

## PARKINSONISM – A REVIEW

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### INTRODUCTION

The purpose of this article is to set forth a concise discussion of Parkinsonism; yet in doing so, we also hope to reveal the magnitude and the complexity of the disease.

James Parkinson, a practicing physician in London, England from 1785-1824 was the first man to describe the classical form of the disease also known as "Paralysis Agitans" in 1817 (*An Essay on the Shaking Palsy*). The first symptoms of paralysis agitans appeared in him in 1814 and as a result, his article is not only composed of objective observations, but also subjective ones.

### AETIOLOGY

The aetiology may be divided into three main types - 1) Idiopathic Parkinsonism, 2) Post-Encephalitis Parkinsonism, and 3) Symptomatic Parkinsonism.

1) **Idiopathic Parkinsonism**, or paralysis agitans, occurs between the ages of 40-70 years, with an average onset of 53 years in the male and 60 years in the female. The Juvenile type has a strong familial tendency, and may have an onset as early as seven years of age. (Onuaguluchi, G. 1964). The aetiology of paralysis agitans is in essence unknown, and it accounts for over 50% of the Parkinsonism cases. (Garland, H. G. 1952).

2) **Post-encephalitic Parkinsonism** as its name implies, is a sequela of Encephalitis Lethargica. Parkinsonism however, may follow Encephalitis Lethargica immediately, after a long time, or not at all. 30% of Parkinsonism cases may be attributed to this condition.

3) **Symptomatic Parkinsonism** may be caused by many factors. Carbon monoxide poisoning, manganese poisoning, or lesions in the extra pyramidal system especially in the mid brain, such as brain tumors, syphilis or trauma.

Patrick and Levy (1922) showed a history of trauma in 22 of 146 cases of Parkinsonism, excluding from the sample those cases due to Encephalitis Lethargica. A virus is even implicated in the aetiology of Parkinsonism which occurs in a small percentage of Behçet's Syndrome. A disruption of the blood supply to cortical veins of the extra-pyramidal system, as with arteriosclerosis, or endarteritis as sometimes happens with syphilis, may both cause Parkinsonism.

The **Phenothiazine** group of drugs may induce a temporary Parkinsonism which will generally disappear with withdrawal of the drug. (Munch-Peterson, S., 1956).

### ECOLOGY AND EPIDEMIOLOGY

Parkinsonism is world wide, but some areas have a greater incidence than others. In the U.S.A. Doshay (1961) estimated that 1 in 200 people have Parkinsonism while Kurland estimates that 1 in 40 will develop the disease. Merritt (1963), Garland (1952), estimated that the incidence in the U.K. is 1 in 1800 and in Sweden, in the population over 50 years of age, the incidence is 1 in 625.

Race and heredity seem to play a part in the incidence of the disease, as illustrated in the U.K. where there is a greater incidence of Parkinsonism among those of Anglo-Saxon descent. There is a higher incidence of Parkinsonism in Europe and North America than in West Africa, but obviously there are environmental, and life expectancy factors involved here, as well as heredity. (Critchley, M. 1955).

Mjones of Sweden, Erb and Gowers all consider heredity to be of significance. The latter two authorities estimated that familial tendencies are apparent in 15% of the cases. One report estimates this incidence to be as high as 38%. (Merritt, H. H. 1963).

The sex ratio shows a male predominance of 5: 3.5 (Ironsides 1952) with an average male onset seven years earlier.

### PHYSIOLOGY

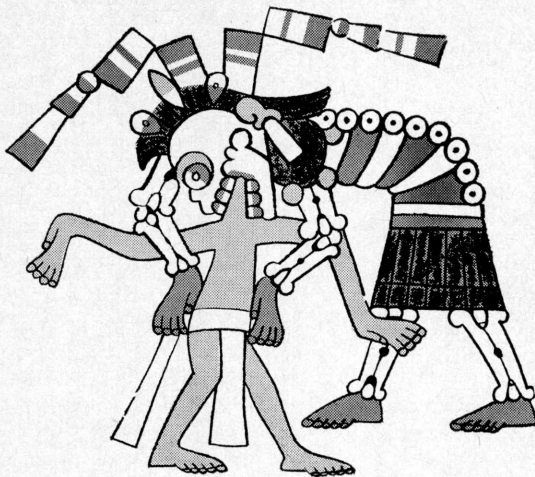
Parkinson's Disease is classified as a disease of the extra-pyramidal system, which

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manifests itself classically as tremor weakness and rigidity of skeletal muscle.

