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Postencephalitic parkinsonism

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SENIOR THESIS

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POSTENCEPHALITIC PARKINSONISM

WITH

CASE REPORTS

1931.

POSTENCEPHALITIC PARKINSONISM

This condition may be differently spoken of as mesencephalitic Parkinsonism or as chronic encephalitis lethargica exhibiting a Parkinson's syndrome. It is also known as encephalitic or postencephalitic paralysis agitata.

The Parkinsonism of postencephalitis is considered generally as a sequel of acute epidemic encephalitis. Later studies, clinically and pathologically, have given rise to the belief that a Parkinsonism following encephalitis (acute epidemic) may not be a sequel, but a symptom syndrome due to chronic inflammatory changes which are a part of the acute encephalitic entity.

For the sake of past studies of the disease, it shall be considered for the present as a sequel of acute epidemic encephalitis with a pathology of degeneration changes in the basal ganglia. Subsequent facts are to be presented in support of more recent interpretation, i.e., chronic inflammatory process.

Postencephalitic Parkinsonism is very probably an incurable disease state which may appear any time after an attack of the acute disease. From the infant of two or three weeks to the person of eighty years no one is exempt. It would seem that the teen-age and the young adult are more often afflicted.

The disease is characterized by the insidious onset of tremor, muscular rigidity and weakness, giving rise to a peculiar gait, attitude and facial expressions. Sialorrhoea and rigidity, also, particularly mark the Parkinsonism of postencephalitis. This syndrome may come on immediately after the onset of the acute epidemic encephalitis, or even after two to four years. There

may be a prodromal attack of Parkinsonian symptoms as the lethargy of the primary disease clears, followed by a quiescent period, that is ended by a typical syndrome of Parkinsonism.

Since this postencephalitic sequel embodies the necessity of a previous attack of encephalitis of the acute epidemic or lethargica type, it is reasonable to note some of the defining characteristics of that disease. It must be known, however, that very typical cases of postencephalitic Parkinsonism exist without a truly diagnosed preceding encephalitis lethargica. Von Economo in 1918 summarized and ably defined the usual symptoms of cases seen then. He noted three stages or types of the primary disease entity, namely (1) In the first there were fever, delirium and hypomania for one or more days. (2) Then followed a variable period of a few days to many weeks during which there was marked hyperactivity, often associated with insomnia, choreic or athetotic manifestations and myoclonus. (3) Finally the patients lapsed into a lethargic state during which there palsies of the cranial nerves, associated with bulbar and cerebellar symptoms.

The exceedingly great variations, however, in the individual cases render this outline of little or no value, except as a study locus. Many patients exhibit but one or two of the three stages; others present a remarkable paucity of diagnostic data until postencephalitic manifestations are seen.

No attack of acute epidemic encephalitis is too slight to be free from late effects, and the interval or the period of apparent complete recovery is often as long as three years or longer, and on the other hand there may be no interval, the acute picture merging into the chronic state imperceptively.

The first epidemic of encephalitis followed in the wake of

of the great epidemic of influenza which swept over the world. From the fact that each succeeding crop of cases varied to some extent from the preceding one, that varying sequels attend different outbreaks and that the disease has become somewhat milder, it is inferred that the causative agent has undergone some mutation or attenuation.

Thus, in defining the rather protean encephalitis lethargica, a better understanding of its sequellae can be had. The background of postencephalitic Parkinsonism becomes more distinct in the light of an interpretation of the primary disease entity.

A. W. (47) Young has noted that in thirty-nine selected cases of postencephalitic Parkinsonism, that the symptoms at the time of the original illness consisted of somnolence in twenty-six cases, diplopia in nineteen, delirium in eleven, restlessness in six, headache in five and giddiness in two. Further, that the interval between the original illness and the onset of the Parkinsonian symptoms amounted to four years and four months as a maximum; in eighteen cases no interval was noted. The average interval then worked out as 7.2 months.

In the past many cases of Parkinsonism with absence of, or poorly traced etiology, could have in all probability, been attributed to encephalitis lethargica.

Acute epidemic encephalitis or encephalitis lethargica has been known of in varying degrees of definition and clarity since the time of Galen and Hippocrates. There are records of outbreaks suggesting this disease in 1712 in Germany and in 1890 in parts of southern Europe. (To which the name Nona was given). Until Von Economo recognized it as a disease entity and described its clinical types in 1917-18, it had been known as Nona. It had been partly recognized and discussed after the pandemic of influenza of 1889-1892.

Parkinson's original description of his syndrome in 1817 was based on observation of chiefly aged patients, whose symptoms were due to senile changes, ie., idiopathic paralysis agitans.

Interest in the sequellae of acute epidemic encephalitis has been transferred from Von Economo's reports in the spring of 1917 at Vienna, on to France in 1918, England in 1918-1919 and to our own shores in 1919-1920. This marks somewhat the path of the epidemic spread of the disease, although all parts of the world were touched and left marked by cases of postencephalitic paralysis agitans. The acute epidemic encephalitis touched Austria and France in 1916, England in the spring of 1918 and was recognized in the United States about the end of 1918. Reports of sequellae appeared after about one year as previously noted.

In England and Wales there were 1470 cases of the acute disease in 1921, and in France it is estimated that there were 10,000 cases up to 1920.

In earlier reports on encephalitis lethargica, it apparently paralleled the influenza epidemics of 1917 and 1919. There has been no proven likeness or interdependence in their etiology.

Males and females are attacked in about equal numbers. This is in contradistinction to Parkinson's paralysis agitans, in which males were stricken twice as frequently as females.

In striking contrast to poliomyelitis it is rare in young children and most common in young adults. Cases met today are chiefly in the age range of twenty to thirty-five years. (1931). Cases in patients over the age of fifty are not uncommon, however.

It seems true that from forty to sixty percent of cases of acute epidemic encephalitis sooner or later manifest Parkinsonism.

Chronic encephalitic Parkinsonism not infrequently makes its first appearance with pregnancy and if present may be aggravated by it. (5) (10)

Heredity nor feeble-mindedness seem not to have any influence upon the incidence of the disease.

Although cases occur sporadically throughout the entire year, the greatest number have always occurred during the winter months. (48) Charted incidence of cases point to December and January as peak months. Head colds, grippe and influenzae are active then, also, but no proven alliance has been cited.

Encephalitis lethargica is undoubtedly infectious, but hardly contagious. The very rare occurrence of more than one case in a family can better be explained on the ground of coincidence. Nor has direct contagion been observed in hospital wards.

