn for 2 mm. and the needle was rotated through one full circle to enable its exposed cutting edge to cut a slice of brain approximately 2 mm. in diameter. This tissue was sucked into the needle (air tight suction being provided by a nylon washer) by withdrawing the inner piston for another few mm. The entire biopsy needle was now withdrawn and the specimen ejected out by gentle pushing of the inner piston to the tip of the needle. Usually, with gentle suction, biopsies of 2 to 3 mm. in size, weighing approximately 5 to 25 mgrms, were obtainable. Often tremor was seen to subside or lessen as soon as a biopsy was taken from the target site in the thalamus.

Immediately after withdrawing the biopsy needle, the coagulating needle was introduced to the same point to act as a tampon to prevent any bleeding at the site of biopsy.

If a biopsy was attempted from a track where another instrument had already been introduced, it was occasionally

difficult to obtain good specimens. Sometimes an attempt at biopsy produced only blood clot especially if it was attempted within 3 mm. of a coagulation lesion done a few seconds earlier. In cases of dyskinesias one was not always successful in obtaining a biopsy from the capsular region. This conformed to our experience that the resistance to the passage of the electrode near the posterior limb of the internal capsule and through it was greater in dyskinesias than in cases of Parkinsonism. Apart from these exceptions it was possible to get a biopsy from the thalamus, capsule or pallidum whenever it was attempted.

No special postoperative complications were encountered in any of the 155 biopsies. There were no cases of hemorrhage at the site of biopsy (as shown by extreme drowsiness with shift in position of steel marker left at conclusion of operation or by transient moderately severe hemiparesis). It was interesting to note that there were 10 cases of hemorrhage as judged by the above criteria in the 405 stereotaxic operations in which no biopsy was taken.

The specimens were fixed with formalin and stained with hematoxylin and eosin. Distinction between grey and white matter was relatively easy under ordinary microscopy.

Of the 155 biopsies, II were not available for ordinary microscopy either because they were subjected to electron microscopy or because fixation with formalin was not satisfactory. Four biopsies were taken during a leucotomy and are not considered any further. One was from an area of previous electrocoagulation and showed no definite normal

tissue.

This study was therefore concerned with the remaining 139 biopsies which were reported upon. A preliminary analysis was first carried out classifying the biopsies according to the histological report.

Of these, 81 were identified as grey matter due to the presence of ganglion cells. Fifteen were identified as white matter due to predominance of fiber bundles with absence of ganglion cells. Twenty-eight specimens showed a mixture of both grey and white matter. Fifteen biopsies could not be classified definitely as grey and/or white matter since they consisted largely of blood clot with only a minute quantity of neural tissue. Fifteen attempts out of 155 therefore produced inadequate material for examination. Biopsy failure rate was therefore less than 10%.

Of the 81 biopsies which were identified as grey matter, 61 were from the thalamus and 20 from the pallidum. All postoperative lesions were charted on the atlas of Schalatenbrand and Bailey (1959), working back on the X-rays showing the opaque steel marker left at the conclusion of the operative procedure. It was seen that all the 20 pallidal biopsies corresponded to the pallidum on the stereotaxic atlas.

Fifty-two out of the 61 thalamic biopsies conformed to the corresponding site on the atlas. Of the nine cases which did not, five biopsies were taken after withdrawal of another instrument introduced in the track before the biopsy cannula. One was known to be a case of abnormal

anatomy in that region and another case has had two previous stereotaxic operations on the same hemisphere. Fifty-two out of fifty-four biopsies therefore coincided with the atlas in the thalamus after these cases had been excluded.

Since all the thalamic and pallidal biopsies were taken at or very near the thalamic or pallidal targets defined radiologically, this information gave us very good confirmation of the great accuracy of the radiological methods used.

Of the 15 biopsies reported as white matter, 11 conformed to the internal capsule on atlas charting. One was probably a bit of subcortical tissue pushed in by the needle. Three were from the capsule although intended to be from pallidum. One was from the capsule although intended to be from the thalamus. Individual anatomical differences between the patients probably accounted for this.

At the junctional tissue between grey and white matter the greatest degree of lack of correlation with the atlas brain occurred. In only 5 out of these 28 cases, the biopsy was intended to be from junctional tissue. In 23 cases the biopsy was intended to be from grey or white matter as per the atlas.

The effect of previous instrumentation along the electrode track on accuracy of biopsy report was now considered.

Previous Instrumentation	Report compared to atlas charting		Total
	Correct	Not Correct	
Done	70	18	88
Not done	18	16	34
TOTAL	88	34	122

Application of the χ^2 test gave the following results.

$\chi^2 = 7.362$ $n = 1$ $P<0.01$

Previous instrumentation along the electrode track was therefore associated with a significantly larger number of incorrect biopsy reports as compared to stereotaxic atlas charting.

These results therefore could now be classified in a different way to assess the correlation between the atlas and the histology report. In this final analysis all biopsies taken when the biopsy cannula was not the first instrument to be introduced were omitted. The biopsies which were after a previous operation as also the piece of subcortical tissue were excluded. The remaining 88 cases were analysed as shown in the table.

Correlation between intended site of biopsy as per stereotaxic atlas and histology report (88 biopsies)

Intended site	Histological Report		
	Grey Matter	White Matter	Both
Thalamus (46 biopsies)	44	0	2
Pallidum (25 biopsies)	14	3	8
Capsule (11 biopsies)	2	7	2
Junctional Tissue (6 biopsies)	1	0	5

This suggested that the degree of accuracy with radiological localisation with this method decreased in the following order for targets in our method of stereotaxy - thalamus, pallidum and capsule.

From this study therefore the following conclusions could be drawn.

(I) Stereotaxic biopsy was a safe method of obtaining tissue for examination from intended sites from the depth of the brain.

(2) When properly carried out the failure rate was less than 10% and accuracy of the order of 1 to 2 mm.

(3) In order to get reliable results, the biopsy cannula should be the first instrument to be introduced along the planned track.

(4) The thalamic target and to a less extent the pallidal target as defined radiologically in the Guiot-Gillingham method were accurate.

(5) The borders of the internal capsule however could not be predicted with the same accuracy by the radiological method alone due to individual anatomical variations.

(f) Creation of Reversible and Irreversible Lesions

For the creation of the therapeutic lesion, diathermy was used. Studies in eg, white and animals as well as examination of autopsy material by Maloney (1960) revealed that the calibration of the diathermy could be standardised with reasonable accuracy.

When the knob of the diathermy machine was set at I and coagulation was done for five seconds, a minimal heating effect only was produced. At calibration II, a slightly greater effect was seen with some shrinking of tissue around. Both these created destruction of a few adjacent cells, recognisable macroscopically only if the tissue was examined

immediately afterwards. For practical purposes and to clinical neurological testing, these two types of coagulation produced no permanent effect. In fact, temporary signs were seen depending on the position of the electrode and these disappeared usually within a minute or so. For example, transient tingling in the fingers or corner of the mouth, a slight jerk of the hand, minimal weakness of handgrip, cessation of tremor or slurring of speech might be noted.

If the effect produced was a desirable one like abolition of tremor or rigidity, the coagulation was now continued to calibration III on the machine. This would produce a lesion approximately 4 mm. in diameter around the electrode. This would be a permanent and irreversible lesion. If still no motor, sensory or other neurological deficit was seen, coagulation would be continued to calibration IV when a lesion approximately 5 mm. in diameter was created. The constancy in size of these lesions could be verified on autopsy material as well as in the patient while testing on the pericranium with the different calibrations. The size of the lesion however did not matter with reference to the calculations used in research because whenever an effect was produced the point was assumed to be the centre of the electrode in known position. The effect produced with this gradual process of heating was therefore almost identical with that described by Cooper (1962) for cooling and was achieved with much less technical complexity and cost.

(g) Stimulation Studies

In the first two hundred operations, stimulation was used to help localisation of the position of the electrode with reference to intracerebral structures. The electrode was designed in such a manner that bipolar stimulation between two points at the tip of the electrode 0.5 mm. apart could be done and the same electrode at the same position could be used for coagulation for a length of 4 mm. around its tip. A Siemens Edison Swan stimulator was used which could deliver 'single shocks' of variable voltage from 0.5 to 12 during a variable time of 0.5 to 10 seconds. Although such stimulation was in general helpful, it could not be fully relied upon for the following reasons. In some patients, whatever strength of current was used, no response was obtained in any position of the electrode. Subsequent coagulation, however, sometimes produced motor or sensory deficits. In some patients the same response, for example, tingling in the face, was seen over a fairly long distance of the electrode track. This was presumably due to leakage of current along a preferential pathway. Therefore, from the point of view of localisation for research purposes, the methods of deep recording, biopsy and creation of reversible and irreversible lesions were better since they gave the required information in almost 100% of the cases while stimulation was successful only in about 60% of cases. When stimulation was successful, however, it gave accurate localisation and was dependable.

(h) Value of Scattergrams

Despite such accurate measurement, corrections and

charting of lesions, general conclusions could not be drawn because of the unknown factor of individual anatomical variation between the patient's brain and the atlas brain. This factor could be overcome in one of two ways.

The first method used successfully by Hughes (1961) was to pool the information available from several atlases and brains and to refer the patient's lesion to this standardised data. Guiot (1961) and Van Buren and Maccubbin (1962) used similar methods.

The second method is identical in principle but is the reverse of the first. This was to pool the information from several lesions and to relate it to a standard diagram. This method of scattergram was used as the main method in this work.

It is interesting to note that some of the classical work in neurology has been done by the method of scattergram under similar circumstances. It is particularly applicable when the 'lesion' producing a neurological phenomenon is quite large in relation to the exact minimal neural tissue damage required to produce such an effect and the individual variation in the shape and size of the neural structures between individuals necessitates the reference to a standard diagram. Russell and Espir (1961) in their work on traumatic aphasia from war injuries of the brain used this method. Holmes (1918) and Holmes and Lister (1916) in their classical work on disturbances of vision by cerebral lesions followed the same technique. Hess (1957) in his Nobel prize winning work on the diencephalon applied the

same principle in some of his studies.

(i) Survey of Clinical Material

The present study is concerned with 334 patients who underwent 449 second stage stereotaxic operations by the Guiot-Gillingham method. A classification by diagnosis is shown below.

Parkinsonism	301 cases
Dyskinesias	23 cases
Pain	3 cases
Mental Disorders	7 cases

The cases of dyskinesias were a mixed group. They included the following syndromes.

Torsion Dystonia	8 cases
Choreoathetosis	3 cases
Familial Tremor	2 cases
Wilson's Disease	2 cases
Spasmodic Torticollis	2 cases
Disseminated Sclerosis	2 cases
Stiff Man Syndrome	1 case
Mixed Syndromes	3 cases

The cases of Parkinsonism are classified by age, sex and handedness below.

Sex	Total No.	Left handedness	Age at the time of the first stereotaxic operation			
			60 and above	50-59	40-49	39 and below
Male	185	9	48	101	29	7
Female	116	4	47	47	19	3
Total	301	13	95	148	48	10

Of the total number, 175 were investigated with lumbar pneumoencephalography and the rest with myodil ventriculography, prior to the second stage stereotaxic procedure.

In 68 of these patients, the disease could be attributed to a post-encephalitic basis.

294 of the 301 patients suffered from tremor while 298 showed rigidity.

Moderate degree of involvement of both sides was present in 241 patients. In 29, the disease was predominantly right sided and in 31 the left side was mainly affected.

Thirty patients suffered from oculogyric crises. Speech was involved in 228 cases. Dribbling of saliva occurred in 120 patients.

Only 28 patients of the 300, were free from manifestations of Parkinsonism other than tremor and rigidity.

In addition to these 445 lesions, 30 other lesions were studied only during the operation and by subsequent charting. They were not followed up closely on postoperative or longterm assessment. Figures from these 30 cases are included only with reference to data elicited during the operation.

(j) Method of Follow-up

All these patients were examined by a senior member of the staff at least one month after the stereotaxic procedure. Apart from a few patients who came from abroad, everyone was again assessed as an outpatient three to six months after the operation. Some of them were readmitted for detailed assessments. In a considerable proportion of cases, repeated follow-up examinations were done. In

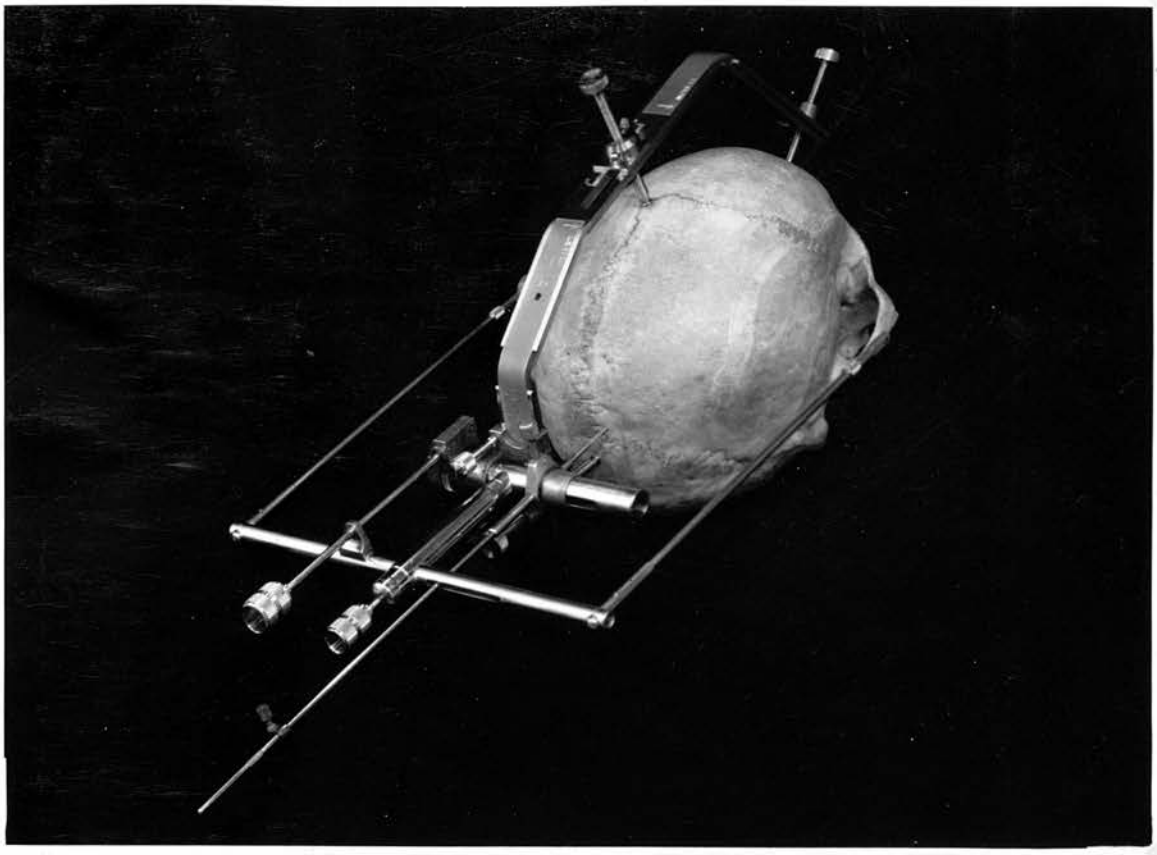
addition, a follow-up form (see Appendix B) was sent to the patient, the patient's near relative and the general practitioner at least six months after the operation. The combined information from all these sources was taken in assessing the effects of treatment. The non-response rate was very low and the number of patients on whom there was not reliable information on a six month follow-up was only one whose post-operative address could not be traced.

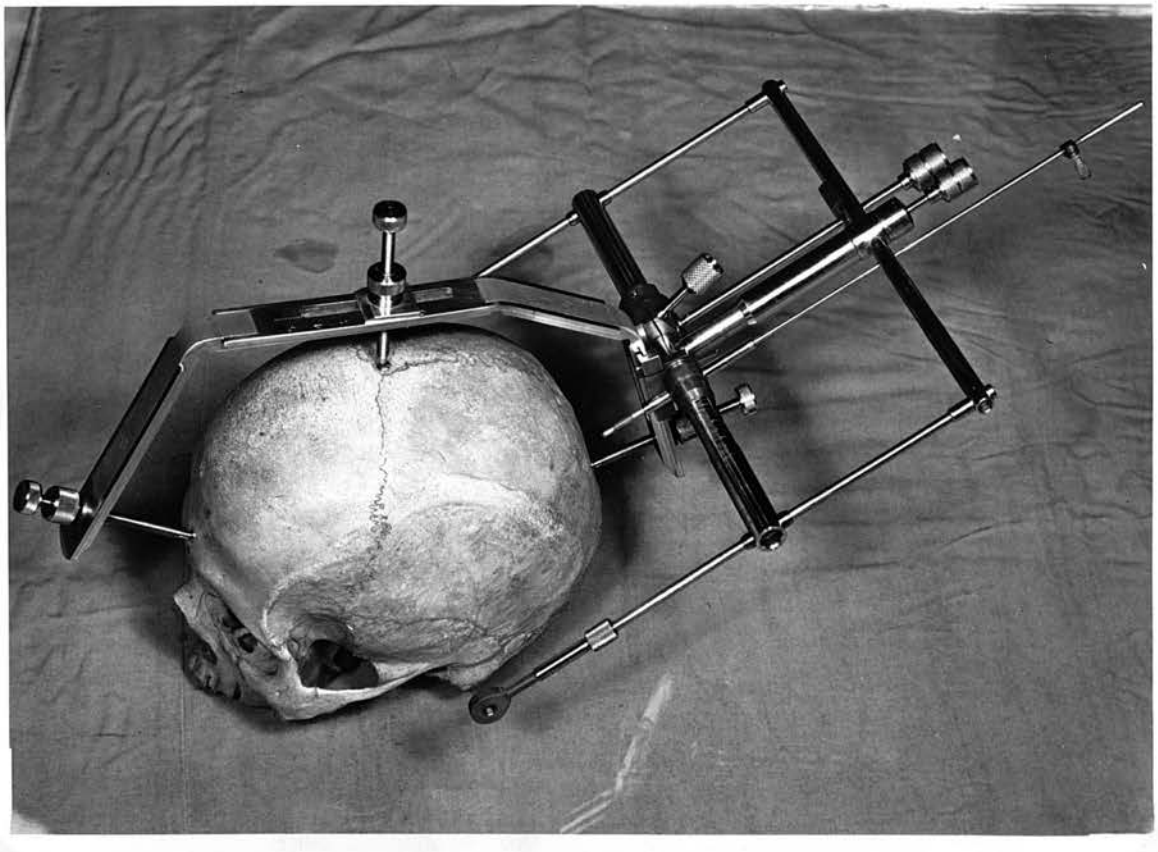
(k) Summary

The various methods of study of the internal capsule and adjacent diencephalon during human stereotaxic surgery were outlined. The radiological, surgical and electrophysiological procedures were described in detail. The methods of clinical follow-up were given.

For extreme degrees of accuracy, the method of depth electrical recording was to be preferred. Stereotaxic biopsy was also quite reliable and useful.

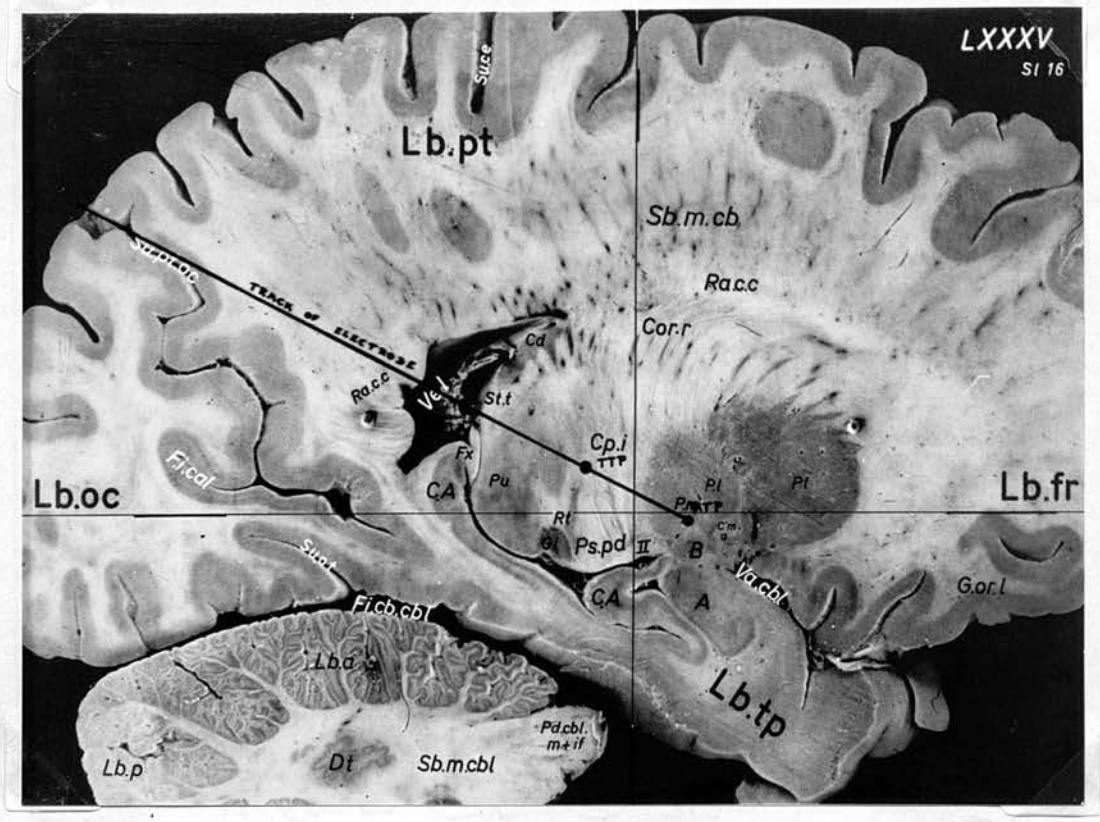
In view of the individual variations, the importance of drawing conclusions only from statistical analysis and scattergrams was stressed.

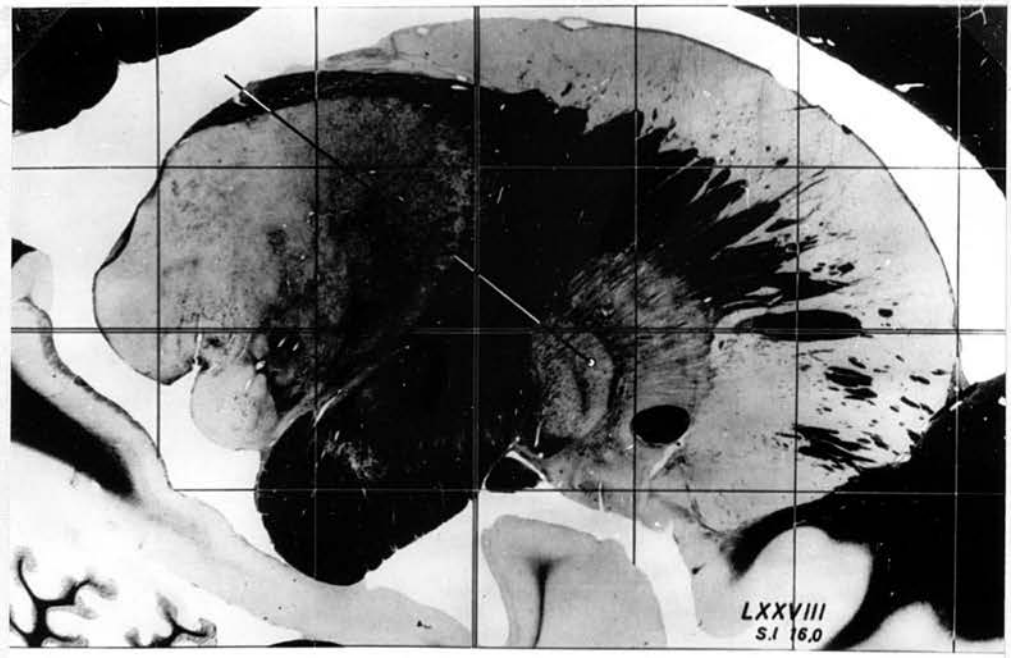




Two views of the Guiot-Gillingham stereotaxic machine.

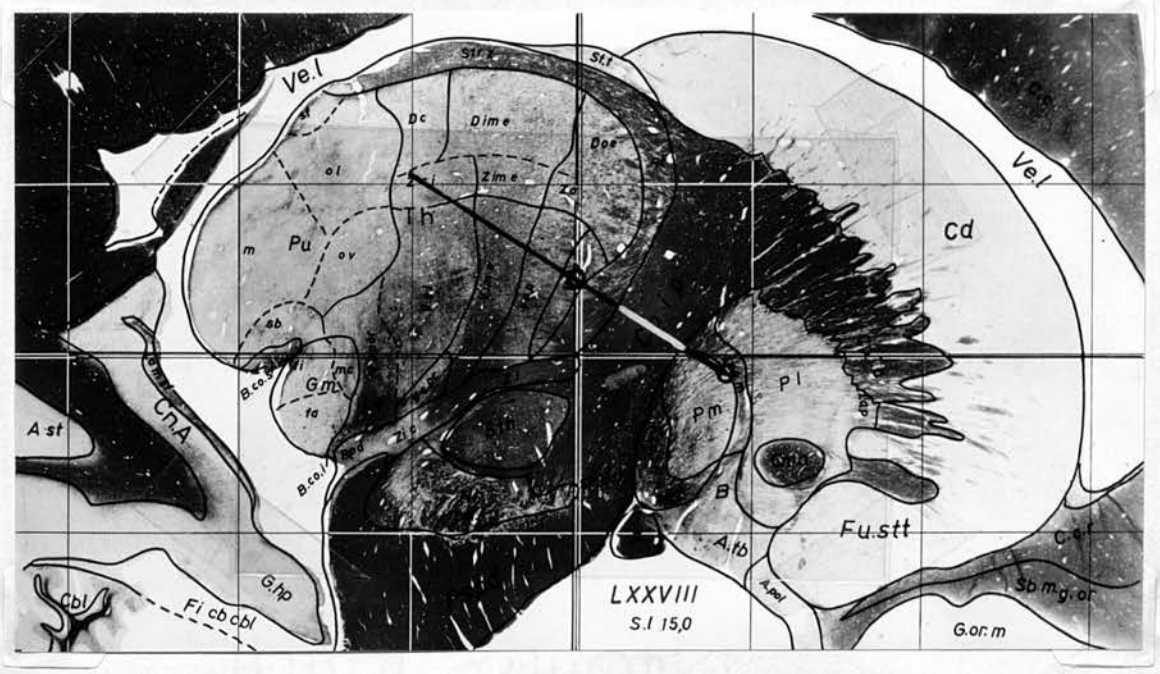
The machine in the lower figure is the latest model.

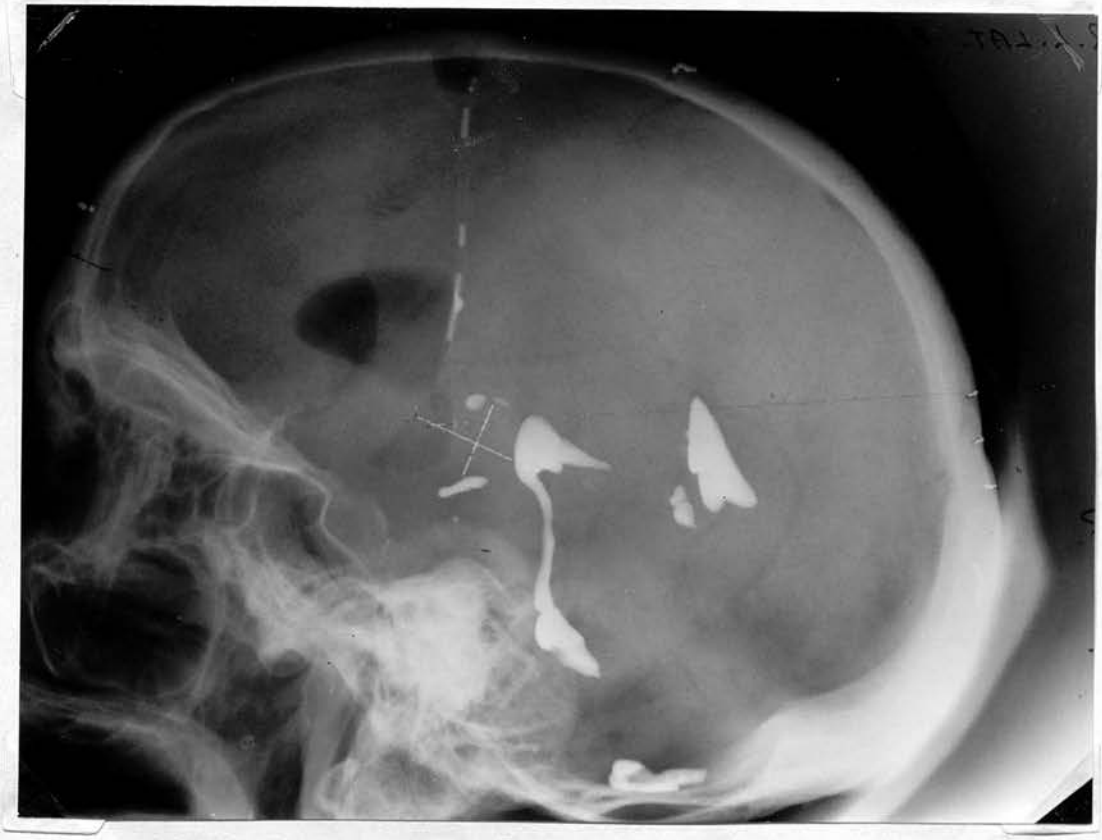




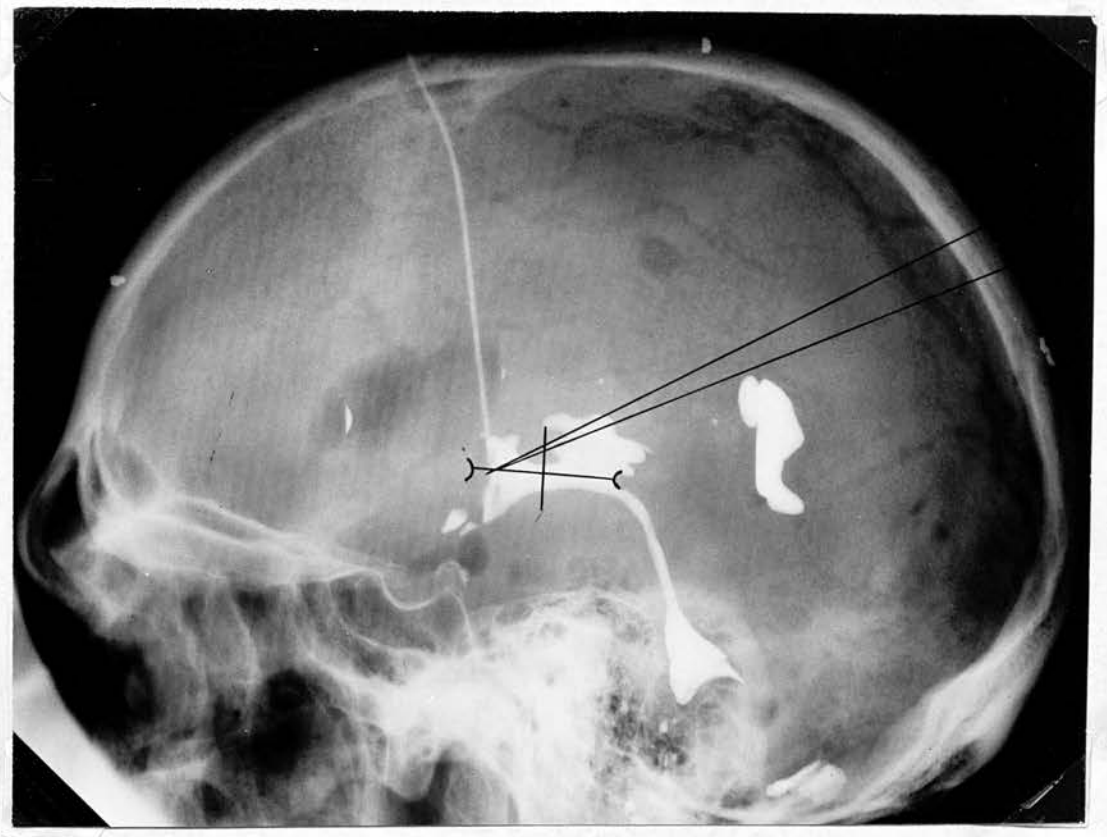
Sagittal section (6X) 16 mm. from the midline and parallel to it, showing the track of the electrode passing to its target in the globus pallidus through the thalamus and internal capsule.

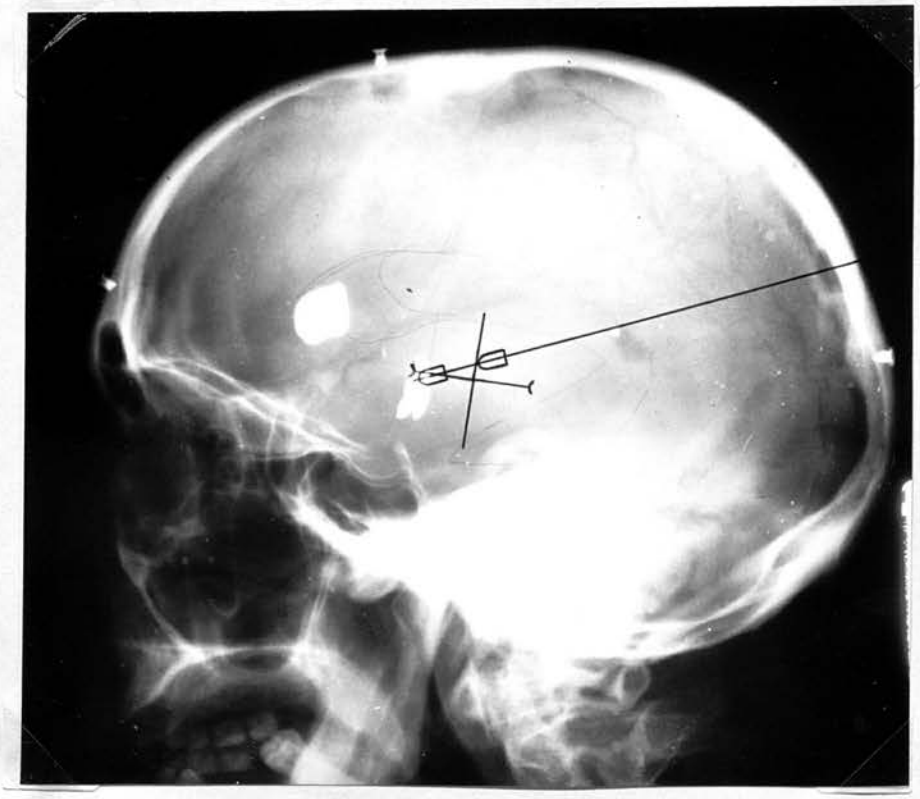
Track of electrode marked on stereotaxic atlas of Schaltenbrand and Bailey.
Above: Plate 16 Sl 16. Below: Plate 48 Sl 16.
TTP - Thalamic Target Point. PTP - Pallidal Target Point.





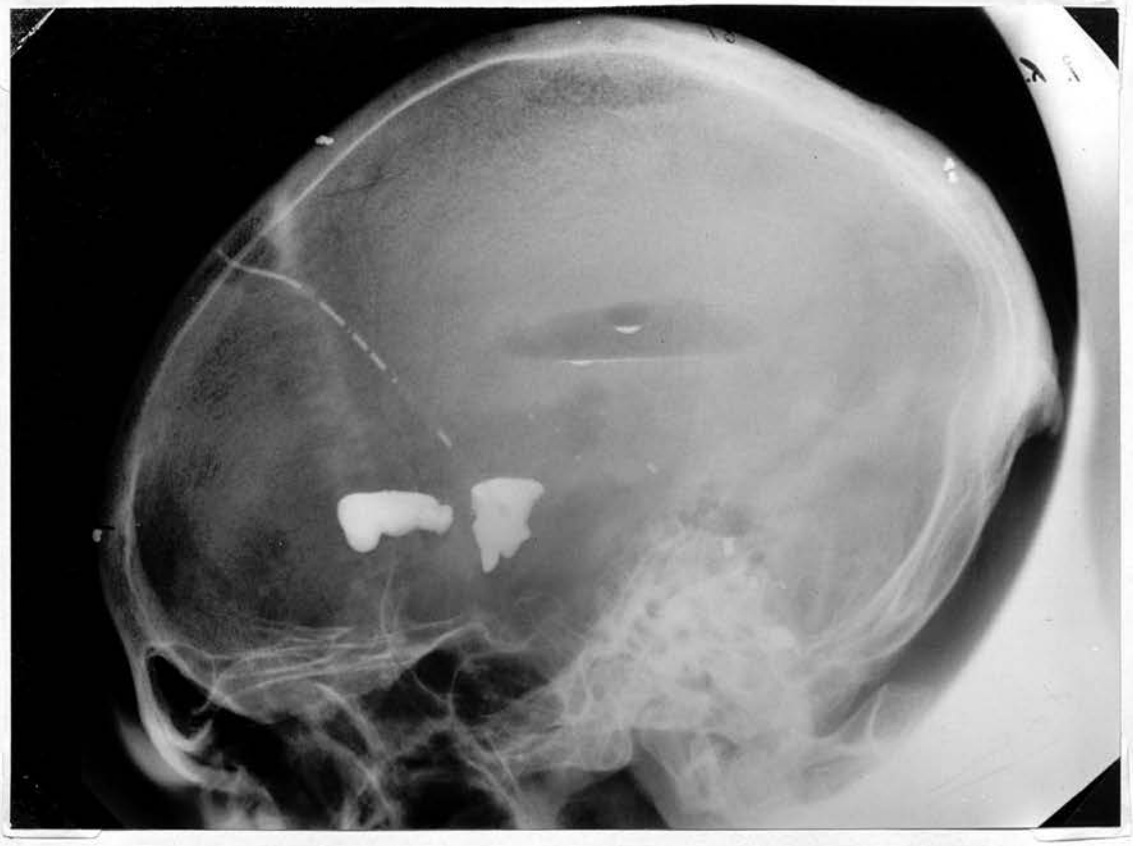
Track of electrode. Above: On Plate 47 Sl 15 of stereotaxic atlas.
Below: On X-ray showing commissures outlined by air and myodil.

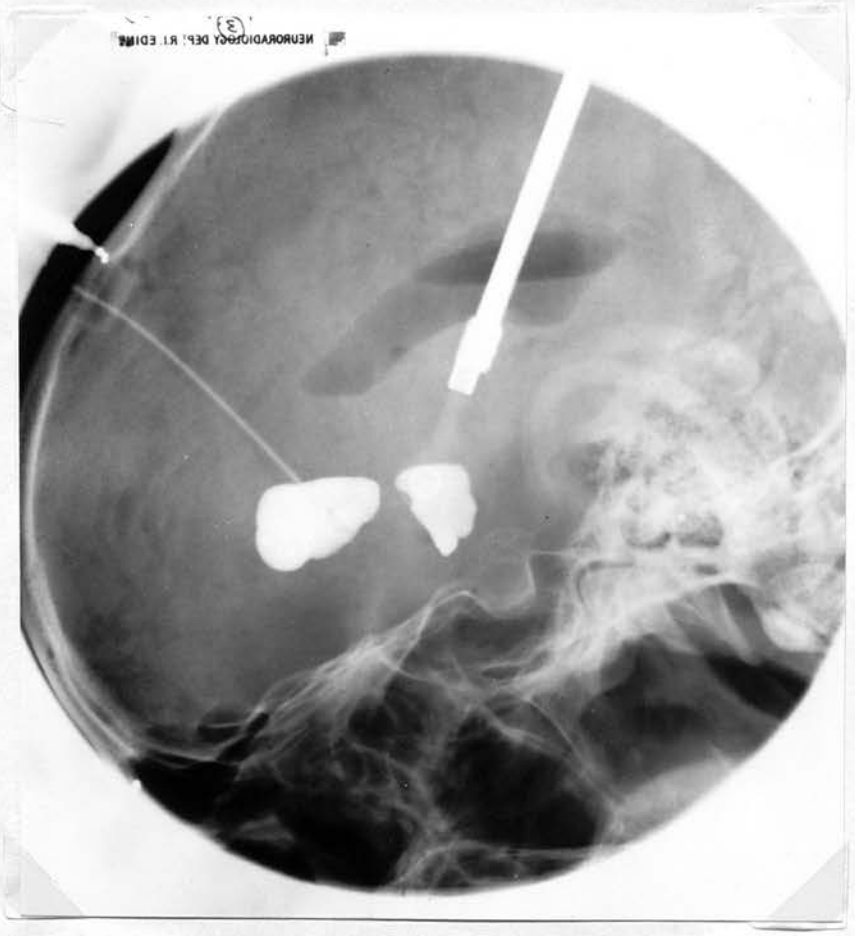




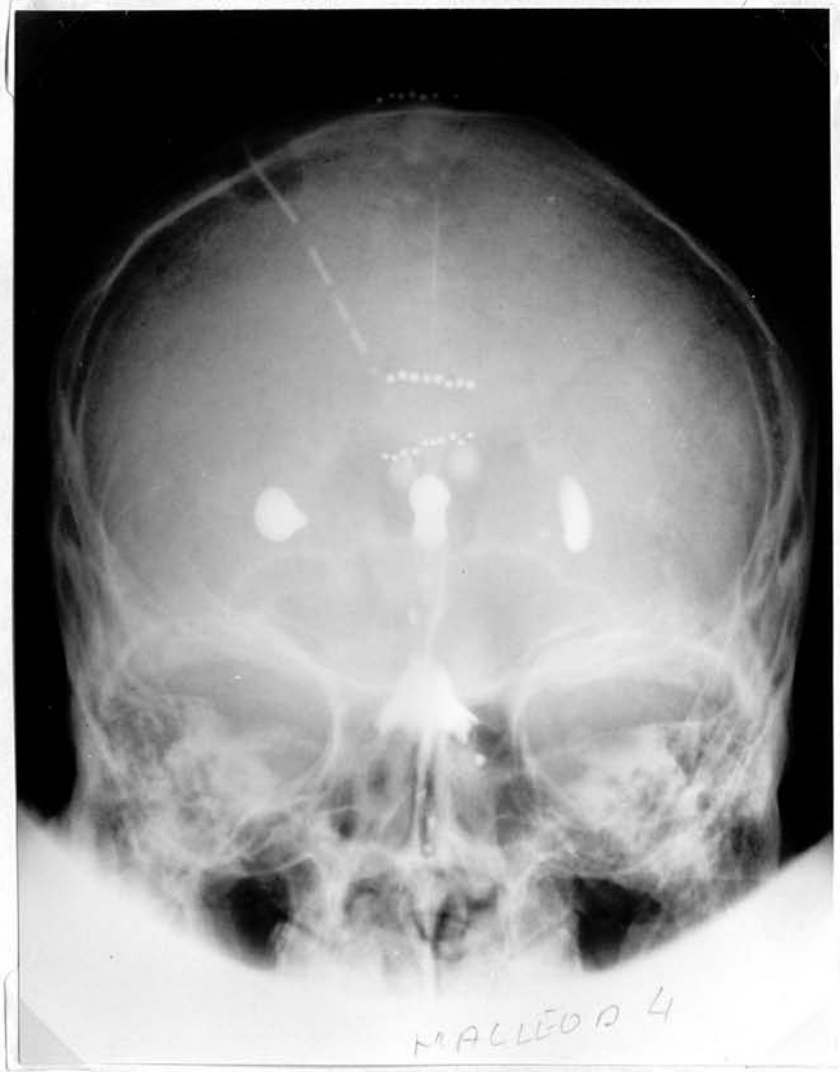
Above: Two tracks of electrode on X-ray showing CP outlined by myodil.

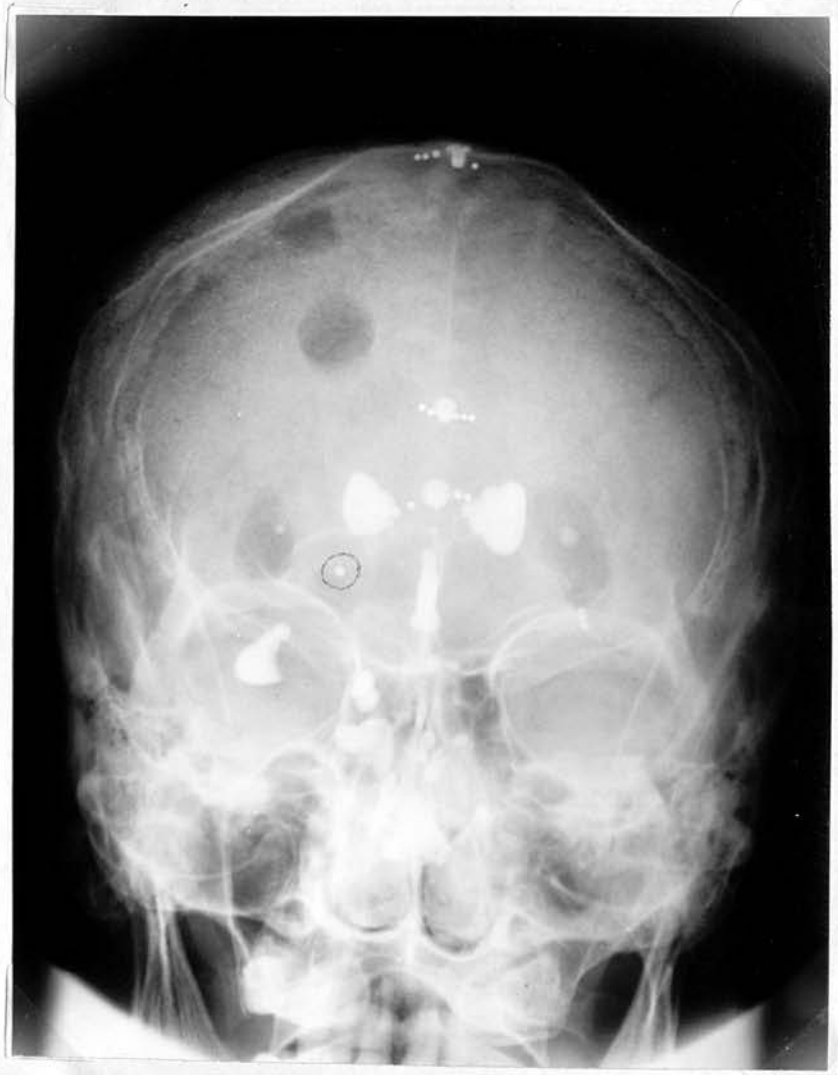
Below: Track of electrode on X-ray showing CA outlined by myodil.
Thalamic and pallidal lesions are also marked.



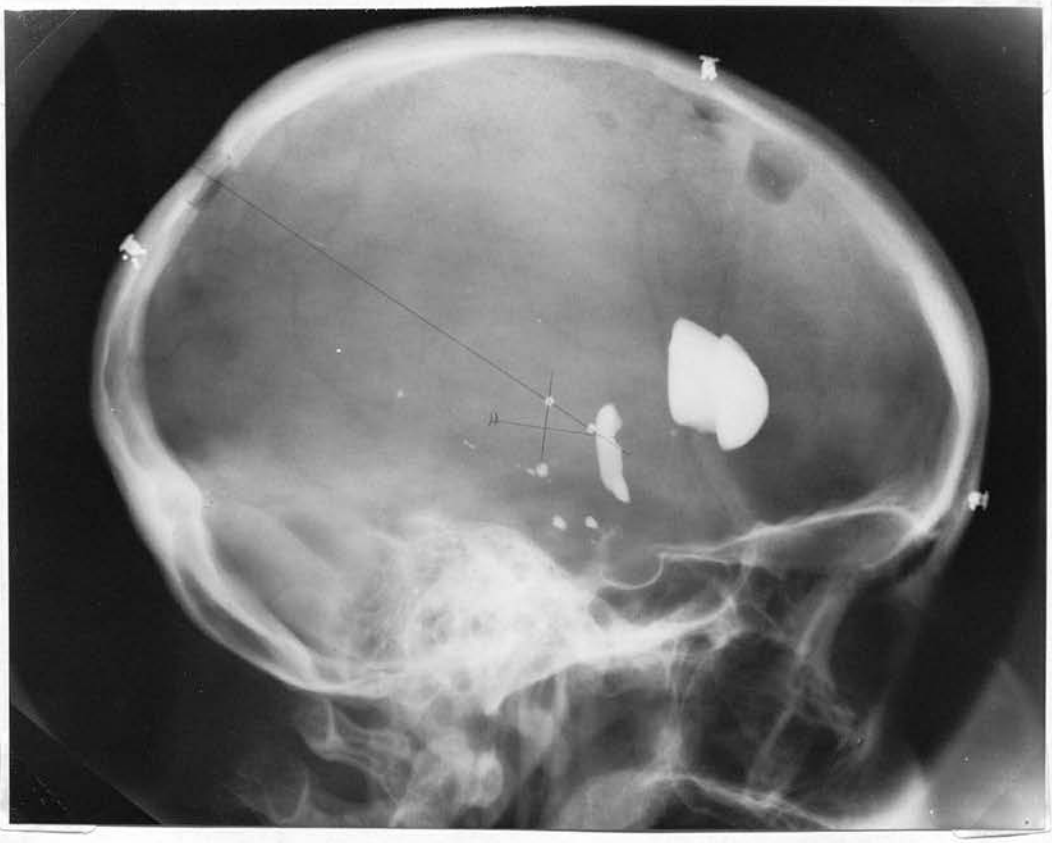


Above: Myodil outlining CA on X-ray.
Below: Sights of stereotaxic frame aligned against pallidal target using image intensifier. Myodil outlining CA.



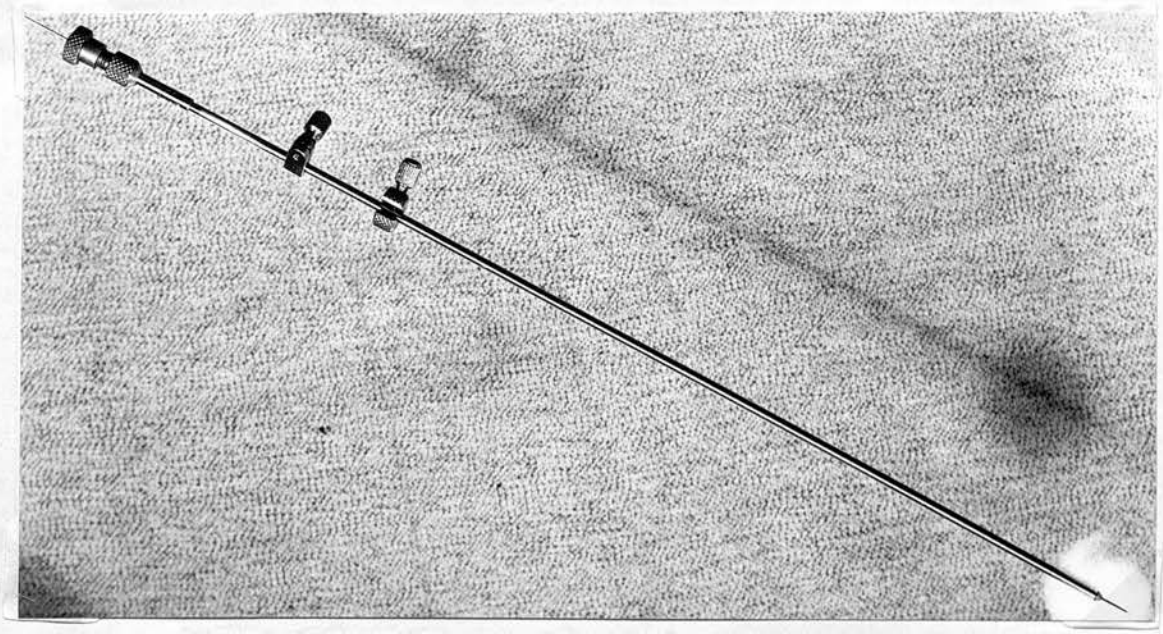


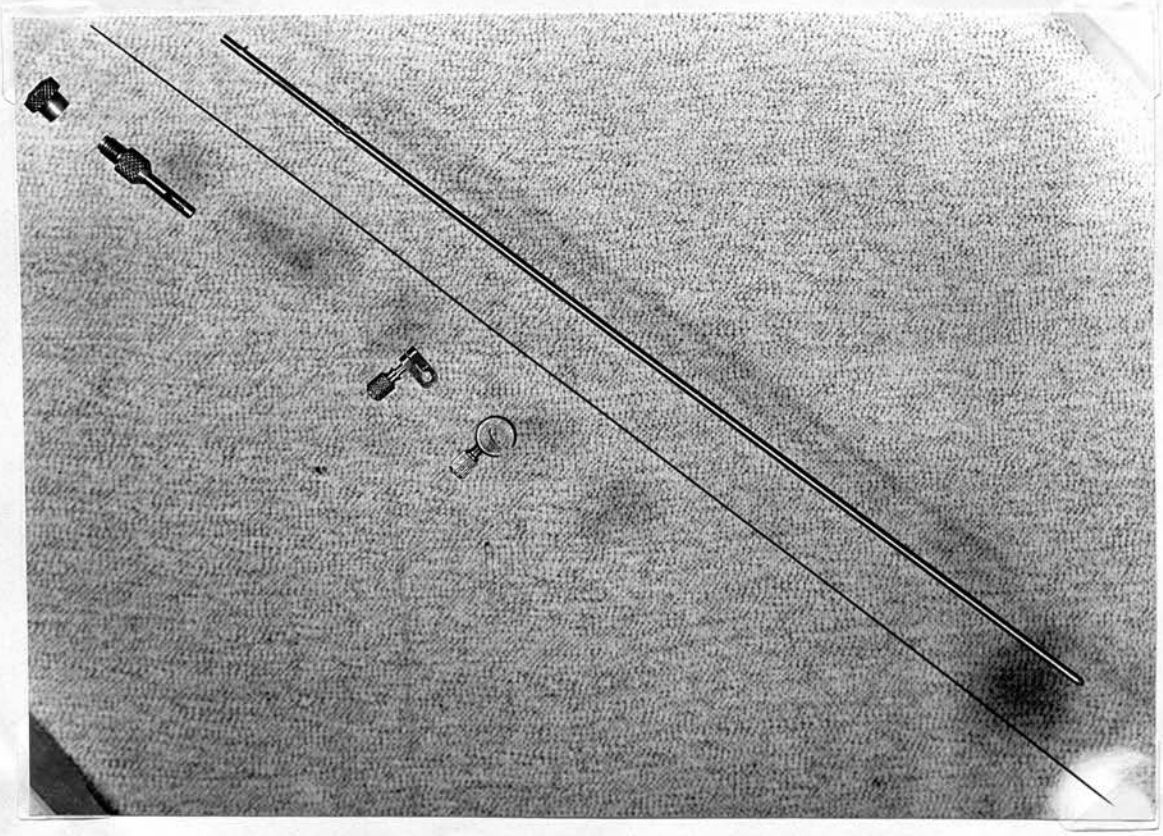
Left: Myodil and air ventriculogram to determine midline markers
Right: Opaque marker at pallidal target seen in postoperative X-ray (encircled by ring)



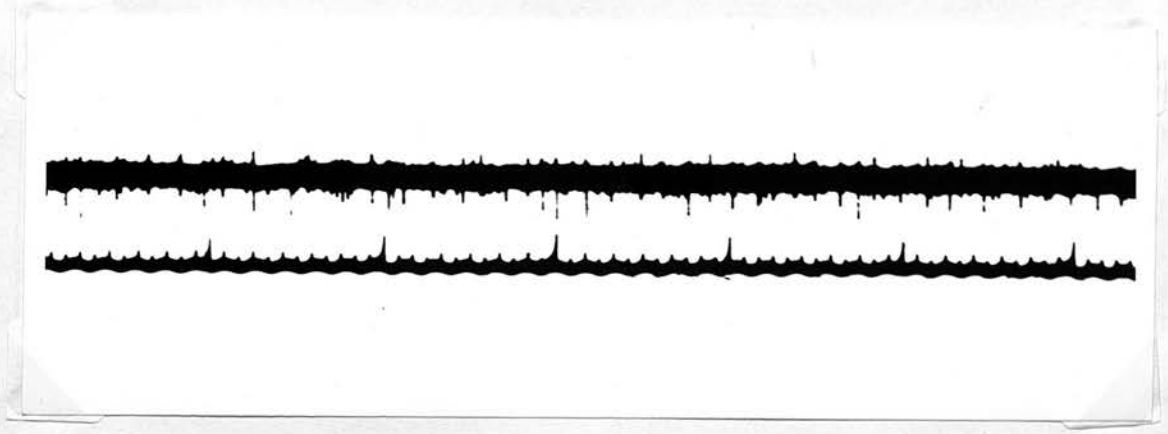
Above: Lateral view showing two markers at most anterior sites of coagulation in a bilaterally operated case; one marker in capsule and the other in thalamus.

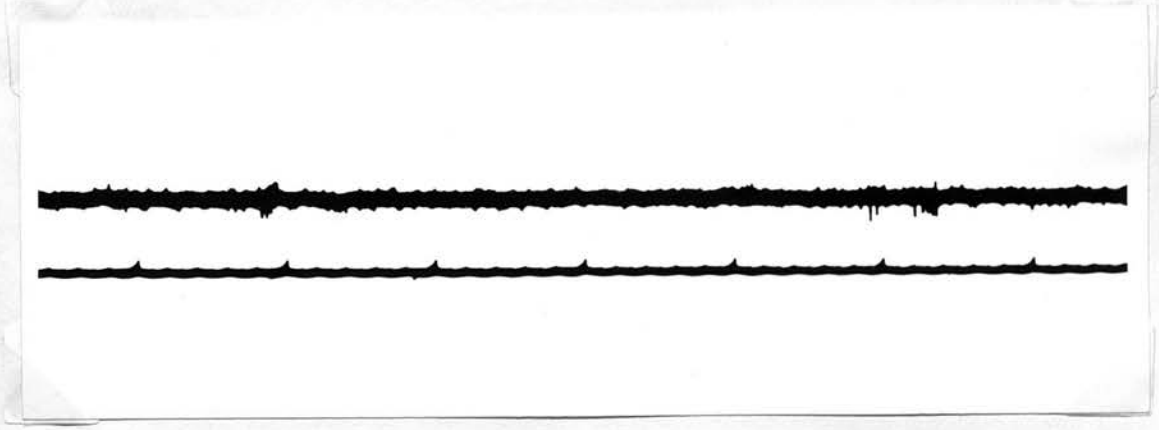
Below: Microscopic appearance of stereotaxic coagulation lesion by diathermy showing the well circumscribed discrete nature of lesion.





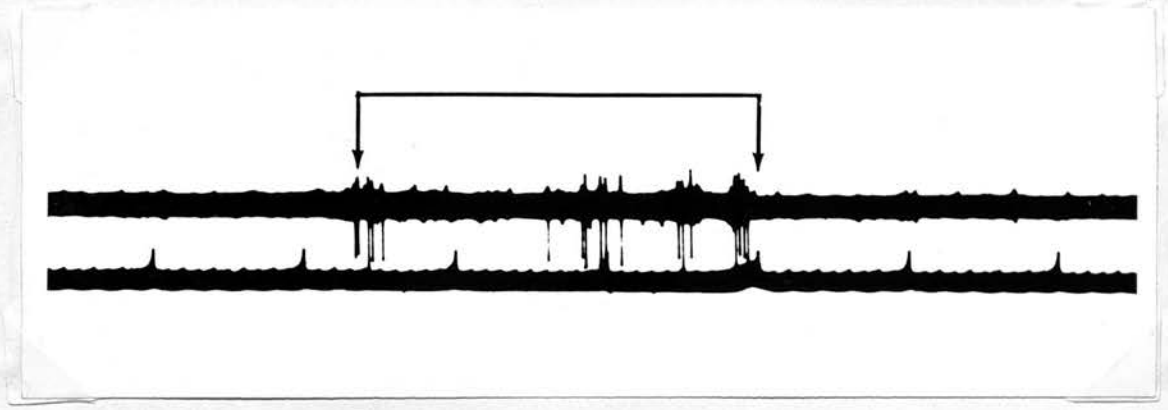
Above: Recording Electrode assembled.
Below: Recording Electrode dismantled.





(The lower trace in each photograph is the time mark in 100 and 10 msec.)

Photographs of oscillographic records during depth electrical recording with audiomonitoring: Above: Spontaneous cellular activity in thalamus
Below: Absence of cellular activity in internal capsule.