The muscle tone is controlled at different levels of the nervous system. Postural muscle tone is effected by a reflex arc from muscle spindles through segmental origin in the spinal cord. Movement is elicited by the alpha fibers, and there is also postural tonic interaction from the gamma fibers to modify this segmental reflex tone. This movement control and reflex system involves two efferent fibers to the muscle. One is the alpha fiber, or muscle-effector fiber, which is a direct continuation of the pyramidal system, and secondly, the extra-pyramidal projection to the intrafusal fibers of the muscle spindle. The annulospiral fiber is afferent and joins the annulospiral area of the spindle, via the dorsal root, to impinge upon the perikaryon of the muscle effector fiber, first mentioned. In this reflex, the efferent fiber to the intrafusal area of the spindle is of the gamma type, but the afferent annulospiral fiber is alpha. Rostrally, the extra-pyramidal system is split into two main functional classes: the inhibitory mechanism and excitory mechanism. Schreiner, Lindsley and Magoun (1949) showed that the reticular formation augmented cortical motor impulses, by way of the reticulo-spinal tract. Magoun (1950) later placed the inhibitory mechanism medial to the excitory fibres in these areas.

The inhibitory area is confined to the bulbar region of the reticular formation. This inhibitory system receives fibers from the cortex areas 4s, 24s, and 19s, the caudate, tectum and cerebellum. (Peterson, Magoun, McCulloch, and Lindsley, 1946). The areas of the reticular formation extend through all levels of the brain stem, (Lindsley, Schreiner, and Magoun, 1949) and are connected to area 6, sensory motor associational areas, limbic, and auditory areas of the cortex. The influence of these connections seems to be bilateral.

It is important, however, to go another step and look at the areas which send impulses to the highest areas of the extra pyramidal system, for it is these areas at which surgery is aimed. This will be dealt with later in this paper.

Rigidity therefore is caused by elevated fiber impulses acting on the spindle reflex. Suda, Koizumin and Brooks (1958) showed a descending discharge with reticular formation

stimulation. This indicated the pathway from the reticular formation which modulates the reflex activity of the muscle spindle.

The physiology of tremor is poorly understood. James Parkinson in 1817 was the first to operate the tremor of "Paralysis Agitans" from other types of tremor and since then various authorities have attributed it to various different causes in different areas.

Meyers (1940) reported a Parkinsonian tremor decrease when the head of the caudate was resected. He considered the resection was an interruption of impulses from the pre-frontal and premotor areas, because he showed that E.E.G. rhythm of 4 per sec. coming in spindles from these areas were synchronous with exaggerated phases of the tremor. Minkler and Klemme (1943) excised the premotor cortex and also relieved the Parkinson tremor. The reason for this was thought to be a connection of the reticular formation to the premotor cortex.

Ward, McCulloch and Magoun (1948) showed that a lesion below the basal ganglia destroying part of the pontine and mesencephalic tegmentum in monkeys will produce tremors at rest, and E.M.G. studies of these monkeys by Peterson, Magoun, McCulloch and Lindsley (1949) showed them to be regular alternation of the antagonist and agonist muscles at 6-8 oscillations per sec. This movement disappeared with action, but their findings were not constant because the lesions varied. If the superior cerebellar peduncle was involved by the lesion, then action tremor was observed as well.

Jenker and Ward (1953) found that tremor-like movements, induced by high frequency stimulation of the medial reticular formation, were reduced or abolished by anticholinergic drugs. Their hypothesis was that interruption of the connections from the cortex and basal ganglia to the reticular formation caused the tremor of Parkinsonism, and the deafferented neurons become hypersensitized to acetylcholine and therefore this hypersensitivity was reduced by anticholinergic drugs.