Cases of postencephalitic sequellae when grouped show rather high incidence of Parkinsonism, pure or mixed. Riley cites 129 cases (35) 70 of which were Parkinsonian type. Another set of 92 cases reveals 42 paralysis agitans syndromes.

(17) Hill reports 67 cases of postencephalitic sequellae with 26 behaviour disorder cases; 23 pure Parkinsonian cases; 9 a mixture of these two and 5 with serious mental retardation. Thus 32 out of 67 cases can be said to fall within the scope of this discussion.

Obviously, postencephalitic Parkinsonism depends for its existence on a previous attack of encephalitis lethargica, whether mild or severe and whether diagnosed or not.

(47) Of A. W. Young's 39 cases of proven postencephalitic Parkinsonism, all gave an account of earlier illness, diagnosed at the time as encephalitis lethargica in 29 instances, influenza in 5, rheumatic fever in 2, and appendicitis in one. One patient had no

previous illness.

(12) Eaves and Croll list ten selected cases of postencephalitic paralysis agitans, 3 of which were not diagnosed as chronic epidemic encephalitis until Parkinsonism appeared.

The frequent occurrence of the Parkinsonian syndrome coincident with the recent world-wide epidemic of encephalitis lethargica has called attention to the important role which this infection plays in the production of a special type of paralysis agitans. Unquestionably many of the cases occurring in young adults not heretofore understood were due to a mild encephalitis which, occurring several years before, had passed unnoticed.

Whether we choose to interpret postencephalitic Parkinsonism as a sequel of the acute epidemic form of encephalitis or as a manifestation of inflammatory changes which are a part of chronic stages of the disease, we may well look further into the etiology of the acute disease entity.

Several different organisms have been described and held responsible for the infections. However, no one of these has been generally accepted. In the past, encephalitis of this type has been associated with or has followed epidemics of influenza. This is without any proven parallelism of diseases or any apparent gradation of virulence of organism.

No known etiological factor seems to play a part in the incidence, communication, contagion or spread of the disease.

(13) Wechsler admits that the cause of epidemic encephalitis is unknown, but states that we may assume that the causative agent is a filtrable virus. Strauss and Lowe in this country have described

and ultramicroscopic virus which they isolated from the nose and throat washings and brains of patients who had encephalitis, and succeeded in transmitting the disease to animals thru several generations. Part of this work has been confirmed by McIntosh and Turnbull, Levaditi and Harvier, Ottolenghi, Doerr, and Kling, but no one has been able to grow the organism in cultures. In view of the fact that, as has been definitely shown, rabbits and monkeys are apt to develop, spontaneously or through infection, encephalitis closely simulating the epidemic form, animal experimentation with the virus loses much of its force.

Rosenow is of the opinion that encephalitis is caused by a strepto coccus. He has produced almost as good evidence as other experimenters, but, like them, he failed to prove his case. It was variously believed that the virus of encephalitis is closely related to that of poliomyelitis, but neither the symptomatology nor the epidemiology support this view. Nor can the poliomyelitis virus be neutralized with convalescent encephalitis serum, although Neustadter is convinced to the contrary. The relationship of encephalitis to influenza has also been discussed, but here again temporal association is interpreted as a causal link. The occurrence of "Nona" which bore some resemblance to encephalitis, after the great epidemic of influenza in 1890, is adduced as further proof of the relationship of the two diseases. If we admit the truth; we do not know. The work of Szymanowski and Zilberblast-Zand identified the relationship of encephalitis and herpes febrilis. The same cannot be said of chicken-pox, though here to the speculation is plentiful. To sum up, epidemic encephalitis seems to be clinically and epidemiologically a fairly distinct entity. It is quite possible that the virus stands in some

biologic relationship with other viruses, but proof is entirely lacking. Whether we are dealing with some mutation forms can only be guessed at for the present.

(25) In 1919, Rosenow began his study of the etiology of epidemic encephalitis. With a somewhat peculiar strepto coccus isolated from infected tonsils, teeth and nasopharynx he succeeded in producing typical symptoms and lesions of encephalitis in animals. Although the various strains isolated were of low virulence, they had considerable antigenic power. Rabbits were successfully immunized following the injection of homologous and certain heterogenous strains. On the basis of these determinations treatment by active immunization (vaccine) has been tried in the more chronic types of the disease, with a view to preventing recurrences and sequelae. No dependable data are available concerning the efficacy of treatment by vaccine. On the basis of letters received from patients and their family physicians apparently little can be expected. (Mayo Clinic's Correspondence)

After all these considerations no single etiological or contributing factor can be cited as a causative agent.

(29) We must agree with Osler and MacCrae that the cause is unknown. Whether virus or streptococcus, we have no legitimate proof on which to base a positive statement.

That a true postencephalitic Parkinson's syndrome may follow an attack of the acute entity is apparent whether the acute attack be severe or mild, diagnosed or unrecognized.

The pathology of a Parkinson's syndrome following epidemic encephalitis has given rise to considerable discussion and research. The chief cause of this, in all probability was the dearth of cases coming to autopsy. In 1925 (February) when Hohman read his paper (18) before the Johns Hopkins Medical Society, there had been but 20 cases

authentically reported, and those in groups of one or two.

Many of the early cases which became study material for the pathologist, were older persons where senile changes of degeneration and arteriosclerosis complicated the picture. Idiopathic paralysis agitans cases were also confused with the postencephalitic ones. More recently, proven cases of postencephalitic Parkinsonism have come to autopsy, which have been from the young adult group insofar as the primary infection was concerned. These give the best possible opportunity for checking clinical findings against pathological data. Early pathological interpretations of this Parkinsonism of postencephalitis were based on the findings of true paralysis agitans. Hence the belief that Parkinson's syndrome following acute epidemic encephalitis was only a sequel and represented idiopathic changes of the brain stem.

At present, the pathologists are rather generally agreed that the brain tissue changes of chronic encephalitic paralysis agitans are of inflammatory origin and represent both acute and chronic stages of activity of the original infection. There is controversy still, however, as to the exact site of the chief lesion of postencephalitic Parkinsonism. The location of the lesions of the various other manifestations of chronic encephalitis lethargica also give much material to current literature.

We now turn to the various investigations of the pathology of this disease, for their decisions and findings. They represent recent, and more remote studies of autopsy material.

