BILATERAL THALAMOTOMY AND PALLIDOTOMY AS TREATMENT FOR BILATERAL PARKINSONISM* 

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Clinical experience concerning the results of bilateral stereotaxic operations for parkinsonism to the present day is very limited. But meager information can be derived from the literature available. The question of bilateral thalamotomy is still open to discussion. Hassler and Riechert were the first to report postoperative states of impaired consciousness. According to their experience, these authors do not regard it justifiable to perform bilateral thalamic operations for bilateral parkinsonism, and they have advocated the combination of pallidotomy on one side and thalamotomy on the opposite. This opinion, which is stated again in their last paper, is also shared by Walker. Spiegel discussed the possibility of producing the effect of a leucotomy by cutting the thalamocortical pathway with a lesion located in the area of the nucleus ventralis anterior of the thalamus. Most of Cooper and Bravo's patients with bilateral parkinsonism have had the pallidal operation on one side and the thalamotomy on the other. The feeling of Cooper has been that he would hesitate to sacrifice the same structure bilaterally, no matter what that structure may be.

It is the purpose of this paper to review and compare the results of bilateral operations in our own series. In this series of 263 cases of parkinsonism, bilateral operations have been performed on 51 patients. Of these 51 patients, 23 have had bilateral thalamic operations, and in 28 cases pallidotomy was performed on one side and thalamotomy on the opposite. The bilateral operation was carried out usually with an interval of 6 months or more between both interventions. Omitting the early period of our experiences, only those patients who tolerated the initial operation without complications were selected.

TECHNIQUE OF OPERATION, EXPLORATORY STIMULATION AND HIGH-FREQUENCY COAGULATION

Well-defined lesions of predetermined size are produced within the thalamus by resorting to the combined use of the Riechert stereotaxic instrument for the introduction of the coagulating electrode, the Wyss stimulator for exploratory stimulation, and the Wyss coagulator for high-frequency coagulation. A specially designed unipolar electrode is used, the tip of which is placed accurately by means of roentgen-ray checks and subsequent electrical stimulation of the target area. High-frequency coagulation is then carried out with this electrode.

The technique is based upon the following principles:

The high degree of accuracy attained in stereotaxic localization (± 0.5 mm.) demands equally high precision in electrical stimulation and methods of coagulation. The unipolar technique has proved particularly suitable for coagulation, for it ensures quantitative relation between the strength of high-frequency current applied, the active surface of the electrode used, and the volume of coagulation produced. The unipolar array has, however, also proved suitable for exploratory stimulation. The technique of selective stimulation of nervous tissue, as originally contrived by Hess, developed further by Wyss, and applied to stimulation of the motor cortex by Wyss and Obra-

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dor;\textsuperscript{28} has been simplified in view of its application to general research of the brain as well as to stereotaxic neurosurgery.\textsuperscript{26,27,29} For the sake of convenience only one electrode is used for both stimulation and coagulation. It serves as the active electrode, the indifferent electrode, of large surface, being placed on the back of the neck of the patient.

Because of the high-current intensity necessary for coagulation and the correspondingly large area of the bared tip of the electrode, resistance to flow of current through the tissue becomes rather low and an even lower output impedance is required for both stimulator and coagulator. Thus constant-voltage device is ensured for stimulation as well as coagulation and, consequently, voltage of output does not depend on resistance of load, the stimulator’s voltage of output refers directly to strength of stimulus, and measurement of the voltage of output of the coagulator can be dispensed with. Coagulation with continuous high-frequency current has the advantage of being electrically nonstimulating.\textsuperscript{22,23,29} Transient excitatory effects, which may appear at sudden make or break, are avoided by slowly increasing and decreasing the intensity of the coagulating current. This also avoids stimulation by heat, i.e., by too fast a rise of local temperature. The frequency, however, should not exceed half a megacycle per second, in order to ensure sufficient accuracy in measuring the intensity of the coagulating current. Duration of flow of current depends on the size of lesion to be obtained; it varies from a few seconds for small lesions up to half a minute for large lesions.

Physical problems encountered in high-frequency coagulation of brain tissue have been studied theoretically and experimentally. Unaware of the earlier work of Hunsperger and Wyss\textsuperscript{8} on quantitative high-frequency coagulation of brain tissue in the cat, and of the further improvement of the technique by Wyss\textsuperscript{24} Aronow\textsuperscript{1} recently studied the physical problem of making radio-frequency lesions in the brain. Theoretical assumptions made by this author are questionable, insofar as conduction of heat through the shaft of the electrode was not taken into account and temperature was therefore thought to be highest around the bared tip of the electrode. It was even claimed that boiling of the tissue fluid may occur at the conducting surface of the electrode, and that this would be an advantageous safety feature! From the experimental data obtained by Hunsperger and Wyss,\textsuperscript{8} however, it is clear that temperature can attain highest values only at a certain distance from the electrode, and that steam-bubble formation should by no means occur in a coagulation performed carefully. It also should be noted that a low output impedance of the coagulator would in cases of overheating with the effect of boiling prevent increase of voltage because of increase in resistance of tissue. Another type of increase of resistance may, however, take place at temperatures below the boiling point, probably because a thin layer of gas (CO\textsubscript{2}, O\textsubscript{2}, N\textsubscript{2}) is formed over the conducting surface of the electrode. Contrary to the effect of boiling, this type of increase of resistance is perfectly reversible, and the neurosurgeon must be aware of the fact that
an unexpected fall of the correctly applied coagulating current does not mean serious failure of conduction or damage to the tissue. In such instances the procedure of coagulation has to be interrupted and repeated after a few minutes’ interval.

For technical reasons as well as for practical convenience, stimulator and coagulator should on no account be confined within the same apparatus.