Hassler, Reichert, Munding, Umbach and Gangleberger (1960) found that stimulation of the pallidum sometimes increased, and sometimes blocked the tremor, and that the ventral medial nucleus of the thalamus also affected this tremor. Cooper and colleagues (1958) showed abolition or reduction in tremor



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with 45% of cases treated with chemopallidectomy or electrocoagulation of the globus pallidus, but chemothalamectomy or electrocoagulation of the thalamus abolishes tremor in 70%. Tremor is then, regular alternating antagonistic muscular contractions, which are increased with adrenalin, or with an increase in emotion. For instance, when the patient knows he is being watched. The tremor ceases during sleep, and cannot be induced in experiment animals by lesions in the striopallidum. However, induction of tremor may be evoked by lesions of the reticular formation, and so the tremor may be ascribed to the associated degenerative lesions in the reticular formation and its ascending connections. (Samson Wright, 1961).

The weakness, the third of the classical symptoms, results in early defects of fine movements of the fingers, and lack of facial expression. This weakness is often accompanied by rigidity, but it is not certain that rigidity is the sole cause of this weakness. (Onuaguluchi, G. 1964).

Barbeau, Murphy and Sourkes (1961) suggested that the cause of Parkinsonism is a metabolic defect. They demonstrated a significant decrease of urinary excretion of dopamine in patients with Parkinsonism compared to the normal, and that dopa administered to Parkinsonian patients temporarily reduced their rigidity. When dopa was administered it was noticed that Parkinsonian patients excreted less dopamine than the normal control group, and from this it was suggested that these patients may be deficient in dopa decarboxylase. Barbeau (1961) and Horykewicz (1962) showed that the concentrations of dopamine and 5-hydroxytryptamine in the basal ganglia of the patients are significantly below normal.

McGreer and Zeldowicz (1964) tried to treat Parkinsonism with dihydroxyphenylalanine, with little success, but patients treated with alpha methyl dopa did show an improvement. (March, Schneider, and Marshall 1963).

From the evidence of the concentration dopamine and 5-hydroxytryptamine in the basal ganglia and in the urine, and the fact that anticholinergic and antihistamine drugs benefit Parkinsonian patients, as described later in this paper, it is interesting, to consider the possibility of a dual, but inter-related system of the control, because occasionally

a patient may manifest solely, rigidity or tremor. Perhaps an abnormal inter-relationship between a dual system of control would produce such symptoms.



A Lewy Body in the Subthalamic Nucleus seen in the middle of this plate.

(Courtesy of

Dr. A. J. Lewis, Dept. Pathology, Dalhousie University)

## PATHOLOGY

James Parkinson himself had no knowledge of the pathology of his disease, and it was not until 70 years after his death that Blocq and Morinesco (1894) demonstrated a case of unilateral Parkinson's Disease. The 38 year old male patient had an olive-sized tuberculoma, which destroyed principally the right substantia nigra. Holmes (1904) was the first to consider that lesions in the substantia nigra were almost invariably associated with Parkinsonism.

The lesions in the substantia nigra were not constant. There is general atrophy, and shrinkage of the cell bodies, with neuroglial scarring in areas of neuron degeneration, especially around vessels. Concentric hyaline cytoplasmic inclusion material is seen in the substantia nigra, dorsal vagal nucleus and sub-thalamic nuclei. These are Lewy Bodies (see plate above). Lewy (1914) also described a shrinkage of the caudate nucleus, putamen and globus pallidus. Lesions in the substantia nigra may produce atrophy in the

globus pallidus, which in turn may affect the putamen and caudate nucleus, due to the important fiber tracts which project to the substantia nigra, via the globus pallidus, from the caudate and putamen.