(18)Hohman, working in Prof. Marburg's laboratory at the Neurological Institute of Vienna was able to confirm the most of the findings of investigators (a) that the process, is a very widespread one which involves practically every part of the central nervous system - cortex,

basal ganglia, mid brain, cerebellum, medulla, and cord, with especial localization in the basal ganglia and midbrain tegmental structures; and (b) that the process affects the parenchyma primarily and the cell changes are for the most part of the severe chronic degenerative sort with cell shrinkage, and sclerosis, on the one hand, and cell dissolution, on the other, with neuronphagia. Along with this constant picture of chronic degeneration Hohman also found more acute alteration with swelling and axonal alteration. The nerve fibers were found to show all stages from acute swelling to a complete resorption of large areas of white matter. The process may be a loss of myelin in a somewhat discontinuous fashion or a more radical loss resulting in a cribriform appearance. This formation of holes in the tissues may or may not implicate blood vessels. The glia were, on the whole, found to show surprisingly little activity. Hohman found some increased activity about the blood vessels with the production of glia fibers, but in general the glial increase was mainly cellular, manifesting itself diffusely or in the formation of small glia nodules.

Hohman further found that the mesenchymal elements gave relatively little evidence of change. There was some slight thickening and the appearance of calcareous deposits in the walls of blood vessels, which he could not account for by the age of the patients. The pia also gave evidence of irritation with minimal evidences of meningitis.

A most striking feature was the universal finding in Hohman's cases of persistent signs of acute and subacute inflammatory reaction even after months or years of the disease. In every case where diligent search was instituted, he found definite areas of round-cell infiltration with plasma cells and lymphocytes. In some cases he had to search through many sections before finding it,

but it was always present. Sometimes when infiltration was massive, he found an occasional polymorphonuclear leucocyte as proof of the relative acuteness of the process. The infiltration was noted to be almost exclusively perivascular. No part of the nervous system was thought to escape this inflammatory process from cortex to cord. He also found a striking fatty change in cells, which varied from a mere lipoid increase to a fatty dystrophy. Even taking into account the fact that the patient as a result of prolonged illness associated with difficulties in deglutition were profoundly marantic, the fatty changes were deemed out of proportion to the amount one could expect from this cause.

Hohman states that the schools of neuropathologists who have studied the akinetic hypertonicity syndrome of Parkinson have dealt with, for the most part, senile brains. These investigators, led by the Vogts and Ramsey Hunt, had been impressed with the lesions in the globus pallidus and large cells of the caudate and putamen. The French School of Pierre Marie and his pupil Tretiakoff, on the other hand, in their study of the syndrome in senile cases and in postencephalitic cases had postulated the most important lesion in the substantia nigra.

There had been practical agreement by all other authors, that in cases of encephalitic Parkinsonism described up to date, (Hohman, 1925) the substantia nigra was the most profoundly involved. Hohman thinks, however, that there is no necessity for believing that both sides may not be correct, and that the localization in the two diseases is not different. He notes that the fact that, whatever the pathways involved in the extra-pyramidal syndromes, they are constituted by a number of links, would make it easy to understand that an interruption at one of several places might result in the

same final outcome.

Having localized the pathological lesion of postencephalitic Parkinsonism, Hohman reports that in all his cases (12 reports), there was most profound and constant degeneration in the substantia nigra. The degeneration in the substantia nigra as he describes it, is manifested by the disappearance of every great number of the large pigmented cells, destruction of many of the fibers and an increase of the glial elements with pigment inclusions and pigment masses lying free in the tissues. Further, cases were found in which the pallidum was practically intact. Next in order of severity of involvement was the striatum (putamen and candatus) and then, thirdly, pallidum cerebral cortex, the mid-brain tegmental structures, (nucleus interstitialis, and nucleus ruber); than the cerebellum with the dentate, the nuclei of the medulla (dorsal vagus nucleus and ambiguus) and, finally, the cord.

Hohman's summary suggests that the correct concept of the histopathology of postencephalitic Parkinson's syndrome is found to consist in:

(a) An essentially chronic degenerative parenchymatous process with constant persistent evidences of inflammatory reaction.

(b) Widespread distribution of the lesions in every part of the central nervous system.

(c) The region of maximal and constant involvement is the substantia nigra.

Other authorities are to be quoted that we may get a conception of the protean understanding of the pathology of the acute to chronic encephalitis exhibiting Parkinson's syndrome.

(6) Edwin Bramwell, considering the pathology of encephalitis lethargica feels that the anatomical considerations demonstrate that this affection is dependent on an encephalitis which shows a

special though not invariable predilection for the brain-stem.

In discussing the symptom of tremor, Glendenning⁽⁷⁾ notes that it is met in a sequel of epidemic encephalitis. He also asserts that this dyskinesia of encephalitis is due to a lesion or lesions in the corpora striata.

⁽¹²⁾ Eaves and Croll in a study of ten cases find great destruction of nerve-cells, especially in the substantia nigra in which there was practically total disappearance of the nerve-cells and an increase of the neuroglia. They also mention perivascular round cell infiltration. Many fibers were obviously abnormal, as they presented varicosities and constrictions, the appearance being similar to the 'beaded' effect observed by Mc Alpine in the substantia nigra of other cases. They summarize by stating that the hypothalamic region of the brain in chronic epidemic encephalitis is more severely affected than any other area except the substantia nigra of the mid brain region. The change in the two regions are usually though not invariably parallel.

⁽²⁸⁾ Michels, Creighton pathologist recently (1929) found that as far as poliomyelitis and encephalitis are concerned, vascular endothelium cannot be held responsible for the production of the exudate cells met with in the lesions occurring in the central nervous system. On the contrary, the vast majority of the infiltration cells represent recently emigrated lymphocytes and large mononuclears, with a quota to be interpreted as homoplastic derivatives of previously extravasated lymphoid cells. This finding would seem to favor the presence of the acute or subacute inflammatory processes which are met in chronic encephalitic paralysis agitans.

⁽¹³⁾ F. G. Ebaugh is considering the pathology of chronic epidemic encephalitis has noted that the basal ganglia show paren-

chymatous changes to a marked degree, but that there are also severe interstitial changes consisting of intense perivascular infiltration mainly of lymphocytes and plasma cells - confined at times to the Virchow - Robin spaces, but at other times flowing over into the tissue substance. He points out that in places it can be made out, as in the region of the substantia nigra, that the tissue itself is invaded by these cellular elements. Although this opinion is quite like previous authorities cited, it adds one supporter to the side of chronic inflammatory changes in Postencephalitic Parkinsonism.