The stimulator (Fig. 1) is fully transistorized and operates on the mains. The stimulating impulses are variations of delayed rise and fall of current derived from a low impedance-output potentiometer (middle knob). The duration of impulse can be varied from 0.1 to 20 msec. by a 6-step selector switch (to the right). The frequency of impulses is chosen between 2 and 40 per sec. by another 6-step selector switch (to the left). The main switch (bottom to the left) serves repetitive operation directly, audible control of frequency of the stimulus is provided with an attenuator of volume (bottom to the right). The positive output terminal is connected to ground, whereas a large condenser is inserted between the impulse-negative pole of the potentiometer and the negative output terminal. This prevents the component of direct current passing through the stimulated tissue.

The coagulator (Fig. 2) is a high-frequency generator operating on the mains. Output terminal 1 (left) must be connected to ground and to indifferent electrode. Output terminal 2 (right) is the active pole, and must be connected with the coagulating electrode. The current of 500,000 c./sec. is delivered automatically. On pressing button on bottom of front panel, the intensity of current rises continuously during 5 sec., remains at constant level for a variable time—pre-set by 6-step selector switch (on the left)—viz. between 5 and 60 sec. as measured from the beginning of flow of current, and then falls within 1 sec. to zero. Intensity of current at constant level controlled on milliammeter is adjusted by turning power dial (on the right) to appropriate position. The electrodes (Fig. 3) are made either of insulated metal rods (a) or of Teflon tubes with metal core (b). For the pallidotomy or the thalamotomy dealt with in this paper, their overall diameter is 2 mm., their length is 200 mm. Each electrode is borne by a Teflon holder which in turn is fixed to the stereotaxic instrument by means of a special adapter ensuring low capacity to earth. The surface of the uninsulated tip of the electrode should be kept clean, and, whenever necessary, be polished before use. Sterilization, either wet or dry, up to 300°C. is possible. The resistance of brain tissue amounts, in unipolar array, to some 500 ohms.

The exploratory stimulation is carried out as soon as the electrode has been placed within the target area and the position of its tip has been controlled by roentgen-ray checks. One chooses a middle position for duration of impulse and starts stimulation with a low frequency of a few impulses per sec., not exceeding 2 volts on the output potentiometer. One then proceeds to higher voltage and frequency in order to obtain a more pronounced response. More than 4 or 5 volts, however, should not be used. Trials of stimulation should be separated by intervals of at least 30 sec. The response to stimulation of the ventrolateral nucleus of the thalamus consists of an increase or decrease of spontaneous motor activity, such as tremor or rigor, whereas direct motor effects indicate probable spread of current to pyramidal-tract fibres in their course through the internal capsule, i.e., high-stimulus intensity. If, therefore, stimulation of the ventrolateral nucleus of the thalamus with adequate strength does not elicit direct motor responses of the pyramidal type, one may conclude that the electrode is located at a sufficient distance from the internal capsule, and that subsequent coagulation will not encroach upon this structure.

As soon as the right position of the tip of the electrode is confirmed by roentgen ray and exploratory stimulation, the coagulator is substituted for the stimulator. In order to ensure successful destruction within the ventrolateral nucleus of the thalamus, a lesion of 200 to 250 mm. is sufficient. This size of lesion is obtained by us-
ing one of the electrodes described above, with an active area of 20 mm.\textsuperscript{2}, and by passing a high-frequency current of 150 ma. for the insulated metal type of electrode, and 125 ma. for the Teflon-tube type. In either case the total duration of flow of current should be approximately 30 sec. Whenever the first coagulation does not seem sufficient clinically, it may be enlarged by subsequent coagulation, carried out in the same way, after the tip of the electrode has been somewhat lowered or raised. Whenever smaller electrodes, such as the stylet electrodes of the Riechert instrument, are used for supplementary coagulations, weaker current is adequate. The intensity required for optimum coagulation, however, depends not only on the size of the active area, but also on the thermal conductivity of the electrode employed, and should be determined for each type of electrode by previous experimental tests on an animal (cat). After successful coagulation, the electrode is withdrawn and the bared tip is controlled carefully; the metal surface must be clean and free from adherent tissue.

High-frequency current has been applied to stereotaxic neurosurgery by several groups of authors. Talairach et al.\textsuperscript{17} employed a former model of the Wyss coagulator, whereas Leksell\textsuperscript{18} and Riechert and Mundinger\textsuperscript{19} used standard electrosurgical high-frequency apparatus. Hassler et al.\textsuperscript{6} sometimes resorted to a more recent type of the Wyss coagulator. Sweet et al.\textsuperscript{18} started with the high-frequency isolation unit of the Grass stimulator (SIU 4A) fed from a power supply of direct current; this preliminary set-up has been re-placed by a 2 megacycle lesion generator designed by Aronow.\textsuperscript{4} Only Yaşargil et al.,\textsuperscript{29} however, founding their work on the earlier experimental investigations of Hunsperger and Wyss,\textsuperscript{8} succeeded in obtaining well-defined coagulation-lesions of predetermined size free from effects of boiling and burning. Their success was the result of attention to the quantitative side of the electrical procedure of high-frequency coagulation, the use of appropriately selected electrodes with sufficiently large areas of contact, and the carefully controlled intensity of the coagulating current.

RESULTS

a) Anatomical Localization of Coagulation-Lesions within the Oral Ventrolateral Nuclei of the Thalamus. Three autopsy specimens demonstrate our target area for thalamotomy. In Fig. 4 the lesion in the ventrolateral nucleus (v.o.p. of Hassler\textsuperscript{4}) of the left thalamus is shown in the case of a 38-year-old female patient who had been suffering for 11 years from a progressive bilateral postencephalitic parkinsonism. On Oct. 8, 1959 two coagulations with main electrodes and two coagulations with side electrodes were made in the ventrolateral nucleus of the left thalamus and the operation was followed by complete loss of tremor and rigidity in the limbs of the right side. The patient recovered very
well. She was able again to care for herself. Four months after the operation, on Feb. 2, 1960, the patient died from influenzal pneumonia in another hospital. The autopsy specimen shows that the coagulation-lesion was of small size and well-placed within the target area of the left oral ventral thalamic nucleus (v.o.p.).