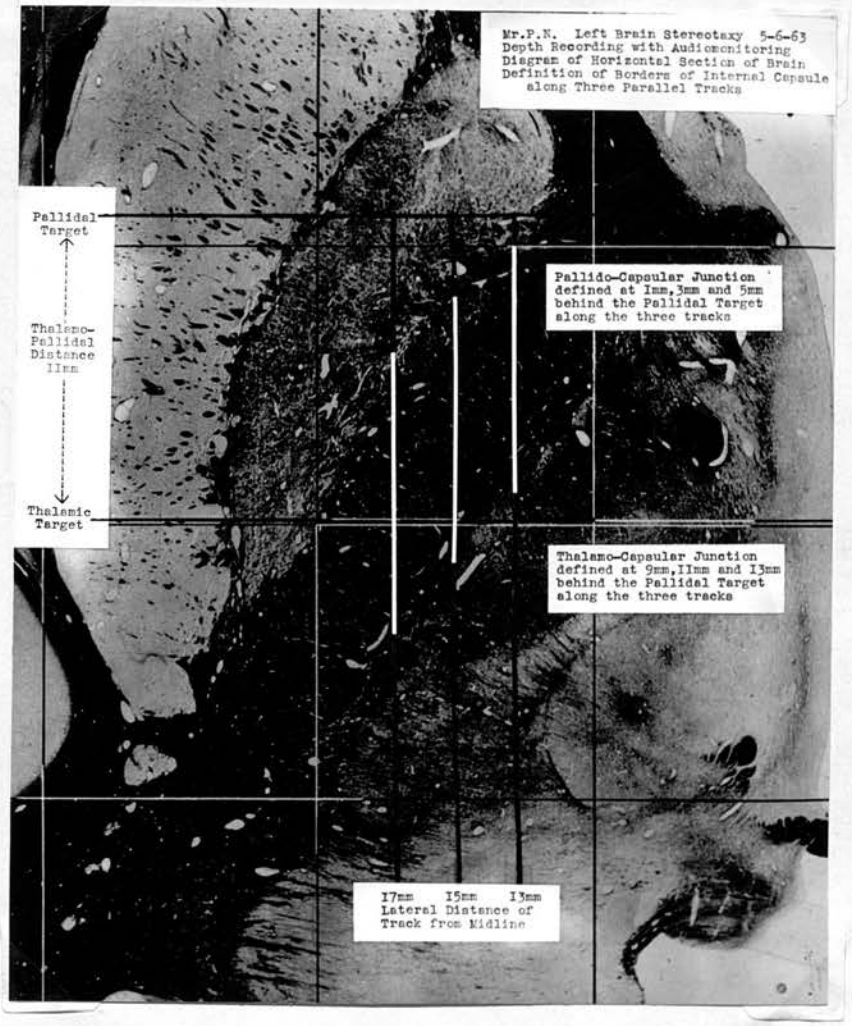


(The lower trace in each photograph is the time mark in 100 and 10 msec.)

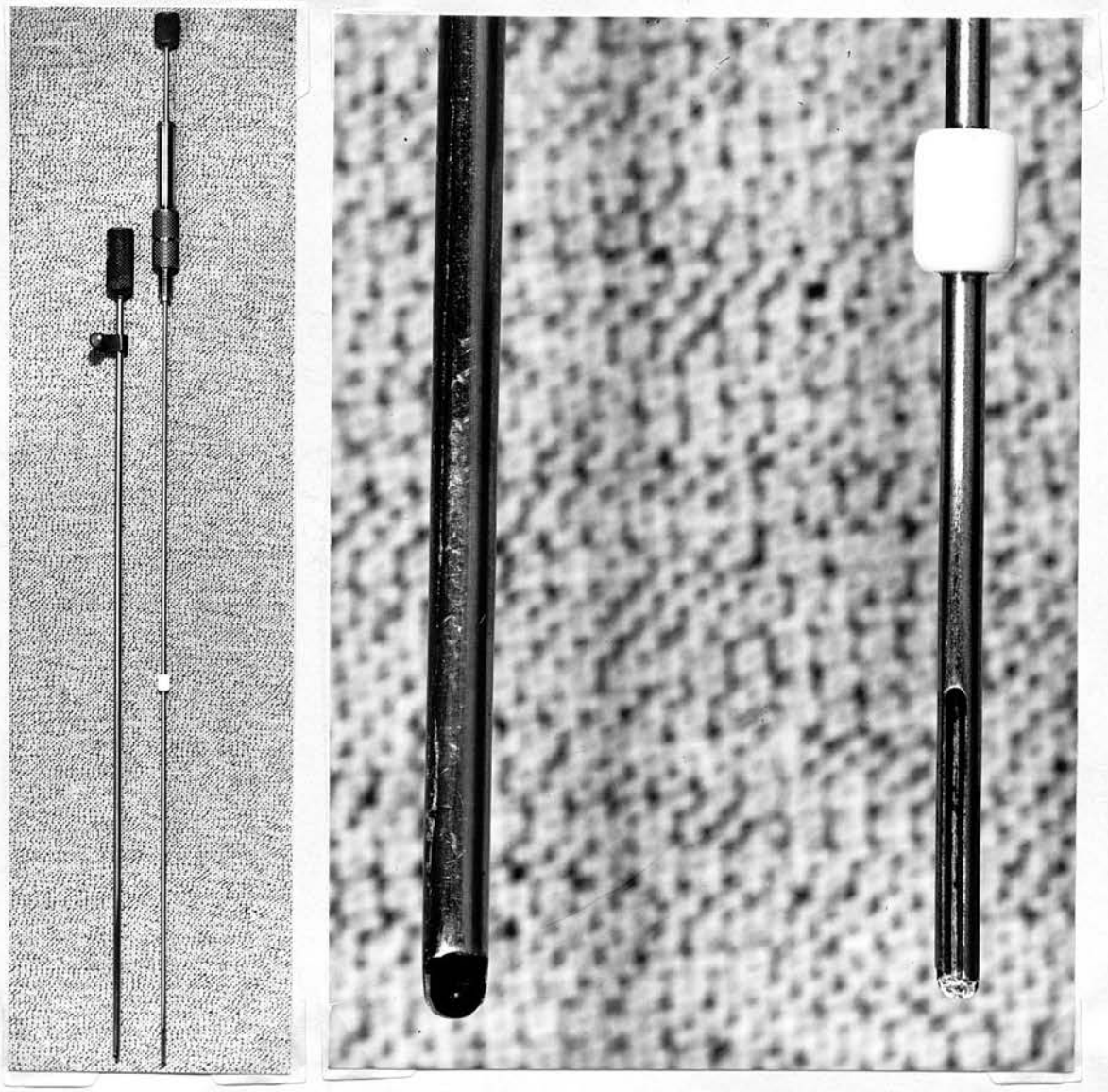
Photographs of oscillographic records during depth electrical recording with audiomonitoring from sensory relay nucleus.

Above: Evoked potentials from tapping on hand

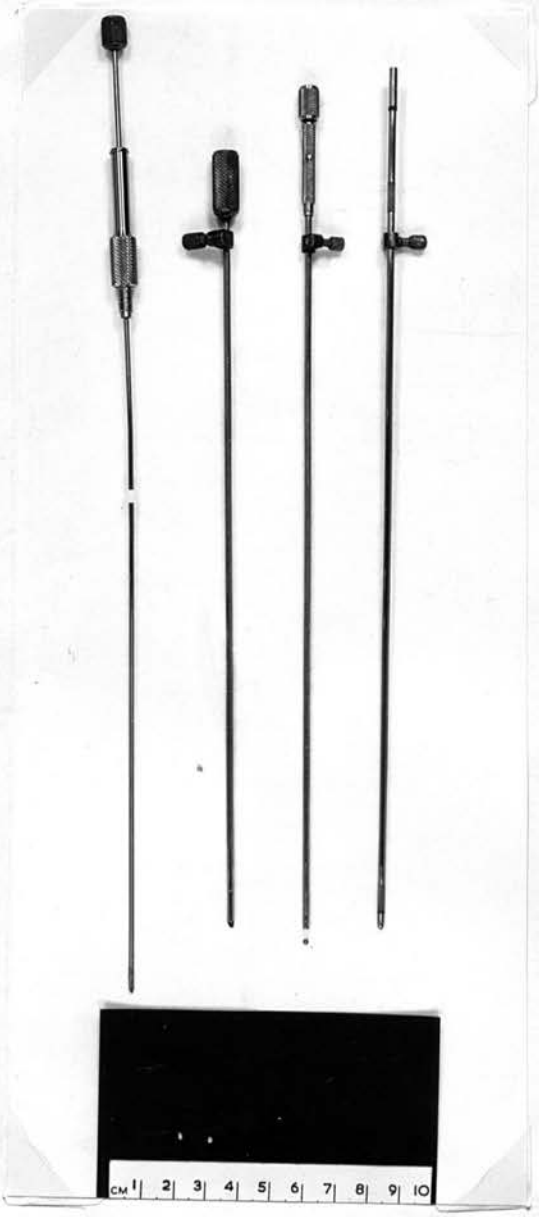
Below: Inhibition of spontaneous activity by tapping on face.



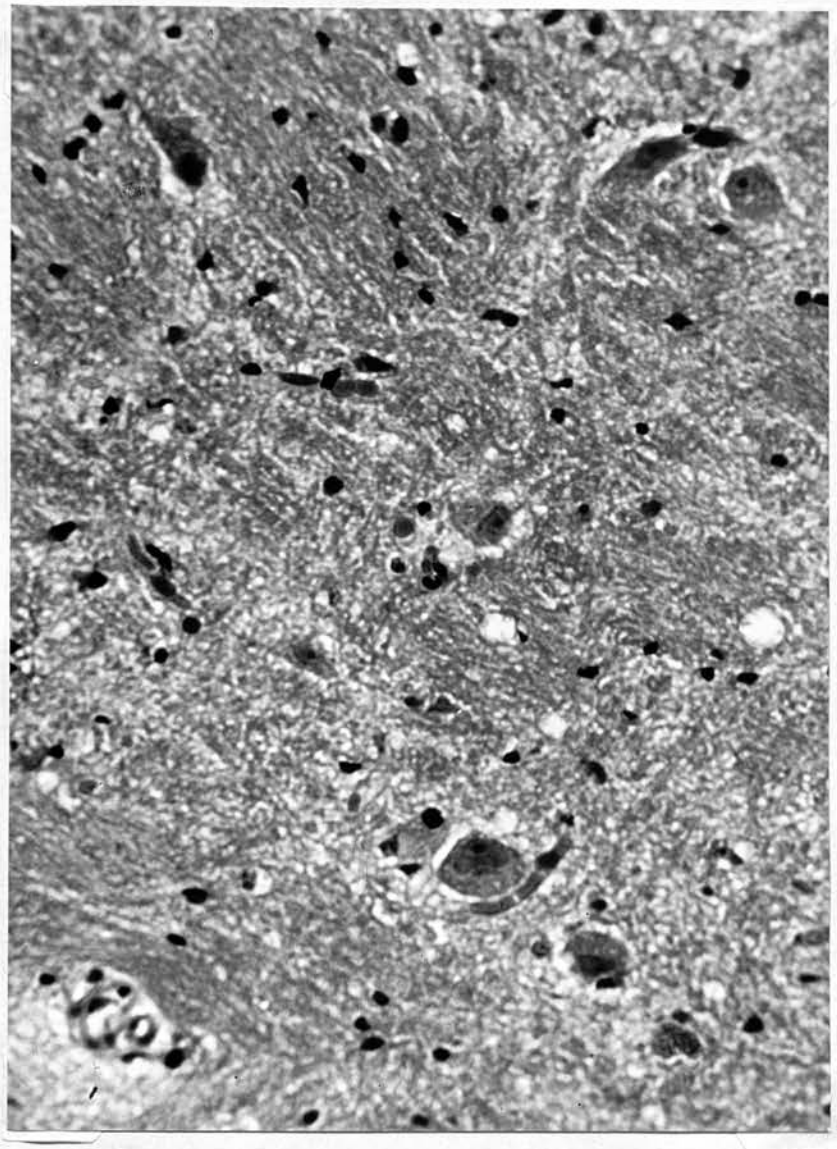
Three parallel electrode tracks defined by audiomonitoring.
Chart superimposed on Plate from stereotaxic atlas (diagrammatic).



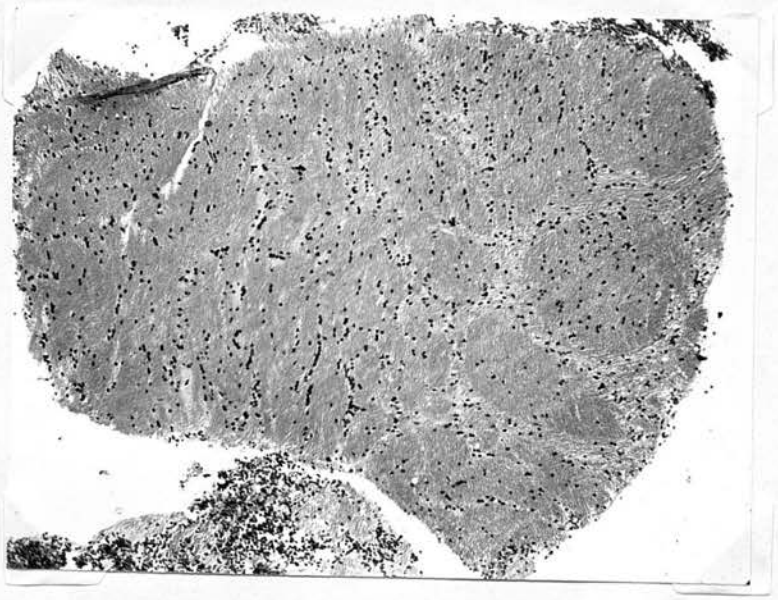
Left: Biopsy forceps dismantled.
Right: Operating end of components of biopsy forceps.

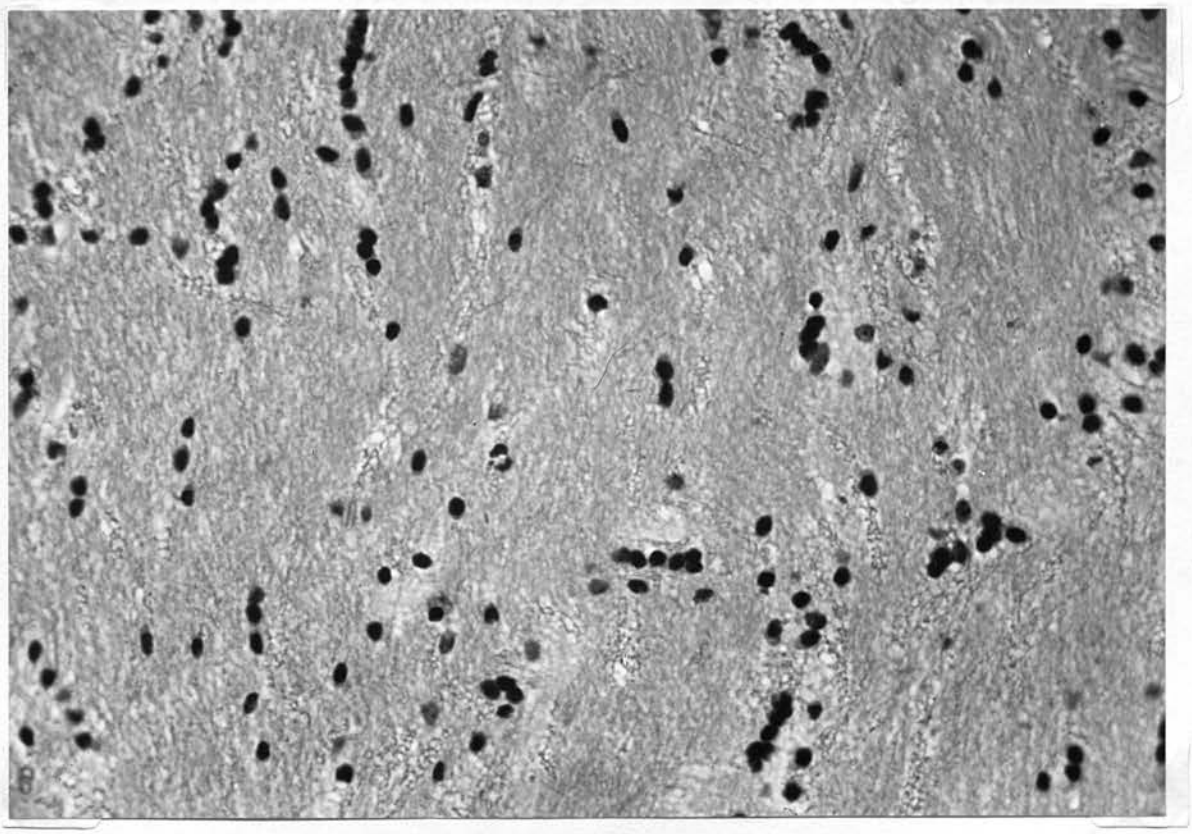


Left: Biopsy forceps assembled.
Right: Biopsy forceps, opaque marker carrier and coagulating electrode. Two pieces of biopsy are shown against the black background above the scale.



High Power view of stereotaxic biopsy from grey matter showing ganglion cells.





Stereotaxic Biopsy of White matter. Above: Low Power Below: High Power No ganglion cells seen

REFERENCES

1. Albe-Fessard, D., Arfel, G., Guiot, G., Hardy, J., Vourc'h, G., Hertzog, E., Aleonard, P. and Derome, P. (1962). Rev. Neurol., 106, 89.

2. Bailey, P. and Stein, S.W. (1951). Studies in Medicine. C.C. Thomas, Springfield, Illinois, 40.

3. Bertrand, C.M. (1958). J. Neurosurg., 15, 251.

4. Brown, A.J. (1963). Second European Congress of Neurological Surgery, Rome, 1963. Excerpta Medica International Congress Series No. 60, 114.

5. Cooper, I.S. (1955). Science, 121, 217.

6. Cooper, I.S. (1962). J. Am. M. Ass., 181, 600.

7. Delmas, A. and Pertuiset, B. (1959). Craniocerebral Topometry in Man. Blackwell Scientific Publications, Oxford.

8. Donaldson, A.A. and Gillingham, F.J. (1960). Brit. J. Radiol., 33, 157.

9. Ervin, F.R. and Mark, V.H. (1960). Arch. Neurol., 3, 368.

10. Gaze, R.M. and Gordon, G. (1952). J. Physiol., 118, 48P.

11. Gaze, R.M. and Gordon, G. (1954). Quart. J. exp. Physiol., 39, 279.

12. Gaze, R.M. and Gordon, G. (1955). Quart. J. exp. Physiol., 40, 187.

13.Gillingham, F.J., Watson, W.S., Donaldson, A.A. and Naughton, J.A.L. (1960). Brit. med. J., 2, 1395.

14.Guiot, G. (1958). Neurochirurgia (Stuttgart), I, 94.

15.Guiot, G., Brion, S. and Akerman, M. (1961). Ann. Chir., 15, 557; 703.

16.Guiot, G., Albe-Fessard, D., Arfel, G., Hardy, J., Hertzog, E., Vourc'h, G., Derome, P. and Aleonard, P. (1962). Rev. Neurol., 107, 84.

17.Guiot, G. (1963). Personal Communication.

18.Hankinson, J., Pearce, G.W. and Rowbotham, G.F. (1960). J. Neurol. Neurosurg. and Psychiat., 23, 352.

19.Heath, R.G., John, S. and Foss, O. (1961). Arch. Neurol., 4, 291.

20.Hess, W.R. (1957). The Functional Organisation of the Diencephalon. Grune and Stratton, London.

21.Hider, C. and Kalyanaraman, S. (1963). Unpublished information.

22.Holmes, G. (1918). Brit. J. Ophthal., 2, 353.

23.Holmes, G. and Lister, W.T. (1916). Brain, 39, 34.

24.Horsley, V. and Clarke, R.H. (1908). Brain, 31, 45.

25.Housepian, E.M. and Pool, J.L. (1960). J. Nerv. & Ment. Dis., 130, 520.

26.Housepian, E.M. and Pool, J.L. (1961). First International Symposium on Stereoencephalotomy. Philadelphia 1961. S. Karger, New York, 1962. 171. Confin. Neurol., 22,171, 1962.

27. Hughes, B. (1961). In Carling, E.R. and Ross, J.P. Ed. British Surgical Practice, Surgical Progress 1961. Butterworth, London 129.

28. Hughes, B. (1961). Second International Congress of Neurological Surgery. Washington, 1961. Excerpta Medica International Congress Series No. 36, E 88.

29. Jinnai, D., Nishimoto, A., Matsumoto, K. and Handa, S. (1961). Second International Congress of Neurological Surgery. Washington 1961. Excerpta Medica International Congress Series No. 36, E 94.

30. Laksell, L. (1949). Acta. chir. scand., 99, 229.

31. McCaul, I.R. (1959). J. Neurol. Neurosurg. Psychiat., 212, 109.

32. Maloney, A.F.J. (1960). Personal Communication.

33. Mark, V.H., McPherson, P.A. and Sweet, W.H. (1954). Amer. J. Roentgenol., 71, 435.

34. Narabayashi, H., Okuma, T. and Shikiba, S. (1956). Arch. Neurol. & Psychiat., 75, 36.

35. Platt, R. quoted by Hill, A.B. Principles of Medical Statistics. Seventh Edition. The Lancet Limited, London, 1961. 9.

36. Riechert, T. and Mundinger, F. (1959). In Schaltenbrand, G. and Bailey, P. Ed. Introduction to Stereotaxis with an Atlas of the Human Brain. Thieme Stuttgart, 421.

37. Russell, W.R. and Espir, M.L.E. (1961). Traumatic Aphasia. Oxford University Press, London.

38. Sachs, B. quoted by Bing, R. and Wartenberg, R. in foreword to Haymaker, W. — Bing's Local Diagnosis in Neurological Diseases. H. Kimpton, London, 1956. 7.

39. Schaltenbrand, G. (1959). — In Schaltenbrand, G. and Bailey, P. Ed. Introduction to Stereotaxis with an Atlas of the Human Brain. Thieme, Stuttgart. 448.

40. Schaltenbrand, G. and Bailey, P. (1959). — Introduction to Stereotaxis with an Atlas of the Human Brain. Grune and Stratton, New York.

41. Spiegel, E.A., Wycis, H.T., Marks, M. and Lee, A.J. (1947). — Science, 106, 349.

42. Spiegel, E.A. and Wycis, H.T. (1952). — Stereoencephalotomy (Thalamotomy and Related Procedures) Part I. Methods and Stereotaxic Atlas of the Human Brain. Grune and Stratton, New York.

43. Spiegel, E.A. and Wycis, H.T. (1961). — In Sheer, D.E. Ed. Electrical Stimulation of the Brain, Austin, University of Texas Press. 487.

44. Talairach, J., de Ajuriaguerra, J. and David, M. (1952). — Presse med., 60, 605.

45. Talairach, J., David, M., Tournoux, P., Corredor, H. and Kvasina, T. (1957). — Atlas D'Anatomie Stereotaxique. Masson & Cie. Paris.

46. Van Buren, J.M. and Maccubbin, D.A. (1962). — J. Neurosurg., 19, 811.

47. Vourc'h, G., Hardy, J. and Denavit, M. (1963). — Brit. J. Anaesth., 35, 208.

C H A P T E R 3

INDIVIDUAL VARIATIONS

"All science is measurement"

.... Helmholtz.

"All true measurement is essentially comparative"

.... Sir Henry Dale.

(a) Radiological Magnification and Distortion

All methods of stereotaxy face this problem. They depend on the outlining of the ventricular system by air or myodil (or similar form of contrast) and calculation of target co-ordinates from reference points in relation to the ventricular system and the instrument.

In the Guiot-Gillingham method, the distortion due to divergent beams is practically eliminated. This is achieved by keeping the structures needing to be outlined like the anterior commissure always in the centre of the radiological field. No calibrations of the instrument are included in the X-ray so that there is no problem of increasing magnification as the periphery of the film is approached. Leksell's spiral diagram is probably the most exact method of overcoming errors from this source but in view of the factors noted above, no such correction was needed.

With respect to small distances in the centre of the field, a constant magnification factor could be used for the correction of X-ray measurements to correspond to actual measurements, provided the tube-plate distance was kept constant. Again, almost all modern methods of stereotaxy incorporate this feature. Leksell included,

in his method, this principle. Others like Walsh (1962) felt that, even, then, the central beam had to be calculated afresh for each case, since the light weight metal beam could bend and produce distortions of the order of a mm. or two. In the method used in this project, the tube-plate distance was kept constant. On the other hand, the head was not rigidly fixed to the X-ray apparatus and this could have given rise to small variations in the magnification factor. There was also the possibilities of slight tilting of the head or its rotation.

It was decided to ignore these factors for the following two reasons. Firstly, even in cases where the stereotaxic frame is rigidly fixed to the skull, no allowance is made or can be made for differences in the sizes of the heads or ventricular systems, to produce constancy of distance between anatomical structures and the X-ray plate. Secondly, when obvious tilt, rotation or non-centering were eliminated, the variation in the magnification factor did not amount to more than 5% at any time. Taking an example, the upper limit of co-ordinate measurements, not corrected for atlas, used in this method was 10 mm. If the magnification was calculated wrongly in such a case as 10% instead of as 15%, the result would be 9 mm. instead of 8.5 mm. Therefore, the maximum possible theoretical error was of the order of 0.5 mm. The accuracy of measurement on the X-ray was only of the same order and hence such an error was not considered to be significant. The usual error was more likely to be of the order of 2% of 5 mm. or 0.1 mm. which was negligible.

The actual correction factor for magnification used in these cases was 10%. The best guide as to the exact magnification at the plane of the site of operation was calculated from cases where two markers were introduced simultaneously or within a very short interval in the same brain. In such cases the factors of instrumental or radiological variation were minimal.

The following measurements were taken from such cases.

No.	Nature of bilateral operation	Actual distance between tracks (in mms.)	Distance between the markers in post-operative X-ray (in mms)	Correction factor for magnification
1.	Thalamotomy	32.0	35.5	9.9%
2.	Leucotomy	34.0	38.0	10.5%
3.	Leucotomy	42.0	47.0	10.6%
4.	Pallidotomy	32.0	35.5	9.9%
5.	Pallidotomy	31.5	35.0	10.0%

These figures showed that for practical purposes, the correction factor used for X-ray magnification was sufficiently accurate.

The degree of possible error due to radiological magnification was further reduced in this project by two other methods. Firstly, all intercommissural distances were standardised to that of the atlas brain for purposes of calculation so that X-ray factors were fully eliminated in that co-ordinate. Secondly, conclusions were drawn only from scattergrams or statistical analyses. Thus the factors of individual variations were accounted for as much as possible.

(b) Variability of Intercommissural Distance

Calculations in this project depended on the

intercommissural distance as the horizontal antero-posterior axis. Either this line or the line joining the foramen of Monro with the posterior commissure is now used by the majority of stereotaxic surgeons as their antero-posterior reference line. Pineal calcification used by some in earlier days, has now been generally discarded in view of its unreliability of presence and inconstant position.

The line joining the posterior border of the anterior commissure with the anterior border of the posterior commissure in the lateral film was measured in 350 patients. The frequency distribution of this measurement was plotted from the data and the characteristics as regards the 'normality' of the series were studied.

Intercommissural distance in mms. uncorrected for X-ray magnification.	Number of cases
20	1
21	1
22	1
23	0
24	5
25	10
26	28
27	56
28	73
29	61
30	46
31	34
32	18
33	7
34	6
35	1
36	1
37	1

Mean	28.68 mm.
S.D.	2.238
C.V.	7.802
Standard error of the mean	0.12 mm.

These figures indicated that the population studied was as near a 'normal' one as might be expected in a biological series.

It was interesting to compare these measurements with similar figures of other authors. Allowing 10% magnification, the mean CA-CP length of the cases under study was 25.8 mm. Van Buren and Maccubin (1962) gave the measurements for 16 brains at autopsy with a mean of 23.5 mm. The range was 21.5 - 27 mm. They, however, measured the distance between the centres of the commissures and admitted that some distortion during fixation of the brain was possible. Spiegel and Wycis (1952) gave the range for autopsy brains as 23 to 29 mm. with an average of 25.2 mm. They also measured the distance between the centres of the commissures. Their figures for lateral Roentgenograms were : Range 22.5 to 28.5 mm. and average 24.8 mm. Their study was based on 30 brains at autopsy. Talairach (1955) in a study of 26 brains gave the range of the distance between the centres of the commissures as 23 - 28.5 mm. with an average of 25.5.mm. Amador, Blundell and Wahren (1959) in 31 brains studied at autopsy gave the range (border to border of commissures) as 21 - 25 mm. with an average of 23 mm. These figures were somewhat smaller than the ones derived in the present study. On the other hand Guiot (1963) felt that the average CA-CP length of French brains studied by him at autopsy was 25 - 26 mm.

In view of the variability of the CA-CP distance the immediate corollary which occurred to all stereotaxic surgeons was whether reduction of the measurements of the

co-ordinates of a target point to correspond to a standard CA-CP distance was likely to increase the accuracy of coincidence of the radiological target with the anatomical structure aimed at. Wahren and Braitenberg (1959) found a general reduction of scattering of more than 50% when the surface sulci were plotted in relation to the intercommissural system as compared with Reid's system. Talairach (1961) found as a result of the study of 130 undistorted brains that proportional correction of the anteroposterior co-ordinate of the thalamic target with respect to the intercommissural distance was relevant. Van Buren and Maccubbin (1961), on the other hand, did not feel that statistical analysis proved this to be true. Hughes (1963) as a result of the study of autopsy brains as well as information from several standard atlases felt that the scatter of the oral ventral nucleus of the thalamus was reduced if a proportionate correction for the CA-CP length was applied. Caracalos, Levita and Cooper (1962) also felt that correction of the anteroposterior co-ordinate for the CA-CP length increased the accuracy of charting on the atlas of Schaltenbrand and Bailey.

These differing opinions and statistical results made it necessary that the importance or otherwise of the correction factor should be established for this study. During depth electrical recording with audiomonitoring it was possible to define accurately the thalamocapsular junction in 22 cases and the pallidocapsular junction in 24 cases, as a basis for this particular part of the study. The lesions were charted on the atlas of Schaltenbrand and

Bailey and the co-ordinates of the thalamocapsular and pallidocapsular junctions were determined before and after proportionate reduction of the antero-posterior co-ordinate for the CA-CP length of the standard brain. In each case, the distance of this point from the corresponding junction in the atlas was measured. These paired observations were subjected to the student test and degree of significance was determined in each series. It was felt that while the mean of each series was shifted towards that of the atlas after correction for CA-CP, the degree of scatter was not significantly altered.

Case No.	Distance between Thalamo capsular junction defined by audiomonitoring and charted lesion and the Thalamocapsular junction of atlas brain on the track (in mm. x 4)	Same measurement in each case after correction of antero-posterior co-ordinate for CA-CP length.
1.	- 3.5	- 6.0
2.	- 3.5	- 5.5
3.	+ 4.0	- 3.5
4.	- 4.0	-10.5
5.	+ 1.5	0
6.	+ 7.0	+ 6.0
7.	0	- 3.0
8.	+ 2.0	0
9.	- 6.0	- 6.0
10.	+17.0	+14.5
11.	+17.5	+16.0
12.	+ 7.0	+ 3.0
13.	-19.5	-24.0
14.	+ 6.0	0
15.	+20.0	+18.0
16.	- 1.0	- 3.0
17.	+ 6.0	+ 2.5
18.	+14.0	+11.5
19.	+ 6.5	+ 6.0
20.	- 4.0	- 6.0
21.	-28.5	-26.0
22.	+22.0	+17.5

Mean	+ 2.75	+ 0.07
S.D.	12.01	11.64
Difference between the means		2.68
Standard Error of the difference between the means		0.476

Applying the t-test, the probability that a difference as great as that observed should arise through chance was less than 0.01.

These 22 cases were also compared with the general population of 350 cases with respect to their CA-CP length, The following were the essential data for comparison.

	350 cases	22 cases
Mean	28.68 mm.	28.32 mm.
S.D.	2.238	1.81
Difference between the means		0.36
Standard error of difference between the means		0.404

Twice the standard error was greater than the difference between the means.

Case No.	Distance between Pallidocapsular junction defined by audiomonitoring and charted on atlas and the Pallidocapsular junction of the atlas brain on the track (in mms. x 4)	Same measurement in each case after correction of AP co-ordinate for CA-CP length
1.	+ 6.5	+ 4.5
2.	+12.0	+10.0
3.	+ 8.0	+ 5.0
4.	- 5.0	-12.0
5.	+12.5	+11.0
6.	+14.0	+12.0
7.	+14.5	+13.0
8.	+ 0.5	- 3.5
9.	+ 7.5	+ 3.5
10.	+ 3.5	0
11.	+17.0	+17.0
12.	+24.5	+22.5

13.	+17.5	+15.5
14.	+ 8.5	+ 5.0
15.	- 3.0	- 6.5
16.	+21.0	+16.5
17.	+21.5	+19.5
18.	+13.0	+11.0
19.	+12.0	+10.0
20.	+16.0	+14.0
21.	+14.0	+13.0
22.	+16.0	+14.0
23.	+ 1.0	- 1.5
24.	-12.0	-13.0
Mean	+10.04	+ 7.52
S.D.	8.926	9.552

Difference between the means 2.52

Standard error of the difference between the means 0.2925

From the t-tables, the probability that a difference as great as that observed should arise through chance was less than 0.01

The characteristics of this sample of 24 cases as compared to the general population of 350 cases with reference to the CA-CP length were studied.

	350 cases	24 cases
Mean	28.68 mm.	28.13 mm.
S.D.	2.238	1.569

Difference between the means 0.55 mm.

Standard Error of the difference between the means 0.341

Twice the standard error was greater than the difference between the means.

It was therefore concluded that the proportionate correction of the anteroposterior co-ordinate approximated the individual case to the atlas brain more closely. Such

a correction slightly decreased the scatter for the thalamocapsular junction and slightly increased it for the pallidocapsular junction.

(c) Maximum Breadth of the Body of the Lateral Ventricle

Cooper and Bravo (1958) advocated the location of the pallidal target with reference to a line drawn in the AP X-ray parallel to the midline through the lateralmost part of the body of the lateral ventricle. Talairach (1955) advocated a correction with reference to the ventricular system for paraventricular structures. Hughes (1961) corrected the position of the ventral oral nucleus of the thalamus with reference to the width of the body of the lateral ventricle in order to make the site of his radiofrequency lesion more accurate.

Talairach and his colleagues (1957) subsequently did not find the breadth of the lateral ventricles to be dependable in correcting for transverse anatomical variations. A similar conclusion was reached by Brierley and Beck (1959). Van Buren and Maccubbin (1962) found no statistical correlation between anatomical variations and lateral ventricular size. Spiegel and Wycis (1962) felt that any such correction did not ensure that the lesion would not encroach on the capsule.

Amador, Blundell and Wahren (1959) did not feel that any correction for ventricular size would be a simple one.

In this project, the problem was approached in the same manner as was done in the question of correction for the intercommissural distance. The maximum breadth of the

body of the lateral ventricle was measured in 240 cases included in this study. The results were as follows.

Breadth in mms. (uncorrected for X-ray magnification)	No. of cases
14	1
15	1
16	5
17	8
18	17
19	39
20	31
21	26
22	26
23	24
24	17
25	18
26	17
27	2
28	6
29	2
Total	240

Mean 21.51 mm.

S.D. 2.963

Coefficient of Variation = 13.77

Davidoff and Dyke (1951) observed that the outermost limits of the bodies of the lateral ventricles measured by them on X-rays ranged from 17.5 to 22.5 mm., the average being 20 mm. They considered measurements greater than 22.5 mm. to be larger than normal.

Davidoff and Epstein (1955) observed that the lateral ventricles were dilated due to atrophy in basal ganglia disease but did not give any definite measurements since they had no series of pneumoencephalograms done in cases of

Parkinsonism or other basal ganglia disease.

Nineteen cases were available for which the lateral ventricle breadth was known and in which the position of the thalamocapsular junction had been defined by audiomonitoring. This point was therefore plotted on the atlas of Schaltenbrand and Bailey before and after correction of the co-ordinates of the point for the ventricular breadth. The correction applied was proportionate reduction of the lateral co-ordinate based on ratio of the ventricular sizes of the case and the standard atlas brain. The following results were obtained.

Case No.	Lat. Vent. breadth (in mms.) (uncorrected for X-ray magnification)	Thalamocapsular junction difference before correction (in mms. x 4)	Same measurement after correction (in mms. x 4)
1.	15	0.0	+60.0
2.	16	- 3.0	+23.0
3.	16	0.0	+16.0
4.	18	0.0	+13.0
5.	18	+ 6.0	+25.5
6.	18	- 6.0	+ 8.5
7.	18	+ 2.5	+16.0
8.	19	+16.0	+22.5
9.	20	- 3.0	- 3.0
10.	20	+ 6.0	+ 6.0
11.	20	- 6.0	- 6.0
12.	21	-24.0	-30.5
13.	21	+11.5	+ 5.0
14.	22	- 5.5	-13.5
15.	22	+18.0	+ 4.5
16.	23	+ 3.0	- 6.0
17.	24	- 3.5	-15.0
18.	24	-10.5	-13.5
19.	24	+14.5	- 2.5

Mean	19,95	+ 0.842	+ 5.895
S.D.	2.798	10.04	19.91

Difference between the means was 5.053

Standard error of the difference between the means was 4.167

Applying the t-test for paired observations the probability that a difference as great as that observed should arise through chance was seen to be between 0.50 and 0.20. Although the shift of the mean away from the thalamocapsular junction could not be definitely ascribed to the correction, it was very suggestive that the correction increased the error by nearly doubling the standard deviation or increasing the scatter very much.

There was another factor to be considered. Although the mean of these 19 cases approximated very closely to the atlas lateral ventricle breadth of 20 mm. (reverse correction applied for X-ray magnification), the group was not representative of the 'universe' of lateral ventricles as judged by the bigger sample of 240 cases. The following figures made this evident.

	240 cases	19 cases
Mean	21.51	19.95
S.D.	2.963	2.799

Difference between the means 1.56

Standard error of the difference between the means was 0.6701

Difference between the means was greater than twice the standard error.

This was probably due to inclusion of dilated ventricles in the bigger sample. While the 19 cases had a relatively 'normal' distribution, the 240 cases had a proportion of

cases with dilated ventricles which made the frequency distribution curve positively skewed.

The t-test was therefore reapplied for these 'hydrocephalic' brains in the group of 19 cases. Eight cases which had a lateral ventricular breadth greater than that of the atlas brain were studied as a separate group. These were shown below both the interrupted lines in the table listing all the 19 cases.

For these 8 cases alone, the following were the relevant figures.

	Ventric. breadth	Difference before correction	Difference after correction
Mean	22.63	+ 0.4375	- 8.938
S.D.	1.302	14.16	11.76

Difference between the means 9.375

Standard error of the difference between the means 1.574

Applying the t-test for paired observations, the probability that a difference as great as that observed should arise through chance was seen to be less than 0.01.

From this study, therefore, a correction for the thalamocapsular junction for the maximum breadth of the body of the lateral ventricle was not seen to be justified.

The same procedure was followed with reference to the pallidocapsular junction defined by audiomonitoring. 21 cases were available where the pallidocapsular junction by depth recording and the breadth of the lateral ventricle by pneumoventriculography were known. The difference between the junction of the case and that of the atlas brain, on

charting, was calculated in each instance before and after correction for the maximum breadth of the lateral ventricle of the case. The results are shown in the table below. The maximum breadth of the body of the lateral ventricle is expressed in mms. without correction for X-ray magnification and the differences between the pallidocapsular junctions of the case and the atlas brain in mms. x 4.

Case No.	Breadth of Lat. Ventricle	Difference between Pallidocapsular junctions Before Correction	After Correction
1.	15.0	+16.5	+35.0
2.	16.0	- 3.5	+10.0
3.	16.0	+ 3.5	+16.5
4.	16.0	0	+14.5
5.	18.0	+11.0	+18.5
6.	18.0	+12.0	+21.0
7.	18.0	+13.0	+21.5
8.	18.0	+17.0	+26.5
9.	18.0	+10.0	+16.0
10.	19.0	+15.5	+21.0
11.	20.0	+11.0	+11.0
12.	20.0	+13.0	+13.0
13.	20.0	+14.0	+14.0
14.	21.0	- 6.5	-10.0
15.	21.0	+14.0	+ 7.5
16.	22.0	+ 5.0	- 1.0
17.	22.0	+19.5	+11.0
18.	23.0	+10.0	- 2.0
19.	23.0	+ 5.0	- 4.5
20.	24.0	-12.0	-17.0
21.	24.0	+22.5	+ 6.5
Mean	19.62	+ 9.07	+10.90
S.D.	2.748	8.765	12.40

Difference between the means 1.83

Standard error of the difference between the means 2.116

Applying the t-test for paired observations, the probability that a difference as great as that observed should arise through chance was between 0.50 and 0.20.

Although the shift of the mean from the pallidocapsular junction could not be definitely attributed to the correction, it is to be noted the scatter was very much increased with a large increase of the standard deviation.

As was shown for the 19 cases of thalamocapsular junction, the 21 cases of pallidocapsular junction were not also representative of the larger series of lateral ventricles as the following figures indicate.

	240 cases	21 cases
Mean	21.51	19.62
S.D.	2.963	2.748

Difference between the means 1.89

Standard error of the difference between the means 0.6295

Difference between the means was greater than twice the standard error.

Again the group of 'hydrocephalic' brains alone were considered. These are shown below the interrupted line in the table showing all the 21 cases.

For these 8 cases alone, the following were the relevant figures.

	Ventric. breadth	Difference before correction	Difference after correction
Mean	22.5	+ 7.188	- 1.188
S.D.	1.195	11.98	9.43

Difference between the means 8.375

Standard error of the difference between the means 1.445

Applying the t-test for paired observations, the probability that a difference as great as that observed should arise through chance was seen to be less than 0.1.

Although statistically speaking, such a correction would appear valid for 'hydrocephalic' brains, it was not actually done due to the following reasons.

(1) The breadth of the internal capsule of the atlas brain was less than the mean of the cases operated with the help of depth electrical recording. This difference was evident mostly at the pallidal end of the capsule and ANY correction therefore which reduced the co-ordinates would shift the pallidocapsular junction towards the smaller atlas brain. If the correction factor was valid, it should have been so for the entire group.

(a) The standard deviation or scatter was not very much reduced by applying the correction.

(3) Unlike in the case of the thalamus, there was no anatomical reason why the pallidum should be shifted by a dilation of the lateral ventricle. Correction factors, anatomically speaking, should be valid only for immediate paraventricular structures.

(4) The number of cases studied, eight, were too few to base conclusions for the entire series of cases under consideration.

(d) Breadth of the Third Ventricle

Correction factors based on this measurement are still very controversial topics among stereotaxic surgeons.

Hughes (1963) found no reduction in the scatter of the oral ventral nucleus of the thalamus when allowance was

made for the width of the third ventricle. Spiegel and Wycis (1962) found wide scattering of the distance of the medial corner of the pallidum from the midline. When the distances were corrected for the width of the third ventricle, the scattering was no less considerable.

Gillingham and his colleagues (1960) felt that a correction for the third ventricular width was reasonable, especially at the pallidal level. Gillingham (1963) did not however apply this correction when the ventricular size was within normal limits. Guiot (1963) used to correct for this factor but had ceased to do so recently since he felt it did not increase the accuracy of placement of his lesion.

Talairach (1962) felt that in cases of distension of the third ventricle, the medial limit of the lesion should be located at least 5 to 6 mm. outside the wall of the third ventricle to avoid encroachment on the hypothalamus.

Hassler (1963) and Yasargil and Krayenbuhl (1963) strongly advocated correction for the width of the third ventricle. They calculated the lateral distance of the lesion from the wall of the third ventricle. Hassler measured the width of the middle of the ventricle for thalamic targets and the lower part of the ventricle for pallidal targets.

Amador, Blundell and Wahren (1959) opined that the correction necessitated by variations in ventricular size was not a simple one. According to these authors, the structures nearest to the third ventricle did not move perpendicularly away from the midline with increasing

ventricular size. The displacement was the resultant of vertical and horizontal movement and the correction therefore was complicated.

In this project, the problem was approached in the same manner as was done for the lateral ventricle. The maximum breadth of the body of the third ventricle adequately outlined by air or myodil was measured in 185 Roentgenograms. The results are given below.

Breadth in mms. (uncorrected for X-ray magnification)	Number of cases
5	6
6	15
7	25
8	38
9	31
10	21
11	20
12	8
13	9
14	4
15	6
16	2
Total	185
Mean	9.184
S.D.	2.438
Coefficient of Variation	26.55

As in the case of the frequency distribution of the lateral ventricular size, the frequency distribution of the third ventricular size was also seen to be positively skewed, with a larger proportion of "dilated" ventricles.

It was interesting to compare these results with those of other authors. Twining (1939) considered 10 mm. as the upper limit of normality. Davidoff and Dyke (1951) gave

the range measured on the X-ray as 2 to 8 mm. for normal ventricles. Amador, Blundell and Wahren (1959) found a range of 1.5 to 8.5 mm. for autopsy sections. They felt that the width was dependant on the presence or absence of a connexus interthalamicus. In cases where a massa intermedia was present, the range was 1.5 to 4.0 mm. Where it was absent, the range was 7.0 to 8.5 mm.

19 cases were available in whom both the position of the thalamocapsular junction by depth electrical recording and the width of the body of the third ventricle by air or myodil ventriculography were known. The thalamocapsular junction was plotted on the atlas of Schaltenbrand and Bailey before and after correction for the size of the third ventricle.

The following results were obtained.

Case No.	Maximum breadth of Third Ventricle uncorrected for X-ray magnification	Difference between thalamo-capsular Junctions on charting between case and atlas brain. Before correction (in mms. x 4)	After correction (in mms. x 4)
1.	5	+16.0	+20.5
2.	5	+ 3.0	+ 8.0
3.	6	- 6.0	- 4.0
4.	6	+18.0	+21.0
5.	7	0	- 1.0
6.	7	+ 6.0	+ 3.0
7.	7	-24.0	-25.0
8.	7	+ 2.5	+ 2.0
9.	8	0	- 6.0
10.	8	+11.5	+ 7.0
11.	9	- 5.5	-12.0
12.	9	- 3.5	-11.5
13.	9	-10.5	-12.5

14.	9	- 3.0	-11.0
15.	10	- 3.0	-13.5
16.	10	0	- 3.5
17.	10	+14.5	- 0.5
18.	10	- 6.0	- 8.5
19.	11	+ 6.0	- 4.5
Mean	8.053	+ 0.842	- 2.737
S.D.	1.810	10.04	11.46

Difference between the means 3.579

Standard error of the difference between the means 1.234

It was noted that the mean was shifted from the thalamocapsular junction by application of the correction and the scatter as judged by the standard deviation was increased. Applying the t-test for paired observations, it was seen that the probability that a difference as great as that observed should arise through chance, was less than 0.01. Correction for third ventricular breadth therefore increased the error due to anatomical variations in the case of the thalamocapsular junction.

The question arose whether this series of 19 cases was representative of the 'universe' of third ventricles as shown by the larger sample of 185 cases. The relevant figures were studied.

	185 cases	19 cases
Mean	9.184	8.053
S.D.	2.439	1.809

Difference between the means 1.131

Standard error of the difference between the means 0.4521

Since the difference between the means was greater than twice the standard error, it was decided that the sample

was not representative of the 'universe' of Parkinsonism cases loaded with dilated third ventricles.

As was done in the case of the lateral ventricle study, the group of cases with third ventricles larger than that of the atlas brain were now separately studied. There were 15 cases in this group shown below the interrupted line in the table listing all the 19 cases. For these 15 cases, the relevant figures were analysed.

	Ventricular breadth	Difference before correction	Difference after correction
Mean	8.733	- 1.0	- 6.5
S.D.	1.335	9.215	8.155

Difference between the means 5.5

Standard error of the difference between the means 1.095

Applying the t-test for paired observations, the probability, that a difference as great as that observed should arise through chance was less than 0.01. Therefore the correction of target site for the breadth of the third ventricle was not justified by this study.

A study on identical lines was done for the pallido-capsular junction and the third ventricular width. 21 cases were available in this series. The details are shown below.

Case No.	Third ventricular Breadth in mms. uncorrected for X-ray magnification	Difference between pallidocapsular junctions of case and the atlas brain on charting (in mms. x 4) Before correction	After correction
1.	5	+ 15.5	+ 19.0
2.	5	+ 5.0	+ 8.0
3.	6	+ 17.0	+ 18.0

4.	6	+ 19.5	+ 21.0
5.	7	+ 11.0	+ 11.0
6.	7	+ 12.0	+ 11.0
7.	7	+ 13.0	+ 12.0
8.	7	- 6.5	- 7.5
9.	7	+ 10.0	+ 9.0
10.	8	+ 16.5	+ 11.0
11.	8	+ 14.0	+ 9.0
12.	9	+ 5.0	- 1.0
13.	9	- 12.0	- 15.0
14.	9	+ 11.0	+ 4.0
15.	10	- 3.5	- 12.5
16.	10	+ 3.5	- 3.5
17.	10	0	- 1.0
18.	10	+ 22.5	+ 8.0
19.	10	+ 14.0	+ 11.5
20.	11	+ 13.0	+ 6.0
21.	13	+ 10.0	- 2.0
Mean	8.286	+ 9.071	+ 5.524
S.D.	2.077	8.768	9.804

Difference between the means 3.547

Standard error of the difference between the means 1.04

It was seen that there was a shift of the mean towards the pallidocapsular junction of the atlas brain but simultaneously there was an increase in scatter as judged by the standard deviation. Applying the t-test for paired observations, the probability that a difference as great as that observed should arise through chance was seen to be less than 0.01.

These 21 cases were compared with the larger sample of 105 cases for their third ventricle width.

	105 cases	21 cases
Mean	9.184	8.286
S.D.	2.439	2.077

Difference between the means	0.898
Standard error of the difference between the means	0.4874

The difference between the means was less than twice the standard error and therefore the likelihood that the sample was not representative of the 'universe' of third ventricles was not proved.

In view of the increase in scatter on the application of the correction, there was no justification to apply it.

(e) Height of Thalamus

Hassler (1963) and Yasargil and Krayenbuhl (1963) attached great importance to this as a correction factor for the vertical co-ordinate of their thalamic targets. Hughes (1963) used 20% of theheight of the thalamus in calculation of his thalamic target. He felt however that the variability of this measurement was very much less than those of the ventricles. Brierley and Beck (1959) felt that the vertical co-ordinate could be accurately corrected by the TOTAL thalamic height which could not be measured in the X-ray, but could be measured only at autopsy. The thalamic height used by stereotaxic surgeons (maximum height of the floor of the body of the lateral ventricle above the intercommissural line in the lateral X-ray) was not considered by them as a valid correction factor.

Van Buren and Maccubbin (1962) found no statistical support for any correction factor based on pneumoencephalograms and introduced the concept of a "sphere of error".

The thalamic heights of 175 cases were measured in this

study. The frequency distribution is shown below.

Height of thalamus in mms. uncorrected for X-ray magnification	No. of cases
14	1
15	2
16	7
17	18
18	24
19	44
20	35
21	30
22	11
23	2
24	1
Total	175
Mean	19.27
S.D.	1.72
Coefficient of Variation	8.926

The present study therefore supported the opinion of Hughes that the variation in thalamic height was much less than that of the size of the ventricles as judged by the corresponding coefficients of variation. It was seen that nearly 97% of cases were within the range of two standard deviations or 3.44 mm. The usual height of thalamic target in the series of cases under analysis was 4 mm., after correction for X-ray. Application of the thalamic height correction factor therefore would not have changed the vertical co-ordinate by more than 0.7 mm. In as many as 60% of cases, it would be only half of this. Since the accuracy of radiological measurement and final instrumental error was more than this amount, there was no

justification for the introduction of a correction factor too minute in proportion to the other variables.

Furthermore the major part of this study was concerned with the internal capsule. At this level the electrode track was on an average 0 to 3 mm. above the intercommissural line. Apart from the very doubtful assumption that a thalamic correction factor was applicable to the capsule, even such a correction would be of the order of a fraction of a millimeter. This was considered to be a negligible variation and consequently no thalamic height correction factor was introduced in this study.

(f) Cranio-Cerebral Topometry

Apart from Delmas and Pertuiset (1959), no stereotaxic surgeon found the cephalic indices useful or reliable in the determination of intracerebral targets. No attempt was therefore made in the present study to introduce any such localisation or correction factor.

(g) Width of the Internal Capsule

Guiot, Brion and Akerman (1961) found the range in the width of the posterior limb of the internal capsule along their electrode tracks to be widely variable. Their trajectory was closely similar to the one used in the present study. From 2 mm., on a medial track, it could be as much as 17 mm. on a lateral track.

Guiot (1963) using bipolar depth recording on a track more oblique than the present one considered the mean width of the capsule to be about 9 mm.

26 instances were available in this project where both the thalamocapsular and pallidocapsular junctions were

defined with accuracy and thus the breadth of the capsule measured. The following results were obtained.

Case No.	Breadth of capsule (in mms.)	Lateral distance of track (in mms. uncorrected for X-ray magnification)	Angle between electrode track and intercommissural line (in degrees)
1.	3.5	17.5	11.5
2.	5.0	16.0	11.0
3.	5.0	17.5	26.0
4.	5.0	18.0	11.0
5.	5.5	19.0	20.5
6.	6.5	17.5	26.0
7.	6.5	19.0	25.0
8.	7.0	17.5	20.0
9.	7.0	18.5	18.5
10.	7.5	17.5	26.0
11.	8.0	17.5	10.0
12.	8.0	17.5	26.0
13.	8.0	18.0	20.0
14.	8.0	19.0	23.5
15.	8.0	20.0	20.0
16.	8.0	20.0	26.0
17.	8.0	20.0	26.5
18.	9.0	18.0	24.0
19.	9.5	15.0	27.5
20.	10.0	16.0	20.0
21.	10.5	18.0	27.0
22.	10.5	18.0	29.0
23.	10.5	19.5	17.0
24.	10.5	21.5	22.5
25.	12.0	17.5	26.0
26.	12.0	17.5	27.0

For Breadth of Capsule

Mean	8.038
S.D.	2.231
C.V.	27.76

It was seen that the variability of the internal capsule was much greater than any of the previous measurements referred to in this study. The number of cases were too few to define exact degrees of correlation between the variables. Scatter diagrams were drawn and these indicated some trends. Within the limits of the lateral distances used in this series for the entry of the electrode, there was no definite correlation between the width of the capsule and the laterality of the track. The angle of entry, however, showed some trend when compared with the width of the capsule. The larger angles were associated with greater capsular widths.

(h) Summary

In the study of correction factors to be applied for accurate localisation of stereotaxic targets, the inter-commissural distance, the maximum breadth of the body of the lateral ventricle, the width of the third ventricle and the height of thalamus were considered.

Each factor was analysed with reference to the shift of the mean and scatter produced before and after correction, the points used being the thalamocapsular and pallidocapsular junctions defined by audiomonitoring and transferred on charting to the standard atlas brain.

The anatomical basis for each correction was also taken into consideration in the final decision as to its applicability.

It was decided that with the information available, under the conditions of operation used in the present

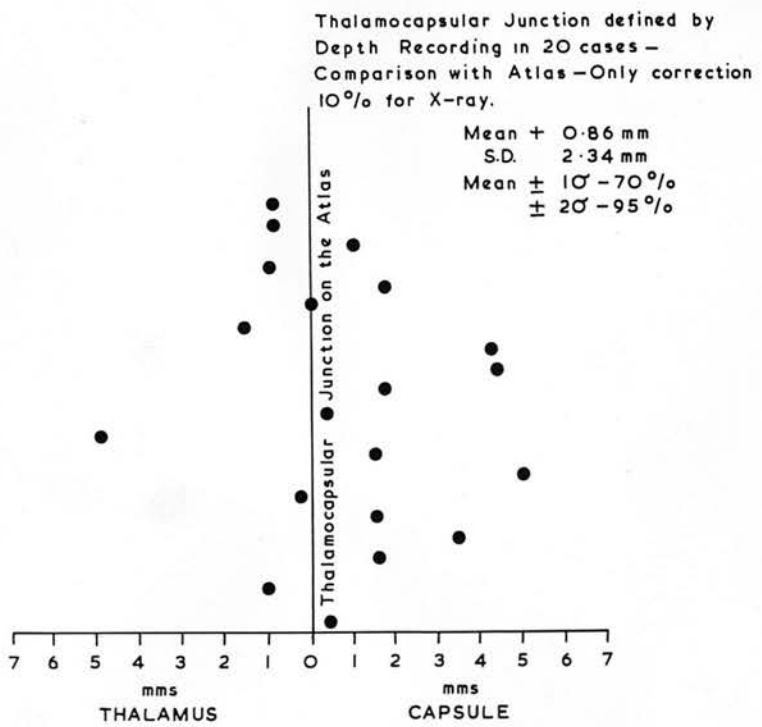
series, and for comparison with the standard brain of Schaltenbrand and Bailey's atlas (brain no. LXXVIII), only one correction was justifiable in the study of the thalamus, internal capsule and pallidum. This correction was the proportionate reduction of the anteroposterior co-ordinate with reference to the intercommissural length.

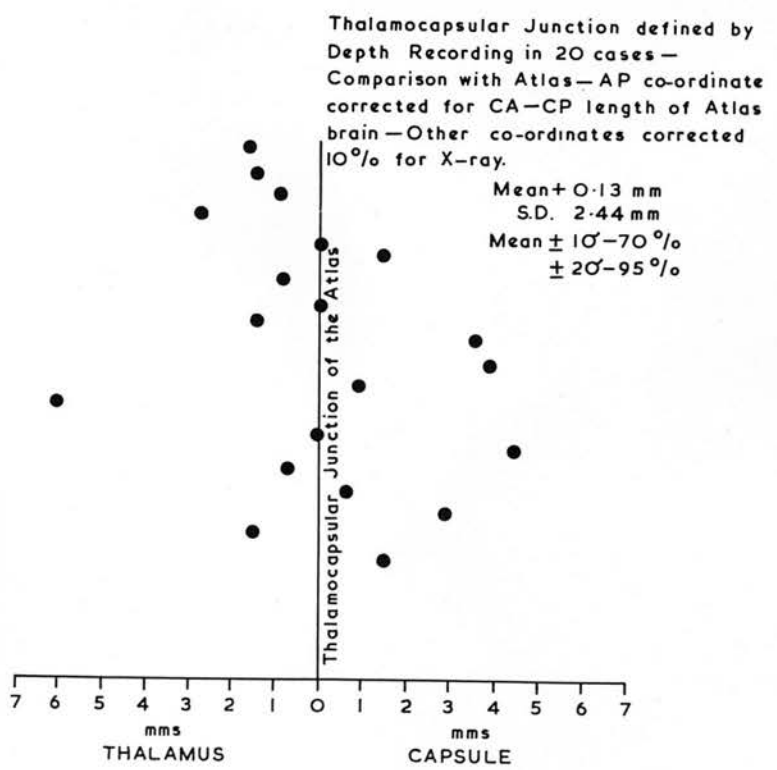
Only this correction has been carried out in the scattergrams in the subsequent parts of this work. All other values of co-ordinates were absolute being corrected only for X-ray magnification.

Figures of Chapter 3

Explanatory Note

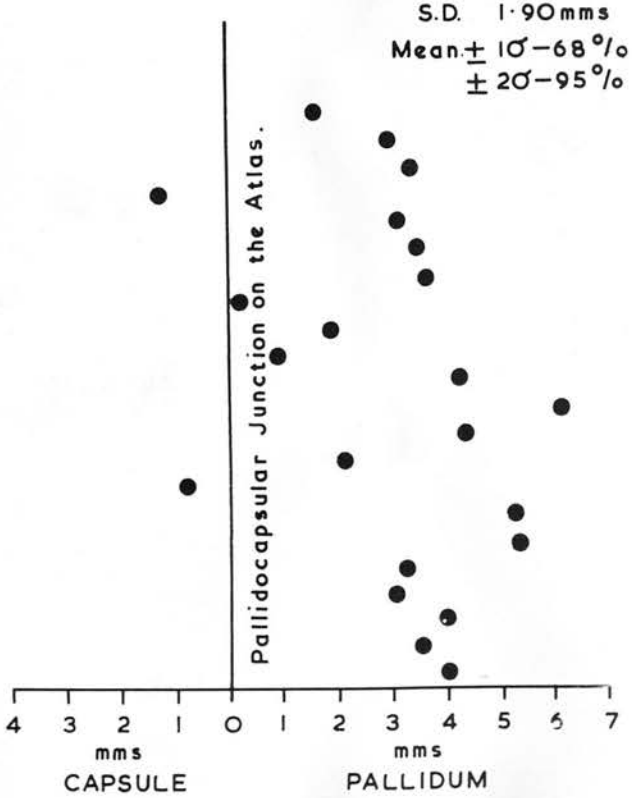
The scattergrams and bar charts which follow, explain the general principles described in the chapter. The number of cases from which they are constructed is slightly less, in each instance, than the actual numbers used in the statistical calculations in the chapter. The last few cases are included for statistical analysis but not in the diagrams. The mean, standard deviation, etc. on the photographs are therefore slightly different from the corresponding figures in the text. The scattergrams and bar charts illustrate the general point that there is no obvious reduction in scatter on application of correction factors. The applicability or otherwise of each correction factor has therefore to be decided on statistical tests as has been done in the text of the chapter.