(46) Wood considers both past and present conceptions of the pathology of Parkinsonisms of chronic lethargic encephalitis. He notes that the first investigations of these cases suggested that the pathological condition was due to a selective atrophy of the motor cells of the pallidal system. Subsequent studies, he points out, have shown that the lesion is situated chiefly in the substantia nigra and that the changes in the globus pallidus are to be considered secondary to those of this region or merely incidental findings. This authority finds that still further investigations have shown that the lesions are often much more extensive, involving the internal capsule, thalamus, candate nucleus, and the frontal lobe. Under these circumstances, he feels that careful quantitative estimations of the cellular changes are important and it has been found that in three cases of postencephalitic paralysis an average decrease in the number of neurons from 57 to 87% has occurred in the substantia nigra without significant lesions in the globus pallidus. Wood mentions here that it is interesting to note at this point, that in patients with a general paralysis definite pathological changes have been found in the basal ganglia, which may be responsible for the inexpressive facies

and the fine tremors about the mouth and eyes occasionally seen in this disease. (Posten. Park.)

Wood has embodied in his considerations of the pathology of postencephalitic Parkinsonism some recent interpretations of its pathology. He states that some observers consider the classic Parkinsonism lesion a chronic progressive degenerative condition of the entire central nervous system; a position that is well supported by the clinical symptoms in advanced cases and that it is not, therefore, proper to look on paralysis agitans merely as an end-result of encephalitis. Evidently under this general title there are numerous closely related clinical conditions associated together, of which the classic paralysis agitans is due to a degenerative type of lesion involving the extra-pyramidal system, mainly the basal ganglia and especially the substantia nigra, while the lesion in the postencephalitic syndrome though degenerative in a degree, is also combined with inflammatory phenomena.

Thus, through the pathological findings of this disease we may state that the present conception of postencephalitic Parkinsonism is that its symptom syndrome is due to chronic progressive inflammatory processes in acute and subacute stages which are a part of the primary encephalitis. Hence, the standing of postencephalitic paralysis agitans as a sequel of encephalitis lethargica loses stability in the light of recent autopsy findings.

The symptomatology of idiopathic paralysis agitans and of postencephalitic Parkinsonism is nearly identical. If we exclude for the moment the rapidity of onset, which in the idiopathic type of paralysis agitans is slow and in the encephalitic type is either acute or insidious following the disease, the two conditions present parallel findings and signs.

At first tremor appears in one limb or only in the fingers or toes, or even in the thumb above or little finger. This tremor generally is rhythmic, fine, or moderate; it may be one of flexion or extension, adduction or abduction, pronation or supination, with a rate of four or five to the second. There is a certain stereotypy to it. It used to be graphically described as "pill rolling" in character, after this particular pharmaceutical maneuver. The tremor is frequently one of rest, but not always, is momentarily stopped by effort or voluntary movement, and aggravated by emotion. It generally ceases at night. It may cease spontaneously for a while, and then resume the restless oscillations. Generally, the tremor spreads up one limb, gradually involves another, possibly becomes limited to one side for a time (hemiparkinson), but finally involves the other limbs. Very characteristic when present is a tremor of the jaw. Occasionally there is tremor of the tongue. The head partakes of the general body tremor, but isolated shakings of the head, such as is seen, for instance, in multiple sclerosis, does not occur. Occasionally tremor is altogether absent; wherefore the name paralysis agitans sine agitane. This is not uncommon in the postencephalitic type. Very rarely one may observe tremor of the eyelids, more often in the encephalitic cases.

Setting in at the same time with the tremor, very often preceding it and usually constituting the most significant feature of paralysis agitans, is a change in the attitude or posture and the movements of the patient. The arm in which tremor has already set in or will ultimately develop does not swing in walking. There is no paralysis, but associated movements of the arms are impaired or lost.

Soon after the other arm fails to move automatically in walking. The face begins to lose its mobile emotional expression, its conative activity in mimicry and gesture, in laughing and crying; it becomes ironed out, waxy, mask-like (early in encephalitis) rigid. The eyelids may be only partly opened, giving rise to a sleepy expression. The head tends to bend forward, the shoulders begin to stoop, the body to flex anteriorly. The arms are extended and adducted, the forearms are somewhat flexed at the elbow, the hands are flexed, the distal phalanges extended and the fingers adducted. (It is said that very seldom there is extension of the body and extremities instead of flexion.) The gait becomes slowed the steps are short; rising from a chair or sitting down is done seemingly with deliberation. All movements are slowed, but there is especial loss of initiation. Underlying all this is a gradually increasing rigidity of the body musculature. Wigton, Boston, and Anders note this rigidity as more typical of early postencephalitic Parkinsonism than the tremor. The patient walks and moves as if in one piece. On attempting to walk there is a certain hesitation, as if the person was rooted to the ground, then festination. With this there may be rapid pulling of the body forward, propulsion, or backward, retropulsion, or sidewise, laterpulsion, so that the gait becomes accelerated. Forward movements of such a patient is characterized as giving the appearance that the patient is continually chasing his center of gravity which just does manage to keep ahead of him.

The want of associated movements and changes in posture and tonus represent the most characteristic features of paralysis agitans. This applies to the encephalitic variety especially the increased tonus or rigidity. In a study of several cases Wechsler and Brock came to the conclusion that the loss of the so-called automatic

movements and the postural disturbances represent the loss of a stell-reflex or righting reflex, as conceived by Magnus and de Kleijn. This loss of postural reflex may be observed even in patients whose bodily musculature does not show hypertonia or rigidity. Generally, however, the rigidity is marked, and one may elicit the cogwheel phenomenon on suddenly trying to overcome the rigidity of the flexed forearm. Sometimes the Souque sign can be demonstrated: Suddenly throwing the patient back while he is sitting in a chair, the lower extremities fail to kick out or extend as they do normally. Occasionally micrographia is a characteristic sign of paralysis agitans, classic or encephalitic types.

While there is not any actual paralysis voluntary motor power is lowered and performed with effort. This may be partly due to atrophy of muscle fibers although that seems to be a very slowly progressive change. It is only in the very late stages that real paralysis or paresis may be observed. The eyeballs move more slowly, the eyelids rarely blink; but if the patient is asked to look quickly to one side or the other winking may occasionally be observed (Wilson). The deep reflexes are preserved, the abdominal reflexes are present and lively, and there is no Babinski sign. This latter applies to the idiopathic type, while in the encephalitic paralysis agitans the Babinski is frequently positive. Similarly, there are no pupillary anomalies, no cranial nerve palsies, no sensory disturbances, again excepting chronic encephalitic manifestations to the contrary, and possibly arterio-sclerotic cases.