Foix and Nicolascio (1925) summed up their essay on "The Pathology of Parkinson's Disease" by describing two main types of lesion. Firstly, there is a diffuse type, very similar to changes found with senility in the premotor, and also cerebellar cortex. Secondly, there are localized lesions in melanin-bearing cells of the brain stem. These observations are in agreement with the subsequent studies of the subject up to the present day.

## CLINICALLY SYMPTOMS

The early symptoms of Parkinsonism are of an insidious onset, and the subjective manifestations are thought to be due to muscle rigidity. These include mild diffuse aches in the muscles, a slight disparity in the swiftness of motion and a mild stiffness. Added to this, may be the complaint of some loss of dexterity in performing small coordinated movements. There is an extreme variability in the muscle groups of the body which are involved in the initial stage. Usually, however, the progression of symptoms is from one extremity to the ultimate involvement of the total body musculature, taking a period of several months to several years. Occasionally, the presenting complaint is that of tremor - usually in one extremity.

It is important to note, that the problem in diagnosis of Parkinsonism occurs at this stage. It is not an arduous task to make the diagnosis in the face of the fully developed clinical picture which will be discussed subsequently. By this time the symptoms are quite evident from these clinical signs.

## SIGNS

The cranial nerves dealing with motor function are all at least somewhat involved, especially in the advanced cases of Parkinsonism.

### CRANIAL NERVES III, IV, VI

Involvement of the eye muscles causes a degree of impaired convergence in some cases of Parkinsonism. Also the eye movements may be limited in range and the movements

themselves may be slightly jerky (described by Merritt as "cog-wheel rigidity of the eye muscles").

Oculogyric crises occur in about 16% of Parkinsonism patients. Women are affected about twice as commonly as men. The clinical features of this condition are essentially divided into two stages. The Prodromal Stage, during which the patient experiences some degree of emotional change, which is always unpleasant and accompanied by nystagmus. The Stage of Tonic Deviation or Rotation of the Eyes follows. In the majority of cases the eyes are rotated directly upwards and laterally. This is accompanied by an increase in the muscular rigidity and a characteristic positioning of the head. Also there is a simultaneously occurring tachycardia and a significant increase in blood pressure. The patient may appear demented and emit moans or plaintive cries. Onuaguluchi (1961) has proposed a Vestibular Ocular-Reflex Theory of pathogenesis.

### CRANIAL NERVE VII

The facial expression is fixed and classically described as "mask-like facies" and attributed, at least in part, to muscular rigidity. Emotional changes in facial expression such as smiling, take a longer time to be executed, appear inadequate and remain fixed for a longer period of time than normal. There is also an infrequency of the normal involuntary blinking movements of the eyes and as a result the patient may appear to be staring. Myerson's Sign (spasms of the orbicularis oculi muscles or repeated blinking movements may be evoked if a finger is thrust quickly towards the eyes or if the bridge of the nose is tapped with a reflex hammer) may usually be elicited.

### CRANIAL NERVES V, VII, IX, X, XII

The speech may be monotonous and the words may be enunciated more quickly - in some cases the volume of the voice is greatly lessened. Normal, involuntary deglutition movements are decreased in frequency and as a result, the patient may "drool" saliva but there is usually little if any, dysphagia with solid or liquid foods.

The Motor System is affected by 3 characteristic features of Parkinsonism:

(1) Rigidity - is present in practically all cases of the disease. It varies in its severity from case to case. As mentioned before,

the generalized weakness that the patient experiences is at least in some degree caused by the rigidity. Face, neck and trunk muscles as well as the larger proximal muscles of the limbs are the most severely affected. This rigidity is classically described as being of the "cog-wheel" or "lead pipe" type - there is a resistance to passive movement of the joints. Rigidity is most marked in Post-Encephalitic Parkinsonism. The muscles of respiration are often involved in the rigidity and weakness found in Parkinsonism. As a result, respiratory effort as well as coughing is impaired. This accounts for the predisposition these patients have for pulmonary infection.