Thalamocapsular Junction defined by Depth Recording in 20 cases — Comparison with Atlas — Only correction 10% for X-ray.
Mean + 0·86 mm
S.D. 2·34 mm
Mean ± 1σ — 70%
± 2σ — 95%
Thalamocapsular Junction on the Atlas
7 6 5 4 3 2 1 0 1 2 3 4 5 6 7
mms
THALAMUS
mms
CAPSULE


Thalamocapsular Junction defined by Depth Recording in 20 cases — Comparison with Atlas — AP co-ordinate corrected for CA—CP length of Atlas brain — Other co-ordinates corrected 10% for X-ray.
Mean + 0·13 mm
S.D. 2·44 mm
Mean ± 1σ — 70%
± 2σ — 95%
Thalamocapsular Junction of the Atlas
7 6 5 4 3 2 1 0 1 2 3 4 5 6 7
mms
THALAMUS
mms
CAPSULE

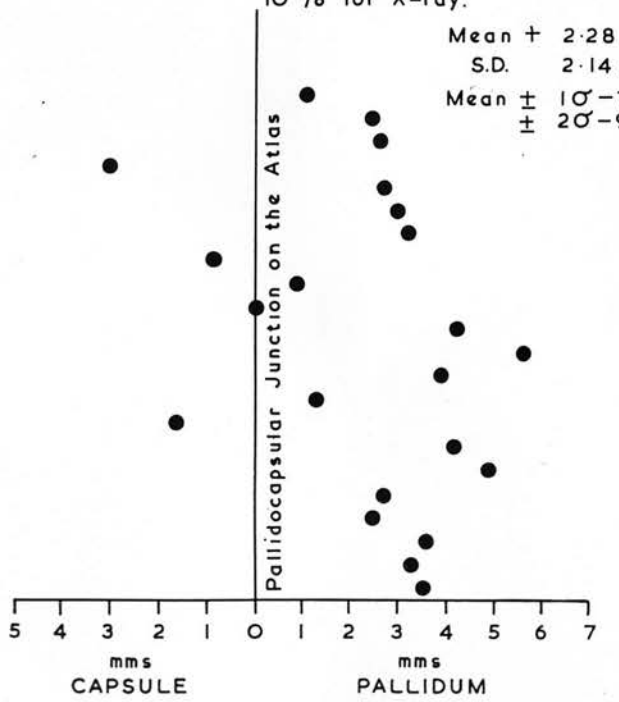
Pallidocapsular Junction defined by Depth recording in 22 cases – Comparison with Atlas – Only correction 10 % for X-ray

Mean + 2·93 mms
S.D. 1·90 mms
Mean ± 1σ – 68 %
± 2σ – 95 %

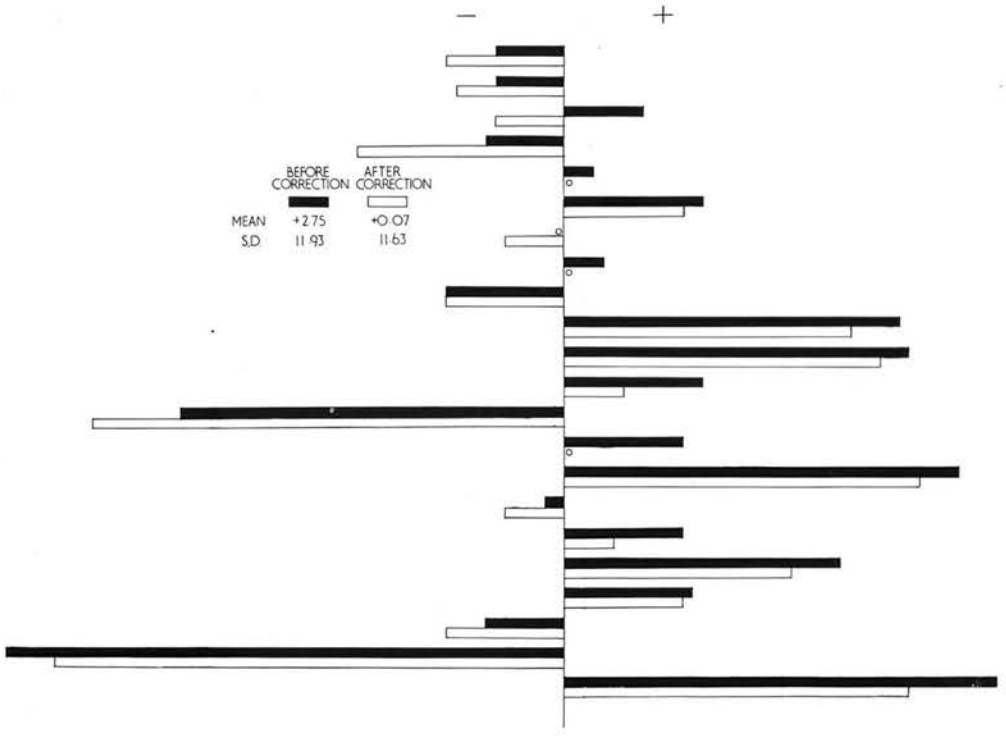


Pallidocapsular Junction defined by Depth Recording in 22 cases — Comparison with Atlas — AP co-ordinate corrected for CA – CP length of Atlas brain — Other co-ordinates corrected 10 % for X-ray.

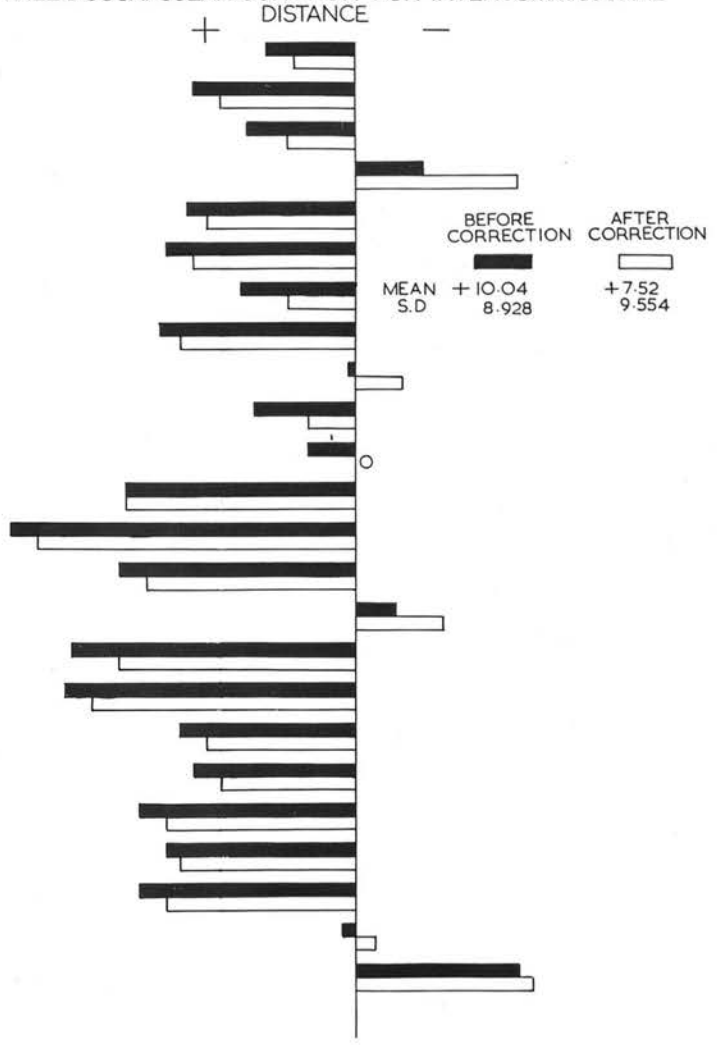
Mean + 2·28 mms
S.D. 2·14 mms
Mean ± 1σ – 73 %
± 2σ – 95 %



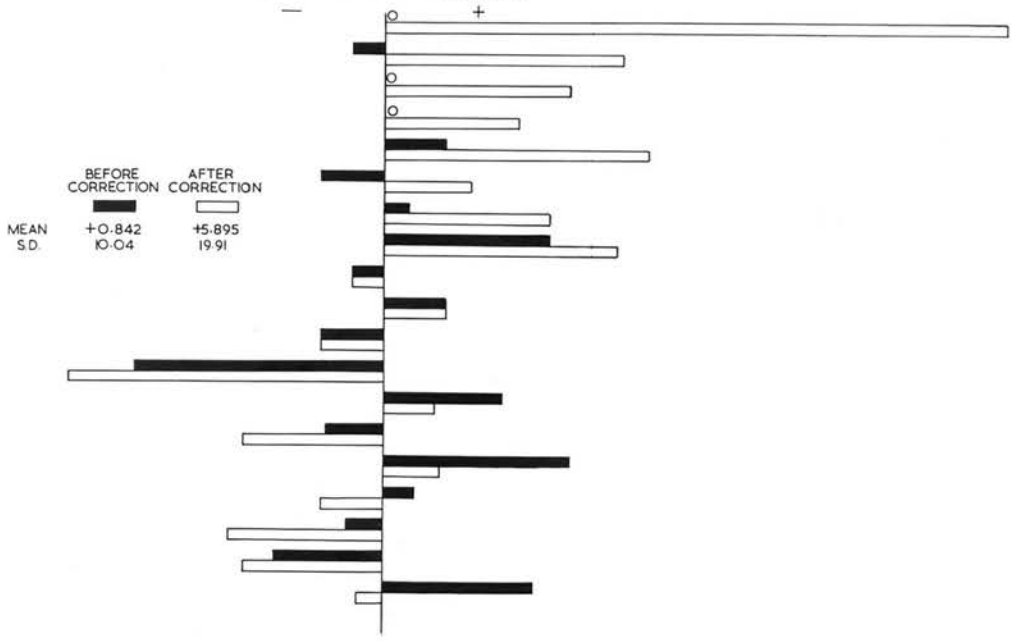
BAR CHART SHOWING NECESSITY FOR CORRECTION OF THALAMOCAP-SULAR JUNCTION FOR INTERCOMMISSURAL DISTANCE.



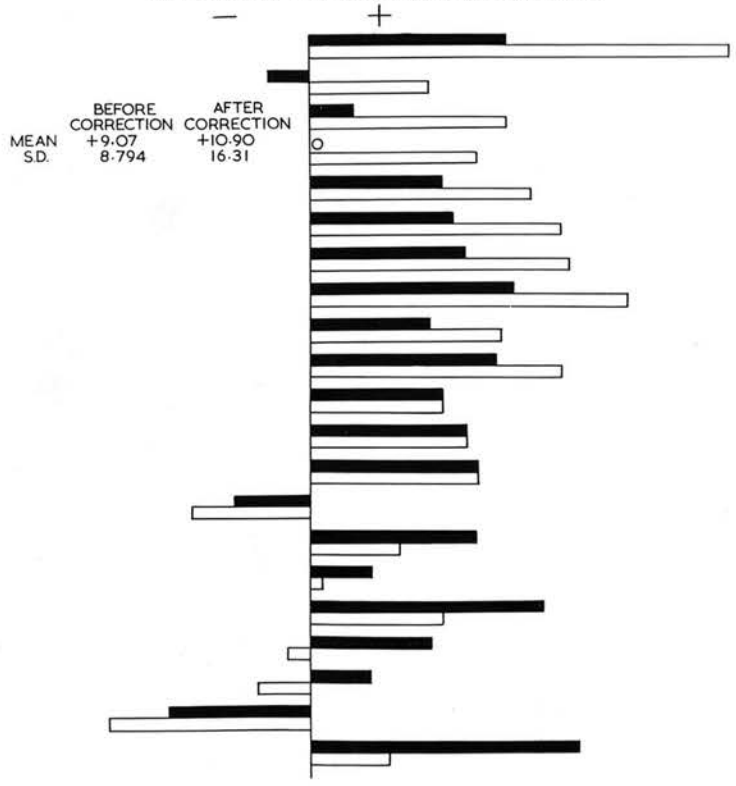
BAR CHART SHOWING NECESSITY FOR CORRECTION OF PALLIDOSCAPSULAR JUNCTION FOR INTERCOMMISSURAL DISTANCE



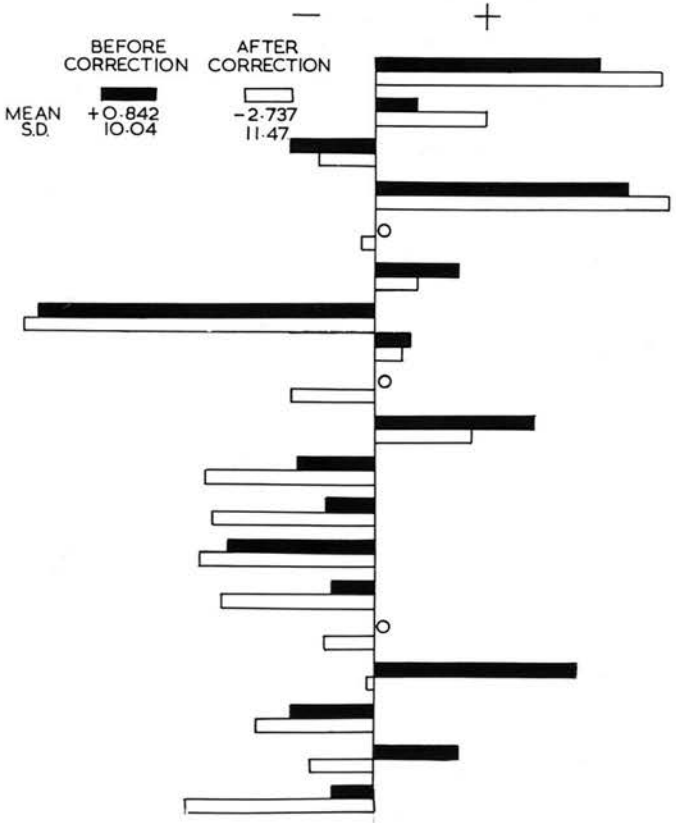
BAR CHART SHOWING SHIFT OF MEAN FROM THALAMOCAPSULAR JUNCTION AND INCREASE OF SCATTER ON CORRECTION FOR BREADTH OF LATERAL VENTRICLE



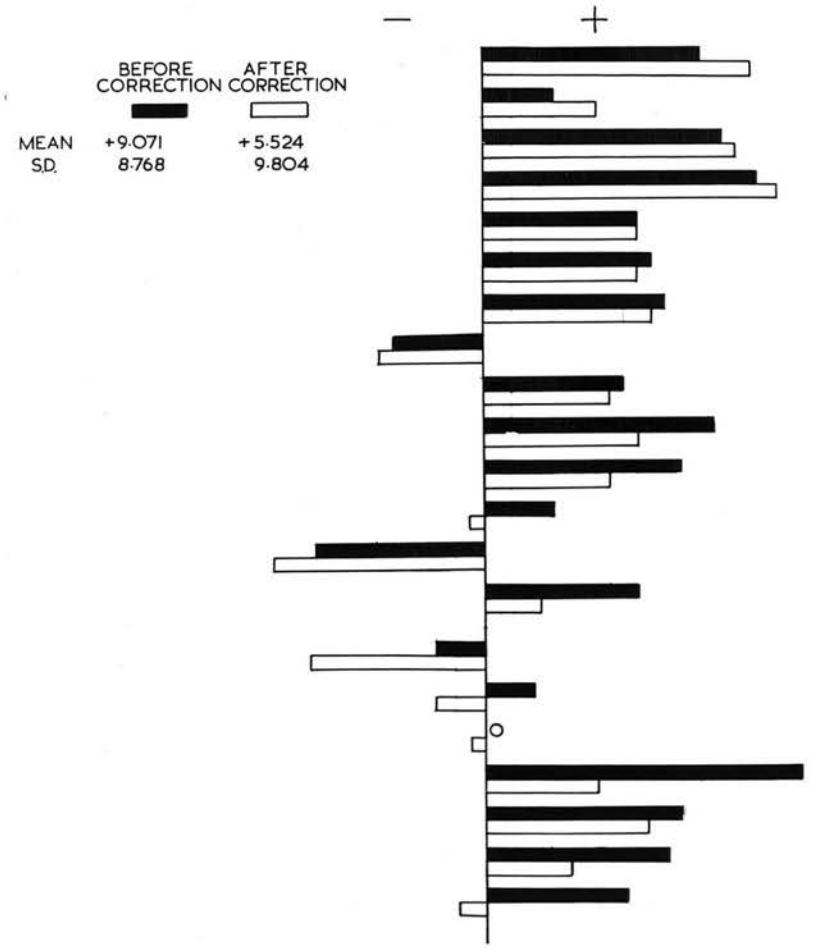
BAR CHART SHOWING SHIFT OF MEAN FROM PALLIDOCAPSULAR JUNCTION AND INCREASE IN SCATTER ON CORRECTION FOR LATERAL VENTRICULAR BREADTH.



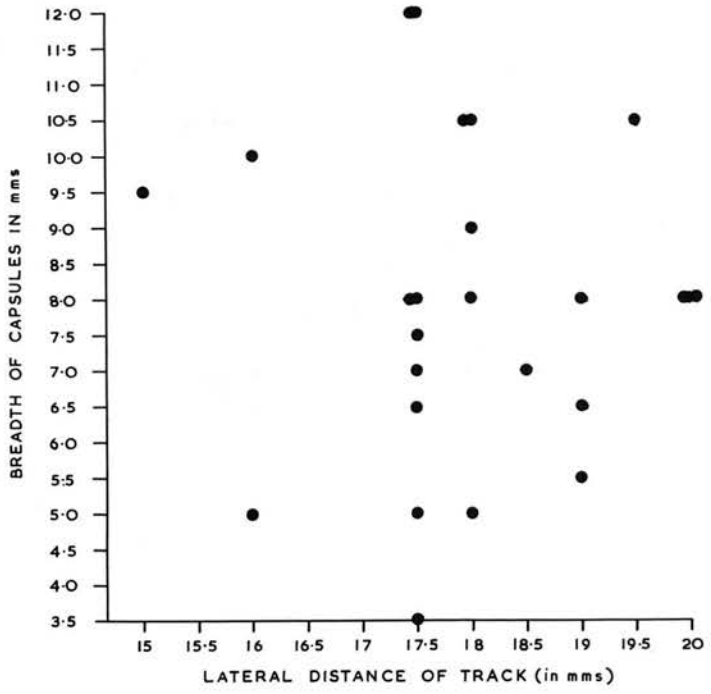
BAR CHART SHOWING SHIFT OF MEAN FROM THALAMOCAPSULAR JUNCTION OF ATLAS ON CORRECTION FOR THIRD VENTRICLE BREADTH.



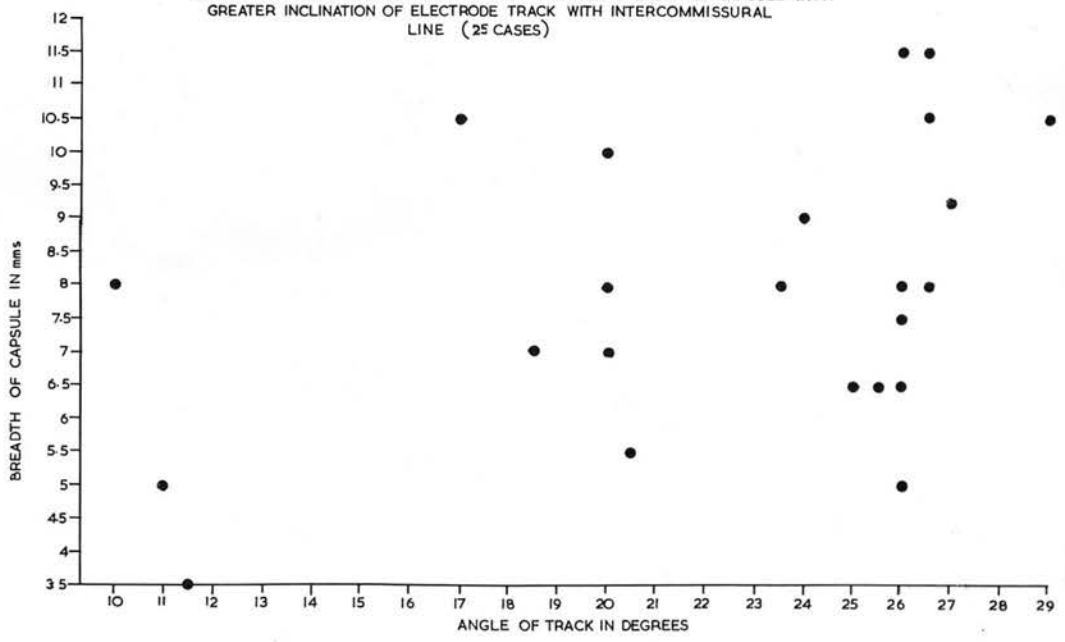
BAR CHART SHOWING SHIFT OF MEAN TOWARDS THE PALLIDOCAPSULAR JUNCTION ON CORRECTION FOR THIRD VENTRICULAR BREADTH.



SCATTER DIAGRAM SHOWING NO CORRELATION BETWEEN LATERAL DISTANCE OF ELECTRODE TRACK (WITHIN THE RANGE USED AT OPERATION) AND BREADTH OF INTERNAL CAPSULE (MEASURED BY DEPTH ELECTRICAL RECORDING) (25 CASES)



SCATTER DIAGRAM SHOWING TENDENCY OF GREATER BREADTH OF CAPSULE WITH GREATER INCLINATION OF ELECTRODE TRACK WITH INTERCOMMISSURAL LINE (25 CASES)



REFERENCES

1. Amador, L.V., Blundell, J.E. and Wahren, W. (1959). In Schaltenbrand, G. and Bailey, P. Introduction to stereotaxis with an atlas of the human brain. Thieme, Stuttgart. I, 16.

2. Brierley, J. and Beck, E. (1959). J. Neurol. Neurosurg. & Psychiat., 22, 287.

3. Caracalos, A., Levita, E. and Cooper, I.S. (1962). St. Barnabas Hospital Medical Bulletin, I, 3, 24.

4. Cooper, I.S. and Bravo, C.J. (1958). In Fields, W.S. Ed. Pathogenesis and treatment of Parkinsonism. C.C. Thomas, Springfield, Illinois, 325.

5. Dale, H. quoted by Hill, A.B. (1961). Principles of medical statistics. The Lancet Limited, London, 7th Edition. 5.

6. Davidoff, L.M. and Dyke, C.C. (1951). The Normal Encephalogram, Lea and Febiger, Philadelphia, 3rd Edition. 99,108.

7. Davidoff, L.M. and Epstein, E.S. (1955). The Abnormal Pneumoencephalogram. H. Kimpton, London, 2nd Edition. 426.

8. Delmas, A. and Pertuiset, B. (1959). Craniocerebral topometry in man. Blackwell Scientific Publications. Oxford.

9. Gillingham, F.J., Watson, W.S., Donaldson, A.M. and Naughton, J.A.L. (1960). Brit. med. J., 2, 1395.

10. Gillingham, F.J. (1963). Personal Communication.

11. Guiot, G. (1963).	Personal Communication.
12. Guiot, G., Brion, S. and Akerman, M. (1961).	Ann. Chir., 15, 557;703.
13. Hassler, R. (1963).	Personal Communication.
14. Helmholtz quoted by Hill, A.B. (1961).	Principles of medical statistics. The Lancet Limited, London, 7th Edition. 5.
15. Hughes, B. (1961).	In Carling, E.R. and Ross, J.P. Ed. British Surgical Practice. Surgical Progress 1961. Butterworth, London. 129.
16. Hughes, B. (1963).	Personal Communication.
17. Spiegel, E.A. and Wycis, H.T. (1952).	Stereoencephalotomy (Thalamotomy and related Procedures) Part I. Methods and stereotaxic atlas of the human brain. Grune and Stratton, New York. 148.
18. Spiegel, E.A. and Wycis, H.T. (1962).	Stereoencephalotomy. Part II. Clinical and physiological applications. Grune and Stratton, New York. 253.
19. Talairach, J. (1955). quoted by Amador, L.V., Blundell, J.E. and Wahren, W.	In Schaltenbrand, G. and Bailey, P. Introduction to stereotaxis with an atlas of the human brain. Thieme, Stuttgart, 1959, I, 16.
20. Talairach, J. (1961).	First International Symposium on Stereoencephalotomy. Philadelphia. Confin. Neurol., 22, 264.

21. Talairach, J. (1962). quoted by Spiegel, E.A. and Wycis, H.T. — Stereoencephalotomy. Part II. Clinical and physiological applications. Grune and Stratton, New York. 1962. 268.

22. Talairach, J., David, M., Tournoux, P., Corredor, H. and Kvasina, T. (1957). — Atlas d'anatomie stereotaxique. Paris. Masson & Cie.

23. Twining, E.W. (1939). — Brit. J. Radiol., 12, 585.

24. Van Buren, J.M. and Maccubbin, D.A. (1961). — First International Symposium on Stereoencephalotomy. Philadelphia. Confin. Neurol., 22, 259. 1962.

25. Van Buren, J.M. and Maccubbin, D.A. (1962). — J. Neurosurg., 19, 811.

26. Wahren, W. and Braitenberg, V. (1959). — In Schaltenbrand, G. and Bailey P. Introduction to stereotaxis with an atlas of the human brain. Thieme, Stuttgart, I, 42.

27. Yasargil, M.G. and Krayenbuhl, H. (1963). — Personal Communication.

PART II

NORMAL ANATOMY AND PHYSIOLOGY

Chapter 4: Studies of the Sensory System

Chapter 5: Studies of the Motor System

CHAPTER 4

STUDIES OF THE SENSORY SYSTEM

"Theories are very attractive to most persons because they are the philosophy, and often the poetry, of science. Even the most captivating theories are subject to eternal change. Recorded observations, on the other hand, although primarily addressed to the rational part of one's nature, have values which endure."

.... Eugene Gley (French Physiologist)

(a) Scope of Exploration

The therapeutic nature of the procedure completely negativated all possibilities of exploration along any other track other than that required for treatment. All the observations made in this project and recorded in this thesis were from a small but important volume of tissue in the diencephalon. The volume of tissue explored could be approximately likened to a truncated cone of tissue. This tissue was situated in an oblique direction downwards and forwards in a parasagittal plane parallel to the midsagittal plane of the brain. It included part of the thalamus, the posterior limb of the internal capsule and the globus pallidus.

Passage of the electrode through the ventricular cavity was achieved with minimal resistance. In many cases there was a definite palpable resistance to its passage as the ependymal covering of the pulvinar was penetrated. In such cases the pulvino-pallidal target distance could be measured as the distance between the ventricular surface of the pulvinar along the electrode

track and the radiological pallidal target. However it was not always possible to measure this distance. It was usually between 20 and 30 mm. In cases of depth electrical recording studies were usually started at 28 mm. behind the pallidal target whereas for stimulation and coagulation, 20 mm. behind the pallidal target was the common starting point.

The lateral distance of the electrode track varied with the phase of evolution of stereotaxic surgery at Edinburgh. The lateral distances of the tracks used, as judged by the position of the radio-opaque steel marker in the post-operative PA film, were analysed in 475 operations. It was seen that the great majority (96.6%) of the explorations could be referred to one of three atlas plates (S1 13.5, S1 16.0 and S1 18.5) being within the range 12.5 to 19.5 mm. from the midsagittal plane of the brain. The few tracks lateral to these, however, gave us very important information regarding the motor tracts. This point would be further elaborated in a subsequent part of the study.

Frequency Distribution of the Lateral Distance of the Track of the Electrode from the Midventricular Line in 475 operations.

Lateral Distance in mms. (uncorrected for X-ray magnification)	Number of cases
11.0 & 11.5	1
12.0 & 12.5	1
13.0 & 13.5	4
14.0 & 14.5	7
15.0 & 15.5	13
16.0 & 16.5	45
17.0 & 17.5	109
18.0 & 18.5	123

19.0 & 19.5	98
20.0 & 20.5	47
21.0 & 21.5	17
22.0 & 22.5	5
23.0 & 23.5	2
24.0 & 24.5	3

These lateral distances could be regrouped into the following five groups.

Laterality of Track	Atlas Plate	Number of cases
1. Electrode track too medial (Marker in X-ray between 11.0 and 13.5 mm., corresponding to a parasagittal plane 11.0 mm. from the midline)	S1 11.0	6
2. Electrode track medial (Marker in X-ray between 14.0 and 16.0 mm., corresponding to a parasagittal plane 13.5 mm. from the midline)	S1 13.5	41
3. Electrode track in standard plane (Marker in X-ray between 16.5 and 19.0 mm. corresponding to a parasagittal plane 16.0 mm. from the midline)	S1 16.0	323

4. Electrode track lateral (Marker in X-ray between 19.5 and 21.5 mm. corresponding to a plane 18.5 mm. from the midline)	S1 18.5	95
5. Electrode track too lateral (Marker in X-ray between 22.0 and 24.5 mm., corresponding to a plane 21.0 mm. from the midline)	S1 21.0 (S1 21.5)	10
Total		475

The angle of inclination of the electrode track again varied with the height of the thalamic target above the intercommissural line. An analysis of this angle is given below.

Frequency Distribution of the Angle of Inclination of the Electrode Track with the Intercommissural Line in 475 Operations.

Angle of entry	No. of cases
10.0 - 11.5	4
12.0 - 13.5	2
14.0 - 15.5	7
16.0 - 17.5	18
18.0 - 19.5	32
20.0 - 21.5	56
22.0 - 23.5	51
24.0 - 25.5	67
26.0 - 27.5	78
28.0 - 29.5	49

30.0 - 31.5	43
32.0 - 33.5	11
34.0 - 35.5	14
36.0 - 37.5	10
38.0 - 39.5	4
40.0 - 41.5	5
42.0 - 43.5	8
44.0 - 45.5	5
46.0 - 47.5	3
48.0 - 49.5	4
50.0 - 51.5	3
52.0 - 53.5	1

In each case the height at which (after application of corrections for X-ray and CA-CP distance) the electrode track crossed the midcommissural plane was measured. The following were the values for this figure in 475 operations.

Frequency distribution of height of intersection of electrode track and midcommissural plane in 475 operations.

Height of intersection of electrode track and mid-commissural plane above the intercommissural line (in mms.)	No. of cases
-6.25 & -6.0	1
-5.75 & -5.5	1
-5.25 & -5.0	0
-4.75 & -4.5	1
-4.25 & -4.0	1
-3.75 & -3.5	0
-3.25 & -3.0	1
-2.75 & -2.5	1
-2.25 & -2.0	1
-1.75 & -1.5	2
-1.25 & -1.0	3
-0.75 & -0.5	3
-0.25 & 0	5

+0.25	&	0.5	8
0.75	&	1.0	8
1.25	&	1.5	24
1.75	&	2.0	26
2.25	&	2.5	43
2.75	&	3.0	50
3.25	&	3.5	41
3.75	&	4.0	55
4.25	&	4.5	50
4.75	&	5.0	37
5.25	&	5.5	28
5.75	&	6.0	25
6.25	&	6.5	15
6.75	&	7.0	8
7.25	&	7.5	12
7.75	&	8.0	9
8.25	&	8.5	3
8.75	&	9.0	3
9.25	&	9.5	6
9.75	&	10.0	0
10.25	&	10.5	0
10.75	&	11.0	1
11.25	&	11.5	3

83% of these tracks crossed between the range of 1.25 mm. and 6.5 mm. This did not, of course, necessarily mean that lesions were made in the midcommissural plane at all these heights. The majority of variations from the middle range were cases of pain or involuntary movements other than Parkinsonism. The values quoted are the heights at which the electrode track would have intersected the midcommissural plane if the trajectory had been continued further forward from its thalamic lesion site. The values are quoted to provide an idea of the extent of

the diencephalon explored by this method of stereotaxy.

(b) Sensory Phenomena during Stereotaxic Surgery

The somatotrophic arrangement of parts of the sensory relay nucleus and the fibres of the thalamoparietal projection has not been studied extensively in the human. Prior to 1958 all knowledge in this field was derived solely from animal experiments or degeneration studies.

Mountcastle and Henneman (1952) using the evoked potential technique, presented evidence to show that the sensory relay nucleus in the thalamus of the macaque monkey could be divided into an external and an internal part. In the external portion, the body segments were represented in an orderly fashion with the cervical segments most medial and the sacral, most lateral. The thoracic and lumbar regions were represented only dorsally while the regions for the peripheral parts of the limbs were represented in an expanded manner ventrally. The medial or arcuate portion of the ventrobasal complex contained the representation of the head, face and intraoral structures. They found very few evoked responses outside the region of the sensory relay nucleus.

Gaze and Gordon (1954) investigated the electrical activity of single neural units in the thalamus of the cat and the monkey. In contrast to the previous workers, they found some evoked potentials outside the region of the sensory nucleus, these being derived presumably from fibre systems transmitting sensory impulses. They confirmed the somatotrophic arrangement of the face, upper limb and lower limb regions from the medial to the lateral aspect but found

a considerable degree of overlapping between these regions.

Cohen and Grundfest (1954) on similar work arrived at different conclusions. They observed a very considerable degree of overlap in the topographical representation of the thalamic sensory nucleus of cats. They were at conflict with the 'strict localisation school' and felt that the thalamus, a primary way station for sensory reporting, did not separate the reports of sensory activity from different areas into discrete trains of messages to higher levels.

This conflict between the 'strict localisation school' and the 'poor localisation school' is also apparent among the stereotaxic surgeons studying human material.

Hassler and Riechert (1959) documented their cases of stimulation in this area and were of the opinion that the somatotopographical segmentation of the sensory thalamic nuclei in the human was, in general, in agreement with that described in animals.

Mark and Hackett (1959) found that lesions placed in the ventral posteromedial nucleus produced sensory loss in the contralateral face and more laterally placed lesions caused hypesthesia in the distal part of the upper extremity.

Ervin and Mark (1960), using stereotaxic thalamic stimulation and audiomonitoring, reported fairly clear separation of areas in this region. On minimal stimulation, they obtained face and hand sensations at 5 to 8 mm. from the midline, shoulder, arm and hand sensations at 8 to 12 mm. from the midline and lower limb sensations at 12 to 17 mm. from the midline.

Gillingham (1962) proposed a new concept of the topographical arrangement of sensory fibres in the internal capsule with the face, upper limb and lower limb fibres arranged in that order from above downwards.

Guiot (1963) confirmed previously reported medio-lateral topographic arrangement. He further reported that diffuse, poorly localised and large area responses from most parts of the body were obtained from the postero-superior part of the nucleus. In the antero-inferior part, precisely localised, sharply defined and small area responses were elicited from (a) lips and corners of the mouth, (b) fingers and hand and (c) toes.

Spiegel and Wycis (1962) studied the spinothalamic tract immediately below the thalamus. They could find no sharp delimitation of topographic representation although they were in general agreement with previous workers.

Bertrand, Hardy and Martinez (1963) presented their data regarding sensory response points on thalamic stimulation. They reported on antero-posterior face-arm by distribution with considerable overlap. Their region of exploration however was 21 mm. behind the anterior commissure, 11 mm. below the intercommissural line and 13 - 14 mm. lateral to the midline. Previous work therefore was in general confusing and contradictory. Different authors have reported face-arm-leg sensory response distribution as lying (a) from the superior to the inferior (b) from the medial to the lateral (c) from the anterior to the posterior planes and (d) so overlapping as no definite classification to be possible.

In the present study the sensory responses noted during and after stereotaxic surgery were divided into three groups.

(a) Sensory responses during operation as a result of mechanical disruption of fibres, stereotaxic biopsy, two-point stimulation, minimal reversible heating lesions and irreversible therapeutic coagulation lesions.

(b) Evoked potentials from peripheral stimuli recorded by intracerebral unipolar microelectrodes.

(c) Postoperative subjective and objective sensory phenomena.

These three groups were considered separately.

During 475 operations, sensory phenomena described in group (a) above were noted in 182 instances. Their classification by site of sensation and laterality of track is given below.

	Lateral distance of track in mm.					
Site of sensory response / Grade: Atlas: X-ray:	Very medial Sl 11.0 (13.5 -11.0)	Medial Sl 13.5 (16.0- 14.0)	Standard Sl 16.0 (19.0- 16.5)	Lateral Sl 18.5 (21.5- 19.5)	Very Lateral Sl 21.0 (24.5- 22.0)	Total
Face only	0	3	22	3	0	28
Face and upper limb	0	3	16	4	0	23
Upper limb only	0	11	87	24	0	122
Upper and lower limbs	0	0	3	5	1	9
Total number of response points	0	17	128	36	1	182
Total number of explorations	6	41	323	95	10	475

The responses were described by the patients mostly as tingling, very often confined to the contralateral face (especially corners of mouth), hand (especially fingers) and lower limb (especially leg and toes). Sometimes the feeling would be described as an electric shock. Rarely a feeling of subjective numbness was noted by the patient although no objective sensory deficit could be demonstrated in the same area. Very rarely objective sensory deficit to testing in the form of hypoesthesia was present. In one case, defective proprioception was noted. In no instance was a sensation of pain, touch, heat, or cold described. The qualitative nature of these responses were in full agreement with those described by other authors.

It was seen from scattergram studies superimposed on the plates of the atlas of Schaltenbrand and Bailey (1959) that these responses were obtained both from the sensory relay nucleus and the thalamoparietal projection.

In view of the medio-lateral somatotrophic arrangement described by most workers, an attempt was first made to classify these responses according to the laterality of their tracks from the midline, after charting each sensory response point and defining its three Cartesian co-ordinates.

Classification in the broad groups outlined in the table above did not give any statistical significance or correlation. An attempt was therefore made to split the groups into smaller ones to see whether any particular trend was discernible within the large groups. This classification is given below.

Site of Response	Lateral distance of track in mm. on X-ray												
	110 to 135	140 & 145	150 & 155	160 & 165	170 & 175	180 & 185	190 & 195	200 & 205	210 & 215	220 & 225	230 & 235	240 & 245	Total
Face only	0	1	0	3	6	11	6	1	0	0	0	0	28
Face & U.L.	0	2	1	2	5	7	2	3	1	0	0	0	23
U.L. only	0	2	0	11	30	33	32	11	3	0	0	0	122
U.L. & L.L.	0	0	0	0	0	0	4	2	1	0	1	1	9
Total no. of responses	0	5	1	16	41	51	44	17	5	0	1	1	182
Total no. of explorations	6	7	13	45	109	123	98	47	17	5	2	3	475

It was seen from this table that the lower limb responses tended to be elicited from more lateral tracks (X-ray range 19.0 mm. to 24.5 mm.; actual range 17.0 mm. to 22.0 mm.). The face and upper limb responses were elicited in more or less equal proportion from tracks in the middle groups (X-ray range 14.0 to 21.5 mm.; actual range 12.5 to 19.5 mm.). There was no significant difference in laterality between the face and upper limb responses as shown by the following figures.

Site of response	Number of observations	Mean in mm. on X-ray	Standard deviation
Face	28	17.96	1.243
Upper limb	122	18.27	1.312
Difference between the means			0.31
Standard error of the difference between the means			0.26

The sensory response points were next classified according to their vertical height above the intercommissural line as shown below.

Medial Tracks (S1 13.5)

Site of response	Number of observations	Mean vertical height in mm.	Standard deviation of entire group
Face	3	8.29	3.36
Upper limb	11	5.34	

Standard Tracks (S1 16.0)

Site of response	Number of observations	Mean vertical height in mm.	Standard deviation
Face	22	6.71	4.01
Upper limb	87	5.14	2.54

Lateral Tracks (S1 18.5)

Site of response	Number of observations	Mean vertical height in mm.	Standard deviation of entire group
Face	3	6.88	2.53
Upper limb	24	4.75	

Application of tests for the significance of the difference between the means in each of these groups did not rule out chance factors causing such difference. It was noted however that in each group, the mean of the face responses stood at a higher level from the intercommissural line than the mean of the upper limb responses. The figures for all the tracks were now considered together and regrouped in mean heights correct to the nearest millimeter to facilitate calculations. The results are tabulated below.

Height in mm. above CA-CP line	Number of sensory responses noted Face	Upper limb
- 2	0	2
- 1	1	3
0	1	0
+ 1	0	6
+ 2	1	7
+ 3	1	12
+ 4	2	20
+ 5	6	14
+ 6	1	24
+ 7	3	15
+ 8	1	7
+ 9	2	6
+10	5	5
+11	0	0
+12	2	1
+13	1	0
+14	1	0
Total	28	122
Mean	7.00	5.082
S.D.	3.78	2.65

Difference between the means 1.918

Standard error of the difference between the means 0.7536

The difference between the means was seen to be more than 2.5 times the standard error.

It was concluded from this study that at the region explored by the present stereotaxic technique, the face and upper limb responses were obtained medially and the lower limb responses were obtained laterally. In the medial region, the face responses were obtained at a higher level with reference to the intercommissural line than the upper limb responses.

Talairach, Tournoux and Bancaud (1960) reported on observations during parietal subcortical stereotaxic surgery

for pain that the somatotrophic arrangement of fibres for the face, upper limb and lower limb was from before backwards in that region.

If the medio-laterally arranged face, upper limb and lower limb fibres of the spinothalamic tract develop an antero-posterior orientation in the subcortical region, one would expect them to turn round in that direction in the thalamoparietal projection. The findings of the present study are in conformity with such a concept.

The mean of the vertical height of the nine sensory response points where lower limb sensations were elicited, was 6.875 mm. and their standard deviation 11.4. In view of their lateral nature in comparison to face and upper limb responses, the heights were not strictly comparable. It is likely, although unproved, that the present tracks of exploration were not sufficiently low in the thalamoparietal projection anteriorly to involve the lower limb fibres as they sweep below the upper limb fibres.

The co-ordinates of the sensory response points were next analysed to see whether there was an anteroposterior somatotrophic arrangement as well. The results are shown below.

Distribution of anteroposterior co-ordinates of sensory response points in relation to the midcommissural plane

Distance in mm.	Number of points		
	Face	Upper limb	Lower limb
-20	1	0	0
-19	0	0	0
-18	0	0	0
-17	0	1	0
-16	0	0	0

-15	0	0	0
-14	1	0	0
-13	0	1	0
-12	1	1	1
-11	1	3	1
-10	2	6	0
- 9	2	9	1
- 8	1	8	0
- 7	3	13	0
- 6	4	13	0
- 5	2	17	1
- 4	1	11	4
- 3	5	7	0
- 2	0	7	0
- 1	1	8	0
0	1	3	1
+ 1	0	6	0
+ 2	2	3	0
+ 3	0	0	0
+ 4	0	1	0
+ 5	0	2	0
+ 6	0	1	0
+ 7	0	0	0
+ 8	0	0	0
+ 9	0	1	0
Total	28	122	9
Mean	-6.214	-4.754	-5.889
S.D.	4.779	4.122	3.919

Difference between the means for face and upper limb 1.46 mm.

Standard error of the difference between the means 0.9772

The difference between the means was less than twice the standard error and was therefore not considered significant.

One also has to remember that when an oblique track parallel to the mid-sagittal plane and moving downwards and forwards crosses a vertically disposed somatotrophic arrangement with the face fibres above those of the upper limb, the former would be encountered at more posterior levels. This may account for the slight difference in the means with respect to the anteroposterior co-ordinates for the face and upper limb sensory response points.

The findings from this study should not be interpreted to mean that the medio-lateral somatotrophic arrangement in the sensory relay nucleus of the thalamus described by other authors was inaccurate. The plane of exploration used by most workers was different from the present one. The sensory response points described in the present study in this section were situated not only in the cells of the sensory relay nucleus but also in the fibres travelling through the sensory nucleus, the nuclei in front of it and the internal capsule. This would explain the wide scatter of these points in the scattergrams. It is also significant, as would be shown in the next section, that the mean of these points for the upper limb was situated anterior to the mean of the evoked potentials for the upper limb from the sensory relay nucleus only.

In one case, two parallel tracks were made for therapeutic purposes at different lateral planes but with otherwise identical measurements. It was seen that face responses were elicited in the medial track while upper and lower limb responses were elicited from the second track

2 mm. lateral to it. The number of patients in whom more than one electrode track was made for therapeutic reasons was very small and therefore no further data was available on multiple track explorations of the same thalamus.

(c) Evoked Potentials from the Thalamus

Chang (1959) defined an evoked potential as the detectable electrical change of any part of the brain in response to deliberate stimulation of a peripheral sense organ, a sensory nerve, a point on the sensory pathway or any related structure of the sensory system.

The technique used in the present study was described in detail in Chapter 2. Previous similar work on the thalamus of animals using microelectrodes and the evoked potential technique has been done by Gaze and Gordon (1952, 1954, 1955), Mountcastle and Henneman (1949, 1952), Rose and Mountcastle (1952), Hunt and O'Leary (1952) and Cohen and Grundfest (1954).

Depth recording in the human thalamus with microelectrodes has been successfully accomplished by Guiot, Hardy and Abbe-Fessard (1962), Ervin and Mark (1960), Hankinson (1961), Bertrand, Hardy and Martinez (1963), Jasper (1963) and Spiegel (1963).

In the present series, depth electrical recording was attempted in 30 cases. In 4, no recording was at all obtained presumably due to undetected faults in the electrical connections or insulations. In the 26 cases where successful recording was obtained, evoked potentials from peripheral stimulation were recorded from the thalamus

in 18 cases. Of these, 7 were from the face and 11 from the upper limb.

One case of this group was operated under local anaesthesia and one had an intravenous drip of methohexitone as well. In the other 16, local anaesthesia was supplemented by intravenous nauroleptanalgesia using dehydrobenzperidol and phenopyridine. No difference was noted in the quality of recorded evoked potentials under the different forms of anaesthesia used.

The quality of the spontaneous background activity changed distinctly as the recording electrode left the pulvinar and entered the LP or VP nucleus. Some microns behind the sensory relay nucleus faint responses to peripheral stimulation were audible and visible. This warned the surgeon to reduce the forward movement each time to a few microns. When the sensory relay nucleus was entered, there was no doubt about the presence of evoked potentials which were quite distinct on peripheral stimulation both in the oscilloscope and in the audioamplifier. The demarcation between the sites where evoked potentials were present and absent was very sharp, of the order of 50-100 microns. This was true both for the entry and for the exit of the recording electrode with reference to the sensory relay nucleus.

All responses recorded were presumably from the sensory relay nucleus and not from the thalamoparietal projection in front, as judged by the charted position of these strips superimposed on the stereotaxic atlas. It

was remarkable how closely they coincided with the position of the sensory relay nucleus of the atlas brain, thus giving further indirect confirmation of the accuracy of the method of charting used and the corrections applied for it.

In no instance were evoked potentials elicitable from the homolateral face or limbs. In almost all of the similar work done on animals, ipsilateral stimulation has produced evoked potentials at a few sites in the thalamus. No stereotaxic surgeon however has yet reported similar ipsilateral response in the human.

In 3 out of the 11 cases where evoked potentials were elicited from the upper limb, the response was mainly to touch on the skin surface. In 2 cases the response was elicited mainly by movement of joints. In the other six, responses were obtained on both exteroceptive and proprioceptive stimuli. Often the positions of the tip of the electrode which responded best to exteroceptive and proprioceptive stimuli were different. From the small number of cases studied there was no suggestive correlation for differential spatial representation for exteroception and proprioception in the sensory relay nucleus of the human thalamus. Similarly, in the face, evoked potentials were sometimes best elicited by maintained pressure than by touch.

The distances over which responses were elicited along the track varied widely from 0.1 mm. to 5.5 mm. in the 18 cases, presumably depending on which part of the nucleus the electrode was passing through. In no instance

were definite responses from both face and upper limb obtained on the same track. Guiot (1963) had one such case in more than a hundred explorations. Probably the direction of exploration in the Guiot-Gillingham technique did not favour this in view of the somatotrophic arrangement in the nucleus. In one case, there was a suggestion that evoked potentials from the upper limb were elicited at a point just behind the area where definite evoked potentials from the face were obtained. This would be in accordance with Guiot's finding and the present author's concept, elaborated later, that the face part of the nucleus is in front of the upper limb representation. However, in view of the fact that the upper limb response in this case was not definite and clear, it was not taken into consideration for scattergram or statistical studies.

When the responses were recorded over a length greater than 0.25 mm. along the track, the area from which evoked potentials were best elicited changed as the recording needle was moved forwards. For example, in one case, initially responses were obtained only from the thumb. A few microns further forwards, evoked potentials were obtainable from the radial three fingers but maximally os from the index finger. Further movement forwards gave evoked potentials from all fingers but maximally from the middle one. More anteriorly, the response from the radial fingers completely disappeared and maximal responses were obtained from the little finger. A few microns further forwards, the electrode recorded stimuli only from the little finger or movement of the wrist.

A similar passage of electrode through the various areas for the face was also observed on several occasions.

Although the number of cases available for study was small, very suggestive results about somatotrophic representation were obtained on statistical analysis. Final acceptance or rejection of these results would depend on the analysis of larger number of cases in future.

The mean heights above the intercommissural line of the central points of the distances over which evoked potentials were elicited for the face and upper limb were first compared, for the plane S1 16.0 (marker in X-ray between 16.5 and 19.0 mm.)

	Face	Upper limb
Mean	6.43 mm.	5.0 mm.
S.D. for entire group	1.936	
No. of cases	7	6

Applying the t-text, $0.5 > P > 0.2$

This suggested that the face responses were perhaps obtained at a higher level than the upper limb responses as was seen in the previous section for sensory response points on stimulation and coagulation. The probability that a difference as big as that observed should arise through chance was however very great. They could not be compared for the plane S1 18.5 (marker on X-ray between 19.5 and 21.5 mm.) since no responses from the face were obtained at this level.

	Face	Upper limb
Mean	---	8.28 mm.
No. of cases	0	5

It is interesting to note that at this level some of the upper limb responses were obtained at a lateral plane but not further down whereas in a more anterior level, (see previous section) all upper limb responses were obtained at a lower plane to the face responses but not further lateral to it. At this level however the lower limb responses were obtained on a more lateral plane but not at a lower level. Although the numbers are too few to draw any definite conclusions, it is suggestive that this is due to the rotation of the fibres as they take up a different position to each other as they pass to the thalamoparietal projection from the sensory relay nucleus.

The lateral distances of the tracks where evoked potentials were elicited were then compared for the face and upper limb.

	Face	Upper limb
Mean (marker in X-ray)	17.79 mm.	19.09 mm.
S.D. for entire group	1.362	
No. of cases	7	11

Applying the t-test, $0.10 > P > 0.05$

The result was near the conventionally accepted level of significance. 6 out of the 11 cases of upper limb responses were obtained on tracks at 19.0 mm. or further lateral while all the 7 face responses were obtained on tracks at 18.5 mm. or further medial. Such a finding suggested that with greater number of cases one may well find a medio-lateral statistically significant difference for the face and upper limb regions in the sensory relay nucleus.

The central points of the distances over which evoked potentials were elicited, were next compared for the face and upper limb for their position behind the mid-commissural plane.

	Face	Upper limb
Mean distance behind Midcommissural plane	6.74 mm.	10.29 mm.
S.D. for entire group	2.365	
No. of cases	7	11

Applying the t-test, $P < 0.01$

This difference was therefore quite significant.

This group of central points of evoked potentials (CPEP) was then compared with the group of sensory response points on stimulation and coagulation (SRPS) which were analysed in the previous section of this chapter. They were compared in all the three Cartesian co-ordinates and for face and upper limb separately.

Mean Height above CA-CP line

Face Responses

	SRPS	CPEP
Mean	7.00 mm.	6.429 mm.
S.D.	3.78	1.043
No. of cases	28	7

Difference between the means 0.571 mm.

Standard Error of the difference between the means 0.8158

Result not significant

Mean Height above CA-CP line

Upper limb Responses

	SRPS	CPEP
Mean	5.082 mm.	6.47 mm.
S.D.	2.65	2.605
No. of cases	122	11

Difference between the means 1.388 mm.

Standard Error of the difference between the means 0.8213

Result not significant

Lateral Distance from Midsagittal Plane

Face Responses

	SRPS	CPEP
Mean (marker in X-ray)	17.96 mm.	17.79 mm.
No. of cases	28	7

Difference between the means 0.17 mm. in X-ray was too small to merit further analysis.

Lateral Distance from Midsagittal Plane

Upper limb Responses

	SRPS	CPEP
Mean (marker in X-ray)	18.27 mm.	19.09 mm.
S.D.	1.312	1.53
No. of cases	122	11

Difference between the means 0.82 mm.

Standard Error of the difference between the means 0.4763

Result not significant

Distance behind Midcommissural Plane

Face Responses

	SRPS	CPEP
Mean	6.214 mm.	6.743 mm.
S.D.	4.779	1.516
No. of cases	28	7

Difference between the means 0.529 mm.

Standard Error of the difference between the means 1.069

Result not significant

Distance behind Midcommissural Plane

Upper limb Responses

	SRPS	CPEP
Mean	4.754 mm.	10.29 mm.
S.D.	4.122	1.647
No. of cases	122	11

Difference between the means 5.536 mm.

Standard Error of the difference between the means 0.6212

Difference between the means was more than eight times the standard error and was therefore highly significant.

Considering the results analysed in the previous and the present sections of this chapter, as well as inspecting the scatter diagrams the following conclusions seem valid or at least highly suggestive. (See figures at end of chapter)

(1) The sensory responses to stimulation and small reversible heating lesions for the upper limb were elicited mainly from the thalamoparietal projection. The evoked potentials for the upper limb were elicited from the sensory relay nucleus.

(2) The sensory responses to stimulation and small reversible heating lesions for the face were elicited from the sensory relay nucleus and probably from the thalamoparietal projection as well. The evoked potentials for the face were elicited mainly from the sensory relay nucleus.

(3) Within the nucleus, the face responses were elicited from sites which were in general superior, medial and anterior to the sites from which upper limb responses were elicited. There was considerable degree of

overlapping between these two regions. It was likely that some of this overlapping was artificial in the sense that the individual anatomical variation was so wide that the figures in the analysis were bound to overlap. On the other hand it seems very significant that under the conditions of recording with the present technique, not even in one patient out of the 17 cases where evoked potentials were recorded, they were done from both the face and upper limb from different parts of the nucleus along the same electrode track.

(4) In the thalamoparietal projection, face responses were obtained from areas above those from which upper limb responses were obtained. The lower limb responses were obtained lateral to both these areas. Again there was considerable overlapping between the different areas.

From these conclusions, one is tempted to speculate a little. Is it likely that in the sensory relay nucleus of the human, the body is represented with the face above, medially and forwards with the lower limb below, laterally and backwards while the upper limb representation lies in between? Is it likely that the fibres arising in the nucleus to form the thalamoparietal projection regroup themselves with the upper limb fibres first getting below those for the face while the lower limb fibres get below those of the upper limb further anteriorly?

Only such a hypothesis can explain all the findings of the present study. Only such a hypothesis can explain why Bertrand and his colleagues (1963) found an antero-posterior arrangement in the thalamus for sensory responses

somatotrophically, while Guiot (1963) found a medio-lateral arrangement and Gillingham (1962) found a supero-inferior arrangement, further forwards.

(d) Sensory Phenomena after Stereotaxic Surgery

In comparison to the vast amount of literature which has been published on stereotaxic surgery, reports of postoperative sensory disturbances have been surprisingly few.

Cooper (1961) found lasting sensory abnormalities to be present in less than 2% of cases in a series of one thousand patients. He observed that it was surprising that so few sensory disturbances were encountered since more than half the cases had relatively large lesions in the ventrolateral region of the thalamus.

Bertrand and Martinez (1962) reported permanent sensory loss over small areas in 3 patients out of a series of 250. Bertrand, Martinez and Hardy (1963) reported again 4 cases of hypoesthesia in a series of 150, a percentage of 2.7.

Markham and Rand (1963) found exteroceptive sensory loss in 2.6% of their patients. All the cases appeared to be in relation to lesions in the posterior part of the thalamic lateral nucleus.

Cassinari, Infuso and Pagni (1963) reported on thalamic lesions for intractable pain. Probably due to the large size of their lesions as well as to the fact that the sensory relay nucleus was itself the target, they found exteroceptive and proprioceptive sensory loss in a

large proportion of their patients.

In a series of publications, Mark, Ervin and Hackett (1960), Mark and Ervin (1961) and Mark, Ervin and Yakovlev (1962, 1963) analysed their findings in stereotaxic operations for intractable pain. They found, in general, comparatively little sensory disturbances after lesions in the sensory relay nucleus. Three distinct clinical syndromes were described by them on clinico-anatomical correlations. The first syndrome was characterised by profound sensory loss with mediocre or little pain relief after thalamotomy. The chief anatomical lesion here was in the sensory relay nuclei of the thalamus. The second syndrome included little or no sensory loss with good pain relief and the main sites of lesions included the parafascicular nucleus and the intralaminar nucleus. The third syndrome of the dorsomedial and anterior nucleus was characterised by a pronounced change in affect with little modification of perception of pain but with development of an indifference to it.

Talairach and his colleagues (1959) reported that subcortical interruption of the thalamoparietal projection relieved painful syndromes but was not followed by a modification of the primary sensibilities or by gnostic disturbances.

Gillingham (1960) achieved supranuclear interruption of the thalamoparietal projection at the level of the posterior limb of the internal capsule with modification of intractable pain without subjective or objective sensory disturbances.

From the perusal of the literature it appears that reports of postoperative sensory distrubances after stereotaxy were rare due to the following reasons.

(a) Most authors used some form of physiological testing before making a permanent lesion and therefore avoided this complication.

(b) Limited damage to the thalamoparietal projection was not associated with subjective or objective sensory disturbances.

(c) It was difficult to produce permanent sensory loss, even deliberately, by small lesions of the thalamic relay nucleus.

(d) Paresthesiae felt for a few days postoperatively, especially if unassociated with objective sensory loss, was not reported by workers as a postoperative complication on longterm assessment.

In the present study, 48 operations out of 448 were followed by some form of sensory disturbance postoperatively. At first sight, this appears excessive (10.7%). The cases however could be classified into the following groups.

Group A: Five patients who did not have any sensory disturbance in the immediate postoperative period but had a delayed onset of tingling of the hands a few months after operation. The tingling was very mild and was reported by the patient, only on specific questioning. It is not clear whether this was due to scar formation or associated irritative lesion at the site of or near the therapeutic lesion. The syndrome appears to be analagous to postoperative pain or paresthesiae after operations on the

spinal cord and peripheral nerves and to postoperative epilepsy after operations on the cerebral hemispheres.

Group B: Four cases followed the reintroduction of the electrode along another track 2 mm. lateral or medial to the first track during the same operation. Apart from sensory loss these cases of multiple electrode insertions were followed by other more serious complications. This practice has now been abandoned.

Group C: Four patients had sensory disturbances as part of a syndrome secondary to a minor hemorrhage at the site of the lesion. They all had associated hemiparesis.

Group D: Thirty five cases belonged to this group.

Group D_1: In eighteen patients of this subgroup the sensory disturbances cleared up completely within a few days.

Group D_2: Eleven patients of this subgroup had total resolution of their sensory disturbances within two years after operation.

Group D_3: Six patients belonged to this group. Four of these had subjective sensory features without objective deficit. Two had both objective and subjective loss. In all six, the complication persisted for more than two years. This is the group usually reported in the literature as having sensory complications after stereotaxy and amounts to 1.34% of the present series.

The most important point which became obvious on the study of these cases was that in thirty four cases of group D which were operated under local anaesthesia, in twenty-seven the sensory disturbance could perhaps have been

avoided. In these 27 cases, coagulation at one site was continued despite tingling or numbness during stimulation or coagulation because tremor was also abolished simultaneously and completely. All the six patients with permanent deficit belonged to this group.

A consideration of the Gillingham electrode track makes the reason for the difficulty obvious. Normally when the tip of the electrode is passed to the radiological thalamic target its coagulating portion should be clear of the sensory relay nucleus. However, if the electrode swung medially due to faulty midsagittal plane alignment, or, if extreme anatomical variations existed, the electrode could lie with part of its coagulating surface in the oral ventral nucleus and part in the sensory relay nucleus. Coagulation at the radiological thalamic target therefore would abolish tremor and at the same time produce sensory features.

This difficulty could not be solved simply by moving the electrode further forward. For the electrode might not have been in the sensory relay nucleus at all, the tingling sensation arising from the thalamoparietal projection. In fact, moving the electrode forward would then take the coagulating portion out of the oral ventral nucleus. Gillingham (1961) overcame this difficulty by moving his electrode track further upwards, altering his thalamic target from 3 mm. to 5 mm. above the intercommissural line on the X-ray. This made the electrode reach the target above the sensory relay nucleus and the thalamoparietal projection and thereafter sensory phenomena

during operation were quite rare.

The present study confirms these ideas statistically, as shown in the following table.

Type of case	No. of cases	Mean height of intersection of electrode track with mid-commissural plane (in mm. above CA-CP line in postoperative chart)	Standard Deviation
(1) Cases without any postoperative sensory features	400	3.76 mm.	2.337
(2) Cases with immediate postoperative sensory features not attributable to hemorrhage or multiple electrode insertion	35	2.89 mm.	2.124
(a) sensory features temporary	29	3.10 mm.	1.934
(b) sensory features permanent	6	1.83 mm.	2.858
(3) Cases with delayed onset of tingling	5	3.80 mm.	5.550
(4) Cases where sensory features could be attributed to hemorrhage or multiple electrode insertion	8		
Total number of operations	448		
Track advocated at present by Gillingham		4.5 mm. (5.0 mm. on X-ray)	

Groups (1) and (2) were compared.

Difference between the means was 0.87 mm.

Standard error of the difference between the means was 0.38

The difference was therefore considered significant.

The analysis of the co-ordinates for evidence as to somatotrophic distribution was unrewarding in these postoperative cases as seen in the following table.

Site of sensory phenomenon	Number of cases	Mean lateral distance of track (marker in X-ray)	Mean height of intersection above CA-CP line of electrode track with plane midway between Thalamic target and posterior commissure
Face only	4	18.75 mm.	5.72 mm.
Face and upper limb	14	18.39 mm.	6.16 mm.
Upper limb only	13	18.23 mm.	5.77 mm.
Upper and lower limbs	4	20.00 mm.	4.10 mm.

This table further suggested that the lower limb distribution in the sensory relay nucleus is below and lateral to the face and upper limb. The number of cases however were too few to apply statistical tests to confirm this. The difference between the means for the face and upper limb cases was too small for any opinion to be suggested. In fact, the means were remarkably close together considering the small number of cases studied. This confirms the impression of other workers that a relatively large lesion in the lateral part of the sensory relay nucleus is needed to produce any sensory deficit at all.

(e) Visual Sensations during Stereotaxic Surgery

Ten patients in this series experienced sensations of 'flashes of light' during stimulation or coagulation at the pallidal target. A study of the scattergram of these points suggested that the phenomenon was probably due to spread of current or heat to the optic tract due to inaccurate alignment or anatomical variations. This phenomenon has not been observed since November 1959 during the last 390 consecutive operations.

One patient with a familial tremor reported sensation of 'flashes of light' in both eyes during coagulation a few mm. (4 and 12 on the two sides) behind the pallidal target and this sensation persisted for a few days post-operatively. Presumably this was due to involvement of the optic radiation in the posterior limb of the internal capsule.

(f) Summary

Sensory features with reference to stereotaxic surgery can appear either during operation or after it. During operation, they may be the result of stimulation or coagulation. They could also be specifically located by recording evoked potentials with microelectrodes.