As the condition progresses the rigidity becomes more marked and the tremor widespread and intense; the patient complains of tiredness and frequently of dull but severe pains which keep him awake at night. Gradually the speech becomes monotonous, and toward the end

true dysarthria and even dysphagia may be present. There are no bladder disturbances, except toward the very end. Trophic and vasomotor disturbances are not common. A feeling of heat, perspiration cyanosis of the extremities, localized edema, and atrophy of the muscles of the hand may be observed. The mentality is normal, although the patient is often depressed, naturally by the illness. Nevertheless, in senile, arteriosclerotic, and especially in encephalitic cases, delusional paranoid trends may be observed. (3) Ebaugh especially notes behavior disorders in children which are inversely proportional in seriousness to the intensity of the Parkinsonism. Salivation or sialorrhea is frequently a distressing symptom and is almost pathognomonic of postencephalitis as noted by Wigton. (4) Excessive sweating may be occasionally encountered. Finally muscular contractures, especially of the hands and feet, develop on the basis of muscular rigidities.

Laboratory findings as generally reported and as we have found in a study of our own hospital cases are essentially negative. The spinal fluid may show an increased glucose content and also a paretic colloidal gold curve. The cell count in the spinal fluid is rarely abnormally increased in postencephalitic Parkinsonism, nor is the globulin content ever more than slightly raised.

The diagnosis of a case of postencephalitic Parkinsonism is relatively easy. First, one should attempt to get a history of the acute epidemic encephalitic attack. This history may not be typical nor will the case, in all probability, have been diagnosed as encephalitis at the time. An attempt should be made to find out if there was an illness in which suppression of urine for one to three days occurred and if there was a period of 40 to 150 hours of failure to sleep or of inability to keep awake. The patient should be asked concerning eye tremors, lid tremors, difficulty in swallowing and in speech at the time of the acute attack. This attack will have preceded

the development of the chronic syndrome by from a few days to seven or more years.

The paralysis agitans syndrome of encephalitis lethargica having established itself with tremor or more probably without, the diagnosis then turns to the features of the typical case. The syndrome may have set in acutely, have developed during the illness or have appeared after a variable latent period. Rigidity of the body and limbs will have been noted as interfering with the usual tasks. Postural disturbance and abnormal gait will be noted. There will be impairment of associated movements. An inability to laugh or cry i.e., "mask-facies" will be noted or complained of by the patient. The handwriting will be slow, irregular and the letters will be very small and cramped. Frequently an oily skin or oily hair will be a complaint in the syndrome. Drooling of saliva will often be a bitter complaint coming from the patient.

Various other postencephalitic manifestations may add to or nearly obscure the Parkinson syndrome. These will be noted further on.

(2) Boston and Anders state that in the Parkinsonism of post-encephalitis the tremor is less constant than in the idiopathic type. Also that in this hypokinetic or akinesic manifestation of chronic encephalitis the increase of the plastic tone is usually much greater than that noted in the idiopathic paralysis agitans.

(3) Wigton, in a personal statement in our Dispensary neurological clinic, states that rigidity and sialorrhoea in a Parkinsonian syndrome with reasonable evidence of a previous attack of encephalitis lethargica are pathognomonic of postencephalitic paralysis agitans while tremor characterizes the classic Parkinson's disease.

There seems to be no typical diagnostic therapeutic test.

(10)(21) Ornsteen describes three signs that aid the early diagnosis of chronic encephalitic Parkinsonism. In the first test, with patient standing at attention, though relaxed, in early Parkinsonism the hand on the affected side shows an increase in the degree of flexion of the interphalangeal joints. In the second test, a finger-spacing test, there is definite asymmetry between the two hands, with greater irregularity of the spacing of the affected hand. In the third test, when the fore-finger is touched rapidly and repeatedly to the thumb, with hands held up in front of the face, there is a definite limitation of agility in the movement and reduction in amplitude on the affected side.

Differentiation of the postencephalitic Parkinsonism from the classic paralysis agitans has been given. In the former, rigidity and sialorrhoea are in comparison to the marked tremor of the latter. Pathologically the lesion of the former is in the substantia nigra while that of the latter is a degenerative lesion in the globus pallidus.

The acute and chronic manifestations and the sequelae of epidemic encephalitis may simulate practically every basal ganglion syndrome.

Syphilis may cause a syndrome of paralysis agitans. It is ruled out of the present consideration by a history of previous acute epidemic encephalitis which is a pretty general differential necessity. The blood Wassermann test would be needed.

The cysticercus has been proven to be the cause of a unilateral paralysis agitans by Oppenheim. Ruled out by the absence of eosinophilia and a history of acute encephalitis.

The arteriosclerotic paralysis agitans of past middle age would be ruled out by the age, the rapidity of onset, the mental symptoms and history of the acute entity.

Carbon monoxide poisoning may cause symmetric softening of the leuticular nuclei (pallidus) with a resulting syndrome of paralysis agitans. This could be verified by a history of the exposure to the gas, or on the other, a history of encephalitis lethargica.

Very rarely a patch of multiple sclerosis in the basal ganglia may also give rise to a syndrome of paralysis agitans, but the usual atoxic tremor and the signs of dissemination, together with the remissions, speak for the multiple sclerosis.

The tremor seen in hysteria, more particularly traumatic neurosis, with an acute onset, emotional background, psychotic origin, and the absence of rigidity, rule out the organic entity.

(19) (39) Hoffman, Wohlwill, and Schuster report cases of frontal brain tumor with Parkinsonian Symptoms, in which the chronic encephalitis entity was ruled out by spinal fluid pressure readings and later by histological research. (39) Schuster found that in his two cases the tumor did not exist, but that there was basal ganglia changes of undetermined origin.

(2) Postencephalitic manifestations may be grouped, viz:

(a) Hypokinetic state (the present study).