(2) Akinesia - may be described as a sudden interruption or onset of purposeful movements. For example, when the patient is eating, his movements may be fixed into several random stages of no apparent design. Handwriting may be affected in much the same manner and in the more severe cases is impossible. Another point is that there is difficulty in performing two or more actions simultaneously.

(3) Tremor - is classically described as being of the "pill-rolling type", having a frequency of about 3-6 per second and is produced by alternating movements of the agonist and antagonist muscles at the finger joints. There may also be a tremor of the wrist or even of a whole limb. Characteristically, the tremor is present when the limb is at rest, disappears when a voluntary movement is initiated and usually returns as soon as the movement is completed. Dramatically, the tremor ceases in sleep. Tremor is most prominent in Idiopathic Parkinsonism and may only be slight in Post-Encephalitic Parkinsonism.

In the early stages of the disease, posture and gait may be normal. As the disease progresses, however, the gait becomes shuffling, slow and is effected in small steps. The arms remain stationary. In some cases the patient shuffles along in small short paces as if hurrying to catch up with his center of gravity. This is called the Festinant Gait of Parkinsonism. The disturbances in gait in this disease may be explained in terms of the akinesia, rigidity and generalized weakness present. The body posture is usually in the general attitude of flexion in the well advanced case.

Contracture deformities of the limbs such as main d'accoucheur and plantar flexion of

the foot occur in a number of cases. Their severity is thought to be proportional to the amount of rigidity that the patient exhibits. Deformities of the spine such as scoliosis and lateral flexion of the neck also occur.

**Sensory System:** There is no loss of cutaneous or deep sensibility.

**Reflex Status:** This is usually normal even in the presence of excessive rigidity.

**Autonomic Nervous System:** There may be some heat intolerance especially in Post-Encephalitic Parkinsonism and hyperhidrosis is not uncommon. Sweating crises occur in a small percentage of patients.

**Mental State:** The disease process does not affect the patient's intellect from the functional point of view. Due to the diminution in facial expression, dysarthria and impaired handwriting, communication is made difficult. Sleep disturbances such as insomnia and reversal of sleep patterns occur. There is a certain predilection for depressive and paranoid psychoses.

**Investigations:** There are few, if any, abnormal results of Laboratory investigation in Parkinsonism. Electromyographic studies may be of diagnostic value in early or doubtful cases of the disease. Subclinical rigidity and slight tremor may be ascertained by this procedure. There is no pathognomonic electroencephalographic record in Parkinsonism. The EEG may be normal or abnormal in the disease. The degree of abnormality has been found to be related to the amount of rigidity and akinesia exhibited by the patient. Frankly abnormal EEG records indicate that poor results may be expected from both drug and surgical therapy.

## DIAGNOSIS

The diagnosis of Parkinsonism is not difficult in the well advanced case. As previously mentioned, the difficulty arises in the early stages of the disease. The differentiation between Idiopathic, Symptomatic and Post-Encephalitic forms of Parkinsonism often is quite difficult. Usually, however, the Post-Encephalitic form may be recognized from a history of a preceding acute febrile illness, a description of which is in keeping with Encephalitis Lethargica. A causal history in Symptomatic Parkinsonism is commonly extremely hard to elicit. Differentiation

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between Arteriosclerotic and Idiopathic Parkinsonism is to be discouraged, as many authors feel that they are synonymous.

Other conditions that may mimic Parkinsonism and must be excluded are: Wilson's Disease (Hepatolenticular Degeneration), Hemiplegia (due to an intracranial tumor), Disseminated Sclerosis, Senile Tremor, Heredo-Familial Tremor and in some cases Myxedema.

### COURSE AND PROGNOSIS

The course of Parkinsonism is implacably progressive from the initial non-specific symptoms until the patient is so incapacitated that he cannot fulfill a useful occupation. The length of time for the incapacitating symptoms to evolve is usually about 10 to 15 years. Since its average age of onset is in the 5th decade, the disease has little tendency to shorten life. Death usually results from intercurrent illness such as pneumonia. A certain proportion die from cerebrovascular accidents.

Drug treatment and surgery are essentially palliative. There is no curative treatment, as the cause or causes of the disease are not known.