Sensory responses as a result of stimulation and coagulation could arise either from the relay nucleus or the thalamoparietal projection. Evoked potentials and postoperative phenomena were due mainly to the nucleus.

Comparison of the Cartesian co-ordinates of these points, statistically, suggests that in the nucleus there

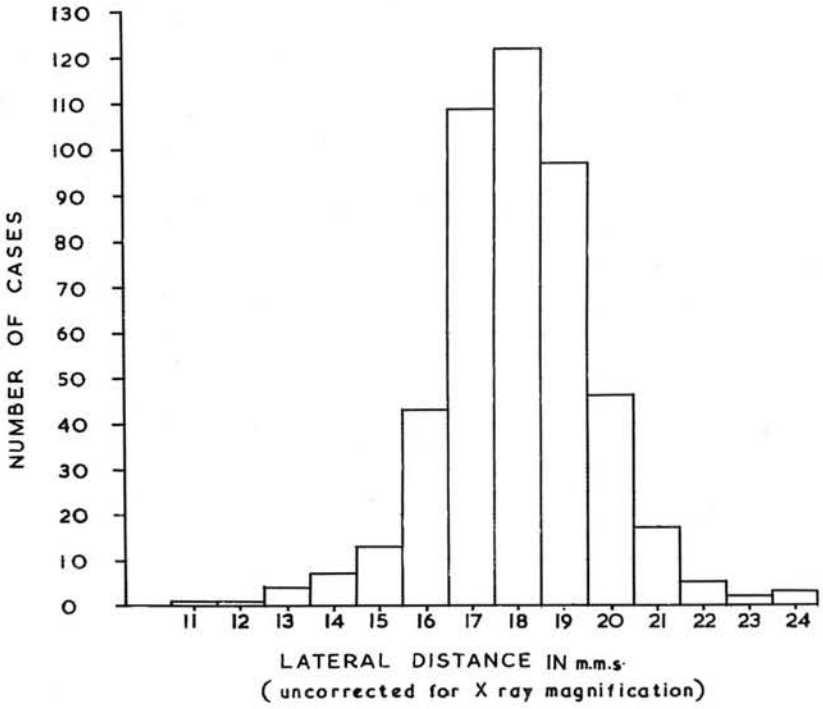
is a definite though overlapping somatotrophic representation. Face was represented above, medially and forwards while the lower limb was represented below, laterally and probably behind. The representation for the upper limb lay in between.

There is evidence to suggest that the fibres in the thalamoparietal projection are arranged from above downwards for the face, upper limb and lower limb in that order. At this level, there seems to be no lateral or anteroposterior somatotrophic arrangement.

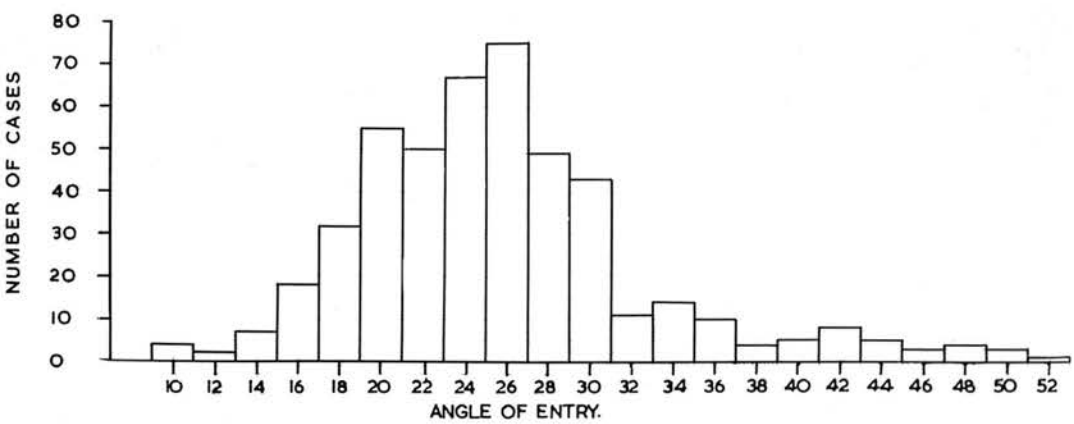
Post-operative sensory deficit was largely due to persisting in coagulation at a site despite 'warning' signs of tingling reported by the patient. There is statistical support for overcoming this difficulty by moving the thalamic target upwards. The lower the track in this operation, the greater were the probability and severity of postoperative sensory disturbances.

Visual sensations during stereotaxy were almost always due to inaccurate alignment or leakage of current.

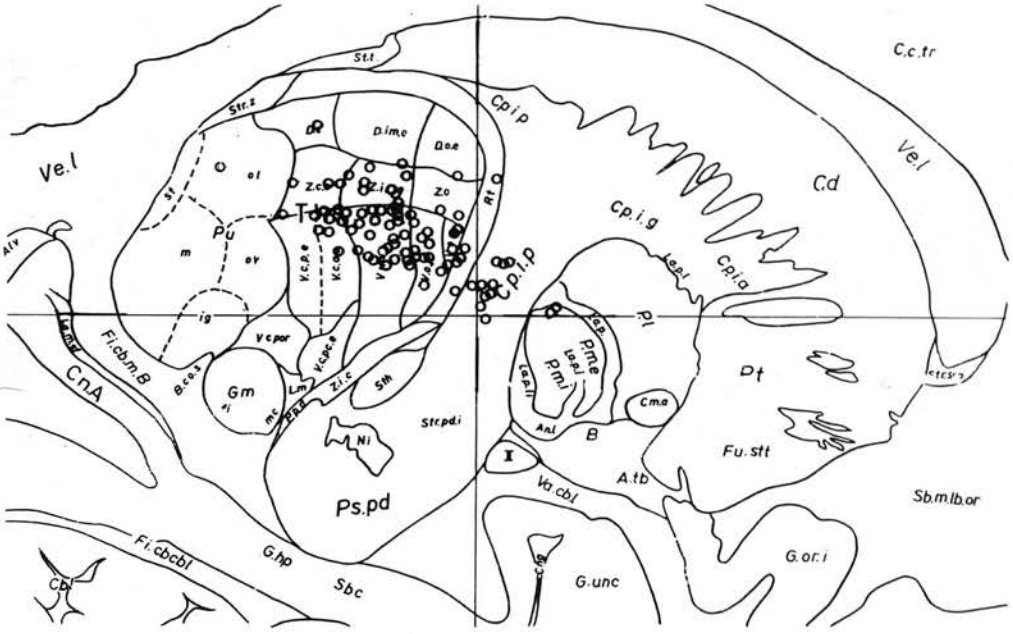
HISTOGRAM SHOWING FREQUENCY DISTRIBUTION OF THE LATERAL DISTANCE FROM THE MID-VENTRICULAR LINE OF THE TRACK OF THE ELECTRODE IN 470 OPERATIONS.

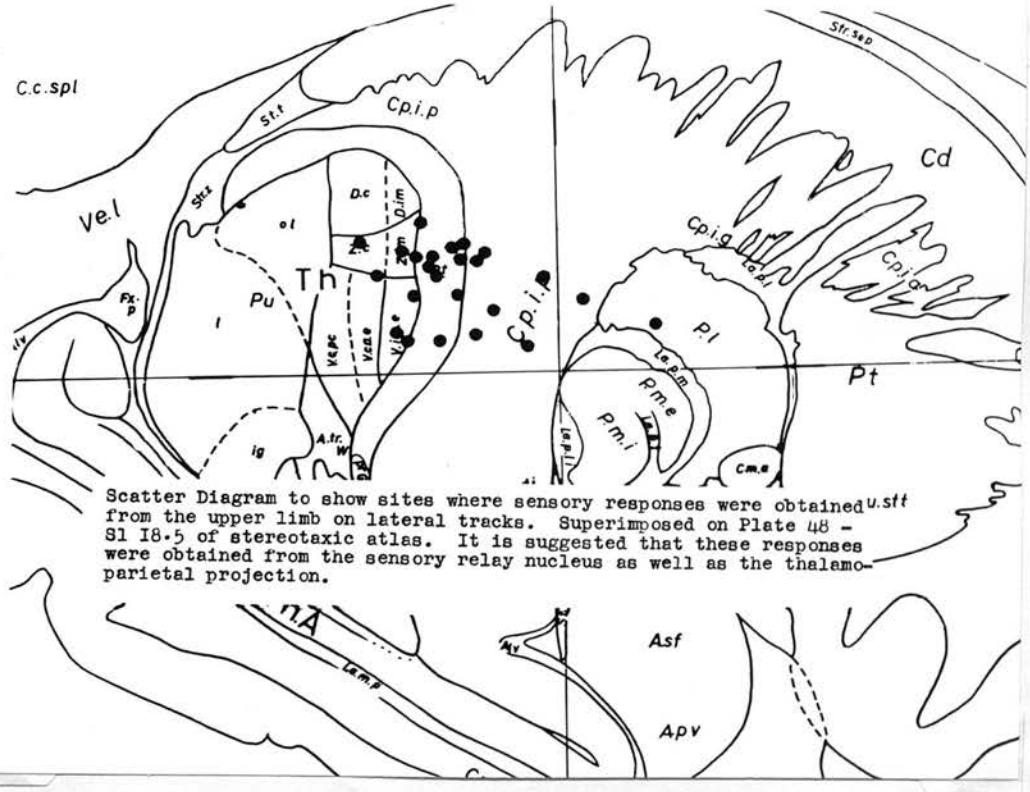


HISTOGRAM SHOWING FREQUENCY DISTRIBUTION OF THE ANGLE OF INCLINATION OF THE ELECTRODE TRACK WITH THE INTERCOMMISSURAL LINE IN 470 OPERATIONS.



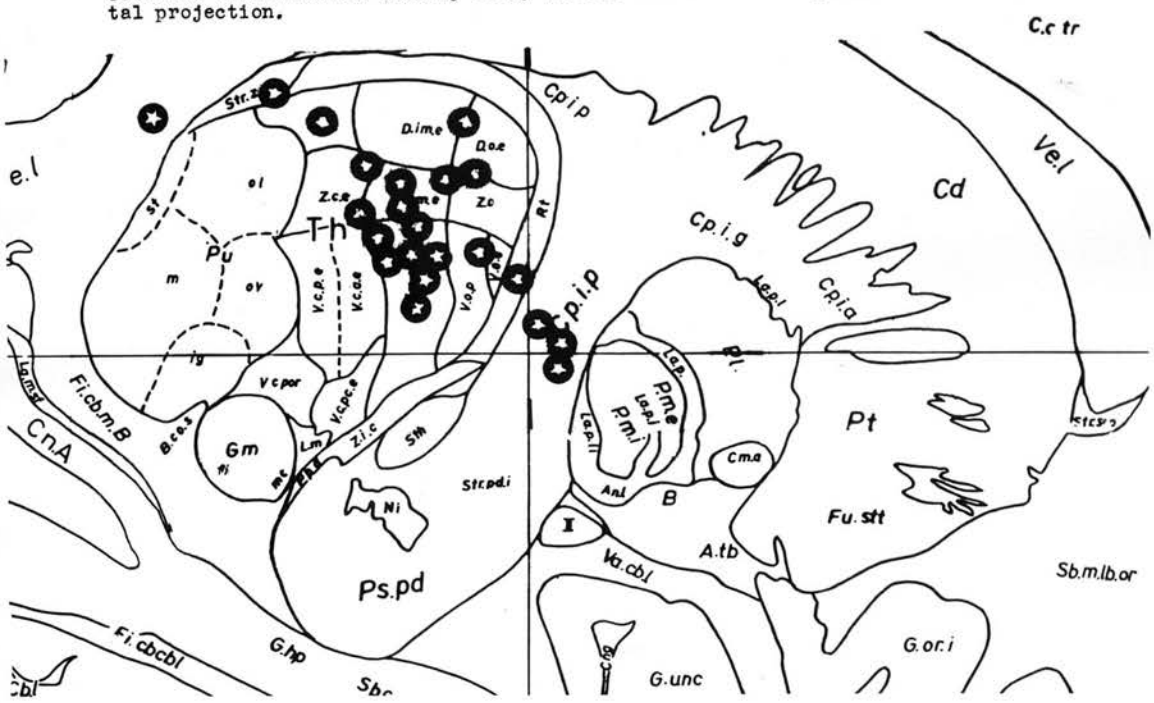
Scatter diagram showing sites where sensory responses were obtained from the upper limb on stimulation and creation of reversible and irreversible lesions. (Superimposed on Plate 48 - Sl I6 of stereotaxic atlas.



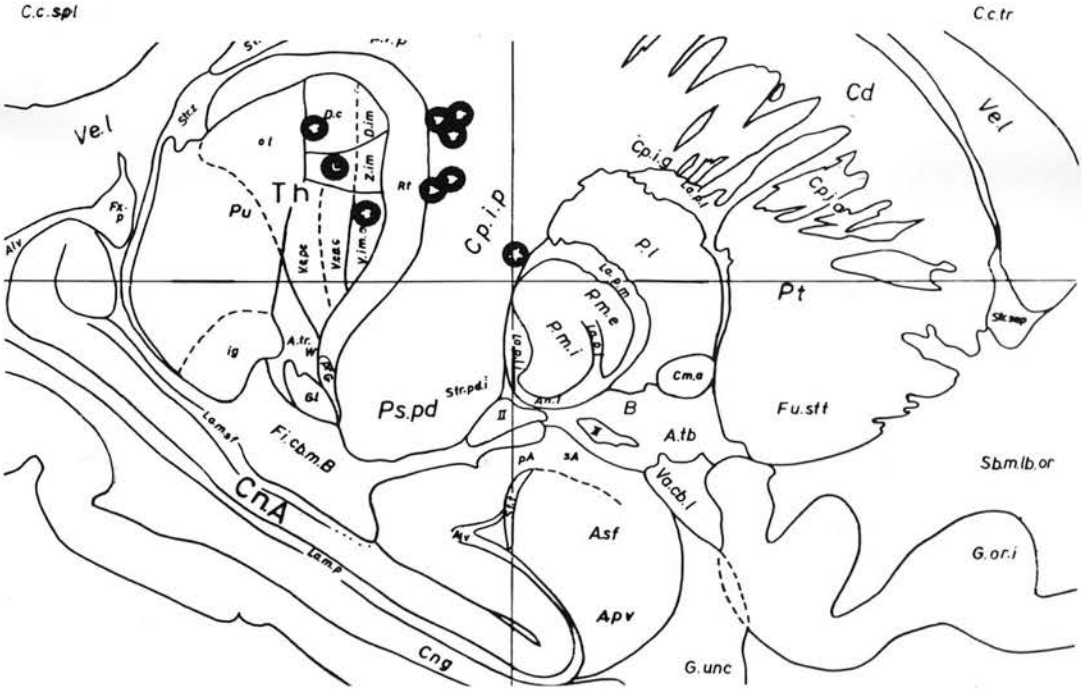


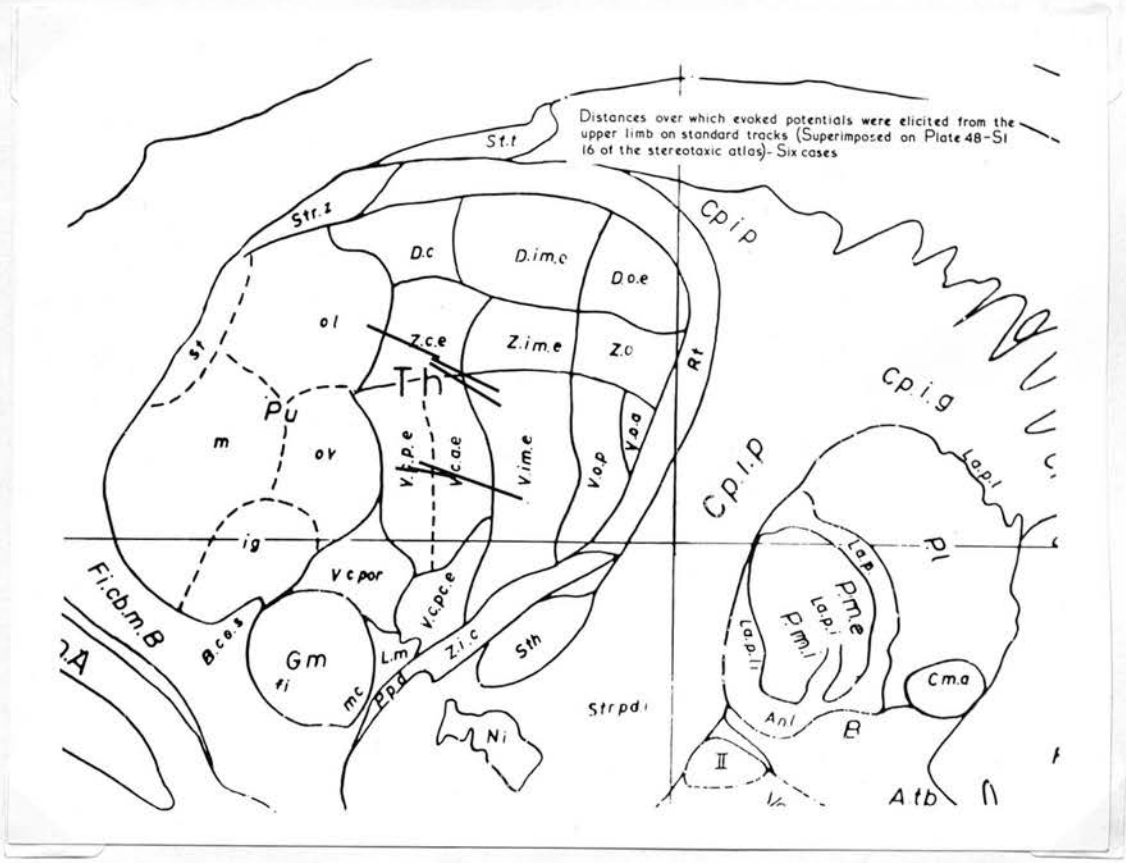
Scatter Diagram to show sites where sensory responses were obtained from the upper limb on lateral tracks. Superimposed on Plate 48 - Sl I8.5 of stereotaxic atlas. It is suggested that these responses were obtained from the sensory relay nucleus as well as the thalamo-parietal projection.

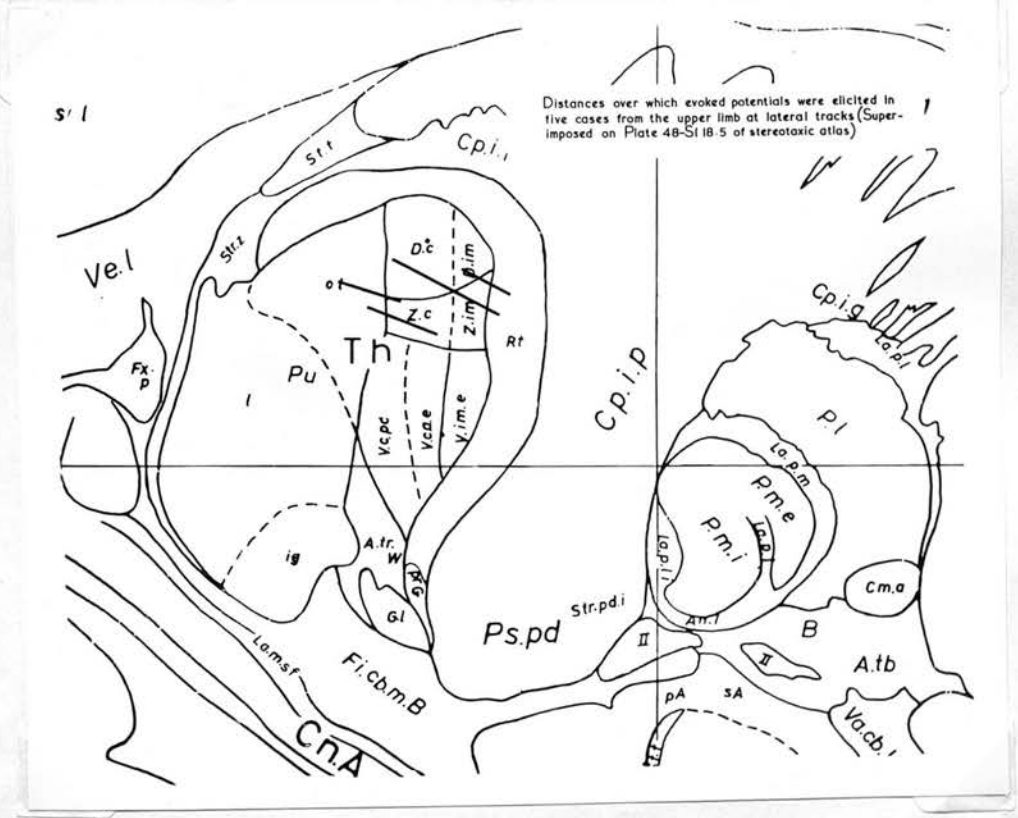
Points where sensory responses were obtained from the face on stimulation and creation of reversible and irreversible lesions. Scatter diagram superimposed on Plate 48 - Sl I6 of stereotaxic atlas. Total 22 cases. It is suggested that these responses were obtained both from the sensory relay nucleus and the thalamoparietal projection.

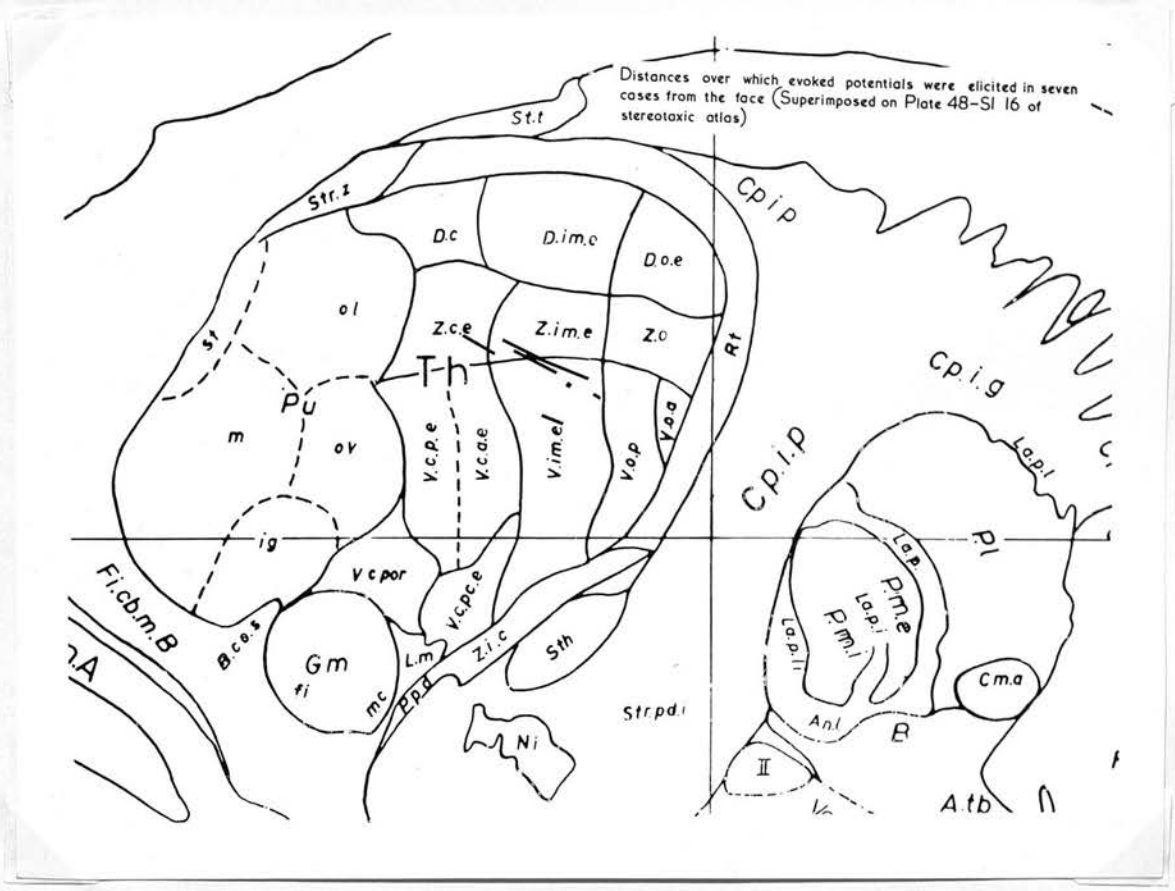


Sites where sensory responses were obtained from the lower limb in nine cases on stimulation and creation of reversible and irreversible lesions. Scatter diagram superimposed on Plate 48 - Sl I8·5 of stereotaxic atlas. It is suggested that these responses were obtained both from the sensory relay nucleus and the thalamoparietal projection.

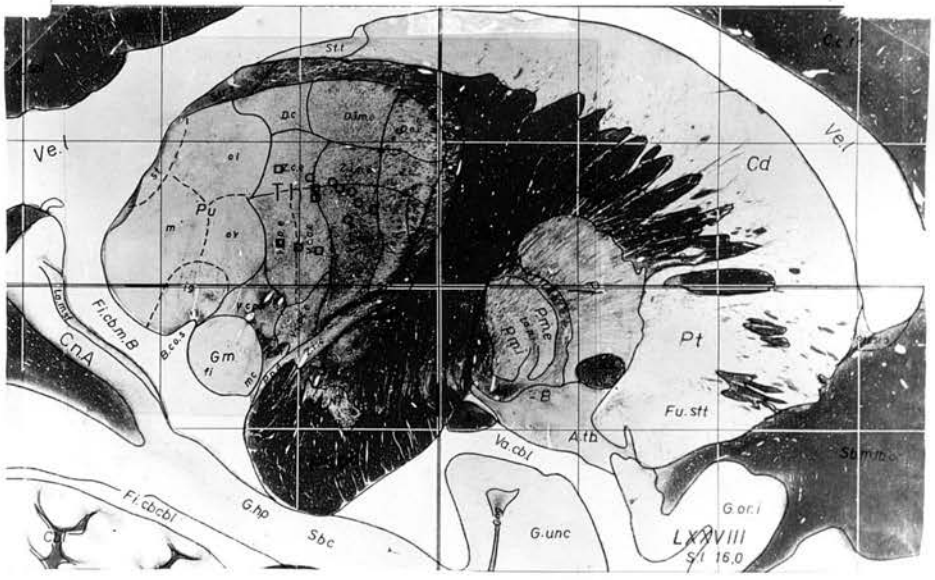


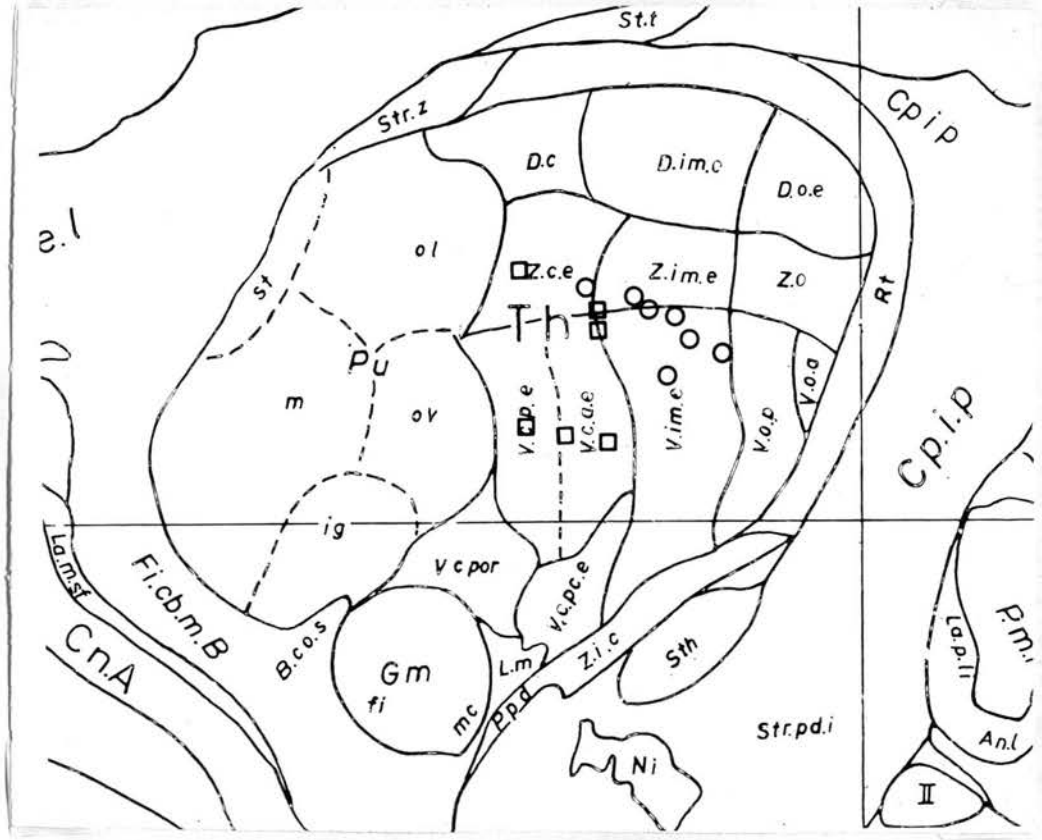

Distances over which evoked potentials were elicited from the upper limb on standard tracks (Superimposed on Plate 48-SI 16 of the stereotaxic atlas)- Six cases
St.t
Str.z
D.c
D.im.c
D.o.e
Z.c.e
Z.im.e
Z.o
Rt
Th
Pu
m
ol
ov
ig
V.c.p.e
V.c.a.e
V.im.e
V.o.p
V.o.a
V.c.por
V.c.pc.e
Z.i.c
Sth
Gm
fi
mc
L.m
Pp.d
Ni
Str.pd.i
Fi.cb.m.B
B.c.e.s
Cp.i.p
Cp.i.g
La.p.l
Pl
La.p
Pm.e
La.p.i
Pm.l
La.p.l
Cm.a
An.l
B
II
A.tb


Distances over which evoked potentials were elicited in five cases from the upper limb at lateral tracks (Superimposed on Plate 48-SI 18-5 of stereotaxic atlas)
St.t
Cp.i.
Ve.l
Str.z
D.c
D.im
o.t
Z.c
Z.im
Rt
Th
Pu
Fx.p
Vc.pc
Vca.e
V.im.e
ig
A.tr.W
Gl
Ps.pd
Str.pd.i
Lam.sf
Fi.cb.m.B
CnA
Cp.i.p
Cp.i.g
La.p.l
Pl
La.p.m
Pm.e
Pm.i
La.p.l
La.p.l
Cm.a
An.l
B
II
A.tb
pA
sA
Va.cb.l



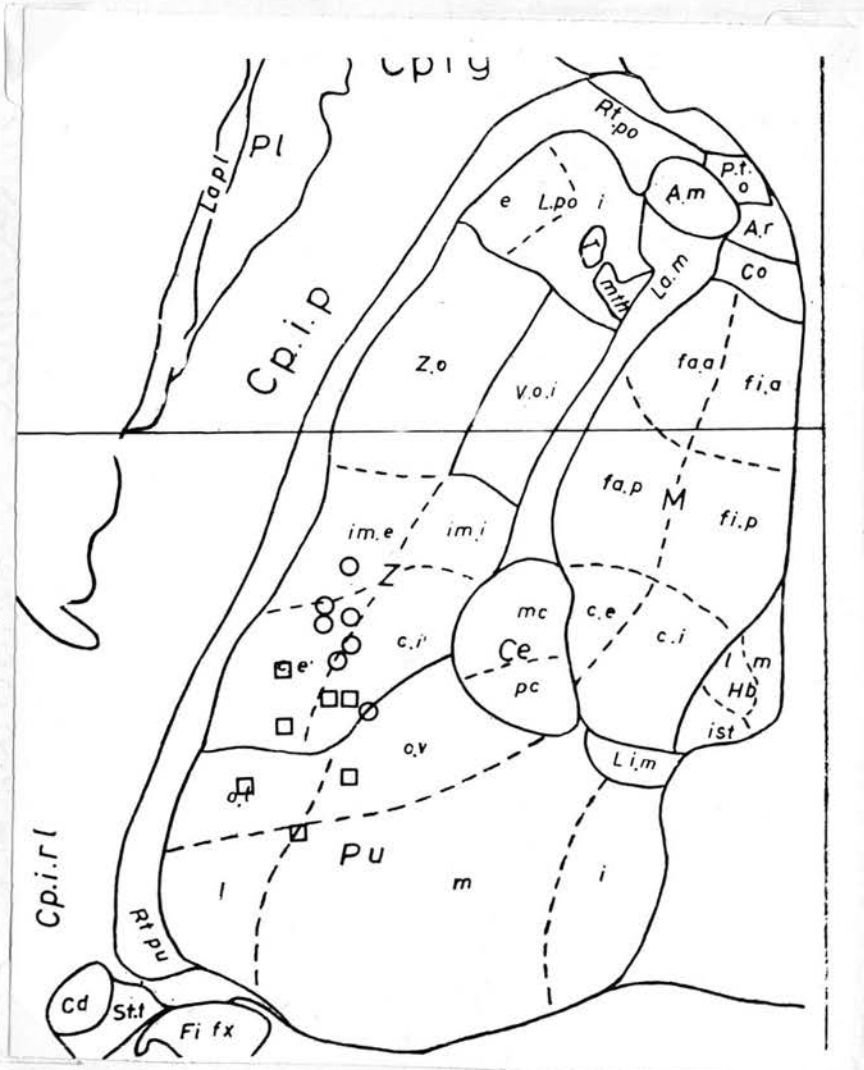
Central points of sites where evoked potentials were elicited at parasagittal plane 16 mm. from midline (Superimposed on Plate 48 S1 I6 of stereotaxic atlas). Face responses shown as circles and upper limb responses as squares. It is suggested that the face fibres end in an area above and in front of the area where upper limb fibres end in the sensory relay nucleus at plane S1 I6.





Scatter diagrams of central points of distances over which evoked potentials were elicited superimposed on plates of stereotaxic atlas. Circles represent responses from the face and squares represent responses from the upper limb.

Left: Sl 16 (Parasagittal section 16 mm. lateral to midsagittal plane)

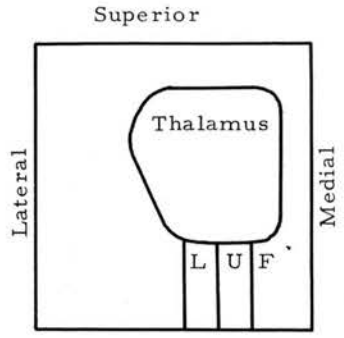


Right: Hd + 6.5 (Horizontal section 6.5 mm. above intercommissural line)

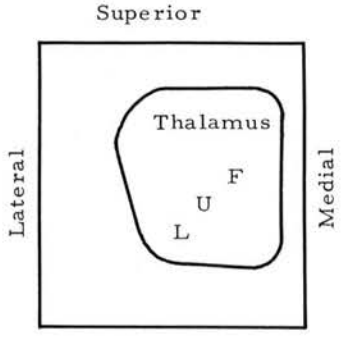
Diagrammatic Representation of the Relative Position of the Face, Upper Limb and Lower limb Fibres in the Spinothalamic Tract, Sensory Relay Nucleus and Thalamoparietal Projection.

Four Frontal Sections are shown from behind forwards.

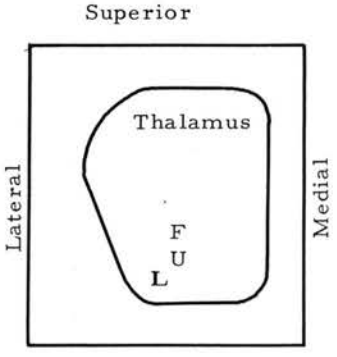
F - Face fibres: U - Upper limb fibres: L - Lower limb fibres.



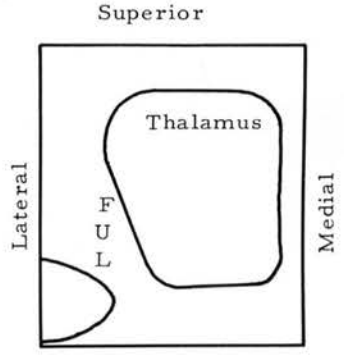
(1) In the spinothalamic tract.



(2) In the sensory relay nucleus



(3) In front of the relay nucleus



(4) In the internal capsule

It is suggested from the findings during stereotaxic surgery that the mediolateral arrangement for the face, upper limb and lower limb changes to a superoinferior arrangement between the spinothalamic tract just below the thalamus and the thalamoparietal projection just in front of the thalamus.

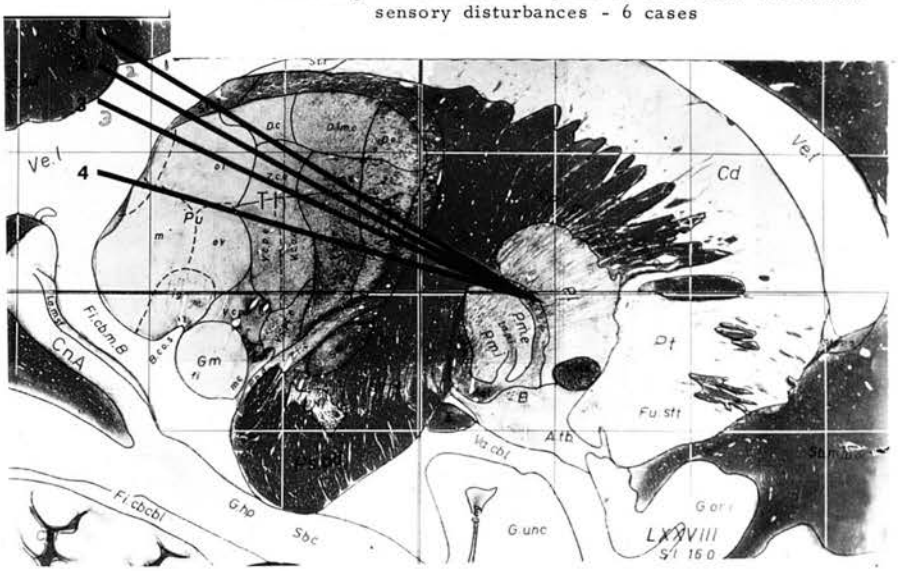
Diagram to show relationship of height of electrode track to incidence of postoperative sensory complications (Superimposition of mean tracks of postoperative charts on nuclear outline of Plate 48-S1 I6 of stereotaxic atlas).

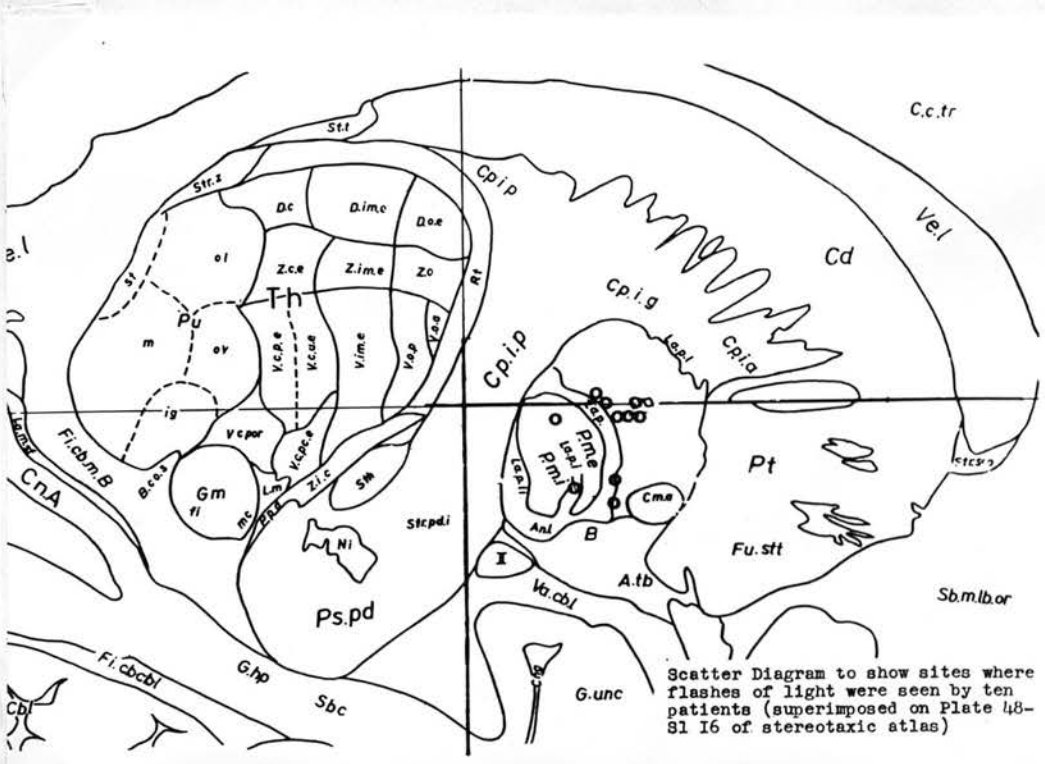
Track 1-Crossing midcommissural plane at 4.50 mm. Present track

Track 2-Crossing midcommissural plane at 3.76 mm. No sensory disturbances - 400 cases

Track 3-Crossing midcommissural plane at 3.10 mm. Temporary sensory disturbances - 29 cases

Track 4-Crossing midcommissural plane at 1.83 mm. Permanent sensory disturbances - 6 cases





Scatter Diagram to show sites where flashes of light were seen by ten patients (superimposed on Plate 48-S1 I6 of stereotaxic atlas)

REFERENCES

1. Bertrand, C. and Martinez, N. (1962). Confin. Neurol., 22, 274.

2. Bertrand, C., Martinez, N. and Hardy, J. (1963). J. Neurol. Neurosurg. Psychiat., 26, 552.

3. Cassinari, V., Infuso, L. and Pagni, C.A. (1963). Excerpta Medica International Congress Series No. 60. Second European Congress of Neurological Surgery. 119.

4. Chang, H.T. (1959). In Field, J., Magoun, H.W. and Hall, V.E. Ed. Handbook of Physiology, American Physiological Society. I, 1, 299.

5. Cohen, S.M. and Grundfest, .. (1954). J. Neurophysiol., 17, 193.

6. Cooper, I.S. (1961). Parkinsonism. Its Medical and Surgical therapy, C.C. Thomas, Springfield, Illinois. 117.

7. Ervin, F.R. and Mark, V.H. (1960). Arch. Neurol., 3, 368.

8. Gaze, R.M. and Gordon, G. (1952). J. Physiol., 118, 48P.

9. Gaze, R.M. and Gordon, G. (1954). Quart. J. exp. Physiol., 39, 279.

10. Gaze, R.M. and Gordon, G. (1955). Quart. J. exp. Physiol., 40, 187.

11. Gillingham, F.J. (1960). Personal Communication.

12. Gillingham, F.J. (1961). Personal Communication.

13. Gillingham, F.J. (1962). Confin. Neurol., 22, 385.

14. Gley, E. quoted by Bing, R. In preface to 'Bing's Local Diagnosis in Neurological Diseases', H. Kimpton, London. 1956. 9.

15. Guiot, G., Hardy, J. and Albe-Fessard, D. (1962).	Neurochirurgia, 5, I, I.
16. Guiot, G. (1963).	Personal Communication.
17. Hankinson, J. (1961).	Proc. Roy. Soc. Med., 54, 380.
18. Hassler, R. and Riechert, T. (1959).	Arch. Psychiat. Nervenkr., 200, 93.
19. Hunt, W.E. and O'Leary, J.L. (1952).	J. Comp. Neurol., 97, 491.
20. Jasper, H. (1963). quoted by Gillingham, F.J.	Personal Communication.
21. Mark, V.H. and Hackett, T.P. (1959).	Tr. Am. Neurol. A., 84, 92.
22. Mark, V.H., Ervin, F.R. and Hackett, T.P. (1960).	Arch. Neurol., 3, 351.
23. Mark, V.H. and Ervin, F.R. (1961).	Excerpta Medica International Congress Series No. 36. Second International Congress of Neurological Surgery. E 114.
24. Mark, V.H., Ervin, F.R. and Yakovlev, P.I. (1962).	Confin. Neurol., 22, 238.
25. Mark, V.H., Ervin, F.R. and Yakovlev, P.I. (1963).	Arch. Neurol., 8, 528.
26. Markham, C.H. and Rand, R.W. (1963).	Arch. Neurol., 8, 621.
27. Mountcastle, V.B., and Henneman, E. (1949).	J. Neurophysiol., 12, 85.

28. Mountcastle, V.B. and Henneman, E. (1952). J. Comp. Neurol., 97, 409.

29. Rose, J.E. and Mountcastle, V.B. (1952). J. Comp. Neurol., 97, 441.

30. Schaltenbrand, G. and Bailey, P. (1959). Introduction to stereotaxis with an atlas of the human brain. Thieme, Stuttgart, 2.

31. Spiegel, E.A. and Wycis, H.T. (1962). Stereoencephalotomy. Part II. Clinical and physiological applications. Grune and Stratton, New York. 230.

32. Spiegel, E.A. (1963), in discussion on Spiegel, E.A., Wycis, H.T., Szekely, E.A., Adams, J., Flanagan, M. and Baird III H.W. J. Neurosurg., 20, 871.

33. Talairach, J., Tournoux, P., Bancaud, J. and Djahanachahi, Dj. (1959). Neuro-Chirurgie, 5, 1, 130.

34. Talairach, J., Tournoux, P. and Bancaud, J. (1960). Acta Neurochir., 8, 153.

CHAPTER 5

STUDIES OF THE MOTOR SYSTEM

"You choose a hard question and one which the bedside is far better placed to solve than is the laboratory. I think the Pyramidal tract is such a HUMAN feature"

.... Sir Charles Sherrington.

"The researches of many eminent scientific men have thrown so much darkness on the subject that if they continue their researches we shall soon know nothing."

.... Artemus Ward.

"Is there a pyramidal tract?"

.... Paul Bucy.

(a) Motor Phenomena during Stereotaxic Surgery

Parkinson (1817) noted in his classical essay that the tremulous agitation ceased in the paralysed limbs during the period in which they were paralysed. Before the advent of stereotaxy many efforts at surgical treatment of the syndrome were aimed at deliberate interruption of the corticobulbar and corticospinal pathways at some level.

After the establishment of pallidal and thalamic surgery for treatment of the syndrome, one school led by Spiegel and Wycis (1952) believed that no risk whatever should be taken of the possibility of production of a capsular lesion. These surgeons preferred to work within safety zones which allowed for maximal anatomical variation in the position of the posterior limb of the internal capsule. The other extreme school led by Bucy (1958) believed that all stereo taxic lesions achieved their therapeutic effect by interfering with the pyramidal tract in the capsule. The majority of neurosurgeons, however, were content to

define the position of the internal capsule by stimulation and to avoid the creation of a lesion where motor responses were provoked.

Accumulating experience soon showed that the problem could not be defined in such simple terms. Although Guiot and his colleagues (1959) claimed precise delimitation of the capsule along their electrode track by stimulation, the majority of workers felt that stimulation was not hundred per cent reliable as an agent for avoiding the capsule. Motor responses could not be equated at all times with the electrode in the capsule and stimulation in the capsule did not always evoke motor responses.

Hassler and Riechert (1961) showed that simple motor responses and various other types of associated movements and phenomena could be produced by stimulation of various parts of the thalamus, capsule and pallidum. Although the experience of these workers involved the largest number of stereotaxic cases studied by stimulation, their results could not be totally accepted for two reasons. They did not produce any statistical evidence for their conclusions and reported as if their findings were true in all their cases. They often used a distance of 5 to 7 mm. between the two poles of their stimulating electrode and accurate localisation was then questionable.

Smith (1962) published very important findings from autopsy studies of cases operated by stereotaxy. She demonstrated clearly, what has been suspected by many stereotaxic surgeons for some time, that limited destruction of the posterior limb of the internal capsule was not

necessarily associated with clinical evidence of hemiplegia.

Gillingham (1962) presented for the first time in stereotaxic surgery, a series of cases where deliberate trans-capsular lesions were made. These patients had no clinical evidence of hemiparesis and had good relief of tremor and rigidity. Charting of the lesion showed that it was very difficult to confine it to the capsule without some involvement of the thalamus or pallidum. However it was proved beyond doubt that his transcapsular lesions were not associated with capsular hemiplegia.

Hughes (1963a) presented for the first time, clinical evidence with statistical support that motor responses could be elicited from the lateral thalamic area.

Evidence thus slowly began to gather that movements could be elicited from outside the capsule while lesions within the capsule were not always associated with motor features. The time was therefore ripe for a radical reorganisation of classically accepted ideas of the structure and function of the posterior limb of the internal capsule, and for a clearing of the misconception that the posterior limb of the internal capsule was almost synonymous with the pyramidal tract.

Gillingham (1960), Guiot and his colleagues (1959), Hassler (1962) and Talairach and Szikla (1963) believed that the corticobulbar and corticospinal tracts were confined only to a small part of the posterior limb of the internal capsule.

The present study of the motor responses elicited during stereotaxic surgery was done on 475 second stage stereotaxic procedures, during which motor phenomena were observed on 153 occasions. This number excluded those cases where paresis was observed due to a presumed minor hemorrhage at the site of the lesion. The central point of the electrode was taken when an effect was produced by mechanical disruption of minimal heating lesion. In case of coagulation, consideration was given to the fact that the lesion extended anterior to the electrode but not behind it and therefore the central point of the lesion and not the electrode was taken. This rule was applied throughout the present study - for sensory and motor responses as well as for tremor and rigidity.

A very striking feature became obvious on plotting these points on the stereotaxic atlas of Schaltenbrand and Bailey. The points could be sharply distinguished into two large groups, one which was well inside the thalamus and behind the thalamocapsular junction and the other which was close to the pallidum in the internal capsule. The thalamic movements were distinguished from the capsular movements on the following criteria.

(1) Thalamic movements were elicited several mm. behind the radiological thalamic target.

(2) They were often elicited at or very near points where sensory responses were elicited, from points which lay in the sensory relay nucleus on charting.

(3) They were never associated with facial palsy, weakness of handgrip or extensor plantar response.

(4) Coagulation at a site where a thalamic movement was elicited was never associated with motor deficit or pyramidal signs.

(5) Stimulation or coagulation for several mm. in front of the points where thalamic movements were elicited provoked no motor phenomena. When the corticobulbar and corticospinal tracts were reached by the electrode just in front of the pallidal target, motor reactions and/or deficit could be induced by stimulation and coagulation. This zone of free interval between thalamic and capsular movements was repeatedly demonstrated along the same electrode track during many operations confirming the belief that these were two different types of motor reactions elicited from different parts of the brain.

Despite the above clinical and anatomical evidence to differentiate motor phenomena into those of thalamic and capsular origin, it could still be argued that the individual variation in the position of the posterior limb of the internal capsule was such that a wide range in motor response points was not unexpected.

Statistical evidence however showed that this argument was untenable. The figures analysed below, show clearly that it was extremely unlikely that all muscle twitches elicited during stereotaxy were of capsular origin.

Twentyfour cases were taken as a 'control' group where the borders of the internal capsule had been defined by audiomonitoring. The midpoint between the thalamocapsular junction and the pallidocapsular junction thus defined could be taken as a midcapsular point with three Cartesian

co-ordinates. A consideration of these twentyfour midcapsular points gave a very reasonable guide to the position of the posterior limb of the internal capsule and the variability in its position.

These points were then compared with 116 points where muscle twitches were elicited during stereotaxic surgery. (The term 'muscle twitch' is used in the subsequent discussion to include all motor contractions occurring in the contralateral half of the body during stimulation, mechanical irritation by needle insertion or biopsy, and creation of reversible and irreversible lesions. The term does not include (a) pareses, (b) weakness or (c) muscle contractions occurring after the stimulation, coagulation, etc., have stopped, e.g. ballistic or athetoid movements continuing after creation of a lesion.)

The first essential to be established was that the two samples were comparable in their lateral distances of electrode tracks. The mean lateral co-ordinates of these points were compared as shown by the position of the opaque marker in the postoperative film.

	No. of cases	Mean lateral co-ordinate in mm. uncorrected for X-ray	S.D.
Midcapsular points defined by audio-monitoring	24	18.083	1.275
Muscle twitch points during stereotaxic surgery	116	18.664	1.884

Difference between the means 0.581mm.

Standard error of the difference between the means 0.3134

The difference between the means was less than twice the standard error and was therefore not considered significant. There was no reason to assume that the samples were different.

The antero-posterior co-ordinates were then compared for the midcapsular points defined by audiomonitoring and the muscle twitch points.

	No. of cases	Mean antero-posterior co-ordinate on chart	S.D.
Midcapsular points	24	+ 2.458 mm.	2.084
Muscle twitch points	116	- 1.810 mm.	5.478

Difference between the means 4.268 mm.

Standard error of the difference between the means 0.663

The difference between the means was more than six times the standard error.

It was therefore quite obvious that the whole group of muscle twitch points could not be equated to the posterior limb of the internal capsule. The frequency distribution of the AP co-ordinates of these 116 points also suggested that these responses showed a wide and irregular scatter.

Antero-posterior co-ordinates of muscle twitch points in mm. with reference to the midcommissural plane.	No. of cases
-12	2
-11	0
-10	3
- 9	2
- 8	4
- 7	10
- 6	11
- 5	7

- 4	16
- 3	11
- 2	5
- 1	12
0	3
+ 1	1
+ 2	4
+ 3	3
+ 4	1
+ 5	3
+ 6	3
+ 7	5
+ 8	1
+ 9	5
+10	3
+11	1

Clinical classification of this group into thalamic and capsular twitches revealed the following distribution.

Frequency distribution of anteroposterior co-ordinates with reference to the mid-commissural plane for thalamic twitches

Co-ordinate in mm.	No. of cases	
-12	2	
-11	0	
-10	3	
- 9	2	Mean - 4.818 mm.
- 8	4	S.D. 2.771
- 7	9	
- 6	11	
- 5	7	
- 4	15	
- 3	9	
- 2	5	
- 1	8	
0	1	
+ 1	0	
+ 2	1	
Total	77	

Frequency distribution of anteroposterior co-ordinates with reference to the mid-commissural plane for capsular twitches

Co-ordinate in mm.	No. of cases		
- 7	1		
- 6	0		
- 5	0		
- 4	1		
- 3	2		
- 2	0	Mean	+ 4.128
- 1	4	S.D.	4.578
0	2		
+ 1	1		
+ 2	3		
+ 3	3		
+ 4	1		
+ 5	3		
+ 6	3		
+ 7	5		
+ 8	1		
+ 9	5		
+10	3		
+11	1		
Total	39		

A comparison of these two groups with reference to the anteroposterior co-ordinate showed them to be quite distinct.

	No. of cases	Mean AP co-ordinate	S.D.
Capsular twitches	39	+ 4.128	4.578
Thalamic twitches	77	- 4.818	2.771

Difference between the means 8.946 mm.

Standard error of the difference between the means 0.7982

Difference between the means was more than eleven times the standard error. The probability that a

difference as great as that observed should arise through chance was therefore extremely unlikely.

The capsular twitches and the midcapsular points defined by audiomonitoring were now compared for their AP co-ordinates.

	No. of cases	Mean AP co-ordinate in mm.	S.D.
Midcapsular points	24	+ 2.458	2.084
Capsular twitches	39	+ 4.128	4.578

Difference between the means 1.67 mm.

Standard error of the difference between the means 0.8476

$$\frac{\text{Difference between the means}}{\text{Its Standard Error}} = 1.97$$

Since this result was very close to the conventionally accepted level of significance, it was highly suggestive that capsular twitches arose from the anterior or pallidal side of the capsule in front of the midcapsular point.

To clarify this point further, all capsular motor response points, that is both twitches and pareses, were now compared with the midcapsular points defined by audiomonitoring for their anteroposterior co-ordinates, with reference to the midcommissural plane.

	No. of cases	Mean AP co-ordinate in mm.	S.D.
Midcapsular points	24	+ 2.458	2.084
All capsular motor response points (twitches and pareses)	76	+ 4.961	4.018

Difference between the means 2.503 mm.

Standard error of the difference between the means 0.6272

The difference between the means was nearly four times its standard error and the result was therefore very significant. Translated into anatomical terms, this meant that the corticobulbar and corticospinal tracts did not occupy the entire breadth of the posterior limb of the internal capsule but only a portion on its anterior aspect, that, is close to the pallidal border.

The 153 motor response points elicited during stereotaxic surgery and considered in the present study could therefore be classified as follows.

	Face only	Face and upper limb	Upper limb only	Upper and lower limbs	Entire contra lateral hemibody	Total
Thalamic movements	5	2	63	4	3	77
Capsular movements	7	3	23	6	0	39
Capsular pareses	16	0	21	0	0	37
Total	28	5	107	10	3	153

The vertical co-ordinates with reference to the CA-CP line were then compared for all muscle twitches (thalamic and capsular) and the midcapsular points defined by audiomonitoring.

	No. of cases	Mean vertical co-ordinate in mm.	S.D.
Midcapsular points	24	+ 1.375	1.498
All muscle twitches (thalamic and capsular)	116	+ 4.490	3.283

Difference between the means 3.115 mm.

Standard error of the difference between the means 0.4319

Difference between the means was more than 7 times the standard error and the result was therefore highly significant.

This again was in accordance with the previous results in relation to anteroposterior co-ordinates that muscle twitches during stereotaxy were distinctly separable into thalamic and capsular groups. These two groups were now compared for their vertical co-ordinates.

	No. of cases	Mean vertical co-ordinate in mm. above CA-CP line	S.D.
Thalamic twitches	77	+ 6.14	2.275
Capsular twitches	39	+ 1.23	2.411

Difference between the means + 4.91 mm.

Standard error of the difference between the means 0.4650

Difference between the means was more than ten times the standard error. The probability that a difference as great as that observed should arise through chance was extremely unlikely.

The vertical co-ordinates were then compared for the capsular twitches and the midcapsular points defined by audiomonitoring.

	No. of cases	Mean vertical co-ordinate above CA-CP line	S.D.
Midcapsular points	24	+ 1.375 mm.	1.498
Capsular twitches	39	+ 1.230 mm.	2.411

Difference between the means 0.14 mm.

Such a small difference was not considered important especially as it is universally accepted that the cortico-

bulbar and corticospinal tracts are passing from above downwards in the posterior limb of the internal capsule and that motor phenomena may be elicited from them at any vertical level.

(b) Position of the corticobulbar and corticospinal tracts in the posterior limb of the internal capsule

Lassek (1954) outlined chronologically 49 important investigations on the human pyramidal tract from 400 B.C. to 1950 A.D. Not one of these investigations was predominatly concerned with the position of the tract in the internal capsule.

Until very recently, knowledge of the position and arrangement of the corticobulbar and corticospinal tracts in the posterior limb of the internal capsule was based largely on the studies of Dejerine (1901), Pfeifer (1934), Levin (1936), Meyer, Beck and McLaray (1947) and Barnard and Woolsey (1956).

Dejerine's observations were based upon the study of pyramidal degenerations ensuing on vascular lesions of the cerebral hemispheres. Pfeifer based his conclusions on myelogenetic studies. Studies of others were conducted on animals using stimulation or degeneration techniques.

It was generally accepted that the corticobulbar tract was situated in the genu and the corticospinal tract in the anterior two-thirds of the posterior limb of the internal capsule. The face, arm and leg fibres were believed to be arranged in that order from the anteromedial

to the posterolateral part of this section of the capsule with some degree of overlap. It was also generally agreed that there was some intermingling with cortico-thalamic and pallidothalamic fibres. But the general pattern was universally accepted and is reproduced in all standard textbooks and monograms, e.g., Rasmussen (1942), Brash (1951), Haymaker (1956), Brain (1962), Patton and Amassian (1960), Bucy (1961), Davies and Davies (1962), Ranson and Clark (1959) and Truex (1959).

Prior to 1958, the only dissenting opinion against this universal belief was that of Minckler, Klemme and Minckler (1944). These authors observed from degeneration studies in man that the anterior part of the posterior limb of the internal capsule was occupied by the premotor bundle and not the pyramidal tract. Despite the fact that this was the only human case studied after several decades, their observation was passed over by the majority who had more faith in animal studies, as showing the structure of the capsule in the human.

The only surgeons to operate on the human internal capsule before 1957 were Browder and his colleagues (1953). Their observations were therefore the sole available record of living human physiology of the internal capsule. On a superficial examination, their results appeared to support the classical concept of the structure of the internal capsule. Closer scrutiny of their published material however revealed that this was not true. Operating by a deep transventricular approach, Browder sectioned the anterior limb of the internal capsule progressively

backwards till a slight weakness of the patient's hand appeared on the opposite side. He therefore assumed that he had just reached the posterior limb. There were no autopsy studies of these cases to show where exactly the section was. Another very valuable observation was again misinterpreted because of the universally held belief derived from animal experiments and pathological material in the dead.

Thurel was the first to challenge the concept of the structure of the posterior limb of the capsule. He observed in his paper on the psudobulbar syndrome that the faisceau genicule was a misnomer since it was not situated in the genu but further posteriorly in the internal capsule. Guiot and his colleagues (1958, 1959, 1960) repeatedly confirmed that they obtained motor responses on stimulation of the posterior limb of the internal capsule at 17 mm. lateral to the midline. They obtained responses from the leg, arm and face in that order from behind forwards.

Gillingham (1960) noted that in medial tracks (13 to 15 mm. from the midline) he seldom got any motor response from the internal capsule. In lateral tracks (16 to 18 mm. from the midline) motor responses were often obtained in the capsule. He favoured a concept which confined the pyramidal tract to a small area in the middle of the posterior limb adjacent to the pallidum. He agreed with Guiot and his colleagues (1959) as well as Hassler (1962) that the pyramidal tract in the human was not situated in the genu or the anterior part of the posterior limb. He disagreed with them in that he felt that the thalamic

border of the capsule was free of this tract which was confined to the pallidal border.