(b) Hyperkinetic states such as tic movements, choreiform and athetoid, and epileptiform states, localized spasms, torsion spasms and (31) myoclonia are frequently encountered. But of greater interest are those involving the respiratory mechanism seen especially in children. This variety has only lately received special attention and was regarded at first as rare and frequently labelled hysterical in nature. These disturbances are seen as disorders of the respiratory rate (tachypnoea and bradypnoea); dysrhythmias, or disorders of the respiratory rhythm (Cheyne-Stokes breathing, breathing-holding spells, sighs, forced or noisy expirations, inversion of the inspiration-expiration ratio); respiratory

tics (yawning, hiccough, spasmodic cough, sniffing). Associated with these respiratory disturbances are exaggerated movements of the accessory muscles of respiration, face, limbs and entire body producing the most bizzare pictures (hence the frequent labelling of hysterical). Lesions here are reported as being found in the upper part of the medulla oblongata. The proportion of recovery cases from respiratory disturbances appears to be greater in the cases with a progressive Parkinsonian state than in those without the manifestation of paralysis agitans. (15)

(c) Hyperalgesia sequellae consisting of pains and dysesthesias in all parts of the body accompanied by marked emotional over-reaction.

(d) Disturbances of the sleep mechanism as insomnia, diurnal somnolence, and inversion of the day-night cycle.

(e) Dyspituitarism usually hypofunctional in character, and often of the Froelich's syndrome.

(f) Neurasthenic states and hysterical reaction.

(g) Psychotic trends suggestive of dementia praecox; behavioristic and personality changes, noted especially in children by Ebaugh (13)

(16) Hill found that in children attacked by epidemic encephalitis over 70% of the survivors show some psychological changes and more than 33% show behavior disorders.

(h) Various vegetative phenomena such as salivation, polyuria diarrhoea, vasomotor symptoms, tachycardia, asthenia, and syncopal attacks have been reported.

(l) Oculogyric Crises. (35) (23)
COURSE AND PROGNOSIS

The course of the Parkinsonism of chronic encephalitis is chronic. Cases are known to have exhibited symptoms of the syndrome, or of the syndrome entire ever since their acute encephalitis seven to ten years ago.

(47) Young studied 50 cases of paralysis agitans, 39 of which were

postencephalitis. The duration of the Parkinsonism in these post-encephalitics was seven years in the longest instance and three months in the shortest, making an average of two years and two months. These cases were reported in 1927 and the condition had not had time to exist longer, ie., there is no reason to expect recovery after 7 or even 20 years. Due to the run down condition, these cases frequently die of pneumonia or other intercurrent disease.

The course is one of slow, insidious progression of rigidity, atrophies and cranial nerve palsies. Active treatment may halt the slow advance.

The prognosis is indeed hopeless in that the chronic inflammatory changes in the basal ganglia seem to progress by acute and subacute exacerbations. Neurous and glial fibers lost in the substantia nigra show no power or regeneration of substance or function.

Kennedy has emphasized that the outlook in this condition is bad especially in children. Beginning more or less insidiously it progresses until fully developed. He states, however, that having reached this stage in the child, it appears to come to more or less of a standstill. The general weakness apparently increases, but as most of his child cases, made little effort to exercise, he deems the weakness to be due, at least in part to voluntary inactivity. This would seem to apply to our own dispensary cases which are trying to maintain active convalescence.

In spite of the marked physical impairment and showing slow reaction time noted in most cases, here and elsewhere, there is very little effect on the mentality.

Probably, the most to be hoped for in typical cases is an inhibition of the slow progression, whatever, may be the therapy employed. In the acute encephalitis lethargica, if less than 20% die, at least 60 to 70% show residual signs and symptoms, some of which are indefinitely

more tragic and incapacitating than the illness itself.

TREATMENT

One author in summing up the treatment of acute and chronic states in encephalitis has noted nearly 600 remedies. This at once bespeaks the futility of therapy, especially if specific results are expected.

The treatment is, therefore, basically symptomatic, and palliative.

(17) Clendening uses hypodermic injections of hyscine hydrobromide, and gives rather large doses. After an initial dosage of 1/150 grain twice a day and even more if no untoward effects are observed. However, in the cases so treated in our hospital and dispensary early nausea, or tolerance for the drug shortened its efficiency. He has also noted treatment with parathyroid hormone given with calcium lactate and also pituitary extract. He states that they have been given trial but tabulates no results.

(14) Scopolamine hydrobromide as well as atropine hydrobromide is servicable in the treatment of the rigidity found in postencephalitis Parkinsonism. At times scopolamine action, at times atropine action, is more advantageous in given cases. Recently, some superiority has been claimed for the use of an allied drug, stramonium, which may be given in the form of a tincture. The accepted maximum dose of lcc. (15 minims) is far too small and at times as much as 75 minims or 150 drops may be given three times a day. Occasionally, the extract of belladonna in the form of a rectal suppository 0.065 gm. (1 grain) to the dose, three times a day, results in the amelioration of the rigidity. All of these drugs should be administered in small doses at first, and the dosage gradually increased to the maximum tolerance of the patient. The tremor is often not diminished by these drugs, but it is claimed that stramonium acts better in this direction. Drs. Wigton and Bennett in our own hospital and the dispensary service employ the tincture of stramonium

in these cases. The dosage is by drops, pushing it up to where dryness of the lips and blurring of the vision are noted. One Parkinsonism case takes 160 drops three times a day, which also controls the lateral gaze (oculogyric) and tremor of the jaw (cranial nerve palsy). She was in our hospital in November 1930 for 19 days and now visits our dispensary every three weeks. Her rigidity is well controlled, and checked by an arm raising and lowering test; formerly only 10-12 times in a minute and now up to 30 times.

(6) Bulbocapnine is said to diminish tremor by producing a certain rigidity of the muscles, but results are not universally confirmed. Small doses of veratrum viride and of gelsemin at times diminish tremor.

For the salivation, both hyoscin and atropin may be used.

Warm baths and massage give momentary relief; still they are worth using.

The best kind of occupational therapy is that which is purposeful and gainful. Tremor and rigidity are lessened by jarring and jolting, hence horse back riding, open carriage drives, auto rides, and travel by rail proves beneficial.

(35) The treatment of postencephalitic paralysis agitans with typhoid vaccine maybe given, provided not too much is expected from it. Hypodermic injections of 500,000,000 to 1,000,000,000 bacteria every few days may be tried, but is less effective than smaller doses (10,000,000 to 50,000,000) intravenously every other day. The object is to get a chill after each injection. There are favorable results in a few cases, none in most. (38)

Malarial treatment of postencephalitic rigidities has also been tried. Further results are needed for comparisons.

Patients with incapacitating sequelae need institutionalization, as they are a burden to themselves and their families.

For, irritability, if present, the bromides may be given Codein may have to be given for the painful contractures, and sometimes morphin.

Later experiments have laid aside parathyroid and typhoid therapy as valueless.

General hygiene, the avoidance of irritations and of mental upsets are to be stressed.