Fig. 5 demonstrates the coagulation-lesion in the right oral ventral thalamic nucleus of a female patient, aged 68, who had been suffering from Parkinson's disease over a period of 5 years. On April 23, 1959 three high-frequency coagulations in the above-mentioned target area were made with complete loss of tremor and rigidity on the left side. Nine months later, on Jan. 4, 1960, the patient died in another hospital from diabetes mellitus. Section of the brain shows a small lesion, not more than 3 mm in diameter, located in the target area, and this in spite of the fact that three high-frequency coagulations had been performed.

Fig. 6 is the autopsy specimen of Case 20 (Th.B.) in our Table 1 of bilateral thalamic coagulations. It shows a small lesion placed accurately in the right oral ventral thalamic nucleus, and a fairly large lesion of the left oral ventral thalamic nucleus extending into the medial thalamus. This large lesion obviously gave rise to progressive cerebral hyperthermia which was the cause of death 10 days after the second operation. It must be stated that in this case the determination of the target point was not made accurately enough in the second operation.

These three autopsy specimens demonstrate that a small and accurately placed coagulation-lesion in the region of the ventrolateral nucleus of the thalamus produces complete and enduring relief of parkinsonian tremor and rigidity. As shown in Table 2, in pallidotomy many more lesions—up to 18 coagulations with main and side electrodes—are needed than in thalamotomy, in which one or two coagulations in the oral ventrolateral nuclei are sufficient to produce enduring and complete results. This is in relation
with the size and shape of these nuclei: the narrow and high shape of the internal pallidum between internal capsule, substantia innominata and optic tract makes its complete destruction without injury to the neighboring structures very difficult, whereas the structure of the ventrolateral nucleus is compact.

b) Effects of Bilateral Coagulation within the Oral Ventrolateral Nucleus of Thalamus and within the Pallidum Internum. Our case-material of bilateral operations in the basal ganglia is summarized in Tables 1 and 2. In all cases of thalamotomy the coagulation was made in the posterior portion of the oral ventral nucleus (v.o.p. of Hassler = posterior basal part of VL). According to our experience, this coagulation abolishes rigidity as well as tremor. In Table 1 the bilateral symmetrical thalamic operations are summarized, and in Table 2, the combined operations of unilateral pallidal and contralateral thalamic coagulations.

**ANALYSIS OF RESULTS**

a) In our series of 23 cases of bilateral thalamotomy (Table 1) the postoperative results have been excellent in 15 cases (65
### TABLE 1