Talairach and Szikla (1963) used the anterior approach to the thalamus, crossing the pallidum and the posterior limb of the internal capsule. They used continuous stimulation under light general anaesthesia as the electrode was moved slowly towards the target. They observed motor responses from the capsule only if their track was 18 mm. or further lateral from the midline.

Bucy (1957, 1958) and Walker (1963) did not agree with this concept. According to them, absence of hemiplegia following capsular lesions did not prove that the pyramidal tract was intact. They claimed that their pedunculotomies were not followed by hemiplegia and a similar situation might well occur in the internal capsule.

This apparently valid argument was seen to be completely unacceptable on closer examination of their and others' published literature. Pedunculotomies were always followed by an initial complete flaccid paralysis as reported by Walker (1952, 1955), Bucy (1957) and Kitamura (1961). What Bucy referred to was the longterm assessment of recovery of motor function. On the other hand, the capsular lesions produced by stereotaxic surgeons were often not associated even with a trace of facial paresis provided such lesions were sufficiently medial. Cortical excisions by Horsley (1909), Sachs (1935), Foerster (1936), Putnam (1940), Bucy (1944) and Krynauw (1950) were all followed by paralysis in the immediate postoperative period although there was a

surprising degree of recovery on longterm follow-up.

There was therefore no reason to assume that a section of the pyramidal tract at a level in between the cortex and the peduncle, in the capsule, would not be followed by an initial complete flaccid paralysis. In fact such is the exact clinical state when a hemorrhage is produced in the lateral part of the posterior limb of the internal capsule during stereotaxic surgery. The postoperative recovery then closely parallels the recovery of hemiparesis after cortical excisions and pedunculotomies.

Lassek (1954) observed that the fibres of the pyramidal tract were not compactly arranged but were broken up into a number of bundles in the internal capsule. Recent editions of some of the textbooks quoted earlier have included this concept in their diagrams.

Guiot and his colleagues (1959) tried to explain the discrepancy between their findings and the classical concept of the structure of the posterior limb of the internal capsule. They suggested that at the level at which their electrode traversed the capsule, the anterior limb and the genu no longer existed having become incorporated with the posterior limb at a higher level. They conjectured that the frontopontine tract from the anterior limb had now displaced the corticobulbar and corticospinal tracts to a more lateral place in the capsule.

In the present study, 76 instances were available where motor responses (twitches and pareses) were elicited from the capsule during surgery.

It was already seen in the previous section of this chapter that the mean of these points was situated significantly in front of the midpoint of the capsule suggesting that the pyramidal tract was close to the pallidal border of the capsule.

The lateral co-ordinates of these motor response points were now compared with each other as well as the entire series. Frequency distribution of lateral distances of all capsular motor phenomena (twitches and pareses) compared for their peripheral sites of incidence (Distances in mm. of opaque marker in postoperative X-ray) was as follows:

Site	Very medial Sl 11.0 Marker 11.0 - 13.5	Medial Sl 13.5 Marker 14.0 - 16.0	Standard Sl 16.0 Marker 16.5 - 19.0	Lateral Sl 18.5 Marker 19,5 - 21.5	Very Lateral Sl 21.0 Marker 22.0 - 24.5	Total
Face only	0	2	13	5	3	23
Face and upper limb	0	0	2	1	0	3
Upper limb	0	2	26	12	4	44
Upper and lower limbs	0	0	1	4	1	6
Total number of motor responses	0	4	42	22	8	76
Total number of explorations	6	41	323	95	10	475
Percentage of motor responses	0	9.8%	13.0%	23.2%	80.0%	16.0%

It was seen that the percentage of motor responses obtained rose steadily till at a lateral distance of 21.0 mm. 80% of the explorations were associated with pyramidal tract signs.

The χ^2 test was not applicable to this table as the expected number in one cell was less than 5 and in another cell was less than one. The columns were therefore regrouped as shown below and the χ^2 test was applied.

	Group A Medial tracks	Group B Standard tracks	Group C Lateral tracks
Number of motor responses	4	42	30
Total number of explorations	47	323	105

χ^2 value 16.46 and for n=2, P was less than 0.01.

This was a very significant result.

(c) Somatotrophic Arrangement within the Pyramidal Tract

It is generally agreed by all previous authors who favoured the classical concept of the capsule as well as by the recent authors who challenged it, that there exists a somatotrophic arrangement within the pyramidal tract at the level of the internal capsule. The face fibers are believed to be situated anteromedially and the leg fibers posterolaterally, with the arm fibers in between. It is also agreed that there is a considerable degree of overlap between these groups.

The mutual relationship of the fiber arrangement would vary depending on the following factors. (1) The

lateromedial representation of face-arm-leg in the motor cortex rotates to mediolateral in the internal capsule. Depending on the level of horizontal section during this change, somatotrophy would vary. The anteroposterior somatotrophy continues unchanged at both levels and during the above rotation. (2) The angle made by the axis of the posterior limb of the internal capsule with the midcommissural plane as seen in a horizontal section is variable from person to person and in the same person at different levels. In the brain of the horizontal microseries of the atlas of Schaltenbrand and Bailey (1959) this angle is seen to change from about 60° at the level of Hd +6.5 to about 45° at the level of Hd +0.5. The greater the angle of inclination, the more prominent will be the antero-posterior somatotrophy. The reverse is true for the lateral somatotrophy.

This may be one of the reasons why Guiot, Rougerie, Sachs and Hertzog (1958) found only an antero-posterior somatotrophic arrangement and not a lateral. These authors found on stimulation along their electrode track (as they moved it from behind forwards) responses from the lower limb first, upper limb next and face most anteriorly.

A statistical analysis was done for the antero-posterior and lateral co-ordinates of these motor response points in the capsule. Upper limb response points were taken as those predominantly with the upper limb component and a similar rule was applied for the lower limb response points.

Comparison of lateral co-ordinates of all capsular motor response points

Site of motor response	No. of cases	Mean in mm. (Marker in X-ray uncorrected)	Standard deviation of entire group
Face	23	18.95	
Upper limb	47	18.97	2.029
Lower limb	6	21.17	

Comparison of face and upper limb:

Difference between the means 0.02 mm.

Difference was too small to merit further analysis.

Comparison of face and lower limb:

Difference between the means 2.22 mm.

Standard error of the difference between the means 0.9303

Result significant

Comparison of upper and lower limbs:

Difference between the means 2.20 mm.

Standard error of the difference between the means 0.8796

Result significant

Comparison of anteroposterior co-ordinates of all capsular motor response points.

Site of motor response	No. of cases	Mean in mm. in front of midcommissural plane in chart	Standard deviation of entire group
Face	23	+6.913	
Upper limb	47	+4.170	4.018
Lower limb	6	+3.330	

Comparison of face and upper limb:

Difference between the means 2.743 mm.

Standard error of the difference between the means 1.022

Result significant

Comparison of face and lower limb:

Difference between the means 3.583 mm.

Standard error of the difference between the means 1.842

$$\frac{\text{Difference between means}}{\text{Standard error}} = 1.945$$

Since this value was quite close to the conventionally accepted level of significance, the result was very suggestive that with greater numbers for the lower limb response points, the groups may well show a significant difference.

Comparison of upper and lower limbs:

Difference between the means 0.84 mm.

Standard error of the difference between the means 1.742

Result not significant

These results show that, at the level of exploration with the present technique, the lower limb fibers of the pyramidal tract are situated lateral to the face and upper limb fibers between which there was no significant difference in laterality. The face fibers of the pyramidal tract are situated in front of the upper and lower limb fibers between which there was no anteroposterior differentiation.

(d) Lateral Thalamic Movements

The role of the ventrolateral nucleus of the thalamus in the extrapyramidal motor system is being increasingly recognised. Hassler (1960) and Nathan (1957) summarised the state of knowledge on this subject. It was not surprising therefore that stimulation of this area provoked motor responses.

Hassler and his colleagues (1960, 1961, 1963) elicited

movements from stimulation of the oral ventral nucleus of the thalamus.

Hughes (1963a, 1963b) described motor responses on stimulation of the lateral thalamic area. He found two types of responses. One was confined to the face, upper limb or lower limb and the other involved the whole of one side of the body. The latter was much less frequently obtained than the former. In the first type, he found upper limb responses to be most common, the face responses to be less common and the lower limb responses, least frequent.

He also found a somatotrophic arrangement for these responses with the face area posteromedial and the lower limb area anterolateral. Upper limb responses were obtained in between.

In the present study, movements were obtained from the lateral thalamic area in 77 instances. Their differentiation from capsular movements on anatomical, clinical and statistical grounds was discussed in an earlier section of this chapter.

74 of these responses corresponded to Type I described by Hughes in that they involved predominantly the face, upper limb or the lower limb. 3 responses belonged to Type II of Hughes in that they involved the entire contralateral hemibody.

Most of these responses were elicited during the period when stimulation was used as a routine in the department, that is, prior to 1961. A few were obtained during creation of reversible and irreversible

lesions. Since stimulation was done by means of single shocks, the movement was only a single contraction often described as a jerk or twitch and involving predominatly the flexor group of muscles. It was most frequent in the upper limb (65 cases), much less so in the face (5 cases) and least in the lower limb (4 cases). 3 of the upper limb cases showed slight involvement of the face and all the lower limb cases showed slight involvement of the upper limb as well.

In view of the fact that in the earlier days of stereotaxy in this department, these movements were believed to be of capsular origin the electrode was often moved from the area where a movement was obtained and no effort was made to study the movement in detail as to its pattern or methods of elicitation. In some instances the distance of the electrode was too far from the capsule and a cautious lesion was made at the site where a movement was obtained. Whenever such a lesion was made, paresis was never produced.

While the nature, distribution, types and general pattern of these thalamic movements closely corresponded to that described by Hughes, there was no significant somatotrophic arrangement to be made out on analysis of their co-ordinates. This might have been due to (a) the different parameters of stimulation used (b) the fact that these movements were not particularly sought for and documented in detail (c) the very small number of cases involving the face and the lower limb and (d) the lack of application of a correction factor for lateral ventricular

width. The general pattern however was similar to that sug ested by Hugh s although the r sults were not statistically significant.

Comparison of the anteroposterior co-ordinates of the points where lateral thalamic movements were elicited.

Site of response	No. of cases	Mean AP co-ordinate in mm. behind mid-commissural plane	S.D. for entire group
Face	5	- 4.00	
Upper limb	68	- 4.88	2.771
Lower limb	4	- 4.75	

Comparison between face and upper limb:

Difference between the means 0.88 mm.
Standard error of the difference between the means 1.284

Result not significant

Comparison between face and lower limb:

Numbers too small to merit further analysis.

Comparison between upper and lower limbs:

Difference between the means 0.13 mm.
Difference was too small to merit further analysis.

Comparison of the lateral co-ordinates of the points where lateral thalamic movements were elicited.

Site of response	No. of cases	Mean lateral co-ordinate (Marker in X-ray in mm. uncorrected)	S.D. for entire group
Face	5	18.05	
Upper limb	65	18.16	1.788
Lower limb	4	18.50	

Comparison of face and upper limb:

Difference between the means 0.11 mm.

Difference too small to merit further analysis

Comparison of face and lower limb:

Numbers too small to merit further analysis

Comparison of upper and lower limbs:

Difference between the means 0.34 mm.

Standard error of the difference between the means 0.9212

Result not significant.

While the general nature of the thalamic movements studied in this project conformed to that described by Hughes (1963a, 1963b) the circumstances of elicitation and observation were not standardised sufficiently for a scientific comparison to be made.

(f) Dysarthria during Stereotaxic Surgery

In most patients with Parkinsonism or other dyskinesias, there is usually some preoperative speech disturbance. Rigidity of lips, tongue, jaw, pharynx, vocal cords, chest and abdomen contribute to difficulty in speech. Generalised physical and mental slowing, memory defects, difficulty of initiation and akinesis are other factors affecting speech. The resultant effect is a combination in varying degrees of the different manifestations in one patient. Inability to initiate speech, reduced voice volume, slurring of speech and festination are some of the major components of the speech disorder. Excessive salivation and dryness of mouth due to drugs may further add to the patient's

difficulties in speech.

The surgical procedure may worsen speech in one or more of four main ways postoperatively. These are (1) dysphasia, (2) dysarthria, (3) dysphonia and (4) worsening of preoperative akinesis, inability to initiate and festination of speech. Postoperative speech difficulties, especially after bilateral lesions are well recognised and are discussed in detail in a subsequent chapter.

During operation, speech phenomena have not been extensively studied, probably because the limited time available for clinical testing has been largely taken up in the estimation of motor and sensory functions. What little has been reported in the literature, has been on the subject of changes in speech as a result of stimulation.

Guiot, Hertzog, Rondot and Molina (1961) observed arrest or acceleration of speech evoked by thalamic stimulation. Hassler and Riechert (1961) reported similar phenomena. It is very debatable how much these stimulation studies elucidate the exceedingly complex mechanism of speech. Hughes (1961) warned against postulating simple direct relationships between the effects of stimulation and coagulation at a given site. Probably this applies to speech even more than to other phenomena. Penfield (1963) felt that stimulation of the pulvinar during stereotaxy might result in valuable data regarding the role of the thalamus in speech mechanisms.

There is usually no opportunity during surgery for a detailed testing for dysphasia. In a tired and often sedated patient, voice volume is difficult to measure and compare, especially under operating theatre conditions.

Dysarthria, appearing suddenly during operation for the first time or becoming markedly worse as compared to the preoperative state, is easily recognised and correlated with the position of the electrode. This therefore is the only aspect of speech with which the present study is concerned.

In fortyfive instances in this series, slurring of speech was noted during stereotaxic surgery. The majority of these responses were due to the creation of reversible heating lesions.

There was no obvious relation to the occurrence of dysarthria and the laterality of the electrode track as shown by the table below.

Track used	medial (S1 13.5)	Standard (S1 16)	Lateral (S1 18.5)	Total
Number of cases of dysarthria during surgery	6	29	10	45
Total number of explorations	47	323	105	475

Study of the scattergrams composed of the co-ordinates of the central points of electrode positions when dysarthria was produced was not particularly helpful for the medial and lateral tracks. The number of points were few and they were widely scattered.

The study of these points in the 'standard tracks' group suggested that they tended to concentrate in two separate areas. One was in the distal capsule and the pallidocapsular junction. (In studying this scattergram, one has to remember that the mean breadth of the capsule in this

series as defined by depth electrical recording and audiomonitoring was greater than that of the atlas brain. Points in the pallidum near its posterior edge on the atlas, therefore, would be actually situated in the pallidal border of the internal capsule in our patients.) The other group was clustered around the thalamic nuclei near the capsular border. (See scattergrams at end of chapter)

If only one site or structure is responsible for a phenomenon, it is logical to expect a scatter around one area as was noted in the cases of the evoked potentials from the thalamus, thalamic movements, capsular motor phenomena and visual phenomena. If two sites closely adjacent to each other are responsible for the same phenomenon, one might expect to see a scattergram with a long wide scatter, as was seen in the case of sensory response points in the thalamic relay nucleus and the thalamoparietal projection. In the case of dysarthria, however, the points tended to group around two different structures separated by a clear interval of the thalamic border of the internal capsule.

This suggests that the thalamic nuclei are intimately concerned with speech and their stimulation can produce alterations of speech (Guiot et al, 1961). Permanent coagulation here, especially bilaterally can cause severe slurring of speech (Krayenbuhl et al, 1961). On the other hand, dysarthria at more anterior levels may be produced by interference with the motor fibres to the tongue in the pyramidal tract in the internal capsule (Gillingham 1961). Clinically these two types of dysarthria seem to be

indistinguishable on the operating table. Therefore these points were not subjected to further statistical analysis of their co-ordinates. The analogy of thalamic and capsular movements springs to the mind but in their case, they could be well differentiated from each other. Only a careful study of a large number of cases of dysarthria produced during stereotaxic surgery would show whether this hypothesis, suggested by the scattergram, is true.

(f) Summary

The controversies regarding the interpretation of the presence and absence of motor phenomena during stereotaxic surgery were outlined. The need for an unbiased approach to the study of the structure of the internal capsule was emphasised.

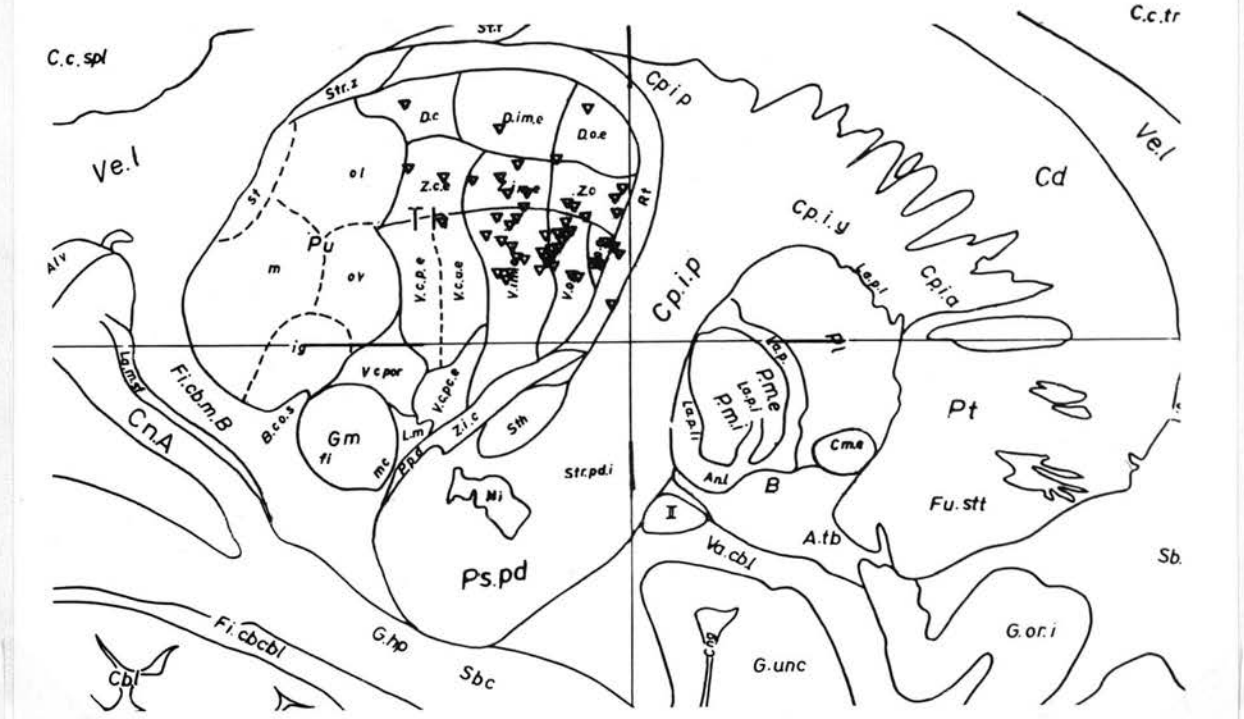
The differentiation between thalamic and capsular movements was stressed.

It was shown that the pyramidal tract is confined to a relatively small portion of the posterior limb of the internal capsule in its middle part and close to the pallidal border.

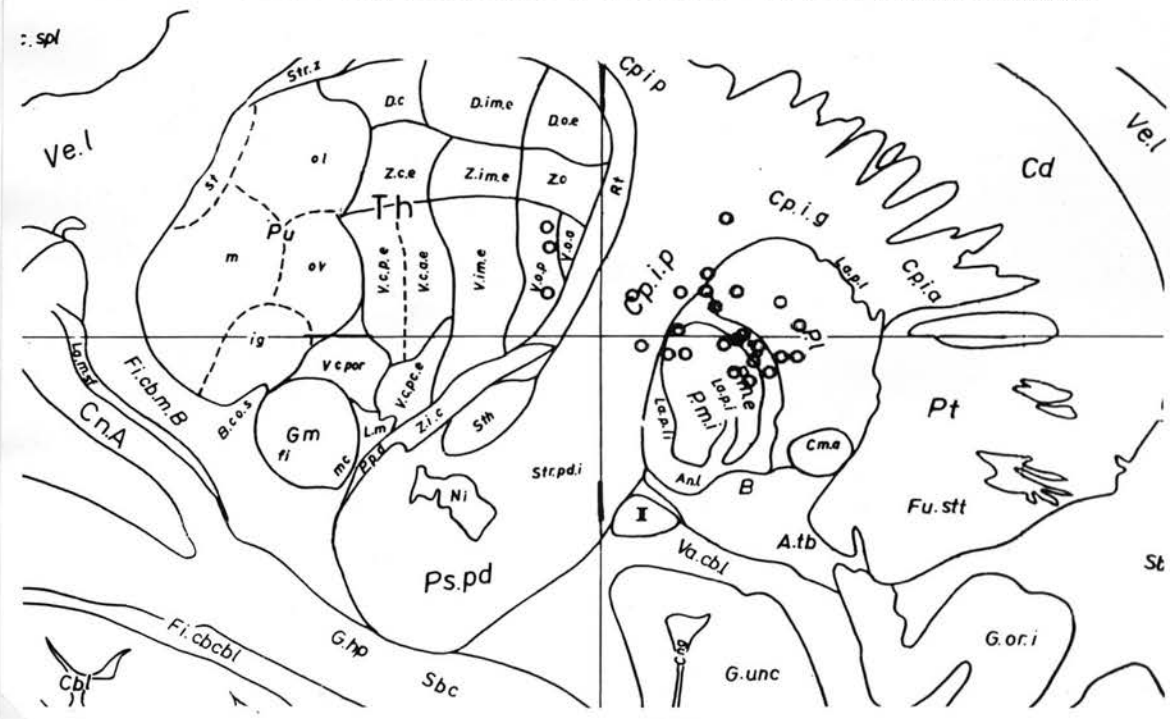
It was shown that the face, upper limb and lower limb fibres within the pyramidal tract are arranged in that order from the anteromedial to the posterolateral direction. There is, however, considerable overlap between the different groups of fibres.

It was suggested that dysarthria during stereotaxic surgery might arise by two different mechanisms. These are (1) the involvement of the thalamic nuclei and (2) the involvement of the motor fibres to the tongue in the pyramidal tract.

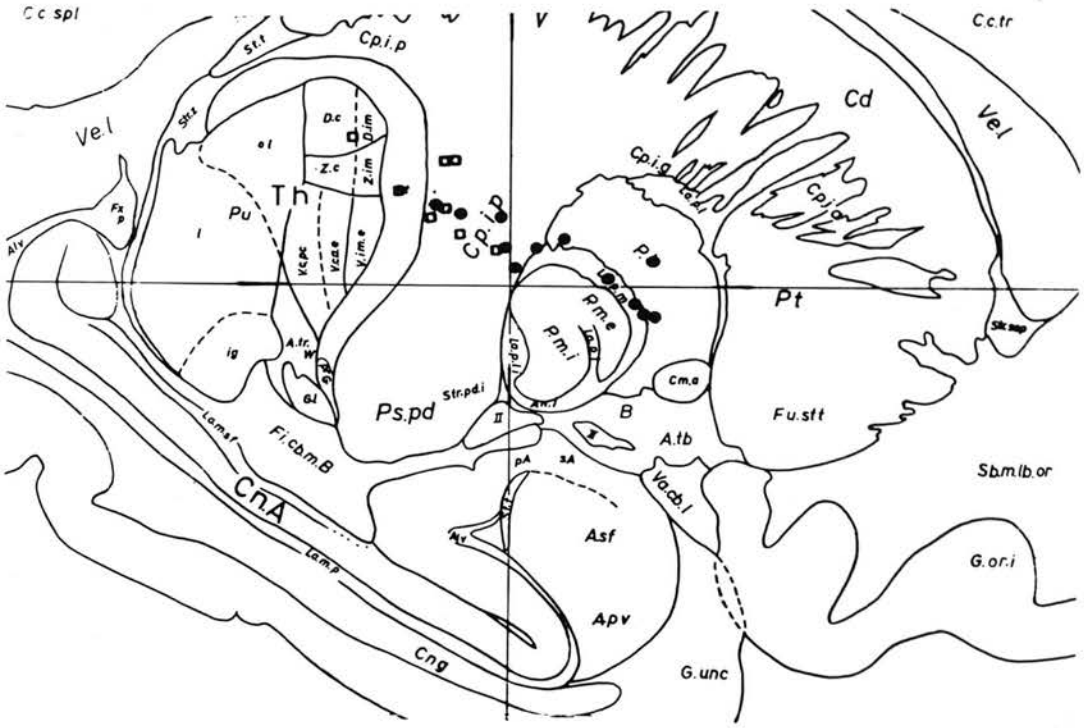
Sites where thalamic movements were elicited from the upper limb.
Scatter diagram superimposed on Plate 48 - Sl I6 of stereotaxic atlas.



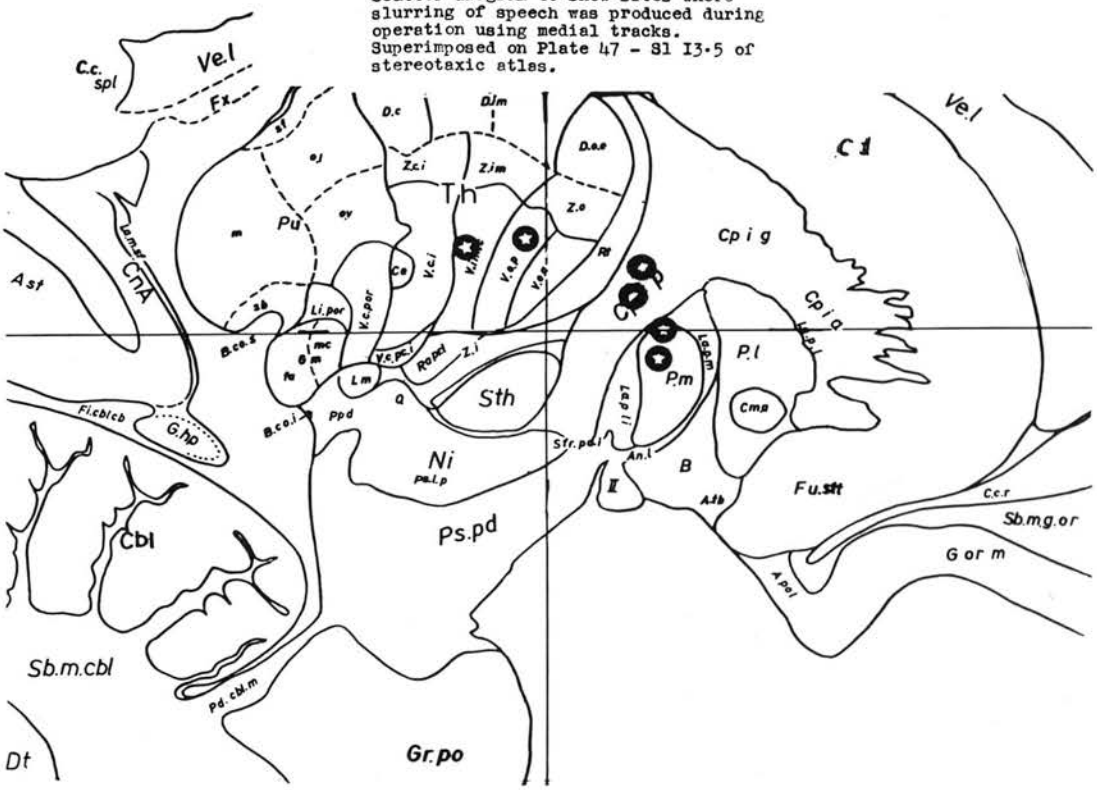
Sites where capsular responses were elicited from the upper limb.
Scatter diagram superimposed on Plate 48 - Sl I6 of stereotaxic atlas.

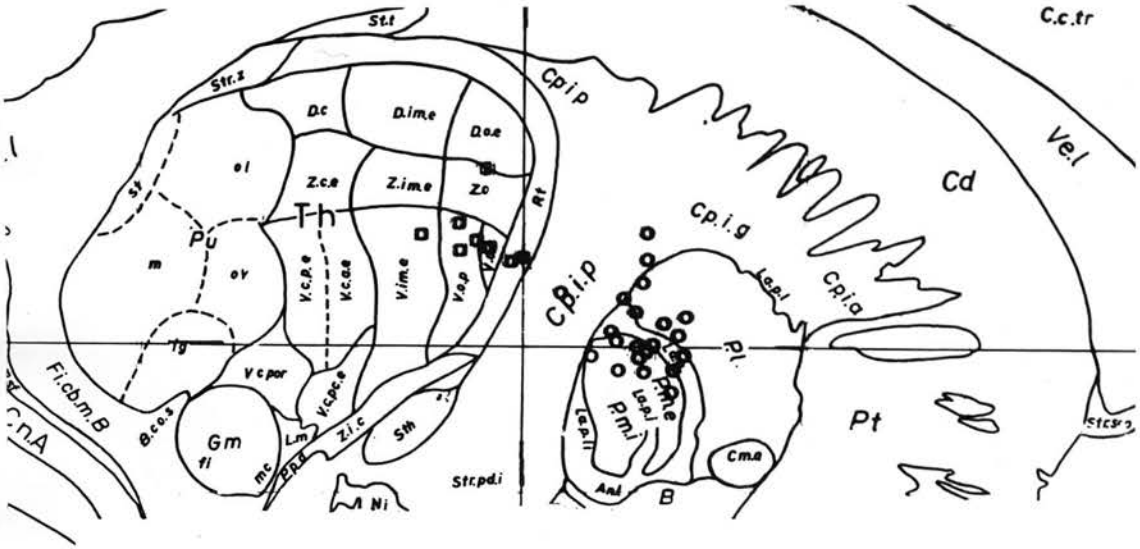


Sites where thalamic (squares) and capsular (circles) motor responses were elicited from lateral tracks in the upper limb. Scatter diagram superimposed on Plate 48 - Sl I8·5 of stereotaxic atlas.



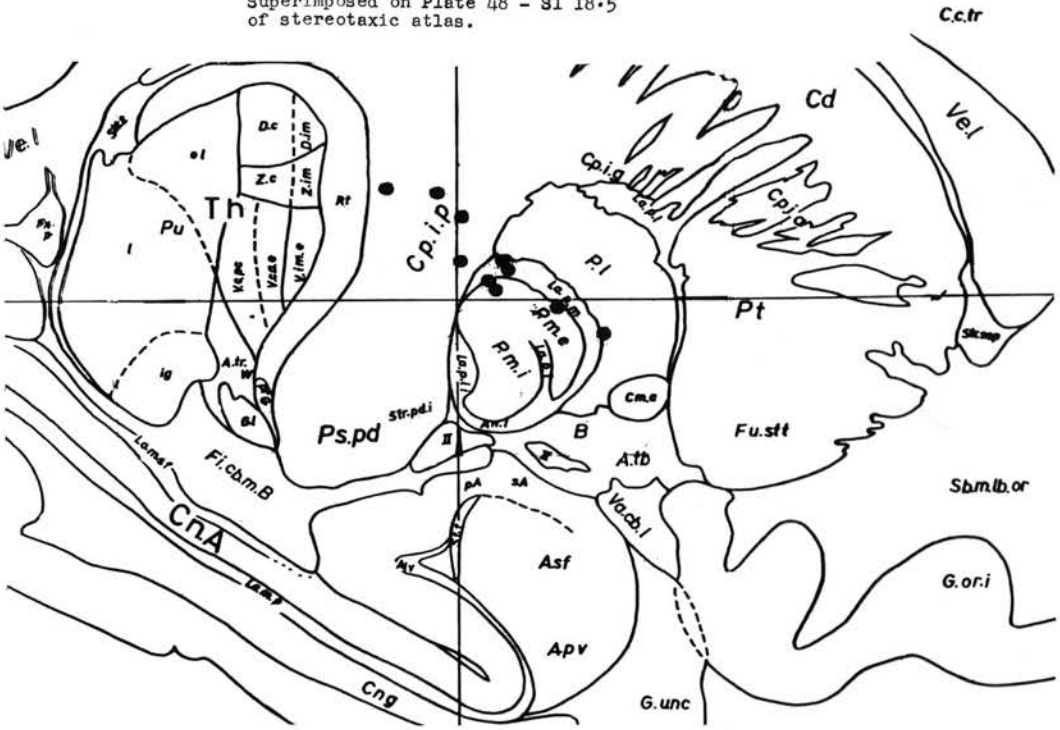
Scatter diagram to show sites where slurring of speech was produced during operation using medial tracks. Superimposed on Plate 47 - Sl I3·5 of stereotaxic atlas.



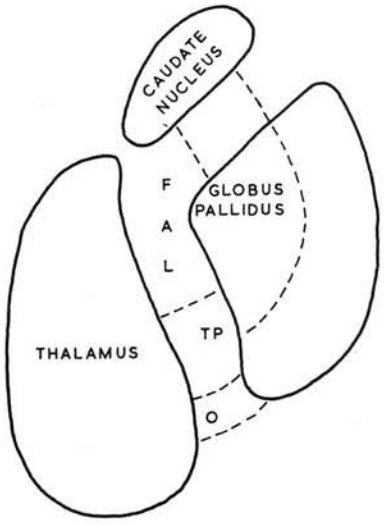


Scatter diagram to show sites where slurring of speech was produced during operation along standard tracks. Superimposed on Plate 48 - Sl I6 of stereotaxic atlas. It is suggested that the responses shown by a circle tend to concentrate around an area in the internal capsule where motor fibres to the tongue may be implicated. It is also suggested that the responses shown by a square tend to group around the oral ventral nucleus of the thalamus and slurring of speech in this region may be produced by a thalamic mechanism.

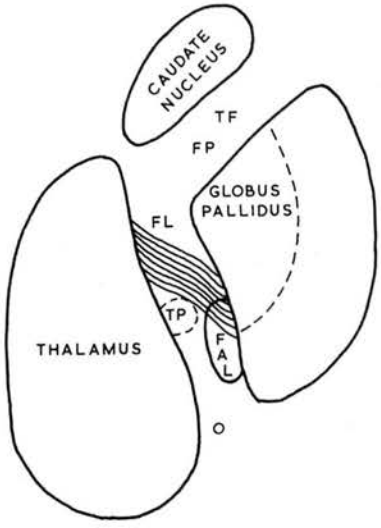
Scatter diagram to show sites where slurring of speech was produced during operation along lateral tracks. Superimposed on Plate 48 - Sl I8·5 of stereotaxic atlas.



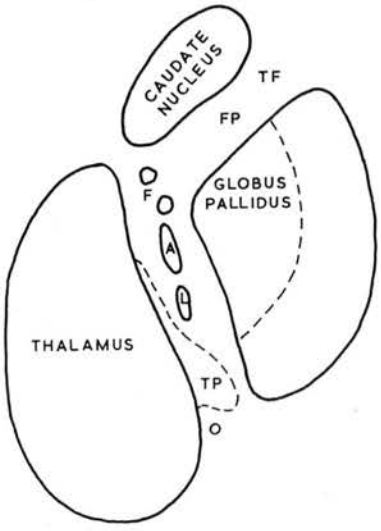
(I) CLASSICAL CONCEPT OF THE INTERNAL CAPSULE.



(III) OUR CONCEPT OF THE INTERNAL CAPSULE.



(II) MODERN CONCEPT OF THE INTERNAL CAPSULE.



TF –THALAMOFRONTAL FIBRES

F –FACE
A –ARM
L –LEG
} FIBRES OF CORTICO–BULBAR AND CORTICOSPINAL TRACTS.

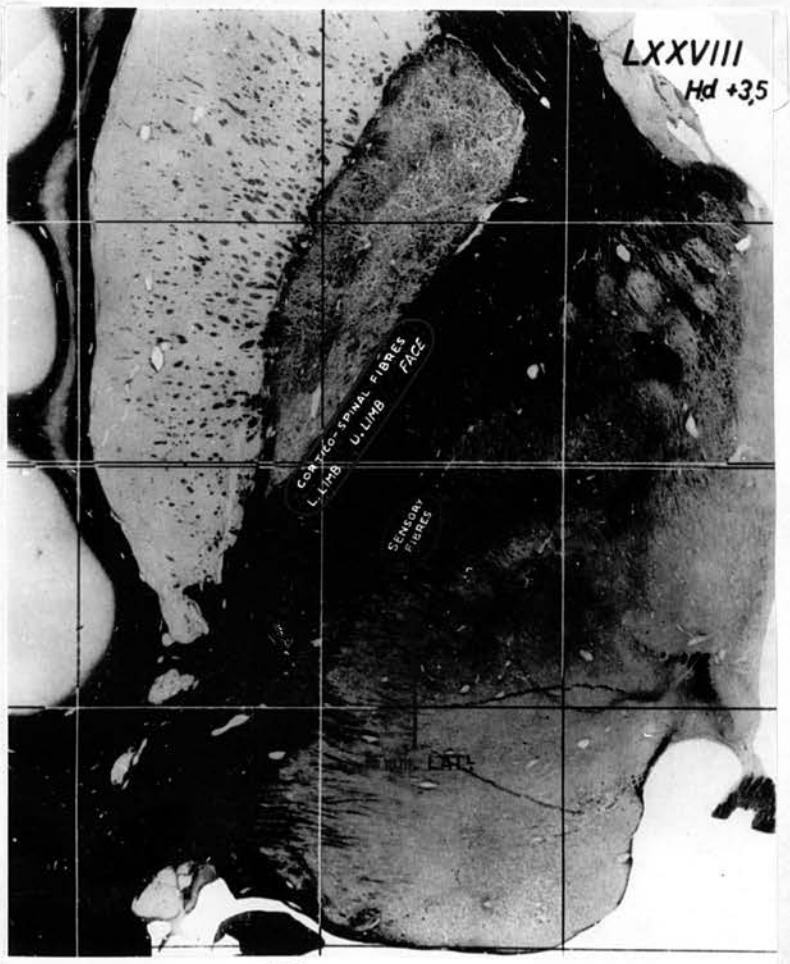
TP –THALAMOPARIETAL PROJECTION

O –OPTIC RADIATION

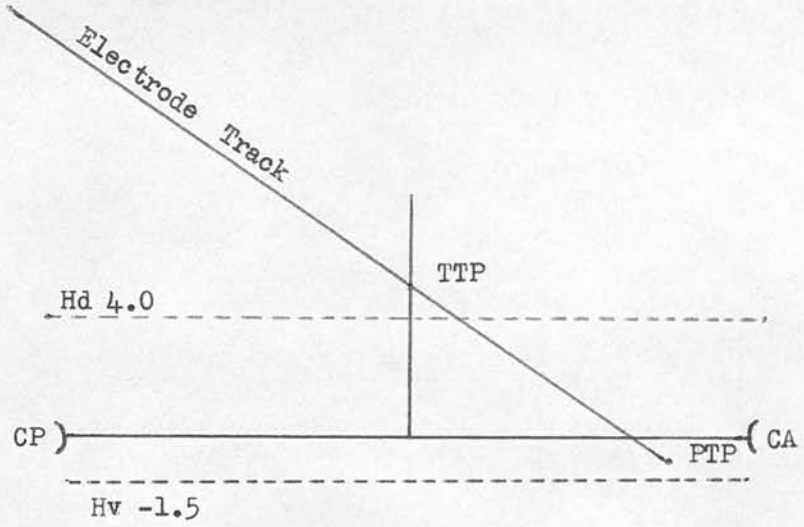
FL –FASCICULUS LENTICULARIS

FP –FRONTOPONTINE FIBRES

DIAGRAMS OF HORIZONTAL SECTIONS THROUGH THE INTERNAL CAPSULE TO SHOW CHANGING CONCEPTS ABOUT ITS STRUCTURE.



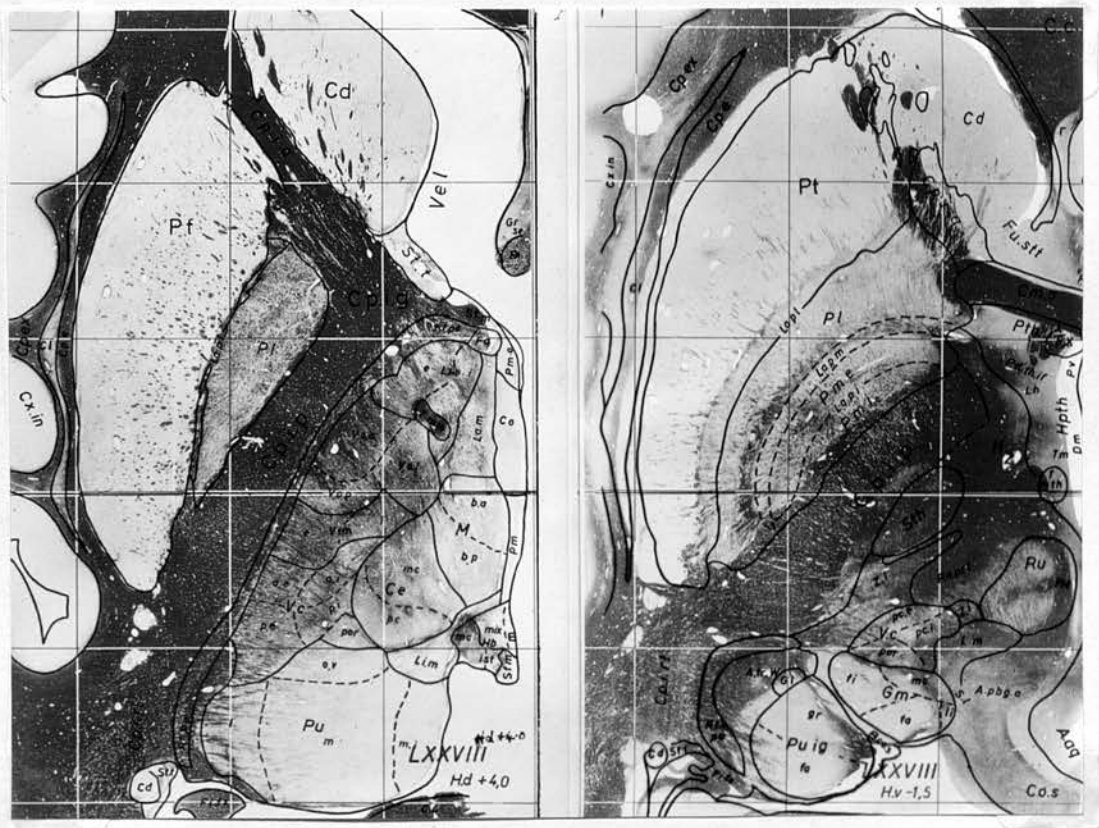
Horizontal section (9X) 3.5 mm. above the intercommissural plane showing the concept of topographical arrangement of cortico-spinal and sensory fibres in the internal capsule



PTP - Pallidal Target Point TTP - Thalamic Target Point

CA - Anterior Commissure CP - Posterior Commissure

Comparison of the two diagrams shows that Guiot's argument is not valid since the capsule is met at Hd 4.0 and not Hv -1.5 in the operations described in the present work.



Comparison of horizontal sections at two levels from the stereotaxic atlas to show disappearance of anterior limb of internal capsule at lower level.
(Guiot's explanation for position of pyramidal tract in posterior limb.)

REFERENCES

1. Barnard, J.W. and Woolsey, C.N. (1956). J. Comp. Neurol., 105, 25.

2. Brain, R. (1962). Diseases of the Nervous System, Oxford University Press, London. Sixth edition. Fig. 3.

3. Brash, J.C. (1951). Cunningham's Textbook of Anatomy. Oxford University Press. London. Ninth edition. 974.

4. Browder, J., Kaplan, H.A. and Rabiner, A.M. (1953). Ann. Surg., 138, 502.

5. Bucy, P.C. (1944). The Precentral Motor Cortex. The University of Illinois Press. Urbana. Illinois. 353.

6. Bucy, P.C. (1957). Brain, 80, 376.

7. Bucy, P.C. (1958). In Fields, W.S. Ed. Pathogenesis and Treatment of Parkinsonism. C.C. Thomas. Springfield, Illinois. 271.

8. Bucy, P.C. (1961). In Taylor, N.B. Ed. The Physiological Basis of Medical Practice. Bailliere, Tindall and Cox Limited. London. Seventh edition. 1186.

9. Davies, D.V. and Davies, F. (1962). Gray's Anatomy. Longmans. London. Thirtythird edition. 1083.

10. Dejerine, J. (1901). Quoted by Nathan, P.W. and Smith, M.C. Brain, 78, 248, 1955.

11. Foerster, (1936). Quoted by Bucy, P.C. The Precentral Motor Cortex. The University of Illinois Press. Urbana. Illinois. 1944, 353.

12. Gillingham, F.J. (1960). Personal Communication.

13. Gillingham, F.J. (1961). Personal Communication.

14. Gillingham, F.J. (1962). Confin. Neurol., 22, 385.

15. Guiot, G., Rougerie, J., Sachs, M. and Hertzog, E. (1958). Rev. Neurol., 98, 222.

16. Guiot, G., Sachs, M., Hertzog, E., Brion, S., Rougerie, J., Dalloz, J.E. and Napoleone, F. (1959). Neurochirurgie, 5, I, 17.

17. Guiot, G., Brion, S. and Hertzog, E. (1960). J. Neurol. Neurosurg. Psychiat., 23, 348.

18. Guiot, G., Hertzog, E., Rondot, P. and Molina, P. (1961). Brain, 84, 363.

19. Hassler, R. (1960). In Jung, R. and Hassler, R. In Field, J., Magoun, H.W. and Hall, V.E. Ed. Handbook of Physiology, Section I. Neurophysiology. American Physiological Society. 863.

20. Hassler, R., Riechert, T., Mundinger, F., Umbach, W. and Ganglberger, J.A. (1960). Brain, 83, 337.

21. Hassler, R. and Riechert, T. (1961). Nervanarzt, 32, 37.

22. Hassler, R. (1962). In French, J.D. Ed. Frontiers in Brain Research. Columbia University Press. New York and London. 242.

23. Hassler, R. (1963). Personal Communication.

24. Haymaker, W. (1956). Bing's Local Diagnosis in Neurological Diseases. H. Kimpton, London. 19.

25. Horsley, V.E. (1909). Brit. med. J., 2, 121.

26. Hughes, B. (1961). IX Congreso Latinoamericano De Neurocirugia. Mexico. 657.

27. Hughes, B. (1963a). Excerpta Medica International Congress Series No. 60. Second European Congress of Neurological Surgery. 124.

28. Hughes, B. (1963b). Personal Communication.

29. Kitamura, K. (1961). Neurologia medico-chirurgica. 3, 152.

30. Krayenbuhl, H., Wyss, O.A.M. and Yasargil, M.G. (1961). J. Neurosurg., 4, 429.

31. Krynauw, R.A. (1950). J. Neurol. Neurosurg. Psychiat., 13, 243.

32. Lassek, A.M. (1954). The Pyramidal Tract. Its Status in Medicine. C.C. Thomas. Springfield, Illinois. 108.

33. Levin, P.M. (1936). J. Comp. Neurol., 63, 369.

34. Meyer, A., Beck, E. and McLardy, T. (1947). Brain, 70, 18.

35. Nathan, P.W. (1957). In Williams, D. Ed. Modern Trends in Neurology. Second Series. Butterworth and Co. London.

36. Parkinson, J. (1817). An Essay on the Shaking Palsy. Sherwood, Neely and Jones, London. Reprinted in Critchley, M. Ed. James Parkinson. Macmillan and Co. London. 1955. 168.

37. Patton, H.D. and Amassian, V.E. (1960). In Field, J., Magoun, H.W. and Hall, V.E. Ed. Handbook of Physiology. Section I. Neurophysiology. American Physiological Society. 2,837.

38. Penfield, W. (1963). Quoted by Gillingham, F.J. Personal Communication.

39. Pfeifer, R.A. (1934). Quoted by Nathan, P.W. and Smith, M.C. Brain, 78, 248, 1955.

40. Putnam, T.J. (1940). Res. Publ. Assoc. Ner. & Ment. Dis., 21, 666.

41. Ranson, S.W. and Clark, S.L. (1959). The Anatomy of the Nervous System. W.B. Saunders and Company. Philadelphia and London. Tenth edition. 320.

42. Rasmussen, A.T. (1942). The Principal Nervous Pathways. The Macmillan Co. New York. 52.

43. Sachs, E. (1935). Brain, 58, 492.

44. Schaltenbrand, G. and Bailey, P. (1959). Introduction to Stereotaxis with an atlas of the human brain. Thieme, Stuttgart, 2.

45. Sherrington, C. Quoted by Walshe, F.M.R. in Garland, H. Ed. Scientific Aspects of Neurology. E. & S. Livingstone Ltd., Edinburgh and London. 1961. 1.

46. Smith, M.C. (1962). Brit. med. J., I, 900.

47. Spiegel, E.A. and Wycis, H.T. (1952). Stereoencephalotomy (Thalamotomy and Related Procedures). Part I. Methods and Stereotaxic Atlas of the Human Brain.

48. Talairach, J. and Szikla, G. (1963). Personal Communication.

49. Thurel, R. Quoted by Guiot, G., Rougerie, J., Sachs, M. and Hertzog, E. (1958). Rev. Neurol., 98, 222.

50. Truex, R.C. (1959). Strong and Elwyn's Human Neuroanatomy. The Williams and Wilkins Company. Baltimore. Fourth edition. 399.

51. Walker, A.E. (1952). J. Nerv. & Ment. Dis., 116, 766.

52. Walker, A.E. (1955). Surg. Gyn. & Obst., 100, 716.

53. Walker, A.E. (1963). Quoted by Gillingham, F.J. Personal Communication.

54. Ward, A. Quoted by Walshe, F.M.R. in Garland, H. Ed. Scientific Aspects of Neurology. E. & S. Livingstone Ltd., Edinburgh and London, 1961. 8.

PART III

EFFECT OF STEREOTAXIC SURGERY

ON "EXTRAPYRAMIDAL" MANIFESTATIONS

CHAPTER 6

Effect of Stereotaxic Surgery on Tremor

"The first symptoms perceived are, a slight sense of weakness, with a proneness to trembling in some particular part; sometimes in the head but most commonly in one of the hands and arms. These symptoms gradually increase in the part first affected; and at an uncertain period, but seldom in less than twelve months or more, the morbid influence is felt in some other part."

.... James Parkinson.

"A perfect operation ought to look like a superb physiological experiment (as indeed it is), the object being to inflict the least possible damage on the patient, whose receovery in the best imaginable condition is the essential object of the proceeding."

.... Geoffrey Jefferson.

(a) Ideal Site of Lesion for Parkinsonian Tremor

The first targets to be destroyed stereotaxically in the treatment of Parkinsonian tremor were the inner segment of the globus pallidus and the ansa lenticularis (Wycis and Spiegel, 1952). It soon became obvious however that rigidity was better controlled by pallidal lesions than tremor (Narabayashi et al 1956). This was confirmed by most stereotaxic surgeons.

Hassler (1955) advocated the creation of a thalamic lesion in Parkinsonism and based his argument on theoretical grounds. His logic was soon confirmed by the creation of such a lesion in the human by Hassler and Riechert (1958) and Cooper (1958).

By 1962, the majority of stereotaxic surgeons were creating lesions in the thalamus, although their target sites differed to some extent. Some preferred an additional pallidal lesion in selected cases. A few aimed at other

sites like the internal capsule or the subthalamic region. A comparison of the sites of lesion preferred by various workers proved to be very interesting.

Bertrand and Martinez (1962) preferred a lesion for tremor to be centred at a point 14 mm. behind, 6 mm. below and 13 mm. lateral to the foramen of Monro.

Cooper (1961) aimed at a point in the thalamus about 3 mm. medial to a perpendicular line drawn from the supero-lateral angle of the body of the lateral ventricle to its floor. In the lateral X-ray, this point was situated about 3 mm. behind the central point of the Monro-pineal line.

Gillingham (1960) considered tremor to be controlled best by a thalamocapsular lesion. His thalamic target was situated 5 mm. (in X-ray) above the intercommissural line on the midcommissural plane, 15 mm. lateral to the midline. A lesion was made with the tip of the electrode at the thalamic target and another 2 mm. further forwards. The central point of the composite lesion was the thalamic target defined above.

Gillingham (1962) also showed that tremor was effectively controlled by his transcapsular lesions.

Guiot (1963) preferred to make a lesion in the oral ventral nucleus of the thalamus, 14 mm. lateral to the midline. He avoided the capsule by (a) demonstrating its position by depth electrical recording with audiomonitoring and (b) insulating one side of the coagulating portion of the electrode. The insulated portion was turned towards the capsule during the creation of a lesion in the thalamus.

Hankinson (1963) used a target 10.5 mm. in front of the

centre of the posterior commissure and 4 mm. above the intercommissural line. The central point of his lesion was approximately 7.5 mm. in front of the anterior border of the posterior commissure and 4.5 mm. above the intercommissural line. The lateral distance was 17 mm. from the midline for cases without dilatation of the lateral ventricle.

Hughes (1963) created lesions in the anterior and posterior parts of the oral ventral nucleus of the thalamus. He calculated the co-ordinates of the targets by means of a specially prepared sets of outlines of these nuclei from brains with different intercommissural lengths and lateral ventricle widths.

Jinnai and his colleagues (1961) preferred thalamocapsulotomy for tremor. Their target was located approximately 1 mm. below the foramen of Monro and 19.5 mm. posterior to it along the line parallel to Reid's line. It was approximately 19 mm. lateral to the midline of the third ventricle.

Krayenbuhl (1962) used a thalamic target 12.5 mm. behind the anterior commissure on the intercommissural line, 11.5 mm. lateral to the wall of the third ventricle.

Leksell and Strang (1963) used a thalamic target, 11 mm. from the midline, 3.5 mm. behind the midcommissural plane and 2.5 mm. above the intercommissural line.

McCaul (1963) used as his thalamic target a point on the midcommissural plane, 3 mm. above the intercommissural line and 12 mm. lateral to the midline.

Narabayashi (1963) preferred a target 3 mm. behind and 2 mm. above the midcommissural point. His lateral distance was approximately 15 mm.

Riechert and Hassler (1963) coagulated the ventral oral posterior nucleus of the thalamus for tremor. According to them, this nucleus was located around a point on the FM-CP line, 12 mm. behind the foramen of Monro and 10 mm. lateral to the middle of the lateral wall of the third ventricle.

Spiegel and Wycis (1962) chose a thalamic target 2 mm. above the intercommissural line and 3 mm. behind the mid-commissural plane, for brains with an intercommissural length, less than 25 mm. This point was situated 12 mm. lateral to the midline.

Spiegel, Wycis and their colleagues (1963) carried out campotomy. The target site was 6 mm. lateral to the midline, 10 mm. anterior to the posterior commissure (for short intercommissural lengths) and was situated on the inter-commissural line.

Talairach and Szikla (1963) used a slightly different target than most workers. They defined their CA-CP line by joining the superior border of the anterior commissure with the inferior border of the posterior commissure between two tangents to the commissures perpendicular to the CA-CP line. They then divided this CA-CP line into three parts. Their thalamic target was situated 10 mm. above the junction of the anterior twothirds and the posterior third of the CA-CP line. It was placed17 mm. lateral to the midline.

Walsh (1962) defined as his thalamic target, a point on the line joining the centres of the commissures and dividing that line in the ratio of 17:10. It was placed 12 mm. lateral to the midline.

A comparison of all these targets made it obvious that there was no single 'ideal' target for abolishing

Parkinsonian tremor. Whether different surgeons are interfering with the same 'neuronal circuit' or tremor conducting pathway at different levels and if so, what the nature of that pathway is, are points for theoretical discussion.

In view of the fact that lesions at all these sites apparently produced total or subtotal abolition of tremor, the 'ideal' site would be decided by the following considerations:

(a) minimal variability of target site with relation to individual variation of anatomical structures

(b) possibility of extension of lesion to an immediately adjacent tremor abolition site if it was not abolished at the aimed target

(c) minimal risk of complications

(d) safety of lesions at the same target in both hemispheres and

(e) possibility of influencing other symptoms like rigidity and oculogyric crises at the target site for tremor.

(b) Scattergram Studies of Sites of Tremor Abolition

In 163 instances in the present series, tremor was dramatically abolished or very considerably reduced (at least 75% by clinical observer's grading) at a single electrode position. Such a result was obtained either by mechanical injury by electrode insertion or by minimal

heating lesions reversible for all practical purposes. Some were due to irreversible coagulation lesions. Stimulation studies were not included in these figures since the results of stimulation were considered unreliable as guides to the placement of therapeutic lesions.

Contrary to general opinion, the lateral distance of the track seemed to have no relation to the abolition of tremor during operation (within the limits of the lateral distance used in the present technique). The following figures illustrate this.

	Number of cases of abolition of tremor at single electrode position during operation	Total no. of explorations
Very Medial Tracks Sl 11.0	2	6
Medial Tracks Sl 13.5	14	41
Standard Tracks Sl 16.0	117	323
Lateral Tracks Sl 18.5	29	95
Very Lateral Tracks Sl 21.0	1	10
TOTAL	163	475

The central points of the electrode positions were plotted for the medial, standard and lateral tracks. Scatter diagrams were drawn and superimposed on the corresponding plates of the stereotaxic atlas of Schaltenbrand and Bailey (1959). (See scattergrams at the end of this chapter)

Consideration of the medial tracks showed that the points were mainly concentrated around the ventral intermediate and the ventral oral nuclei of the thalamus (posterior part more than the anterior part). This agreed well with similar observations of Caracalos, Levita and Cooper (1962) and the opinion of Hassler that the posterior part of the oral ventral nucleus is the ideal site of lesion for tremor.

Examination of the scattergram of the standard tracks showed the concentration of tremor abolition points again around the ventral intermediate and ventral oral nuclei of the thalamus (both anterior and posterior parts), close to the capsular border. The opinion that this is an ideal site for lesion for tremor has been expressed by Gillingham (1960) and by Jinnai and his colleagues (1961).

Inspection of the scattergram for the lateral tracks revealed that an equally satisfactory effect on tremor was produced by lesions in the reticular nucleus of the thalamus and the posterior limb of the internal capsule. Gillingham (1960) was of the opinion that capsular lesions were effective because of interruption of the ansa and fasciculus lenticularis conveying pallidofugal impulses to the thalamus. Aguinis (1963) felt that such a lesion interrupted the cortical projection system from the reticular nucleus of the thalamus and this was the reason for its ability to abolish tremor.

It was felt by the present author that there was no basis for subjecting the co-ordinates of these tremor abolition points to any statistical analysis. It was likely

that they belonged to different structures whose destruction was equally efficient in producing the desired clinical result. It was also likely that they were situated on a neuronal circuit(s) taking a three dimensional course in this part of the diencephalon. In either case, the points were not subject to any further clarification by simple statistical tests.

(c) Tremor Abolition at Pallidal Level

The direction of the electrode track in the Guiot-Gillingham operation made it difficult to evaluate the effect of pure pallidal lesions on tremor, since there was always mechanical injury to the thalamic nuclei by the passage of electrode to reach the pallidum.

However, in common with the experience of other workers (Narabayashi et al 1956; Cooper 1961; Bertrand and Martinez 1962), Gillingham (1960) felt that even when tremor was abolished by a pallidal lesion, the tendency to recurrence was much greater than after a thalamic lesion.

In eight instances in the present series, tremor was not abolished by a lesion in the (presumed site of) thalamus but was abolished dramatically by a lesion in the (presumed site of) capsule or pallidum. These points were studied by drawing scattergrams and superimposing them on the stereotaxic atlas of Schaltenbrand and Bailey (1959). They seemed to be remarkably close together for a scattergram. The cases however were too few for any definite conclusions to be drawn. Even allowing for the smaller width of the internal capsule of the atlas brain as compared to the

present series, these points seemed to be well inside pallidum.

(d) Increase in Ipsilateral Tremor by Stereotaxic Surgery

In fifteen cases in the present series, there was a sudden and pronounced increase of tremor in the ipsilateral limbs at a single electrode position. The effect was seen either after mechanical injury by electrode insertion or after the creation of a reversible or irreversible lesion. Stimulation effects were not taken into consideration in the study of this phenomenon. This was a marked increase in ipsilateral tremor which could not be explained by the stress of the operating theatre atmosphere or as a relative increase in comparison to the opposite limb tremor reduced by surgery. The phenomenon was particularly striking when operating on the second hemisphere. Tremor on the ipsilateral side which had been completely abolished following the first operation might return to a mild degree during and after the second operation. The phenomenon has been recorded by Bertrand and Martinez (1962), French and his colleagues (1962) and Gillingham and Kalyanaraman (1963).

Inspection of the scatter diagram showing the central points of electrode positions when this phenomenon was observed, showed no particular predilliction for any structure to cause this increase of tremor.

Gillingham (1961) suggested that in Parkinsonism, one may be dealing with a 'firing' lesion similar to that in epilepsy. Ruch (1960) suggested the pallidum as the site of a discharging oscillatory lesion producing Parkinsonian

tremor. Gybels (1963) recorded evidence from animal experiments that 'firing' in some cortical cells may be closely related to tremor.