### MEDICAL TREATMENT

Doshay and Boshes (1960) consider there to be three essential forms of Medical treatment for Parkinson's Disease: - Drug Therapy, Physical Therapy and Exercise.

The aims of therapy are: - (1) To delay and minimize the effects of the disease. (2) To prevent development of neuromuscular disabilities, and permanent deformities. (3) To preserve functional activity of the patients. Many complications can be prevented if treatment is instituted at an early stage, hence it is apparent that an early diagnosis is important, as mentioned previously.

Drug therapy is for symptomatic relief and is therefore not an ideal form of therapy. The drugs are aimed at alleviating the patient of tremor and rigidity. For this, two types of drug action are required. Firstly, an antihistamine effect, and secondly a central anticholinergic action. Either or both drug types together may be helpful, but result in only a moderate improvement in 60-80 % of the patients treated. (Goodman, L. S. and A. Gillman 1965).

Up to 1945 Belladonna alkaloids were the most frequently used drugs in this disease, but as would be expected, patients suffered all the side effects of an antimuscarinic drug. Since 1945 many synthetic preparations have made an appearance attempting to reduce the unwanted reactions. All the following drugs mentioned here, are long-term preparations, but with various side effects. Their unfortunate effects may be minimized by manipulating the combinations and dose of the preparations used.

Benzhexol, Cycrinine Biperiden and Procyclidine, are similar drugs in structure and action. The best initial drug to use in Parkinsonism is probably Benzhexol, but 5-10% of patients cannot tolerate the muscarinic type of side effects, in which case another drug in this group may be tried. Mental disturbances have also been reported as side effects of Benzhexol. (Porteous, and Ross 1956). Benzhexol is thought to be an acetylcholine blocker in the CNS. The initial dose of Benzhexol (Trihexyphenidyl) should be 1 or 2 mgs. b.i.d. increasing to 2 mgs. t.i.d. or q.i.d.

Orphenadrine and Chlorphenoxamine are similar chemically and pharmacologically. They tend to reduce the rigidity, but with little effect on the tremor. The drugs also have an euphoric effect which is useful to boost a patient's morale. Orphenadrine gets good results with 60% of Parkinsonian patients. (Onuaguluchi, G. 1964). Doses for both drugs should be initially 50 mgs. t.i.d. increasing to 100 mgs. t.i.d. if necessary.

Ethropropazine is an anticholinergic drug with some antihistamine activity, which is effective against tremor and rigidity. Some clinicians consider this to be the best available control of tremor. The initial dose is started at 40 mgs. per day. Total daily doses have been as much as 1 gm.

Benzotropic Mesylate was synthesized to combine antihistamine and antimuscarinic activity, and is useful for all types of Parkinsonism, especially against tremor. Small, infrequent doses are given because of cumulative effects. Initially, a single daily dose of 0.5 mgs. to 1 mg. is used. This may be increased by daily increments of 0.5 mgs. until the required effects are obtained. The dose rarely exceeds 8 mgs. per day. (Goodman, L. S. and A. Gilman 1965).

Physical therapy is useful for rigidity, but virtually valueless for the 10% of Parkinson patients who manifest tremor only. The modalities of physical rehabilitation include thermotherapy, electrotherapy, massage, and various types of exercise for the retention of functional activity. Even though the treatment is directed at muscular health rather than the comfort of the patient, it is of extreme importance that the patient be continually reassured and encouraged in every possible manner. (von Werssowetz, O. F. 1964)

Exercise, the third of Doshay's "Three Essentials", is designed to increase the motor performance by the improvement of gait, balance and normal posture, this will, in turn, elate the psychic outlook of the patient. The value of physiotherapy is strikingly demonstrated by Doshay (1960) in a comparison between 100 patients given physiotherapy and 100 patients without this treatment. He showed that physiotherapy delays the role of progression of the rigidity and weakness.