**Summary of 23 cases of bilateral symmetrical thalamic operations**

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<tr>
<td>1. G.A.</td>
<td>68 y.</td>
<td>X 3</td>
<td>Bilateral tremor &amp; rigidity, Short stiff gait, incontinence urine, general weakness, reduced working capacity</td>
<td>3</td>
<td>Excellent, No tremor, no rigidity</td>
<td>1</td>
<td>R.v.o.p. 3-5-39</td>
<td>Complete loss tremor &amp; rigidity. On 4th postop. day fell out of bed, left hemiparesis, marked confusion loss of speech, slight euphoria</td>
<td>16 mos. after 2nd op. steady improvement. Normal gait, no incontinence urine, slurred, aphonic speech, slightly euphoric</td>
<td>Good</td>
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<td>2. St.E.</td>
<td>30 y.</td>
<td>XX 7</td>
<td>Bilateral tremor &amp; rigidity, Slightly slurred speech, short stiff gait</td>
<td>£ 2</td>
<td>R.v.o.p. 9-5-39</td>
<td>£ 2</td>
<td>R.v.o.p. 3-14-59</td>
<td>Somnolent 2 wks. Apathetic, salivation, compulsorily crying, no tremor, no rigidity</td>
<td>10 mos. after 2nd op. no tremor &amp; rigidity, no intellectual impairment, writing &amp; speech slightly impaired</td>
<td>Excellent</td>
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<td>3. P.G.</td>
<td>39 y.</td>
<td>XX 15</td>
<td>Bilateral tremor &amp; rigidity, Disturbance speech, swallowing, gait, Impaired writing</td>
<td>£ 3</td>
<td>R.v.o.p. 14-4-38</td>
<td>£ 3</td>
<td>R.v.o.p. 10-1-39</td>
<td>No complications</td>
<td>No tremor, no rigidity, no improvement of speech &amp; writing, gait improved</td>
<td>Excellent</td>
</tr>
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<td>4. D.A.</td>
<td>52 y.</td>
<td>X 5</td>
<td>Bilateral tremor &amp; rigidity, left more than right, great impairment own care &amp; working capacity. Slurred speech, short gait</td>
<td>£ 2</td>
<td>R.v.o.p. 5-10-59</td>
<td>£ 3</td>
<td>R.v.o.p. 3-10-39</td>
<td>No tremor, no rigidity, apathy</td>
<td>3 mos. after 2nd op. no tremor, no rigidity, Propulsion, speech more impaired, no intellectual deficit, 75% working capacity</td>
<td>Fairly good</td>
</tr>
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<td>5. W.P.</td>
<td>61 y.</td>
<td>X 11</td>
<td>Bilateral tremor &amp; rigidity, Salivation, akinesia, unintelligible speech, short gait, pre- &amp; retropulsion, moderate psycho-organic syndrome, unable to work</td>
<td>£ 2</td>
<td>R.v.o.p. 3-24-39</td>
<td>£ 1</td>
<td>R.v.o.p. 10-3-39</td>
<td>No tremor, no rigidity. Few wks. mentally impaired, apathetic, slight right hemiparesis</td>
<td>3 mos. after 2nd op. no propulsion, speech more impaired, mentally dull</td>
<td>Good</td>
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<td>6. St.S.</td>
<td>33 y.</td>
<td>X 9</td>
<td>Bilateral tremor &amp; rigidity, Akinesia, short gait, difficulty writing, own care impossible, impertinent laughing &amp; crying, antero- &amp; retropulsion, unable to work</td>
<td>£ 1</td>
<td>R.v.o.p. 10-29-59</td>
<td>£ 1</td>
<td>R.v.o.p. 10-32-39</td>
<td>No tremor, no rigidity on right side, very slight tremor on left side, markedly improved gait</td>
<td>Excellent. Gait greatly improved, own care normal, speech slightly impaired &amp; aphonic</td>
<td>Good</td>
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<td>7. F.C.</td>
<td>36 y.</td>
<td>XX 14</td>
<td>Bilateral tremor &amp; rigidity, Akinesia, right more than left, monotonic low speech, salivation, tremor lips &amp; tongue, akinesia, short gait, retropulsion, impairment eating &amp; writing</td>
<td>£ 2</td>
<td>R.v.o.p. 10-22-59</td>
<td>£ 1</td>
<td>R.v.o.p. 3-13-60</td>
<td>No tremor, no rigidity. Speech normal, gait impaired, own care normal</td>
<td>Good</td>
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<td>8. P.K.</td>
<td>65 y.</td>
<td>X 7</td>
<td>Bilateral tremor &amp; rigidity, right more than left. Short gait</td>
<td>£ 2</td>
<td>R.v.o.p. 5-14-59</td>
<td>£ 1</td>
<td>R.v.o.p. 1-14-60</td>
<td>Tremor right side, marked mental confusion, incontinence urine, slurred speech</td>
<td>Death 2 mos. after 2nd op.</td>
<td>Bad</td>
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<td>9</td>
<td>P.D.</td>
<td>X</td>
<td>9</td>
<td>Bilateral tremor &amp; rigidity, left more than right, Short gait, akinesia, complete invalid, great impairment eating &amp; writing</td>
<td>2 L.V.O.P. 5-12-59</td>
<td>Excellent</td>
<td>1 R.V.O.P. 12-15-59</td>
<td>Excellent, Speech slightly impaired, monotonous &amp; low, own care normal</td>
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<td>10</td>
<td>B.F.</td>
<td>XX</td>
<td>14</td>
<td>Bilateral tremor &amp; rigidity, right more than left, Slight psychogenic syndrome, slurred speech, short gait, unable to work</td>
<td>2 L.V.O.P. 3-17-59</td>
<td>Excellent, but gait worse, own care again possible</td>
<td>1 R.V.O.P. 11-21-59</td>
<td>Excellent, Speech improved, gait worse</td>
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<td>11</td>
<td>Da.N.</td>
<td>X</td>
<td>7</td>
<td>Bilateral tremor &amp; rigidity, left more than right, Short gait, propulsion, unable to work</td>
<td>2 L.V.O.P. 6-9-59</td>
<td>Excellent, Short gait</td>
<td>2 R.V.O.P. 1-19-60</td>
<td>Very slight tremor left hand, short gait</td>
</tr>
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<td>12</td>
<td>Sp.E.</td>
<td>XX</td>
<td>16</td>
<td>Severe bilateral rigidity &amp; tremor, Complete invalid, unable to care for himself, speech practically impossible, aphonic</td>
<td>2 L.V.O.P. 6-12-59</td>
<td>Excellent, Able to eat by himself</td>
<td>1 R.V.O.P. 1-21-60</td>
<td>No tremor, no rigidity, Transient disorientation &amp; confusion, incontinent, speech nearly normal, gait normal</td>
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<td>13</td>
<td>H.L.</td>
<td>X</td>
<td>8</td>
<td>Severe bilateral tremor &amp; definite rigidity, Short gait, writing impossible, practically complete invalid</td>
<td>1 L.V.O.P. 4-28-59</td>
<td>Excellent, Gait improved</td>
<td>3 R.V.O.P. 11-5-59</td>
<td>Marked tremor left side, otherwise normal</td>
</tr>
<tr>
<td>14</td>
<td>D.M.</td>
<td>XX</td>
<td>26</td>
<td>Bilateral tremor &amp; rigidity, right more than left, Akinesia, short gait, propulsion, monotonous, low speech</td>
<td>3 L.V.O.P. 9-16-58</td>
<td>Excellent</td>
<td>3 R.V.O.P. 11-12-59</td>
<td>No tremor, no rigidity, speech much improved, with articulation</td>
</tr>
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<td>15</td>
<td>T.A.</td>
<td>XX</td>
<td>39</td>
<td>Severe bilateral tremor &amp; rigidity. Tremor of tongue, monotonous speech, akinesis, gait practically impossible, short propulsion, complete invalid</td>
<td>2 L.V.O.P. 5-26-59</td>
<td>Excellent</td>
<td>1 R.V.O.P. 11-26-59</td>
<td>No tremor, no rigidity. 3 days after op, severe mental confusion</td>