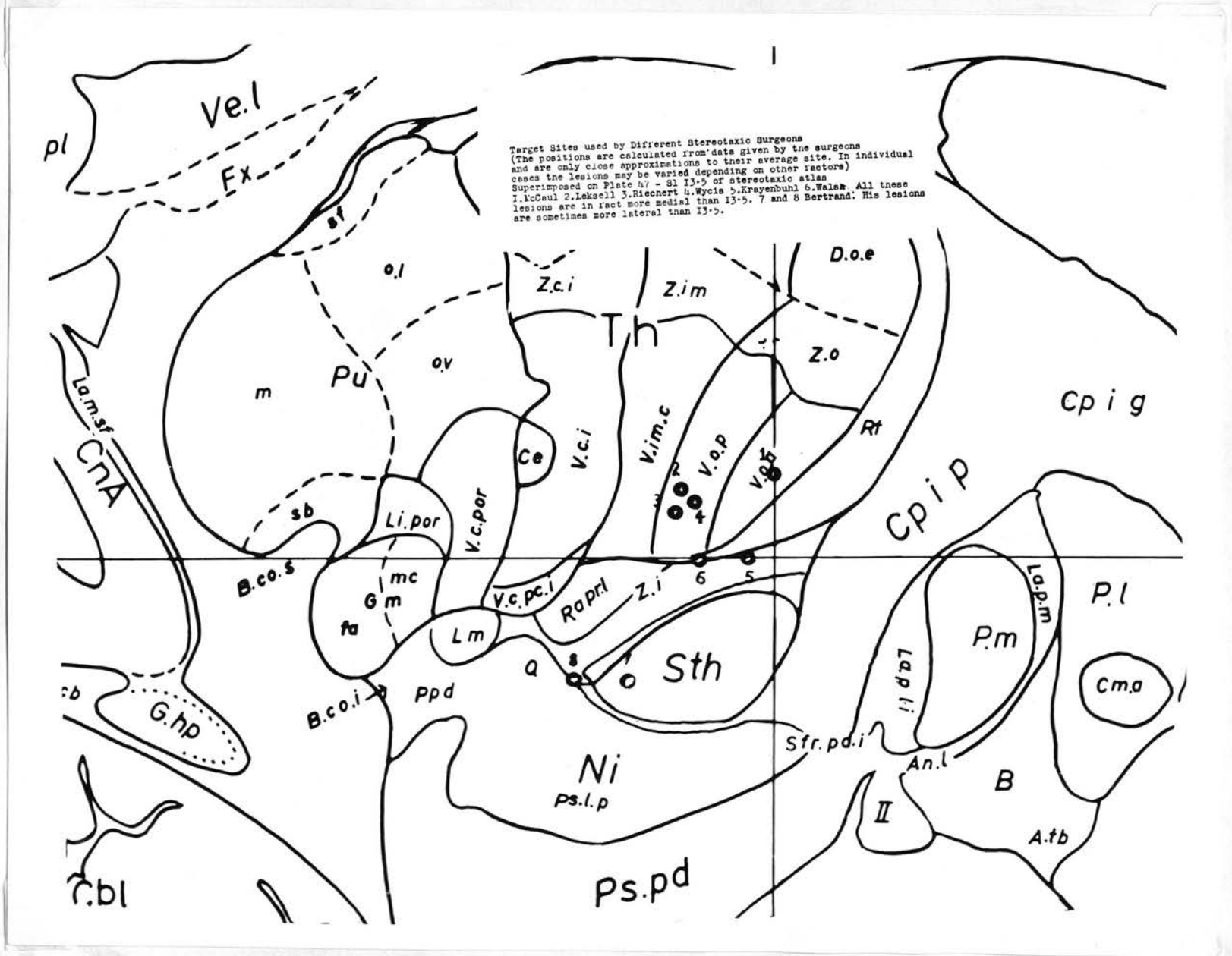
Spiegel and Wycis (1962) postulated that blocking of one path of pain impulses may increase the flow of impulses along an alternative route. Such a conception has been advanced by Gillingham (1960) to explain this phenomenon as well as the incidence of some complications after bilateral surgery like reduction of voice volume, dysphagia, depression, etc. Proof, however, of such a theory can be provided only after we are able to record deep intracerebral 'firing' at several points simultaneously.

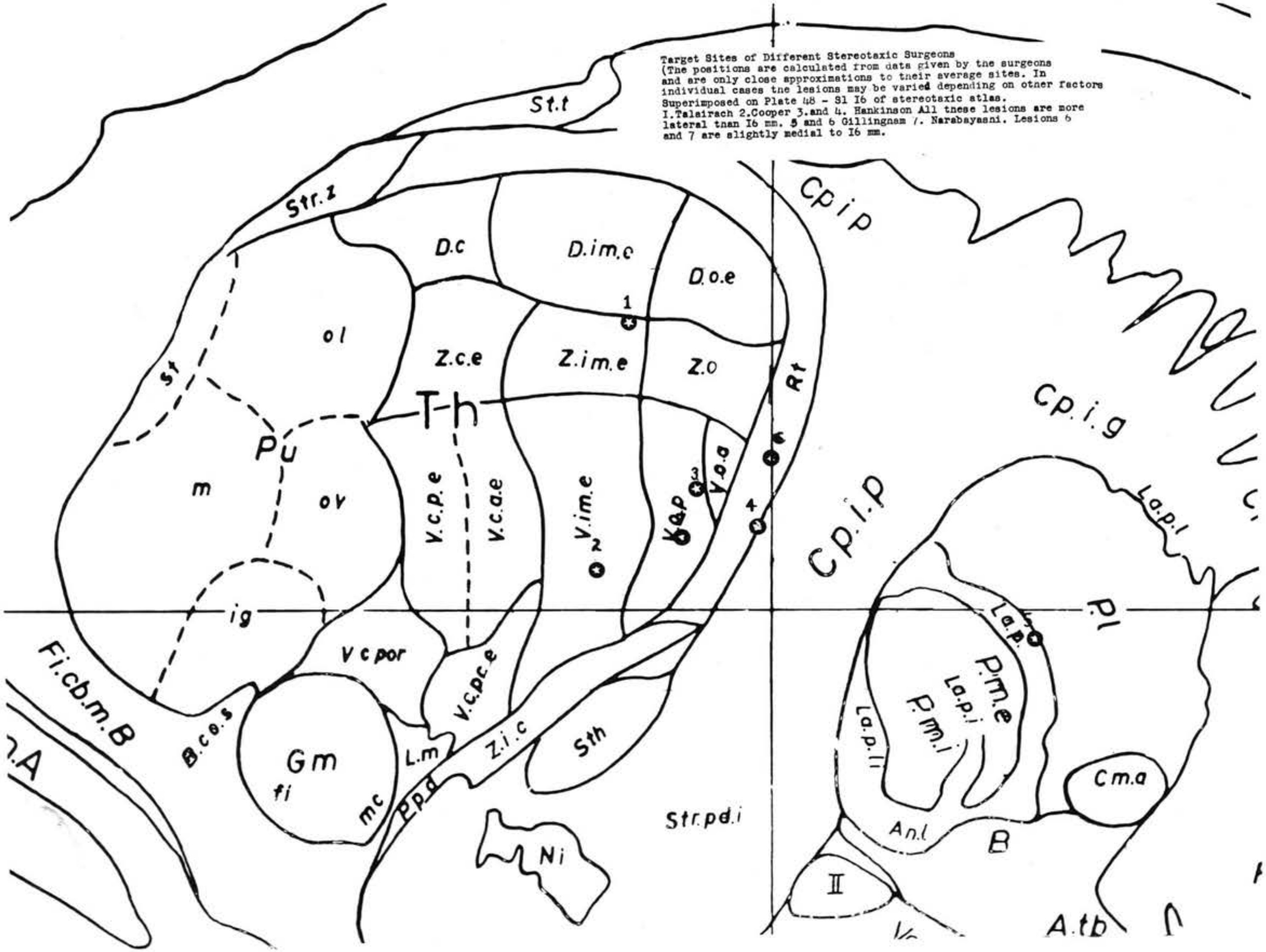
(e) Summary

The 'ideal' sites of lesion for abolition of Parkinsonian tremor by different stereotaxic surgeons were considered. These were found to be widely scattered - in the pallidum, internal capsule, reticular nucleus, oral ventral nucleus, ventral intermediate nucleus, other parts of the ventrolateral nucleus and the subthalamic region.

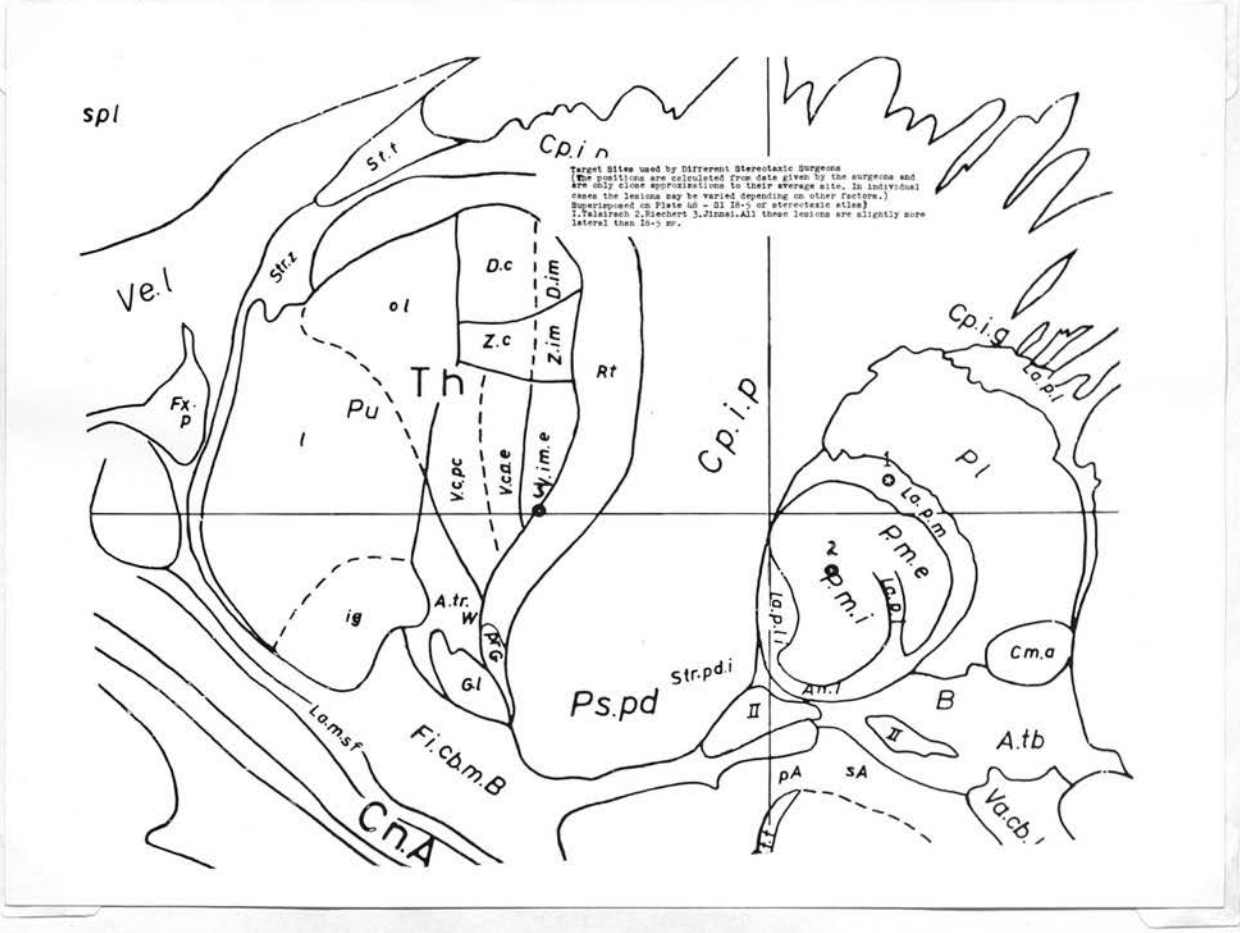
Scattergram studies of tremor abolition sites in the present series showed that tremor might be abolished in the pallidum, capsule or thalamus. At the last site, lateral distances from 12.5 mm. to 19.5 mm. might be equally effective as judged by the immediate effect.

These results suggested that tremor abolition was possible by effective interruption of a complex neuronal circuit(s) at any one of several levels. It was suggested that the ansa and fasciculus lenticularis might be part of such a pathway.

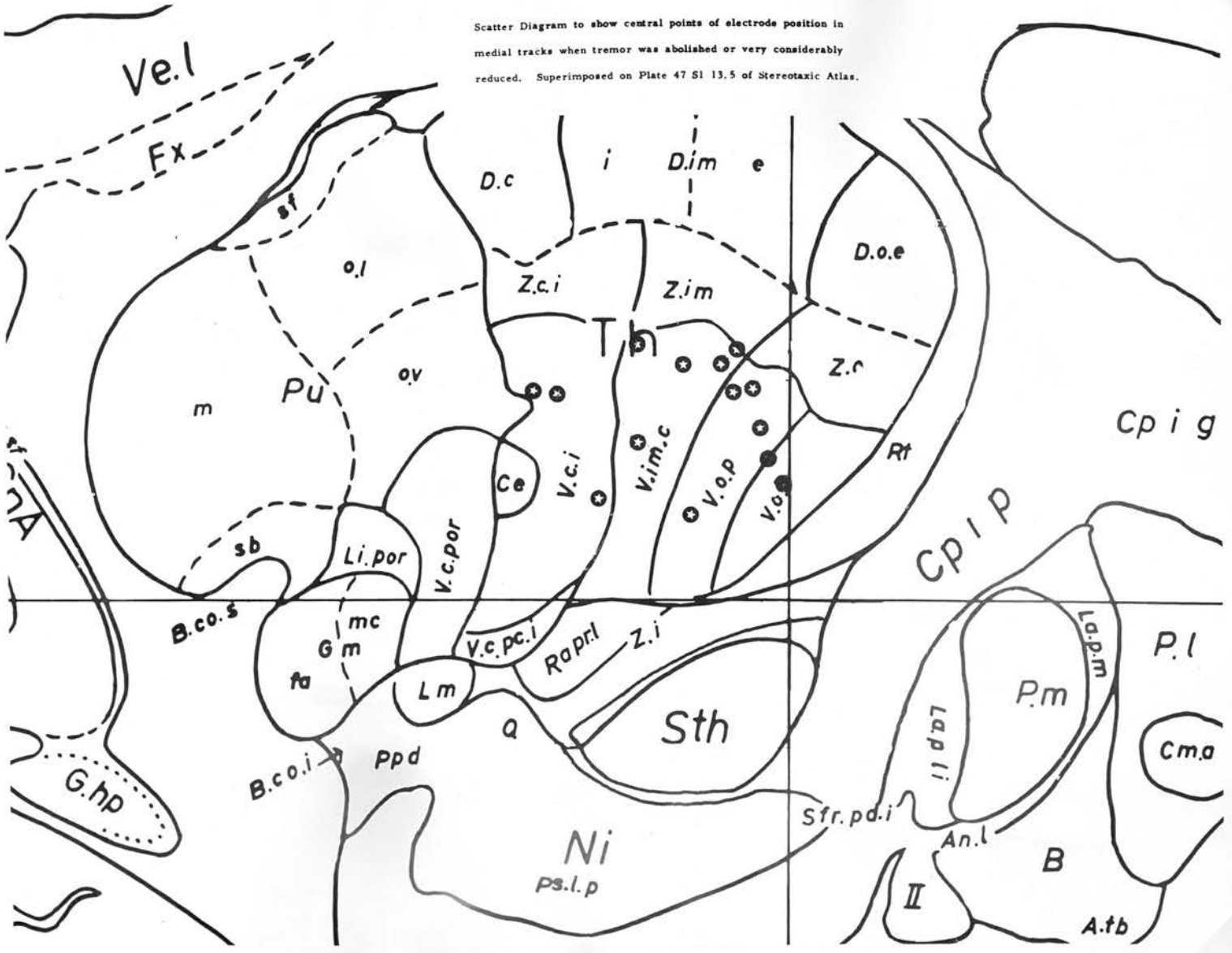

Target Sites used by Different Stereotaxic Surgeons
(The positions are calculated from data given by the surgeons and are only close approximations to their average site. In individual cases the lesions may be varied depending on other factors)
Superimposed on Plate 47 - SI 13·5 of stereotaxic atlas
1.McCaul 2.Leksell 3.Riechert 4.Wycis 5.Krayenbuhl 6.Walsh. All these lesions are in fact more medial than 13·5. 7 and 8 Bertrand. His lesions are sometimes more lateral than 13·5.
Ve.l
Fx
pl
st
o.l
Z.c.i
Z.im
D.o.e
Th
Z.o
Pu
o.v
m
La.m.sf
CnA
Ce
V.c.i
V.im.c
V.o.p
V.o.a
Rt
Cp i g
Cp i p
sb
Li.por
V.c.por
B.co.s
mc
G
m
fa
V.c.pc.i
Rapr.l
Z.i
6
5
Lm
Sth
a
B.co.i
Ppd
G.hp
Ni
Ps.l.p
Ps.pd
C.bl
Sfr.pd.i
An.l
B
II
A.tb
Lap.l
P.m
La.p.m
P.l
Cm.a



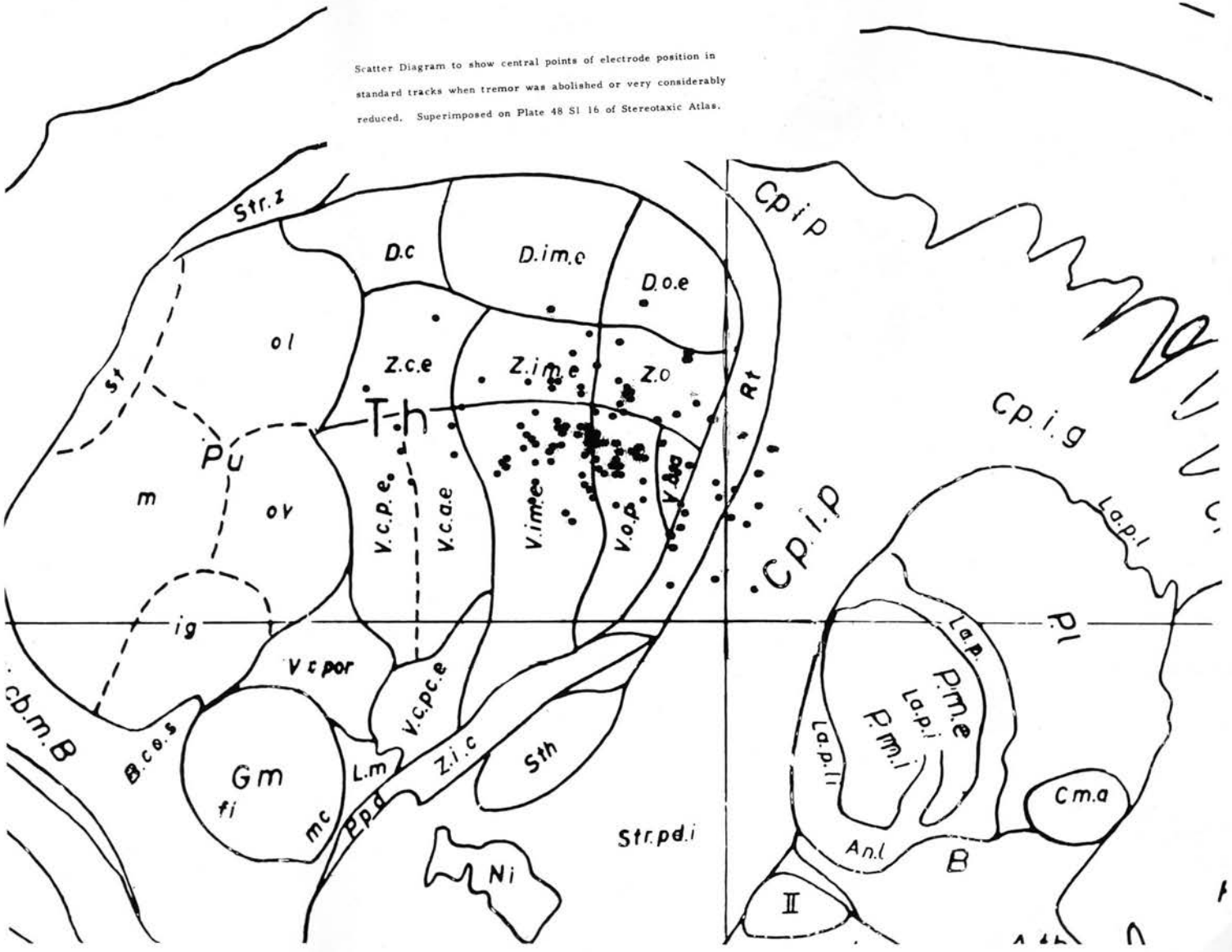
Target Sites of Different Stereotaxic Surgeons
(The positions are calculated from data given by the surgeons and are only close approximations to their average sites. In individual cases the lesions may be varied depending on other factors
Superimposed on Plate 48 - Sl I6 of stereotaxic atlas.
I.Talairach 2.Cooper 3.and 4. Hankinson All these lesions are more lateral than I6 mm. 5 and 6 Gillingham 7. Narabayashi. Lesions 6 and 7 are slightly medial to I6 mm.

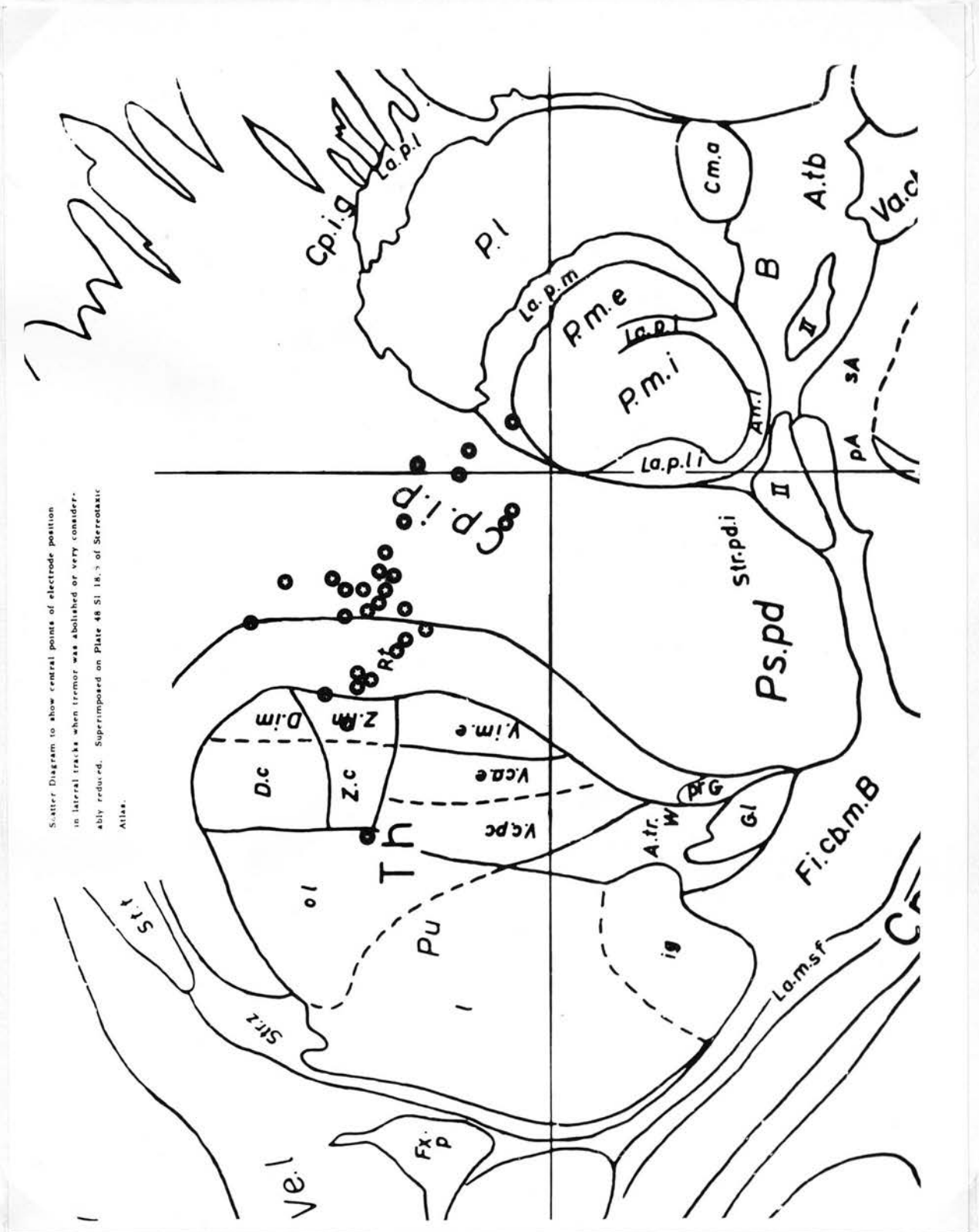

Target Sites used by Different Stereotaxic Surgeons
(The positions are calculated from data given by the surgeons and are only close approximations to their average site. In individual cases the lesions may be varied depending on other factors.)
Superimposed on Plate 46 - AP 10.5 of stereotaxic atlas)
1.Talairach 2.Riechert 3.Jinnai. All these lesions are slightly more lateral than 10.5 mr.
spl
St.t
Cp.i.n
Ve.l
Str.l
o.l
D.c
D.im
Z.c
Z.im
Th
Rt
Fx.p
Pu
l
V.c.pc
V.c.a.e
V.i.m.e
Cp.i.p
Cp.i.g
La.p.l
P.l
La.p.m
P.m.e
P.m.i
La.p.l
La.p.l
A.tr.w
ig
Gl
Str.pd.i
Ps.pd
An.l
II
B
Cm.a
II
A.tb
Va.cb.l
pA
sA
Lam.sf
Fi.cb.m.B
Cn.A

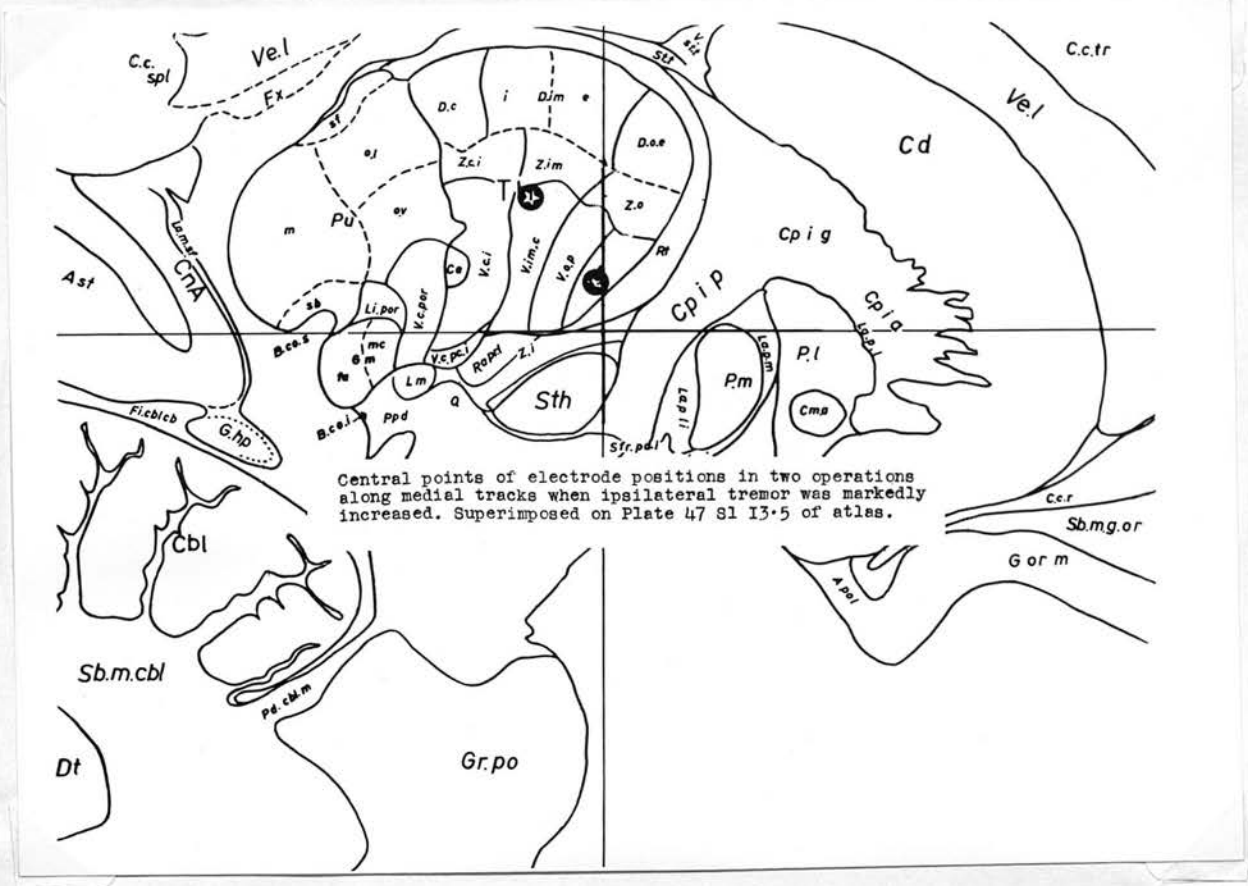
Scatter Diagram to show central points of electrode position in medial tracks when tremor was abolished or very considerably reduced. Superimposed on Plate 47 Sl 13.5 of Stereotaxic Atlas.



Scatter Diagram to show central points of electrode position in standard tracks when tremor was abolished or very considerably reduced. Superimposed on Plate 48 Sl 16 of Stereotaxic Atlas.

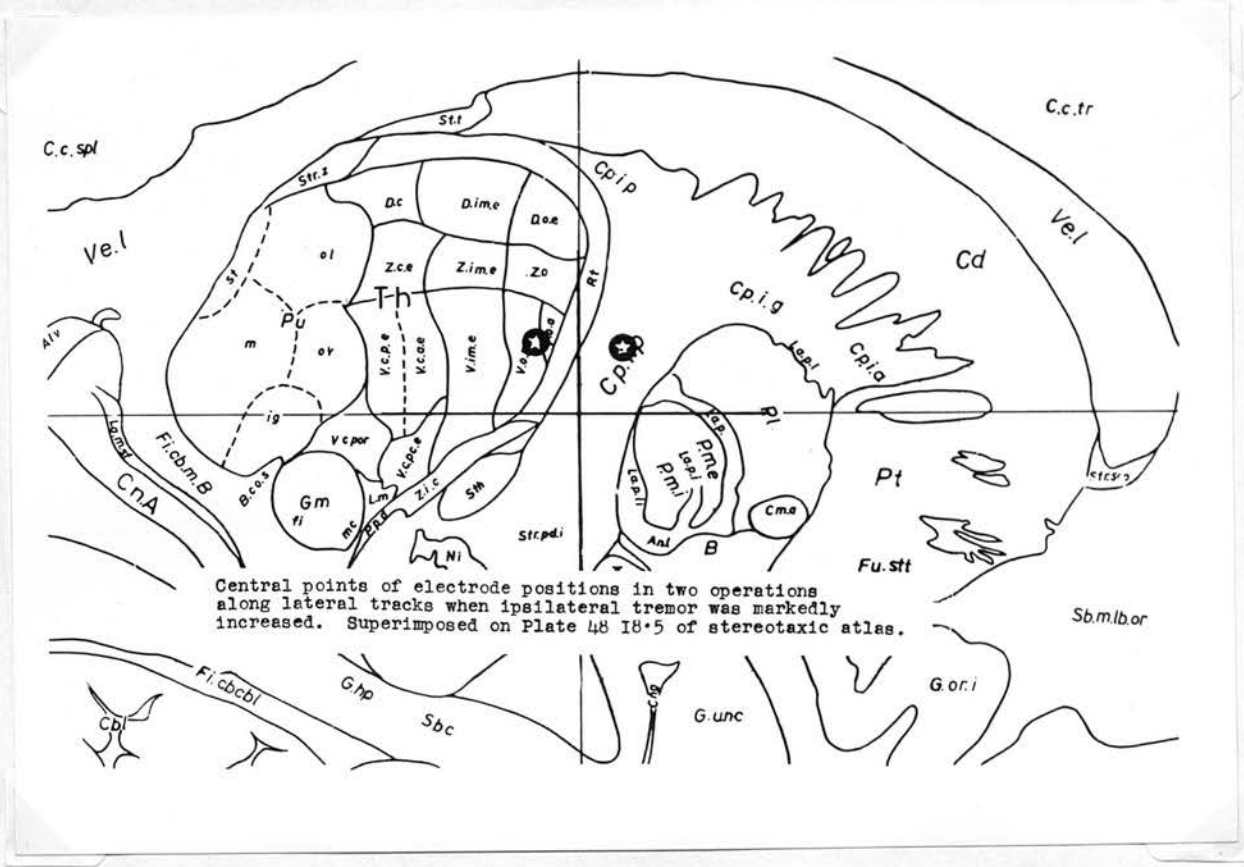



Cp.i.g
La.p.l
P.l
cm.a
A.tb
B
La.p.m
Pm.e
La.p.l
P.m.i
Am.l
II
SA
pA
La.p.l.i
Str.pd.i
Ps.pd
Rb
D.im
Z.m
V.im.e
D.c
Z.c
V.ca.e
V.c.pc
A.tr.
W
pfG
Gl
Fi.cb.m.B
La.m.sf
Str.t
Str.z
Fx.p
Ve.l
Pu
Th
Scatter Diagram to show central points of electrode position
in lateral tracks when tremor was abolished or very consider-
ably reduced. Superimposed on Plate 48 Si 18.3 of Stereotaxic
Atlas.

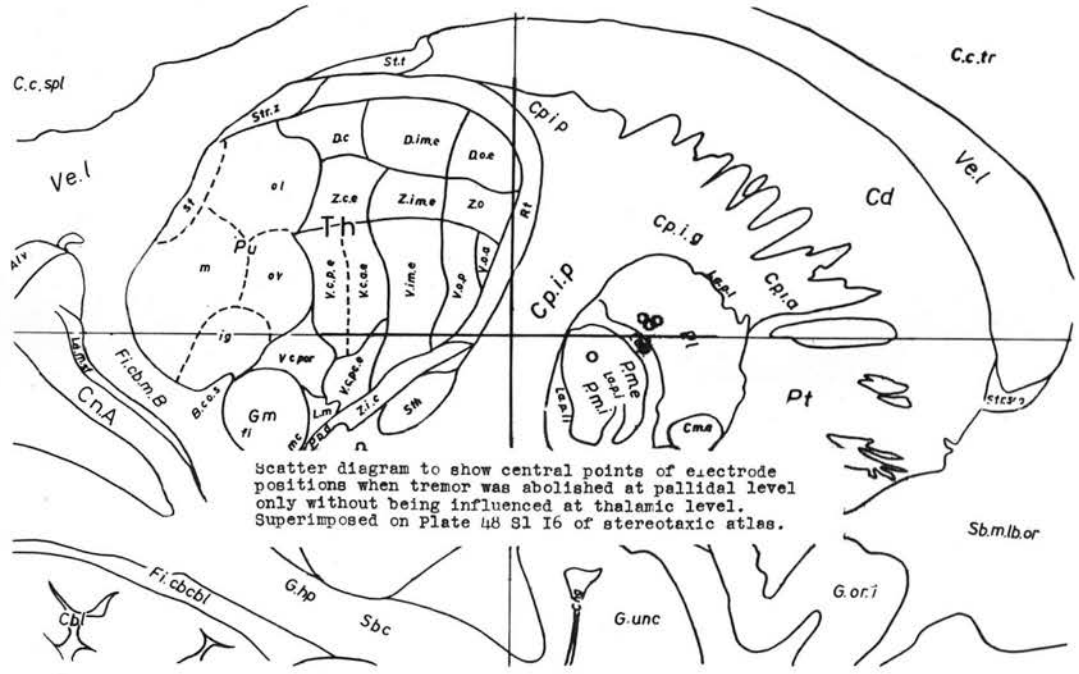


Central points of electrode positions in two operations along medial tracks when ipsilateral tremor was markedly increased. Superimposed on Plate 47 Sl I3·5 of atlas.

Scatter Diagram to show central points of electrode position in II operations along standard tracks when ipsilateral tremor was markedly increased. Superimposed on Plate 48 Sl I6 of stereotaxic atlas.



Central points of electrode positions in two operations along lateral tracks when ipsilateral tremor was markedly increased. Superimposed on Plate 48 I8·5 of stereotaxic atlas.



Scatter diagram to show central points of electrode positions when tremor was abolished at pallidal level only without being influenced at thalamic level. Superimposed on Plate 48 S1 I6 of stereotaxic atlas.

REFERENCES

1. Aguinis, M. (1963). Acta Neurochir., 9, 1, 151.
2. Bertrand, C. and Martinez, M. (1962). Confin. Neurol., 22, 274.
3. Caracalos, A., Levita, E. and Cooper, I.S. (1962). St. Barnabas Hospital Medical Bulletin. 1, 3, 24.
4. Cooper, I.S. (1958). Neurology, 8, 703.
5. Cooper, I.S. (1961). Parkinsonism. Its Medical and Surgical Therapy. C.C. Thomas. Springfield, Illinois. 57.
6. French, L.A., Story, J.L., Galicich, J.H. and Schultz, E.A. (1962). Confin. Neurol., 22, 265.
7. Gillingham, F.J. (1960). Personal Communication.
8. Gillingham, F.J. (1961). Proc. Roy Soc. Med., 54, 375.
9. Gillingham, F.J. (1962). Confin. Neurol., 22, 385.
10. Gillingham, F.J. and Kalyanaraman, S. (1963). Excerpta Medica International Congress Series No. 60. Second European Congress of Neurological Surgery. 115.
11. Guiot, G. (1963). Personal Communication.
12. Gybels, J.M. (1963). The Neural Mechanism of Parkinsonian Tremor. Editions Arscia S.A. Brussells.
13. Hankinson, J. (1963). Personal Communication.
14. Hassler, R. (1955). Proc. 2nd Intr. Congr. Neuropath. London. Part IV, 637. Excerpta Medica. Amsterdam.
15. Hassler. R. and Riechert, T. Quoted by Spiegel, E.A.

(1958). and Wycis, H.T. Stereo-encephalotomy. Part II. Clinical and Physiological Applications. Grune and Stratton. New York. 1962. 328.

16. Hassler, R. (1963). Personal Communication.

17. Hughes, B. (1963). Personal Communication.

18. Jefferson, G. (1956). In Turner, G.G. and Rogers, L.C. Ed. Modern Operative Surgery. Cassell and Company Limited. London. Fourth edition. 2. 1423.

19. Jinnai, D., Nishimoto, A., Matsumoto, K. and Handa, S. (1961). Excerpta Medica International Congress Series No. 36. Second International Congress of Neurological Surgery. E 94.

20. Krayenbuhl, H. (1962). Confin. Neurol., 22, 314.

21. Leksell, L. and Strang, R. (1963). Personal Communication.

22. McCaul, I.R. (1963). Personal Communication.

23. Narabayashi, H., Okuma, T., and Shikiba, S. (1956). A.M.A. Arch. Neurol. Psychiat., 75, 36.

24. Narabayashi, H. (1963). Personal Communication.

25. Parkinson, J. (1817). An Essay on the Shaking Palsy. Sherwood, Neely and Jones. London. Reprinted in Critchley, M. Ed. James Parkinson. Macmillan and Company. London. 1955. 155.

26. Riechert, T. and Hassler, R. (1963). Personal Communication.

27. Ruch, T.C. (1960). In Ruch, T.C. and Fulton, J.F. Ed. Medical Physiology and Biophysics. W.B. Saunders Company. Philadelphia and London. 284.

28. Schaltenbrand, G. and Bailey, P. (1959). Introduction to Stereotaxis with an atlas of the Human Brain. Thieme, Stuttgart. 2.

29. Spiegel, E.A. and Wycis, H.T. (1962). Stereoencephalotomy. Part II. Clinical and Physiological Applications. Grune and Stratton. New York. 338.

30. Spiegel, E.A., Wycis, H.T., Szekely, E.G., Adams, J., Flanagan, M. and Baird III, H.W. (1963). J. Neurosurg., 20, 871.

31. Talairach, J. and Szikla, G. (1963). Personal Communication.

32. Walsh, L.S. (1962). Personal Communication.

33. Wycis, H.T. and Spiegel, E.A. (1952). Confin. Neurol., 12, 245.

CHAPTER 7

EFFECT OF STEREOTAXIC SURGERY ON RIGIDITY

"Hitherto the patient will have experienced but little inconvenience; and befriended by the strong influence of habitual endurance, would perhaps seldom think of his being the subject of disease, except when reminded of it by the unsteadiness of his hand, whilst writing or employing himself in any nicer kind of manipulation. But as the disease proceeds, similar employments are accomplished with considerable difficulty, the hand failing to answer with exactness to the dictates of the will. Walking becomes a task which cannot be performed without considerable attention. The legs are not raised to that height, or with that promptitude which the will directs, so that the utmost care is necessary to prevent frequent falls."

..... James Parkinson.

"Actually, there is a constant play of forces acting on the central nervous system, simulating inactivity when there is a state of equilibrium, appearing as function when there are shifts of equilibrium and manifesting itself as a pathologic symptom when only a disequilibrium exists."

..... W.R. Hess.

(a) Introduction

Parkinson (1817) made the observation that tremor ceased when a state of hemiparesis supervened on limbs affected by Parkinsonism. Numerous operations which followed in the earlier part of this century were based on this observation. All of them aimed principally at interruption of the pyramidal tract at some level.

The cortical operations of Klemme (1940) and of Bucy and Case (1945), the capsular operations of Browder (1948), the peduncular operations of Walker (1952) and the spinal cord sections of Putnam (1938) and of Oliver (1953) are very well known. They were however mainly directed towards the alleviation of tremor. They were followed by a flaccid paralysis, soon replaced by spasticity which was

sometimes severe. Such a state was not always a preferred alternative to rigidity.

Meyers (1942) changed the direction of neurosurgical thinking by his observation that surgical interruption of pallidofugal fibres might modify rigidity and tremor in patients with Parkinsonism. For the first time it was possible to reduce or abolish rigidity without producing paresis or spasticity.

The transventricular approach of Meyers (1951) was associated with a relatively high mortality and morbidity. However, the open subfrontal operations of Fenelon (1950) and of Guiot and Brion (1953) were less hazardous. Stereotaxic procedures markedly increased the safety of surgery for Parkinsonism by greatly reducing the incidence of complications. Pallidal targets were soon changed to thalamic targets to deal with tremor better. Although a large number of surgeons were satisfied with the degree of reduction of rigidity obtained at thalamic levels, a few still preferred to make pallidal lesions, when rigidity was the predominating factor.

(b) Ideal Site of Lesion for Parkinsonian Rigidity

It was interesting to compare the views of various surgeons with regard to what they considered as the optimum site for stereotaxic lesions in case of rigidity.

Bertrand and Martinez (1962) preferred a slightly more anterior lesion for rigidity than for tremor, both lesions being in the thalamus. Their preferred site was 13 mm. behind and 6 mm. below the foramen of Monro and 14 mm. lateral to the midline.

Gillingham (1960) preferred a pallidocapsular lesion for rigidity. His pallidal target was 3 mm. behind and 1 mm. below the posterior border of the anterior commissure. Gillingham (1962) demonstrated that rigidity could be effectively controlled by his transcapsular lesions.

Cooper (1961) did not use separate targets for tremor and rigidity. His lesion was enlarged at the target site in slow degrees over a few days until both rigidity and tremor was controlled without side effects.

Guiot (1963) did not prefer separate target sites for tremor and rigidity. He considered his lesion in the ventral oral nucleus of the thalamus (placed after depth electrical recording and audiomonitoring) to be capable of abolishing both symptoms.

Hankinson (1963) extended his standard lesion used for tremor anteriorly by 2 mm. if rigidity was prominent.

Hughes (1961) felt that lesions in the anterior part of the oral ventral nucleus were more likely to influence rigidity and lesions in the posterior part, more effective for tremor.

Krayenbuhl and Yasargil (1963) made their thalamic lesion at a slightly more anterior site if rigidity was more prominent than tremor.

Riechert and Hassler (1963) preferred a lesion in the anterior part of the oral ventral nucleus for rigidity and in the posterior part for tremor. If in one hemisphere, the anterior part of this nucleus had been coagulated, they preferred to make a pallidal lesion on the second hemisphere if rigidity was the main feature. Their pallidal target

lay 5 mm. behind and 4 mm. below the foramen of Monro with reference to the FM-CP line. Corrections were applied to these figures for the FM-CP length difference between their standard brain and the patient. The target lay 17 mm. lateral to the lower part of the wall of the third ventricle and this distance was again corrected for the lateral ventricular width of the individual patient.

Talairach and Szikla (1963) preferred a pallidal lesion for rigidity. This was calculated as a point 1 mm. above and 1 mm. in front of the junction of the anterior onethird and the posterior twothirds of their CA-CP line. It was 20 mm. lateral to the midline.

Opinions therefore among the various surgeons could be classed into four groups with regard to their choice of targets for tremor and rigidity.

(a) Surgeons like Gillingham and Talairach who preferred pallidal and thalamic lesions respectively for rigidity and tremor.

(b) Surgeons like Riechert who preferred a pallidal lesion for rigidity only in some cases of second hemisphere operation.

(c) Surgeons like Bertrand, Hughes, Hankinson and Krayenbuhl who preferred a thalamic lesion in all cases but placed it slightly more anteriorly for rigidity and posteriorly for tremor.

(d) Surgeons like Cooper and Guiot who used the same target for both rigidity and tremor.

The majority of surgeons belonged to the latter two groups.

A comparison of all these target sites made it obvious that there was no single 'ideal' target. As in the case of tremor, it was likely that one was dealing with a complex neuronal circuit and its effective interruption at any level could achieve the desired clinical result.

(c) Scattergram Studies of the Sites of Reduction of Rigidity

In 181 instances in the present series, rigidity was reduced by 50% or more (clinical observer's grading) at a single electrode position either by mechanical disruption or by the creation of minimal heating lesions, reversible for practical purposes. A few were due to coagulation lesions. An analysis of their incidence according to the laterality of the track is shown below. The small difference in the proportions between the different groups was not considered important enough for statistical tests of significance to be applied.

Tracks	No. of patients in whom rigidity was reduced by 50% or more at a single electrode position	Total no. of explorations
Very Medial Sl 11.0	2	6
Medial Sl 13.5	15	41
Standard Sl 16.0	130	323
Lateral Sl 18.5	31	95
Very Lateral Sl 21.0	3	10
Total	181	475

A study of the scattergrams of the central points of such positions of the electrode along medial tracks showed them to be scattered in a fashion similar to the tremor abolition points. They were grouped around the posterior part of the oral ventral nucleus, the ventral intermediate nucleus and the pallidocapsular junction (allowing for the smaller breadth of the capsule of the atlas brain as compared to our patients).

Examination of the scattergrams for the standard tracks showed these points to be widely scattered in the central intermediate nucleus, the oral ventral nucleus, the ventral intermediate nucleus and the reticular nucleus of the thalamus, the internal capsule and the inner and outer segments of the pallidum.

Scrutiny of the scattergrams for the lateral tracks again showed a wide distribution but mainly centred in and around the internal capsule and the adjacent parts of the thalamus and the pallidum.

Such an arrangement confirmed the opinion that reduction of rigidity could be achieved at different levels and pallidofugal impulses could be interrupted in the pallidum, capsule or the thalamus. One of the main advantages of the method of stereotaxy as practised by Gillingham would therefore seem to be that effective lesions could be placed at one or more of these three levels by a single insertion of the electrode. It might also explain the relatively high percentage of successful abolition of tremor and rigidity.

Such a concept would also be in consonance with the 'firing lesion' theory of Parkinsonism. It could be explained that such a 'firing' could be effectively blocked at several points along its pathway to the periphery. Albe-Fessard and Guiot (1963), Gillingham, Gaze and Kalyanaraman (1963) and Jasper (1963) have detected by microelectrode studies tremor activity in the thalamus. Carrascosa, Carbonell and Sevillano observed irregular slow and sharp waves during an oculogyric crisis by depth recording from the subthalamic region. Spiegel and Wycis (1962) noted in tegmental electrograms slow waves synchronous with peripheral tremor. Another interesting observation (Gillingham and Kalyanaraman, 1963) was that in the present series of more than 300 Parkinsonian patients, there was no case of epilepsy.

Whatever the exact pathophysiological mechanism of tremor, rigidity and other Parkinsonian manifestations may be, there seemed to be little doubt from the scattergram studies that they could be benefited by thalamic, capsular or pallidal lesions.

At the thalamic level, in 95 instances, both tremor and rigidity were benefited at the same electrode position. In 15 explorations rigidity was reduced markedly at a point about 2 mm. (range 1-5 mm.) in front of the point where tremor was reduced. In 5 cases, rigidity was reduced markedly at a point about 2 mm. (range 1-3 mm.) behind the point where tremor was relieved.

In some cases, rigidity was reduced by about 50% at thalamic (or thalamocapsular) level and then was totally

abolished at a pallidal (or pallidocapsular) level several mm. in front. The number of cases where this occurred seemed to bear no definite relation to the laterality of the track except that the proportion seemed to be less on lateral tracks. One might explain this anatomically by the fact that the lateral tracks were less likely to involve the medially placed oral ventral nucleus of the thalamus.

Tracks	No. of cases where total abolition of rigidity was achieved in two stages at thalamic and pallidal levels	Total no. of explorations.
Very Medial Sl 11.0	0	6
Medial Sl 13.5	4	41
Standard Sl 16.0	26	323
Lateral Sl 18.5	5	95
Very lateral Sl 21.0	0	10

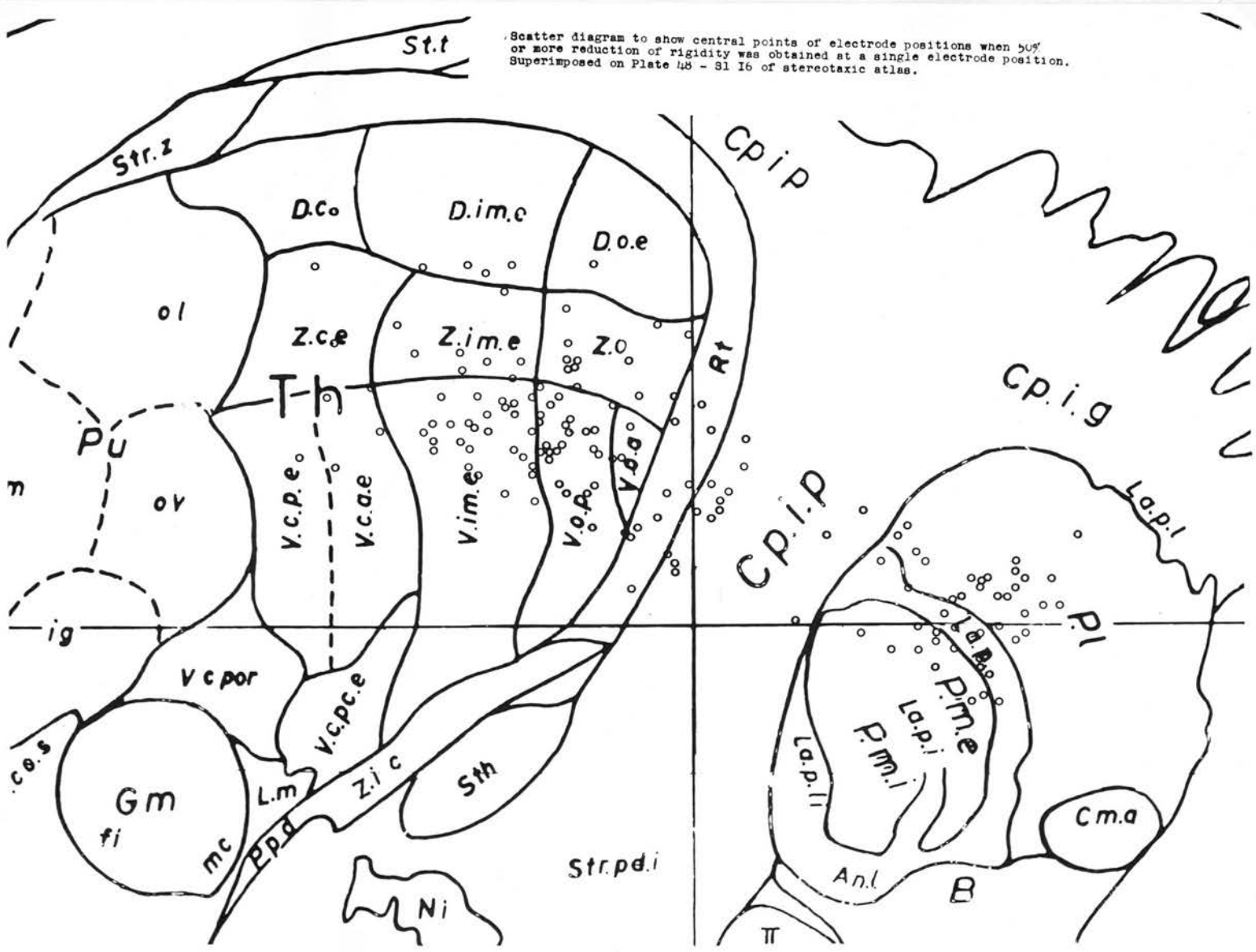
A scattergram was constructed for the central points of the electrode position along the standard tracks where a thalamic lesion reducing rigidity needed to be supplemented by a pallidal lesion for total abolition of rigidity. Examination of this scattergram and its comparison with the scattergram for all rigidity reduction points showed no essential difference in the thalamic site of the lesion. It was likely that in these cases the total volume of the lesion was the crucial factor rather than its position, thus producing a more complete section

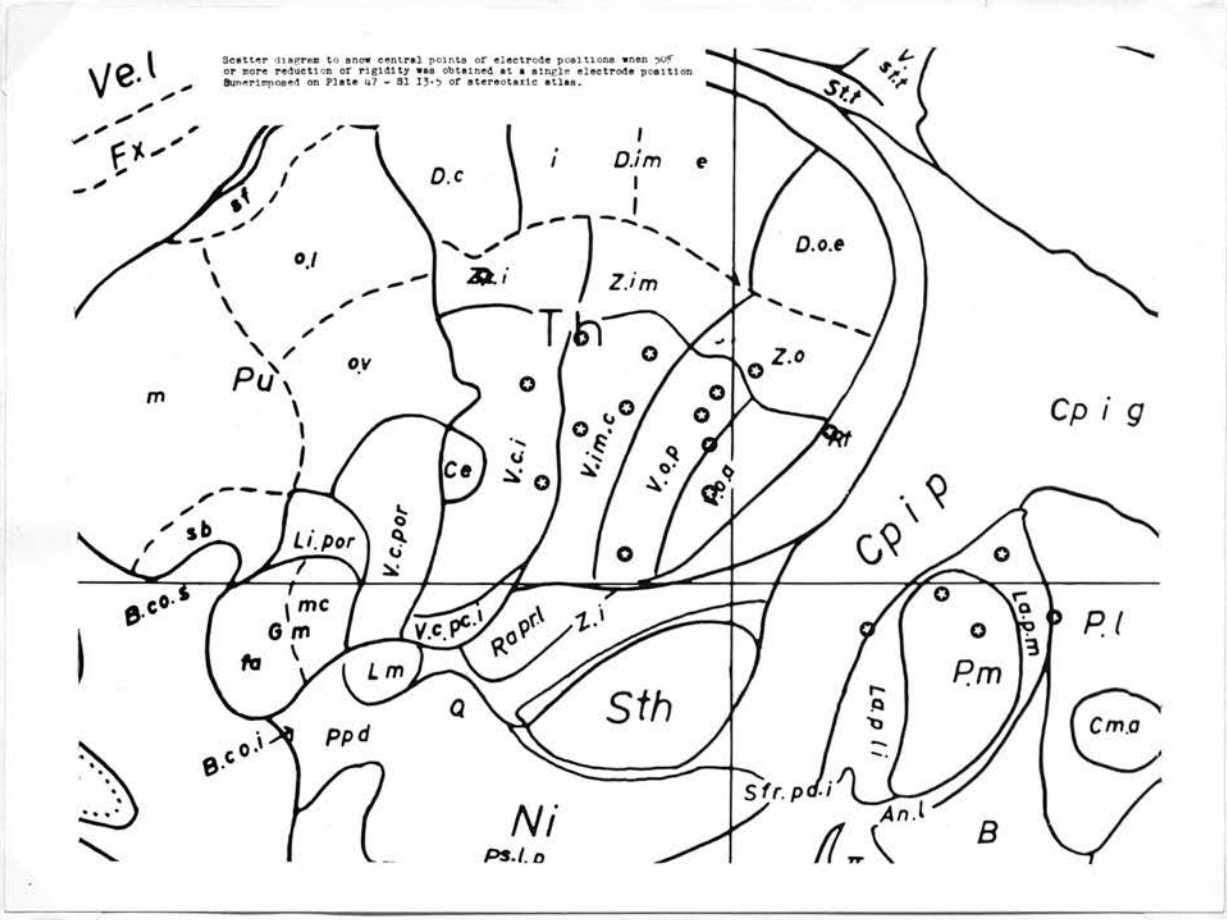
of the pallidofugal fibres.

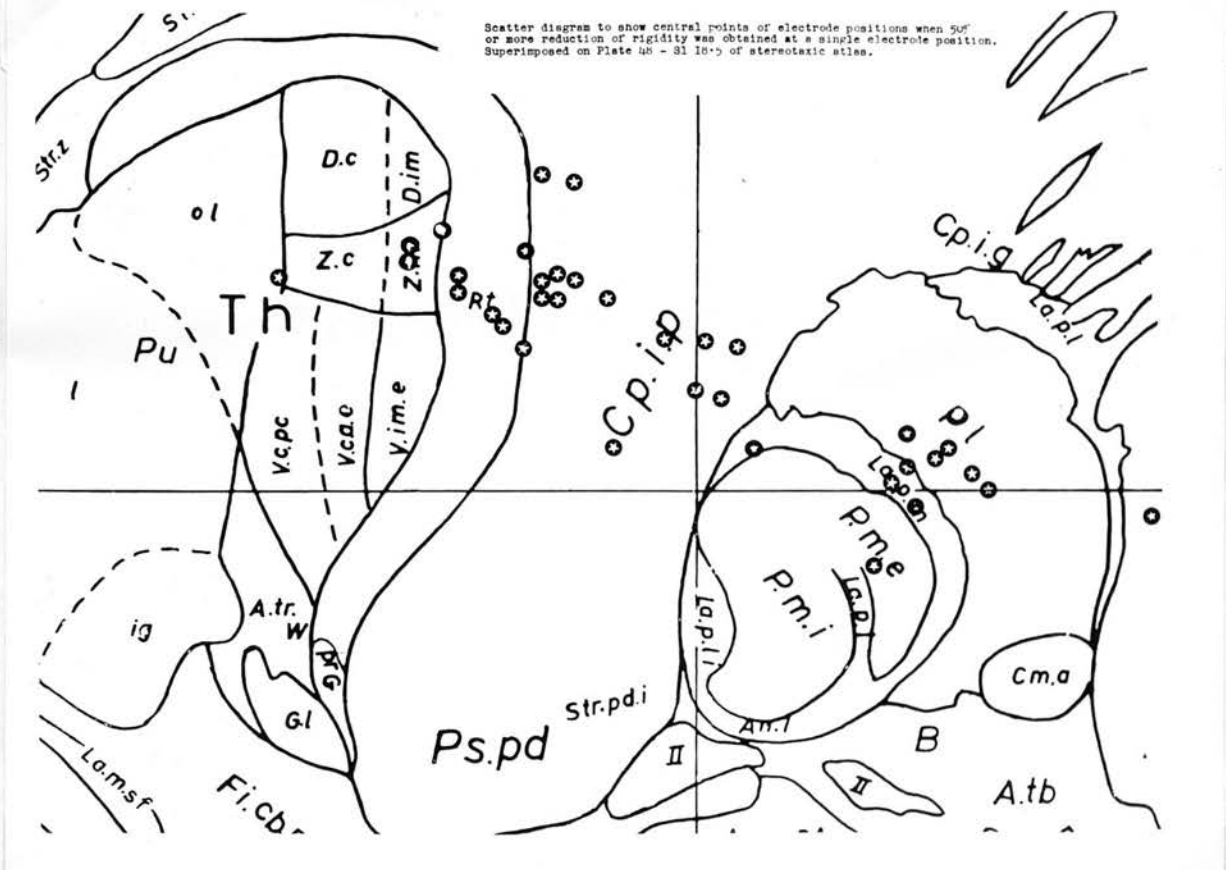
(d) Summary

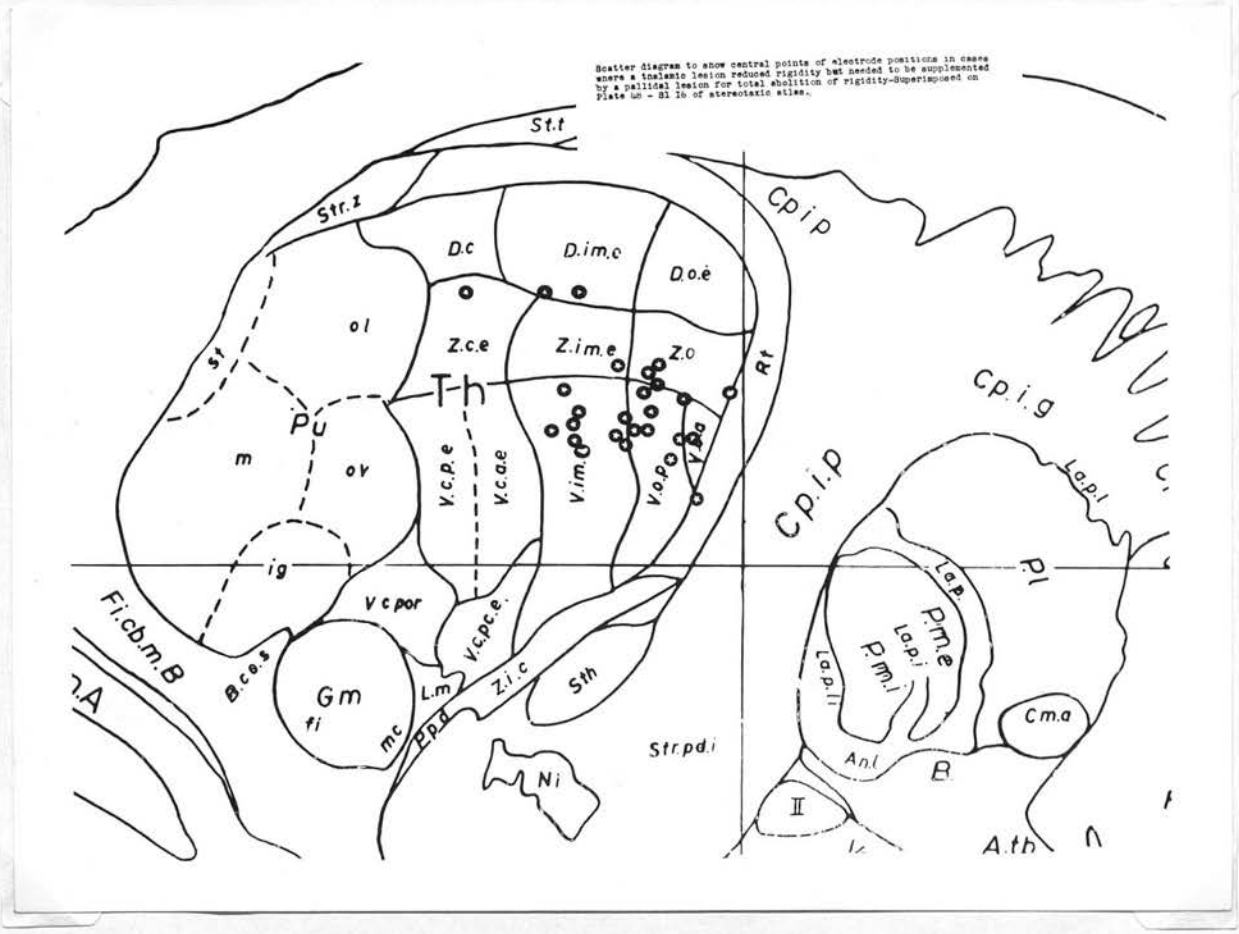
The preferred sites of stereotaxic lesions of various surgeons for abolition of Parkinsonian rigidity were compared.