(10) Alford recommends intravenous hypertonic glucose injections in all stages, including the chronic, and says that a majority of the Parkinsonism patients were benefited.

Lampl gives in detail his method of treating chronic cases during the entire period of treatment the patient takes daily a powder containing scopolamin and calcium lactate. In the beginning of the treatment, fever is induced by the injection of a non-specific protein. A total of six to nine febrile attacks are thus induced. After the last calcium, usually in the form of calcium Sandos, is given intravenously in doses of 10cc. Five or ten injections are given at two or three-day intervals. At the same time, the administration of atropin is begun and continued for two or three months. As regards the benefits of this method, which Lampl claims were considerable, rigidity was relieved to a greater extent than tremor.

(10) Sulfosin is a new drug which Schroeder reports upon very favorably.

(10) Pouppirt has treated 11 cases with hot baths at a temperature that maintained a fever of 104 degrees to 105 degrees F for about forty five minutes. The tremor and the associated pain in the muscles have been helped, and spasticity lessened.

Cacodylate of soda injections have been tried by many clinicians without any remarkable results.

(2) Electric baths are helpful and temporarily diminish the rigidity; general vibratory massage with the electric vibrator also tend to reduce stiffness and counteract the development of deforming postures. Faradism of the muscles may also be of value as an exercising agent for the muscles, but, probably is no more beneficial than the massage of the electric vibrator.

The Wagner-Jauregg method of treatment of metasyphilis by inoculation with the tertian type of malaria has been of some value in the Parkinsonian cases. Intraspinal injection of the serum of convalescent patients has been advised.

(25) Rosenow's serum obtained from horses, immunized by repeated injections of increasing doses of four strains of this worker's peculiar streptococcus, has been used in the treatment of acute epidemic encephalitis cases at the Mayo Clinic and elsewhere. Of 130 patients treated by Rosenow, 85 improved and 43 showed no change. Ther serum has little or no value in the chronic states.

(25) Recently Royle, working in Sydney, Australia, performed a series of experiments on goats to determine the function of the sympathetic fibers supplying the voluntary muscles, and whether that function had any relationship to the abnormal muscular condition met with in spastic paralysis. He suggests that the removal of the influence of the sympathetic innervation may be of some value in certain cases in which disability has resulted from lesions of the corpus striatum, and from disturbances of the upper motor neurone. Hunter is convinced that the relationship of sympathetic innervation of voluntary muscle to muscular tone has been conclusively proved. Sufficient corroborative work has been done to prove that these ideas are tenable, at least in part. If, as seems, probable the rigidity in the Parkinsonian types of encephalitis is due to impulses traveling over the sympathetic system, it may be feasible and ad-

visible to diminish, by surgical means, the crippling hypertonicity in cases no longer progressive. Keegan has not had occasion, as yet to try out this type of surgical therapy.

Because irradiation has been effective in many inflammatory processes (40) Prautmann and Pansdorf employed it in six cases of chronic encephalitis. The rays were applied on three fields, two temporal and one occipital. The distance was 40cm. and the quantity was 10 percent of a unit skin dose. A 0.5mm. copper filter was used. The intervals between irradiations were usually four days. The authors give the case histories of the six patients who were treated in this manner. In none of the cases was improvement noted. Accordingly the authors conclude that in cases of chronic encephalitis roentgen treatment is not effective. This indicates that inflammatory processes in the brain differ from inflammations in the other organs. The authors assume that the efficacy of roentgen rays in inflammations outside of the central nervous system are due to their mobilizing effect on the function of the supporting tissue; their inefficacy in inflammations of the brain is due to the fact that the glia cells do not react to the roentgen ray like other supporting tissues.

(37) Rusetzky studied the influence of calcium chloride and magnesium chloride on the muscle tone in ten cases of postencephalitic Parkinsonism. He found that calcium chloride definitely increases the muscle tone. Magnesium chloride decrease the muscle tone. A mixture of the two solutions causes only a slight relaxation of the muscle tone. He suggests experimenting with solutions of magnesium chloride in the treatment of sequelae of epidemic encephalitis.

Finally we must restate the fact that about all the therapeutic treatment can do is to make the patient comfortable by treating symptoms, allaying pain and irritabilities and giving stimulation when needed. We

cannot hope to cure, but we must attempt to check the insidious progression.

SUMMARY

Since the practicing physician or the specialist must concern himself chiefly with the living patient, the clinical features of postencephalitis Parkinsonism as a chronic infection are to be stressed. Truly enough the student of medicine, though he be in practice must also regard the pathological lesion of his clinical entity. In this paper, considerable authority and experimental data has been set forth to prove the most recent conception of chronic inflammatory changes and degenerative processes in the substantia nigra. Modern pathologists place the histological lesion of postencephalitic paralysis agitans in the substantia nigra.

Therefore, we turn in summary to the clinical features of the lesion which suggest a chronicity of the infection in the brain:

- (a) The gradual appearance and often steady progression of the syndrome many months or several years after an acute encephalitic attack, the patient having, perhaps, been quite well in the interim.
- (b) The insidious development of the syndrome with no history of an acute attack (ie. the infection being chronic from the first).
- (c) A certain amount of evidence of contagion long after the commencement of the illness.
- (d) The appearance and variable course in Parkinsonism of other encephalitic signs and symptoms.
- (e) The occasional recurrence of the disease in an acute form, perhaps months after the first attack.

Conclusions concerning the clinical features are:

- (a) The balance of the evidence must be regarded as being conclusive of a persistence of infection in the central nervous system in some cases of Parkinsonism. The only means of detecting this is by observing clinical signs of progress of the disease.

- (b) The evidence makes it certain that active infection may be present for a considerable length of time before any signs of progress occur.
- (c) It is therefore advisable to treat all cases of epidemic encephalitis including those of Parkinsonism, for some years at least after onset, as cases of active infection in the central nervous system.
- (d) The durable nature of a Parkinsonian syndrome once it has appeared, and the destructive signs found in the substantia nigra, indicate that the signs and symptoms at any moment must be regarded as being almost wholly due to a neuronal destruction, and that no remission of them can be expected from nonspecific or specific treatment of the active infection, even if it were successful.