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<td>16</td>
<td>H.A.</td>
<td>X</td>
<td>3</td>
<td>Left-sided tremor &amp; rigidity, Monotonous, well articulated speech</td>
<td>1 R.V.O.P. 6-2-59</td>
<td>Excellent, Postop amnesia 3 days, 3 mos after op, onset of tremor &amp; rigidity on right</td>
<td>2 L.V.O.P. 5-31-60</td>
<td>Excellent, No tremor, no rigidity, mental disorders, speech unimpaired</td>
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<tr>
<td>17. K.J. M</td>
<td>X</td>
<td>3</td>
<td>Weakness, clumsiness on right, writing greatly impaired, right-sided tremor &amp; rigidity, speech monotonal</td>
<td>L. v.o.p. 241-59</td>
<td>Good, no rigidity, still slight emotional tremor. Able to work 100%. 1 yr. later tremor on left, gait impaired</td>
<td>R. v.o.p. 1-12-59</td>
<td>Excellent. No tremor, no rigidity, gait worse, propulsion more marked</td>
<td>Good. Not yet at work</td>
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<td>18. H.H. M</td>
<td>XX</td>
<td>28</td>
<td>Salivation, bilateral tremor &amp; rigidity, left more than right. Gait short. Complete invalid, unable to write</td>
<td>L. v.o.p. 10-8-59</td>
<td>Good, no tremor &amp; rigidity on right, able to feed himself with right hand &amp; to write</td>
<td>R. v.o.p. 4-21-60</td>
<td>Excellent. Very slight tremor of lips, gait &amp; speech normal</td>
<td>Excellent. Not yet at work, own care normal</td>
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<td>20. Th. B. F</td>
<td>X</td>
<td>3</td>
<td>Bilateral tremor &amp; rigidity, left more than right. Unable to eat &amp; write</td>
<td>R. v.o.p. 6-23-59</td>
<td>Good, No tremor &amp; rigidity on left, gait improved</td>
<td>L. v.o.p. 4-7-60</td>
<td>Good in first postop. days. Again slight tremor on left</td>
<td>On 10th postop. day collapse, hyperthermia Died 10 days after 2nd op. Autopsy: accurately placed lesion in ventrolateral nucleus of right thalamus, but fairly large lesion on left side extending in medial thalamus</td>
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<td>21. R.K. F</td>
<td>X</td>
<td>8</td>
<td>Bilateral tremor &amp; rigidity, right more than left</td>
<td>L. v.o.p. 8-29-59</td>
<td>Excellent. No tremor &amp; rigidity on right</td>
<td>R. v.o.p. 5-10-60</td>
<td>Excellent. Still very slight emotional tremor left hand</td>
<td>Excellent. No mental impairment</td>
<td>Excellent. Able to do housework</td>
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<td>22. L.A. M</td>
<td>XX</td>
<td>18</td>
<td>Bilateral tremor &amp; rigidity, right more than left. Able to do only light work, slightly slurred speech</td>
<td>L. v.o.p. 5-13-59</td>
<td>Good. Apathetic a few days, then great improvement</td>
<td>L. v.o.p. 5-24-60</td>
<td>Good. Still very slight bilateral emotional tremor, for few wks., mental confusion</td>
<td>Probably good. No mental impairment</td>
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<td>23. M.A. M</td>
<td>XX</td>
<td>10</td>
<td>Bilateral tremor, right more than left. Unable to write &amp; feed himself, invalid, monotonous aphonic speech, tremor of lips &amp; tongue</td>
<td>R. v.o.p. 5-24-60</td>
<td>Good, No tremor on right, tremor on left worse, totally aphonic, marked athetosis right hand, gradually decreasing</td>
<td>L. v.o.p. 5-27-60</td>
<td>Good. No tremor, speech improved, slight athetosis right hand</td>
<td>Probably good. No mental impairment</td>
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X = Parkinson's disease.
XX = Postencephalitic parkinsonism.
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<tr>
<td>1</td>
<td>ch.A. M</td>
<td>59 y. Clerk</td>
<td>XX 17</td>
<td>Severe bilateral rigidity &amp; tremor, Tremor lips &amp; tongue, unable to write &amp; feed himself, aphonic speech, salivation, pros- &amp; retro- pulsion, complete invalid</td>
<td>L. Chemo- pallidectomy 11-47-57</td>
<td>Good. Great improvement rigidity &amp; tremor on right, still aphonic</td>
<td>14 R. Pallid. int. 3-18-58</td>
<td>No tremor, no rigidity. Speech greatly improved, able to walk</td>
</tr>
<tr>
<td>2</td>
<td>B.A. M</td>
<td>41 y. Clerk</td>
<td>XX 4</td>
<td>Tremor &amp; rigidity left side, Masked akinesis, short gait, impaired writing</td>
<td>R. Chemo- pallidectomy 5-21-57</td>
<td>Moderately improved tremor after 2nd injec. for 3 mos.</td>
<td>3 R. o.r.o.p. 7-10-58</td>
<td>No tremor, slight rigidity, right side. Akinesia improved, monotounous speech</td>
</tr>
<tr>
<td>3</td>
<td>Tsch.Th. M</td>
<td>54 y. Worker</td>
<td>XX 8</td>
<td>Bilateral tremor &amp; rigidity. Masked face, pros- &amp; retro- pulsion, gait short, unable to work</td>
<td>L. Chemo- pallidectomy 10-16-57</td>
<td>Excellent. No tremor &amp; rigidity right side</td>
<td>2 R. o.r.o.p. 4-1-58</td>
<td>For 4 wks. severe mental confusion, incontinence</td>
</tr>
<tr>
<td>5</td>
<td>B.P. P</td>
<td>54 y. Housewife</td>
<td>X 2</td>
<td>Bilateral tremor &amp; rigidity, right more than left. Monotonous speech, short gait, propulsion</td>
<td>L. Chemo- pallidectomy 10-21-57</td>
<td>Excellent. No tremor on right, increased weight</td>
<td>3 R. o.r.o.p. 4-24-58</td>
<td>Transitory mental confusion, homonymous hemianopsia to left, slurred speech</td>
</tr>
<tr>
<td>6</td>
<td>K.J. M</td>
<td>53 y.</td>
<td>X 8</td>
<td>Severe bilateral tremor &amp; rigidity. Tremor of head, lips &amp; tongue, monotonous speech, short gait, unable to work</td>
<td>L. Chemo- pallidectomy 7-10-58</td>
<td>Good. No rigidity &amp; slight tremor on left</td>
<td>5 R. o.r.o.p. 3-10-59</td>
<td>Improved gait, no tremor, no rigidity on right</td>
</tr>
<tr>
<td>7</td>
<td>W.M. M</td>
<td>58 y. Clerk</td>
<td>XX 9</td>
<td>Bilateral tremor &amp; rigidity, left more than right. Dysarthria, micrography, short gait, depressed</td>
<td>R. Pallid. int. 3-3-58</td>
<td>Improved rigidity &amp; tremor</td>
<td>5 R. o.r.o.p. 2-4-59</td>
<td>Transient mental confusion a few days, no tremor</td>
</tr>
<tr>
<td>8</td>
<td>K.R. F</td>
<td>50 y. Housewife</td>
<td>XX 20</td>
<td>Severe bilateral rigidity. Slight emotional tremor, akinesia, salivation, greatly impaired eating, complete invalid</td>
<td>R. Pallid. int. 3-11-58</td>
<td>Left hemiparesis &amp; hemianopsia</td>
<td>5 R. o.r.o.p. 9-25-58</td>
<td>Very slurred &amp; aphonic speech, mental confusion, no tremor, slight left spastic hemiparesis</td>
</tr>
<tr>
<td>9</td>
<td>H.A. F</td>
<td>68 y. Housewife</td>
<td>X 6</td>
<td>Severe bilateral tremor &amp; rigidity. Unable to walk, invalid</td>
<td>L. o.r.o.p. 4-3-58</td>
<td>Somnolent, confused, incontinence, some improvement tremor</td>
<td>2 R. Pallid. int. 6-26-58</td>
<td>Gait worse, short, rigidity improved, tremor still present</td>
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<td>11. R.M. F 58 y. Housewife</td>