### SURGERY

Selection of patients for surgery is based on several criteria. From the standpoint of age, the patient should be young chronologically or in terms of physiological age. It has been found that patients with unilateral disease exhibit the best results. If bilateral surgery is required, it seems wise to divide the operation into two stages with about a 3-6 month interval between the two operations. In the interim the effect of the first stage operation can be assessed.

There are several contraindications to surgery. Among these are Organic mental deterioration and old age because of the problems in rehabilitation of these patients. "Pseudo-bulbar" palsy resulting in the inability to swallow and difficulty in speech as well as akinesia are two more unfavourable conditions. Finally, patients that do not exhibit rigidity or tremor should not be subjected to surgery even if other manifestations of the disease are present, as these are the two conditions which are most dramatically relieved.

Various surgical procedures have been evolved over the years. These include resection of the premotor cortex (Area 6), ablation of parts of the caudate nucleus, sectioning of the anterior limb of the internal capsule and sectioning of various spinal

tracts. These procedures are no longer performed. In 1952 Irving S. Cooper of New York, while operating on an uncontrolled brain tumor, had to divide and sacrifice the left anterior choroidal artery. The operation was terminated and the effects of the division of this vessel were noted. There was loss of tremor and rigidity in the contralateral extremity with no obvious additive functional impairment. With further anatomical studies, the area of infarction was seen to involve the globus pallidus, the ventrolateral region of the thalamus, the ansa lenticularis as well as the cerebello-thalamic and rubro-thalamic tracts. These afferent tracts are thought to be, at least in part, responsible for the tremor and rigidity.

From this evolved the idea of producing a lesion in the globus pallidus. Subsequently it was found that the same, if not better, results were obtained if the lesion was placed in the ventrolateral nucleus of the thalamus. Also combination lesions in both areas are done and are found to be most effective.

Initially, the surgical procedures were direct, but as such they were attended by a considerable operative mortality. With the evolution of stereotaxic technique, the mortality has been reduced to its present rate of about 2-3%.

There is still a diversity of opinion dealing with the manner in which the lesion is to be effected. The problem lies in the prediction of the size of the lesion and being able to localize it in the desired area. Chemopallidectomy and chemothalamectomy (the injection of chemicals such as alcohol to produce the lesion) were practiced by Cooper for awhile. Recently he has become an advocate of Cryogenic Surgery (where the lesion is produced by the application of a cannula cooled to such temperatures as minus 190° Centigrade with liquid nitrogen).

Spiegel and Wycis advocate the use of electrical methods in the production of the lesion. Research is being done in the fields of ultrasound, Betatron radiation and radioactive isotope and pellet lesions.

The leukotome technique, as described by Claude Bertrand of Montreal, probably gives the most discrete lesion. If it is properly employed, the risk of hemorrhage is less than in most other methods. A wire loop is guided to the area of election and expanded to the

desired size. It is then rotated at about 5-10 degrees at a time, thereby producing the lesion.

The operative results are mainly directed towards the improvement or relief of the tremor and rigidity. Mild or moderate deformities are usually relieved with the relief or rigidity. Sweating crises are also abolished. No improvement is usually seen in the mask-like facies, akinesia, pseudo-bulbar palsy manifestations and oculogyric crises.

In conclusion, it must be stressed that 150 years after the classical description of Paralysis Agitans by Dr. James Parkinson, the real cause of the disease is not known. As a result there is no cure. However, the medical and surgical treatment in use today is of great benefit in the alleviation of several of the incapacitating features of the disease. Suffice it to say, there is still a pressing need in the search for the cause and prophylaxis of Parkinsonism.

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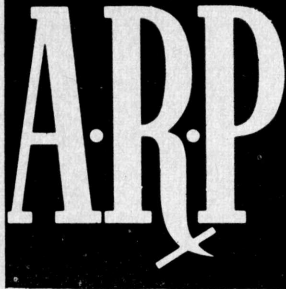
They will tailor-make a programme that meets the needs and desires of each individual client - and, most important, will continue to service and keep the programme up to date.

### REPRESENTATIVES

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Philip C. LeCocq  
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