CASES STUDIED OUTSIDE

- (1) Mrs. H. M. Age 35.
Diagnosis: Postencephalitic Parkinsonism.
Course and Prognosis: Chronic
Symptoms: Rigidity, tremor, sialorrhoea.
Onset of encephalitis: 1924 - slept for 18 days.
Treatment: None.
- (2) Mr. O. M. R. - Bartley, Nebraska - an uncle of the writer. Age 71.
Diagnosis: Paralysis agitans, probably postencephalitis.
Course and Prognosis: Chronic symptoms.
Symptoms: Rigidity, sialorrhoea, mask-like facies, peculiar gait, retro-antero, and lateropulsion, tremor.
Mr. R. is a great lover of the violin, and upon hearing one played of late, his tremor increases and he may cry, but no facial emotion is expressed.
Onset of encephalitis - 1923, diagnosed influenzae and pneumonia - stupor.
Treatment: Untreated.

HOSPITAL CASES REPORTED

- (1) J. J. Age 20 Female Hospital Admission November 11, 1924.
Diag: Postencephalitic syndrome.
Dismissed November 21, 1924. - Improved.
Symptoms: slow moderat tremor more marked in the left hand and arm, than in the right.
Onset: Encephalitis. January 1921. Lethargy.
Treatment: Hyoscine hydrobromide gr.1/200 bid.
- (2) Mr. F. F. Age 24 Male. December 13, 1924.
Diag: Postencephalitic Parkinson Syndrome.
Dismissed January 13, 1925 - same as on entry. (condition).
Symptoms: Tremor of arms and legs. spastic gait.
Onset: Acute encephalitis. Severe Flu - 1929 (sleeping)
Treatment: Hyscine hydrobromide. gr. 1/100 t.i.d.

- (3) J. D. G. Age 12 years. Male Hosp. Admission March 24, 1925
Diag: Postencephalitic syndrome.
Dismissed: April 14, 1925. Slow improvement.
Symptoms: Mask like facies, gait slow. Tremor of hands. Rigidity of legs.
Onset: Acute encephalitis. January 15, 1925.
Treatment: Hyoscine gr. 1/250 am. 8.00 (Colly's serum)
- (4) Mrs. S. Age 29 years. Female Hosp. Admission March 12, 1925.
Diag: Postencephalitis.
Dismissed: April 11, 1925. - Better.
Symptoms: Slow speech.
Onset Encephalitis - January 1925.
Treatment: 30cc. of mercurochrome intravenously initial. 10cc. every third day.
- (5) Mrs. L. M. Age 20 years. Female Hosp. Admission March 11, 1925.
Diag: Postencephalitis and pregnancy at term.
Dismissed: April 1, 1925. Delivered of baby. Still rigid.
Symptoms: Tremor of hands, feet and tongue since 1921 - some muscle rigidity.
Onset: Encephalitis. Flue in 1918.
Treatment: None advised.
- (6) Mr. W. C. W. Age 25 Male Hosp. Adm. Octobe 1, 1925.
Diag: Postencephalitis.
Dismissed: October 26, 1925. Not improved.
Symptoms: Inability ot sleep: mask like facies. Tremor, pill rolling at times. Lead pipe rigidity of arms. Movements slow.
Onset: Encephalitis. flu 1922. Lethargic at the time for one month
Symptoms of chronic state came on at once.
Treatment: Hyscin, which he could not tolerat.
- (7) Mr. J. C. Age 54 Male Hosp. Adm. November 11, 1927.
Diag: Postencephalitis.
Dismissed : December 20, 1927. No change.
Symptoms: Masklike face, cogwheel type of movements, rigidity, tremor.
Onset: Encephalitis. Influenza 1919. Very severe with chronic onset in 1922.
Treatment: Hyoscine hydrobromide one two hundreth of a grain b. i. d.
- (8) Mr. E. P. Age 42. Hosp. Adm. October 25, 1928.
Diag: Postencephalitis.
Dismissed: November 15, 1928. Unimproved.
Symptoms: Tremor, rigidities, slow speech, difficulty in swallowing, salivation, masklike face,
Onset: Encephalitis. Sever cold 1925. weak and tired.
Chronic symptoms: 1927. noted slowness in his work.
Treatment: Hyoscine hydrobromide one one hundredth of a grain b.i.d. 4 injections of typhoid vaccine intravenously. Chills.
- (9) Mrs. F. O. Age 36. Female June 30, 1929.
Diag: Postencephalitis. psychotic reaction.
Dismissed: July 15, 1929.
Symptoms: Parkinsonian slight tremor and rigidity. Facies fairly im- mobile. Posture and gait are.
Onset: Encephalitis.
Treatment: Sedative.

- (10) H. E. Age 18. Female Admission April 16, 1930.
Diag: Postencephalitis - residual effects.
Dismissed: Slightly improved.
Symptoms: Masklike expression. Tendencey to retropulsion. Nystagmus, tremor of tongue, tremor of left foot and knee. Pill rolling .
Atlitude of left hand.
Treatment: MXLV t.i.d. pc.
- (11) R. W. M. Age 48 Male Admission January 31.
Diag: Postencephalitic syndrome.
Symptoms: Spasticity of right leg and arm. Laggin gait. Mask facies.
Dismissed: unimproved.
Onset: Encephalitis. Influenzae in 1920.
Treatment: Tr. Stram. Gtt XL t. i. d. pc.
- (12) F. S. Age 18 Female. Admission July 1930.
Diag: Postencephalitis tremor.
Dismissed: Same date.
Symptoms: Right arm rigid. Tremor right hand speech slow.
Onset: Encephalitis - 1921. 30 hr. uncon. following mastoid operation.
Treatment: Tr. Stramonium gtt XX 5x a day.

DISPENSARY CASES STUDIED AND REPORTED

Case I. Miss I. R. Age 26.

Diag: Postencephalitic Parkinsonism with oculogyric crises.
Course and Prognosis, favorable with rigidity, eye deviation, and sialorrhoea controlled.
Symptoms: rigidity, sialorrhoea, tremor of jaw, tongue, wrists and feet, mask-like facies, spastic gait, retropulsion, oily hair and skin.
Onset of encephalitis: November 1925 with morbid insomnia for 148 hours, then lethargic state for one and one-half weeks. She noted drooping of eyelids and slowing of work in 1927.
Treatment: Tr. Stramonium 160 drops 3 times a day. Tolerance.

Case II. #41587. Mr. P. O. Age 31.

Diagnosis: Chronic encephalitis with Parkinsonian syndrome.
Course and Prognosis: Some progress under stramonium, lessened rigidity.
Symptoms: Rigidity, slow speech. Sialorrhoea, no retropulsion.
Onset of encephalitis: Influenza in 1917 while in the army; moderate He noticed rigidity and salivation early in 1925 with slowing at his work, so that he lost job after job.

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