<td>X</td>
<td>5</td>
<td>Bilateral tremor &amp; rigidity, left more than right. Stiff &amp; short gait, pro &amp; retropropulsion, practically invalid</td>
<td>2 R.s.o.p. 5-12-58</td>
<td>Excellent. No tremor &amp; rigidity on left</td>
<td>16 L. Pallid. int 1-12-59</td>
<td>Slight emotional tremor on right, apathetic, dull</td>
<td>Slight psycho-organic syndrome, able to write, own care normal, speech normal</td>
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<tr>
<td>12. W.R M 63 y. Carpenter</td>
<td>X</td>
<td>98</td>
<td>Bilateral tremor &amp; rigidity, right more than left. Tremor of head &amp; lips, salivation, writing impaired, own care impaired, short stiff gait, invalid</td>
<td>12 L. Pallid. int 3-6-58</td>
<td>Improved tremor &amp; rigidity on right. Salivation improved</td>
<td>4 R.s.o.p. 3-20-58</td>
<td>Weakness left arm, tremor &amp; rigidity unchanged</td>
<td>Psycho-organic syndrome, left hemiparesis, compulsory crying, slurred speech, gait worse</td>
</tr>
<tr>
<td>14. D.A. M 40 y. Merchant</td>
<td>X</td>
<td>5</td>
<td>Bilateral severe rigidity, right more than left. Very slight tremor right hand, impaired writing, short stiff gait, propulsion</td>
<td>5 L. Pallid. int 5-12-58</td>
<td>Transient apathy, improved rigidity</td>
<td>5 R.s.o.p. 1-8-59</td>
<td>Greatly improved gait, writing &amp; speech</td>
<td>No mental impairment</td>
</tr>
<tr>
<td>15. G.M. F 48 y. Housewife</td>
<td>XX</td>
<td>7</td>
<td>Bilateral tremor &amp; rigidity, left more than right. Mentally dull, stiff short gait, own care impaired</td>
<td>4 R.s.o.p. 12-13-58</td>
<td>No tremor &amp; rigidity right. Worse, washing impossible</td>
<td>10 R. Pallid. int 6-12-59</td>
<td>Transient confusion, improved writing &amp; speech</td>
<td>No mental deficit, gait still impaired</td>
</tr>
<tr>
<td>16. H.H. F 58 y. Housewife</td>
<td>XX</td>
<td>11</td>
<td>Severe bilateral tremor &amp; rigidity. Tremor lips &amp; tongue, psycho-organic syndrome, monotonous speech, short gait, invalid</td>
<td>18 L. Pallid. int 10-23-58</td>
<td>Improved tremor &amp; rigidity on right. Speech improved</td>
<td>1 R.s.o.p. 4-16-59</td>
<td>No tremor, no rigidity, Greatly improved speech &amp; gait</td>
<td>No mental impairment, increased weight, able to work</td>
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<td>18. Sch.K. F 28 y. Housewife</td>
<td>X</td>
<td>7</td>
<td>Bilateral tremor, right more than left &amp; slight bilateral rigidity. Short gait, pro &amp; retropropulsion</td>
<td>2 L.s.o.p. 7-3-58</td>
<td>Psycho-organic syndrome, no tremor right side</td>
<td>15 R. Pallid. int 7-5-59</td>
<td>Transient mental confusion, slight left hemiparesis</td>
<td>Apathetic, aphasis speech, mentally impaired, later improved</td>
</tr>
<tr>
<td>19. F.E. M 31 y. Laborer</td>
<td>XX trauma</td>
<td>7</td>
<td>Bilateral tremor &amp; rigidity, right more than left. Shunted speech, short stiff gait</td>
<td>2 L.s.o.p. 5-27-59</td>
<td>No tremor &amp; rigidity on right</td>
<td>1 R. Pallid. int 1-9-59</td>
<td>No tremor, no rigidity</td>
<td>Speech worse, own care possible, salivation</td>
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<td>21. S.J. P 41 v. Housewife</td>
<td>X</td>
<td>5</td>
<td>Bilateral rigidity. No tremor. Slow monotonous speech, salivation, short gait, pro- &amp; retropulsion</td>
<td>3</td>
<td>R. Pallid. int. 7-44-58</td>
<td>Rigidity greatly improved on left</td>
<td>3</td>
<td>L. o.o.p. 2-3-59</td>
</tr>
<tr>
<td>23. S.F. M 50 v. Painter</td>
<td>XX</td>
<td>13</td>
<td>Severe bilateral tremor &amp; rigidity, right more than left. Gait impairment, dragging right foot, propulsion, unable to work 10 yrs.</td>
<td>8</td>
<td>L. o.o.p. 5-19-59</td>
<td>No tremor, no rigidity on right</td>
<td>1</td>
<td>R. Pallid. int. 9-22-59</td>
</tr>
<tr>
<td>24. F.A. M 60 v. Mechanic</td>
<td>XX</td>
<td>12</td>
<td>Bilateral tremor &amp; rigidity, left more than right. Hyperalivation, aphonic speech, pro- &amp; retropulsion</td>
<td>8</td>
<td>R. o.o.p. 4-8-59</td>
<td>No tremor, no rigidity on left</td>
<td>3</td>
<td>L. Pallid. int. 8-20-59</td>
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<td>25. C.A. P 50 v. Housewife</td>
<td>XX</td>
<td>20</td>
<td>Bilateral tremor &amp; rigidity, right more than left. Hyperalivation, monotonous speech, stiff gait, oculogyric crisis, unable to work</td>
<td>3</td>
<td>L. o.o.p. 2-15-59</td>
<td>No tremor &amp; rigidity on right</td>
<td>6</td>
<td>R. Pallid. int. 9-17-59</td>
</tr>
<tr>
<td>26. U.N. M 45 v. Locksmith</td>
<td>XX</td>
<td>7</td>
<td>Severe bilateral rigidity &amp; some tremor. Hyperalivation, akinesia, dystrophic speech, short gait, pro- &amp; retropulsion, own care greatly improved, complete invalid 3 yrs.</td>
<td>2</td>
<td>R. o.o.p. 7-7-59</td>
<td>No tremor &amp; rigidity on left. Speech &amp; gait greatly improved</td>
<td>3</td>
<td>L. Pallid. int. 3-29-59</td>
</tr>
<tr>
<td>27. M.E. F 34 v. Housewife</td>
<td>XX</td>
<td>5</td>
<td>Bilateral tremor &amp; rigidity, left more than right. Monotonous speech, propulsion, short gait</td>
<td>2</td>
<td>R. o.o.p. 4-7-59</td>
<td>No tremor &amp; rigidity on left</td>
<td>2</td>
<td>L. Pallid. int. 10-8-59</td>
</tr>
<tr>
<td>28. A.A. M 39 v. Electrician</td>
<td>X</td>
<td>7</td>
<td>Bilateral rigidity, right more than left &amp; some tremor. Monotonous speech, short gait</td>
<td>9</td>
<td>L. Pallid. int. 9-29-58</td>
<td>No rigidity on right</td>
<td>2</td>
<td>R. o.o.p. 11-26-59</td>
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X = Parkinson's disease.
XX = Postencephalitic parkinsonism
per cent) with permanent abolition of tremor and rigidity. Two patients still showed some tremor on one side, and 1 patient showed a very slight emotional tremor on both sides. In 3 cases the tremor occurred again on one side after the second operation, and in 1 case a slight athetosis of the right hand persisted. Impairment of speech was the most striking complication after the second operation in our series. In 7 cases (1, 2, 4, 5, 6, 8, 9) speech was slightly or considerably worse, slurred, low or fully aphonie. In 7 other cases (7, 10, 12, 14, 17, 19, 23) it was markedly improved by the bilateral thalamotomy; in Cases 12 and 14 the recovery of speech was even spectacular. In all other cases speech was unchanged. The gait was more impaired in 4 instances (4, 7, 10, 17) but slightly or considerably improved in 10 cases (1, 3, 5, 6, 9, 10, 12, 13, 14, 19). One patient (Case 20) died suddenly on the 10th postoperative day from cardiovascular failure and hyperthermia. Autopsy showed that at the second operation the coagulation had produced a fairly large lesion extending from the ventrolateral into the medial thalamus.