Scattergrams, constructed from points where rigidity was reduced at a single electrode position, were studied. It was concluded that rigidity could be dealt with effectively at the thalamic, capsular or pallidal level. It was felt that in some cases, the total volume of the lesion is probably as important as its siting.


Scatter diagram to show central points of electrode positions when 50% or more reduction of rigidity was obtained at a single electrode position. Superimposed on Plate 48 - Sl I6 of stereotaxic atlas.
St.t
Str.z
D.Co
D.im.c
D.o.e
ol
Z.c.e
Z.im.e
Z.o
Th
Pu
ov
V.c.p.e
V.c.a.e
V.im.e
V.o.p
V.b.a
Rt
Cp.i.p
Cp.i.g
La.p.l
ig
V c por
V.c.pc.e
Gm
fi
mc
L.m
Z.i c
Sth
Ppd
Str.pd.i
Ni
Pl
P.l.Bo
P.im.e
La.p.i
P.m.l
La.p.ll
Cm.a
An.l
B
π


Scatter diagram to show central points of electrode positions when 50% or more reduction of rigidity was obtained at a single electrode position Superimposed on Plate 47 - SI 13-5 of stereotaxic atlas.
Ve.l
Fx
D.c
i
D.im
e
st
o.l
Z.i
Z.im
D.o.e
Th
Pu
o.v
Z.o
m
Cp i g
Ce
V.c.i
V.im.c
V.o.p
Rt
sb
Li.por
V.c.por
Cp i p
B.co.s
mc
G
m
V.c.pc.i
Rapr.l
Z.i
La.p.m
P.l
fa
Lm
Q
Sth
P.m
La.p.l
Cm.a
B.co.i
Ppd
Str.pd.i
An.l
Ni
B


Scatter diagram to show central points of electrode positions when 50% or more reduction of rigidity was obtained at a single electrode position. Superimposed on Plate 48 - SI 18-5 of stereotaxic atlas.
Str.z
D.c
D.im
o l
Z.c
Z.im
Th
Rt
Pu
Cp.i.g
La.p.l
Cp.i.p
V.c.pc
V.ca.c
V.im.e
P.l
Lap.m
P.me
P.m.i
La.p.l
La.p.l
A.tr.
W
ig
Cm.a
Str.pd.i
An.l
G.l
Ps.pd
B
La.m.sf
Fi.cb
A.tb


Scatter diagram to show central points of electrode positions in cases where a thalamic lesion reduced rigidity but needed to be supplemented by a pallidal lesion for total abolition of rigidity-Superimposed on Plate 40 - 21 lb of stereotaxic atlas.
St.t
Str.t
D.c
D.im.c
D.o.e
ol
Z.c.e
Z.im.e
Z.o
T-h
St
Pu
m
or
V.c.p.e
V.c.a.e
V.im.
V.p.o
V.Ba
Rt
Cp.i.p
Cp.i.g
La.p.l
ig
Pl
V c por
V.c.pc.e.
Fi.cb.m.B
B.c.s
Gm
fi
mc
L.m
Z.i.c
Sth
Ppd
Str.pd.i
Ni
La.p
P.m.e
La.p.i
P.m.l
La.p.ll
Cm.a
An.l
B
II
A.th

REFERENCES

1. Albe-Fessard, D. and Guiot, G. (1963). Personal Communication.
2. Bertrand, C. and Martinex, N. (1962). Confin. Neurol., 22, 274.
3. Browder, J. (1948). Am. J. Surg., 75, 264.
4. Bucy, P.C. and Case, J.T. (1945). Ann. Surg., 122, 933.
5. Carrascosa, Carbonell and Sevillano. Quoted by Obrador, S. Confin. Neurol., 22, 283, 1962.
6. Cooper, I.S. (1961). Parkinsonism. Its Medical and Surgical Therapy. C.C. Thomas. Springfield, Illinois. 57.
7. Fenelon, F. (1950). Rev. Neurol., 83, 437.
8. Gillingham, F.J. (1960). Personal Communication.
9. Gillingham, F.J. (1962). Confin. Neurol., 22, 385.
10. Gillingham, F.J., Gaze, R.M. and Kalyanaraman, S. (1963). Unpublished Information.
11. Gillingham, F.J. and Kalyanaraman, S. (1963). Unpublished Information.
12. Guiot, G. and Brion, S.(1953). Rev. Neurol., 89, 578.
13. Guiot, G. (1963). Personal Communication.
14. Hankinson, J. (1963). Personal Communication.
15. Hess, W.R. (1957). The Functional Organisation of the Diencephalon. Grune and Stratton, New York. x.

16. Hughes, B. (1961). In Carling, E.R. and Ross, J.P. Ed. British Surgical Practice, Surgical Progress. 1961. Butterworth, London. 129.

17. Jasper, H.H. (1963). Quoted by Gillingham, F.J. Personal Communication.

18. Klemme, R.M. (1940). Arch. Neurol. & Psychiat., 44, 926.

19. Krayenbuhl, H. and Yasargil, M.G. (1963). Personal Communication.

20. Meyers, R. (1942). N.Y. State J. Med., 42, 317.

21. Meyers, R. (1951). Acta Psychiat. et Neurol., Suppl. 67, 1.

22. Oliver, L.C. (1953). Parkinson's Disease and Its Surgical Treatment. Lewis. London.

23. Parkinson, J. (1817). An Essay on the Shaking Palsy. Sherwood, Neely and Jones. London. Reprinted in Critchley, M. Ed. James Parkinson. Macmillan and Co. London. 1955. 156.

24. Putnam, T.J. (1938). Arch. Neurol. & Psychiat., 40, 1049.

25. Riechert, T. and Hassler, R. (1963). Personal Communication.

26. Spiegel, E.A. and Wycis, H.T. (1962). Stereoencephalotomy. Part II. Clinical and Physiological Applications. Grune and Stratton. New York, 407.

27. Talairach, J. and Szikla, G. Personal Communication.

28. Walker, A.E. (1952). J. Nerv. & Ment. Dis., 116, 766.

CHAPTER 8

Effect of Stereotaxic Surgery on Oculogyric Crises

"Before making the attempt to point out the nature and cause of this disease, it is necessary to plead, that it is made under very unfavourable circumstances. Unaided by previous enquiries immediately directed to this disease, and not having had the advantage, in a single case, of that light which anatomical examination yields, opinions and not facts can only be offered. Conjecture, founded on analogy, and an attentive consideration of the peculiar symptoms of the disease, have been the only guides that could be obtained for this research, the result of which is, as it ought to be, offered with hesitation."

.... James Parkinson (1817)

(a) Introduction

The author feels very much like Parkinson when offering his analysis of the results stereotaxic surgery for oculogyric crises. Unlike cases of tremor and rigidity, the numbers available are small and no statistical proof can be given. No definite co-ordinate analysis can be done since the symptom is not modified during surgery whose results are known only much later. Yet there appear some very strong suggestions as to the nature of the successful lesions. It has been worth while including them in the present thesis, if only for the reason that this is the first time any such analysis has been made.

Since the recognition of oculogyric crises as a feature of postencephlitic Parkinsonian syndrome, many reports of this interesting condition have appeared in the literature. Among the more detailed ones are those of Jeliffe (1929), Hall (1931), Wilson (1954) and Onuaguluchi (1961). The clinical features of these attacks were very well documented

by these authors and little further needs to be added to their descriptions.

Diagnostic of the postencephalitic type of the disease, these attacks usually consist of turning upwards of the eyes over which the patient has no control. The duration, frequency and severity of these attacks may vary widely from one patient to another. The attacks are most distressing to the patient and can be dangerous to him if he is alone at the commencement of an attack in a situation when he needs to see clearly. The direction of movement of the eyeballs, associated physical and mental state as well as the methods of termination vary. In general there is no effective way of preventing an attack or shortening its duration after onset. Sporadic reports in the literature have continued to appear of successful methods of aborting a few attacks in individual instances.

Many theories have been offered to explain the pathogenesis of oculogyric crises. The compulsive obsessive theory of Jelifie (1929) was at one time favoured. According to Jeliffe, mental or emotional trauma was necessary to the initial onset of oculogyric attacks. The features of the crises were regarded by him as ritualistic and secondary to a repressed feeling usually of guilt.

Hall (1931) in his Schorstein lecture ascribed the crises to a mechanism similar to or identical with the relaxation of sleep. He collected 206 observations on the position of the eyes during sleep. He came to the conclusion that there was no essential difference between

the position of the eyes during sleep and in oculogyric attacks.

Onuaguluchi (1961) in a detailed analysis of 67 cases from Glasgow was of the opinion that several factors were concerned in the production of these attacks. He favoured the theory that an interpaly of cerebral cortical damage producing impaired cortical inhibition, brain stem lesion with weak extrinsic ocular muscles and excessive wax production or other sources of vestibular stimulation was responsible for these crises. He felt that these factors resulted in stimulation of the reticular formation of the brain stem and diencephalon and this caused the attack which he described as an abnormal vestibulo-ocular reflex with tonic upward glance.

Denny-Brown (1962) identified oculogyric crises with the phenomenon of tonic innervation of head and eyes.

Whatever may be the exact pathogenesis of the syndrome and the theoretical controversies about it, it has been universally accepted in the past that there was no effective treatment.

Lysivane and a number of other anti-Parkinsonism drugs were reported to be beneficial but no statistical study of a series of cases has ever been reported where any drug either prevented or shortened an attack or even reduced the frequency, duration or severity of attacks.

(b) Reports of Successful Surgical Treatment

The first and only successful case reported prior to 1963 of surgical treatment of oculogyric crises was by

Klemme (1941). Through a frontal craniotomy he carried out faradic stimulation of the motor cortex at the junction of the first and second frontal convolutions to reproduce a typical attack. Excision of the premotor cortex only was then carried out. The patient who suffered from severe oculogyric attacks once in three days before operation had none after operation on a one year follow-up.

Wycis and Spiegel (1958) operated three times on a Parkinsonian patient with oculogyric crises. Stimulation and partial elimination of periaqueductal grey matter and mesencephalic tegmentum were carried out bilaterally on the presumption that interference with pathways serving voluntary vertical eye movements may be of benefit. The crises were temporarily abolished but recurred after a few weeks. They noted that the paresis of voluntary upward movement and abolition of oculogyric spasms lasted for the same duration. They suggested that the mechanism innervating the oculogyric crises used the same pathways as the corticofugal impulses for upward movement of the eyeballs.

Matiar (1955) considered the crises to be a general impairment of the vegetative system. He was of the view that the encephalitic process produced a partial disturbance in the diencephalic part of the sympathetic system with a resultant increased excitatory state of the unaffected part of the sympathetic system. He blocked the stellate ganglion with local anaesthesia during an attack and abolished the attack temporarily in the corresponding eye with the simultaneous production of a Horner's syndrome.

Hassler and his colleagues (1960) considered that oculogyric crises were influenced by coagulations of the rostral portion of inner and outer pallidum.

Bertrand (1960) reported improvement in three cases out of five with oculogyric crises as a result of stereotaxic surgery.

Gillingham and Kalyanaraman (1963) reported abolition of oculogyric crises with bilateral stereotaxic lesions. This was the first report of successful treatment of this syndrome in a series of cases.

(c) Present Study

In the present series, 30 patients out of 301 suffered from oculogyric crises. Wilson (1954) reported a varying incidence from 15-20% in different series. The cases in the present group were selected in the sense that patients whose main disability was oculogyric crises and who had only minimal tremor and rigidity were not accepted for stereotaxic surgery.

Longterm follow-up of the results of surgery in these cases revealed the following data:

Group	Effect on oculogyric crises	Patients with unilateral operation	Patients with bilateral operations
(a)	Abolished	5	6
(b)	Improved	5	3
(c)	Unaffected	3	1
(d)	Attacks spontaneously ceased a few months before first operation	2	1
(e)	Attacks started a few months after last operation	1	1
(f)	No definite information available	2	-
	TOTAL	18	12

Of the six bilateral cases in whom the oculogyric spasms were abolished, one had total abolition after operation on the first side and two had considerable improvement after operation on the first side and total abolition after operation on the second side.

The length of follow-up period for groups (a) and (b) is given below.

less than one year	3 cases (1 died)
1 - 2 years	4 cases (2 died)
2 - 3 years	4 cases
3 - 4 years	5 cases
4 - 5 years	3 cases

In no instance was there recurrence of attacks after abolition for more than a few weeks and it is likely that the abolition and improvement are permanent effects in most cases.

The eight patients, who showed improvement but not total abolition, differed in the nature of improvement. One reported improvement in the frequency, duration and severity of attacks. One reported improvement in the frequency and duration of attacks only but not in severity. Two reported improvement in severity only but not in the frequency or duration. Three patients reported improvement in frequency only while the duration and severity remained unchanged. Detailed information about one was not available.

One of the two patients who had improvement after the first operation and abolition after the second, obtained such improvement in frequency, duration and severity.

Information about the other was not available.

When the attacks were abolished or lessened in severity associated obsessional thinking, lack of clear thinking or confusion were also correspondingly benefited.

Of the 30 patients, five were dead at the time of analysis. Only in two, autopsy reports were available. One, who showed clinical improvement in oculogyric crises after surgery, had thalamic and pallidal lesions in one hemisphere and pallidal and capsular lesions in the other hemisphere. The second patient reported spontaneous regression of attacks a few months before operation. He had thalamic, pallidal and capsular lesions on one hemisphere. Six weeks after operation he died of coronary occlusion but had remained free of oculogyric crises till his death.

On charting the lesions on the stereotaxic atlas of Schaltenbrand and Bailey, for the groups (a), (b) and (c), it was seen that only five patients did not show some involvement of the internal capsule by the stereotaxic lesion. There was however no clear distinction on atlas charting between lesions which benefited and lesions which did not benefit oculogyric crises, with reference to capsular involvement as shown on charting. Likewise there was no statistically significant difference in the laterality of the electrode tracks between the two groups.

(d) Discussion

A comparison of the sites of successful lesions, both temporary and permanent, by Klemme (1941), Wycis and Spiegel

(1958), Matiar (1955) and the present study suggests that all were dealing with a corticofugal pathway from the premotor cortex passing through the posterior limb of the internal capsule, the periaqueductal grey matter and the stellate ganglion.

In this connection, a paper by Minckler, Klemme and Minckler (1944) was found to be very interesting. These authors studied sections from the brain of a patient subjected to premotor cortical excision for tremor. They described on their degeneration studies a premotor bundle which started from the area of excision and swept deeply into white matter aggregating in a subcortical position and in descending split over the superior aspect of the lateral ventricle. One part entered the corpus callosum and continued to the opposite premotor cortex. The other part took up a well demonstrated position in the posterior limb of the internal capsule usually ascribed to the pyramidal tract. The degenerating fibres were traced downwards to terminate in the midbrain and spinal cord. They felt that the premotor bundle mediated tremor and was distinct from the pyramidal tract.

It is noteworthy that many authors in their experience of several thousands of cases treated by stereotaxic surgery have not noted total abolition of oculogyric crises although in a few cases they observed improvement. The lesions of most authors were in the pallidum or thalamus and only very rarely encroached to a significant degree on the internal capsule on both sides in the same patient.

It is significant that Guiot (1963) who used a posterior approach but avoided making lesions in the capsule after definition of its borders by depth electrical recording, also considered that oculogyric crises were not benefited by stereotaxic surgery.

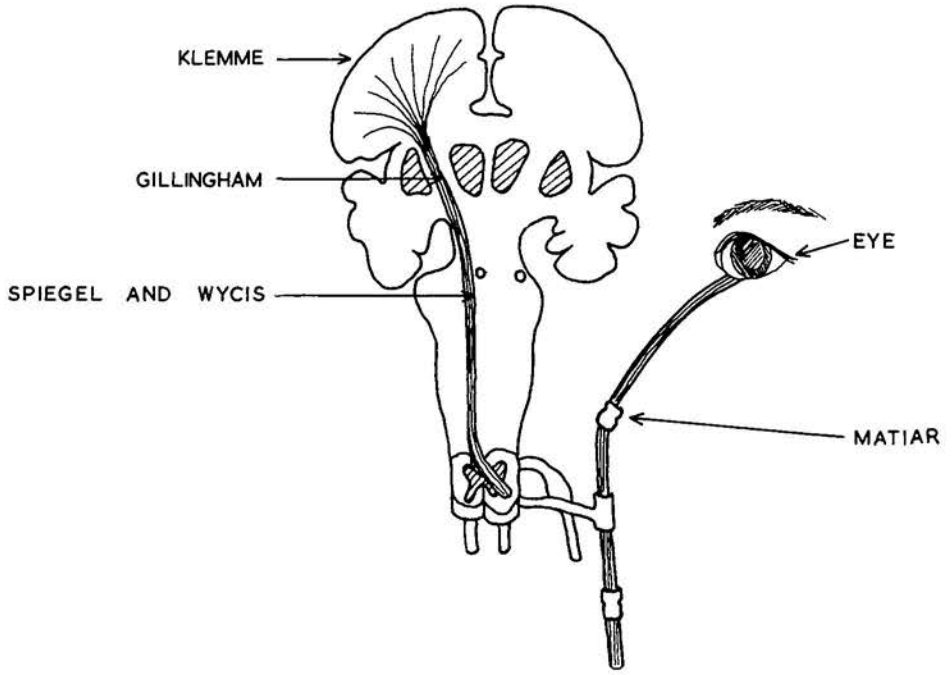
Consideration of the reports of all these authors as well as the present material leads to the conclusion that oculogyric crises can be abolished by bilateral transcapsular lesions in the anterior part of the posterior limb of the internal capsule. This site is well medial to the position of the pyramidal tract as shown by the present study in earlier chapters. Is it likely that if the borders of the internal capsule are accurately defined by depth electrical recording and the lesions are placed across the whole breadth of the internal capsule on both sides along the Gillingham electrode track, we can obtain a 'cure' rate for oculogyric crises as high as we can offer for tremor or rigidity in Parkinsonism by present stereotaxic methods? Only the future will give the answer when a carefully controlled series of cases are operated with depth electrical recording.

(e) Summary

The previous literature on the syndrome of oculogyric crises and its surgical treatment are reviewed.

A series of cases is presented where permanent abolition of oculogyric crises was achieved in a large percentage by the Gillingham method of stereotaxy.

The opinion is expressed that bilateral transcapsular stereotaxic lesions should abolish this symptom in the majority of cases.



LEVEL OF SURGICAL LESION IN REPORTED CASES OF SUCCESSFUL TREATMENT OF OCULOGYRIC CRISES.

AUTHORS	NUMBER OF CASES	LEVEL OF INTERRUPTION	PROCEDURE	UNILATERAL OR BILATERAL LESION	EFFECT
KLEMME (1941)	1	PREMOTOR	EXCISION	UNILATERAL	? PERMANENT
GILLINGHAM (1963)	6	POSTERIOR LIMB OF INTERNAL CAPSULE	STEREOTAXIC LESION	BILATERAL	? PERMANENT
SPIEGEL AND WYCIS (1958)	1	PERIAQUEDUCTAL GREY MATTER	STEREOTAXIC LESION	BILATERAL	TEMPORARY
MATIAR (1955)	2	STELLATE GANGLION	BLOCK BY LOCAL ANAESTHETIC	UNILATERAL	TEMPORARY

Above: Levels of successful lesions for oculogyric crises
Below: Details of previously reported cases of successful surgical treatment for oculogyric crises.

REFERENCES

1. Bertrand, C. (1960). In Elliott, H. and Nasholu, B. Ed. The Shaking Palsy, A Symposium. McGill University Press. Montreal, 115.
2. Denny-Brown, D. (1962). The Basal Ganglia. Oxford University Press, London. 100.
3. Gillingham, F.J. and Kalyanaraman, S. (1963). Excerpta Medica International Congress Series No. 60. Second European Congress of Neurological Surgery. 115.
4. Guiot, G. (1963). Personal Communication.
5. Hall, A.J. (1931). Brit. med. J., 2, 833.
6. Hassler, R., Riechert, T., Mundinger, F., Umbach, W. and Ganglberger, J.A. (1960). Brain, 83, 2, 337.
7. Jeliffe, S.E. (1929). Arch. Neurol. Psychiat., Chicago, 21, 491.
8. Klemme, R.M. (1941). Am. J. Ophthal., 24, 1000.
9. Matiar, H. (1955). Acta neuroveg., 12, 389.
10. Minckler, J., Klemme, R.M. and Minckler, D. (1944). J. Comp. Neurol., 81, 259.
11. Onuaguluchi, G. (1961). Brain, 84, 395.
12. Parkinson, J. (1817). An essay on the shaking palsy. Sherwood, Neely and Jones, London. Reprinted in Critchley, M., James Parkinson, Macmillan and Co. London, 1955, 185.

13. Schaltenbrand, G. and Bailey, P. (1959). Introduction to stereotaxis with an atlas of the human brain. Thieme, Stuttgart, 2.

14. Wilson, S.A.K. (1954). Neurology. Edited by Bruce, A.N. Butterworth & Co., London, 2nd Edition Volume I. 142.

15. Wycis, H.T. and Spiegel, E.A. (1958). Confin. Neurol., 18, 385.

CHAPTER 9

BILATERAL STEREOTAXIC LESIONS

"Nous estimons selon notre expérience qu'une opération bilatérale ne devrait être tentée que chez les malades gravement atteints et nous n'avons d'autre alternative que de mettre en garde les patients et leur famille du risque d'aphonie encouru."

.... Krayenbuhl et al (1963)

"There is no more common error than to assume that, because prolonged and accurate mathematical calculations have been made, the application of the result to some fact of nature is absolutely certain."

.... Whitehead

(a) Introduction

Since the establishment of stereotaxic surgery as a therapeutic procedure of value in the management of Parkinsonism and the dyskinesias, most centres specialising in this operation have carried it out bilaterally in a proportion of their patients. Bilateral stereotaxic surgery, however, has been associated with a certain amount of hazard somewhat greater than in the case of unilateral operations. There has been a general reluctance to subject these patients to operative procedures on both hemispheres and the criteria for selection for surgery have been more strictly applied before the second operation.

In contrast to the very large number of papers dealing with unilateral stereotaxic operations and their results, published reports analysing the problems of bilateral operations have been few.

Wycis and Spiegel (1958) reported 9 cases of bilateral

pallidoansotomy. Broager and Nørholm (1961) referred to 37 cases treated bilaterally in a series of a hundred consecutive patients. Krayenbuhl, Wyss and Yasargil (1961) published a detailed analysis of 51 bilateral stereotaxic procedures. Krayenbuhl, Siegfried and Yasargil (1963) subsequently published a longterm follow-up of these patients. Cooper and Riklan (1962) referred briefly to 200 cases bilaterally treated. Bertrand and Martinez (1962) considered 28 patients who underwent bilateral operations. Gros and his colleagues (1963) reported on a series of 41 cases. Markham and Rand (1963) discussed 35 patients subjected to bilateral procedures. Gillingham and Kalyanaraman (1963) analysed the results of 64 bilateral operations.

(b) Analysis of Eighty-one Cases

The present study is concerned with 81 patients, in a group of 324, who have been treated bilaterally. Seventy-three of these patients had Parkinsonism and 8 suffered from dyskinesias. Their analysis by age at the time of the second hemisphere operation, sex and handedness is given below.

Parkinsonism - Age incidence

Age group	No. of cases
30 - 39	4
40 - 49	19
50 - 59	33
60 - 69	17

Dyskinesias - Age incidence

Age group	No. of cases
0 - 9	1
10 - 19	2
20 - 29	2
30 - 39	2
40 - 49	1

Sex Incidence

Disease	Male	Female
Parkinsonism	43	30
Dyskinesias	4	4

Handedness

Disease	Righthanded	Lefthanded
Parkinsonism	68	5
Dyskinesias	7	1

More than half of the total number with Parkinsonism required constant help for their daily personal routine before operation. Five of these patients were ambulant for only part of the day and required part time nursing care. Only three were doing their full work at the time of the first operation.

All patients with Parkinsonism suffered from rigidity to a greater or lesser degree. All had tremor except one. In each case the symptoms were bilateral. Only four were free from additional disabilities like oculogyric crises (11 cases), festination (28 cases), speech difficulties (63 cases), excessive salivation (39 cases), pain (22 cases) or autonomic phenomena.

The sites of all lesions were assessed by post-operative charting on the atlas of Schaltenbrand and Bailey (1959). The sites of these lesions were further confirmed by operative biopsies in 13 cases, depth electrical recording in 4 cases and autopsy studies in three. Seventy-six of these patients had at least some involvement of the posterior limb of the internal capsule and fourteen had planned bilateral transcapsular lesions.

The incidence of "mirror lesions" were studied by superimposing the cellophane charts for the two hemispheres in 77 cases where both sides were operated in this department.

The details are given below.

Percentage Overlap between the two hemisphere lesions as seen for the smaller lesion.	NUMBER OF CASES	
	Difference between lateral distances of tracks on the two sides less than 2 mm.	Difference between lateral distances of tracks on the two sides more than 2 mm.
0	12	2
25	11	9
50	19	8
75	8 *	2
100	5 *	1

Some of the best results were obtained in the thirteen cases marked by an asterisk. No one of this group of 13 developed any major complications and all of them were good results from the point of view of abolition of tremor and rigidity. The present study would therefore seem to indicate that there was no greater post-operative morbidity

in making the so-called mirror lesions although other authors, notably, Hassler and his colleagues (1957, 1960) caution against this.

The time interval between operations on the two sides varied widely in the present series, as shown below.

Time interval	No. of cases
30 minutes	1
2 - 4 weeks	7
5 - 9 weeks	5
3 - 6 months	8
6 - 12 months	21
1 - 2 years	24
2 - 3 years	9
3 - 4 years	3
4 - 5 years	3

Speed of recovery from the first operation as well as the severity and progress of the disease on the second side were the main deciding factors for the time interval. Spiegel and Wycis (1962) reported one case where bilateral operations were done on the same day without complications. Krayenbuhl and Yasargil (1962) reported a similar performance. Postoperatively their patient suffered from mental confusion, akinesia and aphonia. One patient in the present series, who was operated bilaterally at the same time, had temporary reduction of intellectual performance particularly with regard to recent memory.

(c) Relief of Symptoms in Parkinsonism cases

Rigidity was very much benefited. In 29 patients it was abolished in all four limbs and in 26 it was very

much improved. Fifteen patients, however, had only moderate improvement in rigidity and in 3, it was unaffected or only slightly reduced. Pain associated with rigidity was almost always relieved by surgery.

Tremor was equally amenable to surgery. It was abolished in all four limbs in 22 cases and very much improved in 34. Moderate improvement only was seen in 15 patients and only slight improvement was obvious in 2 cases.

Cillingham (1961) considered a double ipsilateral lesion, in the oral ventral nucleus of the thalamus and the inner segment of the globus pallidus, both encroaching on the corresponding adjacent part of the internal capsule, to be the best lesion in cases of Parkinsonism and especially so for bilateral cases with generalised tremor and rigidity. Cassinari and his colleagues (1963) reported on 26 cases of simultaneous thalamopallidolysis and considered these to be superior to thalamic or pallidal lesions alone in controlling tremor and rigidity.

The effect of bilateral surgery on this symptom was considered in detail in a previous chapter.

Festination was occasionally improved and rarely eliminated. Salivation and other autonomic manifestations were sometimes helped and they were certainly more easily managed by medical therapy, after surgery, than before. Almost always patients needed less medication after operation. It was dangerous, however, to stop or greatly reduce drugs suddenly after operation. Two patients were thus precipitated into an acute Parkinsonistic crisis and needed hospitalisation to get them over it.

Within broad limits, the greater the intellectual and emotional reserve of the patient before surgery, the fuller was the benefit obtained by surgical intervention. Thus the presence of poor initiative, confusion and intellectual deterioration formed a serious contraindication to surgical treatment and especially to bilateral surgery if it was markedly increased after the first procedure. An abnormal electroencephalogram was often helpful as confirmatory evidence against surgery in such cases.

Emphasis was placed throughout on conservative lesions in treating the second side. It was felt that further small lesions could always be added with benefit. In most cases the midline screws were left in position after operation. In many cases the opaque marker left at the pallidal target was seen not to be shifted from that site on taking X-rays before a subsequent operation. These factors made the second or any subsequent operation much easier and shorter than the first procedure. The number of operations done on the 81 patients is shown below.

Total number of operations		Number of patients	
Left hemisphere	Right hemisphere	Parkinsonism	Dyskinesias
1	1	64	5
1	2	3	1
2	1	5	0
2	2	0	1
3	1	0	1
3	2	1	0

While 8 patients with Parkinsonism required one additional lesion after bilateral operation, only one patient needed more than one additional lesion. This was a young woman with advanced disease and she was back to full time activity as a housewife, shortly after her last operation.

(d) Mortality and Morbidity

There was no immediate postoperative mortality. Late postoperative mortality (between three months and a year after the second operation) occurred in four in this series. In two of these the operative result was excellent and death was due to intercurrent chest infection and coronary attack respectively. In one, death was indirectly attributable to progress of disease. In the last (one of the early cases) mortality was traceable to a poorly placed lesion with intraventricular hemorrhage.

Incidence of major postoperative complications was rare. There were no cases of dense or prolonged hemiparesis or sensory changes, hemianopia, large intracranial hemorrhage or pulmonary embolism in this series. Minor hemorrhage at the site of the lesion (presumed by shift in the position of the opaque marker in the postoperative X-ray associated with extreme drowsiness) occurred in 3 cases. One (referred to earlier) died after a few months and the other two recovered fully after a few days.

(e) Postoperative Hemiballismus

This interesting complication appeared in three patients after their second hemisphere operation, an

incidence of 3.7% in contrast to its appearance in one patient after unilateral operation, an incidence of 0.3%. In all four it appeared within a few days after operation, was temporary and was associated with an excellent operative result on longterm followup.

This relatively greater incidence of hemiballismus after second hemisphere surgery was also reported by Dierssen, Gioino and Cooper (1961). Their figures were 4% and 10% for the unilateral and bilateral operations respectively. Hughes (1963) had one case who developed hemiballismus some months after operation while in another case it appeared in the immediate postoperative period. He considered that a combination of factors or lesions was necessary for the clinical appearance of hemiballismus. The initial factors which are not associated with clinical hemiballismus and the last factor which precipitates it may be supplied by progress of disease or the stereotaxic lesion, alone or in combination. Such a theory by Hughes would explain (1) the incidence of hemiballismus without surgery, (2) its appearance immediately after surgery, (3) its appearance some time after surgery with progress of disease and (4) its greater incidence after bilateral surgery than after unilateral surgery.

Charting of the lesions on the stereotaxic atlas of Schaltenbrand and Bailey (1959) did not reveal any involvement of the subthalamic nucleus in these cases. It is now generally recognised that hemiballismus is not always associated with lesions of this nucleus. (Martin 1957; Dierssen, Bergmann, Gioino and Cooper 1961.)

(f) Postoperative Dysarthria and Reduction of Voice Volume

The main risks of operation on the second hemisphere were its effects on speech. Krayenbuhl, Wyss and Yasargil (1961) reported deterioration of speech in 7 out of 23 bilateral thalamotomies and in 17 out of 28 cases with pallidal lesion on one side and thalamic on the other. Krayenbuhl, Siegfried and Yasargil (1963) on a longterm followup of bilateral cases reported aggravation of speech difficulties in 60% of cases with postoperative aphony in 13%. Meyers (1963) found reduction in voice volume and central dysarthria in two-thirds of patients with bilateral lesions in Forel's tegmental field. Cooper (1961) reported incidence of temporary and permanent speech difficulties after unilateral surgery as 1% and 10% respectively. In bilateral surgery these figures increased to 6% and 18%. Approximately one fifth of his patients had dysphasia which was not included in the figures of the other authors quoted above. On the other hand, Gros and his colleagues (1963) and Wycis (1963) did not feel that speech disturbances occurred to a greater degree after second hemisphere surgery. The numbers of cases studied by them, however, were relatively small.

In conformity with the findings of many authors it was seen in the present series that speech was improved especially if the preoperative deficit was mainly due to rigidity of chest, jaw and tongue. However, further reduction of voice volume occurred in patients in whom it was already poor. Of the 73 patients with Parkinsonism treated in this series, this complication occurred in 27

patients. Of these, 21 had some reduction of voice volume before the second operation, usually following the first operation. Reduction of voice volume after the first operation therefore would be a relative contraindication for the second.

A similar situation applied to the presence of slurring. Of 25 patients who had severe and permanent slurring after the second operation, 13 had some degree of slurring before or after the first operation.

Most of these lesions involved both the capsule and the thalamus and it was difficult to determine whether either or both were involved in the production of these complications. Krayenbuhl and his colleagues (1961) implicated the thalamus and pallidum in similar situations. Guiot (1961) and Penfield (1963) implicated the thalamus. Bertrand and Martinez (1962) implicated the capsule. A study of the lesions created after depth electrical recording would provide the final answer to this problem.

(g) Psychological and Intellectual Deterioration

Krayenbuhl and his colleagues (1961) noted psychomotor disturbances in 8 out of 23 bilateral thalamic lesions and 17 out of 28 cases with thalamotomy on one side and pallidotomy on the other. This high incidence of psychological and intellectual complications in sharp contrast to unilateral operations was confirmed by them on longterm followup although some of the disturbances proved to be temporary.

In the present series, gross preoperative psychological and intellectual deficits contraindicated second hemisphere surgery. Nevertheless, these complications tended to occur in a small proportion of patients considered to be

within normal range after the first operation or only slightly deteriorated. Precipitation of mental depression associated with some return of tremor and festination was noted in 9 cases after the second operation. In six of these, the symptoms responded very well to Imipramine (Tofranil) or Amitriptyline (Tryptizol) which restored the patient to his satisfactory preoperative state.

Intellectual or emotional deterioration was noted in six cases. Four of these were affected by such deterioration moderately severely preoperatively (early cases in the series). Such deterioration was aggravated after surgery on the second side. The other two, however, had no such preoperative deficit. In one of these, the lesions were too large. In the other, no definite precipitating factor could be found.

Spiegel and Wycis (1962) suggested that bilateral lesions affecting the nucleus ventralis anterior also interrupted fiber systems connecting the dorsomedial nucleus with the frontal lobe, since these fibers passed through the former nucleus. They were of the opinion that a psychis syndrome similar to that following bilateral thalamofrontal interruption may then ensue. No such complication was encountered in the present series, probably because the lesions were not sufficiently large or medial to affect these fiber systems.

A point of particular interest and importance in this series was that the patients continued to improve for a long period after their second operation, sometimes for more

than a year. Thus, longterm followup studies were much more encouraging than short term assessments when a slight reduction in overall intellectual performance and initiative tended to cloud the early result and suggest a poor prognosis.

(h) Dyskinesias

The other dyskinesias formed a small group and were concerned with the treatment of spasmodic torticollis, Wilson's disease, idiopathic intention tremor, familial tremor, choreoathetosis (2 cases) and torsion dystonia (2 cases). The case of Wilson's disease was extremely ill at the time of operation and responded poorly to it. He eventually died some months later of bronchopneumonia. Spasmodic torticollis was difficult to correct even with bilateral lesions. The others responded satisfactorily, the best results being achieved in cases of intention tremor (familial and idiopathic) in whom the involuntary movements were totally abolished enabling them to resume full and normal activity.

(i) Longterm assessment

In the 73 cases of Parkinsonism operated bilaterally, overall assessments of results could be classified as below.

Grade	No. of cases
1. Cured	0
2. Satisfactory reduction of symptoms without complications	41
(a) Total abolition of tremor and rigidity	4 cases
(b) Satisfactory reduction of tremor and rigidity	37 cases

3. Satisfactory reduction of symptoms with complications 27

(a) Total abolition of tremor and rigidity 8 cases

(b) Satisfactory reduction of tremor and rigidity 19 cases

4. Slight reduction of symptoms 5

TOTAL 73

On a long term followup, the patients (and relatives) classified their own condition as shown below. (66 cases with a minimum period of followup of 5 months)

Classification of Grades:

A - Full working capacity

B - Reduced working capacity

C - Protected circumstances with minor home responsibilities

D - Ambulant but requiring some nursing care

E - Bedridden invalid requiring constant nursing care

Preoperative		Postoperative improved		Postoperative worsened	
Grade	No. of cases	To higher grade	In same grade	In same grade	To lower grade
A	2	0	2	0	0
B	27	5	18	2	2
C	21	6	11 *	3	1
D	16	9	4 *	2	1
E	0	0	0	0	0
TOTAL	66	20	35	7	4

* include one patient who had two coronary attacks and three who sustained fracture of the neck of femur some

weeks after operation. The patient's grading includes disability due to these complications.

(j) Summary

Eightyone cases treated by bilateral stereotaxic lesions, most of them capsular, were analysed. Mirror lesions were shown to be harmless.

Relief of tremor and rigidity was achieved in a large percentage of cases. Other symptoms were benefited to a varying degree.

Postoperative morbidity was quite small. Speech disturbances, intellectual and emotional deterioration and hemiballismus appeared to be complications with a greater incidence after bilateral surgery than after unilateral surgery. Yet they occurred with less frequency than in other published series.

Longterm assessment confirmed the persistence of immediate postoperative improvement and in fact showed in many cases further improvement.

REFERENCES

1. Andy, O.J. (1963). In discussion in Spiegel, E.A., Wycis, H.T., Szekely, E.G., Adams, J. and Baird III, H.W. J. Neurosurg., 20, 871.
2. Bertrand, C. and Martinez, N. (1962). Confin. Neurol., 22, 279.
3. Broager, B. and Norhølm, T. (1961). J. Neurol. Neurosurg. Psychiat., 24, 297.
4. Cassinari, V., Pagni, C.A., Cabrini, G.P. and Pauli, P. (1963). Excerpta Medica International Congress Series No. 60. Second European Congress of Neurological Surgery. 118.
5. Cooper, I.S. (1961). Parkinsonism. Its Medical and Surgical Therapy. C.C. Thomas, Springfield, Illinois. 118.
6. Cooper, I.S. and Riklan, M. (1962). St. Barnabas Hospital Medical Bulletin, 1, 3, 17.
7. Dierssen, G., Gioino, G. and Cooper, I.S. (1961). Neurology, II, 894.
8. Dierssen, G., Bergmann, L.L., Gioino, G. and Cooper, I.S. (1961). Arch. Neurol., 5, 627.
9. Gillingham, F.J. (1961). Personal Communication.
10. Gillingham, F.J. and Kalyanaraman, S. (1963). Excerpta Medica International Congress Series No. 60. Second European Congress of Neurological Surgery. 115.

11.	Gros, C., Serrats, F., Adib-Yazdi and Parker, A. (1963).	Excerpta Medica International Congress Series No. 60. 116.
12.	Guiot, G. (1961).	Rev. Canad. Biol., 20, 395.
13.	Hassler, R. (1957).	Proc. 2nd Int. Congr. Neuropath., London. 1955. Part IV. 637. Excerpta Medica. Amsterdam.
14.	Hassler, R., Riechert, T., Mundinger, F., Umbach, W. and Ganglberger, J.A. (1960).	Brain, 83, 337.
15.	Hughes, B. (1963).	Personal Communication.
16.	Krayenbuhl, H., Wyss, O.A.M. and Yasargil, M.G. (1961).	J. Neurosurg., 18, 429.
17.	Krayenbuhl, H. and Yasargil, M.G. (1962).	Confin. Neurol., 22, 368.
18.	Krayenbuhl, H., Siegfried, J. and Yasargil, M.G. (1963).	Rev. Neurol., 108, 5, 485.
19.	Markham, C.H. and Rand, R.W. (1963).	Arch. Neurol., 8, 621.
20.	Martin, J.P. (1957).	Brain, 80, I, I.
21.	Meyers, R. (1963).	In discussion in Spiegel, E.A., Wycis, H.T., Szekely, E.G., Adams, J., Flanagan, M. and Baird III, H.W. J. Neurosurg., 20, 871.

22. Penfield, W. (1963). Quoted by Gillingham, F.J. Personal Communication.

23. Schaltenbrand, G. and Bailey, P. (1959). Introduction to Stereotaxis with an Atlas of the Human Brain. Thieme, Stuttgart, 2.

24. Spiegel, E.A. and Wycis, H.T. (196_). Stereoencephalotomy. Part II. Clinical and Physiological Applications. Grune and Stratton, New York. 349.

25. Whitehead, A.N. Quoted by Moroney, M.J. Facts from Figures. Penguin Books Ltd., Harmondsworth, Middlesex. 1956. 3rd edition. 271.

26. Wycis, H.T. (1963). In discussion in Spiegel, E.A., Wycis, H.T., Szekely, E.G., Adams, J., Flanagan, M. and Baird III, H.W. J. Neurosurg., 20, 871.

27. Wycis, H.T. and Spiegel, E.A. (1958). In Fields, W.S. Ed. Pathogenesis and Treatment of Parkinsonism. C.C. Thomas, Springfield, Illinois. 294.

PART IV

CONCLUSION

Chapter 10: Pure Capsular Lesions

CHAPTER 10

Pure Capsular Lesions

"In 1935, it appeared, we knew everything; today we know nothing. This is the consummation of a century of investigation upon the pyramidal tract as Bucy now presents it to us. Of course, it is incredible".

-- Walshe.

"If a man will begin with certainties he shall end in doubts; but if he will be content to begin with doubts he shall end in certainties".

-- Bacon.

(a) Introduction

The present study started with many doubts. No previous assertion was accepted just because it was printed in textbooks.

Using the Gillingham method of stereotaxy and analysing the results of 475 operations, several conclusions were reached on scientific and statistical grounds.

It was shown that the sites of lesions could avoid the sensory relay nucleus by eliciting evoked potentials during depth electrical recording. It was also shown that sensory complications could be avoided by using a high track to reach the targets.

The structure of the posterior limb of the internal capsule was shown to be different from the classically accepted view. It was demonstrated repeatedly that capsular lesions in the medial part of the posterior limb

of the internal capsule were situated sufficiently far away from the pyramidal tract to avoid any postoperative pyramidal deficit.

It was demonstrated that tremor could be abolished at different levels. Rigidity was considerably reduced by thalamic lesions but often required additional pallidal or capsular lesions for total abolition. It was shown that both rigidity and tremor could be favourably influenced by capsular lesions, probably by causing interruption of the ansa and fasciculus lenticularis.

Capsular lesions were seen to possess a special advantage as compared to thalmic or pallidal lesions. Only capsular lesions appeared to abolish or improve oculogyric attacks.

Bilateral capsular lesions were not associated with any major deficit. In fact the incidence of complications was shown to be lower than in bilateral lesions at other sites.

Capsular lesions have been carried out in the past by Guiot and his colleagues (1959), Gillingham (1961a) and Jinnai and his colleagues (1961). Only Gillingham carried out transcapsular lesions extending across the whole breadth of the internal capsule. Individual anatomical variations however made it difficult to place a lesion only in the capsule without encroachment on the adjacent thalamus or pallidum.

(b) Pure Capsular Lesions

The development of depth electrical recording with audiomonitoring made such an achievement possible for the first time in neurosurgery. At the suggestion of Professor Gillingham the present author operated on seven cases in whom the borders of the internal capsule were accurately defined and a stereotaxic coagulation lesion was placed entirely in the capsule. This was the logical next step in the evolution of stereotaxic surgical treatment at Edinburgh after the creation of transcapsular lesions by Professor Gillingham, the development of depth electrical recording and the findings of the present author elaborated in the previous chapters of this thesis. The details of the lesions, with their effects on the patients, in these seven instances are given below.

Case 1: Mrs. E.C., aged 57, had suffered from tremor in her right limbs for 22 years. Preoperatively she had advanced rigidity and marked tremor in both right limbs with minimal involvement of the left upper limb. There were no autonomic features or other widespread effects of the disease.

On 20th September 1963, a pure capsular lesion was made on the left side. The anterior end of the oral ventral nucleus was defined at 13.5 mm. behind the pallidal target. The anterior end of the reticular nucleus was defined at 11.5 mm. behind the pallidal target. The pallidocapsular junction was defined at 3.5 mm. behind the pallidal target. A single coagulation with the electrode

tip at 7 mm. behind the pallidal target was done up to calibration 3 on the diathermy machine. It was estimated that the lesion would extend from 5 mm. to 11 mm. behind the pallidal target. This was a pure capsular lesion but did not involve the whole breadth of the capsule or the reticular nucleus.

There was immediate and total abolition of tremor and rigidity. No pyramidal deficit was apparent at the conclusion of the operation, although there was minimal weakness of handgrip during the coagulation.

Forty-eight hours after operation, presumably as œdema extended around the coagulation lesion, she developed slight dysarthria, minimal weakness of the right handgrip and dragging of the right foot. She was incontinent for 36 hours after operation. All these features rapidly and fully resolved over the succeeding 48 hours. There was no evidence of dysphasia at any time.

When reviewed at the convalescent hospital ten days after operation there was slight recurrence of tremor in both right limbs under stress although it was still very much less than the amount of tremor she had preoperatively. Two months after operation she was back to full activity as a housewife. There was still minimal tremor under stress. Rigidity remained fully abolished and there were no signs of any pyramidal deficit.

Twenty weeks after operation she still maintained her improvement and the neurological status was unchanged as compared to the review two months after operation.

Subjectively she felt much more improved and was more active than she had been for many years.

Lateral distance of the track was 15.75 mm.

Case 2: Miss C.S. aged 58 had noticed leftsided tremor for three years associated with some stiffness. Preoperatively she had a moderate degree of tremor and rigidity in the left limbs with only a trace of tremor on the right. There were no autonomic features or other widespread effects of the disease.

On 25th September 1963, depth electrical recording with audiomonitoring was carried out. Thalamocapsular junction was defined at 12.5 mm. behind the pallidal target and the pallidocapsular junction was defined at 4.5 mm. behind the pallidal target. A single coagulation was done with the electrode tip at 7.5 mm. behind the pallidal target up to calibration 4 on the diathermy machine. It was estimated that the lesion would extend from 5 mm. to 11.5 mm. behind the pallidal target. This was a pure capsular lesion which was involving almost the entire breadth of the capsule but not all of it. The position of the reticular nucleus could not be separately defined in this case. Probably it was not involved in the lesion.

During coagulation or immediately after it, there was no evidence of pyramidal deficit. Within 24 hours she developed a minimal facial palsy which cleared rapidly over the next few days. There was slight recurrence of tremor in both left limbs a few days after operation. Reviewed two months after operation she showed no pyramidal deficit.

There was complete absence of rigidity in the left limbs with minimal tremor under stress.

Twenty weeks after operation she maintained her clinical improvement. Her neurological status was unchanged as compared to the review two months after operation. Subjectively however she felt much more improved and was more active than she had been for many years.

Lateral distance of the track was 17.1 mm.

Case 3: Miss J. McA., aged 38, had suffered tremor and stiffness for three years when she was first seen in the department in 1962. In July of that year a left hemisphere stereotaxic lesion was carried out in spite of a reduced voice volume. Tremor and rigidity were fully abolished in the right limbs following this procedure.

Symptoms on the left side continued to progress. She was readmitted for second (right) hemisphere stereotaxy which was done on 4th October, 1963. There were no autonomic features or other evidence of widespread effects of the disease. Psychometric and psychiatric assessment suggested somewhat abnormal personality features.

External medullary lamina, reticular nucleus, thalamocapsular junction and pallidocapsular junction were defined at 9.5 mm., 9 mm., 8.5 mm. and 2.5 mm. behind the pallidal target respectively. A lesion was made with the tip of the electrode at 5 mm. behind the pallidal target using a maximum calibration of 4 on the diathermy machine. It was estimated that the lesion would extend

from 2.5 mm. to 9 mm. behind the pallidal target. This was a pure capsular lesion but involvement of the reticular nucleus could not be ruled out.

During coagulation and immediately after it she did not show any involvement of the pyramidal tract. Voice volume was unaffected by surgery.

Twenty four hours after operation she developed a mild central facial palsy with slight falling of the outstretched arm. These features rapidly resolved over the next few days.

There was total abolition of tremor and rigidity immediately after the creation of the lesion. The effect was fully maintained for several weeks after operation.

Eighteen weeks after operation she complained of mild recurrence of tremor in the left lower limb under conditions of stress. Otherwise she maintained her improvement and showed no evidence of pyramidal or other complications.

Lateral distance of the track was 15.75 mm.

Case 4: Mrs. J.W., aged 56, had shown Parkinsonian symptoms since 1945. Bilateral advanced tremor, generalised rigidity, reduced voice volume, slurring of speech, akinesis, excessive sweating, ankle edema and festination were present.

Depth electrical recording with audiomonitoring was carried out on 8th November 1963. External medullary lamina, reticular nucleus, thalamocapsular junction and pallidocapsular junction were found to be at 16.75 mm.,

15.5 mm., 14.5 mm. and 2.5 mm. behind the pallidal target. Three coagulations were done with the electrode tip at 5 mm., 7.5 mm. and 10 mm. behind the pallidal target, using calibration 4 each time. It was estimated that the lesion would extend from 2.5 mm. to 14 mm. behind the pallidal target. This would be a pure capsular lesion without involvement of the reticular nucleus.

During coagulation or immediately after it, she did not show any facial palsy, weakness of handgrip or shoulder power or extensor plantar response. Both tremor and rigidity were fully abolished by the procedure.

In the early postoperative period, she showed minimal recurrence of tremor. Her main difficulties however were with regard to posture and gait. She showed a tendency to fall to the right. Her voice volume was reduced and slurring was increased. There was however no evidence of dysphasia. She had a long period of convalescence during which time she had intensive physiotherapy, occupational therapy and speech therapy.

At the time of her discharge home two months after operation her posture, speech and gait had improved. There was no evidence of pyramidal deficit. Tremor was very minimal under stress and rigidity remained fully abolished.

Lateral distance of the track was 15.75 mm.

Case 5: Mr. A.G.C., aged 40, had suffered for 18 years from Parkinsonian symptoms. Mild tremor and moderate rigidity was present in all the limbs, the right side being

more affected. There was reduction of voice volume with extrapyramidal dysarthria. There were no autonomic features or other evidence of widespread effects of the disease.

On 29th November 1963, depth electrical recording with audiomonitoring was carried out. Thalamocapsular and pallidocapsular junctions were defined at 13 and 1 mm. respectively behind the pallidal target. The reticular nucleus could not be defined with accuracy. Coagulations were done with the electrode tip at 3.5 mm. and 9 mm. behind the pallidal target using calibration 4. The lesion was expected to extend from 1 mm. to 13 mm. behind the pallidal target, a pure capsular lesion.

During coagulation or for 24 hours afterwards, the patient did not show any evidence of pyramidal deficit. Presumably as the œdema round the lesion extended, he developed a slight central facial palsy 48 hours after operation. This fully resolved in about 4 days. There was total abolition of tremor and rigidity which continued to be absent on a 7 week follow-up. Voice volume was somewhat improved and slurring was unaffected by the operation.

Lateral distance of the track was 15.3 mm.

Case 6: Mr. D.C., aged 53, first noticed dragging of his right foot three years prior to admission. At the time of operation his symptoms and signs were almost exclusively confined to the right limbs and face.

Rigidity was moderately severe but tremor was mild and confined to the upper limb. There were no autonomic features or other widespread effects of the disease.

On 20th December 1963, depth electrical recording revealed the thalamocapsular and pallidocapsular junctions at 11 and 3 mm. respectively behind the pallidal target. Coagulations were done with the electrode tip at 5.5 and 7 mm. behind the pallidal target for calibration 4. It was expected that the lesion would extend from 3 to 11 mm. behind the pallidal target, a pure capsular lesion. The reticular nucleus could not be defined separately in this case.

There was at no time any evidence of pyramidal deficit, during or after operation. Both rigidity and tremor were fully abolished but a trace of rigidity returned within a few days after operation.

At a second review, seven weeks after operation about 50% of preoperative rigidity was seen to have returned and he was again dragging his foot. It was not certain whether the lesion was too small or too far lateral.

Lateral distance of the track was 18 mm.

Case 7: Mrs. A.W. was 45 years old when she was operated on the right hemisphere in our department in January 1961 for predominantly left-sided Parkinsonian symptoms. She had noticed them for 5 years at that time. There was subtotal abolition of tremor and rigidity and this improvement was maintained over the next three years.

She was readmitted for operation on the second side. There was moderately severe tremor and rigidity in the right limbs with no evidence of involvement of the voice or autonomic functions.

On 10th January 1964, depth electrical recording with audiomonitoring was done. External medullary lamina, reticular nucleus, thalamocapsular junction and pallido-capsular junction were defined at 15 mm., 14 mm., 13.75 mm. and 3.25 mm. respectively behind the pallidal target. Coagulation was done up to calibration 4 with the needle tip at 5.75 mm. and 9.75 mm. behind the pallidal target. This was expected to produce a lesion from 3.25 mm. to 13.75 mm. behind the pallidal target, a pure capsular lesion probably without involvement of the reticular nucleus.

There was no pyramidal deficit during or after coagulation. Both tremor and rigidity were fully abolished immediately after the creation of the lesion. A slight degree of tremor under stress reappeared in the early postoperative period.

Lateral distance of the track was 19.35 mm.

A review of these seven cases of Parkinsonism treated by pure capsular lesions suggested that such lesions

(a) were followed by considerable reduction or abolition of tremor and rigidity

(b) were not always capable of causing total abolition of tremor or rigidity

(c) were fully free of pyramidal deficit in the form of even minimal facial palsy, weakness

of limbs or extensor plantar response, and

(d) were fully free of postoperative speech difficulties like dysarthria or reduction of voice volume.

It should be noted that these lesions were 5 mm. in diameter and ranged in length from 6 to 12 mm. All of them were smaller than the standard double ipsilateral thalamic and pallidal lesions or transcapsular lesions done by Gillingham and considerably smaller than the lesions of most stereotaxic surgeons.

(c) Conclusion

The structure of the posterior limb of the internal capsule was deduced by statistical studies and scattergrams in the previous chapters. These pure capsular lesions, entirely confined to the posterior limb of the internal capsule provided the most direct evidence of such a structure.

Will pure capsular lesions be accepted in future as the ideal lesion for Parkinsonism because they seem to interrupt the ansa and fasciculus lenticularis and abolish tremor and rigidity without complications of speech? Will they be definitely preferred to other lesions for second side operation and for cases with oculogyric crises? Will they compare favourably on long-term follow-up with the double ipsilateral (thalamocapsular and pallidocapsular leaving a free strip of capsule in between) advocated by Gillingham (1961b)? Will they be modified to include the

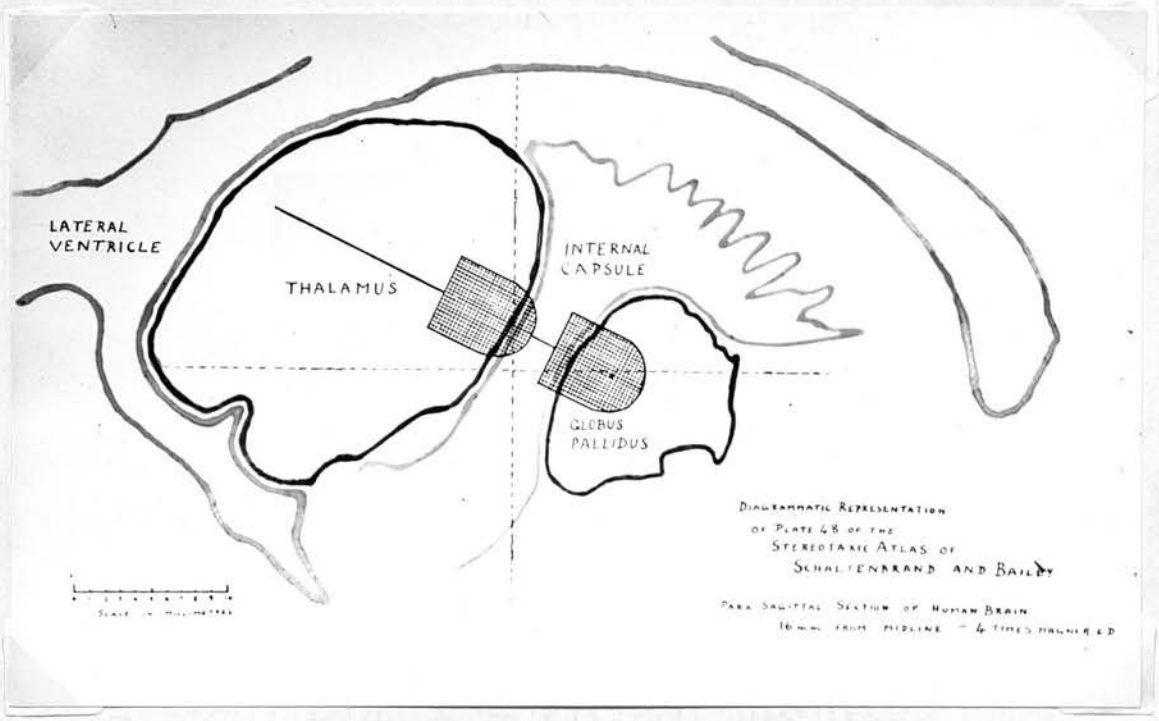
reticular nucleus in each case to produce total abolition of tremor as predicted by Aguinis (1963)? Will the discovery of the ultimate site of origin of abnormal "discharges" in Parkinsonism make capsular lesions obsolete?

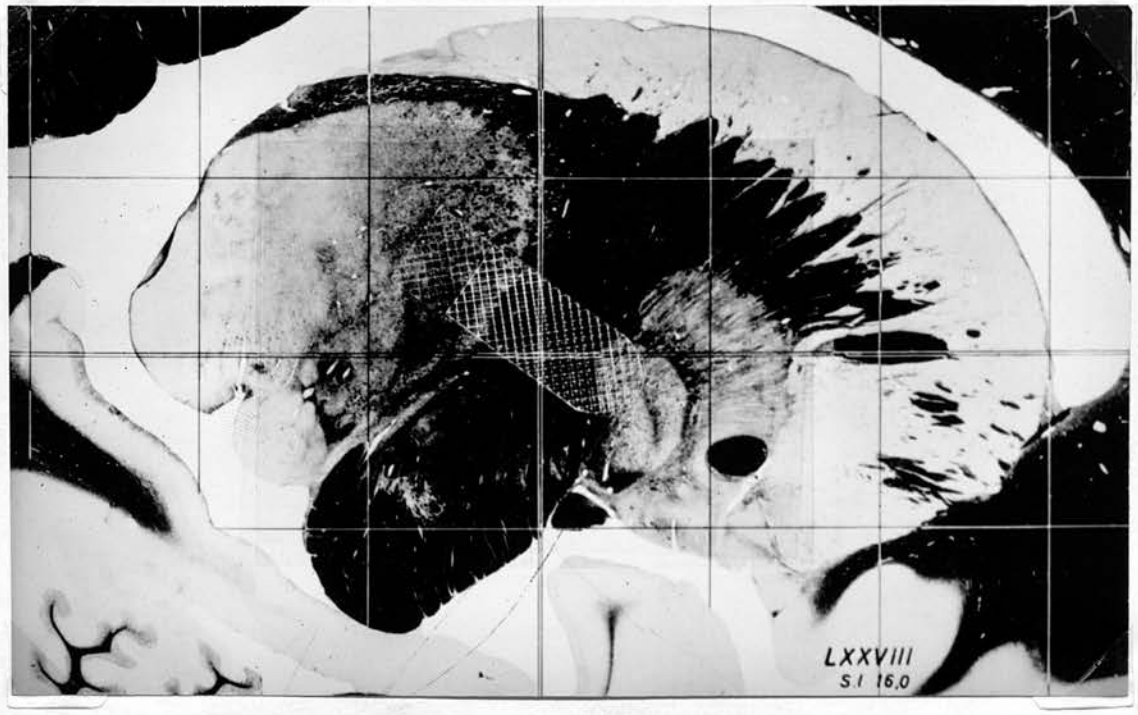
No one can answer these questions adequately now. Long years of painstaking and intelligent research will ultimately provide the answer to the riddle of Parkinsonism. But the new concept of the structure of the internal capsule might soon be accepted by all in the face of increasing evidence in its favour.