Psychomotor disturbances and altered consciousness were present in 8 cases (1, 2, 5, 8, 12, 15, 19, 22); in 6 instances (1, 2, 5, 12, 19, 22) they were only slight and transitory, but in 2 cases (8 and 15) they were serious and long-lasting; in 1 of these 2 cases (15), however, the time of postoperative clinical observation was too short for a definite evaluation. The serious transient confusional state in Case 1 was not the consequence of the surgical intervention but of the cerebral injury from which the patient had been suffering. Case 8 is the only instance in which transient mental confusion already was observed after the first operation. Against our advice, the patient as well as her relatives insisted on the operation on the other side. After this last operation a severe change in personality took place and the patient died 2 months later at home.

As to end-results, there are 12 patients (52 per cent) able to work again, some of them to full capacity. In 8 further cases there was considerable improvement in self-care of the patient. But 2 patients showed a poor result because of severe psychical changes; 1 of these died 2 months later. The other died on the 10th postoperative day from a fairly large, probably thrombotic lesion, extending from the ventrolateral into the medial thalamus.

b) In our second series of 28 combined bilateral operations, i.e., pallidotomy on one side and thalamotomy on the opposite (Table 2), tremor and rigidity were abolished permanently in 14 cases (50 per cent). In the remainder there was still some emotional tremor on one side or some rigidity on one or both sides. In 6 cases the gait was slightly or considerably improved, but in 2 instances it was worse after the second operation.

The influence on speech also is a striking feature in this series of bilateral operations. In 17 cases (2, 3, 4, 5, 6, 8, 10, 12, 13, 17, 18, 19, 20, 21, 23, 25, 26), i.e., in over half, speech was slightly or considerably worse than before the second operation; it was more slurred, dysarthric, or low and aphonie. Of these cases, there is only one (17) in which speech already was worse after the first operation. However, there are also several cases (1, 14, 15, 16, 24, 26) in which speech was slightly or considerably improved by the bilateral operation. This improvement was in many instances associated with an improvement of small script.

Psychomotor disturbances, altered consciousness and a more or less marked psychoorganic syndrome have been observed in 17 cases (1, 2, 3, 4, 5, 7, 8, 11, 12, 13, 15, 18, 20, 21, 22, 23, 25). Fortunately, these changes have been transient in 3 cases (5, 7, 15), and in 3 other cases the definite psycho-organic syndrome was of a slight or moderate degree (1, 3, 11). But in 6 cases the mental impairment was serious and in 2 cases (4 and 22) death occurred 8 months and 5 weeks, respectively, after the second operation. Both patients, however, had very advanced parkinsonism, with a high degree of rigidity and difficulty in speech and swallowing. In Case 13 the psycho-organic syndrome was associated with compulsory crying. In 3 cases (20, 23, 25) the psychic alteration was so
marked that the patient was incapable of
resuming an occupation in spite of a nearly
normal neurological state. Of these cases,
transitory mental confusion already had been
observed in 2 instances (4, 18); but on the
other hand, the psychical changes that oc-
curred after the first operation in Cases 9
and 14 were not repeated after the second
surgical intervention and there was no mental
impairment whatsoever.

The end-results were excellent in 8 cases,
i.e., 28.5 per cent of the patients obtaining
practically full working capacity. In 9 cases
the result was good with considerable im-
provement of the self-care of the patient and
some degree of working capacity, up to 50
per cent. Five cases showed a moderate and
6 cases a bad result. In the latter group were
2 deaths, 1 from pseudoparalysis combined
with respiratory disorders, and the other
from general cachexia.

DISCUSSION

The two series of bilateral stereotaxic oper-
ations reported here demonstrate that severe
psychical disturbances have been far more
frequent in the second than in the first group.
The postoperative psychical syndromes of
the second group have been more frequent
in cases of postencephalitic parkinsonism (11
cases) than in cases of Parkinson's disease
(6 cases); and no correlation between age of
patient and syndrome could be established.
Psychical deterioration occurred in 11 cases
of thalamotomy (as second operation), and
in 6 cases of pallidotomy (as second opera-
tion). In our first group of bilateral thalamot-
omy the psychical disturbances were as fre-
quent in postencephalitic parkinsonism (4
cases) as in Parkinson's disease (4 cases).
When the first operation is followed by se-
vere psychomotor disturbances or altered
consciousness, it is not justifiable to perform
a bilateral stereotaxic procedure. This has
already been stated by Hassler and Riechert.\(^6\)
In addition we hope to get further informa-
tion from the electroencephalographic find-
ings after the first stereotaxic lesion. Accord-
ing to the still limited experience of our co-
worker, R. Hess, it seems that intermittent
delta-rhythms after the first and before the
second operation indicate a poor prognosis
for a second operation. This is in agreement
with the findings of Hassler et al.\(^6\) who stated:
"The majority (86.6 per cent) exhibited
localized delta activity of different intensity
over the fronto-parietal or fronto-temporal
region on the side of coagulation." In both
groups of bilateral operations a striking fea-
ture is the influence on speech. There may
be an improvement, but more often an im-
pairment of speech. No definite correlation
could be found with regard to site or side of
operation. The disorders of speech have not
been accompanied by pyramidal-tract signs
and there is no correlation with right- or
left-handedness. They are characterized by a
more or less pronounced loss of intonation
and by some degree of dysarthria. This com-
plication of speech in the course of bilateral
operations is surprising because in these cases
muscular rigidity had been abolished. The
disability of speech in parkinsonism certainly
may be attributed to muscular rigidity, ac-
cording to Walshe.\(^19\) It is described as loss
of inflections in the voice, the weakness of
phonation and the blurring of articulation.
Our experiences demonstrate that this dis-
order of speech may be present or absent in
spite of the fact that we have rendered the
muscles hypotonic. Penfield\(^14\) has proposed,
as an hypothesis of speech, that the functions
of all three cortical areas of speech in man
are coordinated by projections of each one
to parts of the thalamus, and that by means
of these circuits the elaboration of speech is
somehow carried out. It seems to us that
these bilateral surgical experiments on the
basal ganglia give support to such a concep-
tion.

Comparing these two series of bilateral
stereotaxic operations, we conclude that in
well-selected cases an accurately placed, bi-
lateral lesion of a small, but sufficient size
produces no neurological or intellectual defi-
cit. It is our experience that the thalamic
lesion has a more nearly certain and com-
plete effect on contralateral resting tremor
and rigidity than is the case with the pallidal
lesion. Therefore, today we give preference to
the operation that places the lesion in the ventrolateral nucleus of the thalamus and interrupts all the pallidothalamic and cerebellothalamic fibres, and this also for the second surgical procedure, i.e., the bilateral thalamotomy. Such a conclusion has already been anticipated by Martin:10 “The operation which has interested me more is one which places a lesion at (or near) the ventral portion of the thalamus. This operation ... must interrupt all the pallido-thalamic fibres—both those in the fasciculus lenticularis and those in the ansa—and thus block the whole outflow of impulses from the globus pallidus (with the possible exception of some to the hypothalamus, or its neighbourhood).”

SUMMARY

Experiences with 51 bilateral stereotaxic operations on the basal ganglia are reported. Contrary to the statements in the literature, the results of bilateral thalamotomy are superior to those of combined pallidotomy and thalamotomy. The best results seem to depend mainly on three factors:

1. Well-selected patients without advanced cerebral deterioration.
2. Normal electroencephalographic findings 6 months after the first surgical intervention.
3. Small and accurately placed stereotaxic lesion which is obtained better in thalamotomy than in pallidotomy.

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