I cannot conclude more appropriately than by quoting James Parkinson (1817) from his celebrated 'Essay on the Shaking Palsy'.

"By these repeated observations, he hoped that he had been led to a probable conjecture..... that analogy had suggested such means as might be productive of relief, and perhaps even of cure..... He therefore considered it to be a duty to submit his opinions to the examination of others, even in their present state of immaturity and imperfection".

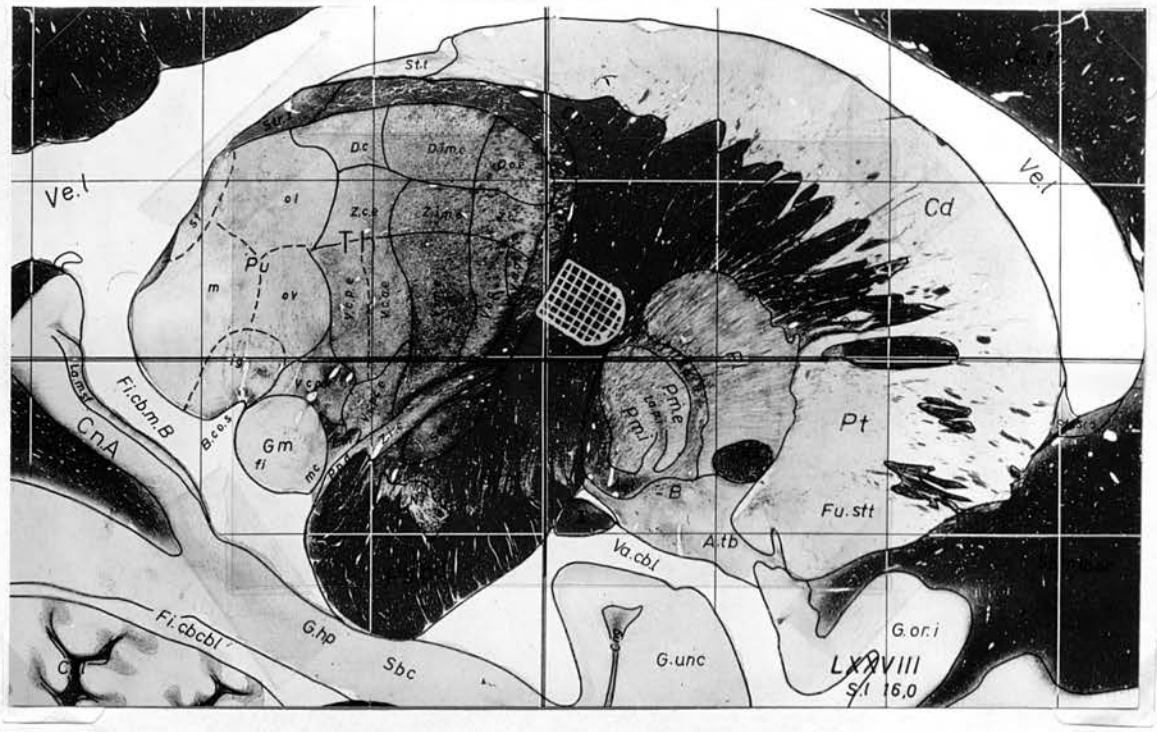
"Should the necessary information be thus obtained, the writer will repine at no censure which the precipitate publication of mere conjectural suggestions may incur; but shall think himself fully rewarded by having excited the attention of those, who may point out the most appropriate means of relieving a tedious and most distressing malady".



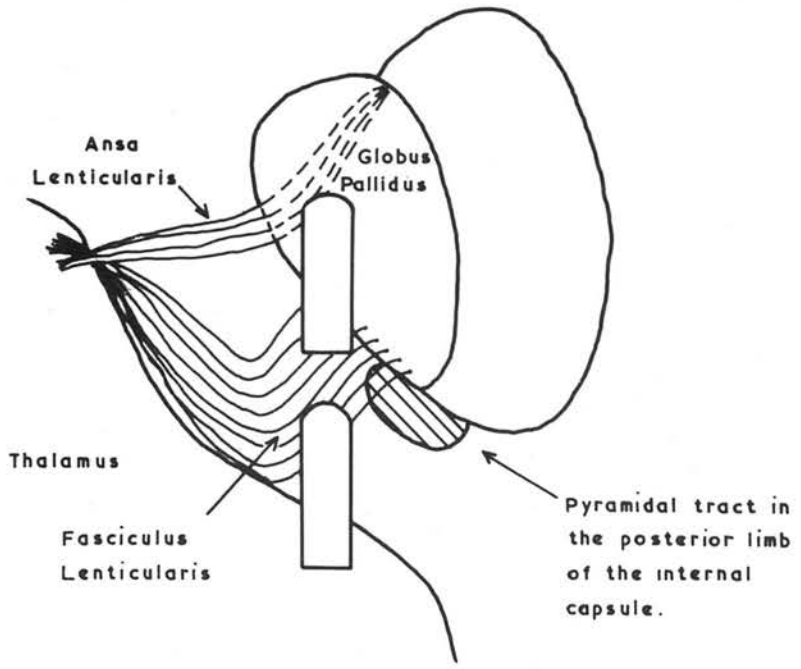


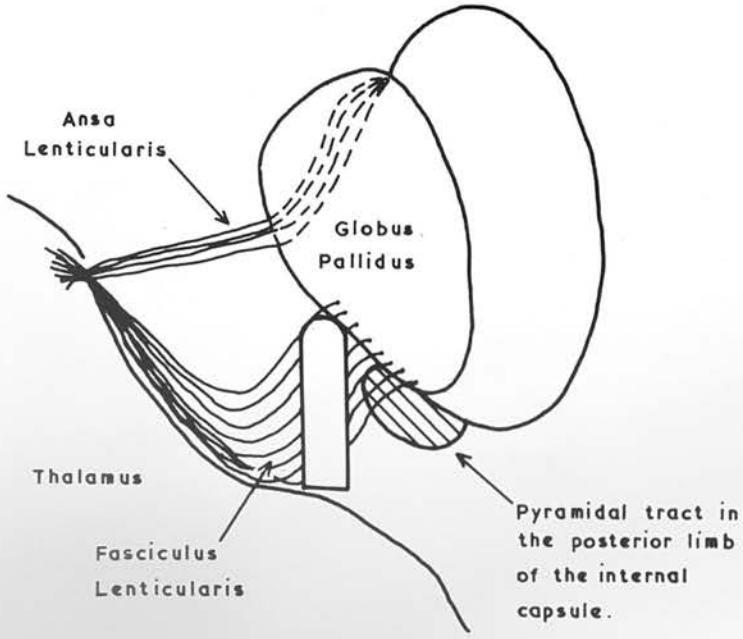
Above: The double ipsilateral lesion advocated by Professor Gillingham.

Below: The long transcapsular lesion first done by Professor Gillingham.



The pure capsular stereotaxic lesion carried out by the author. Diagrammatic representation on the stereotaxic atlas. Plate 48-S1.16.





Diagrams to explain how the double ipsilateral lesion and the transcapsular lesion are effective in abolishing symptoms.

REFERENCES

1. Aguinis, M. (1963). Acta Neurochir., II, I, 151.

2. Bacon, F. Quoted by Moroney, M.J. Facts from Figures. Penguin Books Ltd., Harmondsworth, Middlesex. 1956. 3rd edition. 96.

3. Guiot, G., Sachs, M., Hertzog, E., Brion, S., Rougerie, J., Dalloz, J.C. and Napoleone, F. (1959). Neurochirurgie, 5, I, 17.

4. Gillingham, F.J. (1961a). First International Symposium on Stereoencephalotomy. Philadelphia. Confin. Neurol., 1962, 22, 385.

5. Gillingham, F.J. (1961b). Personal Communication.

6. Jinnai, D., Nishimoto, A., Matsumoto, K., and Handa, S. (1961). Excerpta Medica International Congress Series No. 36. Second International Congress of Neurological Surgery. E 94.

7. Parkinson, J. (1817). An Essay on the Shaking Palsy. Sherwood, Neely and Jones. London. Preface.

8. Walshe, F.M.R. (1961). In Garland, H. Ed. Scientific Aspects of Neurology. E. & S. Livingstone. Edinburgh and London. 8.

APPENDIX A

Method of charting lesions on stereotaxic atlas

As soon as the surgical procedure was completed, a lateral and a PA X-ray were taken of the opaque steel marker left at the most anterior site of coagulation. This was done keeping the patient still prone so that the marker could not move backwards along the electrode track.

The following measurements were then made on these post-operative films.

(1) Distance of the marker behind the posterior border of the anterior commissure along the intercommissural line.

(2) Distance of the marker above or below the intercommissural line.

(3) Distance of the marker from the mid-sagittal plane of the brain determined by the ventricular system and the midline screws inserted at operation.

(4) Angle between the electrode track and the intercommissural line (the electrode track was marked by joining the central point of the posterior burrhole in the X-ray with the marker).

Taking an example; for Mr. P.C., a patient operated on the 17th of August 1962, the following were the relevant measurements:

(a) Intercommissural Distance 27.5 mm.

(b) Marker behind CA 3.0 mm.

(c) Marker below CA-CP line 1.0 mm.

(d) Marker lateral to midline 17.5 mm.

(e) Angle of track 26^{o}

Measurements (a) and (b) above were converted proportionately to suit the CA-CP length of the brain of the atlas of Schaltenbrand and Bailey from which the sagittal and horizontal microseries were prepared. This length was 23 mm. and therefore for a four times magnification for the microseries, the length would be 92 mm. So measurements (a) and (b) for the above case would be changed as follows:

(a) Intercommissural Distance 92 mm.

(b) Marker behind CA = $\frac{92 \times 3}{27.5}$ = 10.03 mm.

Measurements (c) and (d) were reduced 10% for X-ray magnification and then multiplied by 4 to suit the microseries as shown below:

(c) Marker below CA-CP line 3.6 mm.

(d) Marker lateral to midline 63.0 mm.

The angle was kept unchanged since magnification would not affect the angle between two lines.

On a millimeter graph paper, the intercommissural line, CA, CP and the midcommissural line were drawn so that CA-CP = 92 mm.

The marker (in this case at PTP or pallidal target point) was plotted 10 mm. behind and 3.6 mm. below CA. The electrode track was drawn backwards and upwards from the marker so that its angle of inclination with the CA-CP line would be 26^{o}.

It was assumed from autopsy studies that a single coagulation with the tip of the electrode at zero point up to a maximum current strength of 4 on the diathermy machine for a duration of five seconds would produce a bullet shaped lesion resembling a cylinder 4 mm. in length (corresponding to the length of the coagulating surface of the electrode) and 5 mm. in diameter on which was placed end-on a hemisphere 5 mm. in diameter, corresponding to the coagulation made by the tip of the electrode in front of it. It was assumed that there was no corresponding hemisphere of coagulation behind the electrode since this portion of it was insulated.

For a maximum current strength of 3, the diameter was assumed to be 4 mm. and for 2, 1 mm. Current strength 1 produced no permanent macroscopic lesion and current strength 5 was not usually used.

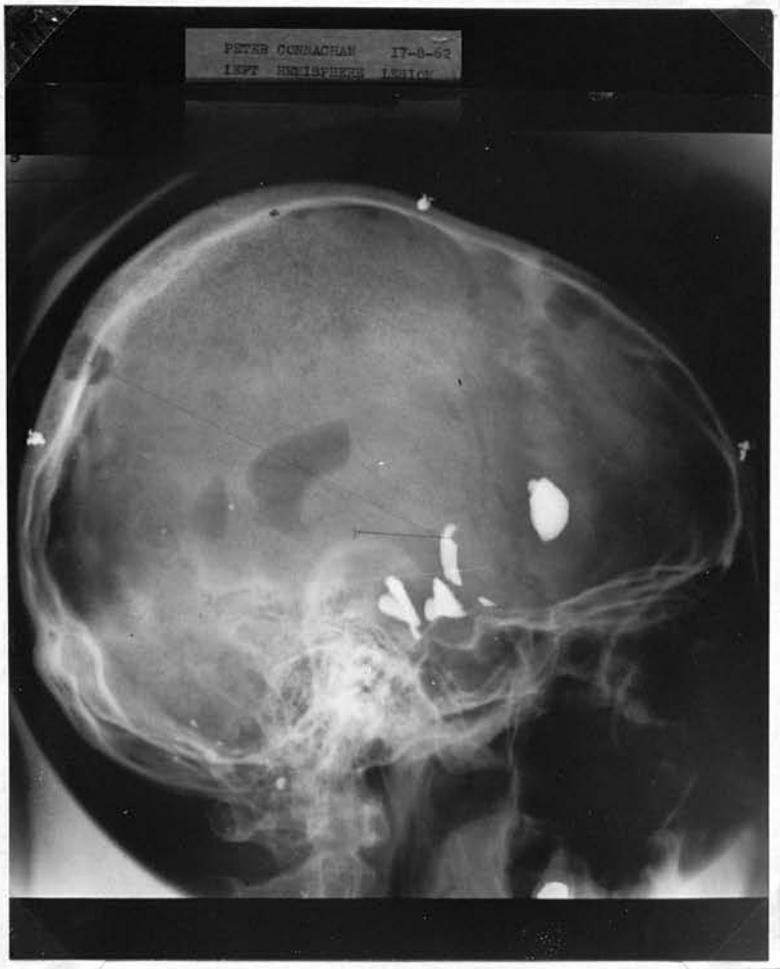
For each point along the electrode track where a lesion was made, such a bullet shaped lesion was drawn and the composite lesion of the coagulations done in Mr. P.C. is shown in the diagram. In his case, coagulations were done as shown below:

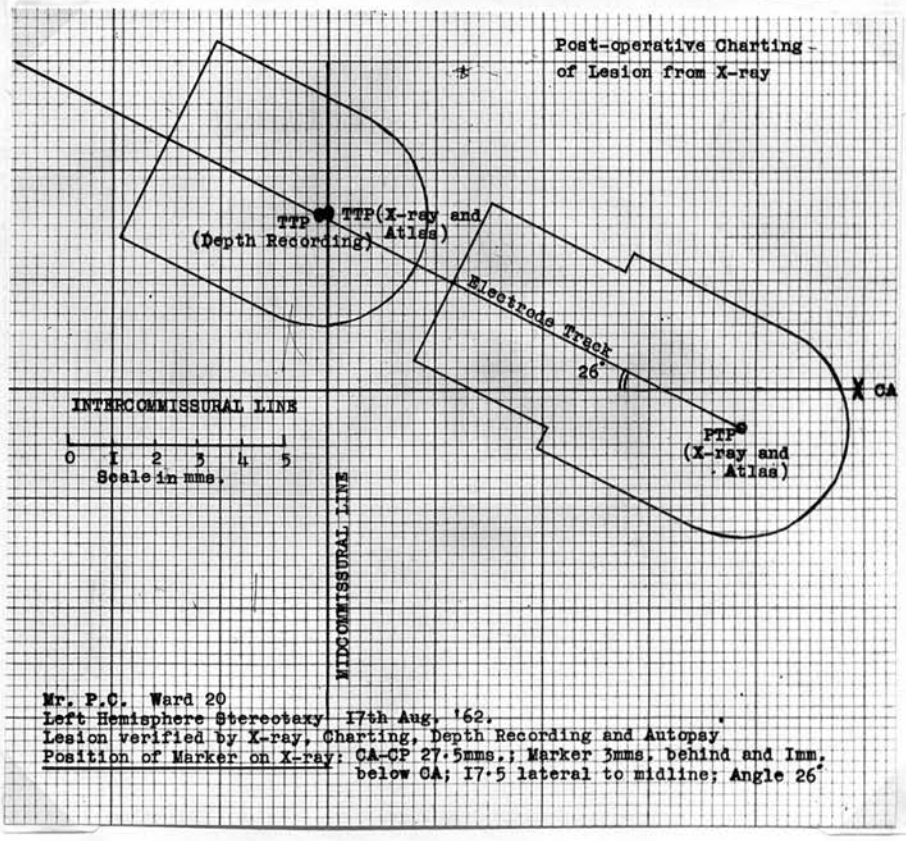
	Maximum current strength
(a) At the pallidal target (opaque marker)	4
(b) 3.5 mm. behind pallidal target	3
(c) At the thalamic target (11.5 mm. behind pallidal target)	4

Since the graph is a four times magnification all distances and diameters were magnified four times for purposes of drawing.

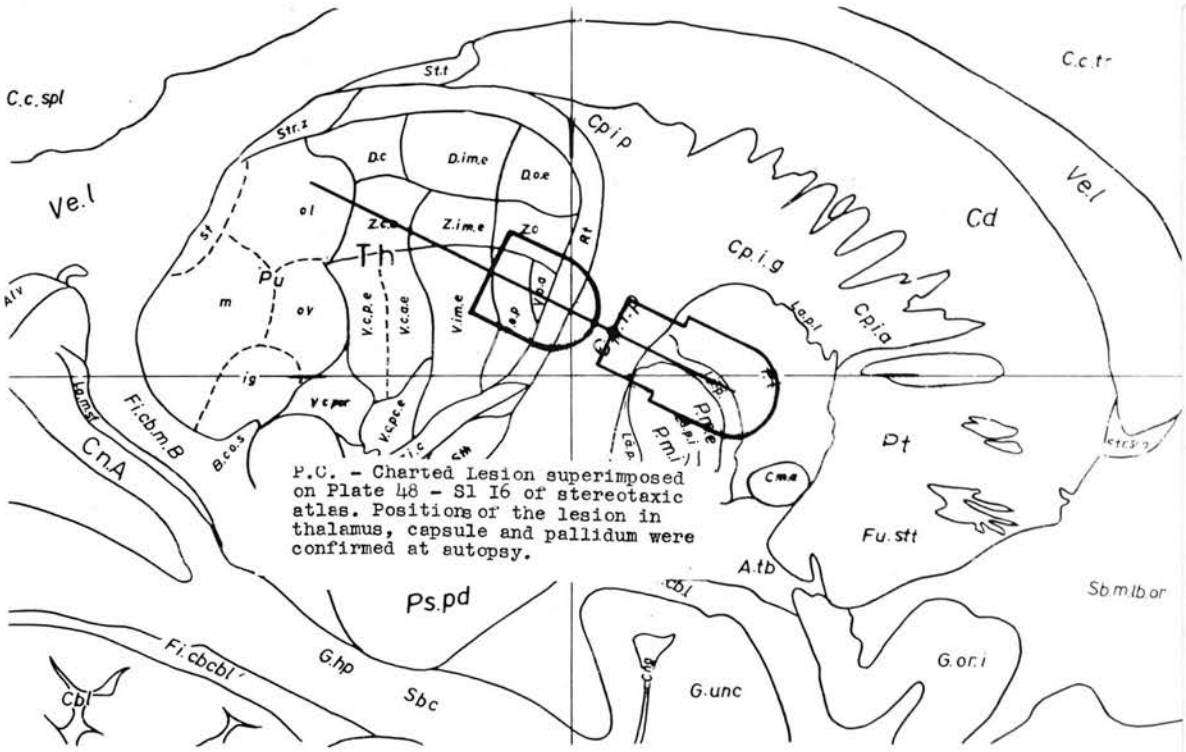
The entire diagram was now copied on cellophane paper and superimposed on the atlas for comparison.

It is to be noted that the size of the lesion and the alleged vagaries of diathermy did not interfere with any of the calculations or conclusions in this thesis for the simple reason that any point studied was taken as the central point of a lesion on the electrode track. The Cartesian co-ordinates of points on the electrode track only were taken into consideration for statistical analyses.

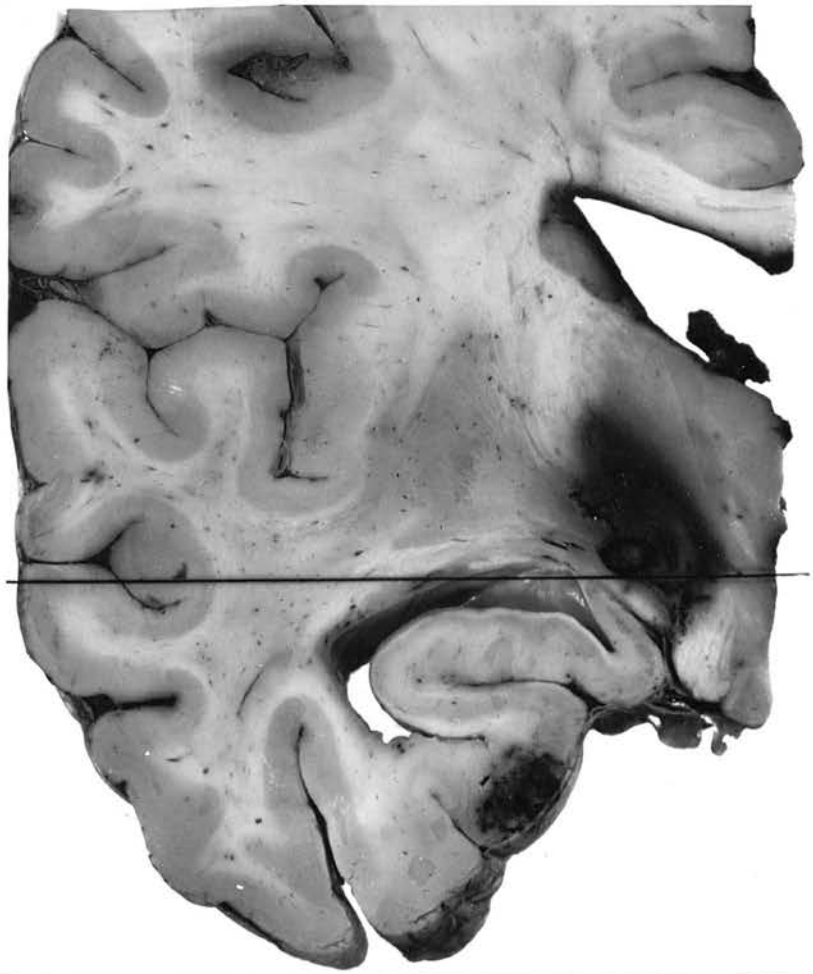




Mr. P.C. Ward 20
Left Hemisphere Stereotaxy 17th Aug, '62.
Lesion verified by X-ray, Charting, Depth Recording and Autopsy
Position of Marker on X-ray: CA-CP 27·5mms.; Marker 3mms. behind and 1mm. below CA; 17·5 lateral to midline; Angle 26°



P.C. - Charted Lesion superimposed on Plate 48 - Sl I6 of stereotaxic atlas. Positions of the lesion in thalamus, capsule and pallidum were confirmed at autopsy.



Coronal section 3.5 mm. in front of midcommissura plane at autopsy showing lesion in pallidum and capsule

APPENDIX B

Questionnaire Used for Follow-up

(i) Proforma to be filled in by patient.

PART A

Ref:

1. Have you any tremor? .....

   If so, is it the same, better or worse since operations? .....

   Which side do you have tremor now? .....

	Right side	Left	Both
First operation			
Second operation			
Third operation			

Worse ... W
Better ... B
Same ... S

2. Have you any stiffness? .....

   If so, is it the same, better or worse since operations? .....

   Which side do you have stiffness now? .....

	Right side	Left	Both
First operation			
Second operation			
Third operation			

Worse ... W
Better ... B
Same ... S

3. Do you have difficulty in speaking now? .....

   If so, is it the same, better or worse since operations? .....

   If any difficulty, was it after the first or second operation? .....

What is the nature of this difficulty? .....

Slurred
Difficulty in finding words
Loss of voice
Hoarseness of voice

4. Do you have difficulty in writing now? .....

If so, is it the same, better or worse since operations? .....

What is the nature of this difficulty? .....

Example - Irregular
Small

5. Do you have difficulty in walking now? .....

If so, is it the same, better or worse since operations? .....

What is the nature of this difficulty? .....

Tendency to tilt (forwards / backwards / sideways) Tendency to run

6. Do you have difficulty in passing water? .....

If so, is it the same, better or worse since operations? .....

What is the nature of this difficulty? .....

Hesitancy/leaking

7. Do you have difficulty in swallowing? .....

If so, is it the same, better or worse since operations? .....

8. Do you have attacks of turning upwards of the eyes?.....

If so, is it the same, better or worse since operations? .....

9. Are you troubled with salivation? .....

   If so, is it the same, better or worse since operations? .....

10. Do you have pain? .....

    If so, is it the same, better or worse since operations? .....

11. Do you get swelling of the foot? .....

    If so, is it the same, better or worse since operations? .....

12. Do you have difficulty with memory or concentration? .....

    If so, is it the same, better or worse since operations? .....

PART B

Please mark in the blank columns below, against each activity, the grade to which you belong.

Grade A: No difficulty at all.

Grade B: Can manage but rather slowly.

Grade C: Can manage but only with assistance.

Grade D: Require help for entire procedure.

ACTIVITY	BEFORE OPERATION			AFTER OPERATION		
	1st	2nd	3rd	1st	2nd	3rd
Walking						
Cutting up meat						
Feeding						
Dressing						
Doing up buttons and shoe laces						
Getting into and out of bed						
Turning over in bed						
Attending the toilet						

PART C

Please state briefly your own assessment as to what extent you have been helped by the operation and any other detail which you think will be of help to us.

APPENDIX B (ii)

Proforma to be filled in by relative

PART A

Ref:

Please mark in the blank columns below, against each activity, the grade to which ..... belongs:-

Grade A: No difficulty at all.

Grade B: Can manage, but rather slowly.

Grade C: Can manage, but only with assistance.

Grade D: Requires help for entire procedure.

ACTIVITY	BEFORE OPERATION			AFTER OPERATION		
	1st	2nd	3rd	1st	2nd	3rd
Walking						
Cutting up meat						
Feeding						
Dressing						
Doing up buttons and shoe laces						
Getting into and out of bed						
Turning over in bed						
Attending the toilet						

PART B

1. Please state briefly, under the following headings what you assess ..... to be since the first, second or third operation.

Has ..... changed and if so, what is the change?

a. Memory.

b. Concentration.

c. Emotions.

d. Personality.

e. General Activity.

f. General Interest in Life.

2. Please state to which Grade ..... belongs to, before and after operations:-

Grade A: Full working capacity.

Grade B: Reduced working capacity.

Grade C: Protected circumstances - minor home responsibilities.

Grade D: Ambulant but requiring some nursing care.

Grade E: Bedridden invalid requiring constant nursing care.

PART C

Please state briefly your own assessment as to what extent ..... has been helped by the operations and any other detail which you think will be of help to us.

APPENDIX B (iii)

Proforma to be filled in by doctor

PART A

Please state against the following signs and symptoms whether the patient has shown any change since operation. If so, what is the nature of this change.

1. Tremor Right Upper Limb.
2. Tremor Right Lower Limb.
3. Tremor Left Upper Limb.
4. Tremor Left Lower Limb.
5. Rigidity Right Upper Limb.
6. Rigidity Right Lower Limb.
7. Rigidity Left Upper Limb.
8. Rigidity Left Lower Limb.
9. Difficulty in speaking.
10. Difficulty in writing.
11. Difficulty in walking.
12. Difficulty in swallowing.
13. Difficulty in micturition.
14. Oculo-gyric crises.
15. Salivation.
16. Pain.
17. Oedema Feet.
18. Memory.
19. Concentration.
20. Drowsiness.
21. Confusion.
22. Dysphasia.
23. Personality changes.
24. Sensory deficit or paraesthesia (face and upper limbs).
25. Motor weakness.
26. Reflex changes (especially extensor plantar).
27. Imbalance.

PART B

Please state briefly your own assessment of the patient before and after operation with regard to any other matter which you think will be of help to us. In particular, I will be glad to know whether the operation has helped in the medical management of the case and whether the patient requires the same dosage of drugs now as before operation.

APPENDIX B (iv)

Proforma regarding attacks of oculogyric crises

(turning upwards of the eyes)

Patient's name:

Present address:

	Before 1st operation	After 1st operation	After 2nd operation
Frequency of attacks			
Duration of attacks			
Severity of attacks			

Are the attacks associated with sleepiness or a feeling of confusion?

What, if any, drug or action relieves these attacks?

How much do these attacks disable you and interfere with your work and daily life?

Reprinted from Excerpta Medica, International Congress Series No. 60 containing abstracts of papers read at the

SECOND EUROPEAN CONGRESS OF NEUROLOGICAL SURGERY

Rome - Italy, April 18-20, 1963

***84. Bilateral stereotactic lesions in the management of Parkinsonism and the dyskinesias**

GILLINGHAM, F. J. and KALYANARAMAN, S. *Edinburgh, Great Britain*

Sixty-four patients have been treated in Edinburgh by bilateral stereotactic operations over the five-year period from 1957. Apart from Krayenbuhl's work no detailed analysis of any large series of patients treated bilaterally have so far been published.

Thirteen of our patients were above the age of sixty. More than half of the total number required constant help for their daily personal routine before operation. All suffered from tremor and rigidity, except for two. In one tremor was predominant, in the other there was only rigidity. Only four were free from additional disabilities such as oculogyric crises, speech difficulties or autonomic phenomena.

The time interval between operations on the two sides was usually between three and twenty-four months, although in one case bilateral procedures were performed at the same operation. Charts of the electro-coagulation lesions from post-operative radiographs were made in all cases on stereotactic atlases. The sites of some lesions were further confirmed by operative biopsies and autopsy studies. Sixty-one of these patients had at least some involvement of the posterior limb of the internal capsule and twelve had planned bilateral transcapsular lesions.

Detailed follow-up studies were carried out in all the patients. Half had been observed for more than two years after the operation in the other hemisphere.

A large lesion did not necessarily mean that it was effective. The first essential to success was strategic siting of the lesion. The targets chosen were the ventro-lateral nucleus of the thalamus, internal capsule or pallidum, and some formed a combination of all three. In general, thalamo-capsular lesions gave the best result for tremor and pallido-capsular lesions for rigidity. The bigger the lesion, the greater were the chances of post-operative complications and neurological deficits. With small lesions, 7 mm. × 5 mm. × 5 mm., permanent neurological deficits were minimal. There was no immediate post-operative mortality.

All patients were benefited. Apart from two patients who had advanced disease and needed permanent institutional care before operation, all were discharged home to an independent life or suitably protected circumstances.

* Selected for presentation.

Rigidity and tremor were always improved and in a number abolished. Oculogyric crises were often reduced in frequency and severity and sometimes abolished after bilateral surgery. Festination was often improved and sometimes eliminated. Autonomic manifestations were sometimes helped but could often be considerably relieved by selective specific medical therapy after surgery. Almost always patients needed less medication after operation. Within broad limits, the greater the intellectual and emotional reserve of the patient before surgery the fuller was the response obtained by stereotaxy.

The main risk of operation on the second side was further reduction of voice volume in cases where it was already poor. Judicious siting and control of size of the lesion could sometimes avoid this. Slight return of tremor at the second operation to the previously treated limbs sometimes worried the perfectionistic type of patient. Another minor risk after bilateral operation was the precipitation of depression, responding however to drug therapy very satisfactorily.

A point of particular interest and importance in this series is that patients continued to improve for a long period after their second operation, sometimes for more than a year. Thus, long-term follow-up studies were much more encouraging than short-term assessments. There is also evidence that in a significant number of cases, rapid pre-operative deterioration had been arrested and stabilised at a satisfactory clinical level. The ultimate duration of control of disability from the dyskinesias would seem to be dependent on accurate siting of the lesion which is large enough to be effective yet not so large as to cause neurological, emotional or intellectual deficit.

Revision Series

Parkinson's Disease

F. JOHN GILLINGHAM, M.B.E., F.R.C.S. Eng. and Ed.

Professor of Neurological Surgery, University of Edinburgh

and

S. KALYANARAMAN, M.S. (Madras), F.R.C.S. Eng. and Ed.

Research Fellow, Dept. of Surgical Neurology, University of Edinburgh

IN 1817 James Parkinson published his celebrated essay on 'the shaking palsy', and this detailed description of the disease remains a classic in medical literature. It was over a hundred years later before surgical treatment was considered, and then it was largely concerned with destruction of the corticospinal pathways. Various operative procedures were devised to interrupt these connections, at the cerebral cortex, internal capsule, cerebral peduncle, and spinal cord. The results of these different methods were not encouraging, and the physiological basis of their application was insufficiently secure to pursue the problem in this way. Russell Meyers of New York (1942) changed the direction of our thinking by the observation that operative interruption of the pallidofugal fibres might modify rigidity and tremor in patients with Parkinsonism. His open operative approach, through the corpus callosum and third ventricle, was formidable and necessarily hazardous. Nevertheless, the early results indicated that his basic concept was correct. In 1920, Horsley and Clark in London devised a stereotaxic instrument for use in animals, and many years later, in 1947, it was adapted by Spiegel and Wycis in Philadelphia for use in the human. Since then many other types of stereotaxic apparatus have been developed, some complex, some of simple design.

Parkinson's disease usually manifests itself in late middle age, but it appears in all age-groups from the mid-twenties to over seventy. The sexes are equally affected. A stooped posture, flexed neck, short shuffling steps, and the absence of automatic arm-swinging, will reveal the disease on sight. A tendency to festination and to falling forwards, backwards, or to one side is often observed. Encephalitis, 'Spanish 'Flu', 'Sleeping Sickness', and, in the older age groups, cerebral atherosclerosis may appear in the patient's history. A very small proportion owe their symptoms to carbon-monoxide poisoning or to drug or metal intoxication, but in our experience no definite causative factor can be detected in a large number of histories.

The Symptoms

The symptoms of Parkinson's disease may be broadly classified into four groups—tremor, rigidity, akinesia, and autonomic phenomena.

Tremor commonly occurs at rest. It is diminished by voluntary muscular effort but it may also be aggravated by action; often the patient endures a

mixture of both types. One or more limbs may be affected as well as the face, eyelids, tongue, jaw, and neck. Tremor is variously increased by tension, excitement, concentration on a mental problem, or by consciousness of being observed, but it is absent during sleep. Unfortunately it is always worse in company and can be a great social nuisance, making the sufferer self-conscious and embarrassed, with the result that he tends to withdraw more and more from his fellows.

Rigidity chiefly affects the limbs. In the face it is apparent as the classical mask with a fixed expression and slow smile. Speech is impaired when the tongue, pharynx, and vocal cords are involved, and rigidity of the respiratory muscles may predispose to the terminal complication of bronchopneumonia.

Lack of initiation, coupled with the inability to produce organised movement, results in the akinetic syndrome which may be a major factor in the disease in some patients.

The excessive production of saliva is frequently associated with kyphosis, a flexed neck, open mouth, and difficulty in swallowing. Voice volume is reduced in the advanced case, sometimes severely. Attacks of involuntary up-turning of the eyes, known as oculogyric crises, occur in some patients for several hours at a time, and are sometimes accompanied by confusion. Often there is difficulty in passing water, or urgency of micturition.

In the early stages of the disease full movement of all joints is observed, the patient being able to move about and attend to his personal daily routine and work, although more slowly and stiffly, leading to increasing difficulty. There is slow or more rapid deterioration, and muscular contractures eventually supervene, resulting in secondary deformities of joints. The patient tends to keep his limbs in a fixed posture best suited to the chair or bed in which he lies. Gradually the disease progresses to a stage when the sufferer has a fixed stare and an open mouth. He is unable to feed himself and requires help for almost every simple human activity. Ultimately he becomes bedridden and succumbs, usually from bronchopneumonia.

The Use of Drugs

Before resorting to surgical treatment, drugs may be administered in an attempt to alleviate the symptoms and may well relieve rigidity and subdue tremor for some time, but the disease progresses undeterred. Some years ago the belladonna group of alkaloids were used, but they have now been replaced by the synthetic drugs, such as Benzhexol (Artane; Pipanol), Orphenadrine (Disipal), Procyclidine (Kemadrin), Benztropine (Cogentin), and Ethopropazine (Lysivane). More recently other drugs have shown themselves to be of value, for example, Dexamphetamine (Dexedrine) for the relief of drowsiness, Chlorphenoxamine (Clorevan) to reduce excessive salivation, and Imipramine (Tofranil) or Amitriptyline (Tryptizol) to combat mental depression.

Stereotaxic Surgery

Stereotaxic surgery has brought new hope to these chronic invalids and there are now many methods of performing such operations. Unless patients are bed- or chair-ridden they are accepted for operative treatment, and even advanced cases benefit to some extent from surgery, although total abolition of symptoms is impossible. In Edinburgh some 320 patients have been treated by means of small ovoid electrocoagulation lesions of 7 mm. × 5 mm. × 5 mm., and seventy-six patients have been treated bilaterally. Lesions are placed in the globus pallidus for the most satisfactory relief of rigidity; in the ventrolateral nucleus of the thalamus for the abolition of tremor; and at both these sites when tremor and rigidity exist together.

Preliminary stages of the operative procedure determine the midline of the brain, as distinct from that of the skull, and delineate radiological target points in the course of the electrode track. Local anaesthesia is used, supplemented by some of the more modern intravenous analgesics, so that the patient is asleep for much of the operation but awake and able to co-operate at certain stages of the procedure. The outer table of the skull is exposed in the frontal, coronal, and lambdoid regions through small incisions, and a row of small dental drill holes made at each site. Small opaque lead markers are inserted into the holes and a rubber catheter is placed in the frontal horn of the lateral ventricle through a burr-hole in the coronal region.

Small quantities of Myodil (a radio-opaque oil containing iodine) and air are inserted into the ventricular system and X-rays are taken to determine the marker at the three sites which correspond to the midline, which is shown by the line of the third ventricle and septum pellucidum. The pallidal and thalamic target points are also determined. The patient is now returned to the operating theatre where a small steel screw with an inset conical head is fixed into the skull at the three selected positions in the midline. The stereotaxic frame is fastened to these points, and radiological screening (using an image amplifier) is employed to align the electrode track. An accuracy of 1 mm. is essential for the complete success of the operation.

Following this, the electrode is introduced by a posterior burr-hole and, with the aid of mechanical and low-voltage electrical stimulation and by recording spontaneous activity of the discharging cells of the grey matter, an ideal site is chosen for the lesion. By means of heat, a small reversible lesion is made. If the symptoms are then relieved

and no untoward effects occur, the lesion is extended and made permanent, using electro-coagulation of increasing intensity. Throughout this part of the procedure the patient is fully conscious and co-operates with the surgeon and his assistants in their efforts, answering questions and performing tasks to demonstrate that consciousness, motor, sensory, and other functions are undisturbed.

In general, lesions in the thalamus and globus pallidus adjacent to and slightly involving the posterior limb of the internal capsule give the best results. At the end of the operation a small opaque marker is left at the site of the lesion, and from post-operative X-rays its exact position is charted on stereotaxic anatomical atlases.

The entire procedure requires about two hours and is tolerated very well, even by the old and infirm.

Rigidity and tremor are nowadays almost always benefited. Autonomic manifestations and akinesia are lessened only in a proportion of patients. Pain from rigidity, especially when severe, is strikingly relieved from the moment of electro-coagulation.

Detailed assessments of the results of operations have been published elsewhere.

Importance of the Team

The successful outcome of the surgical intervention from the practical aspect of the patient's life depends on the skill and co-operation of a team, in which the physiotherapist, occupational therapist, and speech therapist are important members.

The patient is usually kept in hospital for about three weeks for post-operative convalescence, and during this period intensive physiotherapy is given. Before the operation, however, the therapists not only help to prepare the patient to benefit to a maximum degree from the procedure but also co-operate with the surgeon in assessing the exact disability in each individual patient. Thus they are important members of the clinical research team. Post-operatively, apart from the routine care of the chest and limbs which is so necessary, physiotherapy has a vital role to play in the re-education and rehabilitation of these patients who have now found a new freedom of mind, body, and limbs as a result of surgery, for there is often a tendency in some patients to neglect the improved limbs for a period. Physiotherapy is also a useful adjunct to secondary orthopaedic procedures carried out after stereotaxic surgery, although in the moderately disabled most deformities are corrected in a remarkable way by relieving rigidity.

After discharge from hospital the patient is reviewed at regular intervals as an out-patient, and continues with physiotherapy, occupational therapy, or speech therapy for some weeks.

Originally an interval of three to six months was allowed before the patient was treated bilaterally, but in the younger age-groups in which there is no mental deterioration this has been reduced to a month or less.

Bilateral Operations

Bilateral operations which are necessarily performed in patients with advanced and widespread symptoms have often brought great relief by controlling tremor and rigidity in all four limbs. Surprisingly, other features like festination and oculogyric crises are occasionally greatly improved by bilateral operations. It appears from our follow-up of these patients that the disease process is slowed down by surgery, and it would seem that sometimes its progress is arrested. A particular hazard of bilateral operations appears to be the reduction of voice volume, especially in patients in whom it is already low.

In view of the more advanced clinical stage of patients with bilateral disease, greater support and longer treatment are demanded from the nursing, physiotherapy, occupational therapy, and speech therapy members of the team. Such time is well spent, for with patience and perseverance they benefit a great deal, gaining a measure of independence and freedom which gives great pleasure.

BIBLIOGRAPHY

Fénélon, F., and Thiébaut, F. (1950). *Rev. Neurol.* **83**, 280.

Gillingham, F. J., Watson, W. S., Donaldson, A. A., and Naughton, J. A. L. (1960). *Brit. Med. J.* ii, 1395.

Gillingham, F. J. (1961). *The Nursing Mirror*, April 15, 1960.

Gillingham, F. J. (1961). Eighth World Congress, International Society for the Rehabilitation of the Disabled, New York, 1960. *Journal of Chronic Diseases*, **13**, 215-220 (1961).

Gillingham, F. J. (1962). *Transactions of the Medical Society of London*, **77**, 52-56.

Gillingham, F. J. (1962). 1st Int. Symp. Stereoencephalotomy, Philadelphia 1961. *Confin. neurol.* **22**, 385-392.

Gillingham, F. J., and Kalyanaraman, S. (1963). Second European Congress of Neurological Surgery, Rome 1963. *Excerpta Medica. Int. Cong. Series*, No. 60, 84.

Guiot, G. (1958). *Neurochirurgia* (Stuttgart), **1**, 94.

Guiot, G., and Brion, S. (1953). *Rev. Neurol.* **89**, 578.

Hassler, R. (1957). II. *Int. Congr. Neuropath., London 1955.* Amsterdam, Pt. I. 29.

Hassler, R. (1957). II. *Int. Congr. Neuropath., London 1955.* Amsterdam, Pt. II. 637.

Meyers, R. (1942). *N.Y. St. J. Med.* **42**, 317.

Schaltenbrand, G., and Bailey, P. (1959). *Introduction to Stereotaxis with an Atlas of the Human Brain.* Grune and Stratton, New York.

Spiegel, E. A., Wycis, H. T., Marks, M., and Lee, A. J. (1947). *Science*, **106**, 349.

APPENDIX C (iii)

Reprint from *Madras Clinical Jour.*, **30** : (6) 129—134, 1963.

STEREOTACTIC SURGERY *

S. KALYANARAMAN, M. S., F. R. C. S., F. R. C. S. Ed.,
Commonwealth Scholar and Clinical Assistant,
Department of Surgical Neurology, The Royal Infirmary, Edinburgh.

During the past fifteen years a new subspeciality has arisen within neurosurgery. The first human stereotactic operation was performed by Spiegel and Wycis in Philadelphia in 1947. Already the scope of this type of surgery has widened so much that approximately twenty per cent of neurosurgical procedures now in some advanced centres of the world are stereotactic. In Paris, Stockholm, New York, Philadelphia and Tokyo there are clinics which deal only with stereotactic work.

Principle : The principle of stereotactic surgery is very simple. Modern neurosurgery was started by Harvey Cushing in the United States at the beginning of this century. Unlike surgery on other organs and systems of the body, neurosurgery has always had some special difficulties and drawbacks. To enter the intracranial cavity by means of an osteoplastic craniotomy is itself a formidable procedure requiring more time and involving more blood loss and trauma to the patient than most surgical approaches. The surgical approach to deep intracerebral structures like the thalamus and the basal ganglia is fraught with a high incidence of mortality, morbidity and irreversible neurological deficit if carried out by an open method. For, there are no natural planes of cleavage or serous sacs separating the constituent parts of the brain.

Methods : Neurosurgeons have long dreamed of a method which would enable them to reach deep intracerebral targets without the dangers of the open approach. Horsley and Clarke devised the first stereotactic instrument for use in animals. It took forty years before instruments were devised for man and now there are nearly a hundred different varieties. All of them work on the basic principle that a frame is attached rigidly to the skull and by means of air or positive contrast media introduced by lumbar puncture or a frontal burrhole the ventricular

* Specially contributed to this Journal.

system of the brain is outlined. The co-ordinates of the desired deep intracerebral target are now calculated from the X-rays on the stereotactic machine. The surgical instrument is now passed to the target by a single burrhole. Its exact location within the brain is identified by neurophysiological methods like novocaine injection, stimulation, reversible cooling or heating and depth electrical recording. Destruction of the target is then carried out as a therapeutic procedure. The size and extent of the lesion is usually guided by simultaneous neurological testing. Usually the lesions are only a few mm. in size and serve to interrupt pathological neural circuits without producing clinically demonstrable neurological deficit.

The details of the procedure vary in each method. In general stereotactic operations belong to one or other of the following methods. (a) The technique devised by Cooper and various modifications of it. Here temporary inflation of a balloon or moderate cooling by a probe is first done. If this is satisfactory, injection of destructive agents (chemopallidostomy, chemothalamotomy) or deep cooling (cryothalamotomy) achieves a permanent lesion. (b) The instrument and method of Leksell is probably the most widely used. The electrode carrier reaches the centre of a sphere which is adjusted to be the target, in whatever position the burrhole may be placed on the skull. Destruction may be done by radiofrequency, diathermy, cooling, cutting loop, radioactive isotopes or injection of alcohol. (c) The instruments devised by Spiegel and Wycis, Talairach, Riechert, Hughes, Sherwood and others work on similar principles but are relatively more complicated. It is questionable whether they achieve greater accuracy than the groups (b) and (d) despite their cost and size. (d) The methods used by Guiot and Gillingham depend on a much simpler instrument but require the image intensifier for the final part of the alignment. The electrode reaches the target only along a fixed plane. This is an advantage as well as a drawback in these methods. (e) McCaul's instrument is an example of a group which is very simple, small and not costly. These advantages however are achieved at the cost of a slight degree of inaccuracy. (f) Less well known methods. Some of these are practised by only one centre.

There is no single method which would suit all situations requiring stereotactic surgery. Just as different cases of carcinoma of the stomach may require esophagogastrectomy, total gastrectomy, partial gastrectomy, gastrojejunostomy, gastric exclusion, gastrostomy or morphia, different neurological diseases require different degrees of accuracy, methods of approach and number of lesions in stereotaxy. In general three methods-one very accurate, another moderately accurate (with an error of not more than ± 1·0 mm.) but versatile and adaptable and a third very quick and simple method (with an error of not more than ± 2·0 mm.) should serve all situations. In Britain, the Leksell, Gillingham and McCaul methods are the most commonly used. With these three methods any stereotactic procedure from aspiration of a cyst in a few minutes to implantation of multiple depth electrodes for epilepsy requiring a few hours can be satisfactorily done.

Complications: With increasing experience, the risk of mortality and morbidity has been extremely low.

In Professor Gillingham's series in Edinburgh the immediate operative mortality has been 0·25%. This is perhaps a record for any centre but the mortality rate in most clinics has been less than that of laparotomy. Morbidity depends on the primary condition treated, the general condition of the patient and the experience of the surgeon. In Professor Gillingham's series again, hemiparesis (0·5%), hemianopia (nil), hemianesthesia (0·25%), hemiballismus (1%) and other major complications have been extremely rare. Even when they occur, they recover completely in a few months. Minor temporary complications like facial palsy, incontinence, slurring of speech, drowsiness, confusion and dysphasia occur in a slightly larger percentage of cases and again last only for a few days. Compared to the immense benefit of the therapeutic procedure to diseases which have no other treatment, these risks are really very small.

THE PRESENT SCOPE OF STEREOTACTIC SURGERY

Parkinsonism: This is the syndrome most commonly treated by stereotaxy. Several thousands of patients have been operated upon so far in many countries with great benefit. Lesions are now made in one or more of the following structures — oral ventral nucleus, lateral posterior nucleus, and ventral posterior nucleus of the thalamus, the ansa and fasiculus lenticularis, fasciculus thalamicus, inner segment of the globus pallidus and upper part of the midbrain. It is likely that the same extrapyramidal circuits conveying abnormal impulses mediating tremor, rigidity and other manifestations are interfered with by any of the above lesions. Increasing experience has enabled many clinics to perform bilateral stereotactic surgery with minimal complications.

Tremor: A thalamocapsular lesion almost always reduces and often fully abolishes tremor in the contralateral limbs. Tremor of the jaw and tongue is relatively more difficult to control but good results are often obtained. Intention tremor of Parkinsonism can also be treated by a similar lesion.

Rigidity: A pallidocapsular lesion almost always reduces and often fully abolishes rigidity in the entire opposite half of the body – face, jaw, limbs, chest and spine. Action rigidity (akinesis) is more difficult to control than passive rigidity.

Oculogyric Crises: Bilateral lesions from the posterior route fully abolish this symptom in more than half the cases. In other cases the frequency and severity of the crises are reduced.

Blepharospasm: This is another symptom which is very amenable to stereotactic surgery.

Pain: When rigidity of muscles produces pain, it can be greatly relieved by stereotaxy.

Gait: Dragging of the foot, shuffling, lack of swinging of the arms and slowness can be improved by surgery. Propulsion, retropulsion, festination and rooting to the spot are more difficult to treat. Prolonged postoperative physiotherapy can be of great value.

Salivation: Dribbling due to open mouth, flexed neck and difficulty in swallowing can be benefited by operation. Intrinsic hypersecretion is better controlled by drugs or a small dose of radiotherapy.

Posture: Surgery releases the muscles from hypertonus. Exercise and supervised physiotherapy can easily correct the faulty posture in the postoperative period.

Handwriting: When tremor interferes with writing, surgery can produce a steady hand. Micrographia however needs prolonged postoperative training to improve.

Edema: When the ankles are swollen due to the adoption of immobile rigid posture, stereotactic surgery cures this secondarily by mobilising the patient.

Mental Deterioration: This is not benefited by surgery and in fact can be made worse. Advanced mental deterioration is a definite contraindication to operation.

Secondary Deformities: Fixed deformities of joints are more amenable to orthopaedic correction after stereotactic procedure reduces the hypertonus of the muscles involved.

Slurring of Speech: This is sometimes improved but often made worse by surgery, especially when done on both hemispheres.

Reduced Voice Volume: This is very rarely improved and often made worse by stereotactic operations especially if done bilaterally.

Dependance on Drugs: Dosage of anti-Parkinson medication is often reducible after surgery. However, in advanced cases requiring large doses total withdrawal of drugs postoperatively is not advisable.

Obviously surgery does not deal with the fundamental cause of the disease and only aims at producing symptomatic relief. Yet there is some evidence that surgery done early in the course of the pathological process helps to slow down or even arrest its progress.

Intractable Pain: Stereotaxy offers hope in some cases of intractable pain not amenable to other forms of treatment. Post-herpetic and other forms of facial pain and painful inoperable tumours of the head and neck come under this group. The spinothalamic tract in the midbrain, the nucleus ventrocaudalis-parvocellularis, higher portions of the sensory relay nucleus, dorsomedian nucleus, centromedian nucleus, thalamoparietal projection in the internal capsule or at a subcortical level and the thalamofrontal projection have been used as targets for destruction. Results are not uniform. Relief is often obtained only for a few months and often at the cost of some sensory deficit. In cases of intractable pain not treatable otherwise and where life expectancy is anyway limited by incurable malignancy even such results are often worthwhile.

Mental Disorders: The disadvantages of classical leucotomy can be avoided by small discrete lesions strategically placed. These produce the desired effect without personality alterations of an undesirable nature. Targets used include the dorsomedian nucleus of the thalamus and anterior limb of the internal capsule. The aim is to interrupt connexions between the thalamus and the frontal lobe. Stereotactic isolation of area 13 has been achieved by Knight by introduction of radioactive isotopes and is probably effective by interruption of fronto-hypothalamic pathways. Syndromes which are benefited include obsessional states, involutional melancholia, maniac depressive psychoses, selected cases

of schizophrenia, some hysterical manifestations, drug addiction, undifferentiated tension states and excessive emotional reaction to intractable pain. Tranquillisers and other modern drugs have reduced the number of cases requiring psychosurgery but there is still a percentage best treated stereotactically.

Epilepsy: Functional surgery of the brain reaches its greatest complexity in epilepsy. Talairach and Narabayashi have made significant contributions in this field. Epileptic foci not revealed otherwise can be detected and destroyed using chronically implanted multiple deep cerebral electrodes. Some forms of epilepsy and behaviour disorders not amenable to medical or other non-surgical treatment have been cured by unilateral or bilateral amygdalotomy.

Spasmodic Torticollis: This is a particularly difficult disease to manage stereotactically or otherwise. Bilateral stereotactic surgery has recently offered hope to this otherwise intractable condition.

Chorea: A few cases of Huntington's chorea have been reported where pallidal lesions have reduced the involuntary movements. Probably the procedure might be of use in senile chorea and electric chorea as well but too few cases have been recorded. Mixed choreo-athetotic movements certainly benefit from surgery. The exact reason why surgery is able to influence these movements is debatable. As in Parkinsonism we seem to be strategically interfering with extrapyramidal pathways.

Intention tremor of diverse origins: Heredofamilial tremor is particularly amenable to bilateral stereotactic thalamocapsulotomy. Intention tremor due to disseminated sclerosis responds to surgery equally well. Intention tremor of unknown origin and persistent for several years can be similarly abolished. In these cases the aim is to interrupt the cerebellar connexions to the ventral intermediate nucleus of the thalamus (according to some authors).

Other involuntary movements: Athetoid movements of multiple origin usually but not always are reduced. A few cases of Dystonia Musculorum Deformans have strikingly responded while others prove difficult to control. Even in the latter group physiotherapy and sedatives have a greater controlling influence after surgery than before it. Involuntary movements and hypertonus associated with cerebral palsy have been successfully treated especially by the Japanese. When hepatolenticular degeneration is slowly progressive and the condition is not too far advanced, stereotaxy can give some symptomatic relief. Of course surgery cannot affect the basic biochemical disorder of this condition. A few cases of myoclonus have been reported as improved by lesions in the posterior oral ventral nucleus of the thalamus. In general, results in the cases of involuntary movements are not as satisfactory as in cases of Parkinsonism.

Tumours of the Brain: In a small selected group, stereotaxy may be helpful in implanting radioactive seeds as the only measure or as supplementary to subtotal excision. It may also be useful to instil chemotherapeutic agents into deep seated small tumour or abscess or cysts. Rarely it may guide the sites for multiple biopsies.

Stereotactic Surgery of the Pituitary Gland: This is another field where mortality and morbidity has been lowered

considerably with the advent of stereotaxy. Carcinoma of the Breast, Carcinoma of the Prostate, Diabetic Retinopathy, Cushing's Syndrome, Primary Tumours of the Pituitary and severe Thyrotoxicosis have been treated by stereotactic implantation of radioactive seeds into the pituitary fossa either from above or by the transnasal route.

Foreign Bodies: Deepseated foreign bodies of the brain are best removed after localisation by accurate stereotactic guides.

In addition to the vast therapeutic benefit of stereotactic procedures, they have provided unique opportunities to study the neuroanatomy, neurophysiology and the living patholgy of the human brain. Depth electrical recording with audiomonitoring has been successfully practised by Guiot in Paris and Gillingham in Edinburgh. This method has not only eliminated the errors involved in individual variation of the brain but has provided electrophysiological data of fundamental importance regarding the structure of the thalamus and posterior limb of the internal capsule.

The improving economic and health standards in India have raised the life expectancy sharply during the past few years. In the next few decades, when infectious diseases and deficiency disorders are brought under control, we will be faced with the problem of degenerative conditions and neoplasms affecting the older age group and stereotactic surgery would be required to a much greater extent.

I am grateful to Professor F. J. Gillingham for stimulating my interest in this field and for permission to quote his figures.

REFERENCES

(The following list is meant only as an introduction to this fascinating subject. It is by no means comprehensive of all the growing points in such a rapidly advancing field)

1. "Parkinsonism – Its Medical and Surgical Therapy" By Cooper, I. S., C C, Thomas, Springfield, Illinois, 1961.
2. "Pathogenesis and Treatment of Parkinsonism". Edited by Fields, W. S., C. C. Thomas, Springfield, Illinois, 1958.
3. "The Shaking Palsy – A Symposium". Edited by Elliott, H. and Nashold, B., McGill University Press, Montreal, 1959.
4. "Stereoencephalotomy" By Spiegel, E. A. and Wycis, H. T., Grune and Stratton, New York, Part I Methods and Stereotaxic Atlas of the Human Brain. 1952. Part II Clinical and Physiological Applications, 1962.
5. "Introduction to Stereotaxis with an Atlas of the Human Brain". Edited by Schaltenbrand, G. and Bailey, P, Theme, Stuffgard, New York, 1959.
6. "First International Symposium on Stereoencephalotomy". Edited by Spiegel, E. A. and Wycis, H. T., S. Karger, New York, 1963.
7. Hughes, B 'Stereotactic Surgery'. British Surgical Practice, Surgical Progress, 1961. 129—158.
8. Gillingham, F. J., Donaldson, A. A., Watson, W. S. and Naughton, J. A. L. The Surgical Treatment of Parkinsonism. Brit. med. J. 1960, 2, 1395—1402.
9. Gillingham, F. J. and Kalyanaraman, S. Excerpta Medica International Congress Series No. 60. Second European Congress of Neurological Surgery, Rome 1963. Bilateral Stereotactic Lesions in the Management of Parkinsonism and the Dyskinesias. 115—116.

APPENDIX (D)

Nomenclature of Thalamic Nuclei

There is no universal agreement on the nomenclature of human thalamic nuclei. Different methods are used by different authors and this has tended to produce confusion.

Guiot (1963) used a very simple terminology for practical purposes. The sensory relay nuclei (the region where, at operation, he was able to record with a micro-electrode evoked potentials on peripheral stimulation) were together called the VP nucleus. The region posterior to this was the pulvinar. The region above was the LP nucleus. The region in front was the VL nucleus. What was lost in accuracy, was certainly gained in terms of clarity, by such a usage.

Brain (1962) divided the lateral nuclear mass into a larger ventral and a smaller dorsal part. He subdivided the ventral part into (1) the anterior ventral with striatal connections (2) the lateral ventral with cerebellar connections and (3) the posterior part consisting of (a) posteromedial ventral receiving trigeminothalamic fibres and (b) posterolateral ventral receiving the spinothalamic tract and lemniscus.

The terminology followed in this thesis adopted from Hassler (1959). He divided the thalamus into a very large number of subdivisions. From his description, the part of the sensory relay nuclei receiving fibres from the face (corresponding to VPM of cat and monkey) would seem to be V.c.a.i., V.c.p.i. and V.c.p.c.i. The part of the sensory relay nuclei receiving fibres from the trunk and

limbs (corresponding to VPL of cat and monkey) would seem to be V.c.a.e., V.c.p.e. and Z.c. But his description of fiber connections is rather detailed and not very clear and it is difficult to make an accurate comparison or simplified extract. Apart from those of Guiot and his co-workers, the present study is the first detailed one on human sensory relay nuclei by the method of evoked potentials. In our present state of knowledge (or rather, of ignorance) it was felt wise to use the general terms sensory relay nucleus of face, of upper limb, etc. rather than the minute subdivisions of Hassler which cannot be recognised in the living patient by present methods.

The names Voa and Vöp for the anterior and posterior parts of Hassler's oral ventral nucleus have come to be widely accepted by stereotaxic surgeons making thalamic lesions. These terms are therefore freely used in the text.

The following detailed nomenclature is abstracted from Hassler (1959) to facilitate reference to the scattergrams superimposed on the atlas plates.

The lateral nuclear region is subdivided into (a) Dorsal (b) Zentral (c) Ventral and (d) Lateropolar groups.

Dorsal Nucleus

D.o.e.	Dorso-oralis externus
D.o.i.	Dorso-oralis internus
D.im.s.	Dorso-intermedius superior
D.im.e.	Dorso-intermedius externus
D.im.i.	Dorso-intermedius internus
D.c.	Dorso-caudalis
D.sf.	Dorsalis superficialis

Zentral Nucleus

Z.o.	Zentrolateralis oralis
Z.im.e.	Zentrolateralis intermedius externus
Z.im.i.	Zentrolateralis intermedius internus
Z.c.e.	Zentrolateralis caudalis externus
Z.c.i.	Zentrolateralis caudalis internus

Ventral Nucleus

V.o.a.	Ventro-oralis anterior
V.o.i.	Ventro-oralis internus
V.o.p.	Ventro-oralis posterior
V.o.m.	Ventro-oralis medialis
V.im.e.	Ventro-intermedius externus
V.im.i.	Ventro-intermedius internus
V.c.a.	Ventrocaudalis anterior
	V.c.a.e.
	V.c.a.i.
V.c.p.	Ventrocaudalis posterior
	V.c.p.e.
	V.c.p.i.
V.c.pc.	Ventrocaudalis parvocellularis
	V.c.pc.e.
	V.c.pc.i.
V.c.po.	Ventrocaudalis portae

Lateropolar Nucleus

L.po.s.	Lateropolaris superior
L.po.e.	Lateropolaris externus
L.po.i.	Lateropolaris internus
L.po.mc.	Lateropolaris magnocellularis
L.po.b.	Lateropolaris basialis

References

Brain, Lord (1962). Diseases of the Nervous System. Oxford University Press, London. 32-33.

Guiot, G. (1963). Personal Communication.

Hassler, R. (1959). In Schaltenbrand, G. and Bailey, P. Introduction to Stereotaxis with an Atlas of the Human Brain. Thieme, Stuttgart, 230.